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Screening for Environmental Tobacco Smoke Exposure among Inner City Children with Asthma

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Abstract

Background—Environmental tobacco smoke (ETS) causes increased morbidity among children with asthma, however pediatricians do not consistently screen and counsel families of asthmatic children regarding ETS. An index score based on parent report of exposure could help providers efficiently screen for ETS.

Objective—1) To develop an index measure of ETS based on parent self-report of smoking behaviors; 2) To determine whether the index score is associated with children's present and future cotinine levels.

Methods—Data were drawn from a community intervention for inner-city children with persistent asthma (n=226, response rate 72%). Measures of child salivary cotinine and parent self-reported ETS-related behaviors were obtained at baseline and 7–9 months later. To develop the index score, we used a 15-fold cross-validation method on 70% of our data that considered combinations of smoke exposure variables, controlling for demographics. We chose the most parsimonious model that minimized the mean square predictive error. The resulting index score included primary caregiver smoking and home smoking ban status. We validated our model on the remaining 30% of data. ANOVA and multivariate analyses were used to determine the association of the index score with children's cotinine levels.

Results—54% of asthmatic children lived with ≥1 smoker and 51% of caregivers reported a complete home smoking ban. The children's mean baseline cotinine was 1.55ng/ml (range 0.0–21.3). Children's baseline and follow-up cotinine levels increased as scores on the index measure increased. In a linear regression, the index score was significantly and positively associated with children's cotinine measurements at baseline ($p<.001$, model $R^2=.37$) and 7–9 months later ($p<.001$, $R^2=.38$).

Conclusion—An index measure with combined information regarding primary caregiver smoking and household smoking restrictions helps to identify asthmatic children with the greatest exposure to ETS, and can predict children who will have elevated cotinine levels 7–9 months later.

Keywords

Environmental tobacco smoke; asthma; children; primary care; screening

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INTRODUCTION

Environmental tobacco smoke (ETS) exposure is a known risk to health and a preventable cause of morbidity among children.[1,2] More than one quarter of children in the US live with at least one smoker.[2] While many states have restrictions on smoking in public places, the greatest source of ETS exposure for children occurs in the home.[3–5]

ETS exposure is of particular concern for children with asthma. Children with asthma who are exposed to ETS experience more severe symptoms and more frequent exacerbations compared to children without exposure.[6,7] While parents describe a variety of intended ETS harm-reduction strategies when describing their smoking practices, many children, even those with significant asthma, continue to experience exposure to ETS.[8] Not surprisingly, avoiding ETS is recommended as an important component of asthma management.[9]

Exposure to ETS is best measured through the biomarker cotinine. Cotinine is a metabolite of nicotine, which is measured from samples of urine, serum, saliva, and hair. Under conditions of sustained smoke exposure, cotinine levels provide valid and quantitative measures of average ETS exposure.[10–12] Cotinine levels have been positively correlated with risk of ETS-related health complications in children.[10,11] While cotinine measurements now are commonly used in research studies, such measurements are not currently feasible in routine pediatric health care due to cost and time constraints.

Pediatric clinicians are in an ideal position to screen and counsel families regarding childhood ETS exposure. However these practices do not occur consistently during health care visits,[13,14] in part due to limited time and competing priorities.[14] Researchers have designed tools to systematically and accurately assess children's smoke exposure. For example, Johansson et al found that urine cotinine/creatinine levels in children were significantly related to parents' self-reported smoking behaviors on a written questionnaire.[15] In addition, Groner et al derived and validated an office-based tool to identify non-asthmatic young children at risk for ETS exposure.[16] The usefulness of these types of tools for parents of children with asthma has not been established.

The objective of this study was to develop a simple algorithm, based on parent self-report of smoking behaviors, that provides quick and accurate information on ETS exposure and that, unlike biologic measures of exposure, can be easily integrated into clinical practice. We further sought to determine whether the algorithm could be used to predict which children would continue to have high levels of exposure 7–9 months later.

METHODS

Study Population

We analyzed data from 226 children 3–10 years of age with asthma who were participating in a school-based asthma treatment program (the School-Based Asthma Therapy Trial). Children whose parents indicated that they have asthma on their school screening forms were identified for potential enrollment. Parents of these children received a telephone survey to determine if the child met study criteria.

Eligibility for the program required children to have physician-diagnosed asthma with mild persistent to severe persistent symptoms.[9] All children attended school or preschool in the Rochester City School district, and their primary care provider authorized their participation. We excluded children with congenital heart disease, cystic fibrosis, or other chronic lung disease, that could interfere with the assessment of asthma-related outcome measures. We also excluded children whose families were planning to move within the next 6 months. Two

hundred and twenty-six out of 313 eligible children were enrolled (response rate:72%) from August 2006-November 2006.

During a baseline evaluation for this program at the beginning of the school year, we conducted extensive home-based interviews with parents or caregivers (hereafter referred to as 'parents') to obtain information regarding demographics, asthma symptoms and medications, and household smoking behaviors. Saliva samples also were obtained from every child (n=226) for cotinine measurement during the same time period. A follow-up assessment for each child was done 7–9 months after baseline (at the end of the school year, in June/July) during which an additional cotinine measurement was obtained. We were able to obtain follow-up cotinine measurements for 216 children (96%); 7 children had withdrawn and 3 missed the follow-up collection. Because some of the children were eligible for an ETS reduction program as part of their participation in the intervention, we only used follow-up data from the children who did not receive the ETS reduction intervention (n=162). The University of Rochester Institutional Review Board approved the study protocol, and informed consent was obtained from all caregivers.

Smoke Exposure

We assessed ETS exposure by salivary cotinine measurements and parent self-report. Salivary cotinine was used because it is well accepted for the measurement of ETS exposure, non-invasive, and can be done quickly and easily in young children.[17] We collected saliva samples with a sorbette (a wand with a small sponge) using a standard protocol developed for children. Study staff collected three sorbettes from each child to obtain sufficient saliva for analysis. Cotinine analyses were performed with Enzyme Immunoassay techniques by Salimetrics LLC, State College, PA, and reported in ng/ml. The lower limit of cotinine sensitivity is 0.05 ng/ml. Undetectable cotinine values were recorded as 0 (baseline; n=9, follow-up; n=6).

Parent report of children's exposure to ETS in the home was measured by responses to detailed questions asked at the baseline home interview. We asked caregivers how often they smoked (never, occasionally, daily; dichotomized as never vs. occasionally or daily) and the number of smokers living in the child's home (0, 1, 2, 3, ≥4; dichotomized as 0 vs. ≥1).[18] We measured home smoking restrictions[19] by asking parents "which option best describes the situation regarding smoking in this child's home"; 1)smoking is allowed in any common room of the home, 2)smoking is limited to a part of the house where the child rarely goes, and 3)there is no smoking at all. If the parent reports no smoking at all, then we asked if there are any exceptions to this situation. A complete home smoking ban was defined as a parent who reported no smoking at all with no exceptions.[19] Similarly, we asked about smoking rules in the car (for those who owned a car); families with no smoking in the car with no exceptions were defined as having a car smoking ban.

Demographic Measures

Demographic variables included age (preschool, 3–5 years vs. school-age, 6–10 years), gender, race (White, Black, or other), ethnicity (Hispanic or not Hispanic) and insurance status (Medicaid or other). Parental characteristics included parent age (<30 or ≥30 years) and education (< high school or ≥ high school).

Psychosocial Measures

We used the previously validated 10-item Kessler Psychological Distress Scale (K-10)[20] to assess parental symptoms of depression. Parents were asked to think about the prior 30 days and answer on a 5-point likert scale. Scores were summed and categorized into well or mild symptoms of depression (score 10–24) vs. moderate or severe symptoms (score 25–50). We measured parent stress using questions from the Parenting Stress Index.[21] Five items were

scored on a 5-point scale including questions such as: "being a parent is harder than I thought it would be" and "I can't handle making decisions without help". Responses were summed for a total parent stress score (range 0–20) with higher scores indicating increased stress.

Analysis

We performed analyses using SPSS version 15.0 (Statistical Product and Service Solutions 15.0; SPSS Inc, Chicago, Ill), and STATA (version 9, StataCorp, College Station, Texas). We used t-test and ANOVA statistics to compare cotinine values between children of different demographic populations and ages and with different household smoking behaviors. To develop the index score, we used a 15-fold cross-validation method[22] on 70% of our data (158 observations) that looked at the different combinations of the exposure variables, controlling for demographics (age, race and ethnicity). We chose the most parsimonious model that minimized the mean square predictive error. We then validated our model on the remaining 30% of data (68 observations). We ended up with a 2-item index measure, and then evaluated the relationship between the index measure and cotinine values using ANOVA statistics. We determined the relationship between the index score and cotinine values at baseline and follow-up using regression analyses, controlling for the child's age, race, Hispanic ethnicity, parent education, stress and depressive symptoms. Due to the non-normal distribution of cotinine data, we used the natural log function to transform the data prior to analysis. A 2-sided alpha $<.05$ was considered statistically significant.

RESULTS

Table 1 shows the overall demographic characteristics and household smoking behaviors for the children. The mean age was 7 years (SD 1.9). Most children were male (58%), Black (65%) and had Medicaid insurance (74%). More than half of the parents had a high school degree. Thirty-nine percent of caregivers reported smoking either occasionally or daily and 54% of children lived in a home with ≥1 smoker. Complete smoking bans were reported in 51% of children's homes.

Table 1 also shows bivariate comparisons of demographic variables and smoking characteristics by children's cotinine scores. Overall, the children's mean baseline cotinine was 1.55ng/ml (range 0.0–21.3). White children in this sample had the highest mean cotinine values, as compared to black children and children of other racial backgrounds (2.44ng/ml, 1.67ng/ml, 0.82ng/ml respectively, p=.019). The mean cotinine values were significantly higher in children of caregivers who smoked (2.82ng/ml) vs. children whose parents did not smoke (0.72ng/ml, p<.001). Children from households with no smoking bans had higher cotinine levels (2.50ng/ml) than children from household with complete smoking bans (0.63ng/ml, p<.001).

The model that minimized the mean square prediction error from our cross-validation procedure included the exposure variables *primary caregiver smoking* and *smoking ban status*. This model significantly increased prediction of cotinine level from our base model that just included demographic information (p=.0003). We used this model to create our 2-item index score. We validated the index score in the 30% of the sample that were not included in the original development of the measure. Validation of this model suggested little bias.

Figure 1 illustrates the distribution of the index score values and its relationship with children's cotinine measurements. Primary caregiver smoking was given the greatest weight on the algorithm, since it had the lowest mean square prediction error, and smoking ban status was included on the second tier. The index score values range from 1–4. Children whose primary caregiver did not smoke and who had a complete home smoking ban had an index score of 1 (44% of the sample). Children with a non-smoking primary caregiver but no smoking ban in

the home received a score of 2 (16%). Children whose primary caregiver smoked and who lived in a home with a complete smoking ban received a 3 (7%). The maximum score (4) was applied to those children with a primary caregiver who smoked and reported no smoking ban in the home (33%).

We found that mean baseline cotinine values increased as children's scores on the index measure increased. Children with a primary caregiver who smoked who were not protected by a household smoking ban (score of 4) had the highest mean cotinine value (3.06ng/ml), and children with a non-smoking primary caregiver who reported a household smoking ban (score of 1) had the lowest mean cotinine levels (.50ng/ml). Follow-up cotinine values overall were slightly lower than at baseline, due in part to the exclusion of some smoke-exposed children who participated in an ETS reduction program, and the lower levels of exposure often seen during the summer months.[23, 24] Despite this, we found that the follow-up cotinine values 7–9 months after the initial assessment increased as children's scores on the index measure increased. Children with a primary caregiver who smoked and were not protected by a smoking ban at baseline had the highest cotinine values 7–9 months later (2.49ng/ml)

In a multivariate linear regression model estimating baseline cotinine values, the index score was the most significant variable with the largest standardized coefficient (β=.552), even when controlling for demographic characteristics, parent depression and stress ($p<.001$, model R^2=0.37). These findings were substantiated in a similar model with the baseline index score predicting cotinine values 7–9 months following the baseline assessment ($p<.001$, model R^2=0.38).

Figure 2 shows the actual cotinine values (geometric means) by index score, with associated 95% confidence intervals, as well as the cotinine values predicted from the multivariate regression model. Predictions are reasonably accurate, demonstrating the same trend of increasing cotinine values as scores on the index measure increase. There was a similar pattern for follow-up cotinine values (Figure 3).

DISCUSSION

Many children with persistent asthma continue to be exposed to ETS. In this study we found that combined information regarding primary caregiver smoking and household smoking restrictions helps to identify asthmatic children with the greatest exposure to ETS, and can predict those children who will have elevated cotinine levels 7–9 months later. This information could be used to help clinicians estimate risk of exposure among their asthmatic patients and guide their management with two simple questions.

Other studies have similarly shown that questionnaire information can be useful to determine a child's ETS exposure category.[3,16] For example, Groner et al enrolled young children from a primary care office and developed a model based on mother's smoking status, exposure to others smoking, and whether others smoked inside or outside.[16] These questions related to children's cotinine levels; however children with asthma were excluded from the study. Our study builds on this work by specifically considering a group of children with asthma who are at elevated risk from the deleterious effects of ETS, and by also using the index score to predict future cotinine levels. We found that a similar method of inquiry to parents regarding smoking behaviors is useful among urban children with asthma.

Other research supports our finding of the importance of primary caregiver smoking and home smoking rules as significant contributors to children's ETS exposure levels. Prior studies have demonstrated that maternal smoking contributes more to a child's ETS exposure than smoking by others in the household.[25–29] Additionally, a study by Wakefield et al[19] showed that children living in homes with absolute smoking bans had lower cotinine levels than children

from homes with smoking bans to which exceptions were made, and this observation has been substantiated in other studies.[3,29] The prevalence of primary caregiver smoking and smoking bans found in this study is consistent with other studies of young children with asthma. In the inner-city asthma study 59% of children lived with a smoker,[30] and Berman et al[29] found that 47% of smoking caregivers of asthmatic children reported absolute smoking restrictions in the home.

Cotinine is a marker of recent ETS exposure with a half-life of 16–20 hours. Thus cotinine measurements reflect smoke exposure over the past several days. Reported information on past smoking habits has been associated with present cotinine levels in children, suggesting that children's exposure to nicotine is relatively stable.[31] The data presented here demonstrate that parent report of smoking behaviors is strongly associated with children's cotinine levels 7–9 months later, suggesting that smoke exposure among asthmatic children remains relatively stable over time.

There is no level of smoke exposure that is known to be safe for children, and the clinical significance of small differences in cotinine is not clear. Therefore we considered cotinine as a continuous measure in this study. Current methods allow for detection of cotinine as low as . 05ng/ml to quantify ETS exposure. While children with the lowest scores on our index of household smoking behaviors clearly had lower cotinine values compared to children with higher scores, it is important to note that many children in the lowest categories still experienced some exposure to smoke. This suggests the need for ongoing public health efforts around the deleterious effects of ETS exposure, particularly for children with chronic illnesses such as asthma.

Health care provider counseling regarding smoking cessation can be effective in helping smokers quit.[32–36] However there is significant room for improvement in provider counseling and intervention regarding smoke exposure.[14,37–41] Simple screening methods to identify children at high risk could help providers identify those families in greatest need for intervention. We propose the use of two simple questions in the primary care setting: 'How much does the child's primary caregiver smoke'; and 'what are the rules regarding smoking in this child's home'; to help identify asthmatic children's present and future risk for significant smoke exposure. Families with *either* a primary caregiver who smokes or a lack of complete smoking restrictions in the home should be counseled on both smoking cessation and ETS reduction and referred to services as appropriate. New training programs to improve resident physician's tobacco intervention skills are now being evaluated,[37] and these skills will be particularly useful for families of smoke exposed children with asthma.

There are some potential limitations to this study. First, household smoking information was obtained from parents as part of an intervention study to improve preventive care for asthma. Parents may have been subject to social desirability bias and underreported smoking behaviors. Despite this possibility, we still found strong associations between the index measure and children's cotinine levels. All of the children in this study had significant asthma, and therefore we were unable to correlate smoke exposure with symptom severity in this sample. We purposely limited the number of factors in the index score to assure simplicity of use for a busy practitioner. While additional information such as household size might have strengthened the association further, we found that the addition of more variables from our dataset did not change the least mean squared estimate significantly. Further, the majority of children included in the study were African-American, and data suggests that cotinine levels in African-American children may be higher than in children from other racial backgrounds with similar exposure to ETS.[42] Lastly, this sample included children with persistent asthma living in an urban community, and the results can only be generalized to similar populations.

Implications

Pediatricians have an important role to play in protecting children from ETS exposure. The brief index presented here has potential use to help identify children at risk of exposure, and could serve as a guide for counseling in clinical settings. Consistent screening and counseling regarding smoke exposure likely will help to reduce the burden of smoke-related morbidity for all children and in particular for children with asthma.

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Abbreviations

ETS

Environmental Tobacco Smoke

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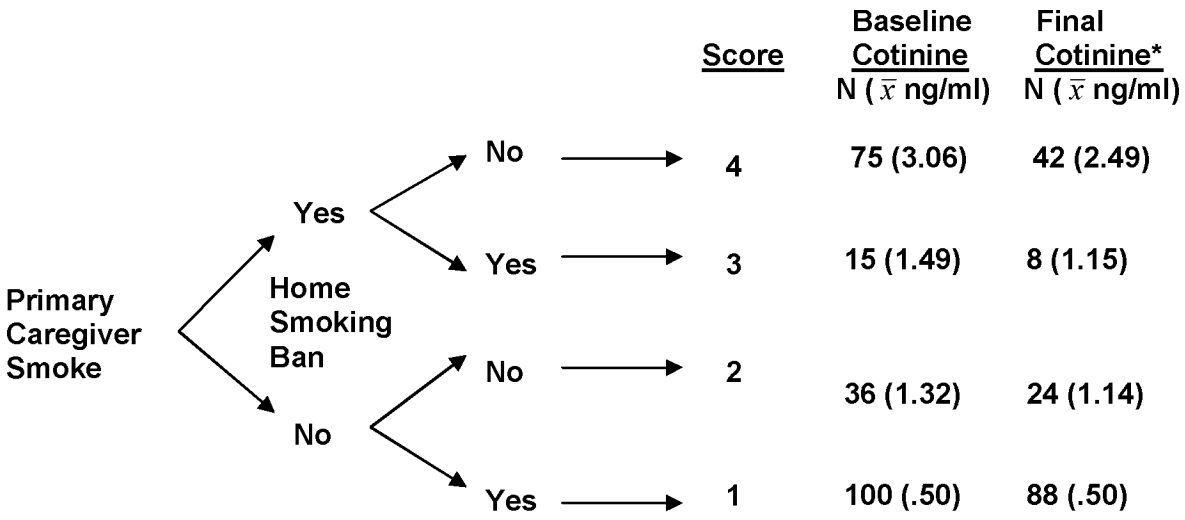

Figure 1.
Algorithm for the Index Score and Associated Mean Cotinine Values

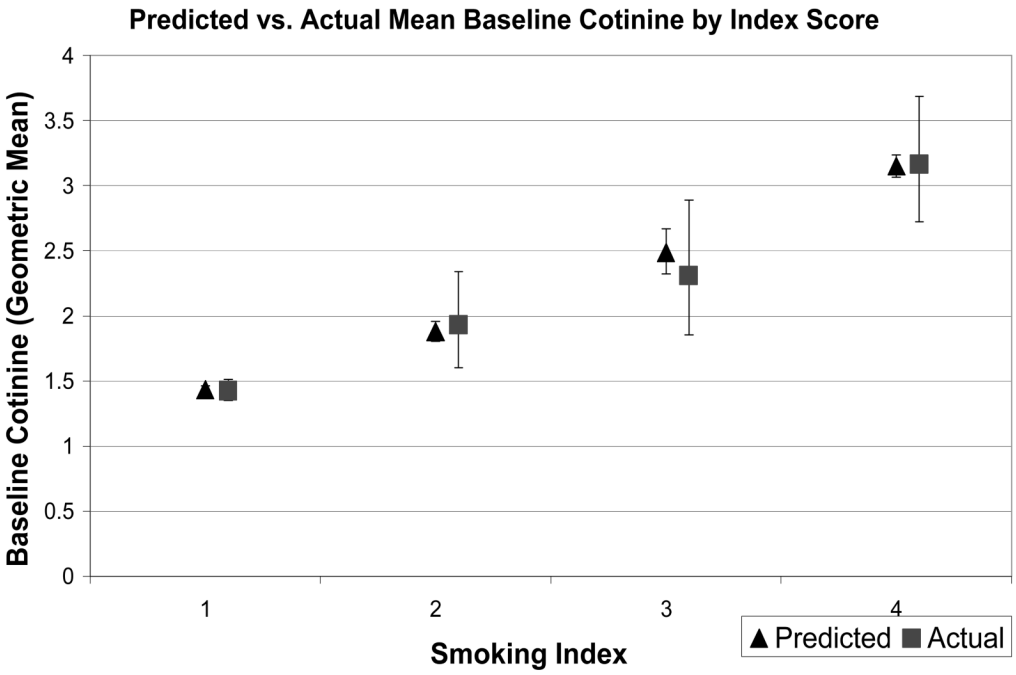

Figure 2.
Predicted and Actual Baseline Cotinine Values by Index Score.

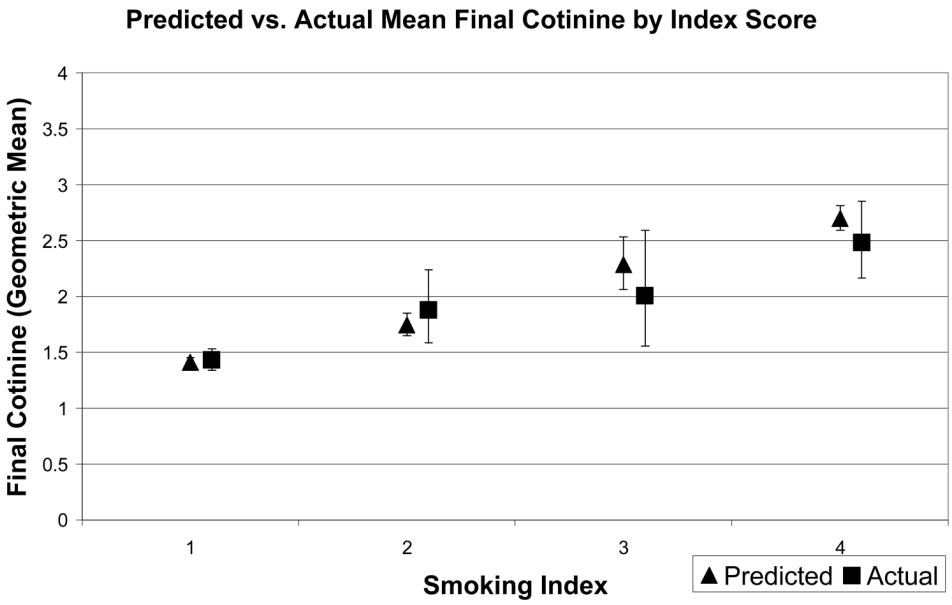

Figure 3.
Predicted and Actual Final Cotinine Values by Baseline Index Score

Table 1

Population Demographics and Mean Cotinine Values

Population Demographics	Overall N (%)	Cotinine ($\bar{\chi}$ng/ml±sd)	p-value
Child Age in years			
3–5	48 (21.2)	1.98 ± 3.71	.328
6–10	178 (78.8)	1.43 ± 2.09	
Child Gender			
Male	131 (58.0)	1.34 ± 2.05	.145
Female	95 (42.0)	1.84 ± 3.05	
Child Race			
White	24 (10.6)	2.44 ± 2.43*	
Black	147 (65.0)	1.67 ± 2.87	.019
Other	55 (24.3)	0.82 ± 0.95	
Child Ethnicity			
Hispanic	63 (27.9)	1.22 ± 1.43	.223
Non-Hispanic	163 (72.1)	1.68 ± 2.83	
Medicaid			
Yes	168 (74.3)	1.67 ± 2.58	.229
No	58 (25.7)	1.20 ± 2.36	
Parent Age			
<30	76 (33.6)	1.80 ± 3.08	.308
≥30	148 (65.5)	1.43 ± 2.20	
Parent Education			
<High school	77 (34.1)	2.01 ± 3.29	.087
≥High school	149 (65.9)	1.31 ± 1.99	
Parent Depression			
None/Mild	159 (70.4)	1.51 ± 2.56	.739
Mod/Severe	67 (29.6)	1.63 ± 2.45	
Smoker in the Home			
Yes	121 (53.5)	2.40 ± 3.15	<.001
No	105 (46.5)	0.56 ± 0.73	
Primary Caregiver is a Smoker			
Yes	89 (39.4)	2.82 ± 3.42	<.001
No	137 (60.6)	0.72 ± 1.10	
Home Smoking Ban			
Yes	115 (50.9)	0.63 ± 0.74	<.001
No	111 (49.1)	2.50 ± 3.27	
Car Smoking Ban			
Yes	146 (64.6)	1.28 ± 2.67	.033
No	80 (35.4)	2.03 ± 2.18	

Table 2

Multivariate Regression Model Predicting Baseline and Final Cotinine Values

	Baseline Index Predicting Baseline Cotinine (N=226)		Baseline Index Predicting Final Cotinine (N=162)	
	Standardized Beta	**P-value**	**Standardized Beta**	**P-value**
Child's Age	−.105	.06	−.213	.001
Child's Race				
Black	-----	-----	-----	-----
White	.050	.41	.050	.49
Other	−.079	.24	.046	.57
Child Hispanic	−.041	.54	−.180	.03
Parent Education	−.116	.04	−.174	.01
Parenting Stress	.029	.61	.036	.60
Parental Depression	.037	.52	−.039	.59
Smoking Index Score	**.552**	**<.001**	**.530**	**<.001**