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Sources and Biomarkers of Secondhand Tobacco Smoke Exposure in Urban Adolescents

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Abstract

Objective: In an urban adolescent population, we evaluated sources of exposure to secondhand smoke exposure (SHS), examined differences in exposure by race/ethnicity, age and sex, and determined the relationship between exposure source (s) and the biomarkers cotinine and NNAL.

Methods: Participants were recruited from a public hospital-based outpatient clinic in San Francisco, CA, USA.

Results: Of a sample of N = 298 adolescents screened, 235 were biologically confirmed to be exposed to tobacco smoke. Of those, N = 16 were active smokers and N = 219 were exposed to SHS; 91 (39%) were heavily SHS exposed (median cotinine = 0.76 ng/mL) and 128 (54%) had light SHS exposure (median cotinine = 0.11 ng/mL). Within those SHS exposed, the most common source of exposure was in a public area. No significant racial/ethnic differences were found, although African American adolescents were more likely to live in a home that allowed smoking. Older adolescents were more likely to be exposed across several difference sources, and females more likely to be exposed in a car and in public areas. Past 7-day exposure in the home, in a car, and current blunt use were significantly related to biomarkers of exposure.

Conclusions: Urban adolescents are exposed to SHS across a variety of sources. Although exposure in a public area is most common, exposure in the home and in cars significantly influences tobacco biomarker levels. Interventions to reduce exposure would have the greatest impact in this population if they focused on reducing exposure in the home and in cars. History of blunt use is a strong determinant of tobacco exposure.

Keywords

adolescents; cotinine; secondhand smoke; sensitive populations

Secondhand smoke exposure (SHS) has been associated with respiratory infections, ear infections, and asthma in children.[1–4] Adolescents are of particular concern as they report higher rates of SHS exposure, and exposure across a greater number of environments than children (less than 11 years old) and adults.[5,6] Adolescents of low socioeconomic status (SES) are even more likely to be exposed[7] as smoking prevalence is higher and smoking in the home is more likely to occur in low SES groups.[8]

Previous research has documented high rates of SHS exposure in low SES adolescents. In a prior study, we collected urine samples from adolescents seen for primary and urgent care at San Francisco's county hospital servicing an economically disadvantaged population. Utilizing the biomarker cotinine (a major metabolite of nicotine), levels consistent with SHS exposure were detected in 76% of the sample.[9] An analysis utilizing the biomarker 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanol (NNAL), a tobacco-specific nitrosamine and metabolite of 4-(methylnitrosamino)-1-(3) pyridyl-1-butanone (NNK), both of which are potent pulmonary carcinogens,[10] found an even higher prevalence.[11] This is much higher than adolescent SHS exposure reported by the World Health organization; 47% internationally and 39% in the Americas.[12] Due to the high prevalence of SHS exposure, the health implications of SHS may disproportionally burden low SES adolescents, supporting the need for SHS screening by care providers and interventions to reduce exposure.

Knowing where adolescents are exposed to SHS is one step toward understanding how to intervene to reduce exposure. Sources of adolescent SHS exposure likely encompass various environments including inside and outside of the home. Previous work has categorized outside of the home exposure as that occurring in private vehicles, public places, and workplaces.[6] Exposure sources may vary by race/ethnic background, as African Americans (AA) are more likely to live in a home without a smoking ban.[8] Additionally, exposure sources may vary by age, with older adolescents encountering more or different environments than younger adolescents. Sex differences may occur as male adolescents are significantly more likely to smoke than females[13] so perhaps they encounter more exposure in their peer groups.

In adolescents, the relationship between the number of and context of SHS exposure sources and biomarker levels is unclear, including how this may vary by race, age, and sex. There is some evidence from child and adult samples. In a study of 4-year-old children, the likelihood of having detectable cotinine was higher for those exposed at home versus not.[14] Children exposed in public places had cotinine above the limit of detection only if the exposures occurred at least 3 times per week.[14] Children living in multiunit housing were more likely to have cotinine levels indicating exposure than those living in attached or detached houses.[15] In a low SES child population, higher cotinine levels were observed in younger children, and in those living in homes with a higher number of smokers.[16] A study of adults found cotinine levels were highest among those who endorsed being exposed in 3 environments (home, work/school, and other public places), and lowest for those exposed

only at work.[17] In one study, adolescents were more likely than younger children to report exposures both inside and outside of the home; however, a higher percentage of children had detectable cotinine levels.[6] Understanding the relationship between source and intensity of exposure and biomarkers is important to informing the design of future SHS-reduction interventions, especially in groups disproportionally affected by SHS.

In the current study, we examined the following aims in a convenience sample of urban, low SES adolescents, and young adults being seen in a hospital-based medical clinic: 1) to identify where adolescents are exposed to SHS and the intensity of the exposure(s); 2) to compare sources and intensity of exposure to SHS by race, age, and sex; and 3) to evaluate which specific sources of exposure are significantly related to the tobacco biomarkers cotinine and NNAL.

Methods

Participants

Participants were enrolled from 2015 to 2017 at the Children's Health Center (CHC) at Zuckerberg San Francisco General Hospital (ZSFG). During this period of enrollment, smoking indoors was prohibited in San Francisco including in restaurants, bars, and workplaces. The CHC serves approximately 11,000 children and adolescents annually, seeing patients up to 21 years of age, with a population of 58.1% Hispanic, 19.1% AA, 11% Asian, 6.5% white, and 5.2% other. The vast majority (96.4%) of the patients have publicly funded health insurance. Participants presented for both preventive and sick care. The research was approved by the UCSF Institutional Review Board.

Measures

Participants completed a basic demographic questionnaire, self-reporting their age, race, and sex. A questionnaire to assess exposure to SHS and product use was created for this study. It listed several environments and asked participants to indicate if they had been exposed to SHS in the past 7 days and past 24 hours in each of the environments. The environments were as follows: in the home, a friend or relative's home, in a car, in a public area, in a club, bar or lounge, and somewhere else. If an environment was endorsed, participants were asked to rate the intensity of the exposure on a 1 to 3 scale; 1 = not irritating, 2 = mildly irritation, and 3 = very irritating. Participants were asked questions about smoking rules in their home; if smoking was allowed some of the time, all of the time, or never, and how many people they lived with were smokers. Product use was assessed by asking participants if they had ever (defined as "at *least one puff or one time*") or currently (defined as *"at least once in the past 30 days"*) smoked tobacco cigarettes, blunts (marijuana rolled in a cigarillo or cigar wrapper),[18] and a variety of other nicotine and tobacco products.

Procedures

Recruitment began May 19, 2015 and ended January 24, 2017. Adolescents and their parents were approached in the clinic waiting room by the research coordinator, given a brief synopsis of the study, and asked if they were interested in participating. If interest was shown, the research coordinator waited until the participant was assigned an exam room to

conduct informed consent confidentially. Parents of those under the age of 18 signed the consent form and HIPAA authorization along with the adolescent participants.

Before administering questionnaires, parents were asked to leave the exam room and the research coordinator reviewed a graphic of SHS with each participant. The graphic depicted sidestream smoke coming off a burning cigarette and mainstream smoke being exhaled out of a smoker's mouth. The research coordinator reviewed examples of the 3 intensity categories with participants; 1 = you notice the smoke but it doesn't bother you; 2 = the smoke makes your throat scratchy or makes you cough; 3 = your throat burns or you cannot stop coughing. This was to ensure that all participants had the same reference point for SHS and intensity categories. All questionnaires were completed on an electronic tablet through the secure electronic data capture system REDCap. Participants were given privacy to complete the questionnaires with the research coordinator on hand if there were questions. After completing questionnaires, participants were asked to provide a urine sample. Urine was collected only after the care providers received samples they required for visit-related testing. After the visit, the research coordinator accessed the participants' electronic medical records to record the reason for their visit to the CHC. All procedures and materials were approved by the University of California San Francisco's Institutional Review Board.

Analytical Chemistry

Urine samples were analyzed for free cotinine and total (free plus conjugated) NNAL by liquid chromatography-tandem mass spectrometry.[19,20] Cotinine, the main proximate metabolite of nicotine, has a half-life of about 16 hours[21] and is a biomarker of ongoing or recent exposure (past 5–6 days), while NNAL has a long half-life (about 10–16 days)[22] and can assess exposure over the past several weeks. The limit of quantitation for cotinine was 0.05 ng/mL (nanograms per milliliter) and for NNAL 0.25 pg/mL (picograms per milliliter).

Statistical Analysis

Frequency data are presented as counts and proportions and are compared across groups using either a chi-square or Fisher's exact test, as appropriate. Continuous variables are reported as median (IQR; interquartile range) or mean ± SD (standard deviation). Based on previous analyses of a similar population,[9] urine cotinine values >30 ng/mL were taken as a cut-point as active smoking and cotinine values <0.05 ng/mL were defined as not exposed. Secondhand smoke exposure analysis was carried out in the population defined as "nonsmoking" and "exposed." Within that group, participants could be heavily SHS exposed (cotinine ranging from 0.25 to 30 ng/mL) or lightly SHS exposed (cotinine ranging from 0.05 to 0.25 ng/mL) as per previously established cut-points.[9]

Either ANOVA or a nonparametric equivalent Kruskal-Wallis, as appropriate, was used to test whether log-transformed cotinine values in exposed, nonsmokers differed across covariates. An intensity "sum score" was created from participants' intensity ratings at each location of past 7-day exposure; "not exposed" were given a score 0, "not irritating exposure" were given a score 1, "mildly irritating exposure" were given a score of 2, and "very irritating exposure" were given a score 3. This sum intensity score incorporates both the number of locations exposed and the intensity of each exposure, and can range from 0

(no exposure at any location) to 18 (very irritating exposure from all 6 locations: *at home, at a friend or relative's home, in a car, at a nightlife venue* (club, bar or lounge), *in or around school/work/ or public,* or *somewhere else).*

A log-linear model of biomarkers (cotinine and NNAL values) in exposed, nonsmokers was developed to assess the relational effects of each source of exposure in overall smoke exposure. Analyses were performed both without and with creatinine normalization and results were similar. Results reported are without creatinine normalization. All statistical analyses were carried out using SAS v. 9.4 (SAS Institute, Inc, Cary, NC). Statistical tests were considered significant at $P < .05$.

Results

Demographics and Product Use

Three hundred participants were recruited from the CHC, with a final sample of N = 298 as cotinine levels were not available for 2 participants (1 sample interference and 1 missing). Participants were: ages 12 to 21 (mean and median age = 16); 143 (48%) male and 155 (52%) female; 195 (66%) Hispanic; 41 (14%) AA; 31 (10%) Asian; and 31 (10%) mixed or other, including white and Native American. The majority of participants (N = 179; 60%) were being seen at the CHC on the day of recruitment for routine well care and a small portion (N = 26; 8%) were seen for an upper respiratory event or infection. The 2 most common products of ever use were tobacco cigarettes (24% ever) and blunts (27% ever). All other products including electronic cigarettes, snuff, snus, cigars, cigarillos, dissolvable tobacco, and/or hookah were used by <5% of the sample.

Secondhand Smoke Exposure

We utilized cotinine as the primary measure to determine the proportion in our sample exposed to SHS following established cut-points previously described.[9] Tobacco smoke exposure was found in 235 (79%) of the sample. Within those exposed, 16 (7%) were actively smoking (median cotinine = 143 ng/mL); 91 (39%) were heavily SHS exposed (median cotinine = 0.76 ng/mL); and 128 (54%) had light SHS exposure (median cotinine = 0.11 ng/mL). Demographics and biomarkers of exposure are shown in Table 1. All further results are reported on our nonsmoking, SHS exposed sample (N = 219).

Sources and Intensity of Exposure

Among those biochemically confirmed to be SHS exposed (N = 219), N = 209 (95%) reported exposure from at least one source in the past 7 days, and N = 129 (59%) in the past 24 hours. The large proportion of participants endorsed exposure from either 1 (45%) or 2 (32%) sources in the past 7 days and 1 source in the past 24 hours (37%). The most common sources of exposure in the past 7 days were *in a public area* (N = 166; 76%) followed by *in the home* (N = 70, 32%); *in a nightlife venue (club, bar, or lounge)* (N = 33, 15%); *in a car* (N = 23, 11%) and *at a friend or relative's home* (N = 20, 9%). Participants described public areas as in or around bus stops, parks, school, work, or on neighborhood streets. A small percentage of participants (17%) reported being exposed *somewhere else* in the past 7 days. The descriptions of *somewhere else* were reviewed and added to applicable categories. The

majority of participants (76%) had intensity sum scores within the 1 to 5 range, indicating a mild level of intensity. Regarding home smoking rules, N = 169 (77%) lived in a home where smoking was never allowed, N = 34 (16%) where smoking was allowed in some places at some times and N = 13 (6%), where smoking was allowed anywhere and at any time.

Racial Differences

No significant differences between race/ethnicity in exposure sources were found; however, nearing significance was that mixed/other, compared to other groups, were more likely to be exposed *in the home* in the past 7 days and 24 hours *P* = .05). AA, compared to other groups, were more likely to live in a home where smoking was allowed some places at some times (*P* = .05). One significant difference in product use emerged such that mixed/other were more likely to have ever smoked than Asians and AA (*P* < .05). Descriptive of source of exposure, intensity, home smoking rules, and product use are shown in Table 2 for the whole sample and by racial/ethnic groups.

Age and Sex Differences

Older adolescents were significantly more likely to be exposed *in the home* (*P* < .05) in the past 7 days. In the past 24 hours of exposure, older adolescents were significantly more likely to be exposed *in public areas* (*P* < .01); *in the home* (*P* < .05); and *in a car* (*P* < .05). In regard to product use, older adolescents were significantly more likely to have ever smoked a cigarette (*P* < .01); currently smoked cigarettes (*P* < .05); ever smoked a blunt (*P* < .01); and currently smoked blunts (*P* < .05).

Females, compared to males, were significantly more likely to be exposed *in a car* (*P* < .05) and *in public areas* (*P* < .01) in the past 7 days. There were no statistically significant differences between males and females in past 24 hours of exposure. Males were significantly more likely to report zero intensity of exposure than females (*P* < .01). In regards to product use, males were significantly more likely to have ever smoked a cigarette (*P* < .05), and females were significantly more likely to have ever smoked a blunt (*P* < .01).

Source of exposure, intensity ratings, home smoking rules, and product use by sex and age are shown in Table 3. All tobacco product use for this group is shown in Supplementary Table 1.

Sensitivity Analysis

We repeated all analyses including only those ages 18 to 21 (N = 52), as 18- to 21-year olds had legal access to purchasing tobacco products and potentially difference exposure sources. Results were similar to that of the entire sample except the following were no longer significant: females were more likely to be exposed *in public areas* and *in a car* in the past 7 days; males were more likely to have ever smoked a cigarette; and females were more likely to have ever smoked a blunt. Patterns of sources of exposure remained the same with the highest endorsements for past 7 days *in public areas* (86%) and *in the home* (40%).

Exposure Sources in Relation to Biomarkers

To determine which independent variables were to be included in our model, we first examined differences in median cotinine and NNAL levels by exposure sources. Cotinine and NNAL levels were significantly higher in those exposed *in the home* in the past 7 days, and in those living in homes where smoking was allowed versus allowed in some places/ times and never. Cotinine and NNAL were significantly higher in those exposed *at a friend or relative's home* in the past 7 days, in those with greater intensity scores, and in current blunt users. Cotinine was significantly higher in those exposed *in a car* in the past 7 days. Comparisons of biomarkers by exposure source are shown in Table 4.

We chose to include the source of exposure (example: car) combined with the intensity rating for that specific exposure as the independent variable. We included past 7 day exposure *in the home* rather than *smoking allowed in the home,* in order to have a dichotomous variable. We chose to not include exposure *at friend or relative's home* in the model, as this had the lowest number of endorsements. We ran models including race; however, it was not significantly associated with the outcomes. Our final model included the independent class variables of age, sex, intensity of past 7-day home exposure, intensity of past 7-day car exposure, and current blunt use. Blunt use had the strongest association with cotinine and NNAL levels, followed by home exposure intensity (Figure and Supplementary Table 2).

Discussion

This work sought to establish sources of and intensity of SHS exposure in a population of urban, low SES adolescents, and to examine these variables by age, sex, and race/ethnicity. The most common source of exposure was *in a public area,* followed by *in the home* for past 7-day and past 24-hour exposures. Most adolescents rated intensity of SHS exposure as mildly irritating. AA were more likely to live in a home that allowed some smoking. Older adolescents were more likely to be exposed *in the home, in public areas*, and *in a car.* Females were more likely than males to be exposed *in public areas* and *in a car*. Males were more likely to rate intensity as zero, meaning they did not endorse exposure from any source.

Product use is important to assess as adolescents with tobacco exposure biomarker levels consistent with SHS may overlap with biomarker levels of adolescents occasionally using tobacco products. Via self-report, current tobacco cigarette smoking and blunt use was low (3% and 11%, respectively); however, blunts were the most popular product of ever use (28%). Males were more likely to have ever smoked cigarettes, and females more likely to have ever used blunts. Older adolescents were more likely to have currently and/or ever used all products. The rates of electronic cigarette use in this sample were low (<5%), demonstrating that usage in low SES adolescents may differ from the larger adolescent population. However, this was before JUUL use became widespread among teens and young adults.

We sought to evaluate which sources of exposure were significantly related to the biomarkers of exposure cotinine and NNAL. While exposure *in a public area* was most

common, it was not significantly related to exposure biomarkers. This is similar to what has been found previously in adult samples[15] in that while exposure outside of the home was most common, cotinine levels were still highest for those exposed in the home. Exposure *in the home* in the past 7 days was significantly associated with both cotinine and NNAL, while exposure *in a car* was significantly associated with cotinine. Current blunt use had the strongest association with both biomarkers. This could be because participants had recently used this tobacco product or been around others who were using it. However, much of the variance in our biomarkers was not explained by our independent variables, indicating other variables that we did not measure may influence exposure.

A novel aspect of this work was identifying which sources of exposure impacted biomarkers within a low SES adolescent population. Prior work has focused on adult and children samples and not surveyed a variety of exposure environments in low SES adolescents. Additionally, the finding of impact of blunt use on biomarkers of exposure within an adolescent population is novel. As blunts are made by filling a cigarillo or cigar wrapper with marijuana, the tobacco content in the wrapper exposes the user to low levels of nicotine and nitrosamines.[23] While the prevalence of blunt use in adolescent populations has been estimated,[24] the impact of blunt use on cotinine and NNAL has not been reported. This is important when estimating SHS exposure, as active blunt use may lead to similar biomarker levels of SHS exposure. A challenge is estimating the impact of SHS exposure versus blunt use on biomarker levels.

Limitations

Our sample had an uneven distribution of race/ethnicity with a high proportion of Hispanic adolescents, reducing power for making comparisons between the groups. Additionally, we enrolled participants above the age of 18 (N = 52, 24%), who may better reflect a young adult population. However, as these participants were being seen along with teens at the CHC, we considered them similar enough to the minors in the sample to be combined. Others have also categorized adolescent samples to include those above the age of 18[6] and our sensitivity analysis indicated that their exposure sources were similar to the sample of minors. As our exposure questionnaire was self-reported, there is potential recall bias; participants may only accurately remember their most recent exposures. Additionally, we primarily asked participants about the location of exposure, rather than persons around them who may be the cause of exposure. We also did not ask how long they were exposed in each environment.

As we asked participants to report the illicit use of tobacco products, there is potential social desirability with participants under reporting or not reporting product use. Finally, we cannot definitively tease out how much our biomarker levels are influenced by product use versus SHS exposure.

Conclusions

In informing potential adolescent screening and intervention for SHS exposure in this low SES population, we found that use of the biomarkers cotinine and NNAL showed, in general, similar results. As mentioned in a previous publication, this indicates that a high

sensitivity cotinine assay would be adequate as a screening measure for tobacco exposure.[9] Our present analysis suggests that while exposure in public areas is most common, it is exposure within the home and within cars that is associated with greater exposure to tobacco smoke. Furthermore, the use of blunts is a strong determinant of biomarker classification as being SHS exposed. In designing an intervention to reduce tobacco smoke exposure in adolescents, a focus on reducing these sources would have the greatest impacts on exposure, and presumably on SHS-related disease risks.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

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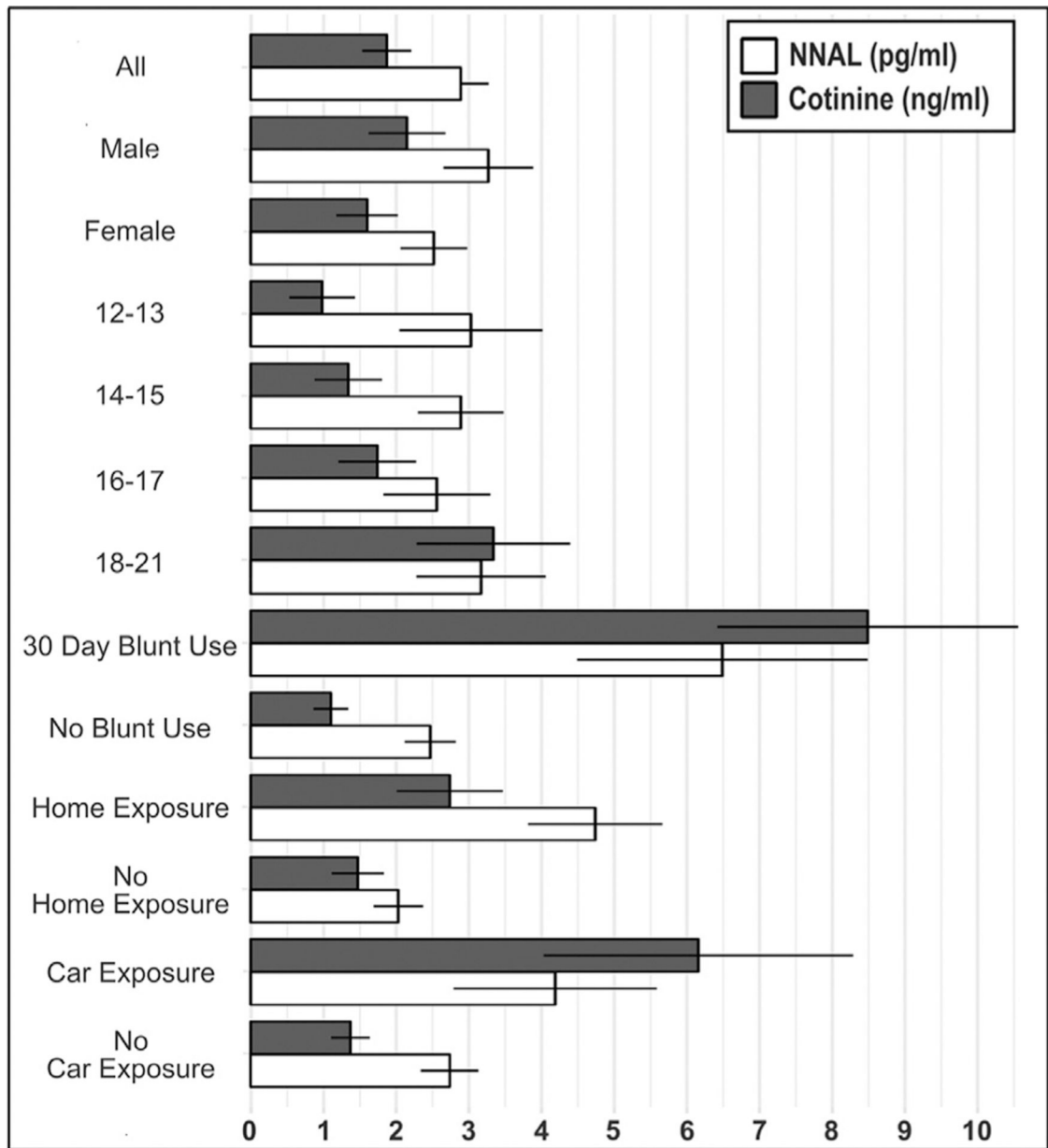
What's New

Exposure to secondhand smoke in the home and in cars, and a history of blunt use significantly predicts biomarkers of exposure within urban adolescents.

Figure. Cotinine and NNAL by sex, age, blunt use, and exposure source
pg/mL = picograms per milliliter; ng/mL = nanograms per milliliter

Table 1.

Demographics, Biomarkers of Exposure, and Product Use

	All Participants N = 298	Smokers (Cot >30) n = 16	Heavy SHS (Cot 0.25–30) n = 91	Light SHS (Cot 0.05–0.25) n = 128	Nonexposed (Cot <0.05) n = 63	P Value
Urine cotinine (ng/mL) median (IQR)	0.15(0.06–0.50)	143 (66–260)	0.76 (0.42–4.18)	0.11 (0.08–0.16)	BLQ	<.001
Urine NNAL (pg/mL) median (IQR)	0.80 (0.18–2.23)	54 (9–85)	2.21 (1.06–5.70)	0.49 (0.18–1.26)	0.28 (0.18–0.73)	<.001
Age (mean ± SD)	15.8 ± 2.4	17.3 ± 2.0	16.1 ± 2.5	15.6 ± 2.4	15.5 ± 2.4	.03
Sex n (% female)	155 (52)	7 (43.8)	44 (48.4)	66 (51.6)	38 (60.3)	.40
Race/ethnicity n (%)						<.01
Asian	31 (10.4)	1 (6.3)	10 (11.0)	12 (9.4)	8 (12.7)	
Black	41 (13.8)	3(18.8)	31 (34.1)	6 (4.7)	1 (1.6)	
Hispanic	195 (65.4)	8 (50.0)	32 (35.2)	103 (80.5)	52 (82.5)	
Mixed/other	31 (10.4)	4 (25.0)	18 (19.8)	7 (5.5)	2 (3.2)	
Ever smoked tobacco cigarette n (%)	74 (24.8)	13(81.3)	22 (24.2)	31(24.7)	8(12.7)	<.001
Ever smoked blunt n (%)	81 (27.4)	13(81.3)	35 (39.3)	27 (21.1)	6 (9.5)	<.001

Cot indicates cotinine; IQR, interquartile range; BLQ, below the lower limit of quantitation; pg/mL, picograms per milliliter; SD, standard deviation.

Table 2.

Exposure Sources, Intensity and Product Use by Race/Ethnicity

	All SHS Exposed n = 219	Hispanic n = 135	African American n = 37	Asian n = 22	Mixed or Other n = 25	P Value
Sources of exposure						
In a public area n (%)						
Past 7 days	166 (75.8)	104 (77.0)	27 (73.0)	17 (77.0)	18 (72.0)	.92
Past 24 hours	81 (36.9)	56 (41.5)	9 (24.3)	9 (41.0)	9 (36.0)	.26
At home n (%)						
Past 7 days	70 (31.9)	34 (25.2)	15 (40.5)	9 (41.0)	12 (48.0)	.05
Past 24 hours	37 (16.9)	18 (13.3)	7 (18.9)	3 (13.6)	9 (36.0)	.05
Nightlife venue (club, bar, or lounge) n (%)						
Past 7 days	33 (15.1)	24 (17.8)	3 (8.2)	1 (4.5)	5 (20.0)	-*
Past 24 hours	9 (4.1)	8 (5.9)	0	0	1 (4.0)	-
At a friend/relatives home n (%)						
Past 7 days	20 (9.1)	9 (6.6)	3 (8.2)	3 (13.6)	5 (20.0)	-
Past 24 hours	7 (3.2)	6 (4.4)	0	0	1 (4.0)	-
In a car n (%)						
Past 7 days	23 (10.5)	12 (8.9)	8 (21.6)	1 (4.5)	2 (8.0)	-
Past 24 hours	9 (4.1)	7 (5.2)	1 (2.7)	1 (4.5)	0	-
Intensity of exposure						.46
0	24 (10.9)	15 (11.1)	4 (10.8)	1 (4.5)	4 (16.0)	
1–5	167 (76.2)	106 (78.5)	25 (67.6)	19 (86.4)	17 (68.0)	
>5	28 (12.8)	14 (10.3)	8 (21.6)	2 (9.1)	4 (16.0)	
Home smoking rules						.05
Never allowed	169 (77.2)	114 (84.4)	21 (56.8)	17 (77.3)	17 (68.0)	
Allowed some of the time	34 (15.5)	14 (10.4)	13 (35.1)	2 (9.1)	5 (20.0)	
Allowed all of the time	13 (6.0)	5 (3.7)	3 (8.1)	2 (9.1)	3 (12.0)	
Product use						
Ever smoked cigarette n (%)	53 (24.2)	39 (28.9)	4 (10.8)	2 (9.1)	8 (32.0)	.04
Smoked cigarette, past 30 days	7 (3.2)	4 (2.9)	1 (2.7)	0	2 (8.0)	.46
Ever smoked blunt n (%)	62 (28.3)	32 (23.7)	17 (46.0)	5 (22.7)	8 (32.0)	.16

	All SHS Exposed n = 219	Hispanic n = 135	African American n = 37	Asian n = 22	Mixed or Other n = 25	*P* Value
Smoked blunt, past 30 days	23 (10.5)	12 (8.9)	6 (16.2)	2 (9.1)	3 (12.0)	.62

*
P value unable to be estimated.

Table 3.

Exposure Sources, Intensity and Product Use by Sex and Age

	All SHS Exposed n = 219	Age <15 (Median = 16) n = 98	Age 16 (Median = 16) n = 121	P Value	Males n = 109	Females n = 110	P Value
Sources of exposure							
In a public area n (%)							
Past 7 days	166 (75.8)	69 (70.4)	97 (80.1)	.09	73 (67.0)	93 (84.5)	.002
Past 24 hours	81 (36.9)	26 (26.5)	55 (45.5)	.004	41 (37.6)	40 (36.4)	.48
At home n (%)							
Past 7 days	70 (31.9)	23 (23.5)	47 (38.8)	.02	29 (26.6)	41 (37.3)	.06
Past 24 hours	37 (16.9)	11 (11.2)	26 (21.5)	.04	15 (13.8)	22 (20.0)	.15
Nightlife venue (club, bar, or lounge) n (%)							
Past 7 days	33 (15.1)	15 (15.3)	18 (14.9)	.93	21 (19.3)	12 (10.9)	.06
Past 24 hours	9 (4.1)	5 (5.1)	4 (3.3)	.51	6 (5.5)	3 (2.7)	.25
At a friend/relatives home n (%)							
Past 7 days	20 (9.1)	7 (7.1)	13 (10.7)	.36	9 (8.3)	11 (10.0)	.42
Past 24 hours	7 (3.2)	2 (2.0)	5 (4.1)	.38	4 (3.7)	3 (2.7)	.50
In a car n (%)							
Past 7 days	23 (10.5)	8 (8.2)	15 (12.4)	.31	7 (6.4)	16 (14.5)	.04
Past 24 hours	9 (4.1)	1 (1.0)	8 (6.6)	.04	3 (2.8)	6 (5.5)	.25
Intensity of exposure							.009
0	24 (10.9)	15 (15.3)	9 (7.4)	.13	19 (17.4)	5 (4.5)	
1–5	167 (76.2)	73 (74.4)	94 (77.7)		78 (71.6)	89 (80.9)	
>5	28 (12.8)	10 (10.2)	18 (14.9)		12 (11.0)	16 (14.5)	
Home smoking rules							.48
Never allowed	169 (77.2)	81 (82.7)	88 (72.8)	.32	88 (80.7)	81 (73.6)	
Allowed some places at some times	34 (15.5)	13 (13.3)	21 (17.4)		19 (17.4)	15 (13.6)	
Allowed anywhere all times	13 (6.0)	4 (4.1)	9 (7.4)		8 (7.3)	5 (4.5)	
Product use							
Ever smoked cigarette n (%)	53 (24.2)	10 (10.2)	43 (35.5)	.001	33 (30.2)	20 (18.2)	.04
Smoked cigarette, past 30 days	7 (3.2)	0	7 (5.8)	.01	3 (2.8)	4 (3.6)	.52
Ever smoked blunt n (%)	62 (28.3)	16 (16.3)	46 (38.0)	.001	20 (18.3)	42 (38.2)	.001

	All SHS Exposed n = 219	Age <15 (Median = 16) n = 98	Age 16 (Median = 16) n = 121	P Value	Males n = 109	Females n = 110	P Value
Smoked Blunt, past 30 days	23 (10.5)	5 (5.1)	18 (14.9)	.02	8 (7.3)	15 (13.6)	.10

Table 4.

Cotinine and NNAL by Exposure Sources and Product Use

Question	Response	n	%	Cotinine ng/mL		NNAL pg/mL	
				Median (IQR)	P Value	Median (IQR)	P Value
Current blunt use (past 30 days)	No	196	89.5	0.17 (0.10–0.44)	<.001	0.86 (0.26–2.05)	<.01
	Yes	23	10.5	4.27 (0.36–12.66)		1.96 (1.12–6.91)	
Smoking *in the home*	Never	169	77.2	0.17 (0.09–0.36)	<.001	0.76 (0.25–1.75)	<.001
	Some Places/times	34	15.5	0.48 (0.13–3.76)		2.28 (1.23–9.19)	
	All places/time	13	5.9	2.20 (0.26–4.27)		4.20 (1.00–8.63)	
Exposed *at home* (past 7 days)	No	149	68.0	0.17 (0.10–0.43)	.01	0.77 (0.18–1.69)	<.001
	Yes	70	32.0	0.26 (0.13–0.96)		1.62 (0.56–3.33)	
Exposed *in a car* (past 7 days)	No	196	89.5	0.17 (0.1–0.45)	<.001	0.97 (0.30–2.21)	.16
	Yes	23	10.5	0.64 (0.15–8.12)		1.48 (0.43–3.80)	
Exposed *in public place* (past 7 days)	No	53	24.2	0.19 (0.12–0.76)	.60	1.22 (0.46–3.64)	.19
	Yes	166	75.8	0.17 (0.10–0.51)		0.99 (0.26–2.20)	
Exposed *at friend or relatives home* (past 7 days)	No	199	90.9	0.17 (0.10–0.49)	<.001	0.86 (0.26–2.04)	<.001
	Yes	20	9.1	0.93 (0.27–7.09)		2.69 (1.26–7.26)	
Exposed *at nightlife venue (club, bar, or lounge)* (Past 7 days)	No	186	84.9	0.19 (0.11–0.58)	.50	1.06 (0.32–2.43)	.60
	Yes	33	15.1	0.18 (0.10–0.60)		1.10 (0.18–1.81)	
Exposed *in a public area* (past 7 days)	No	53	24.2	0.19 (0.12–0.76)	.60	1.22 (0.46–3.64)	.19
	Yes	166	75.8	0.17 (0.10–0.51)		0.99 (0.26–2.20)	
Intensity score	0	24	11.0	0.19 (0.11–0.76)	<.01	1.23 (0.47–3.48)	<.01
	1–5	167	76.3	0.17 (0.10–0.49)		0.85 (0.25–2.04)	
	>5	28	12.8	0.46 (0.15–5.20)		1.84 (0.76–3.49)	
Number of exposure sources	0	24	11.0	0.19 (0.11–0.60)	.01	1.29 (0.54–3.27)	.002
	1	84	38.4	0.13 (0.09–0.35)		0.52 (0.18–1.58)	
	2 or more	111	50.6	0.24 (0.13–0.84)		1.30 (0.48–2.69)	

ng/mL indicates nanograms per milliliter; pg/mL, picograms per milliliter; IQR, interquartile range.