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Thirdhand Smoke in the Homes of Medically Fragile Children: Assessing the Impact of Indoor Smoking Levels and Smoking Bans

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Abstract

Introduction: Thirdhand smoke (THS) residue results from secondhand smoke, and is emerging as a distinct public health hazard, particularly for medically fragile pediatric patients living with smokers. THS is difficult to remove and readily reacts with other pollutants to form carcinogens and ultrafine particles. This study investigated THS found in homes of high-risk infants admitted to a neonatal intensive care unit and their association with characteristics (eg, number of household smokers) hypothesized to influence THS.

Methods: Baseline data from 141 hospitalized infants' homes were analyzed, along with follow-up data ($n = 22$) to explore household smoking characteristics and THS changes in response to indoor smoking ban policies.

Results: Households with an indoor ban, in which not more than 10 cigarettes/d were smoked, had the lowest levels of THS contamination compared to homes with no ban ($P < .001$) and compared to homes with an indoor ban in which greater numbers of cigarettes were smoked ($P < .001$). Importantly, homes with an indoor ban in which at least 11 cigarettes/d were smoked were not different from homes without a ban. The follow-up sample of 22 homes provided initial evidence indicating that, unless a ban was implemented, THS levels in homes continued to increase over time.

Conclusions: Preliminary longitudinal data suggest that THS may continue to accumulate in homes over time and household smoking bans may be protective. However, for homes with high occupant smoking levels, banning indoor smoking may not be fully adequate to protect children from THS. Unless smoking is reduced and bans are implemented, medically fragile children will be exposed to the dangers of THS.

Introduction

Thirdhand smoke (THS) is the residue and particles resulting from secondhand smoke (SHS), and is emerging as a distinct public health hazard,[1–4] and may be particularly problematic for medically fragile pediatric patients who live in smoking households. THS readily reacts with other pollutants to form carcinogens and ultrafine

particles,[5–9] is difficult to remove from indoor deposits,[6,7,10] and reemits as particulate matter[11] or in gaseous form (ie, off-gassing) for months after a cigarette is extinguished.[12] Recent work has shown that nicotine and other THS chemicals (eg, a known carcinogen, 4-[methylnitrosamino]-1-[3-pyridyl]-1-butanone [NNK]) are detectable in common household fabrics for up to one and a half years.[13] The residual nicotine on surfaces serves as a convenient tobacco-specific marker for combusted-tobacco constituents (eg, carcinogenic compounds) that have been collectively referred to as THS.

Simply smoking outdoors is unlikely to protect infants and children from SHS or THS,[12,14] as the ways in which THS is dispersed throughout an indoor space and the routes of exposure are numerous. Smokers often reenter a home immediately after extinguishing a cigarette outdoors, believing that they no longer have to worry about bringing SHS indoors. However, after the final puff from a cigarette, a smoker continues to expel particulate matter for up to 90 seconds,[15] and the breath and clothing of smokers have higher concentrations of benzene (a carcinogenic solvent), Toluene (neurotoxic solvent), 2,5-Dimethylfuran (ie, a neurotoxic and cilatoxic substance [that adversely effects lung cilia in respiration]), and other toxic chemicals for up to 10 minutes.[16] Once inside a home, THS desorbs from residue carried on hands, hair, clothes, and other objects (eg, a cell phone) and adsorbs to new surfaces.[6,7,9] THS is then reemitted over-and-over again.[3,4,17,18] Importantly, common cleaning methods fail to remove nicotine that adsorbs to indoor surfaces such as dust, doors, curtains, upholstery, pillows, mattresses, clothes, and especially carpets and sheet rock walls.[6,7,10,19] Indeed, 80%–90% of combusted cigarette nicotine adsorbs (ie, sticks) to indoor surfaces.[17]

THS bonded to indoor dust and surfaces may expose individuals through ingestion, inhalation, or dermal uptake,[11,20] leading to potential health risks. In one study, nonsmokers occupying homes vacated by smokers had elevations of finger nicotine and urine cotinine weeks after the smoker(s) moved out.[7] Similar findings in hotels have demonstrated that nonsmokers staying in designated smoking rooms had detectable elevations of THS-related carcinogens and nicotine metabolites in their urine, as well as elevated finger surface nicotine levels.[21] Findings for infants and children are more troubling. Even in households with smokers who ban indoor smoking, infants have 5–7 times more THS exposure (THSe) than infants from nonsmoking households.[12] Further, one large study found that children only exposed to THS (ie, no SHS exposure [SHSe]) have more respiratory symptoms than non-exposed children,[1] and long-term, cumulative exposure to THS is worrisome and underexplored. Increased research has been recommended to determine the degree to which THS harms human health,[6,22] especially in children who are more susceptible to THSe and its consequences.[20]

SHSe and active smoking have garnered much of the research attention and THS's contribution to poor health outcomes may be greatly underestimated. Early exposure to nicotine may potentiate smoking later in life, and animal models and data from pregnant women have shown that early nicotine exposure contributes to cognitive impairment, attention deficit disorders, obesity, type-2 diabetes, respiratory dysfunction and impaired fertility.[23–25] Recent evidence has also linked tobacco-specific nitrosamines (found in THS) to pancreatic cancer.[26] Also, exposure levels to nicotine and tobacco-specific nitrosamines (particularly for toddlers who frequently suck on household materials) may be up to 6.8 times and 16 times greater in THS, respectively, compared to passive SHSe.[13] These greater exposure estimates raise concerns as THS has been shown in animal and in vitro assay studies to damage DNA,[27] affect fibroblast migration involved in wound healing[28]; and hinder respiratory development in unborn, premature rat fetuses,[29] and THS may contribute to hyperactivity-related behavior problems.[30] SHSe is thought to contribute to epithelial apoptosis and alter the microbiome,[31] and THS may have similar adverse health consequences, as microbes found on neonatal intensive care unit (NICU) surfaces have later been found in the guts of premature infants.[32]

These preliminary findings are of greatest concern for premature, low-birth-weight infants, who are at elevated risk for harm due to higher respiration rates, immaturity of respiratory functioning, low metabolic capacity, and immature or compromised immune systems. Over 25% of NICU infants are discharged to a home with at least one smoker[14] and similar proportions of medically fragile and/or chronically ill pediatric patients may live with smokers.

The primary aim of this exploratory study was to characterize the level of THS found in the homes of infants admitted to a NICU, and explore the associations of household characteristics that might influence THS levels, including number of smokers living in the home, cigarettes per day (per household), housing type (single family, multiunit), cleaning practices, and in-home smoking ban status (yes/no). Generally, we hypothesized that homes with more smokers, greater cigarette consumption, less frequent cleaning, and homes that permitted indoor smoking would show greater THS levels as indicated by surface nicotine concentration. We hypothesized that multiunit apartments would have greater surface nicotine than single-family homes. A secondary aim was to reexamine THS levels after a 6-month period in a subgroup of participants, during which time families enrolled in a SHS intervention study may have sustained, implemented (for the first time), or discontinued an indoor smoking ban. Our hypotheses were that THS levels would remain stable in homes that sustained a ban, increase in homes that discontinued a smoking ban, and decrease in homes that initiated a ban. This extends work exploring THS levels in the homes of infants[12] by focusing on infants at higher risk for health problems. This study is expected to inform subsequent research aiming to reduce THS contamination and further protect children at great risk for harm from carcinogenic THS.

Methods

Participants and Procedures

Participants were recruited as part of an ongoing SHSe prevention intervention study (*Baby's Breath II*), registered on http://clinicaltrials.gov (NCT01726062). Participants were primary caregivers (ie, most often mothers) to infants admitted between September, 2012 and June, 2014 to a large children's hospital in Houston, Texas with a 128-bed NICU and approximately 1100 admissions per year. The overall trial design and protocol are published.[33] Briefly, research assistants approached caregivers of infants in the NICU to screen for eligibility including: (1) have an infant in the NICU; (2) report at least 1 smoker living in the household; and (3) live within a 50-mile radius of the hospital (due to follow-up home assessments). All participants provided written informed consent in compliance with our local IRB's regulations, the Committee for the Protection of Human Subjects (CPHS) of the University of Texas Medical School, Houston, and the Memorial Hermann Hospital IRB.

Study Design

Baseline data (while the infant was hospitalized in the NICU) were obtained from the first 141 randomized participants with a baseline THS wipe. The overall study participant refusal rate was 14.2%, indicating that a majority of participants found all study procedures

acceptable. Participants were randomized after baseline to conventional NICU care or a Motivational Interviewing intervention targeting the prevention of SHSe/THSe among infants after discharge. There were three additional follow-up assessments at 2 weeks post-NICU discharge (ie, a mid-treatment assessment), 2 months post-NICU discharge (approximately 1 month posttreatment), and 6 months post-NICU discharge (approximately 4 months posttreatment) per trial design.[33] Data collection included interviews, a self-paced computerized questionnaire, a THS (surface-nicotine wipe) sample from the participants' homes, air-nicotine assays, and infant urine cotinine assays. Not all measures were collected at each assessment by trial design (Table 1); for example, infant urine cotinine data were not collected at baseline (while the infant was hospitalized and thus no opportunity for SHSe/THSe).

THS data were collected at the 6-month post-NICU assessment from a subsample of participants' homes (n = 22), referred to as the "follow-up sample." These data were collected for an ancillary pilot study (not part of the original trial design). The 22 homes with 6-month follow-up data were sampled consecutively after trial initiation from homes that had complete baseline and 6-month data. Samples of 20 or fewer are routinely used in THS research,[7,12,21] and this study is comparable to previous designs.

Measures

The research assistant-administered interview and self-paced questionnaire included questions related to participant and household characteristics, psychosocial factors (eg, depressive symptoms), and smoking-related attitudes and behaviors. Variables related to smoking or cleaning with a potential for direct influence on household THS levels were investigated. The number of smokers living in the home was collapsed to 1, 2, and at least 3 (due to a limited number of homes with four or more smokers). The number of cigarettes each smoker used per day (regardless of location) was assessed and combined to form a single composite of total number of cigarettes per day for the entire household. Total household cigarettes (regardless of location) were used due to the transmission of THS on hands, clothing, and other objects to the home. Further, based on our experience, some participants report no smoking indoors even when there is evidence to the contrary (eg, ashtrays). Thus, asking participants to report on total household cigarettes smoked is less susceptible to underreporting than reporting on cigarettes smoked indoors. Number of cigarettes per day across household members was analyzed as a quantitative indicator and was also collapsed to not more than 10 cigarettes/d and at least 11 cigarettes/d, representing light and heavy smoking.[34,35] Type of housing was dichotomized to single-family house or multiunit housing (including duplexes).

Table 1. Study Measurement by Assessment Time Point

	Assessment time point			
Measurement	BL	2-Wk D/C	2-Mo D/C	6-Mo D/C
Interview and self-report	X	X	X	X
Surface nicotine (THS)	X	—	—	X (n = 22)
Air-nicotine monitor	—	—	X	X
Urine cotinine	—	X	X	X

X = data collected; — = data not collected; BL = baseline; 2-Wk D/C = 2 weeks post-NICU discharge; 2-Mo D/C = 2 months post-NICU discharge; 6-Mo D/C = 6 months post-NICU discharge; THS = thirdhand smoke. Air nicotine levels and urine cotinine were not assessed at baseline because the infant was hospitalized in the NICU and had no opportunity for exposure.

Cleaning frequency was assessed by asking how often the participant (or another household member) cleaned the home (ie, daily, several times a week, or weekly or less often). In-home smoking ban status was assessed with a multiple-choice question and two confirmation questions.[36] Only a report of a total indoor home smoking ban, with no exceptions, was counted as having a home smoking ban (yes/no).

THS surface wipe procedures have been established,[7,12,21,37] and samples were taken from the room where the majority of indoor smoking was reported. In homes with indoor bans, samples were taken from the room (ie, the primary room) where the smoker(s) spent the most time. Samples were taken 1.5 meters from the floor from one of the following surfaces (ordered by preferential availability): wooden door, wooden cabinet, door of any material type, closest "wipeable" wood surface (nearest the primary room). Painting, refinishing, sanding, or waxing in the previous 30 days excluded surfaces from being wiped. Research assistants generally took follow-up wipe samples from locations adjacent to the original collection site; however, precise sampling information was not always available and some sampling location overlap was likely to have occurred. Taking a sample of THS surface nicotine involves: (1) preparing a solution of distilled water and 1% ascorbic acid (ie, vitamin C); (2) wetting a screened cotton wipe with the solution; (3) wiping a 100 cm^2 surface; and (4) storing the wipe in a vial for further analysis. Surface nicotine levels are reported in micrograms (μg) per meter squared (m^2). Following Quintana and colleagues'[37] recommendation, field blanks were collected. Specifically, surface wipe procedures were followed with the exception of step 3 (the cotton was exposed to the air but not used to wipe a surface).

Air-nicotine levels are reported in micrograms per cubic meter (m^3) and were obtained from passive air monitors placed in each home approximately 2 weeks before the 2-month and 6-month assessments using standardized procedures.[38] Monitors were clipped to curtains or lamps in the room "where the infant spends a majority of their time" (not including infant bedrooms; most often the living room). Vapor-phase nicotine emissions from tobacco combustions have been established as a valid and reliable marker for passive smoke.[39] Diffusion filters have been found to be an effective and relatively unobtrusive means of assessing household nicotine concentrations[40] and have been used in many SHSe intervention studies.[41] Urine cotinine levels are reported in nanograms (ng) per milliliter (ml), and were collected from infants by extracting urine (via sterile syringe) from two cotton pads placed in the infants' diapers.[42]

Statistical Analyses

All analyses were conducted in SAS, version 9.3, and all statistical tests were evaluated at the alpha (two-tailed) 0.05 level. To control for nonnormal distributions and heterogeneous error variances, we applied natural logarithmic transformations to the THS wipe nicotine, air-nicotine, and urine cotinine values and report geometric means, medians, and interquartile ranges (IQR). Tobit regression analyses for left-censored data (ie, nicotine levels below the level of detection [LOD; ie, 0.10 μg/m^2])[37] were used to test hypotheses about differences in surface nicotine across the household characteristic variables. Dependent samples t tests were also used to compare baseline and follow-up sample data.

Results

The mean age of the caregivers at baseline was 26.1 years (SD = 5.6) and 58.9% had a high school education or less. The

sample was predominantly black/African American (65.3%), followed by Hispanic/Latino (22.7%), white, non-Hispanic (9.2%), and 2.8% were Asian. The mean infant birth weight was 2140.9 g (*SD* = 958.5 g) and a majority of infants (63.8%) were born at low birth weights (<2500 g) with 27.0% born at very low birth weights (<1500 g). Excluding six infants who died while in the NICU, the median number of days from admission to discharge was 29 (IQR: 12–57).

All THS wipe values reported in this work were adjusted by subtracting out the level of nicotine found in field blanks to account for nicotine levels that may already be present in the sampling materials and the air in participants' homes prior to sampling. Field blanks ranged from below the (LOD) to as high as 0.01 μg per cotton wipe (*M* = 0.0021 μg/cotton wipe). After correction, only six (out of 141) baseline wipes and 0 (out of 22) 6-month post-NICU discharge wipes were below the LOD and the maximum surface nicotine level was 1951.4 μg/m^2 (IQR: 0.7–13.7 μg/m^2).

THS Associations With Household Characteristics at Baseline

Detailed descriptive statistics for surface nicotine are presented across all analyzed household characteristics in Table 2. Greater surface nicotine was found in homes with at least three smokers (Geometric Mean [GeoM] = 19.1 μg/m^2; $P < .001$) and two smokers (GeoM = 7.2 μg/m^2; $P < .01$) compared to homes with a single smoker (GeoM = 2.0 μg/m^2). For comparison, homes with no smokers and total smoking bans have been shown to have no detectable surface nicotine in their living rooms and bedrooms.[12] Although the geometric mean for homes with at least three smokers was nearly three times as high as the geometric mean of homes with two smokers, the difference was not statistically significant ($P = .18$). When analyzed as a continuous variable, number of cigarettes smoked per household was positively associated with surface nicotine ($P < .0001$). As described in the Methods, household cigarette use was dichotomized to simplify interpretation. Households that smoked at least 11 or more cigarettes a day had greater surface nicotine (GeoM = 12.6 μg/m^2) than homes that reported not more than 10 cigarettes a day (GeoM = 1.7 μg/m^2; $P < .0001$). Single-family homes had twice as much surface nicotine (GeoM = 5.4 μg/m^2) as homes from multiunit housing developments (GeoM = 2.3 μg/m^2; $P = .03$). Participants that reported cleaning several times/wk had lower surface nicotine levels (1.7 μg/m^2) than homes that reported daily cleaning (4.9 μg/m^2; $P = .04$). No other comparisons of frequency of cleaning were statistically significant. Homes that reported indoor smoking bans had significantly lower surface nicotine levels (1.7 μg/m^2) compared to homes that permitted indoor smoking (9.1 μg/m^2; $P < .0001$).

A multivariable Tobit regression was performed and retained two statistically significant predictors of surface nicotine: household ban status ($P < .01$) and number of household cigarettes smoked ($P < .0001$). When analyzed further, the number of cigarettes smoked collectively across household members had an important impact, even among homes with an indoor smoking ban. Homes with an indoor ban that smoked not more than 10 cigarettes a day (GeoM = 1.0 μg/m^2) had significantly lower surface nicotine than homes with an indoor ban that smoked at least 11 cigarettes a day (GeoM = 9.6 μg/m^2; $P < .001$; Figure 1). Households with a ban that smoked not more than 10 cigarettes a day also had significantly lower surface nicotine than homes without a ban that smoked not more than 10 cigarettes a day (GeoM = 5.1 μg/m^2; $P < .001$) and homes without a ban that smoked at least 11 cigarettes/d (GeoM = 14.6 μg/m^2; $P < .0001$).

Importantly, surface nicotine in homes with an indoor smoking ban in which at least 11 cigarettes were smoked per day were not

Table 2. Surface Nicotine Levels by Household Characteristics

Characteristic	*n* (%)	Surface nicotine (μg/m^2)		*P*
		Geomean (95% CI)	Median (IQR)	
Number of smokers				
1	93 (66.0)	2.0 (1.3–3.2)	2.3 (0.6–7.7)	<.001
2	29 (20.6)	7.2 (2.3–23.1)	6.4 (1.2–88.4)	.18
≥3	19 (13.5)	19.1 (7.9–46.5)	25.4 (3.7–64.1)	R
Number of cigarettes/d				
≤10	89 (63.1)	1.7 (1.0–2.8)	1.9 (0.5–6.4)	<.0001
≥11	52 (36.9)	12.6 (6.8–23.3)	9.1 (2.5–63.3)	R
Housing				
Multiunit	70 (49.7)	2.3 (1.3–4.1)	2.9 (0.6–10.4)	.03
Single-family	71 (50.4)	5.4 (2.9–10.1)	3.7 (1.2–29.3)	R
Cleaning frequency				
Daily	95 (67.4)	4.9 (2.9–8.1)	4.2 (1.0–25.4)	.27
Several times/wk	29 (20.6)	1.7 (0.7–3.9)	2.1 (0.7–4.5)	.62
Weekly or less often	17 (12.1)	2.2 (0.4–10.4)	1.7 (0.6–4.4)	R
Indoor smoking ban				
No	62 (44.0)	9.1 (5.0–16.6)	8.4 (2.9–37.9)	<.0001
Yes	79 (56.0)	1.7 (1.0–2.9)	1.8 (0.4–4.4)	R
Ban × cigarettes/d				
Ban = No, ≥11	34 (24.1)	14.6 (7.3–29.0)	9.7 (3.7–64.1)	<.0001
Ban = No, ≤10	28 (19.9)	5.1 (1.8–14.6)	6.3 (1.4–27.0)	<.001
Ban = Yes, ≥11	18 (12.8)	9.6 (2.6–35.7)	5.2 (1.7–62.5)	<.001
Ban = Yes, ≤10	61 (43.3)	1.0 (0.6–1.8)	1.0 (0.4–3.6)	R

CI = confidence interval; Geomean = Geometric mean; IQR = interquartile range; R = reference group.

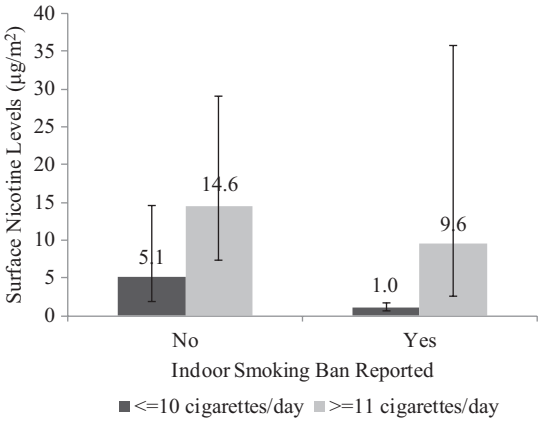

Figure 1. Surface nicotine levels (with 95% confidence limits) by indoor smoking ban status and number of household cigarettes consumed per day.

different from homes without a ban, at both low (P = .41) and high (P = .48) smoking levels. Homes without a smoking ban were not statistically different at high versus low smoking levels (P = .08).

Follow-Up Sample Data (ie, 6-Month Follow-Up)

Twenty-two homes provided surface nicotine samples at baseline and again at 6 months post-NICU discharge. Surface nicotine levels for the majority of homes (68%; N = 15) increased from baseline to 6 months as shown in Table 3. The table is sorted by indoor smoking ban status at baseline and included reported ban status at all post-NICU discharge assessments (ie, 2 weeks, 2 months, and 6 months). Several general trends emerge from this table. First, 11 (out of 12) homes with a ban at baseline had significantly greater levels of surface nicotine at 6 months post-NICU discharge ($t[df = 11] = -3.60$, $P < .01$; median increase= 0.9 µg/m^2; IQR = 0.5–4.0 µg/m^2), and two (out of two) homes that permitted indoor smoking at baseline and never established an indoor ban also had increases (ie, of 7.1 and 395.0 µg/m^2). Second, six (out of eight) homes that permitted indoor smoking at baseline and implemented an indoor ban at a later time had significant decreases in surface nicotine from baseline to 6 months post-NICU discharge ($t[df = 7] = 2.46$, $P < .05$; median decrease= 8.0 µg/m^2; IQR = 0.7–15.7 µg/m^2).

Relations Between Surface Nicotine, Air Nicotine, and Urine Cotinine

Pearson correlations of surface-nicotine levels, air nicotine levels, and urine-cotinine levels (all log-transformed) were undertaken to better understand the relations among these measures of nicotine exposure. Baseline and 6-month surface nicotine were summed to form a "total surface nicotine" variable, which was also log transformed. Positive associations were found between baseline surface nicotine and 2-month air nicotine (n = 98, r = 0.34, $P < .001$) and 6-month air nicotine (n = 80; r = 0.38, $P < .001$) and between total surface nicotine and 6-month air nicotine (n = 21, r = 0.63, $P < .01$). Baseline surface nicotine positively correlated with 2-month urine cotinine levels (n = 109; r = 0.35, $P < .001$) and 6-month urine cotinine (n = 95; r = 0.25, P = .01); total surface nicotine also correlated with 6-month urine cotinine levels (n = 22, r = 0.43, $P < .05$). Further, 2-month air nicotine and 2-month urine cotinine levels correlated strongly (n = 95, r = 0.55, $P < .0001$), as did 6-month air nicotine and 6-month urine cotinine levels (n = 78, r = 0.55, $P < .0001$).

Two (of 21) homes had air-nicotine levels below the LOD (ie, 0.02 µg/m^3) and five additional homes were only slightly above the LOD (≤0.05 µg/m^3), suggesting no (or very low) SHSe in the home (although SHSe in other environments cannot be ruled out). Urine cotinine values for the seven infants from these homes, however, were all above the LOD for urine cotinine (ie, 0.1 ng/ml),[42] raising the possibility that, while SHSe was limited, surface nicotine from THS was absorbed by the infants.

Discussion

This was the first study to examine THS levels in homes of medically fragile pediatric patients, and contributes several novel findings to the THS literature. Infants of smokers returning home after NICU discharge find a THS-polluted home environment, even if caregivers no longer permit indoor smoking. Second, homes that ban indoor smoking but report greater levels of overall household smoking tended to have surface nicotine levels comparable to homes that permit indoor smoking, raising the possibility that indoor bans may not offer sufficient protection from THS for children in these homes. Third, homes with an indoor smoking ban tended to continue accumulating surface nicotine, underscoring that THS may persist as long as household smoking occurs at any level. Conversely, preliminary, longitudinal data suggest that THS accumulation may be reversible by implementing an indoor ban. These objective data, while exploratory, suggest that a relatively large number of households are at risk for significant tobacco-related health disparities,[43] and the results with respect to infants released from NICUs were both intriguing and alarming with profound implications for child health.

There are several potential explanations for why homes that ban indoor smoking have high THS levels, such as the persistence of THS for months to years. Contamination could come from SHS entering the home through open doors/windows and adsorbing to indoor surfaces. The correlation of baseline and total surface nicotine with air monitors at the 6-month time point supports these conclusions. Over 60% of the variance in surface nicotine was not explained by air-nicotine levels, which is not surprising as surface nicotine is often accumulated over a longer period of time and certainly longer than the air nicotine measurement period in this study. As stated previously, THS is known to adsorb to hair, clothes, and skin and desorb from these surfaces (onto new surfaces; eg, the floor of a home) after a smoker returns indoors. It is logical to expect that more household smoking leads to more opportunities for THS contamination and thus greater levels of contamination regardless of where smoking takes place. More information is needed on the smoking practices of homes that ban indoor smoking. For example, what distance from the home is sufficient to eliminate SHS wafting through doors, air vents, and windows? For how long must a smoker remain outdoors after smoking to avoid exhaling particulate matter inside their home? However, particulate matter and residue will still be transported indoors on smokers' clothing, skin, and hair regardless. Also, the finding that single-family homes had greater levels of surface nicotine than multiunit apartments was counter to our hypothesis and deserves comment. It is possible that landlords have rental policies in place to prohibit indoor smoking and in fact the Houston (Texas) Public Housing Authority has banned smoking in all publicly-supported housing (the ban started in January, 2014). Single-family homeowners do not have this same pressure to ban indoor smoking.

The transmission of THS to nonsmokers is documented[7,21]; however, the forms of transmission are less understood. Possible sources

Table 3. Surface Nicotine, Air Nicotine, and Infant Cotinine Levels for the Follow-Up Sample ($n = 22$)

	Surface nicotine (μg/m^2)			Indoor home smoking ban				Per household (at baseline)		Air nicotine(μg/m^3)		Urine cotinine (ng/ml)
PPT	BL	6-Mo D/C	Δ 6-Mo D/C – BL	BL	2-Wk D/C	2-Mo D/C	6-Mo D/C	# of smokers	# of cigs/d	2-Mo D/C	6-Mo D/C	6-Mo D/C
1	6.4	3.9	−2.5	Yes	Yes	Yes	No	2	2	M	0.05	7.82
2	1.8	5.1	3.3	Yes	Yes	Yes	No	2	4	0.43	0.65	5.73
3	1.0	5.7	4.7	Yes	No	Yes	Yes	1	4	M	0.03	0.38
4	3.7	14.7	11.0	Yes	Yes	No	No	1	7	0.34	0.19	2.22
5	67.3	327.0	259.8	Yes	No	M	Yes	1	35	M	1.94	4.28
6	0.7	1.2	0.5	Yes	Yes	Yes	Yes	2	0	0.05	0.01	0.22
7	0.3	1.3	1.0	Yes	Yes	Yes	Yes	1	1	0.27	0.15	1.08
8	1.0	3.8	2.8	Yes	Yes	Yes	Yes	1	2	0.29	0.17	1.46
9	0.0	0.4	0.4	Yes	Yes	Yes	Yes	1	5	0.02	0.01	1.16
10	0.3	0.4	0.1	Yes	Yes	Yes	Yes	1	6	0.09	0.03	3.50
11	0.5	1.1	0.6	Yes	Yes	Yes	Yes	1	10	0.04	0.03	0.11
12	0.2	1.0	0.8	Yes	Yes	Yes	Yes	2	11	0.02	0.13	4.26
13	10.4	1.0	−9.4	No	Yes	No	Yes	1	5	0.56	0.13	7.52
14	4.4	2.0	−2.4	No	Yes	Yes	No	1	5	0.73	0.36	0.41
15	0.6	1.6	1.0	No	Yes	Yes	Yes	1	7	0.21	0.11	5.35
16	9.5	0.9	−8.6	No	Yes	Yes	Yes	2	14	0.48	0.33	2.62
17	35.5	13.4	−22.1	No	Yes	No	Yes	1	15	0.20	1.20	26.16
18	3.8	6.9	3.2	No	Yes	Yes	Yes	1	20	0.40	0.18	7.25
19	7.9	0.6	−7.3	No	Yes	Yes	No	1	30	0.07	0.05	1.05
20	24.7	1.9	−22.8	No	Yes	Yes	Yes	3	60	0.20	0.21	5.06
21	5.3	12.4	7.1	No	M	M	No	1	5	M	M	10.85
22	129.6	524.5	395.0	No	No	No	No	2	6	0.04	0.13	4.57

Δ = change; BL = baseline; 2-Wk D/C = 2 weeks post-NICU discharge; 2-Mo D/C = 2 months post-NICU discharge; 6-Mo D/C = 6 months post-NICU discharge; M = missing. Number of smokers and number of cigarettes per day are reported from baseline measurements; these measures remained stable for most households over later assessments.

include THSe that is not visible or often not detected when off-gassing (ie, returning to gaseous form) takes place or from direct contact (eg, toddler mouth-to-fabric contact) with contaminated clothing, carpets, etc.[13,44] The moderately sized correlation of baseline and total surface nicotine with urine cotinine was consistent with THS contributing to tobacco smoke exposure for these infants. However, given the complexities of THSe, these bivariate associations should not be over interpreted. Another source (especially for very young children) could be skin-to-skin exposure when the child is held or touched by family members or others who smoke. Urine cotinine was above the LOD for all 22 infants whose homes were sampled at baseline and 6 months post-NICU discharge. While SHSe in other locations cannot be ruled out, this suggests that ingestion (hand-to-mouth), hand-to-eye, hand-to-nose, inhalation, and dermal exposure is a real THS-exposure risk for infants residing in these homes, putting them at risk for DNA damage and impaired wound healing among other possible health risks.

Regardless of how THS enters homes or the body, THS measurements may be important targets for interventions as well as an outcome measure. These preliminary data indicate the potential for THS levels to decrease in a real-world setting following the establishment of an indoor ban. This observation informs future clinical trials designed specifically to reduce THSe as well as SHSe. For many homes in which a smoking ban was initiated, the level of surface nicotine dropped to levels that were similar to homes with a smoking ban and low levels of smoking, which may reduce the potential for health-related harm. There is no safe level of SHSe and whether there is a safe level of THSe is unknown but doubtful and certainly not worth the risk for immuno-compromised infants. THSe should be minimized to the greatest extent possible. As demonstrated by this work as well as research in hotels with partial bans,[21] however, the only way to completely eliminate THS contamination is for all household members to abstain from smoking, and within multiunit housing complexes, infiltration from neighbors who smoke must be eliminated.

The relationship between household cleaning frequency and surface nicotine would seem counterintuitive, as homes that reported less frequent cleaning had lower levels. A majority of participants reported "daily" cleaning practices (eg, 68%) and this question was likely influenced heavily by participant bias to present themselves in a positive light to research staff. There are also a wide variety of cleaning methods, and one problem may occur when dusting, sweeping, or vacuuming (without well-maintained HEPA filters) resuspends ultrafine particulate matter, increasing exposure. Indeed, it is plausible that homes engaging in daily vacuuming may reaerate ultrafine particles and increase airborne THSe compared to homes with less frequently cleaned surfaces; future research should explore the relationship between specific cleaning practices (eg, vacuuming) and THS levels. Other methods of assessing household cleaning approaches may prove necessary to determine whether or not traditional cleaning practices reduce THS levels. Water-based (aqueous) remediation of THS from cotton may be sufficient to eliminate THS pollution[13] but less is known about remediating myriad other household surfaces and materials. It is highly possible that greater efforts will be needed to remove THS.

The variability in surface nicotine across homes is worth noting, especially for participants 5 and 22, whose homes had follow-up surface nicotine levels well above other participants (but not uncommonly high, as reported in other THS work).[7,21] Although Participant 5's home ban status was variable across study visits, the air nicotine value was high and she reported relatively high levels of household smoking, thus supporting the elevated surface nicotine value. Participant 22 reported no home smoking ban at any of the visits which is in line with the surface nicotine results. Measurements from the air-nicotine dosimeters (which hang for 2 weeks), however, were relatively low for participant 22's household, perhaps suggesting measurement reactivity in this home, whereby indoor smoking was reduced only for the period during which the monitors hung. Regardless, the discordance between some measures highlights the need for multiple measurement methods in SHS and THS studies.

THS research is in its infancy and many questions remain open for exploration. This study emphasizes the need for other, large sample, controlled studies to examine these questions and measure additional biomarkers (eg, 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanol [NNAL] which metabolizes the carcinogenic tobacco-specific nitrosamine, NNK).[27] Specifically, research needs to determine the amounts of THSe and length of exposure time likely to cause significant health-related harm. It is possible that the associations between smoking and SHSe and adverse health outcomes (eg, breast cancer) have overestimated the contributions of smoking and/or SHSe while ignoring THSe's contributions to health problems and carcinogenesis.[26] Future studies will elucidate over what period of time (and how much cumulative exposure) is necessary, and during what critical periods (eg, in utero, puberty) THSe may heighten risk. Also, the length of time necessary for THS deposits to fully degrade from indoor environments to nontoxic compounds should be explored. Only studies of this nature will help inform public policy to best eradicate THS from all offices, public buildings, homes, cars, and public transportation to protect human health. Ideally, these remediation efforts would also serve to increase cessation and lower smoking potentiation and initiation.[45]

This exploratory work was not without limitations. For example, we relied on a single marker of THS contamination (ie, surface nicotine levels) and it is possible that other THS constituents decay more quickly or remain for longer periods of time. Internal validity was reduced as we did not assess some variables shown to be important elsewhere, such as the age of the homes. Age has been shown to be an important determinant of THS levels in previous studies in cars[46] and may similarly correlate with THS levels in homes. Assessing other potential moderators, such as the time since ban implementation, may also yield important information. The validity of self-reported home bans may be questioned, although studies have demonstrated high correlations between parent self-report and more objective measures.[47] Further, validity was partially supported by significantly lower levels of THS residue found in the homes of participants who reported bans, compared to those who did not. The results presented were largely from cross-sectional baseline data, and the subsample of homes with repeated THS measurements was too small to be representative of populations or draw definitive conclusions. Specifically, the small samples (particularly the follow-up sample) may have resulted in estimates with low precision and may be susceptible to selection bias, setting the stage for additional research. Also, future work with a larger sample of homes may demonstrate differences between homes with bans and those without, regardless of total household smoking levels.

Conclusion

We replicated earlier work that indoor home smoking bans will only be partially effective for reducing THS contamination, and revealed

that children in homes with an indoor ban but with high levels of smoking may not be sufficiently protected from increasing levels of THS. Finally, for a subset of homes in our current study for which we collected follow-up THS data and in which a smoking ban was instituted post-discharge, we observed lower THS levels. Results indicate both the potential for substantial harm to fragile infants discharged from the NICU as well as potential for mitigating harm from such exposure. This preliminary work provides the impetus for numerous larger-scale prevention and intervention studies of THSe in the NICU and other vulnerable pediatric populations.

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Declaration of Interests

None declared.

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