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# Children's Exposure to Secondhand and Thirdhand Smoke Carcinogens and Toxicants in Homes of Hookah Smokers

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## ABSTRACT

**Introduction:** We examined homes of hookah-only smokers and nonsmokers for levels of indoor air nicotine (a marker of secondhand smoke) and indoor surface nicotine (a marker of thirdhand smoke), child uptake of nicotine, the carcinogen 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone (NNK), and the toxicant acrolein by analyzing their corresponding metabolites cotinine, 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanol (NNAL) and NNAL-glucuronides (total NNAL) and 3-hydroxypropylmercapturic acid.

**Methods:** Data were collected at 3 home visits during a 7-day study period from a convenience sample of 24 households with a child 5 years or younger. Three child urine samples and 2 air and surface samples from the living room and the child bedroom were taken in homes of nonsmokers ($n = 5$) and hookah-only smokers ($n = 19$) comprised of daily hookah smokers ($n = 8$) and weekly/monthly hookah smokers ($n = 11$).

**Results:** Nicotine levels in indoor air and on surfaces in the child bedrooms in homes of daily hookah smokers were significantly higher than in homes of nonsmokers. Uptake of nicotine, NNK, and acrolein in children living in daily hookah smoker homes was significantly higher than in children living in nonsmoker homes. Uptake of nicotine and NNK in children living in weekly/monthly hookah smoker homes was significantly higher than in children living in nonsmoker homes.

**Conclusions:** Our data provide the first evidence for uptake of nicotine, the tobacco-specific lung carcinogen NNK, and the ciliatoxic and cardiotoxic agent acrolein in children living in homes of hookah smokers. Our findings suggest that daily and occasional hookah use in homes present a serious, emerging threat to children's long-term health.

## INTRODUCTION

Secondhand smoke (SHS), a by-product of tobacco smoking, is an indoor toxic air contaminant, contains human carcinogens associated with illnesses in children (California Environmental Protection Agency, 2006; Centers for Disease Control and Prevention [CDC], 2010a; Environmental Protection Agency, 1992). There are no known safe levels of SHS exposure; SHS is a toxic mix of more than 7,000 chemicals that kills each year more than 600,000 nonsmokers globally (CDC, 2010a; World Health Organization [WHO], 2013). The WHO (1999) estimated that 700 million or nearly half of the world's children may be exposed to SHS, particularly at home. In the United States, during 2007–2008, the National Health and Nutrition Examination Survey (NHANES) found that about half (53.6%) of children aged 3–11 years were exposed to SHS (CDC, 2010b). Children living in homes of smokers are additionally at risk of exposure to thirdhand smoke (THS). THS consists of residual or aged tobacco smoke particles and toxicants, including nicotine, which remains after tobacco has been smoked and adheres to and is emitted from household surfaces (Matt et al., 2011a). Hang et al. (2013) reported that exposure to THS is genotoxic in human cell

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lines. Research on SHS and THS tends to focus on cigarettes; however, hookah smoking, another method of tobacco use, as a source of SHS and THS has not been studied.

A hookah smoker inhales tobacco smoke through a hose attached to a hookah stem topped with hookah tobacco covered with perforated aluminum foil and inserted into a glass base partially filled with water (Khater, Abd El-Aziz, Al-Sewaidan, & Chaouachi, 2008; Shihadeh, 2003). Burning charcoal is placed on top of the aluminum foil to heat the hookah tobacco, which produces the smoke that the user inhales. Tobacco used for hookah smoking is either unflavored or flavored. Flavored tobacco, known as "Moassel," is a mixture of fruit-flavored tobacco, molasses, artificial flavoring substances, and humectants such as glycerol (Khater et al., 2008; Maziak, Ward, Afifi Soweid, & Eissenberg, 2004; Schubert, Luch, & Schulz, 2013).

Hookah tobacco smoking is on the rise globally (Cobb, Ward, Maziak, Shihadeh, & Eissenberg, 2010; Maziak, 2011). In the United States, in 2013, 26.6% of male and 23.2% of female college students nationally reported ever hookah use (American College Health Association, 2013). Among adults, in 2008, the California Tobacco Surveys showed that 11.2% of males and 2.8% of females ever used hookah (Al-Delaimy et al., 2010). Among middle and high school students, in 2011, the National Youth Tobacco Survey showed that 8.1% of males and 6.6% of females nationally ever used hookah (Amrock, Gordon, Zelikoff, & Weitzman, 2014).

Many hookah smokers are smoking hookah in home settings. In the United States, 43.4%–79.0% of hookah smoker university students surveyed reported smoking hookah at home or in their dormitory (Heinz et al., 2013; Lipkus, Eissenberg, Schwartz-Bloom, Prokhorov, & Levy, 2011). In Syria, nearly half (49.2%) of daily hookah smokers surveyed (mean age, 30.1 years) reported that their usual place of smoking hookah was mainly home (Maziak, Ward, & Eissenberg, 2004). This is potentially alarming particularly with respect to potential exposure of children living in these homes.

Hookah tobacco smoking has been associated with increased risk for periodontal disease, lung and oral cancers, coronary heart and pulmonary disease (Akl et al., 2010; Raad et al., 2011; Shaikh, Vijayaraghavan, Sulaiman, Kazi, & Shafi, 2008). Studies have identified carcinogens and toxicants such as polyaromatic hydrocarbons and volatile aldehydes in hookah tobacco smoke and biomarkers of these toxicants in the urine of hookah smokers (Al Rashidi, Shihadeh, & Saliba, 2008; Daher et al., 2010; Jacob et al., 2011, 2013).

Nicotine and 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone (NNK) are the most important tobacco-specific markers of SHS and SHS exposure (Benowitz & Jacob, 1994; Hecht, 2002). Nicotine is the main addictive constituent of tobacco products; its metabolite, cotinine, is the most widely used biological marker of recent SHS exposure (Benowitz & Jacob 1994). NNK is a potent tobacco-specific pulmonary carcinogen (Hecht, 2002). Its metabolites, 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanol (NNAL) and NNAL-glucuronides (total NNAL) are consistently elevated in adult nonsmokers and children exposed to SHS (Bernert et al., 2010; Hecht, 2002). Another toxic compound, acrolein, is a strong ciliatoxic agent, also believed to contribute to lung carcinogenesis via DNA damage and inhibition of DNA repair, as well as to cardiovascular disease via its oxidant properties (Feng, Hu, Hu, & Tang, 2006; Luo et al., 2007). Acrolein is one of the most abundant, reactive toxicants in cigarette smoke (Feng et al., 2006). A major metabolite of acrolein used for biomonitoring is 3-hydroxypropylmercapturic acid (3-HPMA), which can be measured in urine (Carmella et al., 2007).

We are not aware of any previous studies that have examined SHS, THS, and carcinogen and other toxicant uptake by children who live in homes of hookah-only smokers. These measurements are crucial for assessing the possible negative health consequences to children who live in these homes.

## METHODS

### Study Design

A cross-sectional nonequivalent group comparison study was employed. Research assistants (RAs) collected data from a convenience sample ($N$ = 24) of households with hookah-only smokers ($n$ = 19): (daily hookah smokers [$n$ = 8], weekly/monthly hookah smokers [$n$ = 11]), and nonsmokers ($n$ = 5). Data were collected during three home visits during a 7-day study period between 2010 and 2011: tobacco use, demographics, a 7-Day Home Tobacco Use Diary, a 7-Day Child Observation Diary, home and household characteristics, two environmental samples (air and surface) per home, and three urine samples per child. Adult participants received $150 as an incentive. The study was approved by the Institutional Review Board of San Diego State University (SDSU).

### Inclusion Criteria

Homes were eligible if participants had lived in their current home for at least 6 months and their home was either a "smoker home" or a "nonsmoker home" as described previously (Matt et al., 2011b). "Smoker homes" were those in which residents have smoked hookah-only tobacco indoors or outdoors (on the patio or balcony) during at least 5 of the past 6 months, including the current most recent month, and have not smoked cigarettes or any other tobacco products anywhere inside the home or outdoors in the past 6 months. "Nonsmoker homes" were those in which no smokers lived and no visitors were allowed to smoke indoors or outdoors. Eligible households consisted of either a male or female, 18 years or older, hookah-only smoker or nonsmoker living with a healthy child 5 years old or younger. Hookah-only smokers were eligible if they smoked only hookah at least once a month and did not smoke any other tobacco product during the past 6 months. Nonsmokers and their children were eligible if they were not exposed to SHS from any tobacco product indoors or outdoors at their home for at least the past 6 months. "Hookah-only" smokers are referred to as "hookah smokers" throughout the remaining manuscript.

### Recruitment Efforts and Screening

The RAs recruited participants from the community via intercept brief screening interviews at community colleges, universities, malls, Arab American churches and mosques, Middle Eastern restaurants and grocery stores, and community events in the cities of San Diego, La Mesa, and El Cajon, CA. The RAs contacted eligible households by phone to invite them to participate and coordinate the first home visit to obtain an informed consent and start the study (see Supplementary Material for details on parent reported measures, child SHS exposure measures, and home visits protocol).

### Environmental Measures

*Air Nicotine*

Two air samples were collected with passive diffusion monitor badges to measure air nicotine, one in the living room and one in the child bedroom. A blank nonanalyzed badge was placed in a third room as a bogus pipeline (Matt et al., 2004). Using a home sketch floor plan drawn by the RAs identifying locations of windows, doors, smoking area, the RAs placed the monitors in each home during the first home visit for the duration of 7 days. Monitors were hung in the air about 2 m from the floor, out of reach of the children and away from doors, windows, and corners and hours in the room recorded. The badges consisted of a modified 37-mm diffusive cassette with a sodium bisulfate–treated Teflon-coated glass fiber filter. Badges were transported in ice coolers to SDSU laboratory for analyses. Nicotine was extracted into hexane and analyzed on a gas chromatograph with a nitrogen detector similar to published methods (Bernert, McGuffey, Morrison, & Pirkle, 2000). The limit of detection (LOD) was 0.02 µg/m$^3$.

*Surface Nicotine*

RAs collected two surface wipes, one from the living room for both indoor smokers and outdoor smokers, and one from the child bedroom area. Surface wipes were taken from window sills or door panels during the first home visit. To preserve the nicotine, wipes were wetted with 10 drops (1 ml) of freshly prepared 0.1% ascorbic acid using sterile pipettes, as described previously (Matt et al., 2004). Surface area wipe samples were collected with a cotton wipe (cosmetic 100% cotton) using vertical and horizontal strokes covering 100 cm$^2$ within a 10 × 10 cm disposable frame. Wipes were placed in sanitized 20 ml amber glass bottles and transported in ice coolers to SDSU laboratory for analysis. Blanks were not used due to a small budget. Nicotine extracts from wipes samples were determined using liquid chromatography tandem mass spectrometry (LC-MS/MS) with electrospray ionization (see details in Matt et al., 2011b). The LOD was 5 ng/100 cm$^2$ (0.5 µg/m$^2$).

### Biological Measures

Three child urine samples were collected by parents on Days 1, 3, and 6 to measure urinary cotinine, total NNAL, and 3-HPMA. Parents stored the urine samples in the freezer until pickup within 48 hr by RAs and transferred in a cooler to our research center laboratory. Urine samples were aliquoted, stored in a freezer (−20 °C), then sent frozen in dry ice to three laboratories.

Laboratory urine analyses for cotinine were conducted by LC-MS/MS at SDSU with a LOD of 0.05 ng/ml as previously described (Bernert et al., 2000). Laboratory urine analyses for total NNAL were conducted by LC-MS/MS at the Clinical Pharmacology Laboratory, University of California San Francisco, with a LOD of 0.25 pg/ml as previously described (Jacob et al., 2008). Laboratory urine analyses for 3-HPMA were conducted by LC-atmospheric pressure chemical ionization-MS/MS-selected reaction monitoring at the Masonic Cancer Center, University of Minnesota, with an LOD of 2 pmol/ml as previously described (Carmella et al., 2007).

### Statistical Analysis

Mann–Whitney *U* tests were used to identify differences in air nicotine, surface nicotine, and biomarkers of hookah tobacco SHS exposure in children living in the three types of homes; Wilcoxon signed-rank tests to identify differences in air and surface nicotine between living rooms and child bedrooms within all homes; Spearman's rho (ρ) correlations to identify associations between air nicotine, surface nicotine, and biomarkers; and independent *t* tests to identify differences in demographics and hookah smoking habits by type of home or type of smoker. Geometric means (GMs) and 95% confidence intervals (CIs) were computed for number of hookah heads and hours smoked in 7 days, air nicotine, surface nicotine, and biomarkers. For nondetectable values, the mean of zero and the LOD was used. All statistical tests were two-tailed with an alpha level of .05. Data analyses were conducted using SPSS version 19.

## RESULTS

Child, parent/guardian, and home characteristics are presented in Table 1. All 24 children were in excellent/good physical and mental health. About half were males with a median age of 3 years. Hookah smokers were mostly parents (94.7%, *n* = 18), males (73.7%, *n* = 14), and of Middle Eastern descent (94.7%, *n* = 18). Both hookah smokers and nonsmokers had been living in the same home for more than 6 months.

Hookah smoking habits are presented in Table 2. Hookah-only smokers were comprised of 8 daily and 11 weekly/monthly smokers. All hookah smokers owned a hookah at home and smoked only flavored hookah tobacco "Moassel" and no other tobacco products. Table 3 presents the GM and 95% CIs of air and surface nicotine levels in living rooms and child bedrooms and number of hookah heads and hours smoked in 7 days. A hookah head was defined as one hookah tobacco serving, which was equivalent to 10–20 g of hookah tobacco (Monzer, Sepetdjian, Saliba, & Shihadeh, 2008).

### Air Nicotine Levels

*Living Rooms*

Nicotine was detected in the air of the living rooms of 88% (7 of 8 homes) of daily hookah smoker homes, 60% (6 of 10 homes; one monitor missing) of the weekly/monthly hookah smoker homes compared to 20% (1 of 5 homes) of the nonsmoker homes. In daily hookah smoker homes, GM air nicotine levels were 14.3 and 4.8 times higher, respectively, than those found in the living rooms of nonsmoker homes and weekly/monthly hookah smoker homes. In weekly/monthly hookah smoker homes, GM air nicotine levels were 3 times higher than those found in nonsmoker homes.

*Child Bedrooms*

Nicotine was detected in the air of the child bedrooms of 88% (7 of 8 homes) of daily hookah smoker homes, 55% (6 of 11 homes) of weekly/monthly smoker homes compared to none (0 of 5 homes) of the nonsmoker homes. In daily hookah smoker homes, the GM air nicotine levels were significantly 41 and 13.7 times higher, respectively, than those found in the child bedrooms of nonsmoker homes and weekly/monthly hookah smoker homes. In weekly/monthly hookah smoker homes, GM air nicotine levels were 3 times higher than those found in the child bedrooms of nonsmoker homes; however, the difference was not significant.

**Table 1. Characteristics of Child Participants, Parents/Guardians, Households, and Homes ($N$ = 24)[a,b]**

| | | | | Frequency of hookah-only smoking | | |
|---|---|---|---|---|---|---|
| | Nonsmoker households ($n$ = 5), $n$ (%) | Hookah-only smoker households ($n$ = 19), $n$ (%) | $p$[c] value | Daily hookah smoker ($n$ = 8), $n$ (%) | Weekly/monthly hookah smoker ($n$ = 11), $n$ (%) | $p$ value |
| Child | | | | | | |
| Age (years) | | | | | | |
| Mean ± *SD* | 3.30 ± 0.7 | 2.68 ± 1.1 | *.262* | 2.38 ± 1.1 | 2.91 ± 1.2 | *.324* |
| Median (range) | 3.0 (2.5–4) | 3.0 (1–4.5) | | 2.3 (1–4) | 3.0 (1–4) | |
| Gender | | | | | | |
| Male | 2 (40.0) | 9 (47.4) | | 3 (37.5) | 6 (54.5) | |
| Female | 3 (60.0) | 10 (52.6) | | 5 (62.5) | 5 (45.5) | |
| Physical health | | | | | | |
| Excellent | 4 (80.0) | 10 (52.6) | | 3 (37.5) | 7 (63.6) | |
| Good | 1 (20.0) | 9 (47.4) | | 5 (62.5 | 4 (36.4) | |
| Mental health | | | | | | |
| Excellent | 4 (80.0) | 11 (57.9) | | 4 (50.0) | 7 (63.6) | |
| Good | 1 (20.0) | 8 (42.1) | | 4 (50.0) | 4 (36.4) | |
| Parent or guardian | | | | | | |
| Age (years) | | | | | | |
| Mean ± *SD* | 36.4 ± 1.5 | 39.2 ± 8.2 | *.181* | 43.1 ± 5.3 | 36.3 ± 8.9 | *.069* |
| Median | 37.0 | 40.0 | | 42 | 34 | |
| Gender | | | | | | |
| Male | 0 (0.0) | 14 (73.7) | | 4 (50.0) | 10 (90.9) | |
| Female | 5 (100) | 5 (26.3) | | 4 (50.0) | 1 (9.1) | |
| Ethnicity | | | | | | |
| Middle Eastern | 0 (0.0) | 18 (94.7) | | 8 (100) | 10 (90.9) | |
| Non-Middle Eastern White | 5 (100) | 1 (5.3) | | 0 (0.0) | 1 (9.1) | |
| Relationship to child | | | | | | |
| Mother | 5 (100) | 5 (26.3) | | 0 (0.0) | 5 (45.5) | |
| Father | 0 (0.0) | 13 (68.4) | | 7 (87.5) | 6 (54.5) | |
| Other | 0 (0.0) | 1 (5.3) | | 1 (12.5) | 0 (0.0) | |
| Household and home | | | | | | |
| Months residing in home | | | | | | |
| Mean ± *SD* | 43.0 ± 43.4 | 20.1 ± 18.4 | *.308* | 15.4 ± 10.8 | 23.5 ± 22.3 | *.355* |
| Median | 18.0 | 13.0 | | 12.5 | 13.0 | |
| Number of hookah smokers | | | | | | |
| One | 0 (0.0) | 10 (52.6) | | 4 (50.0) | 6 (60.0) | |
| Two | 0 (0.0) | 9 (47.4) | | 4 (50.0) | 4 (40.0) | |
| Type of home | | | | | | |
| House | 4 (80.0) | 7 (36.8) | | 1 (12.5) | 6 (54.5) | |
| Apartment | 1 (20.0) | 12 (63.2) | | 7 (87.5) | 5 (45.5) | |
| Number of bedrooms | | | | | | |
| Two | 1 (20.0) | 10 (52.6) | | 5 (62.5) | 5 (45.5) | |
| Three or more | 4 (80.0) | 9 (47.4) | | 3 (37.5) | 6 (54.5) | |

[a]Hookah Smoking History Questionnaire, we used open-ended questions for continuous variables.
[b]Home and Household Characteristics Form.
[c]Independent samples $t$ test for significance at $p < .05$.

Correlations among air nicotine, surface nicotine, and biomarkers are presented in Table 4. Air nicotine levels in the living rooms were positively correlated with the total number of hookah heads smoked during the 7 days, and air nicotine levels in the child bedrooms were positively correlated with air nicotine levels in the living rooms.

### Surface Nicotine Levels

*Living Rooms*

Nicotine was detected on surfaces in the living rooms of 100% (8 of 8 homes) of daily hookah smoker homes, 91% (10 of 11 homes) of weekly/monthly smoker homes compared to 40% (2 of 5 homes) of nonsmoker homes. In daily hookah smoker homes, GM surface nicotine levels in living rooms were significantly 61.1 times higher than those found in nonsmoker homes and 5.4 times higher than found in weekly/monthly hookah smoker homes. In week/monthly hookah smoker homes, GM surface nicotine levels were significantly 11.4 times higher than those found in the living rooms of nonsmoker homes.

*Child Bedrooms*

Nicotine was detected on surfaces in the child bedrooms of 100% (8 of 8 homes) of daily hookah smoker homes, 91% (10 of 11

**Table 2.** Hookah Smoking Habits of Parents/Guardians ($N$ = 24)[a]

| | | | Frequency of hookah-only smoking | | |
|---|---|---|---|---|---|
| | Nonsmoker ($n$ = 5), $n$ (%) | Hookah-only smoker ($n$ = 19), $n$ (%) | Daily hookah smoker ($n$ = 8), $n$ (%) | Weekly/monthly hookah smoker ($n$ = 11), $n$ (%) | $p$ value[b] |
| Do you currently smoke hookah daily, weekly, monthly, or not at all? | | | | | |
| Daily | N/A | 8 (42.1) | 8 (100) | 0 (0.0) | |
| Weekly | | 5 (26.3) | 0 (0.0) | 5 (45.5) | |
| Monthly | | 6 (31.6) | 0 (0.0) | 6 (54.5) | |
| How many hookah heads do you usually smoke on the day you smoke? | | | | | |
| Means ± *SD* (hookah heads) | N/A | 1.47 ± 0.772 | 2.13 ± 0.835 | 1 ± 0.0 | ***<.001*** |
| Median (range) | | 1 (1–3) | 2 (1–3) | 1 (1–1) | |
| How long do you smoke hookah on the day you smoke? | | | | | |
| Means ± *SD* (minutes) | N/A | 87 ± 84.2 | 106.9 ± 121.4 | 72.7 ± 43.6 | *.397* |
| Median (range) | | 60 (15–300) | 45 (15–300) | 60 (30–180) | |
| Which days of the week do you usually smoke hookah at home? | | | | | |
| Monday–Thursday | N/A | 12 (66.7) | 7 (87.5) | 5 (50.0) | |
| Friday | | 12 (66.7) | 7 (87.5) | 5 (50.0) | |
| Saturday | | 16 (88.9) | 8 (100.0) | 8 (80.0) | |
| Sunday | | 14 (77.8) | 8 (100.0) | 6 (60.0) | |
| What times do you usually smoke hookah at home? | | | | | |
| Mornings (6 a.m.–12:59 p.m.) | N/A | 4 (21.1) | 4 (50.0) | 0 (0.0) | |
| Afternoons (1 p.m.–5:59 p.m.) | | 7 (36.8) | 6 (75.0) | 1 (9.1) | |
| Evenings (6 p.m.–12:59 a.m.) | | 14 (73.7) | 4 (50.0) | 10 (90.9) | |
| Nights (1 a.m.–5:59 a.m.) | | 2 (10.5) | 1 (12.5) | 1 (9.1) | |
| Did you smoke hookah during the past 7 days? | | | | | |
| Yes | N/A | 16 (84.2) | 8 (100) | 8 (72.7) | |
| No | | 3 (15.8) | 0 (0.0) | 3 (27.3) | |
| Do you currently own a hookah at your home? | | | | | |
| Yes | 0 (0.0) | 19 (100) | 8 (100) | 11 (100) | |
| No | 5 (100) | 0 (0.0) | 0 (0.0) | 0 (0.0) | |
| What type of hookah tobacco do you currently smoke? | | | | | |
| Smoke only flavored hookah tobacco (Moassel) | N/A | 19 (100) | 8 (100) | 11 (100) | |
| Smoke only unflavored hookah tobacco (Ajami) | | 0 (0.0) | 0 (0.0) | 0 (0.0) | |
| Smoke both equally | | 0 (0.0) | 0 (0.0) | 0 (0.0) | |
| In the past 6 months, have any other tobacco products (cigarettes, cigar, pipe, etc.) been smoked inside your home? | | | | | |
| Yes | 0 (0.0) | 0 (0.0) | 0 (0.0) | 0 (0.0) | |
| No | 5 (100) | 19 (100) | 8 (100) | 11 (100) | |
| In the past 6 months, was hookah being smoked: | | | | | |
| Mostly inside your home | N/A | 8 (42.1) | 2 (25.0) | 6 (54.5) | |
| Only outdoors at your home | | 11 (57.9) | 6 (75.0) | 5 (45.5) | |
| If smoked outside home on patio or balcony[c] | | | | | |
| How often patio or balcony doors were open? | | | | | |
| Almost always/sometimes | N/A | 11 (100) | 6 (100) | 5 (100) | |
| Never | | 0 (0.0) | 0 (0.0) | 0 (0.0) | |

[a]Hookah Smoking History Questionnaire, we used open-ended questions for continuous variables.
[b]Independent samples $t$ test for significance at $p < .05$; statistically significant $p$ values are noted in bold.
[c]Hookah Smoking Session Form.

homes) of weekly/monthly smoker homes compared to 40% (2 of 5 homes) of nonsmoker homes. In daily hookah smoker homes, GM surface nicotine levels in child bedrooms were significantly 47.5 times higher than those found in nonsmoker homes and 1.9 times higher than found in weekly/monthly hookah smoker homes. In weekly/monthly hookah smoker homes, GM surface nicotine levels were significantly 24.5 times higher than those found in the child bedrooms of nonsmoker homes.

Surface nicotine levels in the living rooms were positively correlated, respectively, with total number of hours hookah

**Table 3. Air and Surface Nicotine Contamination in Homes of Hookah-Only Smokers Compared to Nonsmoker Homes (*N* = 24)**

| | | | | Hookah-only smoker homes by frequency of smoking | | | | |
|---|---|---|---|---|---|---|---|---|
| | Nonsmoker homes ($n$ = 5), GM (95% CI)[a] | Hookah-only smoker homes ($n$ = 19), GM (95% CI) | $p$ value[b] | Daily hookah smoker homes ($n$ = 8), GM (95% CI) | $p$ value[c] | Weekly/monthly hookah smoker homes ($n$ = 11), GM (95% CI) | $p$ value[d] | $p$ value[e] |
| Parent reported number of hookah heads smoked in 7 days | | | | | | | | |
| Heads of hookahs smoked[f] | 0 (0–0) | 4.84 (3.10–7.30) | ***.001*** | 8.48 (5.86–12.10) | ***.002*** | 3.10 (1.57–5.55) | ***.001*** | ***.016*** |
| Median | | 4 | | 7 | | 3 | | |
| Range (heads) | | 1–21 | | 4–21 | | 1–17 | | |
| Parent reported number of hours hookah smoked in 7 days | | | | | | | | |
| Hours hookah smoked[f] | 0 (0–0) | 3.84 (2.50–5.69) | ***.002*** | 5.05 (2.63–9.08) | ***.003*** | 3.12 (1.73–5.20) | ***.002*** | *.231* |
| Median | | 3.92 | | 4.21 | | 3.92 | | |
| Range (hours) | | 0.50–19 | | 1.75–19 | | 0.50–15 | | |
| Air nicotine in 7 days ($\mu g/m^3$)[g] | | | | | | | | |
| Living room | 0.03 (0.00–0.08) | 0.23 (0.05–0.45) | *.117* | 0.43 (0.02–1.00) | *.079* | 0.09 (0.02–0.17) | *.270* | *.248* |
| Range | 0.01–0.13 | 0.00–2.18 | | 0.01–2.18[h] | | 0.00–0.40 | | |
| %, Freq/$n$ above LOD[i,j] | 20%, 1/5 | 72%, 13/18 | | 88%, 7/8[k] | | 60%, 6/10[l] | | |
| Child's room | 0.01 (0.01–0.02) | 0.17 (0.02–0.35) | ***.030*** | 0.41 (0.05–0.88) | ***.005*** | 0.03 (0.01–0.04) | *.193* | ***.017*** |
| Range | 0.01–0.02 | 0.01–1.61 | | 0.02–1.61 | | 0.01–0.08 | | |
| %, Freq/$n$ above LOD | 0%, 0/5 | 68%, 13/19 | | 88%, 7/8 | | 55%, 6/11 | | |
| $p$[m] | *.463* | *.215* | | *.947* | | *.117* | | |

*(Continued)*

**Table 3. Continued**

| | | | | Hookah-only smoker homes by frequency of smoking | | | | |
|---|---|---|---|---|---|---|---|---|
| | Nonsmoker homes ($n$ = 5), GM (95% CI)[a] | Hookah-only smoker homes ($n$ = 19), GM (95% CI) | $p$ value[b] | Daily hookah smoker homes ($n$ = 8), GM (95% CI) | $p$ value[c] | Weekly/monthly hookah smoker homes ($n$ = 11), GM (95% CI) | $p$ value[d] | $p$ value[e] |
| Surface wipes nicotine (μg/m$^2$)[n] | | | | | | | | |
| Living room | 1.37 (0.00–5.85) | 31.98 (11.75–84.33) | ***.014*** | 83.67 (12.50–529.94) | ***.013*** | 15.62 (6.36–36.54) | ***.047*** | *.083* |
| Range | 0.02–13.85 | 0.01–2,741.74 | | 0.09–2,741.74 | | 0.01–158.95 | | |
| %, Freq/$n$ above LOD | 40%, 2/5 | 95%, 18/19 | | 100%, 8/8 | | 91%, 10/11 | | |
| Child's room | 1.36 (0.00–6.61) | 44.06 (16.87–112.63) | ***.012*** | 64.60 (13.25–301.01) | ***.013*** | 33.29 (9.48–111.18) | ***.036*** | *.620* |
| Range | 0.01–20.82 | 0.01–7,180.90 | | 8.10–7,180.90[o] | | 0.01–1,033.76 | | |
| %, Freq/$n$ above LOD | 40%, 2/5 | 95%, 18/19 | | 100%, 8/8 | | 91%, 10/11 | | |
| $p$[p] | *.953* | *.809* | | *.638* | | *.307* | | |

*Note.* LOD = limit of detection.
[a]GM (95% CI) = geometric mean and 95% confidence interval.
[b–e]$p$ values where derived from Mann–Whitney $U$ tests on raw data; two-tailed with an alpha level $p < .05$. $p^b$, $p^c$, $p^d$, $p^e$, respectively, [b]hookah smokers vs. nonsmokers, [c]daily smokers vs. nonsmokers, [d]weekly/monthly smokers vs. nonsmokers, [e]daily smokers vs. weekly/monthly smokers.
[f]Parent reported number of hookah heads and number of hours hookah smoked in 7 days were calculated from the 7-Day Home Tobacco Use Diary.
[g]Nicotine air samples were collected with passive diffusion monitor badges during 7 days and were placed in the living room and child room.
[h]For example, 548 ng × 1 μg/1,000 ng/(0.252 m$^3$) = 2.18 μg/m$^3$.
[i]Percentage (%) of samples with nicotine levels above the LOD: nicotine air LOD = 0.02 μg/m$^3$ per 7 days, nicotine surface wipes LOD = 0.5 μg/m$^2$.
[j]Freq/$n$ = frequency of samples with levels above the LOD/sample size of samples per group.
[k]For example, 1 surface wipe/living room/home × 8 homes = 8 wipes.
[l](1 monitor missing). Air nicotine levels were quantified in nanograms (ng) and were reported in micrograms/cubic meter (μg/m$^3$) as follows: 25 cm$^3$/min (rate of monitor air sampling) × 1,440 min/day × 1 m$^3$/106 cm$^3$ × 7 day = 0.252 m$^3$.
[m]Air nicotine living room vs. child room.
[n]Surface wipes were collected from the living room and child room, one wipe per room using 10 cm by 10 cm surface area for wiping (100 cm$^2$ = 0.01 m$^2$). Surface nicotine levels were quantified in ng and were reported in micrograms/meter squared (μg/m$^2$).
[o]For example, 71,808.9 ng × 1 μg/1,000 ng/(0.01 m$^2$) = 7,180.9 μg/m$^2$. Statistically significant $p$ values are noted in bold.
[p]Surface nicotine living room vs. child bedroom using Wilcoxon log-rank tests on raw data.

**Table 4. Spearman's Rho Correlations of Child Urinary Biomarkers of Hookah-Only Tobacco SHS Exposure With Air and Surface Nicotine**

| | Air nicotine living room | Air nicotine child bedroom | Surface nicotine living room | Surface nicotine child bedroom | Cotinine | Total NNAL | 3-HPMA | Total[a] hours hookah smoked | Total[a] number hookah smoked | Number of bedrooms in homes | Total[a] hours child exposed to SHS | Child age |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Air nicotine living room | 1 | .483[b] | .322 | .133 | .254 | .415 | .043 | .302 | .512 | −.119 | NA | NA |
| | | **.019**[c] | .134 | .544 | .242 | **.049** | .847 | .162 | **.013** | .590 | | |
| Air nicotine child bedroom | | 1 | .416 | .297 | .374 | .417 | .343 | .294 | .377 | −.322 | NA | NA |
| | | | **.043** | .158 | .072 | **.042** | .101 | .164 | .069 | .125 | | |
| Surface nicotine living room | | | 1 | .635 | .358 | .508 | .403 | .404 | .469 | −.122 | NA | NA |
| | | | | **.001** | .086 | **.011** | **.051** | **.050** | **.021** | .572 | | |
| Surface nicotine child bedroom | | | | 1 | .173 | .397 | .203 | .343 | .471 | −.160 | NA | NA |
| | | | | | .419 | **.055** | .340 | .101 | **.020** | .455 | | |
| Cotinine | | | | | 1 | .625 | .157 | .596 | .598 | −.352 | .670 | −.313 |
| | | | | | | **.001** | .465 | **.002** | **.002** | .092 | **<.001** | .137 |
| Total NNAL | | | | | | 1 | .217 | .353 | .582 | −.300 | .521 | −.432 |
| | | | | | | | .310 | .090 | **.003** | .154 | **.009** | **.035** |
| 3-HPMA | | | | | | | 1 | .140 | .131 | −.490 | .314 | −.359 |
| | | | | | | | | .513 | .541 | **.015** | .136 | .085 |

*Note.* 3-HPMA = 3-hydroxypropylmercapturic acid; NA = not applicable; NNAL = 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanol; SHS = secondhand smoke. Biomarkers units: cotinine ng/mg creatinine, NNAL pg/mg creatinine, 3-HPMA pmol/mg creatinine.

[a]In 7 days.

[b]Spearman's rho correlation coefficient (ρ).

[c]*p* value; statistically significant *p* values are noted in bold.

smoked and total number of hookah heads smoked in 7 days. Surface nicotine levels in the child bedrooms were positively correlated with surface nicotine levels in living rooms and to total number of hookah heads smoked in 7 days.

### Child Exposure to Hookah SHS

Urinary levels of cotinine, total NNAL, and 3-HPMA are presented in Table 5. We included uncorrected and creatinine-corrected maximum values of the biomarkers (GM of the highest level of three spot urine samples collected during 1 week), and the average values (GM of the average level of three spot urine samples collected during 1 week). Children living in daily hookah smoker homes were exposed to hookah SHS 1.6 times longer than children living in weekly/monthly hookah smoker homes.

Urine cotinine was detected in 75% (18 of 24 samples) of samples from children living in daily hookah smoker homes compared to 85% (28 of 33 samples) of samples from children living in weekly/monthly hookah smoker homes and 60% (9 of 15 samples) of samples from children living in nonsmoker homes. GM urine cotinine levels in children living in daily hookah smoker homes were significantly 6.5 times higher than those found in children living in nonsmoker homes and 1.8 times higher than in children living in weekly/monthly hookah smoker homes. GM urine cotinine levels in children living in week/monthly hookah smoker homes were significantly 3.7 times higher than those found in children living in nonsmoker homes.

Urine total NNAL levels were detected in 71% (17 of 24 samples) of samples from children living in daily hookah smoker homes compared to 67% (22 of 33 samples) of samples from children living in weekly/monthly hookah smoker homes and 7% (1 of 15 samples) of urine from children living in nonsmoker homes. GM urine total NNAL levels in children living in daily hookah smoker homes were significantly 37.3 higher than those found in children living in nonsmoker homes and 2.2 times higher than in children living in weekly/monthly hookah smoker homes. GM urine total NNAL levels in children living in week/monthly hookah smoker homes were significantly 17 times higher than those found in children living in nonsmoker homes.

Urine 3-HPMA was detected in all children. GM urine 3-HPMA levels in children living in daily hookah smoker homes were significantly 1.9 times higher than those found in children living in nonsmoker homes and 1.4 times higher than in children living in weekly/monthly hookah smoker homes. GM urine 3-HPMA levels in children living in week/monthly hookah smoker homes were 1.37 times higher than those found in children living in nonsmoker homes.

## DISCUSSION

This is the first study to investigate nicotine levels in indoor air and on surfaces inside homes of hookah-only smokers, and carcinogen and other toxicant uptake in children living in these homes. Our results demonstrate significantly higher exposures to nicotine, NNK, and acrolein in children who live in such homes compared to children who live in nonsmoker homes. These results point to potential dangers of hookah smoking in homes with children.

We compared our data from homes of daily hookah smokers to data from studies on daily cigarette smoker households with children. Hookah smoking differs from cigarette smoking in that daily hookah smokers smoke less frequently per day, however, for longer time per smoking session (WHO, 2006). Our sample of daily hookah smokers reported that on the day they smoke, they usually smoked an average of two hookah heads for an average of 100 min (Table 2).

All hookah smokers in our study smoked "Moassel," which contains about 30% tobacco (Khater et al., 2008). Analysis of nicotine content of 11 commercial brands of "Moassel" showed that the average nicotine content of "Moassel" was 3.35 mg/g tobacco (range, 1.8–6.3 mg/g) (Hadidi & Mohammed, 2004). Accordingly, the average nicotine content of one hookah head of 20 g Moassel is 67 mg/hookah head (range, 36–126 mg) compared to 10.2 mg/cigarette (Hadidi & Mohammed, 2004; Kozlowski et al., 1998). To date, the U.S. Food and Drug Administration (FDA) has not regulated hookah tobacco or its many flavors and additives. Although the Family Smoking Prevention and Tobacco Control Act, which gave the FDA the authority to regulate tobacco products to protect public health, became law in 2009, the FDA has been waiting for evidence of toxicological exposures to initiate regulatory practices (FDA, 2013). This study contributes to the evidence of hookah tobacco SHS toxicological exposures in children.

In contrast to beliefs that hookah tobacco is less harmful than cigarettes, the CDC (2011) reported that hookah smoking is not a safe alternative to smoking cigarettes (Akl et al., 2013). We detected relatively high levels of nicotine on surfaces in living rooms and child bedrooms of 18 out of 19 hookah smoker homes. Children living in these homes are at risk for exposure to tobacco THS throughout their homes. GM surface nicotine levels in daily hookah smoker homes were higher than levels found in indoor daily cigarette smoker homes (living rooms: GM, 83.67 vs. 51.3 μg/m$^2$; child bedrooms: GM, 64.6 vs. 41.9 μg/m$^2$, respectively) (Matt et al., 2011b).

Nicotine contamination levels on home surfaces are due to accumulation of THS over time (Quintana et al., 2013). A culturally based aspect of hookah smoking behavior may have influenced the relatively high levels of nicotine on surfaces in homes of hookah smokers. Our study sample of hookah smokers was comprised mainly of Arab Americans. Reported and observed behaviors of hookah smoker participants who smoke outdoors may have allowed hookah tobacco smoke to drift inside the home. Based on home sketches and the location of smoking, outdoor hookah smokers (11 of 19 smokers) reported smoking on the patio or the balcony near their living room door. They also reported that patio or balcony doors were either almost always or sometimes open when smoking (Table 2). Reasons for having the doors open, as identified by the RAs through direct observation, were to allow family members in and out of the home to bring food and drinks, to welcome incoming guests for socializing, or for children to play indoors and outdoors.

The majority of smokers reported that they usually smoked hookah during the afternoon or evening hours and over the weekend, times used for socializing (Table 2). This paper focused on frequency of hookah smoking; however, future investigations are needed to compare SHS and THS levels inside homes of indoor versus outdoor hookah smoker households.

**Table 5. Urinary Levels of Cotinine, Total NNAL, and 3-HPMA in Children (≤5 Years) Living in Homes of Hookah-Only Smokers and Nonsmokers (*N* = 24)**

| | | | | Hookah-only smoker homes by frequency of smoking | | | | |
|---|---|---|---|---|---|---|---|---|
| | Nonsmoker homes (*n* = 5), GM (95% CI)[a] | Hookah-only smoker homes (*n* = 19), GM (95% CI) | *p* value[b] | Daily hookah smoker Homes (*n* = 8), GM (95% CI) | *p* value[c] | Weekly/monthly hookah smoker homes (*n* = 11), GM (95% CI) | *p* value[d] | *p* value[e] |
| Child exposure to hookah SHS | | | | | | | | |
| Hours exposed in 7 days[f] | 0 (0–0) | 3.39 (2.01–5.39) | ***<.001*** | 4.42 (1.92–9.06) | ***.003*** | 2.76 (1.35–5.04) | ***.002*** | *.247* |
| Median | | 2.17 | | 4.21 | | 1.92 | | |
| Range (hours) | | 0.02–19 | | 0.33–19 | | 0.02–15.17 | | |
| Cotinine | | | | | | | | |
| Maximum[g] | | | | | | | | |
| ng/ml | 0.34 (0.07–0.68) | 0.92 (0.51–1.44) | *.082* | 1.21 (0.38–2.52) | *.079* | 0.73 (0.35–1.22) | *.157* | *.620* |
| ng/mg creatinine | 0.90 (0.23–1.92) | 3.26 (1.82–5.43) | ***.036*** | 4.13 (1.52–9.48) | ***.040*** | 2.71 (1.24–5.15) | *.079* | *.563* |
| Average[h] | | | | | | | | |
| ng/ml | 0.16 (0.06–0.26) | 0.65 (0.30–1.08) | *.063* | 0.88 (0.21–1.93) | *.143* | 0.49 (0.17–0.91) | *.062* | *.804* |
| ng/mg creatinine | 0.44 (0.16–0.79) | 2.08 (1.07–3.58) | ***.021*** | 2.85 (0.83–7.12) | ***.028*** | 1.62 (0.71–3.00) | ***.047*** | *.364* |
| Range in ng/mg | 0.05–2.86 | 0.05–28.44 | | 0.05–28.44 | | 0.05–15.85 | | |
| %, Freq/*n* above LOD[i,j] | 60%, 9/15[j] | 81%, 46/57 | | 75%, 18/24 | | 85%, 28/33 | | |
| Total NNAL | | | | | | | | |
| Maximum | | | | | | | | |
| pg/ml[k] | 0.20 (0.0–0.57) | 3.40 (1.59–6.45) | ***.010*** | 4.70 (1.10–14.46) | ***.013*** | 2.64 (1.06–5.45) | ***.012*** | *.455* |
| pg/mg creatinine | 0.56 (0.20–1.03) | 8.53 (3.92–17.47) | ***.014*** | 12.42 (2.98–44.24) | ***.013*** | 6.43 (2.53–14.63) | ***.047*** | *.364* |
| Average | | | | | | | | |
| pg/ml | 0.11 (0.0–0.24) | 2.55 (1.15–4.87) | ***.009*** | 3.70 (0.80–11.30) | ***.013*** | 1.89 (0.71–3.90) | ***.012*** | *.407* |
| pg/mg creatinine | 0.28 (0.14–0.43) | 6.68 (2.93–13.99) | ***.012*** | 10.43 (2.27–38.98) | ***.008*** | 4.75 (1.80–10.81) | ***.047*** | *.215* |
| Range in pg/mg | 0.08–1.43 | 0.08–242.20 | | 0.15–242.20 | | 0.08–66.32 | | |
| %, Freq/*n* above LOD | 7%, 1/15 | 68%, 39/57 | | 71%, 17/24 | | 67%, 22/33 | | |

*(Continued)*

**Table 5. Continued**

| | Nonsmoker homes ($n$ = 5), GM (95% CI)[a] | Hookah-only smoker homes ($n$ = 19), GM (95% CI) | $p$ value[b] | Hookah-only smoker homes by frequency of smoking: Daily hookah smoker Homes ($n$ = 8), GM (95% CI) | $p$ value[c] | Weekly/monthly hookah smoker homes ($n$ = 11), GM (95% CI) | $p$ value[d] | $p$ value[e] |
|---|---|---|---|---|---|---|---|---|
| 3-HPMA | | | | | | | | |
| Maximum | | | | | | | | |
| pmol/ml[l] | 991 (479–2,050) | 1,085 (749–1,573) | *.546* | 950 (668–1,351) | *.999* | 1,196 (658–2,173) | *.336* | *.160* |
| pmol/mg creatinine | 2,306 (1,423–3,737) | 3,665 (2,847–4,718) | *.110* | 4,076 (2,965–5,603) | *.057* | 3,392 (2,329–4,939) | *.282* | *.409* |
| Average | | | | | | | | |
| pmol/ml | 616 (339–1,121) | 714 (495–1,031) | *.414* | 676 (439–1,040) | *.770* | 744 (422–1,311) | *.282* | *.322* |
| pmol/mg creatinine | 1,600 (1,112–2,303) | 2,488 (1,890–3,277) | *.095* | 2,966 (2,074–4,240) | ***.040*** | 2,190 (1,476–3,250) | *.282* | *.186* |
| Range in pmol/mg | 553–5,864 | 300–8,889 | | 778–7,866 | | 300–8,889 | | |
| %, Freq/$n$ above LOD | 100%, 15/15 | 100%, 57/57 | | 100%, 24/24 | | 100%, 33/33 | | |

*Note.* 3-HPMA = 3-hydroxypropylmercapturic acid; LOD = limit of detection; NNAL = 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanol; SHS = secondhand smoke.
[a]GM (95% CI) = geometric mean and 95% confidence interval.
[b–e]$p$ values were derived from Mann–Whitney $U$ tests on raw data; two-tailed with an alpha level $p < .05$; statistically significant $p$ values are noted in bold. $p^b$, $p^c$, $p^d$, $p^e$, respectively,
[b]hookah smokers vs. nonsmokers, [c]daily smokers vs. nonsmokers, [d]weekly/monthly smokers vs. nonsmokers, [ge]daily smokers vs. weekly/monthly smokers.
[f]Number of hours child exposed to hookah SHS in 7 days was calculated from reported data from two diaries: 7-Day Home Tobacco Use Diary and 7-Day Child observation Diary.
[g]Maximum = GM of the highest level of three spot urine samples collected during 1 week, uncorrected and corrected by creatinine.
[h]Average = GM of the average level of three spot urine samples collected during 1 week, uncorrected and corrected by creatinine.
[i]Percentage (% rounded up) of urine samples above the LOD: cotinine LOD = 0.05 ng/ml, NNAL LOD = 0.25 pg/ml, and 3-HPMA LOD = 2 pmol/ml.
[j]Freq/$n$ = frequency of samples with levels above the LOD/sample size of samples per group, for example, three urine sample per child × 5 homes = 15 urine samples.
[k]Molecular weight for NNAL is 209: to convert from pg/ml to pmol/ml divide by 209, for example, 0.25 pg/ml = 0.0012 pmol/ml.
[l]Molecular weight for 3-HPMA is 221: to convert from pmol/ml to pg/ml multiply by 221, for example, 2 pmol/ml × 221 = 442 pg/ml.

Also, seven of eight daily hookah smokers lived in apartments. Of these, almost half (47.4%) reported having two hookah smokers per household. This is of concern in an apartment complex because hookah tobacco smoke could enter other apartments. Nonsmokers who reside in multiunit housing may share the same air space as those who smoke in adjacent units (King, Travers, Cummings, Mahoney, & Hyland, 2010). King et al. (2010) documented that SHS can transfer between living units within the same multiunit housing building. Therefore, emerging smoke-free policies in multiunit housing restricting cigarette smoking to protect residents from exposure to SHS should take into consideration hookah smoking.

Urinary levels of cotinine (GM, 0.88 ng/ml) and total NNAL (GM, 3.7 pg/ml) in children living in homes of daily hookah smokers were lower than those reported in previous studies in children exposed to cigarette SHS. In a study of children ages 10 years or younger living in homes of daily cigarette smokers ($N$ = 79) in Minnesota, the GM urinary levels of total cotinine and total NNAL were 11.9 ng/ml and 0.08 pmol/ml (16.7 pg/ml), respectively (Thomas et al., 2011). Elementary school children with parental reported exposure to SHS ($N$ = 38) in Minnesota had GM urinary levels of total cotinine and total NNAL of 12.6 ng/ml and 0.04 pmol/ml (8.4 pg/ml), respectively (Hecht et al., 2001). Children in Moldova 5–10 years old ($N$ = 7) with reported exposure to SHS at home had GM urinary levels of total cotinine and total NNAL of 4.6 ng/ml and 0.061 pmol/ml (12.8 pg/ml) (Stepanov, Hecht, Duca, & Mardari, 2006). The majority of daily hookah smokers in our sample smoked outdoors ($n$ = 2 indoor smokers, $n$ = 6 outdoor smokers), a larger sample of indoor hookah smokers is warranted in future studies.

Consistent with previous studies, we found a significant positive correlation between urinary cotinine and total NNAL (Hecht et al., 2001; Stepanov et al., 2006; Thomas et al., 2011). We also found significant positive correlations between the child urinary cotinine and total NNAL levels and indicators of tobacco smoke exposure, namely, number of hookah heads smoked and number of hours the child was exposed to hookah tobacco SHS per week. These correlations were stronger than previously observed between child urinary cotinine and total NNAL, respectively, and number of cigarettes (mean ± $SD$, 9.5 ± 5.3 cigarettes) smoked per day at home ($r$ = .37, $p$ < .001; $r$ = .30, $p$ < .01) (Thomas et al., 2011).

Urinary total NNAL levels were positively correlated with surface nicotine in the living rooms and child bedrooms. Thomas et al. (2014) detected the presence of NNK on surfaces in most homes occupied by cigarette smokers. Therefore, more research is needed to identify associations between THS and NNK uptake in nonsmokers living in smokers' homes.

Urinary total NNAL levels were negatively correlated with child age. Data from the NHANES (2007–2008) showed that children had significantly higher concentrations of total NNAL than did adults aged 20 years or older ($p$ < .001) (Bernert et al., 2010). Younger children are more likely to spend more time at home than older children, and less likely to be able to remove themselves from environments in which smoking occurs, thereby being exposed at home for a longer time to tobacco SHS (Bernert et al., 2010).

All children had 3-HPMA in their urine, independent of exposure to hookah SHS. Acrolein occurs endogenously as a lipid peroxidation product and naturally in foods (International Agency for Research on Cancer [IARC], 1995; Pan & Chung, 2002). It is also formed during the combustion of fossil fuels, wood and tobacco and during the heating of cooking oils (IARC, 1995; Yu, Chiu, Au, Wong, & Tang, 2006). Nazaroff and Singer (2004) found that population intake of acrolein from residential SHS appears to be higher than from ambient sources. Our findings are consistent with studies that demonstrate higher acrolein uptake in smokers compared to nonsmokers (Carmella et al., 2007; Hecht, Yuan, & Hatsukami, 2010). Levels of urinary 3-HPMA in our sample of children exposed to daily hookah SHS were significantly higher, respectively, than levels found in children living in nonsmoker homes (GM, 2,966 vs. 1,600 pmol/mg; $p$ = .040) and were 1.6 times higher than levels found in a previous study of 21 adult nonsmokers (mean, 1,900 pmol/mg) (Carmella et al., 2007).

We found higher levels of 3-HPMA in children living in smaller houses or apartments. Urinary 3-HPMA levels were negatively correlated with the number of bedrooms in homes. Using smoking machines, Daher et al. (2010) found that a single hookah use session emitted 1,135 ± 97 µg acrolein in the sidestream smoke; however, two studies found inconsistent average yields of acrolein in the gas phase of mainstream hookah smoke generated using 10 g of flavored hookah tobacco, a low yield of 11.3 and a high yield of 892 µg/smoking session (Al Rashidi et al., 2008; Schubert, Heinke, Bewersdorff, Luch, & Schulz, 2012).

Our findings inform targeted trials to reduce SHS exposure among children, especially those with illnesses, who live in homes of hookah smokers (Stotts et al., 2011, 2013; Tyc et al., 2012). Such trials are important in the context of the studies successfully completed in the last 20 years showing that SHS exposure can be reduced when families are coached to do so (Hovell et al., 1994, 2000, 2002, 2009, 2011, 2013; Klepeis et al., 2013; Meltzer, Hovell, Meltzer, Atkins, & de Peyster, 1993).

### Limitations and Recommendations

This study is limited by a small sample size and the use of a convenience sample, thus limiting power and generalizability. Most measures had wide CIs that most likely explain some of the nonsignificant findings. Our hookah smoking participants were almost exclusively Middle Easterners and the nonsmokers were non-Middle Easterner Whites. The detection of low levels of nicotine in two of five homes of nonsmokers could be due to drifting tobacco smoke or that surface nicotine levels were not corrected with field blanks. A field blank is a cotton round that is handled and transported with actual samples but is not used to wipe target surfaces (Quintana et al., 2013). Quintana et al. (2013) recommended that nicotine levels detected on field blanks are subtracted from those found on actual surface wipes to control for contamination from extraneous nicotine sources other than those targeted by the measure.

Future efforts are needed with larger sample sizes among various populations, housing arrangements, climates, taking into consideration the additive effect of migrating smoke, indoor versus outdoor smoking, and the use of surface wipe blanks to provide a more refined assessment of toxicant and carcinogen exposure from hookah tobacco SHS.

## CONCLUSIONS

Our data provide the first evidence that children living in homes of hookah smokers are at risk of exposure to nicotine and the

tobacco-specific carcinogen NNK. All children had detectable levels of 3-HPMA with the highest levels in children living in homes of daily hookah smokers. Exposure to acrolein in non-smokers warrants further research. Our findings suggest that daily and occasional hookah use in homes present a potentially serious threat to children's long-term health. Our results call for clinical trials to assist hookah smoking families to reduce exposure to children and/or quit smoking completely, call for regulatory actions to limit toxicants in hookah products, and call to action for the implementation of voluntary smoke-free home rules.

## SUPLEMENTARY MATERIAL

Supplementary material can be found online at http://www.ntr.oxfordjournals.org

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## DECLARATION OF INTERESTS

*NLB has provided expert testimony in tobacco litigation related to nicotine addiction in the past 5 years.*

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