Brief Report

Secondhand Smoke Exposure and Hair Nicotine in Children: Age-Dependent Differences

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Abstract

Introduction: A significant proportion of children in the United States remain exposed to secondhand smoke (SHS). We are reporting on relationships observed between parental report of their child's SHS exposure in two groups of children (ages 2–5 years and 9–14 years) with a biological marker of long-term SHS exposure, hair nicotine.

Methods: Participants were healthy children recruited via convenience sampling for two age groups: 2–5 years and 9–14 years. The presence and amount of SHS exposure were assessed by both questionnaire and hair sampling for nicotine determination.

Results: A total of 115 participants were recruited (54 toddlers and 61 youth). The groups were similar in terms of demographics and reported SHS exposure. Hair nicotine levels were significantly different by age group, with toddlers having higher levels than youth. The most important independent determinants of hair nicotine were toddler age group, receiving Medicaid for health insurance, and number of smokers the subject was exposed to in 24 hr.

Conclusions: Our findings suggest that young children who are insured by Medicaid have higher levels of hair nicotine, a biomarker of SHS exposure, when compared with an older age group. Further efforts to protect this vulnerable population and mitigate their lifetime risks of SHS exposure–related morbidities are warranted.

Introduction

Despite an overall decrease in the rates of smoking, a significant proportion of children in the United States remain exposed to secondhand smoke (SHS). Like active smoking, SHS remains

a class-based health risk since exposure among low-income children in urban areas ranges from 30% to 79% (Cornelius, Goldschmidt, & Dempsey, 2003) and varies inversely with family income and parental education (Marano, Schober, Brody, & Zhang, 2009; Soliman, Pollack, & Warner, 2004). While there have been general trends in the United States in reduced tobacco use over the past decade, smoking among adults from lower socioeconomic groups remains prevalent and has changed less than other groups (CDC, 2010). A group of vulnerable children persists, despite national trends of reduced smoking prevalence in adults (Singh, Siahpush, & Kogan, 2010). Therefore, SHS-related morbidities in children could be considered a health risk disparity.

We report on relationships observed between parental report of their child's SHS exposure with a biological marker of long-term SHS exposure (hair nicotine levels) in two age groups of children (ages 2–5 or 9–14 years) from low-income families. This datum was obtained as part of a larger study on SHS exposure and markers of cardiovascular risk in children. These two age groups were of interest because of the high SHS exposure of the younger children based on our previous work (Groner et al., 2004) and the ability of the older group to cooperate with additional cardiovascular testing as part of the larger study.

Methods

Recruitment

Participants were recruited via convenience sampling through recruiting in Nationwide Children's Hospital (NCH, Columbus, OH) Primary Care Network and via advertising in the NCH internal hospital E-mail system. The Network serves low-income, urban children in Columbus, OH. The inclusion criteria were healthy children in two age groups (between 2 and 5 years and between 9 and 14 years), both exposed and unexposed to

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SHS by parental report. The exclusion criteria were presence of one or more of the following: active smoker (referring to child or teen), acute febrile illness or other active infections, congenital heart disease, diabetes (Type 1 or 2) (elevated fasting glucose [>100 mg/dl]), concurrent daily anti-inflammatory prescription or nonprescription medications, and not having enough hair for hair sampling. The project was approved by the NCH IRB; informed consent was obtained by parents and written assent by youth over 9 years old.

SHS Exposure Assessment

The presence and extent of SHS exposure were assessed by both by questionnaire and by hair sampling for nicotine determination. A 'smoker' was defined as an individual who has smoked at least 1 cigarette/day during the previous 7 days. Exposure to SHS was defined as living in a home with a smoker regardless of whether the smoker claimed "indoor or outdoor" smoking or exposure to one or more smokers in 24 hr. The following questions were used to assess smoke exposure: (a) presence of a smoker in the child's home (yes/no), (b) number of people living in the home who smoke (numeric value), (c) maternal smoking (yes/no), (d) number of people the child comes in contact with in 24 hr who smoke (numeric value) and (e) presence of an in-home smoking ban (smoking *never* allowed in the home considered a 'smoking ban').

Hair Nicotine

Hair nicotine was used as a biological marker of SHS exposure. This measure provides a long-term (months) evaluation of smoke exposure since the nicotine in the bloodstream of hair follicle capillaries is incorporated in the growing hair shaft (Al-Delaimy, 2002; Al-Delaimy, Crane, & Woodward, 2000). The half-life of nicotine in body fluids is approximately 2–3 hr and that of cotinine (a major nicotine metabolite) is 1–2 days (Benowitz, 1996). Thus, hair nicotine provides an extended exposure assessment when compared with blood, urinary, or salivary concentrations of nicotine or cotinine. Hair nicotine as a SHS exposure assessment has added advantages for the study of young children because hair samples are easy and noninvasive to obtain, store, and transport, and parental consent is easier to obtain than other approaches. This measure has also been shown to linearly correlate to numbers of cigarettes smoked in active adult smokers (Kintz, Ludes, & Mangin, 1992; Mizuno, Uematsu, Oshima, Nakamura, & Nakashima, 1993) and has been used in recent global epidemiological studies of children's tobacco exposure (Wipfli et al., 2008).

Approximately 20–40 hair shafts 2–3 cm in length were cut at the root at the occipital area, stored, and later sent for assay at established contract research facility (Specialist Biochemistry Laboratory, Wellington Hospital, Wellington, New Zealand). This assay involves washing the hair sample prior to analysis and therefore measures inhaled nicotine and not ambient nicotine adhered to hair (Mahoney & Al-Delaimy, 2001). The method is reversed-phase high-performance liquid chromatography with electrochemical detection as described previously (Mahoney & Al-Delaimy, 2001). All samples were run in duplicates; values ≥ 100 ng/mg were redetermined to confirm values in that range. Hair nicotine level is expressed as nanogram per milligram of hair, and sensitivity limit was 0.01 ng/mg hair when 2 mg of hair is used.

Statistical Analyses

All analyses were performed using Stata Version 10 (StataCorp, College Station, TX). For comparing age cohorts, χ^2 tests were used for categorical and dichotomous variables, and *t*-tests were used for continuous measures. Because hair nicotine values were not normally distributed, log hair nicotine was used in descriptive comparisons and in regression analyses. Ordinary least squares regression was employed to assess the effects of age and other exposure variables on actual measure of hair nicotine concentrations (ng/mg, log transformed), with adjusted SEs for clustering within a family.

Results

Shown in Table 1 are general descriptions of the subjects. A total of 115 were recruited in toddler (2–5 years) or youth (9–14) groups. Gender, racial distributions, and the proportion of children receiving Medicaid or having a very low family income (income $< \$20,000$ per year) were not different between groups. Parental report of child SHS exposure was similar in both age groups of children (Table 1).

Of the 115 children studied, all but two had measurable levels of hair nicotine incorporated in hair. Nicotine levels ranged from 0.11 to 253 ng nicotine/mg hair across both age groups. Within the toddler age group, the range was extremely large (0.30–254 ng/mg; median 1.90 ng/mg) and within the youth age group, the range was much smaller (0.11–5.2 ng/mg, median 0.48 ng/mg). The medians (toddlers 1.90 ng/mg vs. youth 0.48 ng/mg, $p < .01$) and geometric means (toddlers 0.87 ± 1.64 vs. youth 0.32 ± 1.29 ,

Table 1. Description of Sample, N (%)

	Toddlers, age 2–5 ($n = 54$)	Youth, age 9–14 ($n = 61$)	<i>p</i>
Female	29 (54)	31 (51)	.703
Family income $\leq 20,000$ per year, %	37	43	.359
Medicaid, %	74	64	.709
Race			
White	23 (43)	27 (44)	.644
Black	22 (41)	24 (39)	.772
Other	9 (17)	8 (16)	.382
Age in years (mean [SD])	3.46 ± 1.18	11.34 ± 1.82	.000
Child lives with one or more smokers	62	52	.519
No. of smokers living in home			
0	20 (37)	29 (47.5)	.524
1	18 (33)	17 (28)	
2+	16 (30)	15 (24.5)	
No. of smokers exposed to in 24 hr			
0	16 (30)	31 (51)	.070
1	13 (24)	10 (16)	
2+	25 (46)	20 (33)	
Maternal smoking (yes)	24 (44)	22 (37)	.439
Smoking ban present	30 (56)	38 (62)	.463

mean $\pm$ SD, $p < .01$) were significantly different between age groups. Geometric mean hair nicotine levels were also significantly higher for toddlers in homes with maternal smoking, living with 2 or more smokers, and in homes without a smoking ban when compared with youth with the same exposure (Supplementary Figure).

We used multivariate regression analysis to evaluate the relationships between hair nicotine and reported SHS exposure and to control for covariation of report measures and hair nicotine levels clustering within families. This analysis (Table 2) shows several models. Age (toddler vs. youth) and receiving Medicaid were independently associated with hair nicotine. Maternal smoking (Model 1), not having a smoking ban (Model 2), and number of smokers the subject was exposed to in 24 hr (Model 3) were all associated with hair nicotine in separate models. Because of the high correlation of these three variables, all were first entered into the model individually. Model 4 shows the results when all variables are entered simultaneously and explains the most variance in hair nicotine ($R^2 = .47$). While each of the smoking status variables independently predicts hair nicotine values, when entered together, the only variable which remains significant is the number of smokers exposed to in 24 hr. (When 'number of smokers in the home' was entered into the model, none of the SHS exposure variables remain significant; age and Medicaid status did remain significant [data not shown]). Standardized β results indicate that age group (toddler vs. youth) was the strongest predictor of hair nicotine ($-.30$), followed closely by Medicaid status ($.29$), and then number of smokers exposed to in 24 hr ($.22$).

Discussion

In our study, toddlers had significantly higher hair nicotine content than older children, which is consistent with a large international study of SHS exposure wherein younger children had consistently higher hair nicotine levels *even after adjusting for the amount of time spent in the home* (Kim et al., 2009). Age appears to play a role in other biomarkers of SHS. We previously found that hair cotinine levels of infants were as high as their actively smoking mothers and higher than nonsmoking mothers (Groner et al., 2004). Saliva cotinine has also been reported to be higher in younger than older children (Delpisheh,

Kelly, & Brabin, 2006) and National Health and Nutrition Examination Survey III data (Wilkinson, Arheart, & Lee, 2006) similarly show an inverse relationship between age and serum cotinine. 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanol, a carcinogen specific to SHS, has been reported to be two to four times higher in the urine of infants and children than in adults (Hecht et al., 2006; Stepanov, Hecht, Duca, & Mardari, 2006).

Our observation that toddlers have greater levels of an SHS exposure biomarker than older youths is consistent with other research, but the mechanisms involved are not clear. Potential causes that do not reflect true exposure differences could include faster deposition of circulating nicotine into the hair shaft or slower nicotine clearance from blood. Alternatively, younger children spend more hours in the home in the presence of a smoker, which would reflect true increased health risk. The presence of 'third-hand' smoke exposure (Winickoff et al., 2009) wherein there is a contact with surfaces that have nicotine dust due to contamination from the smoker may also contribute. This contamination has been shown to be significant, even with smokers claiming to smoke outside the home (Matt et al., 2004) and since young children have more frequently "mouthing" behavior than older children, their increased hair nicotine levels could be due to this ingestion of nicotine contaminating household surfaces. The increased respiratory rate of younger children (e.g., greater minute ventilation per kilogram body weight) may be an important physiological basis; steady-state plasma cotinine is directly related to minute ventilation (Benowitz, 1996) and therefore one can infer that since cotinine is the main metabolite of nicotine, systemic nicotine absorption itself is directly related to minute ventilation. Since hair nicotine is a reflection of inhaled nicotine, it is therefore reasonable that the discrepancies we have observed between toddlers and youth may be a true reflection of inhaled SHS.

A high proportion of the sample had detectable hair nicotine (98%), while only 59% had a reported exposure to a smoker in 24 hr. This discrepancy may be due to underreporting of SHS exposure due to social desirability in the context of being both recruited and studied at a health facility. This discrepancy may also be related to unknown exposure of children who live in multiunit housing (Wilson, Klein, Blumkin, Gottlieb, & Winickoff, 2011) where there may be potential for seepage of SHS through open doors and shared ventilation systems. Hair nicotine levels

Table 2. Ordinary Least Squares Regression Analysis on Logged Hair Nicotine

	Model 1	Model 2	Model 3	Model 4	β
	b	b	b	b	
Older age (youth vs. toddler)	-1.02***	-1.01***	-1.00***	-0.97***	-.30
Medicaid	1.11***	1.24***	1.09***	0.96***	.29
No maternal smoking	-1.01**			-0.66	-.21
Smoking ban		-0.80*		-0.17	-.05
Number of smokers in 24 hr			0.27***	0.18*	.22
R^2	.45	.41	.44	.47	
N	115	115	115	115	

Note. All SEs are adjusted for clustering within families. Model 1: age group, insured by Medicaid, and maternal smoking status. Model 2: age group, insured by Medicaid, and smoking ban. Model 3: Age group, insured by Medicaid, and number of smokers in 24 hr. Model 4: age group, insured by Medicaid, maternal smoking status, smoking ban, and number of smokers in 24 hr.

* $p < .05$. ** $p < .01$. *** $p < .001$.

found in our convenience sample were comparable to those found by Kim et al. (2009) in a large global epidemiological study. It is interesting to note that almost one third of the toddlers in our study had hair nicotine levels that were approximately as high as adult active smokers reported in other investigations (Kintz et al., 1992).

The strengths of the study are its focus on a highly exposed, low-income group of children in two age groups and the use of a long-term biological measure of SHS exposure. There are several limitations—our subjects were recruited via convenience sample, and we excluded children on chronic anti-inflammatory medication. We did not gather information on paternal smoking, multi-unit housing, on reported number of cigarettes per day of exposure and car exposure, and we did not use an indoor air nicotine monitor or a passive diffusion monitor to measure SHS exposure.

As general societal trends show declines in smoking, there are clearly some subgroups where smoking prevalence persists, and children within these subgroups (such as those from lower socioeconomic groups) are thus at higher risk of SHS exposure. It is clear from our observations that tobacco exposure early in life is a health risk disparity; Medicaid status was a significant independent determinant of child hair nicotine. This may be due to poor ventilation and small room size among families of children receiving Medicaid compared with other children. Our age-dependent findings are consistent with findings using other biomarkers. Regardless of the mechanism, these results raise concern for the long-term health effects for children with early significant SHS exposure and reinforce efforts to eliminate all such exposure during childhood.

Supplementary Material

Supplementary Figure can be found online at <http://ntr.oxfordjournals.org/content/suppl/2012/03/07/ntr269.DC1>

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Declaration of Interests

None of the authors had any conflicts of interest, and there was no corporate sponsorship of this research.

References

- Al-Delaimy, W. K. (2002). Hair as a biomarker for exposure to tobacco smoke. *Tobacco Control*, 11, 176–182. doi: 10.1136/tc.11.3.176
- Al-Delaimy, W. K., Crane, J., & Woodward, A. (2000). Questionnaire and hair measurement of exposure to tobacco smoke. *Journal of Exposure Analysis and Environmental Epidemiology*, 10, 378–384.
- Benowitz, N. L. (1996). Cotinine as a biomarker of environmental tobacco smoke exposure. *Epidemiologic Reviews*, 18, 188–204. Retrieved from <http://epirev.oxfordjournals.org/content/18/2/188.long>
- CDC. (2010). Vital signs: Current cigarette smoking among adults—United States, 2009. *Morbidity & Mortality Weekly Report*, 59, 1135. Retrieved from <http://cdc.gov/mmwr/preview/mmwrhtml/mm5935a3.htm>
- Cornelius, M. D., Goldschmidt, L., & Dempsey, D. A. (2003). Environmental tobacco smoke exposure in low-income 6-year-olds: Parent report and urine cotinine measures. *Nicotine & Tobacco Research*, 5, 333–339. doi: 10.1080/14622200307208
- Delpisheh, A., Kelly, Y., & Brabin, B. J. (2006). Passive cigarette smoke exposure in primary school children in Liverpool. *Public Health*, 120, 65–69. doi: 10.1016/j.puhe.2005.05.003
- Groner, J., Wadwa, P., Hoshaw-Woodard, S., Hayes, J., Klein, J., Koren, G., et al. (2004). Active and passive tobacco smoke exposure: A comparison of maternal and child hair cotinine levels. *Nicotine & Tobacco Research*, 6, 789–795. doi: 10.1080/1462220042000274257
- Hecht, S. S., Carmella, S. G., Le, K.-A., Murphy, S. E., Boettcher, A. J., Le, C., et al. (2006). 4-(Methylnitrosamino)-1-(3-Pyridyl)-1-Butanol and its glucuronides in the urine of infants exposed to environmental tobacco smoke. *Cancer Epidemiology Biomarkers & Prevention*, 15, 988–992. doi: 10.1158/1055-9965.epi-05-0596
- Kim, S., Wipfli, H., Navas-Acien, A., Dominici, F., Avila-Tang, E., Onicescu, G., et al. (2009). Determinants of hair nicotine concentrations in nonsmoking women and children: A multicountry study of secondhand smoke exposure in homes. *Cancer Epidemiology Biomarkers & Prevention*, 18, 3407–3414. doi: 10.1158/1055-9965.epi-09-0337
- Kintz, P., Ludes, B., & Mangin, P. (1992). Evaluation of nicotine and cotinine in human hair. *Journal of Forensic Sciences*, 37, 72–76. doi: 10.1520/jfs13212j
- Mahoney, G. N., & Al-Delaimy, W. (2001). Measurement of nicotine in hair by reversed-phase high-performance liquid chromatography with electrochemical detection. *Journal of Chromatography B: Biomedical Sciences and Applications*, 753, 179–187. doi: 10.1016/S0378-4347(00)00540-5
- Marano, C., Schober, S. E., Brody, D. J., & Zhang, C. (2009). Secondhand tobacco smoke exposure among children and adolescents: United States, 2003–2006. *Pediatrics*, 124, 1299–1305. doi: 10.1542/peds.2009-0880
- Matt, G. E., Quintana, P. J. E., Hovell, M. F., Bernert, J. T., Song, S., Novianti, N., et al. (2004). Households contaminated by environmental tobacco smoke: Sources of infant exposures. *Tobacco Control*, 13, 29–37. doi: 10.1136/tc.2003.003889
- Mizuno, A., Uematsu, T., Oshima, A., Nakamura, M., & Nakashima, M. (1993). Analysis of nicotine content of hair for

assessing individual cigarette-smoking behavior. *Therapeutic Drug Monitoring*, 15, 99.

Singh, G. K., Siahpush, M., & Kogan, M. D. (2010). Disparities in children's exposure to environmental tobacco smoke in the United States, 2007. *Pediatrics*, 126, 4–13. doi: 10.1542/peds.2009-2744

Soliman, S., Pollack, H. A., & Warner, K. E. (2004). Decrease in the prevalence of environmental tobacco smoke exposure in the home during the 1990s in families with children. *American Journal of Public Health*, 94, 314–320. doi: 10.2105/ajph.94.2.314

Stepanov, I., Hecht, S. S., Duca, G., & Mardari, I. (2006). Uptake of the tobacco-specific lung carcinogen 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone by Moldovan children. *Cancer Epidemiology Biomarkers & Prevention*, 15, 7–11. doi: 10.1158/1055-9965.epi-05-0293

Wilkinson, J. D., Arheart, K. L., & Lee, D. J. (2006). Accuracy of parental reporting of secondhand smoke exposure: The National Health and Nutrition Examination Survey III. *Nicotine & Tobacco Research*, 8, 591–597. doi: 10.1080/14622200600790021

Wilson, K. M., Klein, J. D., Blumkin, A. K., Gottlieb, M., & Winickoff, J. P. (2011). Tobacco-smoke exposure in children who live in multiunit housing. *Pediatrics*, 127, 85–92. doi: 10.1542/peds.2010-2046

Winickoff, J. P., Friebely, J., Tanski, S. E., Sherrod, C., Matt, G. E., Hovell, M. F., et al. (2009). Beliefs about the health effects of “thirdhand” smoke and home smoking bans. *Pediatrics*, 123, e74–e79. doi: 10.1542/peds.2008-2184

Wipfli, H., Avila-Tang, E., Navas-Acien, A., Kim, S., Onicescu, G., Yuan, J., et al. (2008). Secondhand smoke exposure among women and children: Evidence from 31 countries. *American Journal of Public Health*, 98, 672–679. doi: 10.2105/ajph.2007.126631