Determinants of Hair Nicotine Concentrations in Nonsmoking Women and Children: A Multicountry Study of Secondhand Smoke Exposure in Homes

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Abstract

The main purpose of this study was to identify and evaluate determinants of hair nicotine concentrations in nonsmoking women and children exposed to secondhand tobacco smoke at home. Hair samples were collected from nonsmoking women (n = 852) and from children (n = 1,017) <11 years of age living in households (n = 1,095) with smokers from 31 countries from July 2005 to October 2006. Participants' ages, activity patterns and socioeconomic characteristics including education and employment status, and hair treatment information were collected. Multilevel linear regression modeling was used to identify the main determinants of hair nicotine concentration measured by gas chromatography coupled with mass spectrometry. Increased indoor air nicotine concentration at home were associated with increased hair nicotine concentrations in nonsmoking women and children. This association was not changed after controlling for other explanatory variables. After controlling for age, length of exposure, and socioeconomic characteristics, hair nicotine concentrations in nonsmoking children and women were estimated to be increased by 3% and 1%, respectively, for a 1 μg/m^3 increase in air nicotine concentration. The association between children's exposure to secondhand tobacco smoke at home and hair nicotine concentration was stronger among younger children and children with longer exposure at home. (Cancer Epidemiol Biomarkers Prev 2009;18(12):3407–14)

Introduction

Exposure to secondhand smoke (SHS) is a worldwide public health problem (1-4). Despite evidence showing causal associations between SHS and diverse adverse health effects in children and adults (5), vulnerable children remain involuntarily exposed to tobacco smoke in home environments. The Global Youth Tobacco Survey found that 46.5% of participants 13 to 15 years of age were living with smoking parents (6). A recent study by Wipfli et al. (7), conducted in 31 countries in Europe, Asia, and Latin America, showed that smoking at home remains a major source of SHS exposure for nonsmoking adults and children worldwide. To quantify levels of SHS exposure among nonsmoking women and children in diverse climates and cultures, Wipfli and colleagues (7) measured concentrations of air nicotine in the home as a tracer of tobacco smoke in the home environment, and concentrations of hair nicotine in women and children as a marker of personal exposure to SHS from all environments including the home. Allowing smoking inside houses was associated with a 13-fold increase in the concentration of air nicotine. Women and children living with at least one smoker also had higher concentrations of nicotine in their hair (7).

Aspects of the behavior of nonsmoking children may affect their exposures to SHS and the relationship between SHS exposure and hair nicotine, a biomarker of internal dose (8-10). Young children may be at greater risk for exposure to SHS in households with smokers because they are physiologically and behaviorally different from adults (11). Young children in general have limited mobility and spend most time indoors at home (10). In addition, even at similar exposure levels, differences in absorption between adults and young children can occur because children's body weight–adjusted respiratory rates are higher than those for adults (12). Women may be unavoidably exposed as they spend time in proximity to smoking husbands. Socioeconomic characteristics of nonsmoking women have been inversely associated with SHS exposure (13-15). In general, more educated women have a lower risk of exposure to SHS (13, 15). In this article, we extended analyses in the 31-country study reported by Wipfli et al. (7) and explored determinants of hair nicotine concentrations. We also assessed the association between air nicotine concentration and the amount of nicotine in hair.

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Materials and Methods

Study Population. A cross-sectional study of adult nonsmoking women and their children was conducted in 31 countries in Latin America, Asia, Eastern Europe, and the Middle East. Details on recruitment of households with and without smokers [~40 per country except China (n = 60), overall 1,284] and sample collection procedures have been previously described (7). For the present study, we selected all households allowing smoking inside the home (n = 1,095) and 852 nonsmoking women and 1,017 children, 11 y of age or younger, living in those households. Smoking restriction policy in the household was defined as "no smoking allowed throughout the entire indoor environment," based on self-report. All recruitment procedures and sampling strategies were approved by the Institutional Review Board of Johns Hopkins Bloomberg School of Public Health and by local Institutional Review Boards or other authorities in each country.

Data Collection. Using a standardized questionnaire for all countries, we collected sociodemographic characteristics including age, total time spent at home, maternal education, and employment status. Information was collected on the total number of cigarettes smoked daily in the household and on hair chemical treatments (i.e., dyed, bleached, straightened, permanent wave, and medicinal treatment) within the past 90 d. Only 3% of children had chemically treated hair. Thus, effects of the chemical hair treatments on hair nicotine concentrations were only evaluated among women.

To assess home exposure levels to SHS, we measured air nicotine concentrations over 7 d using passive air monitors, one monitor per household (16, 17). Air nicotine monitors were installed in the room in which the family congregated most frequently. To assess personal exposure to SHS, we collected hair samples from the nonsmoking primary female caregiver and one child younger than 11 y of age. Approximately 30 to 50 strands of hair were cut near the hair root from the back of the scalp. The hair samples for each participant were placed in separate, clean plastic bags for storage and transportation. Three centimeters of hair from the scalp were used to evaluate dose from exposure levels for the most recent months for each individual (18).

Laboratory Analysis. Laboratory analytical methods have been described in detail elsewhere (7, 19). Nicotine collected by each passive air sampler was extracted and analyzed by gas chromatography coupled with a nitrogen phosphorous detector. The limit of detection (LOD) was 0.001 $\mu g/m^3$ for a 7-d sample. Hair nicotine was extracted using an isotope dilution method (Nicotinine-d3, Supelco; ref. 19). The method was modified from that developed by Kintz (20). Hair nicotine analysis was conducted using gas chromatography/mass spectrometry (GC-17/MS-QP5000, Shimadzu) in SIM and splitless mode. Because we were only interested in the measurement of nicotine accumulated in hair by inhalation, systemic transport, and subsequent incorporation of nicotine into the growing hair, hair samples (30 mg), taken from within 3 cm of the hair root, were washed using 3 mL of dichloromethane and sonication (Aquasonic, Model 250HT) for 30 min to remove nicotine adhering to the surface of the hair, before nicotine extraction and analysis. The LOD was 0.02 ng/mg for a 30-mg hair sample. For both air nicotine and hair nicotine, final concentrations were provided after subtraction of background levels from blank samples.

Data Analysis. Descriptive analyses were conducted for demographic characteristics and smoking behaviors in the household. Measurement distributions were evaluated for normality using the Shapiro-Wilks test. Hair nicotine concentrations were log transformed for the analyses. Multilevel linear models (21, 22) were used to identify factors associated with differences in levels of log-transformed hair nicotine. Separate models were set up for women and children. To evaluate the association of hair nicotine concentrations with indoor air nicotine concentration after controlling for demographic and socioeconomic status factors, we included a continuous variable of age, an ordinal variable of women's education levels, and binary variable of women's employment status in our model. In addition, to take into account SHS exposure strength and time period simultaneously, we included continuous variables for indoor air nicotine concentration and total time spent at home. We also included the binary variable of hair treatment in the model to assess effect of hair treatments on hair nicotine concentration. Because few children had a chemical hair treatment, we excluded the variable of hair treatment in the children's model. For children, we calculated total time spent at home by subtracting time spent in schools, daycare centers, and transportation from 24 h. Because women's daily time activities were not accurately reported, we excluded the variable of time in the women's model.

Therefore, determinants of interest were indoor air nicotine levels at home, age, total time spent at home (children only), education level, employment status, and chemical hair treatment (women only). Equation 1 shows the multilevel model used for data analysis.

$$\text{Log } Y_{jk} = \beta_{0k} + \beta_1 X_{1jk} + \beta_2 X_{2jk} + \beta_3 X_{3jk} + \beta_4 X_{4jk} + \beta_5 X_{5jk} + \beta_6 X_{6jk} + \epsilon_{jk} - \qquad (Eq.1)$$

$$\beta_{0k} = \beta_0 + \delta_k$$

Y_{jk} is the log-transformed hair nicotine concentration for a study participant in a household (j = 1-40) in a country (k = 1-31). X_{1jk} to X_{6jk} represent household air nicotine, age, total time spent at home (children only), education level, employment status, and chemical hair treatment (women only), respectively. The errors ε_{jk} and δ_k represent within-country and between-country variance, respectively, of hair nicotine concentration, assuming a normal distribution with mean 0 and variances of σ^2 and τ^2. First, we specified a crude model without any adjustment to estimate the association between each determinant of interest and hair nicotine concentrations (model 1). Second, we included all explanatory independent variables as an ordinal or a binary scale of fixed effects (model 2). Third, we included the independent variables as continuous variables (model 3). The geometric mean ratios [95% confidence intervals (95% CI)] associated with relevant determinants were estimated in those multilevel models. Before conducting multilevel data analysis with model 3, to make the model intercept more meaningful, we centered the independent variables of indoor air nicotine concentration (0.16 $\mu g/m^3$), woman's or child's age (women, 34 y; children, 6 y), women's education level (middle school), and total time spent at home by children in each household (19 h) with respect to their median values. The statistical package SAS version 9 was used for statistical analyses.

Results

Smoking Characteristics of Households with Smokers and Demographic Characteristics of Women and Children

Smoking Characteristics. The median number of cigarettes smoked daily in households with smokers was 12 cigarettes/day [interquartile range (IQR) = 4-20]; the number was highest in European and Middle Eastern countries (20 cigarettes/day), followed by countries in Asia (10 cigarettes/day), and South America (8 cigarettes/day; Supplement Table S1). Median indoor air nicotine concentration for households in the 31 countries was 0.16 μg/m^3 (IQR = 0.03-0.82). The median air nicotine concentrations were highest among countries in Europe and the Middle East (0.58 μg/m^3, IQR = 0.09-1.96), followed by South America (0.18 μg/m^3, IQR = 0.04-0.77) and Asia (0.08 μg/m^3, IQR = 0.02-0.31). More detailed information on observed air nicotine by country is available in Wipfli et al. (7).

Demographic Characteristics for Women. The median age of nonsmoking women was 34.0 years (IQR = 28.0-40.0). About 48% of women had graduated from middle or elementary school or had no education, 63% were employed, and 32% had chemical hair treatments (Supplement Table S1). Age and employment status were similar across the three continents. Education levels were higher in Europe and the Middle East and more women in Latin America had chemically treated hair.

Demographic Characteristics for Children. Children's median age was 6.0 years (IQR = 3.0-8.0), and the age distribution patterns were similar across the three regions. Half of the children spent at least 19 hours per day in their houses (Supplement Table S1).

Observed Hair Nicotine Concentrations. Median hair nicotine concentrations for women and children living in households with smokers were 0.44 ng/mg (IQR = 0.15-1.13) and 0.80 ng/mg (IQR = 0.27-2.24), respectively (Table 1). Hair nicotine concentrations were higher in women and children from Asia, Europe, and the Middle East. Women and children exposed to higher air nicotine concentrations in the home had higher hair nicotine concentrations. The pooled correlation coefficient between air nicotine in the home and women's hair nicotine concentrations was 0.21 ($P < 0.001$), whereas that for children was 0.31 ($P < 0.001$).

Among women, hair nicotine concentrations were similar by age, education, and employment status and lower among women with chemical hair treatment. Even after stratifying by level of air nicotine concentration in the home (Fig. 1), the hair nicotine concentrations were not different by dichotomous variables mentioned above. Younger children who spent more time at home and children whose mother were unemployed had higher hair nicotine concentrations (Table 1). Those differences were

Table 1. Median and IQR of hair nicotine concentration in nonsmoking women and children living in households with smokers by region, air nicotine concentration at home, and socioeconomic characteristics

	Women			Children		
	n	Median (ng/mg; IQR)	*P*	*n*	Median (ng/mg; IQR)	*P*
Overall	852	0.44 (0.15-1.13)	—	1,017	0.80 (0.27-2.24)	—
% <LOD	—	28.0	—	—	6.7	—
Region						
Asia	418	0.53 (0.23-1.65)	<0.001	427	0.89 (0.32-2.51)	0.001
Latin America	215	0.26 (0.09-0.75)	—	263	0.54 (0.17-1.56)	—
Europe and Middle East	219	0.46 (0.14-1.03)	—	327	1.01 (0.27-2.95)	—
Air nicotine at home (μg/m^3)						
<0.01	132	0.31 (0.14-0.76)	<0.001	144	0.52 (0.16-1.40)	<0.001
0.01 to ≤0.1	252	0.33 (0.14-0.76)	—	288	0.49 (0.14-1.15)	—
0.1 to ≤1.0	297	0.55 (0.19-1.94)	—	357	1.05 (0.42-2.95)	—
1.0 to ≤10.0	163	0.66 (0.25-1.34)	—	212	1.61 (0.50-4.69)	—
>10.0	8	0.66 (0.39-4.22)	—	16	3.70 (0.70-8.26)	—
Children age (y)						
<6	—	—	—	490	1.21 (0.36-3.43)	<0.001
≥6	—	—	—	527	0.63 (0.22-1.63)	—
Total time spent at home (h/d)						
<19	—	—	—	452	0.65 (0.24-1.73)	<0.001
≥19	—	—	—	460	0.94 (0.28-3.18)	—
Women age (y)						
<34	451	0.44 (0.14-1.14)	0.48	—	—	—
≥34	401	0.44 (0.17-1.10)	—	—	—	—
Women education						
Less than high school	391	0.44 (0.17-1.20)	0.27	432	0.81 (0.28-2.29)	0.37
High school or more than high school	410	0.44 (0.14-1.13)	—	531	0.74 (0.24-2.03)	—
Women employment						
Unemployed	295	0.45 (0.14-1.31)	0.67	355	0.96 (0.25-3.50)	0.009
Employed	507	0.44 (0.16-1.12)	—	608	0.68 (0.26-1.81)	—
Women chemical hair treatment						
No	556	0.47 (0.15-1.30)	0.03	—	—	—
Yes	257	0.37 (0.15-0.91)	—	—	—	—

NOTE: *P* values: from tests for heterogeneity of hair nicotine concentrations by participant characteristics ("Region," "SHS level," "Number of cigarettes": The Kruskal Wallis test, Others: The Wilcoxon test).

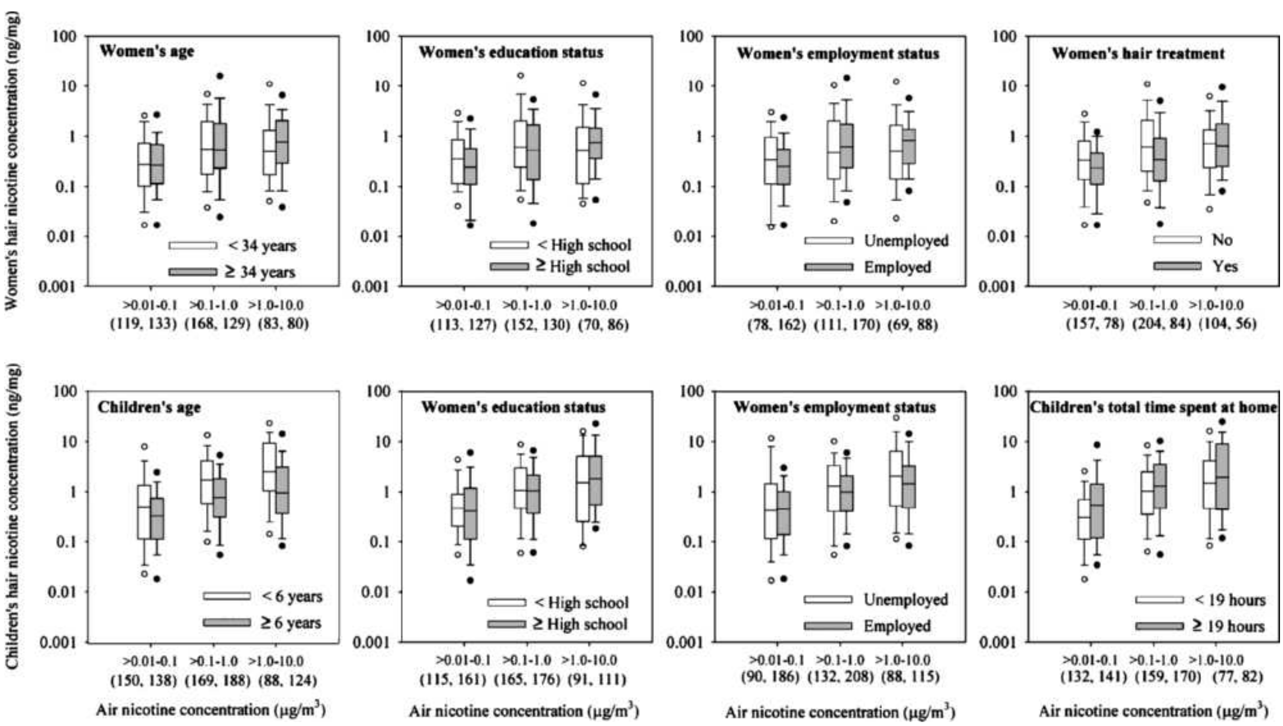

Figure 1. Distribution of hair nicotine concentrations by categories of air nicotine concentration at home and other variables. Number in parenthesis is sample size of each category. Ages, hours, and education status mentioned above are median values. Box plots show median as a center bar, 25th and 75th percentiles as a box, 10th and 90th percentile values as whiskers, and 5th and 95th percentile values as dots.

also observed after stratifying by air nicotine concentrations in the home (Fig. 1).

Determinants of Hair Nicotine Concentrations

Indoor Air. After adjustment for age, education, employment status, and chemical hair treatment, hair nicotine concentrations were 3.0 (95% CI, 1.2-7.6) times higher in women living in households with air nicotine concentrations of >10.0 $\mu g/m^3$ compared with <0.01 $\mu g/m^3$ (Table 2). Our model with air nicotine concentrations as a continuous variable, rather than as a five-level ordinal variable, showed that hair nicotine concentrations increased by 1% (95% CI, 0-3%) per 1 $\mu g/m^3$ increase in air nicotine concentration (Supplement Table S2).

Children's hair nicotine concentrations were 6.8 times (95% CI, 2.9-15.6) higher in households with air nicotine concentrations of >10.0 $\mu g/m^3$ compared with <0.01 $\mu g/m^3$ after controlling for age and total time spent at home (Table 2). A model with air nicotine concentration as a continuous variable estimated that a 1 $\mu g/m^3$ increase in air nicotine concentration was associated with a 3% (95% CI, 2-5%) increase in hair nicotine concentration among children (Supplement Table S3).

Age and Total Time Spent at Home. Among children who spent <19 hours a day at home, those who were <6 years of age had 12% (95% CI, 5-20%) higher hair nicotine concentration per 1 $\mu g/m^3$ increase of air nicotine concentration compared with those who were older (Table 3). In addition, among children ages >6 years, mean hair nicotine concentration for children who spent >19 hours at home were 15% (95% CI, 7-23%) higher than for children who spent <19 hours at home. These results showed that the association of children's indoor air nicotine exposure with hair nicotine concentrations was modified by children's age and length of exposure.

Women's hair nicotine concentration increased by a factor of 1% (95% CI, 1-2%) with a unit increase of women's age centered with respect to the median (34 years) for unemployed women who graduated high school, had untreated hair, and exposed to 0.16 $\mu g/m^3$ air nicotine concentration at home (Supplement Table S2). No significant effect modification (P = 0.23) was observed for the association between women's hair nicotine concentration and air nicotine concentration at home by women's age dichotomized at 34 years.

Women's Education and Employment Status. Hair nicotine concentrations decreased by a factor of 0.9 (95% CI, 0.8-1.0) per unit increase of education level after controlling for indoor air nicotine concentrations, women's age, employment status, and hair treatment status (Supplement Table S2). Employed women showed similar hair nicotine levels to unemployed women by a factor of 1.0 (95% CI, 0.8-1.2) after adjusting for indoor air nicotine concentrations, age, education status, and hair treatment, although this was not statistically significant (Supplement Table S2). There was no statistically significant interaction by women's educational status (less than high school versus high school or more than high school) or employment status (no versus yes) for the association of SHS level at home with hair nicotine concentration in women. In addition, this association in children was not modified by the mother's education or employment status.

Table 2. Geometric means and geometric mean ratios of hair nicotine concentration in nonsmoking women and children by air nicotine concentration at home

	Women* (n = 813)				Children† (n = 906)			
	Crude (model 1)		Adjusted* (model 2)		Crude (model 1)		Adjusted† (model 2)	
	Hair nicotine (ng/mg), GM	GMR (95% CI)	Hair nicotine (ng/mg), GM	GMR (95% CI)	Hair nicotine (ng/mg), GM	GMR (95% CI)	Hair nicotine (ng/mg), GM	GMR (95% CI)
Nicotine concentration (μg/m³)								
<0.01	0.27‡	1.00 (Reference)	0.32‡	1.00 (Reference)	0.39‡	1.00 (Reference)	0.44‡	1.00 (Reference)
0.01 to ≤0.1	0.28	1.01 (0.75 1.36)	0.30	0.95 (0.70-1.31)	0.41	1.05 (0.79-1.41)	0.49	1.10 (0.82-1.48)
0.1 to ≤1.0	0.49‡	1.80 (1.34-2.42)	0.52‡	1.65 (1.21-2.25)	0.87‡	2.22 (1.67-2.97)	1.00‡	2.25 (1.67-3.01)
1.0 to ≤10.0	0.68‡	2.49 (1.77-3.54)	0.75‡	2.37 (1.66-3.39)	1.49‡	3.81 (2.74-5.30)	1.75‡	3.93 (2.77-5.57)
>10.0	0.90§	3.29 (1.27-8.54)	0.94§	2.99 (1.18-7.57)	3.04‡	7.78 (3.80-15.9)	3.00‡	6.75 (2.92-15.58)

Abbreviations: GM, geometric mean; GMR, geometric mean ratio.
*Adjusted for age, education status, employment status, and chemical hair treatment. Age: binary (0, <34; 1, ≥34 y); education status: binary (0, less than high school; 1, high school or more than high school); employment status: binary (0, no; 1, yes]; hair treatment: binary (0, no; 1, yes).
†Adjusted for age and total time spent at home. Age: binary (0, <6; 1, ≥6 y]; total time spent at home: binary (0, <19 h; 1, ≥19 h).
‡P < 0.01.
§0.05 < P ≤ 0.01.

Women's Hair Treatments. Based on the modeling results (Supplement Table S2), we estimated that women with chemically treated hair had 0.9 (95% CI, 0.7-1.2) times lower hair nicotine concentration than women without hair treatment after adjusting for indoor air nicotine concentration, age, education, and employment status; although not statistically significant, this finding indicates that hair nicotine concentration might be slightly underestimated among women with hair chemical treatment.

Discussion

Observed Hair Nicotine Concentrations. For several decades, hair nicotine concentration has been measured as a biomarker of tobacco smoke exposure with the advantage of noninvasive sampling and ease of sample transfer and storage compared with sampling of blood, urine, or saliva. Previous studies have described hair nicotine concentrations for nonsmoking women, children, or infants (23-31). The median hair nicotine concentration (0.44 ng/mg) for women in our study was ~4.4 times lower than the average of 2.0 ng/mg measured in 70 nonsmoking women in Finnish households with smokers (26). The median hair nicotine concentration for children in the current study (0.8 ng/mg) was approximately five times lower than that of children with SHS exposure at home (4.3 ng/mg, n = 196) in New Zealand (23). However, one (26) of the prior studies did not include a hair-washing step in their article. Therefore, our method, which removed nicotine adhering to the surface of the hair before hair nicotine analysis, would be expected to provide a lower concentration that reflects internal dose only. However, a lack of quantitative SHS exposure information in the Finnish and New Zealand studies limits further exploration of the basis for the differences in hair nicotine concentrations between our study and these studies.

The majority of prior studies on SHS exposure used indirect exposure parameters, including number of cigarettes or number of smokers, rather than direct measurement of air nicotine or other chemicals as markers reflecting tobacco smoke exposure levels. Thus, comparison of the magnitude of association between air and hair nicotine concentrations obtained from our study to associations in other studies is substantially limited. As a second possible approach, we compared our correlation coefficients (r = 0.21 for women, 0.31 for children) between the two measurements, i.e., air and hair nicotine, to those obtained from a similar study reporting the association of chamber air nicotine concentrations with serum cotinine among 40 adults who were exposed over 2 hours in a chamber (32). The correlation coefficient (r = 0.44, n = 40) was comparable with that of our study, although the median air nicotine concentrations in our study were ~300 times lower than in the experimental study.

Determinants for Hair Nicotine Concentrations Among Women and Children

Indoor Air Nicotine. In our study, women's and children's hair nicotine concentrations showed exposure-dose relationships such that there were estimated at 1% (95% CI, 1-3%) and 3% (95% CI, 1-4%) per 1 μg/m³ increase of air nicotine concentration, respectively. The strong positive association between air nicotine concentration and hair nicotine concentration was not changed after

Table 3. Geometric means and geometric mean ratios of children's hair nicotine concentration by age and total time spent at home

	Hair nicotine (ng/mg), GM (95% CI)	*P*	GMR (95% CI)
Per 1 μg/m^3 increase in air nicotine at home			
Children ≥6 y, who spent <19 h/d (*n* = 311)	1.01 (1.00, 1.03)	0.0014	1.00 (Reference)
Children <6 y, who spent <19 h/d (*n* = 135)	1.14 (1.06, 1.23)	<0.001	1.12 (1.05, 1.20)
Children ≥6 y, who spent ≥19 h/d (*n* = 151)	1.16 (1.09, 1.24)	<0.001	1.15 (1.07, 1.23)
Children <6 y, who spent ≥19 h/d (*n* = 309)	1.31 (1.20, 1.42)	<0.001	1.29 (1.12, 1.48)
At centered median SHS exposure level of 0.16 μg/m^3			
Children ≥6 y, who spent <19 h/d at home	0.46 (0.32, 0.68)	<0.001	1.00 (Reference)
Children <6 y, who spent <19 h/d at home	0.68 (0.46, 1.02)	0.07	1.47 (1.19, 1.82)
Children ≥6 y, who spent ≥19 h/d at home	0.53 (0.35, 0.79)	0.008	1.14 (0.90, 1.43)
Children <6 y, who spent ≥19 h/d at home	0.78 (0.53, 1.13)	0.27	1.67 (1.15, 2.06)

controlling for age, total time spent at home, and socioeconomic characteristics. Among women without chemical hair treatment (*n* = 556), the same positive association of hair nicotine concentration with air nicotine concentration was also observed (data not shown). The use of air nicotine concentration at home as an overall indicator for SHS exposure has been well described (7, 33-35). Because components of SHS can settle on and be absorbed by carpets, walls, and furnishings, and then be re-emitted weeks or months later, susceptible populations, especially young children, may absorb nicotine by inhalation, ingestion, or skin contact after smoking has occurred at a room (10). This can occur even if a child did not spend time in the room with a smoker (10).

Age and Total Time Spent at Home. Even after adjusting for indoor air nicotine concentrations and total time spent at home, there was an inverse association between children's age and hair nicotine concentrations, which is consistent with findings by Groner et al. (36). In that study, the hair nicotine level in the children ages 2 to 5 years was approximately six times higher than in older children, ages 9 to 14 years, at the same levels of parental smoking. In addition, Coultas et al. (37) found that the median of saliva cotinine concentrations among nonsmoking children ages <5 years was about twice higher, compared with that among older children ages 6 to 12 years when there was one cigarette smoker in the home. Coultas et al. (37) also reported that presence and the concentration of saliva cotinine were affected by all household smokers at home. Such body burden differences between older children and younger children under similar external environmental exposures may be due to relatively closer proximity of younger children to smokers and/or a higher inhalation rate in younger children per body mass compared with older children (11, 37). Children's hair nicotine concentrations showed a positive association with total time spent at home, indicating that higher hair nicotine levels may reflect the time-activity patterns among young children (37, 38).

Women's Education Levels and Employment Status. Previous studies on the association between education levels and SHS exposure levels have had inconsistent findings. Simoni et al. (39) found that educated Italian women had a higher risk of exposure to SHS than less educated Italian women, as more educated women work in occupational settings where other persons smoke. However, Twose et al. (13) reported that more educated women had a lower risk of exposure to SHS than less educated women in Spain. In our study, women with lower education levels or being unemployed had higher hair nicotine concentrations. More research is needed to evaluate the associations between a person's exposure level and education and/or employment status. The research should include a country-by-country comparison, as laws governing occupational smoking vary among countries.

Women's Hair Treatments. Chemical hair treatments were associated with reduction in hair nicotine levels by a factor of 0.9 (95% CI, 0.7-1.2) after controlling for SHS exposure levels, age, and other sociodemographic characteristics of nonsmoking women (Supplement Table S2). This finding was comparable with the experimental result of Jurado et al. (40), who reported an average decrease of 30% in hair nicotine concentrations after bleaching naturally colored hair samples among eight French smokers. Pichini et al. (41) found that hair nicotine concentrations were affected by treatment (dyes, permanent wave, and hydrogen peroxide), but the results were not reproducible. On the basis of this study and other experimental results in earlier articles, it is likely that women's hair nicotine concentrations in this study might be slightly underestimated.

One strength of this study lies in the use of a standard protocol for a large study population from multiple countries with diverse cultures and climates. However, this study was limited by the small sample size per country, collection of samples on a convenience basis due to sampling feasibility as well as resources availability, and the lack of information on gender among children, and hair growth rate per ethnic group. Since the amount of nicotine extracted from hair actually reflects the total amount of nicotine accumulated by a participant's exposure to multiple sources—i.e., not only in the home but in other indoor and outdoor locations—indoor air nicotine might under-represent a participant's true total exposure level. Therefore, if a future study measures air nicotine levels by personal exposure monitoring with time-activity records, rather than home indoor air nicotine concentrations alone, the association with biomarker levels may be stronger than found in our study. Nonetheless, this study showed that the home remains an important determinant of SHS exposure among nonsmoking women and children. Also, to further validate hair nicotine as a biomarker of tobacco smoke, it would be useful to evaluate the association of hair nicotine with cotinine concentration in saliva, serum, or urine. Additionally, further data are needed on the consistency of hair nicotine levels over time.

Conclusions

This study showed a positive association between SHS exposure and hair nicotine concentrations in a setting outside of the laboratory and showed that home exposure to SHS was an important determinant of hair nicotine among nonsmoking women and children from 31 countries around the world. Under global efforts to reduce the burden of tobacco use led by the Framework Convention on Tobacco Control, a large number of countries have implemented smoking bans in public places including workplaces, restaurants, schools, and hospitals (42, 43). Private microenvironments, such as homes, cars, and child care in private residences, are outside the jurisdiction of imposed smoking bans, although banning smoking on private property may be preferred by the owners of homes or cars (10, 44). As SHS in the home can spread quickly throughout the closed microenvironment (35), parents should implement complete home-smoking bans to make the environment around children smoke free (45). This analysis, along with previous evidence (4, 7), provides adequate evidence to support educational and policy initiatives aimed at encouraging home smoking bans and reducing exposure to SHS, especially among young children.

Disclosure of Potential Conflicts of Interest

No potential conflicts of interest were disclosed.

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