



When smokers move out and non-smokers move in: residential thirdhand smoke pollution and exposure

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ABSTRACT

Background This study examined whether thirdhand smoke (THS) persists in smokers' homes after they move out and non-smokers move in, and whether new non-smoking residents are exposed to THS in these homes.

Methods The homes of 100 smokers and 50 non-smokers were visited before the residents moved out. Dust, surfaces, air and participants' fingers were measured for nicotine and children's urine samples were analysed for cotinine. The new residents who moved into these homes were recruited if they were non-smokers. Dust, surfaces, air and new residents' fingers were examined for nicotine in 25 former smoker and 16 former non-smoker homes. A urine sample was collected from the youngest resident.

Results Smoker homes' dust, surface and air nicotine levels decreased after the change of occupancy ($p < 0.001$); however dust and surfaces showed higher contamination levels in former smoker homes than former non-smoker homes ($p < 0.05$). Non-smoking participants' finger nicotine was higher in former smoker homes compared to former non-smoker homes ($p < 0.05$). Finger nicotine levels among non-smokers living in former smoker homes were significantly correlated with dust and surface nicotine and urine cotinine.

Conclusions These findings indicate that THS accumulates in smokers' homes and persists when smokers move out even after homes remain vacant for 2 months and are cleaned and prepared for new residents. When non-smokers move into homes formerly occupied by smokers, they encounter indoor environments with THS polluted surfaces and dust. Results suggest that non-smokers living in former smoker homes are exposed to THS in dust and on surfaces.

INTRODUCTION

Secondhand smoke (SHS) is composed of side-stream smoke emitted from the smouldering tip of a cigarette (80% to 90%) and exhaled mainstream smoke (10% to 20%). It contains a complex and dynamic mixture of more than 4000 chemical compounds in the form of gases and particulate matter, and has been classified as a human carcinogen and an indoor air pollutant.^{1–4} Immediately after emission, tobacco smoke undergoes physical and chemical changes, and the mixture of chemical compounds interacts with the environment.

The combination of tobacco smoke pollutants remaining in an indoor environment has been referred to as residual tobacco smoke pollution or,

more popularly, thirdhand smoke (THS).^{5–6} THS includes a mixture of semivolatile compounds found in SHS that have sorbed or settled on surfaces of an indoor space and are later re-emitted into the air. THS also encompasses particulate matter that has deposited and accumulated on surfaces and in dust, or has become trapped in carpets, upholstery, fabrics and other porous materials commonly found in indoor environments. THS also may contain secondary pollutants created from reactions of tobacco smoke pollutants with oxidants and other compounds in the environment.

The constituents of THS that have been identified so far include nicotine, 3-ethenylpyridine (3-EP), phenol, cresols, naphthalene, formaldehyde and tobacco-specific nitrosamines (some absent in freshly emitted tobacco smoke).^{7–8} THS exposure results from the involuntary inhalation, ingestion, or dermal uptake of THS pollutants in the air, in dust and on surfaces. It includes inhalation exposure to compounds re-emitted into the air from indoor surfaces and particles resuspended from deposits, and dermal and ingestion exposure to compounds partially derived from cigarette smoke and resulting particles that have settled, deposited and accumulated on surfaces and dust. Some of the compounds in THS are odorant and are experienced as an unpleasant, stale tobacco smoke odour on smokers, in rooms in which smoking has occurred, or on non-smokers or objects that have been in smokers' environments.

Research suggests that THS pollutants in dust, air and on surfaces in homes and cars may persist as long as months after the last known tobacco use occurred.^{9–10} Evidence collected in field and controlled laboratory studies shows that indoor environments in which tobacco is regularly smoked become reservoirs of tobacco smoke pollutants, potentially leading to the involuntary exposure of non-smokers to THS in the absence of concurrent smoking and long after smoking has taken place.^{11–13} Our previous research found that infants of smoking mothers were exposed to tobacco smoke pollutants through THS even though their mothers had strict indoor smoking bans and never smoked near their children.⁹

This study examined homes of smokers and non-smokers who were about to move out to better understand the persistence of THS during a change of occupancy. Before the first occupants moved out, we measured levels of THS in their homes and the extent to which non-smoking residents were involuntarily exposed to tobacco smoke. We revisited these homes after new non-smoking residents

moved in to determine the extent to which the homes remained polluted with THS and the extent to which new non-smoking residents were exposed to THS.

METHODS

Study design

This study relied on a quasiexperimental design, comparing non-smoker and smoker homes and their residents before (part 1) and after (part 2) a change of occupancy. For part 1, 150 participants were recruited who were planning to move out of a private residence (ie, house, condominium, or apartment) within the next month. Participants were interviewed, environmental sampling was conducted and children's urine samples were collected for analysis of cotinine concentration. For part 2, we recruited the new residents who moved into the part 1 homes. These residents were interviewed, environmental sampling was conducted, and urine samples were collected from the youngest residents.

Inclusion criteria

For part 1, residents were eligible to participate if they were age 18 or older, spoke English, had lived in their current home for at least 6 months, reported that everyone in their household was planning to move within the next month and also that (to the best of their knowledge) the home would be reoccupied after they moved out. In addition, they met criteria for classification as either a 'smoker home' (n=100) or a 'non-smoker home' (n=50). Smoker homes were those in which residents had smoked indoors during at least 5 of the past 6 months, including the current and most recent month, and had smoked a minimum of seven cigarettes per week inside the home during the week prior to study measures. Non-smoker homes were those where no smokers had lived and no visitors had smoked indoors during the past 6 months, and where a *target child* (under age 12, not breastfeeding) who had not been exposed to any SHS in the past month resided full time. For smoker homes, a target child was selected if there was a resident under age 12 who lived in the home full time and was not breastfeeding. Six smoker homes that were measured in part 1 were disqualified because residents smoked fewer than seven cigarettes inside the home during the week preceding study measures, and their data were not included in the following analyses.

For part 2, new residents were eligible if they were age 18 or older, spoke English or Spanish, had not smoked any cigarettes since they moved into the home and if no visitors had smoked inside the home since the new residents moved in. The youngest resident who lived in the home full time and was not breastfeeding was designated the target child.

Participants

Participants received US\$100–US\$200 for completing an interview, providing urine samples and allowing the collection of environmental samples. All procedures were approved by the San Diego State University Institutional Review Board.

Part 1 recruitment

For part 1, participants were recruited through advertisements in local print (n=82) and electronic news media (n=4), San Diego County Women, Infants, and Children Supplemental Food and Nutrition Program (WIC) offices (n=52), referrals from friends, relatives, or coworkers (n=4), flyers distributed in military housing (n=1) and postcard mailers to a commercially available list of smokers (n=1).

Part 2 recruitment

After part 1 residents confirmed they had moved, research assistants delivered or mailed up to 12 recruitment letters and flyers to the same homes, requesting that new residents contact the research office by telephone for eligibility screening. Homes were visited at varied times of the day on weekdays and weekends, and screening was conducted in person if the new residents were present and agreed. If a home was still vacant and we were unable to gain access through the property manager or owner (6%) or new residents had not responded 6 months after part 1 measures were completed (12%), the home was disqualified from part 2. New residents of 26% of homes were disqualified due to smoking, the part 1 residents did not move from 18% of homes, the new residents declined to participate in 6% of homes, we were unable to schedule measures with 2%, and 1% of homes were completely renovated.

Part 2 measures were completed for 25 former smoker homes and 16 former non-smoker homes. Seven of these homes (four non-smoker and three smoker) were measured while vacant, with permission from the property manager or owner, as no new residents had moved in after 3 months. There were no statistically significant differences in air, surface, finger, or dust nicotine contamination for homes that were measured while vacant versus occupied (all $p>0.23$).

There were no significant differences for any part 1 measures of home contamination or target children's SHS exposure between smoker homes that did or did not participate in part 2. Compared to non-smoker homes that did not participate in part 2, those that participated exhibited higher mean nicotine concentration levels in living room air ($p=0.031$) and on residents' fingers ($p=0.014$) at part 1.

Participant and home characteristics

See table 1 for participant and home characteristics at part 1 and part 2.

Table 1 Participant and home characteristics

Characteristic	Part 1		Part 2	
	Non-smoker homes N = 50	Smoker homes N = 94	Non-smoker homes N = 16	Smoker homes N = 25
Participant				
Female	86%	75%	85%	76%
Age, years* †	33	38	26	27
Race/ethnicity				
White	38%	37%	46%	38%
Hispanic	28%	12%	46%	19%
Black	24%	31%	8%	29%
Other	10%	20%	0%	14%
Target child				
Female	44%	44%	29%	0%
Age, years*	4.0	4.3	2.9	3.4
Race/ethnicity				
White	24%	19%	29%	14%
Hispanic	26%	25%	43%	57%
Black	22%	31%	0%	29%
Other	28%	25%	29%	0%
Number of residents living in home* †	4	2	3	2
Square footage of home* †	767	591	764	666
Household income* †	US\$37220	US\$25500	US\$32000	US\$34000

*Median.

† $p<0.01$ (two sided) part 1 smoker versus non-smoker homes.

Measures

Pairs of research assistants visited participants' homes to conduct in-person interviews and collect environmental samples. Interviews were primarily conducted with the eligible resident who agreed to participate, however questions about smoking inside the home and SHS exposure of non-smokers were asked of each smoker who agreed to participate. If a smoker resident was unavailable, participants provided proxy reports. In smoker homes, samples were collected in the living room and one bedroom (the target child's or a non-smoker's, or the smoker's bedroom in homes with no non-smokers). In non-smoker homes, samples were collected in the living room only.

Indoor smoking and SHS exposure

At each interview, primary interview participants and other parents (spouses or partners living in the home) reported their smoking and the target child's SHS exposure on typical work and non-work days (or week and weekend days if participants didn't work outside the home) during the past 7 days, including exposure from other residents and visitors, and outside of the home including in the car. SHS exposure was defined as the number of cigarettes smoked while the target child was in the same indoor room or car. The target child's weekly exposure to cigarettes in the home and 'total exposure' to all cigarettes in the home, car and elsewhere were computed. These measures have shown acceptable test—retest reliability and validity in relation to cotinine and nicotine assays in our past studies.^{14–16}

To examine the test—retest reliability of our measures, selected smoking and SHS exposure questions were asked by telephone again for 32 part 1 respondents who agreed to participate 24–72 h following their home interview. Pearson correlation coefficients for participants' reports at the part 1 interview and 24–72 h retest were $r=0.95$ for participants' smoking rate inside the home, $r=0.92$ for other parents' smoking rate inside the home, $r=0.90$ for visitors' smoking rate inside the home, $r=0.97$ for participants' overall smoking rate, $r=0.89$ for other parents' overall smoking rate and $r=0.98$ for children's SHS exposure from visitors inside the home. Validity correlations between part 1 outcome variables were $r=0.61$ for living room surface nicotine with dust nicotine, $r=0.54$ for living room surface nicotine with air nicotine, $r=0.63$ for living room dust nicotine with air nicotine and $r=0.89$ for urine cotinine with reported indoor smoking.

Surface nicotine in living room and bedroom

Prescreened cotton wipes (cosmetic 100% cotton facial wipes) were wetted with 1.5 ml of 1% ascorbic acid and wiped over a 100 cm² area, typically a wooden door or cabinet unlikely to be frequently cleaned.⁹ Nicotine-d₄ was added as an internal standard, then 0.1% aqueous formic acid was added, mixed, and the wipe removed from solution. Then, 1 M KOH (aqueous) was added, vortexed, and 2 ml was transferred to a precleaned solid phase extraction (SPE) column (Isolute C8, International Sorbent Technologies, Hengoed, UK). The column was washed, then the nicotine eluted with acetonitrile/pH4 20 mM ammonium acetate buffer into an amber autosampler vial. Samples were stored at -20°C in the dark until analysis. For part 2, samples were collected in a 100 cm² area directly adjacent to the area sampled in part 1.

Finger nicotine concentration

A wipe sample of the participant's dominant hand index finger was taken at the home visit. In part 1, this was the smoker or non-smoker about to move out. In part 2, this was the new

non-smoking resident. Wipes were prepared and processed as above.

Dust nicotine in living room and bedroom

Dust samples were collected from a 1 m by 1 m area (or from a larger area if needed to collect approximately one-quarter of an inch of dust in a collection bottle) with a High-Volume-Small Surface-Sampler (HVS4, CS3 Inc., Venice, Florida, USA) into methanol-washed amber bottles. Samples were transported cooled, then were weighed and sieved with a stainless steel, methanol-washed, 150 µm mesh sieve to remove large debris such as pet hairs, and weighed again. Samples were stored at -20°C until analysis. For analysis of nicotine, 50 mg of sieved dust were used. Samples were processed and analysed in a manner similar to wipe samples except the inlet end of the SPE columns were coupled to a filter cartridge containing a medium porosity filter paper to retain the particulate. Dust concentrations are reported as µg/g (concentration) as well as µg/m² (loading). For part 2, samples were collected directly adjacent to the area sampled in part 1.

Air nicotine in living room and bedroom

A passive diffusion monitor badge was used, consisting of a modified 37 mm 3M Organic Vapour Monitor (3-M, St. Paul, Minnesota, USA) with a glass fibre filter coated with a glycerol/phosphoric acid mixture (filter collector was modified from Kuusimäki *et al.*).¹⁷ The sampling rate was empirically determined to be 18.4 ml/min. At the home visit, research assistants taped monitors to a wall about 1.5 m (5 feet) above the ground, out of children's reach and away from windows, corners, doors and ashtrays. Inactive monitors were placed in all other rooms of the study homes to enhance reporting accuracy. Research assistants visited the homes 7 days later to retrieve the monitors, and the time in minutes the badge was placed were recorded. Extraction took place as for wipes, as discussed above. For part 2 measures, air monitors were placed in the same exact locations as for part 1.

Urine cotinine concentration

At each part 1 and part 2 home visit, a urine sample was collected from the target child. Samples were obtained using a standard collection cup for older children and adults, or by placing two pieces of a 12.7 cm by 22.9 cm (5 inch by 9 inch) pad (cut into four pieces) in the diaper (TenderSorb Wet-Pruf Abdominal Pads, Kendall # 9190, Kendall, Covidien, Mansfield, Massachusetts, USA). Wet pads were packed into separate sterile 20 ml syringes and expressed into sterile 5 ml plastic phials.

Laboratory analyses

Samples were analysed by D Chatfield at San Diego State University. The method of analysis was by liquid chromatography tandem mass spectrometry (LC-MS-MS) using electrospray ionisation (ESI; Thermo Fisher Scientific, Waltham, Massachusetts, USA). Nicotine was quantified against the internal standard, nicotine-d₄ (CDN Isotopes Inc., Pointe-Claire, Quebec, Canada). The final extracts after sample preparation were injected (1–5 µl) onto a LC silica column (Hypersil, Thermo Fisher Scientific, Waltham, Massachusetts, USA) and separated in hydrophilic interaction chromatography (HILIC) mode using acetonitrile:pH4 20 mM acetate buffer of 70:30 (v/v) at 150 µl/min. Selected reaction monitoring of the MS-MS transitions at 16V collision-induced dissociation (CID) of m/z 163.2 to m/z 117.1 and 130.1 and m/z 167.1 to m/z 121.1 and m/z 134.1 was used for nicotine and the deuterated analogue, respectively. Standard calibration curves were linear over the

concentration range studied, 0.1 to 1000 ng/ml with $R^2=0.997$. Limits of detection were approximately 0.1 μg nicotine/ m^2 for wipe samples, 0.2 μg nicotine/g dust and 0.0053 $\mu\text{g}/\text{m}^3$ in air for a 7 day exposure. The detection limit for urine cotinine was approximately 0.05 ng/ml.

Statistical analyses

Results are presented for study homes that had part 1 and part 2 measures ($N=41$), and for all part 1 homes ($N=144$). To control for non-normal distributions and heterogeneous error variances, we subjected response variables to logarithmic transformation and report geometric means. We examined differences in THS pollution and exposure between smoker and non-smoker homes before (part 1) and after (part 2) the change of occupancy using two-sample t tests with unequal variances. Mean changes in THS pollution from part 1 to part 2 were examined with paired t tests. Quantile and Tobit regression analyses for left-censored data were used to explore the contribution of dust, surface and air contamination to participants' finger nicotine and urine cotinine levels. Quantile regression models were examined for 50th and 75th percentiles. Analyses were conducted with Stata IC V. 10.0 and SPSS V. 15.0 statistical software.^{18, 19} The type I error rate was set at $\alpha=0.05$, and comparisons between non-smoker and smoker homes were conducted based on directional (one-tailed) hypotheses regarding differences in THS pollution and exposure between non-smoker and smoker homes and between non-smokers residing in former smoker and non-smoker homes. All other hypotheses were tested in a non-directional (two-tailed) fashion.

To investigate how well environmental and biological markers of THS pollution and exposure discriminate between smoker and non-smoker environments, we determined cut-off values for urine cotinine and finger, air, dust and surface nicotine levels that yield the largest per cent difference between correctly identified smoker homes (ie, hits) and incorrectly identified non-smoker homes (ie, false alarms).

RESULTS

Tobacco smoke pollution in homes

Tobacco smoke pollution in smoker and non-smoker homes before the change of occupancy (part 1)

Table 2 shows the geometric means and 95% CIs for the number of cigarettes smoked indoors at home, as well as for nicotine levels in the air, dust and on the surfaces of smoker and non-smoker homes (ie, part 1). Data are reported for all smoker and non-smoker homes, and also separately for the subset of homes for which part 1 and part 2 data were available.

In part 1 smoker homes, participants reported that an average of 60 cigarettes/week were smoked indoors; 52% had 1 smoking resident, 44% had 2 and 4% had 5 smoking residents. In part 1 non-smoker homes, participants reported that no residents had smoked at all in the past 6 months, and that no cigarettes were smoked inside the home for at least 6 months prior to study measures.

Replicating findings from our earlier research, smoker homes showed significantly elevated levels (all $p<0.001$) of nicotine in the air, in household dust and on surfaces. Air nicotine concentrations were 35–98 times higher than those found in non-smoker homes. The 2 major reservoirs for THS in smoker homes, dust and surfaces, showed nicotine levels approximately 12–21 and 30–150 times higher, respectively, than the reference levels in non-smoker homes. Note that nicotine concentrations in dust were approximately equivalent in living rooms and bedrooms.

Table 2 Tobacco smoke pollution in smoker and non-smoker homes before (part 1) and after (part 2) the change of occupancy

	Part 1: original occupants, N mean (95% CI)	Part 2: new non-smoker occupants, N mean (95% CI)
Indoor smoking, cigarettes/week		
All non-smoker homes	50 0	16 0
All smoker homes	94 60.17 (49.60 to 72.96)	25 0
Same non-smoker homes	16 0	16 0
Same smoker homes	25 68.57 (46.94 to 99.94)	25 0
Air nicotine, $\mu\text{g}/\text{m}^3$		
Living room:		
All non-smoker homes	50 0.02 (0.01 to 0.03)	16 0.14 (0.00 to 0.34)
All smoker homes	81* 1.86 (1.38 to 2.44)	23 0.20 (0.07 to 0.34)
Same non-smoker homes	16 0.04 (0.00 to 0.07)	16 0.14 (0.00 to 0.34)
Same smoker homes	19 1.96 (1.01 to 3.34)	19 0.23 (0.07 to 0.41)
Bedroom:		
All smoker homes	74† 1.44 (1.00 to 1.97)	22 0.12 (0.04 to 0.19)
Same smoker homes	19 1.55 (0.75 to 2.73)	19 0.13 (0.05 to 0.22)
Surface nicotine, $\mu\text{g}/\text{m}^2$		
Living room:		
All non-smoker homes	50 1.6 (0.8 to 3.0)	16 1.5‡ (0.4 to 3.7)
All smoker homes	94 98.7 (61.2 to 158.6)	24 10.0‡ (3.1 to 28.6)
Same non-smoker homes	16 1.4 (0.3 to 3.6)	16 1.5‡ (0.4 to 3.7)
Same smoker homes	24 211.7 (85.2 to 523.9)	24 10.0‡ (3.1 to 28.6)
Bedroom:		
All smoker homes	87 50.1 (29.4 to 84.7)	23 7.5 (1.9 to 24.4)
Same smoker homes	23 66.1 (24.8 to 173.5)	23 7.5 (1.9 to 24.4)
Dust nicotine, $\mu\text{g}/\text{g}$		
Living room:		
All non-smoker homes	50 2.9 (1.1 to 4.0)	16 2.3§ (1.0 to 4.4)
All smoker homes	93 39.6 (30.0 to 52.2)	25 10.9§ (6.4 to 18.2)
Same non-smoker homes	16 2.7 (1.1 to 5.3)	16 2.3§ (1.0 to 4.4)
Same smoker homes	25 47.6 (26.6 to 84.7)	25 10.9§ (6.4 to 18.2)
Bedroom:		
All smoker homes	76 30.7 (22.2 to 42.2)	23 11.0 (6.0 to 19.6)
Same smoker homes	23 40.4 (23.1 to 70.2)	23 11.0 (6.0 to 19.6)
Dust nicotine, $\mu\text{g}/\text{m}^2$		
Living room:		
All non-smoker homes	49 3.6 (2.2 to 5.6)	16 3.1 (0.8 to 8.3)
All smoker homes	92 58.8 (40.9 to 84.3)	25 7.6 (3.6 to 15.3)
Same non-smoker homes	15 4.2 (1.3 to 10.6)	15 3.4 (0.8 to 9.6)
Same smoker homes	25 76.2 (33.1 to 173.8)	25 7.6 (3.6 to 15.3)
Bedroom:		
All smoker homes	73 51.0 (34.7 to 74.8)	21 7.3 (3.0 to 16.3)
Same smoker homes	21 75.4 (36.7 to 153.9)	21 7.3 (3.0 to 16.3)

*Part 1 living room air monitors were not placed in nine smoker homes because residents were moving in <7 days, and air monitors were not returned by residents of four smoker homes.

†Part 1 bedroom air monitors were not placed in nine smoker homes because residents were moving in <7 days, or in six studio apartments, and were not returned by residents of five smoker homes.

‡ $p=0.0059$ (directional) part 2 non-smoker versus former smoker homes.

§ $p=0.0002$ (directional) part 2 non-smoker versus former smoker homes.

Change in tobacco smoke pollution when smokers moved out and non-smokers moved in (part 1 vs part 2)

Of the homes that participated in part 2, smoker homes were vacant a median of 62 days and non-smoker homes were vacant a median of 34 days after part 1 residents moved out. Part 2 measures were obtained a median of 33 days after new residents moved into former smoker homes, and a median of 32 days after new residents moved into former non-smoker homes. Smoker homes were more likely than non-smoker homes to get new flooring in the bedroom, kitchen and living room, and were more likely to have the kitchen painted (as reported by part 2 participants; all $\chi^2 p<0.05$).

Table 2 shows that tobacco pollutants as measured by nicotine concentrations significantly decreased when smokers moved out (part 1) and new non-smoking residents moved into the same homes (part 2) (all $p < 0.001$). The largest reductions in smoker homes were observed for nicotine on living room surfaces (95% reduction), and the smallest for dust nicotine concentration (i.e., nicotine per gram of dust) in living rooms and bedrooms (75% reduction). For former non-smoker homes, nicotine levels stayed approximately equivalent to their original levels, suggesting stable levels of background nicotine pollution.

Thirdhand smoke pollution in former smoker homes compared to former non-smoker homes (part 2)

Table 2 shows results comparing THS levels in homes of non-smokers (part 2) who moved into former smoker and non-smoker homes. Homes formerly occupied by smokers showed significantly higher levels of nicotine on living room surfaces (1.52 vs 10.04 $\mu\text{g}/\text{m}^2$, $p = 0.0059$) and in living room dust (2.27 vs 10.94 $\mu\text{g}/\text{g}$, $p = 0.0002$). On average, nicotine contamination was seven times higher on living room surfaces and five times higher in living room dust in former smoker homes compared to former non-smoker homes. Dust nicotine loadings (ie, nicotine per m^2) were higher in smoker as compared to non-smoker homes, but this elevation was not as marked as for dust concentration and was not statistically significant ($p = 0.07$).

Exposure to tobacco smoke pollutants in homes

SHS and THS exposure in smoker and non-smoker homes before change of occupancy (part 1)

Table 3 shows urine cotinine and finger nicotine levels, and reported measures of involuntary exposure to tobacco smoke among the target children in smoker and non-smoker homes. Data are reported for participants in all non-smoker and smoker homes, and also separately for the subset of participants in homes for which part 1 and part 2 data were available.

Children living in homes with active smokers were reportedly exposed to an average of 14 cigarettes/week at home. No exposure was reported for children living in non-smoker homes. Geometric mean urine cotinine levels among children in smoker homes were 5.42 ng/ml, compared to 0.15 ng/ml among children

in non-smoker homes. Finger nicotine levels were, on average, 660.21 ng/wipe among smokers in smoker homes, compared to 0.47 ng/wipe among non-smokers in non-smoker homes. Part 1 smoker and non-smoker homes differed significantly on urine cotinine ($p = 0.002$) and finger nicotine ($p < 0.001$).

Residents' exposure to tobacco smoke pollutants after the change of occupancy (part 1 vs part 2)

Table 3 shows that the geometric mean urine cotinine concentrations of new non-smoking youngest residents in former smoker homes (part 2) were lower than the levels exhibited by the children who previously resided in these same homes ($p < 0.05$ all homes). New residents' finger nicotine levels were also lower in part 2 smoker homes ($p < 0.001$). In non-smoker homes, there were no differences in mean urine cotinine levels ($p > 0.20$) or finger nicotine levels ($p > 0.20$) between part 1 and part 2.

THS exposure among non-smokers occupying former smoker and non-smoker homes (part 2)

Table 3 shows urine cotinine and finger nicotine levels among non-smokers who moved into homes formerly occupied by smokers and non-smokers. Nicotine levels found on the index fingers of non-smokers residing in former smoker homes were 7–8 times higher than for those residing in former non-smoker homes (same homes: 5.85 vs 0.75 ng/wipe, $p = 0.0339$; all homes: 5.19 vs 0.75 ng/wipe, $p = 0.0402$). Urine cotinine levels were 3–5 times higher among the youngest occupants of former smoker homes compared to former non-smoker homes (same homes: 0.61 vs 0.13 ng/ml, $p = 0.1176$; all homes: 0.13 vs 0.45 ng/ml, $p = 0.0344$).

Reported tobacco odour and discolouration

The new residents of four former smoker homes reported tobacco odour in their homes, and the new residents of one additional former smoker home reported tobacco discolouration (yellow spots on the living room and dining room ceilings). No residents of former non-smoker homes reported tobacco odour or discolouration.

Exploring the contribution of dust, surface and air contamination to overall thirdhand smoke exposure

To explore how THS in dust, air and on surfaces may contribute to non-smokers' overall exposure to THS, we first examined the associations between finger nicotine levels and THS on surfaces and in dust. Tobit regression models of finger nicotine levels showed statistically significant associations with surface nicotine levels (pseudo $R^2 = 0.08$, $p = 0.037$) and dust nicotine levels (pseudo $R^2 = 0.11$, $p = 0.009$). When entered jointly, surface and dust nicotine yielded a statistically significant model fit (pseudo $R^2 = 0.13$, $p = 0.025$).

We then examined the associations between urine cotinine levels and THS, as measured by dust and surface nicotine levels. Using Tobit regression models, urine cotinine showed statistically significant associations with dust nicotine (pseudo $R^2 = 0.18$, $p = 0.035$) and surface nicotine (pseudo $R^2 = 0.21$, $p = 0.027$). In a Tobit regression model, dust and surface nicotine levels jointly produced a statistically significant model fit (pseudo $R^2 = 0.29$, $p = 0.031$).

Lastly, we examined the association between urine cotinine and finger nicotine. Tobit (pseudo $R^2 = 0.69$, $p < 0.001$) and quantile regression (pseudo $R^2 = 0.28$, $p < 0.001$) models, as well as Pearson ($r = 0.70$, $p < 0.001$) and Spearman ($r = 0.67$, $p < 0.001$) correlations showed a strong association between nicotine on part 2 residents' fingers and their urine cotinine levels.

Table 3 Exposure to tobacco smoke pollution in smoker and non-smoker homes before (part 1) and after (part 2) occupants move

	Part 1: original occupants, N mean (95% CI)	Part 2: new non-smoker occupants, N mean (95% CI)
Urine cotinine, ng/ml		
All non-smoker homes	50 0.15 (0.09 to 0.21)	13 0.13* (0.00 to 0.27)
All smoker homes	31 5.42 (3.88 to 7.46)	20 0.45* (0.13 to 0.86)
Same non-smoker homes	13 0.14 (0.00 to 0.29)	13 0.13† (0.00 to 0.27)
Same smoker homes	5 3.66 (1.49 to 7.70)	5 0.61‡ (0.00 to 2.26)
Finger nicotine, ng/wipe		
All non-smoker homes	50 0.47 (0.04 to 1.08)	11 0.75‡ (0.00 to 3.06)
All smoker homes	91 660.21 (441.58 to 986.84)	19 5.19‡ (0.81 to 20.12)
Same non-smoker homes	11 1.35 (0.00 to 8.02)	11 0.75§ (0.00 to 3.06)
Same smoker homes	18 803.85 (387.84 to 1664.96)	18 5.85§ (0.90 to 23.72)
Reported exposure, cigarettes/week		
All non-smoker homes	50 0	12 0
All smoker homes	31 14.19 (7.16 to 27.28)	20 0.40 (0.00 to 1.15)
Same non-smoker homes	12 0	12 0
Same smoker homes	5 18.49 (0.10 to 343.13)	5 1.52 (0.00 to 20.01)

* $p = 0.0344$ (one sided) part 2 smoker versus part 2 non-smoker homes.

† $p = 0.1176$ (one sided) part 2 smoker versus part 2 non-smoker homes.

‡ $p = 0.0402$ (one sided) part 2 smoker versus part 2 non-smoker homes.

§ $p = 0.0339$ (one sided) part 2 smoker versus part 2 non-smoker homes.

When urine cotinine was regressed on finger nicotine, surface nicotine and dust nicotine as explanatory variables, only finger nicotine level was statistically significant ($p=0.001$; dust and surface nicotine, both $p>0.20$). This suggests that finger nicotine in non-smokers may be a robust measure of THS on polluted surfaces and dust.

In part 2 homes, air nicotine levels were not associated with urine cotinine or finger nicotine levels. Models that included reported SHS exposure and reported number of days participants smelled smoke drifting inside the home were not statistically significant, nor were bivariate correlations of these variables with urine cotinine.

Cut-off levels discriminating between smoker and non-smoker homes

Table 4 shows the percentages of smoker and non-smoker homes with above threshold levels of air, surface and dust nicotine, urine cotinine and finger nicotine. These findings indicate that dust nicotine best discriminates between smoker and non-smoker homes. Specifically, 84% of smoker homes' living rooms still exhibited above threshold levels of nicotine in dust when non-smokers moved in (part 2), compared to 90% when smokers still lived there (part 1) and 19% of part 2 non-smoker homes. Similarly, 54% of the former smoker homes' living rooms (part 2) had surfaces above threshold levels, compared to 19% of

Table 4 Percentage of homes with detectable levels of cotinine in non-smoker's urine, nicotine on non-smoker's fingers and nicotine in house household dust, air and surfaces

	Cut-off*	Part 1: original occupants, percentage \geq cut-off value	Part 2: new non-smoker occupants, percentage \geq cut-off value
Urine cotinine	0.30 ng/ml		
Non-smoker homes		10	8
Smoker homes		97	40
Finger nicotine	50.0 ng/wipe†		
Non-smoker homes		2	0
Smoker homes		93	35
Air nicotine living room	0.10 $\mu\text{g}/\text{m}^3$		
Non-smoker homes		6	25
Smoker homes		90	44
Air nicotine bedroom	0.10 $\mu\text{g}/\text{m}^3$		
Non-smoker homes		NA	NA
Smoker homes		78	39
Surface nicotine living room	5.0 $\mu\text{g}/\text{m}^2$		
Non-smoker homes		16	19
Smoker homes		86	54
Surface nicotine bedroom	5.0 $\mu\text{g}/\text{m}^2$		
Non-smoker homes		NA	NA
Smoker homes		75	44
Dust nicotine living room	5.0 $\mu\text{g}/\text{g}$		
Non-smoker homes		28	19
Smoker homes		90	84
Dust nicotine bedroom	5.0 $\mu\text{g}/\text{g}$		
Non-smoker homes		NA	NA
Smoker homes		84	70
Dust nicotine living room	5.0 $\mu\text{g}/\text{m}^2$		
Non-smoker homes		31	25
Smoker homes		91	60
Dust nicotine bedroom	5.0 $\mu\text{g}/\text{m}^2$		
Non-smoker homes		NA	NA
Smoker homes		64	52

*Cut-offs were established to discriminate between smoker and non-smoker homes.

†Wipes were 0.1 m \times 0.1 m; 50 ng/wipe is equivalent to 5.0 $\mu\text{g}/\text{m}^2$.

former non-smoker homes. Among the part 2 occupants of smoker homes (all non-smokers), 40% had above threshold levels of THS exposure (urine cotinine) and 35% had above threshold levels of finger nicotine. This compares to 8% and 0%, respectively, among occupants of part 2 non-smoker homes.

DISCUSSION

This was the first study to examine residential THS pollution and exposure after smokers moved out and non-smokers moved in. Findings replicate those from an earlier study of smoking mothers with infants,⁹ showing that smoker homes have become significant reservoirs of THS pollutants at the time smokers prepare to move out.

Even 2 months after smokers moved out and non-smokers moved in, nicotine in dust and on surfaces still exceeded threshold levels in 84% and 54% of homes, respectively. Even though mean levels of nicotine significantly declined when non-smokers moved into former smoker homes, dust and surface nicotine levels were still significantly higher than in non-smoker homes that underwent a similar change of occupancy. This is particularly notable because these homes were vacant for an average of 2 months during the change of occupancy, and because all of these homes underwent cleaning and many were repainted and had carpets replaced before new occupants moved in (especially smoker homes). In summary, these findings demonstrate that smokers leave behind a legacy of THS in the dust and on the surfaces of their homes that persists over weeks and months.

Non-smokers moving into former smoker homes are exposed to the THS left in dust and on surfaces by the former smoker occupants. This is shown by increased finger nicotine and urine cotinine levels among non-smokers living in former smoker homes. This exposure pathway is further supported by significant correlations of dust and surface nicotine levels with finger nicotine levels, and between finger nicotine and urine cotinine levels. Air nicotine levels were not associated with biological exposure measures. This suggests that the main reservoirs of exposure to THS are in dust and surfaces. Air concentrations of THS may remain low relative to dust and surfaces because airborne THS is more rapidly transported outside the home through passive air exchanges and active ventilation.

It should be noted that smoker homes in this study were more expensive to prepare for new occupants than non-smoker homes. Smoker homes remained vacant for on average an extra month, and they were more likely to get new flooring in the bedroom, kitchen and living room and to have the kitchen painted. These findings parallel results from our study of the resale value of used cars sold by smokers, showing that their cars lost 7% to 9% in value relative to non-smoker cars of equivalent age, make, model and condition.²⁰ These results suggest economic consequences for owners, sellers and renters of cars and homes. Theoretically, such economic penalties, if communicated to the community, create incentives to reduce smoking as well as THS contamination of cars and homes.²¹

Limitations

Markers of THS have not been comprehensively studied, and there remain important questions regarding the extent to which nicotine represents other chemical compounds known and suspected in THS. Similarly, it is unclear how well cotinine represents biological exposure to THS compounds beyond nicotine, such as tobacco-specific nitrosamines.^{7 8} This study was not designed to investigate health outcomes of exposure to THS. Future research on surface chemistry and biological mechanisms, as well as behavioural studies of exposure

pathways are needed to better understand the nature of THS, associated health outcomes, and the behavioural and economic factors influencing THS pollution and exposure in the field.

The subject matter of this field study precluded a randomised trial, creating some ambiguity about the causal origins of the THS pollutants detected in part 1 homes. The fact that the THS marker is tobacco specific (ie, nicotine) and strongly associated with reported smoking behaviour of part 1 occupants makes this validity concern implausible. The voluntary nature of participation in this study, typical vacancy rates in the housing market, participation refusals and our efforts to exclude from part 2 participants who were exposed to SHS decreased sample sizes for part 2 analyses. This lowered the statistical power of our hypothesis tests and could have contributed to differential attrition. To address these issues, we report findings based on data collected from all eligible homes and from homes for which part 1 and part 2 data were available. We also report geometric means with 95% CIs and exact p values of hypothesis tests to allow the reader to evaluate their statistical and practical significance, given the relatively small sample sizes. We examined and found no plausible evidence for differential attrition.

Conclusions

Homes remain reservoirs of tobacco smoke pollutants after smokers move out, creating a source for involuntary exposure to non-smokers moving into these homes. Infants and young children are likely most at risk for exposure to THS in dust and surfaces and its health consequences because of age-specific behaviours (eg, crawling, sucking, ingesting non-food items, hand-to-mouth contact). Known susceptibility of infants due to immature respiratory and immune systems, lower metabolic capacity and the many years of life remaining make exposure to the potent carcinogens reported in THS a concern. It has been previously demonstrated that house dust can be a major route of exposure to lead for young children.^{22 23}

What this paper adds

- ▶ Thirdhand smoke (THS) consists of tobacco smoke pollutants that remain on surfaces and in dust after tobacco has been smoked, are re-emitted and resuspended back into the air, or react with oxidants and other compounds in the environment to yield secondary pollutants.
- ▶ Evidence collected in field and controlled laboratory studies shows that indoor environments in which tobacco is regularly smoked become reservoirs of THS, potentially leading to the involuntary exposure of non-smokers to THS in the absence of concurrent smoking and long after smoking has taken place.
- ▶ This study is the first to examine whether private homes of smokers remain contaminated with THS after the smokers move out and non-smokers move in, and whether non-smokers who move into homes formerly occupied by smokers are exposed to THS through contaminated dust, surfaces and air in these homes.
- ▶ Findings indicate that THS accumulates in smokers' homes and persists when smokers move out even after homes remain vacant for 2 months and are cleaned and prepared for new residents. When non-smokers moved into homes formerly occupied by smokers, they encountered indoor environments with measurable THS polluted surfaces and dust. Results suggest that non-smokers living in former smoker homes are exposed to THS in dust and on surfaces.

Based on the current limited evidence on the chemistry, biology and behavioural science of THS, it is premature to rule on its significance as a cause, moderator, mediator, or contributor to health outcomes. This and other studies suggest caution in trivialising the relatively low levels of pollutants found 2 months after the last cigarette was smoked. The limited existing research warrants rigorous further investigations into the chemical, physical, biological, environmental, behavioural and economic aspects of THS to more comprehensively understand its impact on human health in the social and policy contexts in which smoking occurs throughout the world.

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REFERENCES

1. **US Environmental Protection Agency.** *Respiratory health effects of passive smoking: lung cancer and other disorders.* Washington, DC: U.S. Dept. of Health and Human Services, U.S. Environmental Protection Agency, 1993.
2. **National Cancer Institute (NCI).** *Health Effects of Exposure to Environmental Tobacco Smoke: The Report of the California Environmental Protection Agency. Smoking and Tobacco Control Monograph No. 10, National Institutes of Health (NIH) Publication No. 99-4645.* Bethesda, MD: NCI, NIH, U.S. Department of Health and Human Services (USDHHS), 1999.
3. **State of California Air Resources Board.** *Technical support document for the "Proposed Identification of Environmental Tobacco Smoke as a Toxic Air Contaminant", Part A-Exposure Assessment.* State of California Air Resources Board, 2005.
4. **International Agency for Research on Cancer.** *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans: Tobacco Smoke and Involuntary Smoking.* Lyon (France): International Agency for Research on Cancer, 2004.
5. **New York Times.** *The 9th Annual Year in Ideas. Thirdhand Smoke.* New York: The New York Times Magazine, 2009:66–7.
6. **Szabo L.** *Babies may absorb smoke residue in home.* USA Today, 2006.
7. **Destailats H, Singer BC, Lee SK, et al.** Effect of ozone on nicotine desorption from model surfaces: evidence for heterogeneous chemistry. *Environ Sci Technol* 2006;**40**:1799–805.
8. **Sleiman M, Gundel LA, Pankow JF, et al.** Formation of carcinogens indoors by surface-mediated reactions of nicotine with nitrous acid, leading to potential thirdhand smoke hazards. *Proc Natl Acad Sci* 2010;**107**:6576–81.
9. **Matt GE, Quintana PJ, Hovell MF, et al.** Households contaminated by environmental tobacco smoke: sources of infant exposures. *Tob Control* 2004;**13**:29–37.
10. **Matt GE, Quintana PJE, Hovell MF, et al.** Residual tobacco smoke pollution in used cars for sale: air, dust, and surfaces. *Nicotine Tob Res* 2008;**10**:1467–75.
11. **Van Loy MD, Lee VC, Gundel LA, et al.** Dynamic behavior of semi-volatile organic compounds in indoor air. 1: nicotine in stainless steel chamber. *Environ Sci Technol* 1997;**31**:2554–61.
12. **Singer BC, Hodgson AT, Guevarra KS, et al.** Gas-phase organics in environmental tobacco smoke: 1. Effects of smoking rate, ventilation, and furnishing level on emission factors. *Environ Sci Technol* 2002;**36**:846–53.
13. **Singer BC, Hodgson AT, Nazaroff WW.** Gas-phase organics in environmental tobacco smoke: 2. Exposure-relevant emission factors and indirect exposures from habitual smoking. *Atmos Environ* 2003;**37**:5551–61.
14. **Emerson JA, Hovell MF, Meltzer SB, et al.** The accuracy of environmental tobacco smoke exposure measures among asthmatic children. *J Clin Epidemiol* 1995;**48**:1251–9.
15. **Matt GE, Hovell MF, Zakarian JM, et al.** Measuring secondhand smoke exposure in babies: the reliability and validity of mother reports in a sample of low-income families. *Health Psychol* 2000;**19**:232–41.
16. **Hovell MF, Zakarian JM, Matt GE, et al.** Counseling to reduce children's secondhand smoke exposure and help parents quit smoking: a controlled trial. *Nicotine Tob Res* 2009;**11**:1395–406.
17. **Kuusimäki L, Pfaffli P, Froshaug M, et al.** Determination of nicotine as an indicator of environmental tobacco smoke in restaurants. *Am J Ind Med* 1999;(Suppl 1):152–4.
18. **STATA Corp.** *Stata statistical software. Release 10.0.* College Station, TX: Stata Corporation, 2007.

19. **SPSS Inc.** *SPSS for Windows. Release 15.0.* Chicago, IL: SPSS Inc, 2006.
20. **Matt GE**, Romero R, Ma DS, *et al.* Tobacco use and asking prices of used cars: prevalence, costs, and new opportunities for changing smoking behavior. *Tob Induc Dis* 2008;**4**:2.
21. **Hovell MF**, Hughes SC. The behavioral ecology of secondhand smoke exposure: a pathway to complete tobacco control. *Nicotine Tob Res* 2009;**11**:1254–64.
22. **Lanphear BP**, Matte TD, Rogers J, *et al.* The contribution of lead-contaminated house dust and residential soil to children's blood lead levels: a pooled analysis of 12 epidemiologic studies. *Environ Res* 1998;**79**:51–68.
23. Childhood lead poisoning associated with lead dust contamination of family vehicles and child safety seats—Maine, 2008. *MMWR: Morb Mortal Wkly Rep* 2009;**58**:890–3.