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Levels of Urine Cotinine from Hookah Smoking and Exposure to Hookah Tobacco Secondhand Smoke in Hookah Lounges and Homes

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Abstract

Background: Nicotine, an addictive drug, is present in all forms of tobacco products, including hookah tobacco, which is not yet regulated in the United States.
Objectives: This study aimed to investigate the uptake of nicotine in hookah smokers and non-smokers exposed to secondhand smoke (SHS) at indoor hookah social events in natural settings where hookah tobacco was smoked exclusively.
Patients and Methods: We quantified cotinine, a metabolite of nicotine, in the urine of 105 hookah smokers and 103 non-smokers. Participants provided spot urine samples the morning of and the morning after attending an indoor hookah-only smoking social event at a hookah lounge or in a private home.
Results: Following a social event where exclusively hookah tobacco was smoked, urinary cotinine levels increased significantly 8.5 times (geometric mean (GM): 16.0 ng/mg to 136.1 ng/mg) among hookah smokers, and 2.5 times (GM: 0.4 ng/mg to 1.0 ng/mg) among non-smokers exposed exclusively to hookah tobacco SHS. Among hookah smokers, the highest increase in urinary cotinine levels post a hookah event was found in occasional hookah smokers in which GM levels increased significantly 31.2 times post smoking (from 2.0 ng/mg to 62.3 ng/mg). Reported reasons for preference to smoke hookah at home by hookah smokers who attended a hookah social event in a private home included recreational purposes, socializing with friends and family, 'Me' time and relaxing at home, more comfortable to smoke hookah at home, owning a hookah and hookah tobacco, eating and drinking while smoking hookah, and saving money by smoking at home and not going to hookah lounges.
Conclusions: Hookah tobacco smoke is a source of substantial nicotine exposure. Our results call for protecting hookah smokers' and non-smokers' health by requiring accurate hookah tobacco labels, raising taxes on hookah tobacco, reducing the spread of hookah lounges, and encouraging voluntary bans on smoking hookah tobacco in private homes.

Keywords: Hookah Smoking, Waterpipe Smoking, Cotinine, Nicotine, Secondhand Smoke

1. Background

Nicotine, an addictive drug, is present in all forms of tobacco products, including hookah tobacco (1-4). The nicotine content of flavored hookah tobacco varies (3, 4). In contrast to the misleading false ingredient statement '0.05% nicotine' that is portrayed on most hookah tobacco packages (5, 6), the average nicotine content of 11 brands of flavored hookah tobacco was 3.4 mg/g tobacco (range, 1.8 - 6.3) (4). This nicotine concentration is less than that reported for cigarettes (mean, 13.8 mg/g tobacco; range, 9.8 - 18.2) (4). However, hookah smokers smoke 10 - 20 g hookah tobacco head per one hookah smoking session (3). The average nicotine content of 20 g flavored hookah head was 67 mg ranging from 36 to 126 mg (4).

Cotinine, the major metabolite of nicotine, is the most widely used biomarker of recent tobacco use and exposure to secondhand tobacco smoke (SHS) (7, 8). A review paper of four studies that measured urine cotinine levels associated with hookah smoking in four countries (Lebanon, Jordan, Kuwait, and India) indicated that daily use of the hookah tobacco produced a 24-hr urinary cotinine level of 785 ng/mL (95% CI: 578 - 991 ng/mL) (9). Two clinical studies and one laboratory research study in the United States (U.S.) demonstrated elevated plasma cotinine levels following smoking hookah tobacco (10-12).

Studies investigating hookah smoking in natural set-

tings are lacking (13). In the U.S., we identified only one study that measured urinary cotinine among hookah smokers in a natural setting in a hookah lounge, and found that urinary cotinine increased significantly 4.7 times after smoking hookah tobacco (Geometric mean (95% CI), 11.8 ng/mg creatinine (7.21 - 19.2) to 55.3 ng/mg creatinine (33.9 - 90.1)) (14).

Hookah smokers smoke at hookah lounges as well as in private homes (14-16). We previously found that 34.3% (n = 261) of a representative sample of 760 U.S. college student ever hookah users, and 56% (n = 210) of 458 Arab American hookah smokers, reported that they first tried to smoke hookah in a home setting, either at their home or at a friend's home (15, 17). Two other studies found that 43.4% to 79.0% of U.S. hookah-smoking university students surveyed reported smoking hookah at home or in their dormitory (18, 19). A study in Canada and England, and another in Syria found that private homes represent a social setting where hookah tobacco smoking takes place (20, 21).

There is limited research on the impact of SHS exposure from hookah tobacco use on non-smokers, particularly in private homes (13). The centers for disease control and prevention (CDC) reported that exposure to SHS has been causally linked to cancer, respiratory, and cardiovascular diseases (22). The CDC report was based on tobacco products other than hookah tobacco. Research on SHS tends to focus on cigarettes; however, hookah smoking, another method of tobacco use, has not been sufficiently studied as a source of SHS.

Hookah (waterpipe) smoking is the inhaling of hookah tobacco smoke that has been generated by heating hookah tobacco with burning charcoal and passed through a partially-filled water jar. Hookah tobacco smoking has been associated with increased risk for lung and oral cancers, coronary heart disease, and pulmonary disease (23, 24). Hookah smoking is on the rise in the U.S. In 2015 ever hookah use was reported nationally by 33.8% of male and 28.4% of female undergraduate college students (25). This is alarming since a 2016 consensus statement on assessment of hookah smoking concluded that accumulating evidence suggests that hookah smoking can lead to nicotine dependence (26).

2. Objectives

This is the first study, to our knowledge, that aimed to measure cotinine in the urine of hookah smokers and non-smokers exposed exclusively to hookah tobacco SHS during indoor hookah smoking social events in private homes compared to their counterparts in hookah lounges.

We previously reported in detail the reasons hookah smokers smoke in hookah lounges (15). In this paper, we report the reasons for preference to smoke hookah inside private homes, thereby informing points of intervention to control hookah use, and SHS exposure to non-smokers socializing or living in hookah smokers' homes.

3. Patients and Methods

We have previously published a detailed description of the methods used for this study (27). Briefly, we employed a pre and post group comparison study design and collected data from a convenience sample (N = 208) of adult exclusive hookah smokers (n = 105) and non-smokers (n = 103) residing in San Diego County, California. Participants received $75 as an incentive. San Diego State University (SDSU) Institutional Review Board approved the study protocol.

We recruited hookah smokers and their non-smoker relatives and/or friends from the community via brief intercept screening interviews. Eligible participants were 18 years or older, hookah smokers, or non-smokers. Hookah smokers were eligible if they had smoked exclusively hookah tobacco and had not used any other tobacco product in the past 30 days. Non-smokers were eligible if they had not been exposed to SHS from any tobacco product other than hookah tobacco in the past 30 days. Non-smokers with $\leq$ 10 ng/mL saliva cotinine were included in the study.

During a group training on data collection in our research center, participants provided informed consent, received two coded urine cups, and completed a tobacco use history questionnaire that included past and current hookah and other tobacco product use, smoking rules in homes, and demographic questions.

Participants in groups of 6 to 12, comprised of hookah smokers and non-smokers, attended indoor social events either in a hookah lounge or in a private home, during the evening hours, where flavored hookah tobacco (*Moassel*) was exclusively smoked. Each hookah smoker ordered at least one flavored hookah tobacco head packed in a hookah with one hose; however, almost all participants (92.9%) reported sharing with other smokers. To observe any evidence of other tobacco use or non-tobacco "herbal" use during the events, two research assistants (RAs) were present during the entire event at hookah lounges and homes. During the hookah event, hookah smokers counted the number of hookah heads they and other patrons smoked as described previously (27).

Participants provided two first-void spot urine samples the morning of the hookah event day and the following morning. Participants stored the samples in a freezer until transferred frozen to our laboratory. Urine samples were aliquoted and stored in a freezer (-20°C), then sent frozen

to SDSU laboratory for analyses. The SDSU laboratory conducted urinary analyses for cotinine by LC-MS/MS with a limit of detection (LOD) of 0.05 ng/mL as previously described (28), and conducted urinary analyses for creatinine by LC-MS/MS that was linear from 0.3 - 1000 mg/dL.

3.1. Statistical Analyses

The following analyses were conducted using SPSS version 23 and Stata version 11: Wilcoxon signed-rank tests to identify within-person differences in cotinine levels pre and post hookah events; Mann-Whitney U tests to identify differences in pre-to-post event change in cotinine levels by location of hookah event and by hookah use pattern; Pearson correlation coefficients (r) to determine associations of cotinine levels in a, post hookah events and b, pre-to-post event change in cotinine levels with time spent at events, and with number of hookah heads smoked by the participant, and by other hookah smokers; independent t-tests or chi-square tests, as appropriate, to identify differences in demographics and hookah smoking behaviors by smoking status; Spearman's Rho coefficients (ρ) to determine associations of post hookah event cotinine and pre-to-post event change in cotinine with corresponding cotinine levels. Uncorrected (ng/mL) and creatinine-corrected (ng/mg creatinine) arithmetic means and standard deviations (SD), geometric means (GM) and 95% confidence intervals (CI), medians and 5th and 95th percentiles, and minimum and maximum levels were computed for cotinine. Monthly and occasional hookah smokers were combined and renamed occasional hookah smokers. All statistical tests were two-tailed; statistical significance was set to $\alpha \leq 0.05$.

For open-ended questions, an a priori codebook was developed by the principal investigator (PI) and reviewed by the study team. Participants' responses were manually grouped into categories by 2 coders comprised of the PI and the data manager. The code book was updated by emerging themes. Category percentages and direct quotes are presented.

Throughout the remainder of the manuscript, location of hookah event is referred to as either a hookah lounge or a private home; 'ng/mg creatinine' as 'ng/mg'; 'indoor hookah-only smoking social events' as 'hookah events', 'hookah lounge events' or 'home events'; 'hookah tobacco smoking' as 'hookah smoking'; and 'pre-to-post hookah event change in urinary cotinine levels' as 'pre-to-post change in cotinine'. Creatinine-corrected cotinine findings are discussed below.

4. Results

Detailed description of demographics and hookah smoking behaviors during the hookah smoking event were previously published (27). Table 1 presents a brief description of the demographics. Hookah smokers and non-smokers did not differ significantly by gender, racial/ethnic makeup, body mass index or time spent at hookah events (median, 180 minutes). Hookah smokers were significantly younger than non-smokers (median, 22 years vs. 28 years), respectively. About half of the hookah smokers (50.5%) and about a third of non-smokers (38.8%) were Arab Americans, followed by Whites (17.5%, 24.3%), respectively. Hookah smokers were daily (19.1%), weekly (43.8%), or monthly/occasional (37.1%) smokers who exclusively smoked flavored hookah tobacco (*Moassel*).

Daily hookah smokers at hookah lounges smoked more hookah heads than their counterparts in homes (median, 10 hookah heads vs. 2 hookah heads), respectively. No significant difference was found in number of hookah heads smoked by location of hookah event among weekly or occasional smokers (27). Daily hookah smokers smoked more hookah heads than weekly (median, 10 hookah heads vs. 3 hookah heads) and occasional (median, 10 hookah heads vs. 2 hookah heads) hookah smokers at hookah lounges; however, no significant difference was found between groups in home events (median, daily: 2 hookah heads, weekly: 2 hookah heads, occasional: 3 hookah heads) (27).

Among hookah smokers overall, pre-to-post event change in cotinine levels was positively correlated with number of hookah heads smoked at home events (r = 0.328, P = 0.028); the correlation was not significant for hookah lounge events (P = 0.803). This may be explained in that the reported number of hookah heads smoked by hookah smokers other than the participants during the hookah events was higher in hookah lounges than in homes (median, 81 hookah heads vs. 21 hookah heads), respectively.

4.1. Exposure to Nicotine

Creatinine-corrected cotinine values pre and post a hookah event are presented in Table 2 (see supplementary file Appendix 1 for uncorrected cotinine values). All hookah smokers and non-smokers in our study had cotinine in their urine after attending a hookah event. In hookah smokers, overall, GM urinary cotinine levels increased significantly 8.5 times post hookah event (from 16.0 ng/mg to 136.4 ng/mg).

Among daily and weekly hookah smokers, GM urinary cotinine levels increased significantly 2.7 and 4.9 times post a hookah event, respectively. The highest increase

Table 1. Characteristics of Hookah Smokers and Non-Smokers (N = 208)[a,b]

Variables	Hookah Smokers (N = 105)	Non-Smokers (N = 103)	P Value[c]
Age, y			0.001[d]
Mean (± SD)	26.9 (± 10.5)	32.0 (± 12.0)	
Median (minimum - maximum)	22 (18 - 61)	28 (18 - 67)	
Gender			0.333
Male	57 (54.3)	49 (47.6)	
Female	48 (45.7)	54 (52.4)	
Race/ethnicity			0.179
Arab American	52 (50.5)	40 (38.8)	
White, Caucasian	18 (17.5)	25 (24.3)	
Mexican, Hispanic, or Latino	8 (7.8)	13 (12.6)	
Black or African American	2 (1.9)	6 (5.8)	
Other	23 (22.3)	19 (18.5)	
Do you currently smoke hookah?[e]			
Daily	20 (19.1)	0 (0.0)	
Weekly	46 (43.8)	0 (0.0)	
Monthly/Occasionally	39 (37.1)	0 (0.0)	
Did you smoke hookah during the past 7 days?			
Yes	76 (72.4)	0 (0.0)	
No	29 (27.6)	103 (100)	
Home hookah smoking restriction			< 0.001[d]
Allowed everywhere/ certain location	86 (86.0)	38 (38.8)	
Not allowed anywhere	14 (14.0)	60 (61.2)	
Time spent at a hookah lounge event, min			0.228
Median (5 - 95 percentile)	180.0 (175 - 200)	180.0 (175 - 205)	
Time spent at a hookah home event, min			0.908
Median (5 - 95 percentile)	180.0 (180 - 226)	180.0 (180 - 200)	
Number of hookah heads smoked by participants			-
Median (5 - 95 percentile)	2 (1 - 12)	-	
Did you share the hookah with anyone?			-
Yes	92 (92.9)	-	
No	7 (7.1)	-	

[a] Values are expressed as No. (%).
[b] Due to missing values, number of categories of some variables do not sum to the total sample size
[c] P Smokers vs. non-smokers: P values were derived from Mann-Whitney U tests; two-tailed alpha level P < 0.05
[d] Significant levels.
[e] Daily, at least once each day; Weekly, at least once each week but less than daily; Monthly, at least once each month but less than weekly; Occasionally, at least once a year but less than monthly; monthly and occasional hookah smokers were combined and renamed occasional hookah smokers in the manuscript.

post a hookah event was among occasional hookah smokers in which GM urinary cotinine levels increased significantly 31.2 times post hookah event (from 2.0 ng/mg to 62.3 ng/mg).

The highest pre and post hookah event GM urinary cotinine levels were among daily hookah smokers (106.0 ng/mg and 285.9 ng/mg), respectively. Pre hookah event GM urinary cotinine levels among daily hookah smokers were significantly 53 and 265 times higher, respectively, than those found in occasional smokers and non-smokers. Also, post hookah event GM urinary cotinine levels among daily hookah smokers were significantly 4.9 and 286 times higher, respectively, than those found in occasional smokers and non-smokers.

Among non-smokers, overall, GM urinary cotinine levels increased significantly 2.5 times post hookah event (from 0.4 ng/mg to 1.0 ng/mg).

4.2. Exposure to Nicotine by Location of Event

Creatinine-corrected cotinine values pre and post a hookah event by smoking status and location of event are presented in Table 3 (see supplementary file Appendix 2 for uncorrected cotinine values).

The change in pre-to-post event cotinine levels among hookah smokers was not significantly different between

Table 2. Creatinine-Corrected Urinary Cotinine[a] levels in Adults (≥ 18 Years) Pre and Post an Indoor Hookah-Only Social Event, by Smoking Status (N = 208)

Variables	Hookah-Only Social Event (N = 208), ng/mg creatinine[b]		Ratio[c]	P Value[d]
	Pre Event	Post Event		
All hookah smokers (n = 105)				< 0.001[e]
Mean ± SD[f]	207.7 ± 539.4	318.3 ± 430.9		
GM (95% CI)[g]	16.0 (8.9 - 28.6)	136.4 (100.5 - 185.2)	8.5	
Median (5 - 95 percentile)	48.3 (0.2 - 876.8)	136.1 (15.1 - 1187.1)	2.8	
Minimum - Maximum	0.02 - 4558.3	0.1 - 2410.5)		
% above LOD (Freq/n)[h,i,j]	98 (101/103)	100 (104/104)		
Daily hookah smokers (n = 20)				0.001[e]
Mean ± SD	231.9 ± 244.8	455.9 ± 369.27		
GM (95% CI)	106.0 (46.5 - 241.7)	285.9 (144.8 - 564.4)	2.7	
Median (5 - 95 percentile)	156.9 (1.4 - 829.3)	346.4 (32.4 - 1315.9)	2.2	
Minimum - Maximum	0.7 - 900.4	1.2 - 1319.3		
% above LOD (Freq/n)	100 (20/20)	100 (20/20)		
Weekly hookah smokers (n = 46)				0.002[e]
Mean ± SD	308.4 ± 762.11	375.4 ± 485.7		
GM (95% CI)	38.3 (18.0 - 81.6)	189.0 (132.5 - 269.5)	4.9	
Median (5 - 95 percentile)	77.5 (0.5 - 1195.8)	196.7 (40.2-1187.1)	2.5	
Minimum - Maximum	0.2 - 4558.3	27.2 - 2410.5		
% above LOD (Freq/n)	100 (46/46)	100 (46/46)		
Occasional hookah smokers (n = 39)				< 0.001[e]
M ± SD	69.5 ± 183.72	176.6 ± 353.7		
GM (95% CI)	2.0 (0.8 - 4.9)	62.3 (35.3 - 110.0)	31.2	
Median (5 - 95 percentile)	1.0 (0.03 - 542.0)	90.3 (1.7 - 1143.8)	90.3	
Minimum - Maximum	0.02 - 833.8	0.1 - 1956.4		
% above LOD (Freq/n)	95 (35/37)	100 (38/38)		
Non-smokers (n = 103)				< 0.001[e]
M ± SD	1.1 ± 1.78	2.4 ± 4.66		
GM (95% CI)	0.4 (0.3 - 0.5)	1.0 (0.8 - 1.2)	2.5	
Median (5 - 95 percentile)	0.3 (0.02 -5.4)	0.9 (0.1 - 10.8)	3.0	
Minimum - Maximum	0.01 - 9.2	0.04 - 25.5		
% above LOD (Freq/n)	98 (100/102)	100 (93/93)		
	P value[k]	P value[k]	P value[l]	
Daily vs. weekly	0.135	0.048[e]	0.276	
Daily vs. occasional	< 0.001[e]	< 0.001[e]	0.017[e]	
Daily vs. non-smoker	< 0.001[e]	< 0.001[e]	< 0.001[e]	
Weekly vs. occasional	< 0.001[e]	0.002[e]	0.116	
Weekly vs. non-smoker	< 0.001[e]	< 0.001[e]	< 0.001[e]	
Occasional vs. non-smoker	0.001[e]	< 0.001[e]	< 0.001[e]	

[a] Cotinine is a metabolite of nicotine.
[b] Cotinine values are corrected with creatinine (ng/mg creatinine).
[c] Ratio, Ratio of post to pre hookah event cotinine GMs and medians.
[d] P Hookah events: pre vs. post event.
[d] P values were derived from Wilcoxon signed-rank tests; two-tailed alpha level P < 0.05.
[e] Significant levels.
[f] Mean ± SD, Arithmetic mean and standard deviation.
[g] GM (95% CI), Geometric mean and 95% confidence interval.
[h] % Above LOD, Percentage of urine samples above the limit of detection (LOD); cotinine LOD, 0.1 ng/mL.
[i] Freq/n, Frequency of samples with levels above the LOD / n-size of samples per group.
[j] Missing values due to interference (n = 4) or missing urine samples (n = 1).
[k] P cotinine levels by smoking frequency status. P values were derived from Mann-Whitney U tests; two-tailed alpha level P < 0.05.
[l] P Pre to post event change in cotinine levels by smoking frequency status. P values were derived from Mann-Whitney U tests; two-tailed alpha level P < 0.05.

hookah lounges and homes, however was significant in non-smokers (P = 0.013).

Among hookah smokers, GM urinary cotinine levels increased significantly 8.6 and 8.4 times post hookah event (hookah lounge, from 14.5 ng/mg to 124.7 ng/mg; home, from 17.8 ng/mg to 150.2 ng/mg).

Among non-smokers, GM urinary cotinine levels increased significantly 3.3 and 1.8 times post hookah event (hookah lounge, from 0.4 ng/mg to 1.3 ng/mg; home, from 0.4 ng/mg to 0.7 ng/mg).

4.3. Reasons for Smoking Hookah at Home

Supplementary file Appendix 1 presents responses by hookah smokers in home events (n = 50) to the open-ended question: 'What are the reasons you prefer to smoke hookah at home?' About half of the responses indicated that participants preferred to smoke hookah at home for recreational purposes (26.9%) and to socialize with friends and family (24.1%). Responses ranged from doing smoke tricks, smoking while watching TV, smoke at home for fun and when bored, and smoking at home while socializing with friends or family members.

'Me' time and relaxing at home (19.9%) was the third most reported reason for smoking at home. Responses ranged from simply "to have some 'Me' time" to "I smoke hookah at home to relax after a long day". A total of 12.8% of the responses indicated that participants felt more comfortable to smoke hookah at home especially when they did not want to drive or stay outside the house late.

Other reasons included owning a hookah and hookah tobacco (7.1%), eating and drinking while smoking hookah (5.7%), and saving money by smoking at home and not going to hookah lounges (3.5%).

5. Discussion

We quantified uptake of nicotine in hookah smokers and non-smokers exposed exclusively to hookah tobacco SHS in indoor hookah smoking social events in natural settings: private homes and hookah lounges. Our results demonstrated higher exposures to nicotine post hookah events in both hookah smokers and non-smokers exposed to hookah tobacco SHS in both home and hookah lounge settings. Both before and after hookah events, GM urinary cotinine levels in daily and weekly hookah smokers were significantly higher than in non-smokers. Furthermore, among hookah smokers overall, pre-to-post event change in cotinine levels was positively correlated with number of hookah heads smoked at home events. These results suggest that hookah tobacco smoking is a source of exposure to the addictive drug nicotine and should be included in tobacco control strategies.

We identified only one study in the U.S. that assessed levels of urine cotinine resulting from hookah smoking in a natural setting in a hookah lounge (14). The study reported a significant increase (4 times) in the excretion of cotinine after smoking hookah tobacco in a hookah lounge (n = 47); the urinary cotinine levels were similar to our study in pre-exposure levels (GM, 14.4 ng/mg vs. 14.5 ng/mg), however, post-exposure levels were 2.1 times lower than observed in our study (GM, 59.3 ng/mg vs. 124.7 ng/mg), respectively (14). The overall trend is higher in our study, showing an 8.6-fold increase vs. a 4-fold increase in GMs post a hookah lounge visit (14). This variability may be explained in part in that participants in our study spent more time during the hookah lounge visit (mean, 182 minutes vs. 101 minutes), and smoked more hookah heads (mean, 3.67 heads vs. 1.5 heads) (14, 27).

To date, we did not identify studies in the U.S. that assessed levels of urine cotinine resulting from hookah smoking in private homes. Beside hookah lounges, hookah smokers smoke hookah tobacco while socializing in their homes or in friends' or relatives' homes (15, 16). We did not find a significant difference in change in urine cotinine levels pre-to-post hookah event between hookah smokers in hookah lounges vs. in private homes. Therefore, future research and hookah tobacco preventive measures and control should include both natural locations where hookah smoking is allowed in hookah lounges and in homes.

We also were not able to find data on urinary cotinine levels in tobacco smokers and non-tobacco smokers exposed to tobacco SHS in a nationally representative sample of the U.S. population via the National Health and Nutrition Examination Survey (NHANES). NHANES provides serum cotinine levels in tobacco smokers (cigarettes, cigars) (29). Because collecting urine samples are less invasive than blood samples, and in order to compare our results to a representative sample of tobacco smokers and non-tobacco users exposed to SHS in the U.S., we suggest that NHANES and other national surveys that measure plasma cotinine also provide urine cotinine values, and include hookah tobacco smoking in future assessments.

5.1. Hookah Tobacco SHS Exposure

To date, research focusing on the impact of SHS exposure from hookah tobacco smoking on non-smokers, particularly in natural settings is limited (13). We found that passive exposure to hookah tobacco SHS in non-smokers resulted in a significant increase, 3.3 times and 1.8 times, respectively, in GM urinary cotinine levels post hookah social event in hookah lounges and in homes. Urine cotinine levels among non-smokers exposed to hookah tobacco SHS

Table 4. Responses to the Open-Ended Question: Why do You Prefer to Smoke Hookah at Home? (N = 50)[a]

Variables	No. (%)	Quotes
Recreation	38 (26.9)	'Recreational, practicing my "o" s, practice doing smoke tricks, I smoke hookah while watching TV, because I enjoy it at home, I like it at home, I smoke at home because it is fun, it is fun at home, playful, entertaining, have a good time a home, my hobby at home, it is a time passing activity, when nothing to do at home I smoke, when I am bored I smoke.'
Socialization	34 (24.1)	'Good for socializing, hookah parties at home, more fun with friends at home, I enjoy smoking hookah with all my friends at home, I like to smoke at home to meet up with my friends in my apartment, it is something to do when my friends are hanging out with me at home, I usually hang out with my friends when I smoke, my friends always come over to smoke and play games, it is something chilling to do with a group of friends, spending time with friends, I only smoke with friends and family never alone, have a good time with friends, I enjoy it with my mother in law, I like to smoke at home when my sons and daughters gather, I like to smoke hookah at home because sometimes my brothers come over and smoke hookah, I like it because I smoke with my husband in the house.'
'Me' time and relaxing at home	28 (19.9)	'To have some "me" time, it is a priority for me, I am passionate about it, I don't smoke outside the house because I get shy, I like to smoke alone in the house, because there is no noise at home, I love hookah because I learned to smoke it at home since I was little, I smoke hookah at home to relax after a long day, I smoke hookah at home because I feel more relaxed, keeps me thinking about my life, just to relax on the porch after a hard day at work, if I don't smoke hookah I get nervous, a daily thing.'
More comfortable at home	18 (12.8)	'I prefer to smoke at my home because I would be more comfortable, it is much more comfortable to be at home, I am more comfortable at home, I like smoking at home, sometimes I smoke hookah at my friend's home, but I like to smoke hookah at home, I smoke hookah at home when I feel tired or do not need to go out, it is easier for me than going out, I do not worry about having to drive home late at night from a hookah lounge, I smoke hookah at home because I can stay out of trouble that happens in hookah lounges.'
Own hookah and hookah tobacco	10 (7.1)	'We own a hookah, I like to smoke it at home because I like to make it by myself, love the different flavors I buy, I love the taste of the tobacco I have, I like to smoke at home because it smells nice.'
Food and drinks	8 (5.7)	'Goes good with drinking beer, I prefer to smoke hookah at home and drink coffee comfortably at home, I smoke hookah while I am drinking at home, I smoke hookah after a heavy meal, I eat while smoking as I wish, I drink tea or whisky sometimes, to enjoy it with food and drinks.'
Save money	5 (3.5)	'I can save money when I smoke at home because it is about $15 - 20 every time I go, it costs too much to go to hookah lounge, cheaper at home.'

[a] Participants who smoked at home (n = 50) provided more than one response for a total of 141 responses.

ranged from 0.12 - 25.5 ng/mg post hookah lounge event, and 0.04 - 13.1 ng/mg post home hookah event.

We were also the first to find that GM urinary cotinine levels in children living in daily hookah smoker homes and weekly/monthly hookah smoker homes were significantly 6.5 times and 3.7 times higher, respectively, than those found in children living in non-smoker homes (16). Since there is no level of exposure to tobacco smoke considered to be risk free (30), exposure to SHS should be minimized in order to protect the health of non-smoker adults and children socializing or living with hookah smokers.

Furthermore, optimal cut-off points for biomarker values to distinguish tobacco use versus no tobacco use have been determined for tobacco use other than hookah use. For example, a urinary cotinine of 50 ng/mL and 31.5 ng/mL were determined, respectively, to discriminate smokers from non-smokers, and smokers from non-smokers exposed to SHS (31, 32). We suggest that future research identify urine cotinine cut-off values to distinguish among hookah smokers, non-smokers exposed to hookah tobacco SHS, and non-smokers. Additionally, for disease epidemiology, it will be important to consider investigating the adverse effect of the cumulative dose of low cotinine levels due to chronic exposure to hookah tobacco SHS.

5.2. Multidimensional Stimuli to Dependence

The causes of nicotine dependence among hookah smokers are likely multidimensional (3, 20, 33). Therefore, studies are needed to investigate the effect of chronic nicotine exposure within the context of various stimuli that may induce tobacco dependence in daily hookah smokers versus in occasional hookah smokers with intermittent nicotine exposure. We found that among daily hookah smokers, GM urinary cotinine levels increased 2.7 times post event in daily hookah smokers, as compared to 31.2 times in occasional hookah smokers. This variation by hookah smoking status in changes in GM urinary cotinine levels due to smoking hookah was partly the result of differences in pre hookah event cotinine levels; pre-event GM urinary cotinine levels were 53 times higher in daily hookah smokers than in occasional smokers (106.0 ng/mg vs. 2.0 ng/mg).

We have previously identified stimuli to practice the habit of hookah smoking in hookah lounges, such as the high density of hookah lounges and proximity to colleges and homes, social aspects, and the availability of a variety of hookah tobacco flavors (15). In this paper, we identified stimuli to smoke in private homes, such as socializing while smoking with family and friends who prefer to smoke at home, being more comfortable smoking at home, eating dinner/lunch while smoking hookah

(hookah lounges in the U.S. are not allowed to sell foods to their hookah smoking customers), owning a hookah, and saving money by smoking at home instead of going to hookah lounges. These stimuli could be included as points of intervention in public health programs to curb the spread of hookah use in private homes.

A few of our participants tried to save money by smoking in their private homes. Such stimulus that encourages hookah smoking at home suggests raising excise taxes on hookah tobacco products to increase the burden of smoking. A study in Lebanon estimated that a 10% rise in the price of hookah tobacco would result in a 14.5% relative decrease in its home-based consumption (34, 35).

Hookah tobacco smoke inside homes is hazardous to the health of non-smokers who live or socialize with hookah smokers in their homes (16). While previously we suggested curbing the spread of hookah lounges (15, 27), our previous and present findings reported in this paper suggest encouraging banning hookah tobacco smoking inside private homes (27). Efforts to pass regulations to ban smoking in public housing, and to encourage voluntary bans of smoking in private homes (36), should be extended to include hookah tobacco smoking.

5.3. Limitations

Generalizability of this study is limited by convenience sampling. We have a small sample size for daily hookah smokers (n = 20). Additional research is needed with larger sample sizes by smoking frequency status to enable a more rigorous assessment of nicotine exposure from hookah tobacco smoking.

5.4. Conclusions

Hookah tobacco smoke is a source of nicotine exposure. Those attending social smoking events in hookah lounges and private homes are at risk of nicotine intake from exposure to hookah tobacco SHS, and smokers absorb even more nicotine through direct inhalation. GM urinary cotinine levels in hookah smokers and non-smokers increased significantly 8.5 times and 2.5 times, respectively, following a hookah social event. Among hookah smokers, the greatest change in urinary cotinine levels was found in occasional hookah smokers, in which GM levels increased 31.2 times. Our results call for protecting hookah smokers' and non-smokers' health by requiring accurate hookah tobacco labels for nicotine content, raising taxes on hookah tobacco, reducing the spread of hookah lounges, and encouraging voluntary bans on smoking hookah tobacco in private homes.

Supplementary Material

Supplementary material(s) is available here [To read supplementary materials, please refer to the journal website and open PDF/HTML].

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Footnotes

Authors' Contribution: Conception, design and development of methodology: Nada O F Kassem, Noura O Kassem, Melbourne F Hovell; acquisition of data, study supervision, and administrative, technical, material or Laboratory support: Nada O F Kassem, Noura O Kassem, Alexander Ivan B Posis, Sheila R Jackson, Dale A Chatfield; analysis and interpretation of data: Nada O F Kassem, Noura O Kassem, Sheila R Jackson, Sandy Liles, Melbourne F Hovell. All authors were involved in writing, review, and/or revision of the manuscript.

Declaration of Interests: The authors declare no competing interests associated with this study.

Implication for Health Policy Makers/Practice/Research/Medical Education: Our results call for protecting hookah smokers' and non-smokers' health by requiring accurate hookah tobacco labels, raising taxes on hookah tobacco, reducing the spread of hookah lounges, and encouraging voluntary bans on smoking hookah tobacco in private homes.

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Table 3. Creatinine-Corrected Urinary Levels of Cotinine[a] in Adults pre and post an Indoor Hookah-Only Social Event, by Smoking Status and Location of Event (N = 208)

Variables	Hookah Lounge, Hookah-Only Social Event (N = 108), ng/mg Creatinine		Ratio[b]	P[c]	Home, Hookah-Only Social Event (N = 100), ng/mg Creatinine		Ratio[b]	P[d]	P[e]
	Pre Event	Post Event			Pre Event	Post Event			
Hookah Smokers (n = 105)				< 0.001[f]				< 0.001[f]	0.576
Mean ± SD[g]	178.9 ± 386.7				238.3 ± 667.2	332.6 ± 399.2			
GM (95% CI)[h]	14.5 (6.4 - 32.7)	124.7 (79.8 - 195.0)	8.6		17.8 (7.6 - 41.9)	150.2 (97.9 - 230.6)	8.4		
Median (5 - 95 percentile)	48.3 (0.2 - 928.1)	133.6 (7.7 - 1187.1)	2.8		48.6 (0.14 - 876.8)	170.0 (15.8 - 1312.6)	3.5		
Minimum - Maximum	0.02 - 2342.7	(0.09 - 2410.5)			0.03 - 4558.3	1.2 - 1576.0			
% above LOD[i] (Freq/n)[j, k]	98 (52/53)	100 (54/54)			98 (49/50)	100 (50/50)			
Non-Smokers (n = 103)				0.001[f]				0.001[f]	0.013[f]
Mean ± SD	1.1 ± 1.9	3.2 ± 5.9			1.0 ± 1.7	1.6 ± 2.7			
GM (95% CI)	0.4 (0.3 - 0.6)	1.3 (0.9 - 1.8)	3.3		0.4 (0.2 - 0.5)	0.7 (0.5 - 1.0)	1.8		
Median (5 - 95 percentile)	0.4 (0.02 - 6.0)	(0.13 - 22.8)	2.8		0.3 (0.02 - 5.4)	0.7 (0.1 - 8.7)	2.3		
Minimum - Maximum	0.02 - 9.2	0.12 - 25.5			0.01 - 8.9	0.04 - 13.1			
% above LOD (Freq/n)	85 (44/52)	100 (52/52)			82 (41/50)	96 (48/50)			

[a] Cotinine is a metabolite of nicotine. Cotinine values are corrected with creatinine (ng/mg creatinine).

[b] Ratio, Ratio of post to pre hookah event cotinine GMs and medians.

[c] P Hookah lounge: pre vs. post event. P values were derived from Wilcoxon signed-rank tests; two-tailed alpha level P < 0.05.

[d] P Home: pre v post event. P values were derived from Wilcoxon signed-rank tests; two-tailed alpha level P < 0.05.

[e] P Change in cotinine, hookah lounge vs. home. P values were derived from Mann-Whitney U tests; two-tailed alpha level P < 0.05.

[f] Significant levels.

[g] Mean ± SD, Arithmetic mean and standard deviation.

[h] GM (95% CI), Geometric mean and 95% confidence interval.

[i] % Above LOD, Percentage of urine samples above the limit of detection (LOD); cotinine LOD, 0.1 ng/mL.

[j] Freq/n, Frequency of samples with levels above the LOD / n-size of samples per group.

[k] Missing values: interference (n = 4)/missing samples (n = 1).