

UC Irvine

UC Irvine Previously Published Works

Title

Magnitude and Chronicity of Environmental Smoke Exposure Across Infancy and Early Childhood in a Sample of Low-Income Children.

Permalink

<https://escholarship.org/uc/item/2hn954k8>

Journal

Nicotine and Tobacco Research, 21(12)

Authors

Gatzke-Kopp, Lisa
Willoughby, Michael
Warkentien, Siri
[et al.](#)

Publication Date

2019-11-19

DOI

10.1093/ntr/nty228

Peer reviewed



Original investigation

Magnitude and Chronicity of Environmental Smoke Exposure Across Infancy and Early Childhood in a Sample of Low-Income Children

Lisa M. Gatzke-Kopp PhD^{1,○}, Michael T. Willoughby PhD²,
Siri M. Warkentien PhD², Thomas O'Connor PhD³,
Douglas A. Granger PhD^{4,5,6,7}, Clancy Blair PhD⁸

¹Department of Human Development and Family Studies, Pennsylvania State University, University Park, PA; ²Education and Workforce Development, RTI International; Research Triangle Park, NC; ³Department of Psychiatry, University of Rochester, Rochester, NY; ⁴Department of Psychological Science, Institute for Interdisciplinary Salivary Bioscience, University of California, Irvine, CA; ⁵Department of Acute and Chronic Care, Johns Hopkins University School of Nursing, Baltimore, MD; ⁶Department of Pediatrics, Johns Hopkins University School of Medicine, Baltimore, MD; ⁷Department of Population, Family and Reproductive Health, Johns Hopkins University Bloomberg School of Public Health, Baltimore, MD; ⁸Department of Applied Psychology, New York University, New York, NY

Corresponding Author: Lisa M. Gatzke-Kopp, PhD, Department of Human Development and Family Studies, Pennsylvania State University, 119 HDD, University Park, PA 16802, USA. Telephone: 814-867-2371, E-mail: lmk18@psu.edu

Abstract

Introduction: Infants and young children may be at an increased risk for second- and thirdhand exposure to tobacco smoke because of increased respiration rate and exposure to surface residue. However, relatively fewer studies have examined biomarkers of exposure (cotinine) in children under age 4 years. This study examines the magnitude and chronicity of exposure across early childhood among children from low-income families in order to better characterize contextual risk factors associated with exposure.

Methods: A total of 1292 families were recruited in six nonurban counties of Pennsylvania and North Carolina. Cotinine was assayed from infant saliva at 6, 15, 24, and 48 months of age ($N = 1218$), and categorized as low (≤ 0.45 ng/mL), moderate (0.46–12 ng/mL), or high (≥ 12 ng/mL) at each time point. Categories were highly correlated across time. Latent class analysis was used to summarize patterns of exposure categories across time.

Results: Magnitude of exposure in this sample was high, with approximately 12% of infants registering cotinine values at least 12 ng/mL, consistent with active smoking in adults. Greater exposure was associated with lower income, less education, more residential instability, and more instability in adult occupants in the home, whereas time spent in center-based day care was associated with lower exposure.

Conclusions: Young children from low-income, nonurban communities appear to bear a higher burden of secondhand smoke exposure than previous studies have reported. Results contribute to understanding populations at greater risk, as well as specific, potentially malleable, environmental factors that may be examined as direct contributors to exposure.

Implications: Results suggest that infants from low-income, nonurban families have higher risk for environmental smoke exposure than data from nationally representative samples. Predictors of exposure offer insights into specific factors that may be targeted for risk reduction efforts, specifically conditions of children's physical space. In addition to considering the increases in risk when an adult

smoker lives in a child's home, families should also attend to the possible risk embedded within the home itself, such as residual smoke from previous occupants. For high-risk children, day care appears to mitigate the magnitude of exposure by providing extended time in a smoke-free environment.

Introduction

Exposure to secondhand cigarette smoke increases children's risk for a range of health problems including asthma, respiratory and ear infections, and sudden infant death syndrome¹ and may be a source of lead exposure.² Beyond the effects of the smoke itself, nicotine absorption in children is associated with metabolic and immune indicators of cardiovascular risk,³ periodontal disease,⁴ susceptibility to insulin resistance,⁵ and neurobehavioral changes.^{6,7} Although epidemiological data exist on the magnitude of environmental exposure among children and adolescents, less is known about exposure among infants and young children, under the age of 4 years. Primate research indicates that the developing brain continues to be vulnerable to the adverse effects of nicotine exposure well into the postnatal period.⁸ Ongoing brain development in the early years of life may make young children especially vulnerable to the programming effects of nicotine through the sensitization of cholinergic receptors, which increases susceptibility to the development of addiction later in life.^{9,10} Thus, infancy and toddlerhood may represent a time of enhanced vulnerability to the effects of environmental smoke, and research is needed to understand the prevalence of exposure in this age range, as well as circumstantial factors associated with risk or protection that may be suitable targets for policy and preventive education.

Recent research has documented that exposure to environmental smoke includes both secondhand smoke, consisting of smoke emitted from the lit cigarette and from the smoker, and thirdhand smoke, consisting of the invisible residue that settles onto floors, furniture, and clothing, where it can remain for extended periods of time.¹¹ As such, nicotine residue can be carried unwittingly into environments even where active smoking is prohibited and linger in environments previously occupied by a smoker.¹²⁻¹⁴ Parents may not recognize such sources of exposure, making them more difficult to avoid. Thirdhand routes of exposure may be especially relevant for infants and toddlers, as young children spend significant amounts of time in close physical proximity to parents, whose hair and clothing could carry nicotine residue, and often spend a significant amount of time on the floor where nicotine residue in carpeted surfaces may be persistent. Furthermore, infants often explore objects orally, increasing exposure to the invisible residue of thirdhand smoke.^{5,15} These factors suggest that young children are more likely than older children to experience both respiratory and dermal routes of exposure, and may therefore carry higher burdens of environmental smoke exposure at this age.

Large-scale epidemiological studies that have assayed cotinine, such as the National Health and Nutrition Examination Survey (NHANES), reported detectable levels of serum cotinine in one-third to one-half of the sample, depending on the year, but did not examine children under the age of 3 years.¹⁶ Several studies have sought to address this lack of knowledge by examining blood samples of children under the age of 3 years receiving routine lead screening. In a study of 1541 children, cotinine was detected in 61% of the samples, suggesting that this age group may experience greater nicotine exposure than estimated in the NHANES sample.¹⁷ This study also found higher exposure among children receiving Medicare, and among those residing in states with higher rates of smoking, suggesting that the burden of environmental smoke exposure is disproportionately carried by

lower-income children in certain geographical regions. Another study examined approximately 500 children receiving care at a free public health clinic in an urban region of California.¹⁸ Despite California being characterized by relatively lower prevalence of smoking, and statewide bans on smoking in public places, cotinine was detected in over one-half of the children sampled, again highlighting the extent to which lower socioeconomic status may be associated with greater risk for environmental smoke exposure.

Although studies have examined the prevalence of environmental smoke exposure among children, many studies classify children as "exposed," with less attention to the magnitude, or dose, of exposure. In studies of adults, cotinine values are frequently used to differentiate between active and passive exposure.¹⁹ A threshold of 12 ng/mL in saliva has been shown to have maximal sensitivity and specificity in differentiating between smokers and nonsmokers,²⁰ with research indicating that salivary and serum assays can be comparable in scale.²¹ Because children's exposure is restricted to passive sources, many studies simply define exposure as any detectable cotinine, without attention to the observed range. For example, the NHANES sample differentiated low exposure (<1.0 ng/mL) from moderate exposure, defined only as more than 1.0 ng/mL. However, levels of cotinine detected in children under the age of 3 years ranged from 0.3 to 41 ng/mL,¹⁷ indicating that young children can reach nicotine absorption levels consistent with being an active smoker. Furthermore, research on secondhand exposure among children has relied almost exclusively on single-time-point assessments, and it is not clear the extent to which a single assessment reflects sustained exposure across early childhood.

This study examines environmental smoke exposure among young children from low-income, nonurban communities. Cotinine was assayed from saliva that was collected during home visits when infants were approximately 6, 15, 24, and 48 months of age, and used to develop latent classes that describe the magnitude of secondhand smoke exposure across infancy and early childhood. Multiple factors were examined as potential predictors of exposure severity. Demographic variables including income/needs ratio (INR) and maternal education were examined as potential predictors of latent class membership. Additional measures of children's physical environments across this age range were examined to determine whether such factors predicted class membership even after accounting for socioeconomic indicators. Because young children may be particularly prone to thirdhand exposure, which persists in environments even after a smoker has departed or moved out,¹³ it was hypothesized that moving frequently to new homes, and having a greater number of adults residing with the child over time, would be associated with a greater risk for exposure. In addition, the proportion of time the child spent in an institutional day care setting, presumed to be a smoke-free environment, was examined as a potential protective factor.

Methods

Participants

The Family Life Project is an ongoing longitudinal study of rural poverty that involves families who delivered a new child between

September 2003 and August 2004 in one of six rural counties in eastern North Carolina or central Pennsylvania. A detailed characterization of the sampling plan and study has been detailed elsewhere.²² Briefly, a representative sample of 1292 children was recruited from area hospitals at the time of the child's birth. Using brief screening data collected by a study representative who visited the mother in the hospital after the child's birth, families were oversampled for low-income status (income less than or equal to twice the federal poverty limit for household size, receipt of government services, or neither parent having completed high school) in both states, and of African American families in North Carolina. All procedures were approved by the institutional review board at the University of North Carolina at Chapel Hill. Parents provided consent at each visit.

This study makes use of data that were collected in home visits when children were 2, 6, 15, 24, 36, and 48 months of age. This study is limited to 1218 children for whom at least one measure of salivary cotinine was available, which was used to define the primary outcome. Participating children did not differ from nonparticipating children with respect to state of residence (40% vs. 36% residing in Pennsylvania, $p = .51$), living in a household that was recruited into the low-income stratum (78% vs. 73% poor, $p = .33$), primary caregiver educational status at study enrollment (80% vs. 82% with a high school degree or general educational development credential, $p = .62$), sex of the child (51% vs. 54% male, $p = .57$), race of the child (43% vs. 36% African American, $p = .28$), or child first-born status (39% vs. 46%, $p = .25$).

Procedures

Following hospital screening, participants who were selected and agreed to participate were formally enrolled into the study by having a researcher complete a home visit when the target child was approximately 2 months old. Participating families received additional home visits when their child was approximately 6 and 15 months old, as well as annually from 2 to 4 years of age.

Measures

Residential Instability

At each home visit, participants were asked to report any moves since the previous home data assessment. Residential instability was calculated as the total number of residential moves children experienced in the first 4 years, and ranged from 0 to 11 ($M = 1.58$, $SD = 3.29$).

Occupant Instability

At each home visit, an inventory was taken of all children and adults (of any age) who resided in the home, where residence was defined by sleeping at the household three or more nights per week. Consistent with our previous work in this sample,²³ we created a composite variable from the data at each home visit in order to index the total number of individuals who had moved in and out of the household across the child's first 4 years of life. Values ranged from 0 to 29 ($M = 3.13$, $SD = 18.81$).

Caregiver Education

Primary caregivers reported their education level at each visit. The highest obtained level of education was used, and was classified into one of three categories. Approximately 10% of the sample did not complete their high school degree, 69% graduated from high school but did not obtain a higher degree, and 21% had completed a

bachelor's degree or greater. Two dummy codes were created indicating (1) whether or not the caregiver completed high school and (2) whether or not the caregiver completed college.

Income/Needs Ratio

Household poverty levels were defined by summing the income of all residents and dividing it by the federal poverty threshold (for each calendar year) for a given family size to create the INR. Household income information was collected at the 6-month home visit and every home visit thereafter. The mean INR value across assessments ranged from 0 to 13.60 ($M = 2.13$, $SD = 2.55$) and was used to index household poverty. Across the first 4 years of the child's life, 23% of the families lived consistently below the poverty line ($INR \leq 1$) and another 36% of the sample had an average value more than 1.0 but less than or equal to 2.0 times the federal poverty limit.

Center-Based Child Care

At each home visit, primary caregivers reported on the location of any out-of-home care the child attended. Consistent with our previous work in this sample,²⁴ a composite variable was constructed to represent the proportion of time that children attended center-based care across the full 4 years of cotinine assessments ($M = 0.19$, $SD = 0.07$).

Maternal Smoking

At the intake assessment (child age 2 months) mothers completed the pregnancy and delivery module of the Missouri Assessment of Genetics Interview for Children, which included items related to cigarette use during pregnancy.²⁵ Reich et al. reported good short- and long-term reliability for self-reports of pregnancy behaviors. Approximately 25% of mothers reported smoking during pregnancy. Mothers were also asked to report whether they currently smoked, along with the approximate number of cigarettes smoked per day when the child was 6, 24, and 35 months of age ($M = 3.52$, $SD = 37.69$).

Child Cotinine

Following Granger et al.,²⁶ cotinine in saliva was assayed using a commercially available diagnostic immunoassay (US Food, Drug and Cosmetic Act §501(k); conforms with European health and safety requirements [CE Marked]) that was specifically designed for use with saliva, without modification to the manufacturer's recommended protocol (Salimetrics, Carlsbad, CA). The assay has a test volume of 20 μ L, range of calibrators from 0.8 to 200 ng/mL, and lower limit of detection of 0.15 ng/mL. The inter- and intra-assay coefficients of variation computed for this sample were 6.7% and 6.8%, respectively.

Cotinine data are naturally highly skewed, and as such, distributions cannot be accurately characterized by the sample mean. Therefore Table 1 contains the median raw cotinine value, observed range, and first and third quartile thresholds for each assessment. To facilitate interpretation and to address the highly skewed nature of the data, each observed cotinine value was categorized into a three-level ordinal variable that reflected low exposure (≤ 0.45 ng/mL), moderate exposure (0.46–12 ng/mL), and high exposure (≥ 12 ng/mL). At 6 months, only 8% of children had cotinine values below the limit of detection (ie, undifferentiated from zero). Furthermore, only 20% of children with no detectable cotinine at 6 months continued to have no detectable cotinine at 15 months, indicating that a failure

to detect exposure in a given assay may not accurately represent the child's typical environment.¹⁴ However, of children with no detectable cotinine at 6 months, 75% had cotinine values at or below 0.45 ng/mL at 15 months, indicating that they continued to have relatively low exposure compared to the rest of the sample. Thus the boundary for low exposure was defined, arbitrarily, at 0.45 ng/mL. The threshold for defining high exposure was based on recommendations for definitions of active smoking.²⁰

Analysis Strategy

A series of conditional latent class analysis models was estimated to summarize children's environmental smoke exposure between 6 and 48 months of age.²⁷ Maternal reports of prenatal smoking along with the maternal report of the average number of cigarettes smoked per day after birth through age 3 years were used as covariates to improve latent class estimation. Following best practice,²⁸ we relied on the Bayesian information criterion to identify the optimal number of latent classes and ensured that the resulting model was substantively meaningful. Cotinine-defined latent classes were regressed on contextual risk factors using the three-step method.²⁹ This approach is conceptually similar to a multinomial regression model in which nominal latent classes are the outcomes that are regressed on predictor variables. It differs from a traditional multinomial regression model because in this case the outcome (latent classes) is probabilistic. Multiple imputation methods were used (25 imputed datasets) to deal with missing data among cotinine values and predictor variables. All analyses, including multiple imputation, were conducted using Mplus version 7 software and accounted for the complex sampling design (probability weights and stratification variables).

Results

Descriptive Statistics

The proportion of the sample classified with low, moderate, and high exposure at each assessment is reported in Table 1. As indicated, values ranged from below the limit of detection to as high as 559.58 ng/mL. Values in excess of 12 ng/mL were observed at all four time points and characterized up to 12% of the sample at ages 6 and 15 months. Exposure was lowest at the 48-month assessment, with a decline in the range of raw values, as well as the proportion of children classified with moderate or high exposure.

As reported in Table 2, polychoric correlations among ordinal exposure categories across assessments were high ($r_s = .52-.66$), as

well as with maternal reports of smoking during and after pregnancy ($r_s = .41-.69$). As expected, caregiver education and household income were moderately negatively associated with all measures of smoking ($r_s = -.14$ to $-.44$). Composite variables of residential moves and household composition were moderately positively associated with all measures of smoking and negatively associated with caregiver education and household income. The proportion of time that children spent in center-based care was weakly associated with all predictor and outcome variables ($r_s = -.12$ to $.11$).

Latent Class Analyses

We began by identifying the optimal number of classes to describe cotinine exposure during children's first 4 years. Our conditional latent class model relied on three-level ordinal indicators of observed cotinine levels at 6, 15, 24, and 48 months. On the basis of indices of model fit, and model interpretability and parsimony, we determined that three latent classes optimally described the patterns of cotinine exposure. Specifically, the Bayesian information criterion was minimized at the three-class solution; the entropy index indicated sufficient separation of classes; the three classes were substantively meaningful; and the classes had high average probabilities for most likely latent class membership (>0.85) (Table 3). The first class, *High Exposure*, represented the approximately 15% of children who had the highest probability of exposure to levels of cotinine of 12 ng/mL or more from 6 to 48 months. The second class, *Moderate Exposure*, represented the 48% of children who consistently had probabilities between 0.8 and 1.0 of moderate cotinine exposure (0.46–12 ng/mL) from 6 to 48 months. The third class, *Low Exposure*, represented the 37% of children who consistently had the highest probabilities of undetectable or low (≤ 0.45 ng/mL) levels of cotinine from 6 to 48 months.

Predicting Latent Class Membership

Table 4 presents odds ratios and 95% confidence intervals for six covariates predicting membership in each class (ie, High vs. Low Exposure; Moderate vs. Low Exposure; High vs. Moderate Exposure). With respect to socioeconomic predictors, both primary caregiver education and household income significantly predicted children's cotinine exposure class. Conditional on all other covariates, having a high school degree was associated with an 85% decrease in the odds of having a child in the High Exposure class relative to both the Low and Moderate Exposure classes compared with caregivers who did not graduate. Moreover, having achieved a 4-year college degree was associated an 85% decrease in the odds of

Table 1. Descriptive Data for Cotinine Exposure Across Time Points

	Raw cotinine values (ng/mL)				
	N	Median	Range	25%	75%
6 mo	1118	1.61	0–215.12	0.47	5.84
15 mo	935	1.83	0–90.29	0.47	5.50
24 mo	899	1.66	0–559.58	0.41	5.32
48 mo	932	0.69	0–76.22	0.20	3.18
	Ordinal classification of exposure				
	Low (<0.45 ng/mL)	Moderate (0.45–12 ng/mL)	High (≥ 12 ng/mL)		
6 mo	24%	64%	12%		
15 mo	24%	64%	12%		
24 mo	26%	64%	10%		
48 mo	41%	54%	5%		

Table 2. Associations Among Study Predictors

	1	2	3	4	5	6	7	8	9	10	11	12
1 Cotinine (6 mo)												
2 Cotinine (15 mo)	0.56											
3 Cotinine (24 mo)	0.57	0.66										
4 Cotinine (48 mo)	0.52	0.54	0.59									
5 Prenatal smoking	0.42	0.41	0.43	0.41								
6 Postnatal cigarettes per day ^a	0.46	0.48	0.49	0.49	0.69							
7 High school degree	-0.22	-0.23	-0.22	-0.29	-0.14	-0.19						
8 College degree	-0.38	-0.38	-0.44	-0.40	-0.25	-0.26	0.17					
9 Income/needs ratio ^a	-0.37	-0.40	-0.38	-0.38	-0.20	-0.24	0.22	0.53				
10 Residential instability ^a	0.34	0.33	0.30	0.36	0.23	0.25	-0.22	-0.29	-0.37			
11 Occupant instability ^a	0.31	0.30	0.28	0.31	0.22	0.24	-0.22	-0.25	-0.33	0.58		
12 % time in center care ^a	-0.05	-0.11	-0.12	-0.11	-0.08	-0.09	0.11	0.07	0.09	0.01	-0.04	
N	1118	935	899	932	1209	1214	1218	1218	1212	1057	1057	1218

^aComposite variables aggregated across multiple assessments. Cotinine at each time point reflects the ordinal classification of exposure.

Table 3. Model Fit Statistics for Conditional Latent Class Analysis

Classes	Log likelihood	AIC	BIC	Adjusted BIC	Entropy
1	-8154.8	16333.5	16394.8	16356.7	—
2	-2719.8	5477.7	5574.6	5514.3	0.815
3	-2541.4	5142.7	5295.9	5200.6	0.782
4	-2504.8	5091.7	5301.0	5170.8	0.680

AIC = Akaike’s information criterion, BIC = Bayesian Information Criterion. Results run on 25 imputed datasets.

Table 4. Predictors of Secondhand Smoke Exposure Classes

	High exposure (Reference: low exposure)			Moderate exposure (Reference: low exposure)			High exposure (Reference: moderate exposure)		
	Odds ratio	(95% CI)	<i>p</i>	Odds ratio	(95% CI)	<i>p</i>	Odds ratio	(95% CI)	<i>p</i>
High school degree	0.14	(0.03% to 0.60%)	.01	0.27	(0.06% to 1.21%)	.09	0.52	(0.29% to 0.94%)	.02
College degree	NE	—		0.15	(0.08% to 0.30)	.00	NE	—	
Income/needs ratio ^a	0.58	(0.40% to 0.82%)	.00	0.67	(0.55% to 0.82%)	.00	0.86	(0.61% to 1.23%)	.14
Residential instability ^a	1.43	(1.17% to 1.74%)	.00	1.28	(1.06% to 1.54%)	.01	1.12	(0.98% to 1.27%)	.09
Occupant instability ^a	1.11	(1.01% to 1.21%)	.05	1.07	(0.98% to 1.16%)	.14	1.04	(0.98% to 1.10%)	.21
% time in center care ^a	0.19	(0.05% to 0.71%)	.01	1.32	(0.62% to 2.78%)	.47	0.14	(0.04% to 0.51%)	.04

CI = confidence interval, NE = nonestimable because of small cell size. Adjusted odds ratios are reported, indicating the changes in odds of class membership unique to each predictor accounting for all other predictors in the model.

Bolded values indicate significant odds ratios.

^aComposite variables aggregated across multiple assessments.

having a child in the Moderate relative to the Low Exposure class. Independent of education, each unit increase in INR reduced the odds of being in the High or Moderate Exposure classes by 42% and 33%, respectively, relative to the Low Exposure class.

Controlling for socioeconomic indicators, dynamic household factors independently predicted children’s environmental smoke exposure. Higher levels of residential instability (the number of total times the child moved) and occupant instability (the total number of individuals moving into or out of the child’s home) were

both associated with increased odds of membership in High and Moderate Exposure classes relative to the Low Exposure class. Every additional move increased the odds of membership in the High vs. Low Exposure class by 43% and the Moderate vs. Low Exposure class by 28%. Each additional individual moving into or out of a child’s house increased the odds of membership in the High vs. Low Exposure class by 11%.

Finally, the estimates in Table 4 suggest that whether and where children spend out-of-home time is related to cotinine exposure

during their early years. Net of socioeconomic and family dynamic characteristics, a higher proportion of time spent in center-based care reduced children's odds of membership in the High Exposure class relative to Low Exposure by 81% and of membership in the Moderate Exposure by 86%.

Discussion

This study examined cotinine levels in a longitudinal sample of children from age 6 to 48 months. Results demonstrated a high prevalence and magnitude of exposure, with approximately 15% of the children in the study registering cotinine levels considered to reflect active smoking in studies with adults.²⁰ This study capitalizes on a relatively large, longitudinally followed, and well-characterized sample of families to provide unique insight into the demographic characteristics and contextual factors associated with vulnerability for significant environmental smoke exposure among young children.

Previous studies have reported that children from lower-income families (receiving government-subsidized health care) were more likely to have environmental smoke exposure,^{17,18} an association evident in the current sample as well. Findings from the current study further demonstrate that maternal education predicts children's exposure even after accounting for the effect of income status. Previous studies have also reported exposure to be higher among children residing in states where overall rates of smoking were higher.¹⁷ Children in the present study were drawn from two states with comparably moderate-to-high prevalence rates of smoking, approximately 18%,³⁰ which likely contributed to the high prevalence of exposure observed in this sample relative to samples drawn from broader geographic regions.^{16,17} Beyond these socioeconomic indicators, this study examined additional demographic factors in an effort to better characterize specific contexts and conditions of the physical environment that may contribute exposure risk independent of socioeconomic status indicators. Results indicate that children who moved frequently had greater odds of exposure for both the moderate- and high-exposure groups. Nicotine residue (ie, thirdhand smoke) can remain in a home previously occupied by a smoker on soft surfaces such as carpet or drapery, and in dust in ventilation and heating systems.¹³ As indicated earlier, infants may be more susceptible to these routes of exposure because of the time they spend at ground level and are more likely to explore their environment orally. Although frequency of residential moves was found to increase exposure odds even after controlling for the effects of income and education, residential instability is itself correlated with poverty. These findings may help illuminate specific, and potentially modifiable, processes by which low-income individuals experience greater risk for environmental smoke exposure. Families could be educated to identify exposure risks that may unknowingly reside in their environment and to seek information about smoking status of previous occupants when relocating. Similarly, policies requiring landlords to engage specific cleaning practices between occupants could protect low-income families from thirdhand exposure.

In addition to changes in the physical dwelling, changes in the occupants of the home also predicted increased exposure. Each adult moving in or out of the home increased the odds of being classified with High Exposure by 11% relative to children in the Low Exposure group. High numbers of adult occupants across time increase the chances that the child will be exposed to an adult who smokes. High frequency of change in adult occupants in the home may be more common among lower-income families needing

to share the financial burden of housing expenses and may be an additional process by which poverty creates an array of correlated risk factors. Parents should be educated to recognize risks posed by residents who smoke, regardless of whether they smoke in the direct proximity of the child. Together these findings illustrate the importance of considering a broad range of sources of exposure.

Consistent with the implications of residential conditions regarding increased risk for exposure, children who spent time in center-based day care settings were significantly less likely to be classified in the highest exposure group. This finding suggests that children who register the highest levels of cotinine are likely to be accumulating this exposure throughout the day, and increased time in smoke-free environments reduces this burden. In previous analyses with this dataset, time in child care was associated with a lower rate of problem behavior, lower levels of stress hormones, and higher verbal and executive function at school entry for children from highly chaotic homes.^{24,31} The results from the current study further reinforce the potential protective effects that child care can offer to high-risk families.

Although our results indicated a high level of stability within the exposure classes, exposure was lower at the 48-month assessment. It is possible that this finding is commensurate with developmental changes occurring by age 4 years, when children are less likely to place objects in their mouth and may be more compliant with hand-washing practices than younger children; or it may reflect older children spending more time outside of the home, such as in pre-school settings. However, it is also important to consider this trend in light of broader social changes in policy (eg, banning smoking in public places) and increased social awareness of secondhand smoke risks that were occurring over the time the assessments took place. Epidemiological data illustrate a comparable decline in exposure around the same calendar year the children in the Family Life Project were approximately 48 months.¹⁶

This study provides insight into environmental factors associated with increased environmental exposure to cigarette smoke among infants in low-income families. The repeated assessments across development, the home-based data collection, and the characterization of families' environments contribute to a unique set of data targeted at an underrepresented population. It is important to note, however, that these results are preliminary and should be interpreted in light of several limitations. This sample was recruited as a part of a study designed to understand development in the context of specific socioeconomic and geographical conditions, and may therefore not generalize to other populations. Specifically, it is not clear whether these same predictors would emerge in urban samples. Furthermore, the variables available in this study serve as probable indicators of risk mechanisms that were not able to be measured directly. Future studies should ascertain these risks more directly, such as the smoking status of adult cohabitants, adult exposure to secondhand smoke in their workplace, and smoking status of nonresident relatives who visit the home. In addition, better characterization of the housing environment may provide more information on how exposure can be reduced. Factors such as the size of the home, whether smoking is restricted inside the home, ventilation, and proximity to neighbors may all affect the exposure children encounter.

In addition, the thresholds used to classify smoking status in this study are not presumed to define boundaries associated with developmental risks stemming from exposure. Although the threshold of 12 ng/mL conveys that infants are capable of absorbing nicotine at levels previously assumed to be limited to active smoking, it is not

the case that this threshold is known to differ from lower values with regard to the associated health effects children may suffer from exposure. Thus these findings should be considered a preliminary examination of environmental factors that predict broad classes of exposure.

Research demonstrates that policies to restrict smoking in public places result in measurable decreases in children's exposure to secondhand smoke.³² Given the effectiveness of initiatives to educate and empower parents to reduce children's exposure,³³ programs should be expanded to focus on the risk of thirdhand exposure for very young children. The findings from this study extend previous observations that socioeconomic status is an indicator of increased exposure risk by identifying potential pathways by which risk is increased. These findings suggest that additional consideration may need to be given to policies aimed at thirdhand routes of exposure, and mechanisms by which nonsmoking parents could introduce nicotine into their child's environment. For instance, research has documented that nonsmoking adults experienced increased secondhand exposure in workplaces that permitted smoking, even when precautions were taken to contain smoking to certain areas, and to ensure well-designed ventilation.³⁴ Eliminating environmental smoke exposure in young children will ultimately require a comprehensive understanding of the many environments children encounter, both public and private, and the many ways in which tobacco smoke can infiltrate those spaces.

Funding

Support for this research was provided by the National Institute of Child Health and Human Development (PO1-HD-39667), with co-funding from the National Institute on Drug Abuse. This research was also funded by the National Institutes of Health (1UG3OD02332-01).

Declaration of Interests

In the interest of full disclosure, DAG is the Chief Scientific and Strategy Advisor at Salimetrics LLC and Salivabio LLC. The nature of these relationships is managed by the policies of the committees on Conflict of interest at Johns Hopkins University School of Medicine and the University of California at Irvine.

Acknowledgments

The Family Life Project (FLP) key investigators include Lynne Vernon-Feagans, the University of North Carolina; Mark Greenberg, the Pennsylvania State University; Martha Cox, the University of North Carolina; Clancy Blair, New York University; Margaret Burchinal, the University of North Carolina; Michael Willoughby, the University of North Carolina; Patricia Garrett-Peters, the University of North Carolina; and Roger Mills-Koonce, the University of North Carolina. The FLP investigators and project staff would like to thank all the families and children who participated in this project as well as all the child care providers, all of whom graciously allowed us into their daily lives.

References

1. Banderali G, Martelli A, Landi M, et al. Short and long term health effects of parental tobacco smoking during pregnancy and lactation: a descriptive review. *J Transl Med.* 2015;13:327.
2. Apostolou A, Garcia-Esquinas E, Fadrowski JJ, McLain P, Weaver VM, Navas-Acien A. Secondhand tobacco smoke: a source of lead exposure in US children and adolescents. *Am J Public Health.* 2012;102(4):714–722.
3. Groner JA, Huang H, Joshi MS, Eastman N, Nicholson L, Bauer JA. Secondhand smoke exposure and preclinical markers of cardiovascular risk in toddlers. *J Pediatr.* 2017;189:155–161.
4. Sanders A, Slade G. State cigarette excise tax, secondhand smoke exposure, and periodontitis in US nonsmokers. *Am J Public Health.* 2013;103(4):740–746.
5. Adhami N, Starck SR, Flores C, Martins Green M. A health threat to bystanders living in the homes of smokers: how smoke toxins deposited on surfaces can cause insulin resistance. *PLoS One.* 2016;11(3):e0149510.
6. Gatzke-Kopp LM, Beauchaine TP. Direct and passive prenatal nicotine exposure and the development of externalizing psychopathology. *Child Psychiatry Hum Dev.* 2007;38(4):255–269.
7. Pagani LS. Environmental tobacco smoke exposure and brain development: the case of attention deficit/hyperactivity disorder. *Neurosci Biobehav Rev.* 2014;44:195–205.
8. Slotkin TA, Pinkerton KE, Seidler FJ. Perinatal environmental tobacco smoke exposure in rhesus monkeys: critical periods and regional selectivity for effects on brain cell development and lipid peroxidation. *Environ Health Perspect.* 2006;114(1):34–39.
9. Hellström-Lindahl E, Nordberg A. Smoking during pregnancy: a way to transfer the addiction to the next generation? *Respiration.* 2002;69(4):289–293.
10. Leung CY, Leung GM, Schooling CM. Early second-hand smoke exposure and child and adolescent mental health: evidence from Hong Kong's "Children of 1997" birth cohort. *Addiction.* 2015;110(11):1811–1824.
11. Bush D, Goniewicz ML. A pilot study on nicotine residues in houses of electronic cigarette users, tobacco smokers, and non-users of nicotine-containing products. *Int J Drug Policy.* 2015;26(6):609–611.
12. Matt GE, Quintana PJ, Hovell MF, et al. Residual tobacco smoke pollution in used cars for sale: air, dust, and surfaces. *Nicotine Tob Res.* 2008;10(9):1467–1475.
13. Matt GE, Quintana PJ, Zakarian JM, et al. When smokers move out and non-smokers move in: residential thirdhand smoke pollution and exposure. *Tob Control.* 2011;20(1):e1.
14. Matt GE, Quintana PJ, Fortmann AL, et al. Thirdhand smoke and exposure in California hotels: non-smoking rooms fail to protect non-smoking hotel guests from tobacco smoke exposure. *Tob Control.* 2014;23(3):264–272.
15. Falck AJ, Mooney S, Kapoor SS, White KM, Bearer C, El Metwally D. Developmental exposure to environmental toxicants. *Pediatr Clin North Am.* 2015;62(5):1173–1197.
16. Environmental Protection Agency [US]. *EPA's Report on the Environment (ROE): Serum Cotinine Level.* 2015. <https://cfpub.epa.gov/roe/indicator.cfm?i=26#4>. Accessed January, 2018.
17. Joseph A, Spector L, Wickham K, et al. Biomarker evidence of tobacco smoke exposure in children participating in lead screening. *Am J Public Health.* 2013;103(12):e54–e59.
18. Dempsey DA, Meyers MJ, Oh SS, et al. Determination of tobacco smoke exposure by plasma cotinine levels in infants and children attending urban public hospital clinics. *Arch Pediatr Adolesc Med.* 2012;166(9):851–856.
19. Kim S. Overview of cotinine cutoff values for smoking status classification. *Int J Environ Res Public Health.* 2016;13(12):1236.
20. Jarvis MJ, Fidler J, Mindell J, Feyerabend C, West R. Assessing smoking status in children, adolescents and adults: cotinine cut-points revisited. *Addiction.* 2008;103(9):1553–1561.
21. Benowitz NL. Biomarkers of environmental tobacco smoke exposure. *Environ Health Perspect.* 1999;107 (Suppl 2):349–355.
22. Vernon-Feagans L, Cox M; FLP Key Investigators. The family life project: an epidemiological and developmental study of young children living in poor rural communities. *Monogr Soc Res Child Dev.* 2013;78(5):1–150, vii.
23. Vernon-Feagans L, Garrett-Peters P, Willoughby M, Mills-Koonce R; The Family Life Project Key Investigators. Chaos, poverty, and parenting: predictors of early language development. *Early Child Res Q.* 2012;27(3):339–351.
24. Berry D, Blair C, Willoughby M, Garrett-Peters P, Vernon-Feagans L, Mills-Koonce WR; Family Life Project Key Investigators. Household chaos and children's cognitive and socio-emotional development in early

- childhood: does childcare play a buffering role? *Early Child Res Q.* 2016;34(1 quarter):115–127.
25. Reich W, Todd RD, Joyner CA, Neuman RJ, Heath AC. Reliability and stability of mothers' reports about their pregnancies with twins. *Twin Res.* 2003;6(2):85–88.
26. Granger DA, Blair C, Willoughby M, et al.; Family Life Project Investigators. Individual differences in salivary cortisol and alpha-amylase in mothers and their infants: relation to tobacco smoke exposure. *Dev Psychobiol.* 2007;49(7):692–701.
27. Lanza ST, Bray BC, Collins LM. An introduction to latent class and latent transition analysis. In: Schinka JA, Velicer WF, Weiner IB, eds. *Handbook of Psychology: Research Methods in Psychology.* 2nd ed., Vol. 2. Hoboken, NJ: Wiley; 2012:691–716.
28. Nylund KL, Asparouhov T, Muthen BO. Deciding on the number of classes in latent class analysis and growth mixture modeling: a Monte Carlo simulation study. *Struct Equ Modeling.* 2007;14(4):535–569.
29. Asparouhov T, Muthen B. Auxiliary variables in mixture modeling: three-step approaches using MPlus. *Struct Equ Modeling.* 2014;21(3):329–341.
30. Centers for Disease Control and Prevention. *State Tobacco Activities Tracking & Evaluation (STATE) System. Map of Current Cigarette Use Among Adults (Behavior Risk Factor Surveillance System).* 2016. https://www.cdc.gov/tobacco/data_statistics/fact_sheets/adult_data/cig_smoking/index.htm. Accessed June 21, 2018.
31. Berry D, Blair C, Granger DA; Family Life Project Key Investigators. Child care and cortisol across infancy and toddlerhood: poverty, peers, and developmental timing. *Fam Relat.* 2016;65(1):51–72.
32. Aurekoetxea JJ, Murcia M, Rebagliato M, et al. Second-hand smoke exposure in 4-year-old children in Spain: sources, associated factors and urinary cotinine. *Environ Res.* 2016;145:116–125.
33. Butterfield PG, Hill W, Postma J, Butterfield PW, Odom-Maryon T. Effectiveness of a household environmental health intervention delivered by rural public health nurses. *Am J Public Health.* 2011;101 (Suppl 1):S262–S270.
34. Repace JL. Secondhand smoke in Pennsylvania casinos: a study of non-smokers' exposure, dose, and risk. *Am J Public Health.* 2009;99(8):1478–1485.