

Original Investigation

Racial Differences in Hair Nicotine Concentrations Among Smokers

Benjamin J. Apelberg, Ph.D., M.H.S.,^{1,2} Lisa M. Hepp, M.P.H.,¹ Erika Avila-Tang, Ph.D., M.H.S.,^{1,2} Sungroul Kim, Ph.D.,³ Camille Madsen, M.P.H.,¹ Jiemin Ma, Ph.D., M.H.S.,¹ Jonathan M. Samet, M.D., M.S.,⁴ & Patrick N. Breyse, Ph.D., M.H.S.⁵

¹ Institute for Global Tobacco Control, Department of Health, Behavior and Society, Johns Hopkins Bloomberg School of Public Health, Baltimore, MD

² Department of Epidemiology, Johns Hopkins Bloomberg School of Public Health, Baltimore, MD

³ Department of Environmental Health Sciences, Soonchunhyang University, Asan, South Korea

⁴ Department of Preventive Medicine, Keck School of Medicine, University of Southern California, Los Angeles, CA

⁵ Department of Environmental Health Sciences, Johns Hopkins Bloomberg School of Public Health, Baltimore, MD

Benjamin J. Apelberg is now at the U.S. Food and Drug Administration.

Corresponding Author: Patrick N. Breyse, Ph.D., M.H.S., Department of Environmental Health Sciences, Johns Hopkins Bloomberg School of Public Health, 615 North Wolfe Street, Baltimore, MD 21205, USA. Telephone: 410-955-3608; Fax: 410-955-9334; E-mail: pbreyse@jhsph.edu

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Abstract

Introduction: In the United States, race/ethnicity is a strong determinant of tobacco use patterns, biomarkers of tobacco smoke components and metabolites, and likelihood of successful cessation. Although Black smokers tend to smoke fewer cigarettes than White smokers, they have higher cotinine levels and disease risk and lower cessation success. We examined racial differences in hair nicotine concentrations among daily tobacco smokers ($n = 103$) in Baltimore, Maryland.

Methods: Participants completed a survey, and hair samples were collected and analyzed for nicotine concentration using gas chromatography coupled with mass spectrometry.

Results: After adjustment, hair nicotine concentrations among Black smokers were more than 5 times higher than among White smokers (95% CI 3.0, 10.5). Smokers reporting hair treatments other than coloring (bleaching, permanent, or straightening) in the past 12 months had 66% lower (95% CI 32%, 83%) hair nicotine concentrations. Smokers reporting smoking their first cigarette within 30 min of waking had twice the hair nicotine concentrations of those whose time to first cigarette was greater than 30 min after waking (95% CI 1.1, 4.2). For every additional cigarette smoked per day up to 20, mean hair nicotine concentration among all smokers increased by 4% (95% CI -1%, 9%).

Conclusions: This study demonstrates that Black smokers have substantially higher hair nicotine levels than White smokers, after controlling for cigarettes smoked per day and other exposure sources. Time to first cigarette, cigarettes smoked per day, and use of hair treatments other than coloring were also associated with hair nicotine concentrations among smokers.

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Introduction

In the United States, racial and ethnic differences have been reported in tobacco use, biomarkers of dose, likelihood of successful cessation, and disease risk. Black smokers tend to smoke fewer cigarettes per day than White smokers (Pleis & Lucas, 2009) but have higher serum cotinine concentrations (Caraballo et al., 1998). Black smokers are more likely to make a quit attempt than White smokers (Schoenborn & Adams, 2010; U.S. Department of Health and Human Services [USDHHS], 1998), but rates of successful cessation are consistently higher among Whites (King, Polednak, Bendel, Vilsaint, & Nahata, 2004; USDHHS). This difference is further evidenced by higher quit ratios (defined as the proportion of ever-smokers who have successfully quit) among Whites (Fu et al., 2008; Giovino, 2002). Although Black smokers consume fewer cigarettes per day and are more likely to be non-daily smokers (Pleis & Lucas), they are more likely than Whites to develop some tobacco-related diseases, such as lung cancer and esophageal cancer and cardiovascular disease. For example, in 2008, the age-adjusted incidence rate of lung cancer was approximately 30% higher for Black men compared with White men, even though smoking prevalence among Blacks has been historically lower than among Whites (Fast Stats: An interactive tool for access to SEER cancer statistics). Blacks are also at greater risk for cerebrovascular disease and ischemic heart disease, two diseases caused, in part, by tobacco use (USDHHS, 1998).

Although evidence suggests that race affects serum cotinine concentrations, cessation success, and risk for tobacco-caused diseases, explanations for these differences have not been fully elucidated. Blacks have been shown to metabolize nicotine and cotinine more slowly than Whites (Benowitz, 2008; Benowitz et al., 1999; Berg, Mason, Boettcher, Hatsukami, & Murphy,

2010; Moolchan, Franken, & Jaszyna-Gasior, 2006; Perez-Stable, Herrera, Jacob, & Benowitz, 1998), leading to a greater internal dose of cotinine. It has also been suggested that differences in the type of cigarettes smoked (Clark, Gautam, & Gerson, 1996; Jarvik, Tashkin, Caskey, McCarthy, & Rosenblatt, 1994; McCarthy et al., 1995) and smoking topography (Benowitz, Pérez-Stable, Herrera, & Jacob, 1995) could also play a role in increased exposure. In addition, several studies have shown that nicotine bioaccumulates in melanin-containing tissues, suggesting another pathway for increased exposure among Black smokers (Claffey, Stout, & Ruth, 2001; Dehn, Claffey, Duncan, & Ruth, 2001; Yerger & Malone, 2006).

Although racial disparities in cotinine concentrations have been well characterized, limited data are available for other biomarkers of tobacco exposure. During the past decade, hair nicotine has been used increasingly to assess tobacco exposure because it represents a longer duration of exposure than cotinine in serum, urine, or saliva (Al Delaimy, 2002; Kim, Wipfli, Avila-Tang, Samet, & Breysse, 2009; Kintz, 1992). Furthermore, hair is often easier to collect, transport, and store than other biospecimens. Despite increased use of hair nicotine as a biomarker of tobacco exposure, little information is available on the distribution of hair nicotine levels among Black and White smokers. In addition, the shape of the dose–response relationship between tobacco consumption and hair nicotine concentrations has not been fully described. In this study, we examine racial differences in hair nicotine concentrations among a sample of smokers in the Baltimore metropolitan area.

Methods

Study Population

We conducted a cross-sectional survey of adults 18 years of age and older in Baltimore, Maryland, to assess the use of hair nicotine as a marker of tobacco use and secondhand smoke (SHS) exposure. A total of 300 participants were recruited into this convenience sample from the Baltimore metropolitan area, evenly split between (a) smokers, (b) nonsmokers regularly exposed to SHS (e.g., nonsmokers who live with a smoker or are exposed to SHS in the workplace, such as bar and restaurant workers), and (c) nonsmokers with limited exposure to SHS. Participants were recruited through advertisements in local newspapers and outside of various commercial establishments, such as grocery stores and markets. Recruitment was conducted with stratification to ensure a study population with sufficient racial diversity.

Self-reported users of nicotine replacement therapy and users of smokeless or chewing tobacco were excluded from the study. Pregnant women were also excluded because pregnancy may affect the uptake and metabolism of nicotine (Rebagliato et al., 1998). In addition, those individuals without sufficient hair for analysis (at least 2 cm in length after stretched out) were excluded from the study.

In this analysis, only self-reported daily smokers were included. In total, 103 smokers were included in the study.

Questionnaire

Study protocols were approved by the Johns Hopkins Institutional Review Board, and informed consent was received from

individuals who agreed to participate in the study. An interviewer-administered questionnaire was used to collect information on demographic characteristics (e.g., age, gender, level of education, and race/ethnicity), smoking history, exposure to SHS, and characteristics of respondents' hair. Race was based on self-report, and respondents were allowed to select multiple racial categories. Due to the small sample size for some of the categories, respondents were categorized into three groups for analysis: White, Black or African American, and other. The majority of respondents in the "other" category were Asian or Asian-American. Five daily smokers reported multiple racial groups, all of which included "Black or African American." These participants were categorized as Black. Respondents who were smokers were asked about the number of cigarettes smoked per day (both manufactured and hand rolled). Age at daily smoking initiation was obtained among respondents who reported ever being daily smokers. Time to first cigarette use after waking was reported in categories of <5, 5–30, 31–60, and >60 min. For the analysis, these categories were collapsed to ≤30 and 30+ min after waking. Smokers were also asked whether they typically smoked menthol or regular cigarettes, which was confirmed by asking to see their pack of cigarettes, when possible.

SHS exposure in the home was assessed by asking respondents about the smoking policy in the home and how often individuals smoked in the home (daily, weekly, monthly, less than monthly). At the time of hair collection, respondents were asked about their natural hair color. Categories of hair color were based on self-report and were coded as black, brown/red, or light (e.g., blond, gray). If a respondent reported more than one hair color, it was coded as the darker of the hair colors reported. In a sensitivity analysis, we categorized respondents based on the lighter of the hair colors, but the results did not change. Respondents were also asked if they had any chemical or cosmetic hair treatment applied in the 12 months prior to the survey. This included any of the following: coloring, bleach, highlights, permanent, or straightening. These responses were categorized as hair coloring (coloring or highlights) and other hair treatment (bleach, permanent, or straightening).

Hair Collection

Two small samples of hair (approximately 30–50 strands) were cut near the hair root from the back of the scalp where there is the most uniform growth pattern between individuals. For subjects with short curly hair, hair samples were collected from the base of the neck. The hair samples were taped onto a labeled index card with an arrow pointing to the direction of the scalp end (or placed in an envelope if the hair was too short). The hair samples were immediately placed in a properly marked clean sample collection ziplock bag sealed tightly for storage and transportation. Hair samples were stored in a refrigerator at Johns Hopkins Bloomberg School of Public Health until analysis.

Laboratory Analysis

Hair nicotine was measured in the Exposure Assessment Facility at the Institute for Global Tobacco Control. Levels of nicotine in hair were measured using liquid–liquid extraction methods and gas chromatography coupled with mass spectrometry as described previously (Kim, Wipfli, Avila-Tang, et al., 2009). If

the hair length was ≥ 2 cm, only the 2 cm of hair closest to the scalp was processed. If the hair was < 2 cm length, the entire length was processed. For each sample, 30 mg of hair was weighed, cleaned with dichloromethane, and incubated in 1 M sodium hydroxide at 100 °C for 10 min. After cooling, samples were extracted and d3-nicotine was added as an internal standard. Nicotine was separated on a 30-m ZB-5 capillary column and quantified by a Shimadzu QP-5000 gas chromatography–mass spectrometry system using selected-ion monitoring mode (m/z 84 and 162 for nicotine and 87 and 165 for d3-nicotine, respectively). The hair concentration of nicotine was calculated by dividing the amount of nicotine collected in each hair sample (μg) by the amount of hair sampled (g). For quality control purposes, 10% of hair samples were analyzed in duplicate. The limit of detection was 0.05 ng/mg for a 30-mg hair sample. Accuracy and precision of measurements were evaluated by analyzing checking standards (equal to the calibration standard of 0.5 ng/ml: $n = 8$ within batch and $n = 48$ between batch). The recoveries obtained were 94% and 108%, respectively, for within and between batches with 9% relative standard deviation.

Statistical Analysis

Descriptive statistics (frequencies, percentages, means, medians, and interquartile ranges) and plots were used to characterize the study population. Differences in demographic and smoking-related characteristics by race were determined using Fisher's exact test and the Mann–Whitney U test (Wilcoxon rank-sum test). Statistical significance was based on a p value less than .05.

Linear regression was conducted with log-transformed hair nicotine as the outcome variable to account for the right-skewed distribution of nicotine concentration. Regression coefficients were back-transformed to estimate geometric mean ratios comparing different levels of the independent variable. The following independent variables were included in regression models: age, gender, education level (up to and including a high school degree, more than a high school degree), hair treatment in the past 12 months, and hair color (black, brown, light). In a sensitivity analysis, we conducted the analysis in a subset of the population that reported no change in smoking behavior over the 2 months prior to the survey. No substantial differences in the findings were observed, so the full population is reported here.

Variables that were significant (or of borderline significance) in univariate models were included in the multivariate regression model. All analyses were conducted in SAS version 9.1.

Results

In total, 103 daily smokers were enrolled in the study (33 White, 54 Black, and 16 other). Table 1 describes the demographic and smoking-related characteristics of the study population by racial group. Statistically significant differences ($p < .05$) between racial groups were observed for gender, type of cigarette smoked, smoking exposure in the home at least weekly, and hair color. Black smokers were more likely to be menthol cigarette smokers (92%) and to be exposed to smoking in the home at least weekly (89%). Statistically significant differences in self-reported natural

hair color were observed, as well in hair treatment other than coloring in the last 12 months.

Hair nicotine concentrations were significantly higher among Black smokers (median 37.8 ng/mg), compared with White smokers (median 5.7 ng/mg) and “other” groups (median 9.4 ng/mg), even after adjusting for cigarettes smoked per day (Figure 1a and b).

Table 2 shows the results from linear regression models of log-nicotine regressed on various predictors. In univariate models, race, age, age at initiation, hair coloring, cigarettes smoked per day, time to first cigarette, and smoking exposure in the home were all associated with hair nicotine concentration. Hair treatment other than coloring (bleaching, permanent, or straightening) was of borderline statistical significance. In multivariate models, only race, hair treatment other than coloring, and time to first cigarette remained as significant predictors, while cigarettes smoked per day was of borderline statistical significance. After adjustment for potential confounders, mean hair nicotine concentrations among Black smokers were more than 5 times higher than among White smokers (95% CI 3.0, 10.5). Smokers reporting the use of hair treatments other than coloring in the past 12 months had 66% lower (95% CI 32% to 83%) mean hair nicotine concentrations than smokers who did not use hair treatment. Smokers reporting smoking their first cigarette within 30 min of waking had twice the hair nicotine concentrations of those whose time to first cigarette was greater than 30 min after waking (95% CI 1.1, 4.2). For every additional cigarette smoked per day, mean hair nicotine concentrations increased 4% (95% CI –1% to 9%). No association was observed between cigarettes smoked per day and hair nicotine above one pack per day.

Although we collected information on cigarette type, the lack of variation among Black smokers limited our ability to include menthol cigarette use in multivariate regression analyses. In univariate analyses, we found no difference in hair nicotine concentrations and hair nicotine per cigarette among White smokers. However, among Black smokers, we found a borderline significantly higher hair nicotine concentration among menthol cigarette smokers compared with regular cigarette smokers (median 38.4 vs. 16.4 ng/mg, $p = .06$; 3.8 vs. 1.4 ng/mg per cigarette, $p = .1$). However, these findings are based on only four Black regular cigarette smokers.

Discussion

This study demonstrates that Black smokers have substantially higher levels of hair nicotine than White smokers, after controlling for cigarettes smoked per day and other potential sources of exposure. In addition, we found that, among this population of smokers, time to first cigarette, use of hair treatments other than coloring, and cigarettes smoked per day were important predictors of hair nicotine concentrations.

The greater concentrations of hair nicotine observed in Black smokers have important implications for racial differences in nicotine dependence and, along with differences in access to counseling and treatment (Royce et al., 1993), may contribute to disparities in rates of successful quitting. Although previous studies have found higher levels of serum cotinine

Table 1. Demographic and Smoking-Related Characteristics by Race

	White smokers (N = 33)	Black smokers (N = 54)	Smokers with “other” race/ethnicity (N = 16)
Demographics			
Age (years), mean (SD)	37 (11.7)	41 (12.7)	42 (14.0)
Gender ^a , n (%)			
Male	11 (33)	13 (24)	13 (81)
Female	22 (67)	41 (76)	3 (19)
Bleach, permanent, or straightening ^a (past 12 months), n (%)			
Yes	2 (6)	14 (26)	2 (13)
No	31 (94)	40 (74)	14 (87)
Hair coloring (past 12 months), n (%)			
Yes	13 (39)	12 (22)	6 (38)
No	20 (61)	42 (78)	10 (62)
Hair color ^a , n (%)			
Black	1 (3)	35 (65)	12 (75)
Brown	21 (66)	17 (32)	4 (25)
Light	10 (31)	2 (4)	0 (0)
Smoking behavior and SHS exposure			
Cigarettes smoked per day, mean (SD)	17.9 (9.8)	14.5 (7.9)	17.3 (5.3)
Age at initiation (years), mean (SD)	16.4 (6.1)	16.9 (6.2)	18.7 (6.0)
Time to first cigarette, n (%)			
≤30 min	23 (70)	45 (83)	14 (87)
>30 min	10 (30)	9 (17)	2 (13)
Cigarette type ^a , n (%)			
Menthol	18 (56)	43 (91)	4 (25)
Regular	14 (44)	4 (9)	12 (75)
Home SHS exposure ^a , n (%)			
At least weekly	19 (58)	48 (89)	9 (56)
<Weekly	14 (42)	6 (11)	7 (44)
Biomarker concentrations (ng/mg), median (IQR)			
Hair nicotine ^a	5.7 (1.6–18.0)	37.8 (14.1–76.1)	9.4 (5.9–11.5)
Hair nicotine per cigarette ^a	0.5 (0.2–1.0)	3.5 (0.8–4.7)	0.5 (0.4–0.8)

Note. Missing values: cigarette type (White 1, Black 7); hair color (White 1). IQR = interquartile range; SHS = secondhand smoke.

^aStatistically significant difference between racial groups: *p* < .05.

among Black smokers (Caraballo et al., 1998; Wagenknecht et al., 1990), the magnitude of the difference observed in the present study is substantially greater. Cotinine, as a metabolite, may be more sensitive than nicotine to metabolic variability

among Black smokers because of cytochrome P-450 2A6 variability (Ho et al., 2009). Therefore, hair nicotine could be more sensitive to actual exposure than cotinine in Black smokers.

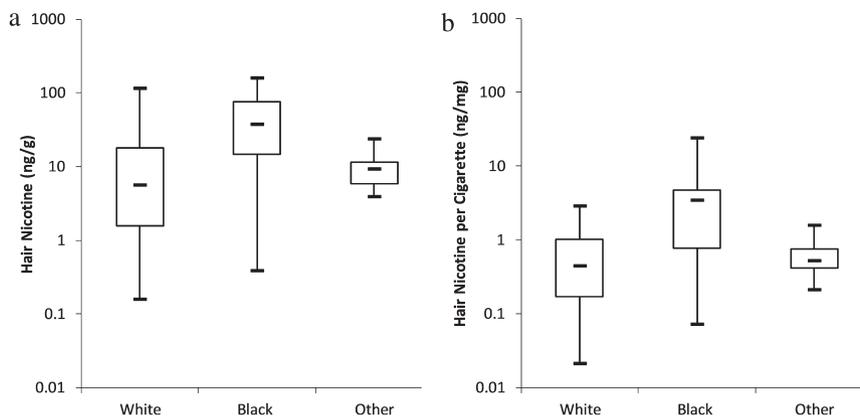


Figure 1. Distribution of hair nicotine (a) and hair nicotine per cigarette (b) among smokers by race. “Other” refers to self-reported race other than White or Black or African American.

Table 2. GM Ratios of Hair Nicotine Concentrations

	Univariate			Multivariate ^a		
	GM ratio	95% CI		GM ratio	95% CI	
Race						
Black	4.69*	2.56	8.59	5.58*	2.97	10.5
White	Ref			Ref		
Other	1.62	0.70	3.73	1.73	0.80	3.76
Gender						
Male	1.48	0.79	2.78			
Female	Ref					
Age						
10-year increase	1.29*	1.02	1.63	1.01	0.82	1.24
Age of initiation						
<18 years	2.04*	1.10	3.78	1.46	0.87	2.46
≥18 years	Ref			Ref		
Hair color						
Black	1.68	0.89	3.20			
Brown	Ref					
Light	1.30	0.48	3.53			
Bleach, permanent, or straightening						
Yes	0.50	0.23	1.10	0.34*	0.17	0.68
No	Ref			Ref		
Hair coloring						
Yes	0.41	0.22	0.78	0.75	0.43	1.28
No	Ref			Ref		
First cigarette						
≤30 min	3.57*	1.79	7.14	2.17*	1.14	4.17
>30 min	Ref			Ref		
Cigarettes/day						
Per cigarette (≤20)	1.05*	1.00	1.11	1.04	0.99	1.09
Per cigarette (>20)	1.03	0.88	1.20	1.01	0.92	1.10
Smoking in home						
At least weekly	2.54*	1.31	4.94	1.01	0.55	1.86
<Weekly	Ref			Ref		

Note. GM = geometric mean; Ref = reference group.

^aVariables included in multivariate model: race, age, age at initiation, bleach/permanent/straightening, hair coloring, time to first cigarette, cigarettes per day, and smoking in the home.

* $p < .05$.

Previous studies have demonstrated clear racial differences in hair cotinine concentrations as well. In a study of asthmatic children enrolled in an intervention trial to reduce SHS exposure, Wilson, Kahn, Khoury, and Lanphear (2007) found hair cotinine concentrations to be approximately fourfold higher in Black children than in White children, after controlling for air nicotine and exposure to SHS in the home. Serum cotinine concentrations were also higher among Black children, but only by about 30%. An analysis of the same data by Kalkbrenner et al. (2010) confirmed that Black race was the strongest predictor of hair cotinine concentration in these children. Experimental studies of other drugs, such as cocaine, have also found racial differences in uptake into hair, possibly due to differences in melanin content (Henderson, Harkey, Zhou, Jones, & Jacob, 1998; Joseph, Su, & Cone, 1996).

The hair nicotine concentrations observed among Black smokers in our study are generally higher than previously observed among smokers of other racial/ethnic backgrounds. Zahlsen and Nilsen (1994) found a mean hair nicotine concentration of

42.4 ng/mg among 13 smokers in Norway. However, the hair samples were not washed and external contamination may have contributed to the measured concentrations. Chetiyankornkul, Toriba, Kizu, Kimura, and Hayakawa (2004) analyzed hair nicotine from 10 Japanese smokers and found a mean hair nicotine concentration of 39.0 ng/mg. Man, Ismail, Harn, Lajis, and Awang (2009) reported mean hair nicotine concentration of 26.3 ng/mg among 17 young adult male smokers in Malaysia. Studies among small samples of smokers in Canada and the United States found hair nicotine concentration of 19.9 ng/mg (mean; Eliopoulos, Klein, & Koren, 1996) and 6.2 ng/mg (geometric mean; Okoli, Hall, Rayens, & Hahn, 2007), respectively. Some of the variation observed is likely to be due to differences in amount smoked as well as analytic methods and procedures. Future research should also examine the role of hair type on nicotine uptake and adsorption.

We found that hair treatment other than coloring (bleaching, permanent, straightening) in the past 12 months was associated with lower concentrations of hair nicotine, after controlling for

cigarettes smoked per day and other factors. This finding is consistent with previous evidence (Al Delaimy, 2002; Jurado, Kintz, Menendez, & Repetto, 1997; Kim, Wipfli, Avila-Tang, et al., 2009; Pichini, Altieri, Pellegrini, Pacifici, & Zuccaro, 1997). For example, Pichini et al. (1997) found that hair treatments were associated with significant decreases in hair nicotine concentration, most likely due to chemical degradation. Jurado et al. (1997) also found an average decrease of 30% in hair nicotine concentrations comparing bleached with unbleached hair. In a study of nonsmoking women from 31 countries, Kim, Wipfli, Avila-Tang, et al. (2009) reported that hair treatment was associated with a slightly lower hair nicotine concentration, which was not statistically significant.

Genetic differences play an important role in the metabolism and clearance of nicotine and cotinine and subsequent nicotine addiction (Benowitz, Lessov-Schlaggar, & Swan, 2008; Benowitz, Swan, Jacob, Lessov-Schlaggar, & Tyndale, 2006). Blacks tend to metabolize nicotine and cotinine more slowly, have a greater nicotine intake per cigarette, and smoke fewer cigarettes than Whites (Benowitz, 2008; Benowitz et al., 1999; Berg et al., 2010; Moolchan et al., 2006; Perez-Stable et al., 1998). Additionally, the majority of Black smokers prefer mentholated cigarettes. Because of the actions of menthol on airway receptors leading to a cooling sensation, it has been suggested that smoking menthol cigarettes may lead to greater depth of inhalation and a greater dose of nicotine and other tobacco-related constituents per cigarette smoked. Although menthol cigarette smoking has been shown to slow down nicotine metabolism (Benowitz, Herrera, & Jacob, 2004), the evidence relating menthol cigarette use to higher cotinine levels is mixed (Ahijevych & Gillespie, 1997; Ahijevych & Parsley, 1999; Ahijevych & Wewers, 1994; Clark et al., 1996; Muscat et al., 2009). In the present study, we collected information on the use of menthol cigarettes, but we were limited by the lack of variability in menthol use by race. In univariate analysis, there was a higher concentration of hair nicotine only among Black menthol smokers, which was of borderline statistical significance. However, there were only four Black smokers of non-menthol cigarettes, limiting our ability to parse any potential impact of menthol cigarette use on nicotine uptake. A recent review of the public health impact of menthol cigarettes by the Food and Drug Administration's Tobacco Products Scientific Advisory Committee found "conflicting evidence regarding the effects of menthol on nicotine exposure levels as measured by cotinine or 24-hr nicotine equivalents" (Tobacco Products Scientific Advisory Committee (TPSAC), 2011). Future studies should explore the role of menthol cigarettes in explaining racial differences in hair nicotine concentrations.

Several studies have demonstrated an affinity between nicotine and melanin, possibly due to partially irreversible binding (Claffey et al., 2001; Dehn et al., 2001). Tobacco-specific nitrosamines and benzo(a)pyrene, a polycyclic aromatic hydrocarbon, have also been shown to accumulate in melanin-containing tissues (Roberto, Larsson, & Tjalve, 1996; Yerger & Malone, 2006). Previous studies have found higher nicotine concentrations in darker hair fibers, attributable to levels of melanin (Gerstenberg, Schepers, Voncken, & Volkel, 1995; Uematsu, Mizuno, Nagashima, Oshima, & Nakamura, 1995). In the present study, we did not find a statistically significant relationship between hair color and nicotine levels, but smokers with black hair tended to have higher concentrations and the

percentage of participants with gray or blond hair was low. However, hair color was based on self-report, rather than melanin content. Future research should analyze nicotine concentration normalized for hair melanin content, rather than nicotine per milligram total hair.

Nicotine is thought to enter and deposit in the hair shaft through the circulation and because human hair grows at approximately 1 cm per month (Uematsu, 1993), the cumulative exposure over a given period of time can be quantified by obtaining the relevant length of hair from the scalp. Hair nicotine has been used widely to characterize exposure to SHS (Al Delaimy, 2002; Al Delaimy, Crane, & Woodward, 2002; Kim, Wipfli, Avila-Tang, et al., 2009; Nafstad et al., 1995; Wipfli et al., 2008). Studies conducted in smokers have generally found a correlation between reported cigarettes smoked per day and hair nicotine concentrations (Ashford et al., 2010; Eliopoulos et al., 1996; Jacqz-Aigrain et al., 2002; Okoli et al., 2007) but not uniformly (Al Delaimy, Crane, & Woodward, 2000). We observed a positive association between cigarettes smoked per day and hair nicotine concentrations, with borderline statistical significance. The association leveled off at about 20 cigarettes/day, likely due to titration of nicotine intake and consistent with what has been observed with cotinine concentrations in smokers from several countries (Blackford et al., 2006). Further, differences in smoking topography would be expected to result in variation in nicotine concentrations, even among smokers reporting the same number of cigarettes smoked per day. Therefore, biomarkers such as hair nicotine can improve exposure assessment.

There are some limitations to this study. We had a relatively small sample of daily smokers, which limited the multivariate analysis and the ability to assess interactions. Additionally, the sample was not randomly selected, but we would not anticipate bias in the relationship between smoking and hair nicotine as a result. Hair samples are washed with dichloromethane to remove nicotine adsorbed onto the outside of the hair. Washing is thought to remove the majority of nicotine deposited onto the hair externally but not all of it (Al Delaimy, 2002). We asked about hair treatment in the previous 12 months, but only treatment within the past 2–3 months would be expected to affect hair nicotine concentrations. However, we were still able to demonstrate a clear association between hair treatment and nicotine levels. Hair color was based on self-report, and small sample sizes and lack of variability limited the inferences to be made regarding the association with nicotine concentrations. In addition, combining brown and red hair may not be appropriate because of the differences in melanin type. However, only one respondent had red hair, and the results did not change when this observation was excluded. We also did not assess the use of other smoked tobacco products, such as hookah, as well as exposure to tobacco smoke outside of the home. As with any survey based on self-report, the potential for reporting error exists. Differential underreporting of the number of cigarettes smoked per day could somewhat bias the findings but could not account for the substantial difference in hair nicotine concentrations observed between racial groups.

Conclusions

In conclusion, this study demonstrated that Black smokers have substantially higher hair nicotine concentrations than White

smokers, after controlling for cigarettes smoked per day and other potential confounders. In addition, time to first cigarette, the use of hair treatment other than color, and cigarettes smoked per day among pack or less per day smokers were predictors of hair nicotine concentrations. Future research among smokers using hair nicotine should take into account race/ethnicity, nicotine dependence, and hair treatment information in the analysis and interpretation of results.

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Declaration of Interests

None declared.

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