An examination of social and environmental determinants of secondhand smoke exposure among non-smoking adolescents

Elizabeth K. Do[1,2], Kennedy C. Bradley[3], Kendall Fugate-Laus[4], Kiranpreet Kaur[5], Matthew S. Halquist[6], Laure Ray[6], Michell A. Pope[7], Rashelle B. Hayes[8], David C. Wheeler[9], Bernard F. Fuemmeler[1,2]

ABSTRACT

INTRODUCTION Adolescents are at increased risk of secondhand smoke exposure (SHS) due to the limited control that they have over social and physical environments. Yet, knowledge regarding determinants of SHS among non-smoking adolescents is limited. This study identifies social and environmental factors associated with SHS among non-smoking adolescents.

METHODS To be included, parents and adolescents (aged 11–17 years) of the Adolescents, Place, and Behavior Study had to have completed surveys between March 2019 and May 2020. Adolescents had to have not reported smoking within the past 30 days and provided a saliva sample assayed for cotinine (≤3 ng/mL). A series of stepwise linear regression models were fit to the data to identify social and environmental determinants of SHS, using log-transformed salivary cotinine.

RESULTS Of the 105 adolescent and parent dyads included, 90.3% were African American, 26.9% of parents reported smoking, 33.3% resided in multi-unit housing, and 67.7% lived in homes where smoking was not permitted. Significant associations were found between parent tobacco use (β=2.56, SE=0.98, p=0.0082) and residing in multi-unit housing (β=1.72, SE=0.86, p=0.0460) with increased log-transformed cotinine levels among non-smoking adolescents. Adolescent age, gender, and race/ethnicity, parental education, peer tobacco use, the number of adults and children in the home, average number of days of self-reported SHS within public spaces outside of the home, and home smoking policies were not significantly associated with cotinine.

CONCLUSIONS Results emphasize the importance of reducing secondhand smoke exposure by reducing parental smoking and altering exposures within social and home environments. Parental tobacco use and residential setting should be considered when developing interventions to reduce secondhand smoke exposure among non-smoking adolescents.

AFFILIATION
1 Department of Health Behavior and Policy, Virginia Commonwealth University, Richmond, United States
2 Massey Cancer Center, Virginia Commonwealth University, Richmond, United States
3 Department of Family Medicine and Population Health, Virginia Commonwealth University, Richmond, United States
4 Department of Psychology, Virginia Commonwealth University, Richmond, United States
5 School of Medicine, Virginia Commonwealth University, Richmond, United States
6 School of Pharmacy, Virginia Commonwealth University, Richmond, United States
7 Research Unlimited, Richmond, United States
8 Department of Psychiatry, Virginia Commonwealth University, Richmond, United States
9 Department of Biostatistics, Virginia Commonwealth University, Richmond, United States

CORRESPONDENCE TO
Bernard F. Fuemmeler. Department of Health Behavior and Policy, Virginia Commonwealth University, Richmond, VA 23298, United States. E-mail: Bernard.Fuemmeler@vcuhealth.org
ORCID ID: https://orcid.org/0000-0002-3550-0107

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INTRODUCTION

Secondhand smoke (SHS) exposure is a widespread public health problem, caused by the inhalation of smoke exhaled by an individual using combustible tobacco products. There is no risk-free level of SHS, which has been linked to an increased risk of coronary

heart disease, stroke, and lung cancer and contributes to >41000 annual deaths in the US[1]. In response to this growing public health problem, smoking restrictions and bans within workplaces, restaurants, and other public places have increased[2]. The implementation of these smoking regulations have been found to be effective in reducing individual-level SHS exposure, as evidenced by the downwards trend in the percentage of non-smokers exposed to SHS[3]. However, individuals may still be exposed to SHS in unregulated areas such as homes and personal vehicles[4].

Adolescents are at heightened risk of SHS exposure due to the limited autonomy and control that they have over their home and social environments. More than half of adolescents report SHS exposure in public places[5]. Research examining SHS determinants has predominantly focused on socioeconomic factors. Exposure to SHS is generally higher among adolescents with lower socioeconomic status, defined as living below the federal poverty level, having lower annual household incomes, lower parental education, lower rates of parental employment, and living in single-parent families[6]. These socioeconomic factors have also been found to be associated with parental smoking, as the prevalence of smoking is generally higher among parents with a high school education or less[7]. Added to this, a disproportionate number of African Americans reside in lower income neighborhoods, where smoking rates are generally higher[8]. According to one study using data from NHANES (1999–2014), African American children are 1.85 times more likely (95% CI: 1.39–2.47) to be exposed to tobacco smoke than non-Hispanic White children[9].

The social and environmental contexts in which children and adolescents live may also influence SHS exposure. According to a recent systematic review, the absence of home smoking policies and the use of tobacco products by parents and peers have been associated with an increased risk of SHS exposure[10]. Type of residence has also been identified as a risk factor for SHS exposure, as children living in rented homes, including multi-unit housing, are 2.23 times more likely (95% CI: 1.85–2.69) to be exposed to SHS[9]. Recent work has been expanded to include potential sources of SHS in locations outside of the home (i.e. vehicles, schools, other public spaces); though, few studies have examined the validity of these self-reported measures using biomarker data. Extant research has heavily relied on the reporting of parental or self-reported SHS exposure, which could potentially under or overestimate actual SHS exposure[11]. Thus, the purpose of this study is to determine the level of agreement between adolescent-reported SHS exposure and the biomarker cotinine and to identify social and environmental determinants of salivary cotinine concentration among a sample of non-smoking adolescents.

METHODS

Data source and study procedures

Data were obtained from the Adolescents, Place, and Behavior (APB) Study, a prospective cohort study funded by the Virginia Foundation for Healthy Youth (www.vfhy.org). Eligibility criteria for the Adolescents, Place, and Behavior study included adolescents (aged 11–17 years at time of enrollment) and parents, residing within 50 miles of Richmond, Virginia. Participants were recruited with the help of Research Unlimited, LLC through outreach events and posted flyers at various community sites. Interested participants were provided with more information about the study. To be enrolled in the study, written parental/guardian consent, written or verbal child assent, and survey completion by parents and adolescents were required. Adolescent participants also had the opportunity to provide a saliva sample at the time of recruitment. Adolescent participants were compensated $10 for survey completion and $15 for saliva collection. Parent participants were compensated up to $10 for survey completion. All study procedures were approved by the Institutional Review Board at Virginia Commonwealth University.

Study participants

To be included in this cross-sectional analysis, parents and adolescents had to have provided survey data between March 2019 and May 2020 on sociodemographic characteristics (e.g. adolescent gender, adolescent race/ethnicity, parent education) and tobacco use variables (e.g. ever use, past 30-day use). Adolescents also had to have provided a saliva sample. Adolescents who had reported using

tobacco products in the past 30 days and those who had cotinine concentration levels indicative of active smoking (i.e. >3 ng/mL) were excluded from analysis. The resulting sample included 105 adolescent and parent dyads.

Compared to the full cohort of the APB Study, this cross-sectional subset had a greater proportion of adolescent participants that indicated that their race/ethnicity was African American (90.4% cross-sectional sample vs 58.7% APB cohort) and a lower proportion indicated that their sex was male (46.7% cross-sectional sample vs 53.4% APB cohort). A lower proportion of parents of adolescent participants in the current study reported having a Bachelor's degree or higher, relative to the full cohort (24.8% cross-sectional sample vs 26.3% APB cohort).

Measures

Salivary cotinine concentration

Adolescent SHS exposure was determined from cotinine concentration levels, assayed from saliva samples. Saliva samples were collected using the Salimetrics Salivabio Passive Drool Collection Aid, according to suggested protocols by the manufacturer[12]. Sample levels of cotinine were determined using liquid chromatography tandem mass spectrometry (LC/MS/MS), preceded by a validated extraction method. The extraction method consisted of thawing samples at room temperature, and a sample volume of 0.050 mL was used for a single liquid extraction for analysis. Deuterated internal standard, base, and 90:10 methyl-t-butyl ether: tetrahydrofuran was added to each sample. The organic layer was poured onto the reconstitution solution and evaporated to dryness under a nitrogen stream. The samples were then reconstituted with 1% formic acid in acetonitrile and a volume of 0.010 mL injected into the LC/MS/MS. The LC/MS/MS method employed electrospray ionization (ESI) positive multiple reaction monitoring (MRM) mode. Nicotine, cotinine, and their respective deuterated internal standards were monitored using the following MRM transitions: nicotine 163→130, nicotine-d4 167→134, cotinine 176→80, and cotinine-d3 179→101. Chromatographic separation was achieved using a Polaris Si-A column (50 mm × 3.0 mm; 5 µm, Agilent Technologies, Palo Alto, CA). Chromatographic separation used hydrophilic interaction liquid chromatography (HILIC). A gradient initially of 100% 1:1 acetonitrile: methanol with 0.05% formic acid slowly changing to 90% over 3 min and 10% 10 mM ammonium format with 0.05% formic acid is used. The linear range used for cotinine was 0.1–1000 ng/mL, respectively, with a 1/x2 weighted regression model. These processes were developed and implemented by the Bioanalytical Shared Resource Laboratory within the School of Pharmacy at Virginia Commonwealth University[13,14]. Resulting cotinine concentration was treated as a continuous variable, measured in mg/mL with greater cotinine concentrations indicative of greater SHS exposure. However, due to the skewedness of the distribution of cotinine values, they were log-transformed (using natural log) prior to analyses.

Social determinants

Potential social determinants included adolescent-reported race/ethnicity and parent-reported educational attainment. Adolescent-reported race/ethnicity was categorized into two groups, due to small sample size (African American vs White, Hispanic, Asian, and other race/ethnicity). Parent-reported educational attainment was determined by asking parents to report their highest educational attainment. Response categories were coded as: less than high school (e.g. no schooling completed, homeschooling, nursery school to 8th grade, 9th to 11th grade, and 12th grade without a diploma), high school graduate or equivalent (high school graduate, general education diploma), some college (some college, associate's degree), and Bachelor's degree or higher (Master's, professional, or doctoral degree). Parents were asked to report whether they have used any tobacco product in the past 30 days. This measure was derived from the Population Assessment of Tobacco and Health Survey[15]. Responses were coded as 'Yes' or 'No'. Adolescent-reported peer tobacco use was determined by asking adolescents to report on how many of their closest four friends use tobacco, as derived from the National Youth Tobacco Survey[16]. Responses were coded as 'Yes' (if at least one friend smokes) or 'No' (if no friends smoke).

Environmental determinants

Potential environmental determinants included sources of SHS exposure, parent-reported smoking

policies within the home, the number of adults and children residing within the same household, and whether the participant resided in multi-unit housing. To characterize the sources of SHS to which adolescents were exposed, adolescents were asked to report the number of days, within the past week, they breathed smoke from someone who was smoking a tobacco product at home, in a vehicle, in school buildings/grounds/parking lots, and in indoor and outdoor public spaces including: stores, restaurants, sports arenas, school grounds, parking lots, stadiums, and parks. These questions were derived from the National Youth Tobacco Survey[16]. Parent-reported home smoking policies were measured by asking parents to report the type of home smoking policy implemented within their household. Parents could select from the following response options: no one is allowed to smoke anywhere, smoking is permitted in some places at some times, and smoking is permitted anywhere. This question was derived from the Tobacco Use Supplement to the Current Population Survey[17]. A binary variable was created indicating that either: no smoking was permitted or that smoking was permitted at least in some places at some times. Parents were also asked to report on the type of housing in which they resided (single-family home, multi-unit housing), as well as how many adults (aged ≥18 years) and children (aged <18 years) resided within the same household.

Statistical analysis

The analysis began with descriptive statistics on all variables, reported as frequencies and percentages for categorical variables and means and standard deviations for continuous variables. Next, either Pearson (between continuous variables) or point biserial correlations (between continuous and categorical variables) were computed to examine the association of all variables to confirm hypothesized associated directions with log-transformed cotinine concentration and to determine any collinearity across predictors. To evaluate the agreement between self-reported SHS exposure and cotinine, an agreement analysis was conducted by computing Cohen's kappa[18]. To do this, the thresholds of ≥1 ng/mL for cotinine level and having reported ≥1 day in the past week from public spaces, home, personal vehicles, or school were coded as binary measures of SHS.

Bivariate linear regression models were fit to the data to determine the independent relationships between each social and environmental factor and SHS exposure, as measured by log-transformed cotinine concentration. A stepwise regression model approach was then used to determine the best-fitting linear regression model, based upon R^2 (the percentage of variability of the dependent variable that is explained by the variation in independent variables), the sum of squared errors (the sum of the squares of residuals, or deviations predicted from actual empirical values of data), and goodness-of-fit, as determined by the lowest estimated Akaike's Information Criteria (AIC)[19]. We also checked for potential interactions between the variables included in the models with age, gender, and race/ethnicity for possible effect modification. All analyses were conducted using SAS 9.4 (SAS Institute Inc., Cary, NC, USA).

RESULTS

Sample characteristics

Table 1 gives the correlations, means and standard deviations, and percentages for the variables included in this study. A total of 105 adolescents (mean age=13.3 years, SD=1.5) were included in this cross-sectional analysis. Most study participants (90.4%) were African American. Of the remaining, 6.7% were White and 2.9% were Hispanic or another race/ethnicity. More than half (53.3%) were female. Less than 25% of parents had earned a Bachelor's degree or higher. About a third of the sample resided in multi-unit housing. The mean number of adults within a household was 1.6 (SD=1.4) and the mean number of children within a household was 2.6 (SD=1.6). Approximately 67.7% of parents reported home smoking policies that do not permit smoking within the home.

Secondhand smoke exposure measures and agreement analysis

On average, in the past week, adolescents reported: 1.1 (SD=2.2) days of SHS exposure at home, 0.7 (SD=1.6) days of SHS exposure within personal vehicles, 1.7 (SD=2.3) days of SHS exposure at school, and 0.8 (SD=1.8) days of SHS exposure in other public spaces. As shown in Table 1, adolescent-reported sources of SHS exposure were found to

Table 1. Correlations, means (standard deviations), and percentages, for study variables: adolescents, place, and behavior study, Virginia, USA, 2019–2020 (N=105)

Variables	Correlations															n (%) or Mean (SD)
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	
1. Log-transformed cotinine concentration [log (ng/mL), range: -9.2–1.08][a]	-															-6.7 (4.0)
Adolescent-reported sources of secondhand smoke exposure (days/week, range: 0–7)																
2. At home (n=90)	0.18	-														1.1 (2.2)
3. Within personal vehicle (n=93)	0.15	0.77**	-													0.7 (1.6)
4. At school (n=92)	0.12	0.37**	0.38**	-												1.7 (2.3)
5. In other public spaces (n=87)	0.15	0.68**	0.53**	0.70**	-											0.8 (1.8)
6. Age (years) (range: 11–17)	0.01	-0.03	0.05	0.11	0.02	-										13.3 (1.5)
7. Adolescent gender	0.01	-0.08	-0.16	-0.04	-0.12	-0.12	-									
Male																49 (46.7)
Female																56 (53.3)
8. Adolescent race/ethnicity	0.01	-0.06	-0.06	-0.11	-0.13	0.02	-0.08	-								
African American																94 (90.4)
Non-African American (e.g. White, Hispanic, Asian, other)																10 (9.6)
9. Parent education	-0.24*	-0.03	-0.35	-0.11	-0.01	0.08	-0.18	-0.09	-							
Less than high school																24 (22.9)
High school diploma/GED																21 (20.0)
Some college																34 (32.4)
Bachelor's degree or higher																26 (24.8)
10. Parent tobacco use	0.37**	0.25*	0.40**	0.15	0.16	-0.03	-0.09	-0.12	-0.24*	-						28 (26.9)
11. Peer tobacco use	-0.02	0.17	0.24*	0.22*	0.18	-0.03	-0.10	-0.08	0.18	-0.02	-					8 (8.2)
12. Resides in multi-unit housing	0.32**	0.36**	0.27*	0.19	0.24*	-0.08	-0.06	0.05	-0.39**	0.33**	0.03	-				34 (33.3)
13. Number of adults within the home (range: 0–6)	-0.11	-0.18	-0.07	0.05	-0.10	0.08	0.14	-0.05	-0.07	-0.14	-0.03	-0.18	-			1.6 (1.4)
14. Number of children within the home (range: 0–6)	0.10	0.03	0.06	0.20	0.13	-0.01	-0.02	-0.01	-0.33**	0.14	0.05	0.05	0.22*	-		2.6 (1.6)
15. Home smoking policy	-0.25*	-0.38**	-0.41**	-0.11	-0.22*	-0.01	-0.03	-0.01	0.02	-0.34**	-0.03	-0.08	-0.03	0/03	-	
No smoking permitted in home																69 (67.7)
Smoking permitted in home sometimes or all the time																33 (32.4)

*p<0.05, ** p<0.01. a Using natural log.

be positively correlated with one another, as well as positively correlated with parental tobacco use, peer tobacco use, residing in multi-unit housing, and negatively correlated with having a home smoking policy that does not permit smoking in the home. Adolescent age, sex, and race/ethnicity were not correlated with these measures.

Salivary cotinine data were available for all 105 adolescent participants included in the analyses. Cotinine concentrations ranged between <0 ng/mL and 2.93 ng/mL (mean=0.29, SD=0.68). Approximately 70.5% (n=74) had cotinine concentrations below the level of detection (e.g. <0 ng/mL). To retain all cotinine values for analyses, individuals with cotinine concentrations of <0 ng/mL were recoded as 0 ng/mL. Then, a value of 0.0001 ng/mL was added to the cotinine concentrations of each participant, prior to applying log-transformation. After log-transformation (using natural log), salivary log cotinine levels ranged from -9.2 to 1.1 (mean=-6.7, SD=4.0). As shown in Table 1, this measure was negatively correlated with parental education and having a home smoking policy that does not permit smoking in the home, and positively correlated with parent tobacco use and residing in multi-unit housing.

To conduct agreement analyses, adolescent-reported SHS exposure and log-transformed salivary cotinine concentration were recoded into binary measures, indicating ≥1 day of SHS exposure in the past week for each variable or having a cotinine concentration ≥1 ng/mL for cotinine level, respectively. Adolescents reported ≥1 day of SHS exposure: in public spaces (39.1%), at home (26.7%), in personal vehicles (21.9%), and at school (18.1%). Overall, adolescent-reported SHS exposure from any of these sources for at least one day in the past week was 49.5%. Meanwhile, approximately 13.3% of adolescent participants had cotinine concentrations that were indicative of passive SHS exposure (i.e. 1–3 ng/mL). As shown in Table 2, agreement analysis between recoded salivary cotinine and self-reported SHS measures yielded a Cohen's kappa value of 0.0026 (95% CI: -0.1284–0.1335), indicating weak agreement between self-reported SHS exposure and the cotinine measure. Among 53 adolescents who reported that they had not been exposed to SHS, 7 were 'under-reporters' and had salivary cotinine levels indicative of SHS exposure. Among the 52 adolescents who reported SHS exposure, 45 were considered 'over-reporters' and did not have salivary cotinine levels indicative of SHS exposure. Overall, 50.4% (53/105) of the self-reported responses regarding SHS exposure were congruent with cotinine levels (i.e. adolescents reporting no SHS exposure, who also had salivary cotinine levels indicative of no exposure), while 49.5% (52/105) of the responses were incongruent with salivary cotinine levels (i.e. adolescents were over- or under-reporting SHS).

Social and environmental determinants of log-transformed salivary cotinine levels

Bivariate linear regression models are shown in Table 3. These models demonstrate independently significant associations between lower parent education level (β=-0.89, SE=0.35, p=0.00125), parent tobacco use within the past 30 days (β=3.29, SE=0.83, p=0.0001), and residing in multi-unit housing (β=2.70, SE=0.81, p=0.0012) with increased log-transformed cotinine levels among non-smoking adolescents. Having a home smoking policy that does not permit smoking within the home was significantly associated with decreased log-transformed cotinine level (β=-2.10, SE=0.83, p=0.0132). Adolescent-reported SHS exposure within personal vehicles,

Table 2. Comparison of self-report passive exposure to salivary cotinine in the adolescents, place, and behavioral study, Virginia, USA, 2019–2020 (N=105)

	Self-reported passive exposure		
Salivary cotinine	*No exposure n (%)*	*Passive exposure n (%)*	*Total n (%)*
No exposure (<1 ng/mL)	46 (50.6)	45 (49.4)	91 (85.7)
Passive exposure (1–3 ng/mL)	7 (50.0)	7 (50.0)	14 (13.3)
Total	52 (50.5)	52 (49.5)	105 (100)

at home, at school, and in other public spaces, age, gender, race/ethnicity, peer tobacco use, and parent-reported number of adults and children within the home were not significantly associated with log-transformed cotinine levels.

Models predicting log-transformed salivary cotinine levels

Stepwise regression models for log-transformed salivary cotinine levels are shown in Table 4. A total of 73 adolescents were included in regression analyses for cotinine, as 32 adolescents had missing values in one or more variables. Of these models, Model 3 is the best-fitting according to R^2, the sum of squared errors, and goodness-of-fit, as determined by lowest AIC. Model 3 explains 21% of the variability in log-transformed cotinine level as explained by the independent variables included in the model. Further, Model 3 demonstrates statistically significant associations between parent tobacco use within the

Table 3. Bivariate linear regression models: adolescents, place, and behavior study, Virginia, USA, 2019–2020 (N=105)

Characteristics	F	df	R^2	Estimate	SE	p
Adolescent-reported secondhand smoke exposure						
Within personal vehicles (n=93)	2.00	91	0.0215	0.34	0.24	0.1611
At home (n=90)	2.87	88	0.0316	0.32	0.19	0.0939
At school (n=92)	1.26	90	0.0138	0.25	0.23	0.2647
In other public spaces (n=87)	1.84	85	0.0212	0.24	0.17	0.1785
Adolescent age (years) (range: 11–17, n=105)	0.01	103	0.0001	0.02	0.27	0.9353
Adolescent gender (Ref: Male, n=105)	0.01	103	0.0001	0.08	0.79	0.9153
Adolescent race/ethnicity (Ref: African American, n=104)	0.02	102	0.0002	0.18	1.32	0.8913
Parent education (Ref: <High school, n=105)	6.47	103	0.0591	-0.89	0.35	**0.0125**
Parent tobacco use (Ref: No, n=104)	15.75	102	0.1338	3.29	0.83	**0.0001**
Peer tobacco use (Ref: No, n=98)	0.05	96	0.0006	-0.35	1.50	0.8172
Resides in multi-unit housing (Ref: No, n=102)	11.14	100	0.1002	2.70	0.81	**0.0012**
Number of adults within home (range: 0–6, n=105)	1.24	103	0.0119	-0.32	0.29	0.2674
Number of children within home (range: 0–6, n=105)	1.05	103	0.0101	0.26	0.25	0.3084
Home smoking policy (Ref: Smoking permitted in the home, n=102)	6.36	100	0.0598	-2.10	0.83	**0.0132**

Bold values indicate statistical significance at p≤0.05.

Table 4. Linear regression models for salivary cotinine: adolescents, place, and behavior study, Virginia, USA, 2019–2020 (N=73)

Characteristics	Model 1 b, SE, p	Model 2 b, SE, p	Model 3* b, SE, p	Model 4 b, SE, p
Intercept	-7.54, 0.44, **<0.0001**	-7.91, 0.48, **<0.0001**	-6.98, 0.79, **<0.0001**	-5.65, 1.38, **<0.0001**
Parent tobacco use (Ref: No)	3.67, 0.86, **<0.0001**	3.02, 0.91, **0.0013**	2.56, 0.96, **0.0082**	2.41, 0.96, **0.0141**
Resides in multi-unit housing (Ref: No)		1.66, 0.86, **0.0500**	1.72, 0.86, **0.0460**	1.40, 0.89, 0.1210
Home smoking policy (Ref: Smoking permitted in the home)			-1.23, 0.84, 0.1452	-1.29, 0.84, 0.1263
Parent education (Ref: <High school)				-0.44, 0.37, 0.2419
F, df, p	18.3, 96, **<0.0001**	11.30, 95, **<0.0001**	8.35, 94, **<0.0001**	6.64, 93, **<0.0001**
R^2	0.18	0.19	0.21	0.20
RMSE	3.75	3.70	3.68	3.67
SSE	1350.87	1299.66	1270.50	1251.83
AIC	261.27	259.32	259.10	259.64

*Model 3 is the best-fitting model, based upon lowest estimated AIC. Bold values indicate statistical significance at p≤0.05. RMSE: root mean squared error. SSE: sum of squared errors. AIC: Akaike's information criteria.

past 30 days (β=2.56, SE=0.98, p=0.0082), and residing in multi-unit housing (β=1.72, SE=0.86, p=0.0460) with increased log-transformed cotinine levels among non-smoking adolescents. Although we checked for potential interactions between the variables included in the models with age, gender, and race/ethnicity for possible effect modification, interaction terms were not statistically significant (details not shown).

DISCUSSION

We sought to determine the level of agreement between adolescent-reported SHS exposure and cotinine concentration and to identify social and environmental determinants of salivary cotinine levels. Results demonstrated weak agreement between adolescent-reported SHS exposure and salivary cotinine levels and identified statistically significant associations between parent tobacco use within the past 30 days and residing in multi-unit housing with log-transformed cotinine concentration among non-smoking adolescents. The best-fitting model for salivary cotinine levels included: having a home smoking policy that does not permit smoking within the home, parental tobacco use, and residing in multi-unit housing. Though, having a home smoking policy that does not permit smoking within the home was not significantly associated with log-transformed salivary cotinine level.

Among this sample, adolescent-reported SHS exposure from sources external to the home environment were common. Adolescents reported exposure to SHS ≥1 day in the past week: in public spaces (39.1%), at home (26.7%), in personal vehicles (21.9%), and at school (18.1%). These estimates are similar to those reported by a previous study using 2013 National Youth Tobacco Survey data that demonstrated that among US middle and high school students, 39.9% reported secondhand smoke exposure for ≥1 day in the past week at a public area, 25.0% in a vehicle, 24.9% at school, and 23.9% at home[20]. This information helps us to better understand the places where adolescents may be exposed to SHS and plan for potential areas for prevention and intervention. For example, smoke-free laws prohibiting smoking in all indoor areas of a venue have been found to fully protect non-smokers from involuntary SHS exposure indoors and legislation regulating public smoking has been found to reduce SHS levels[21]. Despite the successes of these regulations, adolescents within our study report on average, 0.8 days a week where they are exposed to SHS in public places and 1.7 days a week where they are exposed to SHS at school.

The prevalence of secondhand smoke exposure, as measured by cotinine in this sample (13.3%) is lower than that presented in other studies using data from larger national studies [32.0% among those aged 12–19 years, using National Health and Nutrition Examination Survey (NHANES) data from 2013–2014[22]]. The lower rates of exposure found in our sample may be due to the selection of self-reported non-smoking adolescents and the use of a lower cotinine threshold (<3 ng/mL in our study vs <10 ng/mL in NHANES). We used a lower cotinine threshold within our study to allow for potential misclassification bias (e.g. classifying adolescents as non-smokers when they may be actively smoking). Our study is also unique in that it contains information on self-reported measures of SHS exposure and biomarkers among a population that is demographically at higher risk for SHS: mostly African American adolescents, who are at an age that may be at high risk for SHS but have not initiated smoking and may reside in multi-unit housing. Future studies are needed to further validate reported results, especially since existing studies examining the validity of adolescent-reported SHS are limited[11].

Analyses revealed weak agreement between adolescent-reported SHS exposure and salivary cotinine concentration. Correlations between sources of SHS exposure external to the home environment and salivary cotinine ranged from 0.12 to 0.18 but were not statistically significant. Further, 49.5% of self-reported responses under- or over-reported SHS exposure, according to salivary cotinine. These findings suggest that adolescent-reported SHS exposure may not be a reliable proxy for actual SHS exposure, and underscores the importance of assessing the reliability and validity of adolescent-reported exposures in different settings[11].

Prior research has found that participants provide more accurate responses if they are asked to recall SHS exposure occurring within the home or personal vehicles[23]. Alternatively, adolescents are less accurate

in reporting the duration of exposure[24]. Over-reporting may occur when respondents conflate SHS exposure with thirdhand smoke exposure (i.e. residual tobacco smoke particles that settle on surfaces and dust)[23] or recall smelling tobacco smoke near them in specific locations[11]. Recall accuracy can be improved by reducing the timeframe between the discrete event and the length of the recall period. The recommended recall period is a maximum of 7 days within a single assessment[11]. However, given the weak agreement between self-reported SHS and salivary cotinine found in our study (which included a recall period of the past 7 days), it may be useful for future studies to collect and examine self-reported and biomarker-derived data at multiple timeframes and durations of exposure (e.g. past 24 hours, 3 days, and 7 days).

Thus, there are a few possible explanations why we might find weak agreement between self-reported SHS and cotinine. Under-reporting could have occurred if respondents had not realized that they had been exposed to SHS. Studies using NHANES data have found that self-reported exposure estimates are generally under-reported, relative to SHS exposure derived from cotinine[8]. Meanwhile, over-reporting could have occurred if respondents had conflated SHS exposure with thirdhand smoke exposure. Given findings from prior literature, this is likely to have occurred if respondents resided in or frequented areas that smelled like smoke during data collection[11]. The implication of this is that the survey instruments used in future studies may want to differentiate between SHS and thirdhand smoke exposure within specific locations (inside and outside of the home). Another possibility is that over-reporting could have resulted from adolescents reporting on exposures that were brief in duration and/or of low concentration that could not be detected due to the short half-life of cotinine (e.g. 72 hours)[25]. Future studies will need to conduct validity testing between adolescent-reported SHS exposure and cotinine across different race/ethnicity groups. Future studies are also needed to determine the best set of questions to ask adolescents regarding SHS exposure, similar to a recently published article that identified a highly sensitive set of questions for assessing child SHS exposure from parent-report[26].

Regular SHS exposure during adolescence can affect cardiovascular health into adulthood, potentially through negative effects that SHS has on diet, activity level, and percent body fat[27]. As adolescents grow older, they will gain more autonomy over their exposure to SHS[24] and there is some evidence to suggest that interventions focused on informing adolescents how to avoid SHS exposure may help to reduce SHS exposure[28]. However, by focusing interventions solely on adolescents, who have limited capacity to control their outside environments, we miss out on addressing other potentially relevant causes.

Parents and caregivers play a large role in shaping the environmental context of adolescent SHS exposure. Parental tobacco use has been associated with increased SHS exposure in adolescents and children, in our and other studies, even after controlling for the effects of socioeconomic factors[10]. Although not found to be statistically significant in adjusted models within our study, having a home smoking policy that does not permit smoking within the home has been previously associated with a reduction of SHS exposure in other studies[29]. Mixed findings may be associated with differences in the perceived addictiveness of tobacco products. For example, one study finds that adults with higher levels of perceived addictiveness to e-cigarettes are more likely to support complete e-cigarette bans at home, while adults who perceive e-cigarettes as non-addictive are more likely to support no home smoking rules[30]. Differences in perceived addictiveness might also explain why interventions focused on changing parent smoking behaviors to reduce adolescent SHS have yielded mixed results[31,32].

Another complicating factor in addressing adolescent SHS exposure is whether participants live in multi-unit housing (MUH). Our study results align with the current literature, which suggests that the implementation of a home smoking policy that does not permit smoking within the home might not protect MUH residents from SHS exposure[33]. Adolescents who live in multi-unit housing may still be at increased risk of SHS exposure due to the potential transfer of environmental smoke through walls, ductwork, windows, and ventilation systems. Air circulation patterns inside MUH can

facilitate involuntary SHS exposure among residents through shared hallways and ventilation systems. Consequently, physical separation of non-smokers from smokers will not necessarily eliminate SHS exposure among MUH residents[34]. Prohibiting smoking in all buildings of MUH is the only known effective means of protecting non-smokers from SHS exposure. Added to that, an estimated 79% of non-smokers residing in MUH prefer that their building be smoke-free; yet, only 7.1% actually reside in smoke-free buildings[35].

These findings support the need for specific measures to prevent SHS exposure in MUH, such as disseminating information about the short- and long-term health risks involved with SHS exposure among the public, educating MUH residents of their increased risk of SHS exposure, and the promotion of smoke-free MUH legislation and policy. Since current smokers are more likely to live in MUH than non-smokers, smoke-free MUH legislation and policies have the potential for addressing tobacco-related disparities by reducing in-home SHS exposure and smoking prevalence, similar to how clean indoor air legislation and policies prohibiting smoking in workplaces and public spaces have in the realm of de-normalizing smoking in public spaces[3].

Strengths and limitations

This study should be considered within the context of its limitations and strengths. The main limitation of this study is related to the small sample size and its potential generalizability to other populations. The sample includes adolescents who are aged 11–17 years, mostly of African American descent, and residing in the Mid-Atlantic region of the United States. Our sample may represent a population at greater risk for tobacco use; however, due to low sample size, we were unable to assess potential differences by race/ethnicity. Replication studies with more diversity in terms of ethnic populations and geographical location are needed. Additionally, our cross-sectional survey design does not allow investigation into temporality and/or causality between variables.

Despite these limitations, our study is strengthened by its use of self-reported and biomarker derived SHS exposure measures. Cotinine is considered the best valid measure of both tobacco use and exposure to SHS among non-smokers and many existing studies investigating determinants of SHS exposure have relied on self-reported measures of exposure and/or parent-report of adolescent SHS[11]. Self-reported SHS exposure may provide an underestimate, due to the difficulties involved with estimating the intensity, frequency, and duration of exposure. Furthermore, associations between self-reported SHS exposure and cotinine may differ according to specific locations, as self-reported measures of SHS exposure within indoor spaces outside of the home have a greater association with cotinine, relative to SHS exposure in public spaces[36].

CONCLUSIONS

Both accurate quantitative measurement of SHS exposure and a better understanding of its determinants are needed to develop policies and interventions that aim to further reduce SHS exposure among non-smoking adolescents. Results from our study demonstrate that parental tobacco use within the past 30 days and living in multi-unit housing should be considered when developing targeted interventions to reduce SHS exposure among adolescents.

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CONFLICTS OF INTEREST
The authors have completed and submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest and none was reported.

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AUTHORS' CONTRIBUTIONS
EKD performed the statistical analyses, assisted with the literature review, and prepared the introduction, methods, results, discussion, and conclusions sections of the text. KCB, KF-L and KK helped to conduct literature review and contributed to the introduction, analysis, and discussion of the text. RBH, DCW, MP and BF helped to review the introduction, statistical analysis, results, discussion, and conclusion. MH and LR were responsible for the processing of cotinine assays using saliva samples and assisted with editing of the methods, results, and discussion sections of the text.

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