

Children's Exposure to Environmental Tobacco Smoke: Using Diverse Exposure Metrics to Document Ethnic/Racial Differences

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Four metrics were used to assess exposure to environmental tobacco smoke (ETS) for a probability sample ($n = 152$) of elementary school-age children in two economically disadvantaged neighborhoods: *a*) caregiver responses to a baseline questionnaire (BQ) about smoking status and behavior; *b*) 48-hr time-activity (T-A) data on location and time spent by children in the presence of tobacco smoke; *c*) total urinary cotinine as a marker for nicotine uptake; and *d*) urinary NNAL [4-(methylnitrosamino)-1-(3-pyridyl)-1-butanol] + NNAL-Gluc [4-(methylnitrosamino)-1-(3-pyridyl)-1-(*O*- β -D-glucopyranuronosyl)butane] as a marker for uptake of the tobacco-specific lung carcinogen 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone (NNK). Consistent differences in ETS exposure by ethnicity and race were observed. Although data were insufficient to determine differences for NNAL + NNAL-Gluc, BQ responses, T-A data, and cotinine levels all indicated that average ETS exposure was highest for African-American children, moderately high for those designated "other" (white, Southeast Asian, Native American), moderately low for Hispanic children, and lowest for Somali immigrant children. For example, in February 2000, mean cotinine levels were 14.1 ng/mL for African Americans, 12.2 ng/mL for other, 4.8 ng/mL for Hispanics, and 4.4 ng/mL for Somalis. The BQ and T-A data together were reasonably good predictors of total cotinine levels (adjusted $r^2 = 0.69$), and based on limited data, measured total cotinine values were a relatively good predictor of NNAL + NNAL-Gluc (adjusted $r^2 = 0.73$). The results suggest that when children are exposed to ETS primarily in their homes, questionnaires and T-A logs might be effective screening tools for identifying those likely to experience higher uptake of nicotine. **Key words:** children's health, cotinine, environmental tobacco smoke, ethnicity, questionnaires, race. *Environ Health Perspect* 112:392–397 (2004). doi:10.1289/ehp.6473 available via <http://dx.doi.org/> [Online 12 November 2003]

Environmental tobacco smoke (ETS) is a carcinogenic mixture of more than 4,000 chemicals [National Toxicology Program 2000; U.S. Department of Health and Human Services (DHHS) 1986; U.S. Environmental Protection Agency (EPA) 1992] and a potentially important determinant of children's environmental health (Carroquino et al. 1998; Hecht et al. 2001; Landrigan et al. 1998). Although available data are inconclusive (Boffetta et al. 2000), there are legitimate concerns that childhood exposure to ETS might lead to cancer later in life (Sandler et al. 1985; Tang et al. 1999). In addition, ETS may directly harm children by exacerbating preexisting asthma (Gold 2000; Jaakkola et al. 2001) and contributing to neurodevelopmental problems (Eskenazi and Castorina 1999).

Because ETS is a common constituent of the air inside many buildings and vehicles (Cummings et al. 1990; Samet 1999; Subramaniam et al. 2001), most children are exposed to some level of ETS during their normal daily activities (Hecht et al. 2001; Scherer et al. 1999). Although there are many sources of ETS in the environment, parental smoking status (and related in-home ETS concentration) is a strongly associated with biologic markers of exposure for many children, and it

is well established that smoking prevalence rates vary by ethnicity/race, immigrant status, and country of birth (Baluja et al. 2003). Relatively little is known, however, about how these demographic variables affect the magnitude, duration, and frequency of children's exposure to ETS.

Better estimates of childhood ETS exposure, including delineation of the effects of important sociodemographic variables, are needed to help inform decisions about protection of children's environmental health. For example, accurate assessment of ETS exposure (as a causative agent, cofactor, confounder, or some combination) is necessary for ensuring the validity of most epidemiologic studies designed to investigate causal links between air pollution and children's health. Exposure misclassification errors (e.g., erroneously classifying a child as "highly exposed" or "not exposed") can introduce information bias that affects the validity of findings by either exaggerating or understating the true causal link between exposure and effect (Rothman 1986). Diverse methods are available to assess children's ETS exposure, ranging from parental questionnaires about smoking status, which are relatively easy to obtain, inexpensive to analyze, and less accurate, to biomarkers of exposure (cotinine in

urine), which are comparatively harder to collect, expensive to analyze, and more accurate (Benowitz 1999; Daisey 1999; Jarvis et al. 1984; National Research Council 1986, 1991). Given the goals of a particular study and the resources available, investigators must make trade-offs between using exposure metrics that deliver better accuracy at higher cost (e.g., biomarkers) versus those that provide less accuracy at lower cost (e.g., questionnaires).

In the School Health Initiative: Environment, Learning, Disease (SHIELD) study, children's exposure to ETS is assessed using two relatively inexpensive metrics: questionnaires about caregiver smoking status/behavior and time-activity (T-A) logs reporting the time and place of ETS exposure; and two relatively expensive metrics: measurement of urinary total cotinine [cotinine plus pyridyl-*N*- β -D-glucopyranuronosyl-(*S*)-(–)-cotininium inner salt] as an uptake marker of nicotine, and measurement of NNAL [4-(methylnitrosamino)-1-(3-pyridyl)-1-butanol] + NNAL-Gluc-[4-(methylnitrosamino)-1-(3-pyridyl)-1-(*O*- β -D-glucopyranuronosyl)butane] as an uptake marker of the tobacco-specific lung carcinogen NNK [4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone]. Associations between measures in a probability sample of elementary school-age children enrolled in SHIELD are estimated, and ethnic and racial differences in ETS exposure are examined.

Materials and Methods

Subjects and data collection. This study was approved by the University of Minnesota Research Subjects' Protection Program Institutional Review Board: Human Subjects Committee. The participating children were part of the SHIELD study, the details of

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which have been published previously (Sexton et al. 2000, 2003). Two hundred sixteen students in grades 2 through 5 (6–10 years of age) at either the Lyndale or Whittier elementary schools in south Minneapolis, Minnesota, were selected based on a stratified random sampling strategy, and eligible siblings were also allowed to participate. In the fall of 1999, children and their families eligible for SHIELD were identified and contacted based on enrollment information provided by the Student Accounting Department, Minneapolis Public Schools. After successful contact, recruiters met with children and caregivers in their homes to explain the study and answer any questions. For the 152 who agreed to be in the study, recruiters obtained

verbal and written consent, and assent as appropriate, and administered the baseline questionnaire (BQ).

The primary caregiver was asked a series of questions about smoking status and behavior, as well as questions about socioeconomic status, residential characteristics, and the child's health. For example, they were asked "In the past month how many packs of cigarettes did you smoke inside the home in the presence of the child?" and "How many smokers who live with the child smoke inside the child's home?"

During winter (January and February 2000) and spring (April and May 2000) of the next year, children were asked to give 40-mL urine samples at school. In addition, for the 48 hr preceding collection of a urine

sample, children, with the help of caregivers, interviews/translators, and field technicians, were asked to maintain a T-A log recording the location and approximate time they spent in seven different microenvironments and also to answer questions about the location and approximate time they spent in the presence of an active smoker. A summary of smoking-related data from questionnaires and logs used in SHIELD is provided in Table 1.

Laboratory analyses. Total cotinine was measured by gas chromatography–mass spectrometry, as described in previous publications (Hecht et al. 1993, 2001). Because of cost considerations, not all samples could be analyzed for NNAL and NNAL-Gluc. Because we considered 10 ng/mL cotinine to

Table 1. Summary of responses to smoking-related questions from the BQ and the T-A log (includes all smoking-related questions used in the regression models examined in this article).

Question and source	Responded yes or no > 0 ^a [Frequency (%)] ^b				
	All	African American	Somali immigrant	Hispanic	Other ^c
BQ (total no. of respondents)	150	35	39	46	30
Caregiver ever smoked inside the home	25 (22.3)	16 (46.4)	0 (0)	3 (8.5)	6 (18.4)
Current smokers in the home besides caregiver	25 (20.7)	9 (33.1)	0 (0)	9 (19.9)	7 (20.3)
Packs/day in past month by caregiver	22 (19.4)	14 (40.2)	0 (0)	3 (8.5)	5 (14.9)
Pack years smoked before past month by caregiver	22 (18.8)	14 (37.8)	0 (0)	2 (6.0)	6 (18.4)
No. of current smokers who smoke in the home besides caregiver	23 (18.2)	8 (27.4)	0 (0)	8 (17.7)	7 (20.3)
No. of days in month smoked	38 (29.4)	20 (51.9)	0 (0)	8 (20.5)	10 (29.0)
Don't know	5 (2.2)	1 (2.1)	4 (7.5)	0 (0)	0 (0)
No. of smokers inside the home in past month	35 (27.2)	20 (49.9)	0 (0)	6 (15.9)	9 (26.9)
No. of hours in home with smoker	36 (30.5)	21 (58.3)	0 (0)	6 (15.9)	9 (28.6)
Missing	2 (0.6)	0 (0)	2 (3.3)	0 (0)	0 (0)
No. of hours in vehicle with smoker	14 (14.2)	10 (32.4)	0 (0)	0 (0)	4 (12.7)
Missing	3 (1.2)	0 (0)	2 (3.3)	0 (0)	1 (2.5)
No. of hours indoors with smoker not counting the home	10 (12.5)	7 (29.7)	0 (0)	3 (9.5)	0 (0)
Missing	4 (2.0)	1 (2.3)	2 (3.3)	0 (0)	1 (2.5)
T-A log (winter; total no. of respondents)	107	23	23	38	23
Day 1—tobacco smoked in home	25 (26.8)	14 (52.7)	0 (0)	4 (12.2)	7 (26.1)
Day 2—tobacco smoked in home	21 (23.5)	11 (43.7)	0 (0)	3 (10.6)	7 (26.3)
Missing	2 (2.1)	1 (4.0)	1 (4.7)	0 (0)	0 (0)
Day 1—no. of cigarettes smoked in child's presence	26 (27.1)	14 (52.7)	1 (4.0)	5 (14.1)	6 (22.7)
Day 2—no. of cigarettes smoked in child's presence	20 (20.1)	9 (32.3)	1 (4.0)	4 (12.4)	6 (22.9)
Missing	2 (2.1)	1 (4.0)	1 (4.7)	0 (0)	0 (0)
Day 1—no. of cigars smoked in child's presence	1 (0.8)	1 (2.6)	0 (0)	0 (0)	0 (0)
Day 2—no. of cigars smoked in child's presence	1 (0.8)	1 (2.6)	0 (0)	0 (0)	0 (0)
Missing	2 (2.1)	1 (4.0)	1 (4.7)	0 (0)	0 (0)
No. of hours day 1—indoors with smoker	20 (21.6)	13 (49.2)	0 (0)	4 (12.2)	3 (9.7)
Missing	1 (1.3)	0 (0)	0 (0)	0 (0)	1 (5.2)
No. of hours day 2—indoors with smoker	17 (17.2)	10 (34.6)	0 (0)	3 (10.6)	4 (12.9)
Missing	3 (3.4)	1 (4.0)	1 (4.7)	0 (0)	1 (5.2)
No. of hours day 1—in vehicle with smoker	5 (4.0)	2 (6.6)	2 (6.6)	1 (3.0)	0 (0)
Missing	1 (1.3)	0 (0)	0 (0)	0 (0)	1 (5.2)
No. of hours day 2—in vehicle with smoker	4 (3.6)	2 (6.6)	1 (4.0)	1 (3.0)	0 (0)
Missing	3 (3.4)	1 (4.0)	1 (4.7)	0 (0)	1 (5.2)
T-A log (spring; total no. of respondents)	100	22	21	37	20
Day 1—tobacco smoked in home	20 (22.6)	14 (49.1)	0 (0)	2 (7.9)	4 (15.3)
Day 2—tobacco smoked in home	18 (20.9)	13 (46.7)	0 (0)	2 (7.9)	3 (11.6)
Day 1—no. of cigarettes smoked in child's presence	19 (21.0)	13 (45.4)	0 (0)	3 (10.0)	3 (11.6)
Day 2—no. of cigarettes smoked in child's presence	19 (21.5)	13 (46.7)	0 (0)	3 (10.0)	3 (11.6)
Day 1—no. of cigars smoked in child's presence	2 (2.0)	1 (4.1)	0 (0)	1 (2.1)	0 (0)
Day 2—no. of cigars smoked in child's presence	1 (0.6)	0 (0)	0 (0)	1 (2.1)	0 (0)
No. of hours day 1—indoors with smoker	21 (22.2)	13 (45.8)	0 (0)	5 (13.7)	3 (11.6)
Missing	1 (0.9)	1 (3.7)	0 (0)	0 (0)	0 (0)
No. of hours day 2—indoors with smoker	18 (19.5)	11 (39.4)	0 (0)	4 (12.02)	3 (11.6)
No. of hours day 1—in vehicle with smoker	4 (3.8)	1 (2.7)	0 (0)	2 (6.5)	1 (4.7)
Missing	1 (0.9)	0 (0)	0 (0)	0 (0)	1 (3.7)
No. of hours day 2—in vehicle with smoker	3 (2.9)	1 (2.7)	0 (0)	1 (4.6)	1 (3.1)

^aNumber (weighted percentage) of respondents either answering "yes" to a particular question or reporting a value > 0. ^bPercentages are weighted to adjust for selection and response probabilities. ^cIncludes white, Cambodian, Laotian, Native American, and "other."

be an indicator of potentially elevated ETS exposure, all usable samples with total cotinine ≥ 10 ng/mL as well as a selection of those with total cotinine < 10 ng/mL were analyzed for NNAL and NNAL-Gluc, using methods described previously (Hecht et al. 2001). Analysis was by gas chromatography–nitrosamine selective detection, using methods described previously (Hecht et al. 2001).

Statistical analysis and related considerations. Index children were sampled with selection probabilities designed to equally represent strata defined by school, grade, ethnicity (dichotomized as English-speaking vs. non-English-speaking homes), and sex, and analyses were weighted to account for selection and response probabilities. Race/ethnicity was broken down further for analyses addressing this factor specifically, by aggregating categories with fewer than 15 children into the “other” category. Analyses were performed on log-transformed laboratory values to normalize the distributions and to equalize variances, and transformed means were exponentiated to obtain geometric means. Values below detection limits were set to half the limit. Previous analyses showed that results are relatively insensitive to the choice of a substitute value. Confidence intervals were calculated in the transformed scale and back-transformed by taking logs.

Multiple linear regression modeling was applied to the smoking-related variables in the BQ and the T-A log to develop predictive equations for total urinary cotinine within each monitoring session, as well as for the average over both sessions in the subset of children who provided two samples. In addition, questionnaire and T-A variables, both individually and in combination with cotinine values, were used in a similar manner to predict urinary NNAL + NNAL-Gluc, but for winter only because an inadequate number of analyzed samples was available from the spring monitoring session. All possible regressions were examined, and the adjusted multiple r^2 was used as the penalized optimality

criterion because it represents an estimate of the percentage of variability explained by the regression, adjusting for the number of covariates. Some variables were not included in these models because extreme collinearity made the computations unstable. To assure applicability to the entire target population, all regressions were weighted by stratum-specific selection and response probabilities.

Use of multiple predictors can lead to optimistic estimates of the model's predictive capability. An empirical validation of the prediction equation for cotinine was performed by examining how the predicted values might perform relative to the actual values. One potential use of the measured cotinine levels would be to predict future cotinine levels for a cohort of children. With this in mind, we first examined the ability of dichotomized measured cotinine concentrations in the winter to predict dichotomized cotinine values measured in the spring using a two-by-two table. Each winter or spring cotinine value was classified by whether it was above or below 5 ng/mL, and the concordance of winter categorization with that in the spring was quantified. Next, an analogous two-by-two table was constructed based on predicted winter cotinine values (from the BQ) dichotomized above or below 5 ng/mL versus the same spring categorization. Again, the concordance of predicted winter cotinine values with measured spring values was quantified and compared with the previous table. The comparison of model-based predictors versus actual values on this simple prediction problem, summarized by the cross-product ratio, provides a measure of the validity of the model.

Results

Data from BQs were collected for 150 of the randomly selected children (referred to as “index” children) enrolled in the SHIELD study. Two urine samples, one in winter and one in spring, were obtained from 86% of these children, and 66% provided both 48-hr T-A logs (Sexton et al. 2003).

A summary of smoking-related responses on the BQ (administered at the beginning of the study) and the T-A log (completed in both the winter and spring monitoring sessions) is provided in Table 1, including a breakout by ethnic/racial group. Overall, 22% reported on the BQ that the caregiver had ever smoked inside the home, and 21% said that other occupants also smoked inside the home. Fourteen percent stated that their child had at least some exposure to ETS in vehicles, and 13% acknowledged that their child had some ETS exposure in other indoor environments besides the home. More than 20% of those completing T-A logs reported ETS exposure inside the home on both the first and second sampling day for winter and spring.

As shown in Table 1, there were substantial and consistent differences for smoking-related responses among the four ethnic/racial groups. In all instances, a considerably higher percentage of African-American children lived in a home that reported smokers (46% of caregivers smoked, 33% of other occupants smoked) and were reportedly exposed to ETS on both the first and second monitoring day in winter ($> 40\%$) and spring ($> 45\%$). Typically, children in the “other” category (white, Native American, Southeast Asian) were nominally the next most exposed group, followed by Hispanics and Somali immigrants. The evidence shows that Somali families almost never reported ETS exposure for their children on either the BQ or the T-A log.

Mean total cotinine levels by season are summarized in Table 2. Geometric mean cotinine concentrations were < 5 ng/mL for the entire cohort, as well as for each of the ethnic/racial groups, and no major seasonal differences were apparent. Geometric mean cotinine levels were substantially higher for African-American children (3.4 ng/mL in winter, 3.6 ng/mL in spring) compared with children classified as “other” (2.2 ng/mL in winter, 1.4 ng/mL in spring). Mean cotinine values for “other” children were, in turn, substantially higher than for Hispanic (0.6 ng/mL in winter and spring) and Somali immigrant children (0.7 ng/mL in winter, 0.4 ng/mL in spring). This general pattern (African American $>$ “other” $>$ Hispanic and Somali) was similar to that observed for the responses to smoking-related questions on the BQ and information derived from the T-A log (Table 1).

More detailed information on the distributions of urine cotinine by ethnic/racial group is provided in Figure 1. These data show that the general pattern of African-American $>$ “other” $>$ Hispanic and Somali immigrant children persisted for the middle percentiles. At the upper percentiles, total cotinine values began to converge, for example, for 95th percentiles: “other” (46 ng/mL)

Table 2. Mean urine total cotinine concentrations (ng/mL)^a for SHIELD children^b by ethnicity and race.

Season	All SHIELD children	African American	Somali immigrant	Hispanic	Other ^c
February					
No. of children	113	25	29	33	26
Mean cotinine (ng/mL) ^d	9.9	14.1	4.4	4.8	12.2
Geometric mean (ng/mL) ^d	1.6	3.4	0.7	0.6	2.2
95% CI ^d	1.0–2.6	1.2–10.2	0.4–1.5	0.3–1.2	0.9–5.8
May					
No. of children	86	24	19	21	22
Mean cotinine (ng/mL) ^d	9.5	15.2	0.8	7.2	7.2
Geometric mean (ng/mL) ^d	1.5	3.6	0.4	0.6	1.4
95% CI ^d	0.9–2.6	1.2–11.1	0.2–0.7	0.3–1.4	0.5–3.9

^aSamples below the detection limit were assigned a value of 0.2 ng/mL, which is half of the minimum detectable level.

^bIncludes all randomly selected (index) children with at least one urine sample in either the winter or spring. ^cIncludes children classified as white, Cambodian, Laotian, Native American, and other. ^dMeans and percentages are weighted to account for selection and response probabilities.

> African American (38 ng/mL) > Hispanic (35 ng/mL) > Somali immigrant (30 ng/mL). However, estimates of extreme percentiles are less stable, so this ordering may be arbitrary.

A summary of mean cotinine concentrations by season is provided in Table 3 for the stratification variables used to select the SHIELD probability sample. Cotinine levels were relatively constant across season, except for children in the third (2.2 ng/mL in winter, 0.8 ng/mL in spring) and fifth (1.5 ng/mL in winter, 2.8 ng/mL in spring) grades and children enrolled at the Lyndale school (3.0 ng/mL in winter, 1.5 ng/mL in spring). Mean cotinine values tended to be higher *a*) in girls than in boys (1.9 vs. 1.4 ng/mL in winter, 1.9 vs. 1.2 ng/mL in spring), *b*) for students at Lyndale than for those at Whittier in the winter (3.0 vs. 1.0 ng/mL), and *c*) for children from English-speaking compared with those from non-English-speaking families (3.0 vs. 0.9 ng/mL in winter, 2.9 vs. 0.6 ng/mL in spring).

Regression models were used to validate responses to smoking-related questions on the BQ, information from the T-A log, or a combination of the two as predictors of urine cotinine concentrations. The estimates of proportion of explained variance (r^2), adjusted for the number of covariates, are compared in

Table 4 for the models tested. Individually, both the BQ and T-A log predicted cotinine with reasonable reliability (adjusted $r^2 > 0.45$). The BQ was about as good as the T-A log, even though the BQ was administered several weeks before sample collection, whereas the T-A log was completed during the 2 days preceding sample collection. In combination, the BQ and T-A log were better yet. For example, the BQ plus the T-A log (winter and spring) explained 69% (adjusted r^2) of the variance in average (winter and spring) urine cotinine levels compared with 58% for the BQ alone and 66% for the T-A log alone.

We also examined whether concentrations of NNAL + NNAL-Gluc measured in urine could be predicted by total cotinine values or a combination of total cotinine values and either the BQ or T-A log (Table 4). Because relatively few urine samples (< 20) were analyzed for NNAL + NNAL-Gluc from the spring (Hecht et al. 2001), there were insufficient degrees of freedom available to run the full regression model. Therefore, only results from the winter monitoring session are summarized in Table 4. Findings indicate that total cotinine in the winter was a reasonably good predictor (adjusted $r^2 = 0.73$) of measured NNAL + NNAL-Gluc in the winter. A combination of either total cotinine plus BQ smoking variables

(adjusted $r^2 = 0.65$) or total cotinine plus T-A log smoking variables (adjusted $r^2 = 0.33$) was less effective.

Several previous studies suggest that subjects with urinary cotinine values less than about 5 ng/mL are typically unaware of any ambient ETS exposure (Cummings et al. 1990; Jarvis et al. 1984; Pirkle et al. 1996). Consequently, despite the presence of cotinine in their urine, they would be unlikely to report ETS exposure, presumably because they were

Table 3. Mean urine total cotinine concentrations (ng/mL) by season for SHIELD stratification variables.

	Cotinine concentration (ng/mL) ^a	
	February	May
Sex		
Male		
No.	61	43
Mean	9.0	5.6
Geometric mean	1.4	1.2
95% CI	0.8–2.5	0.6–2.2
Female		
No.	52	43
Mean	11.1	13.4
Geometric mean	1.9	1.9
95% CI	0.8–4.4	0.8–4.8
Grade 2		
No.	31	23
Mean	8.3	8.1
Geometric mean	1.2	1.1
95% CI	0.5–3.3	0.3–3.5
Grade 3		
No.	26	21
Mean	13.8	6.4
Geometric mean	2.2	0.8
95% CI	0.7–6.9	0.3–2.3
Grade 4		
No.	27	20
Mean	11.8	9.4
Geometric mean	2.0	2.4
95% CI	0.8–5.0	0.9–6.7
Grade 5		
No.	29	22
Mean	6.74	14.4
Geometric mean	1.5	2.8
95% CI	0.6–3.6	0.9–8.8
Lyndale school		
No.	53	49
Mean	14.1	7.5
Geometric mean	3.0	1.5
95% CI	1.6–5.8	0.8–2.7
Whittier school		
No.	60	37
Mean	6.6	11.6
Geometric mean	1.0	1.5
95% CI	0.5–1.9	0.6–3.9
English-speaking		
No.	38	37
Mean	13.7	13.1
Geometric mean	3.0	2.9
95% CI	1.3–7.0	1.2–7.0
Non-English-speaking		
No.	75	49
Mean	6.1	4.7
Geometric mean	0.9	0.6
95% CI	0.5–1.4	0.4–1.0

^aSamples below the detection limit were assigned a value of 0.2 ng/mL, which is half of the minimum detectable level. Results are weighted to adjust for selection and response probabilities.

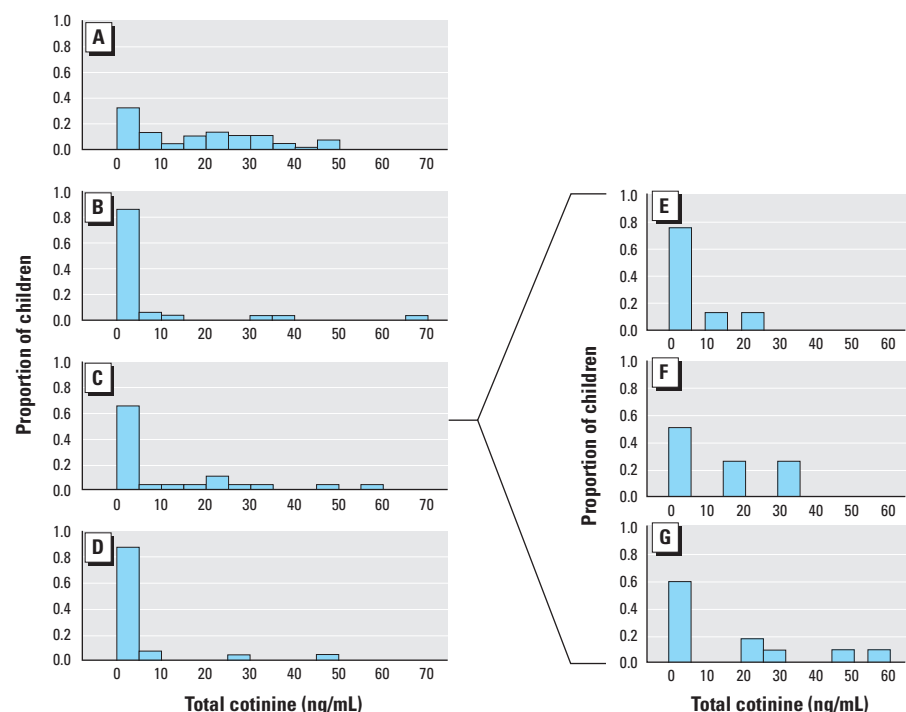


Figure 1. Proportion of children with urine cotinine (ng/mL) for SHIELD children by ethnicity or race [includes all randomly selected (index) children with at least one urine sample in either the winter or spring]. The average cotinine concentration was used for children with two samples. Median values are weighted to adjust for selection and response probabilities. Samples below the detection limit were assigned a value of 0.2 ng/mL, which is half of the minimum detectable level. (A) African-American children ($n = 31$, median = 9.0). (B) Hispanic children ($n = 41$, median = 0.2). (C) "Other" children ($n = 29$, median = 2.2). (D) Somali immigrant children ($n = 33$, median = 0.2). The "other" category (C) includes (E) Asian-Cambodian ($n = 8$, median = 0.9), (F) Asian-Laotian ($n = 4$, median = 19.3), and (G) white ($n = 12$, median = 1.5).

oblivious to inhalation of low levels during routine activities. Using this 5-ng/mL “awareness threshold” as a cutoff, we compared measured cotinine concentrations (< 5 ng/mL vs. ≥ 5 ng/mL) in winter and spring (Table 5) and found that children who had urine cotinine values < 5 ng/mL in the winter also tended to have values < 5 ng/mL in the spring, and vice versa (misclassification error: $8/65 = 0.12$). Then we compared measured cotinine concentrations (< 5 ng/mL and ≥ 5 ng/mL) in the spring versus predicted winter values from the BQ full regression model. The results (Table 6) show that the smoking variables from the BQ did almost as well at predicting spring cotinine values (misclassification error: $9/63 = 0.14$).

Discussion

This article builds on and expands earlier findings among SHIELD children. We focus here on comparing results using four different exposure metrics to assess children's exposure to ETS: BQ, T-A log, measured urinary levels of total cotinine, and measured urinary levels of NNAL + NNAL-Gluc. All four metrics suggest that a substantial fraction of elementary school-age children from two economically disadvantaged and ethnically diverse neighborhoods were routinely exposed to ETS. Moreover, three of the exposure metrics (BQ, T-A log, cotinine) indicated a consistent ethnic/racial pattern for childhood ETS exposure, with African American $>$ “other” (Southeast Asian, white, Native American) $>$ Hispanic $>$ Somali immigrant. The BQ and T-A log data, although not conclusive, suggest that the home environment (e.g., caregiver or others smoking indoors in the presence of the child) was the primary source of ETS exposure for children in all ethnic/racial groups.

There was comparatively little difference in the ability of the BQ and the T-A log to

predict urinary cotinine levels, with both doing a reasonably good job (adjusted $r^2 > 0.45$). A combination of the BQ and T-A log did better than either alone (adjusted $r^2 = 0.69$ for average cotinine). Because of the relatively short half-life of nicotine in the body, one might logically expect the T-A log (which covers the 48 hr preceding urine collection) to be a better predictor of urinary cotinine. The observed results (BQ $\approx$ T-A log) are consistent with a scenario wherein children are exposed to ETS primarily at home, the time they spend at home in proximity to smokers is relatively constant, and the number of smokers in the home and the amount they smoke in the child's presence are relatively stable over time. This scenario is also compatible with the fact that children whose urinary cotinine concentrations were below (or above) the “awareness threshold” (5 ng/mL) in winter were also likely to be below (or above) that value in the spring.

Although fewer urine samples were analyzed for NNAL + NNAL-Gluc, it was possible to use data from the winter monitoring session to examine statistical relationships between these biomarkers of NNK and cotinine as well as smoking-related variables. For this limited data set (winter only), urinary cotinine did a reasonably good job of predicting NNAL + NNAL-Gluc (adjusted $r^2 = 0.73$). However, a combination of cotinine plus the smoking variables from either the BQ or T-A log was less effective in predicting NNAL + NNAL-Gluc.

When choosing an appropriate metric to assess childhood ETS exposure, it is necessary to evaluate the relative costs and benefits of various indicators within the context of study objectives and resource constraints. Although the SHIELD study was not expressly designed to compare the BQ and the T-A log, there were some obvious differences in logistics.

The BQ was administered once at the beginning of the first year of SHIELD, whereas the T-A log was collected twice, once in each monitoring session. The sole respondent to the BQ was the child's caregiver, which made it comparatively easy to administer. Maintaining an accurate 48-hr T-A log of a child's activities was a challenging undertaking that often required the combined efforts of the child, parents, and field staff.

Urine cotinine is commonly considered the most direct and therefore the best indicator of ETS exposure. But collecting urine from children is always challenging, and laboratory analysis of large numbers of samples can be expensive. Moreover, there may be more interest in measuring a urinary biomarker (NNAL + NNAL-Gluc) for the uptake of a tobacco-specific carcinogen (NNK) rather than one for nicotine uptake (cotinine), for example, as in a childhood cancer study. Although more urine is needed from exposed individuals for analysis of NNAL + NNAL-Gluc (10–20 mL vs. 1–2 mL for cotinine), the costs of collecting the requisite urine samples are similar. The costs of laboratory analysis for NNAL + NNAL-Gluc are, however, 3- to 4-fold higher than for total cotinine.

In summary, two major findings from the data are presented here. First, there were apparent ethnic/racial differences in children's exposure to ETS in two economically disadvantaged neighborhoods. Based on multiple exposure indicators (BQ, T-A logs, total urinary cotinine), a clear and consistent pattern emerged: African-American children tended to have the highest exposure, children

Table 4. Comparison of regression models (various self-reported ETS exposure metrics versus urine total cotinine or NNAL + NNAL-Gluc) using proportion of explained variance (r^2).

Model	No.	p-Value	Adjusted r^2
BQ vs. urine total cotinine			
BQ smoking variables vs. average cotinine ^a	63	< 0.0001	0.58
BQ smoking variables vs. cotinine (spring)	83	< 0.0001	0.49
BQ smoking variables vs. cotinine (winter)	110	< 0.0001	0.49
T-A log vs. urine total cotinine			
T-A smoking variables (winter + spring) vs. average cotinine ^a	42	< 0.0001	0.66
T-A smoking variables (spring) vs. cotinine (spring)	64	< 0.0001	0.50
T-A smoking variables (winter) vs. cotinine (winter)	83	< 0.0001	0.46
Combination of BQ and T-A log vs. urine total cotinine			
BQ and TA (winter + spring) vs. average cotinine ^a	41	< 0.0001	0.69
BQ and TA (spring) vs. cotinine (spring)	62	< 0.0001	0.66
BQ and TA (winter) vs. cotinine (spring)	61	< 0.0001	0.54
BQ and TA (winter) vs. cotinine (winter)	80	< 0.0001	0.48
Urine total cotinine vs. urine NNAL + NNAL-Gluc			
Cotinine (winter) vs. NNAL + NNAL-Gluc (winter)	44	< 0.0001	0.73
BQ and cotinine vs. urine NNAL + NNAL-Gluc			
BQ smoking variables + cotinine (winter) vs. NNAL + NNAL-Gluc (winter)	42	< 0.0001	0.65
T-A log and cotinine vs. urine NNAL + NNAL-Gluc			
T-A smoking variables + cotinine (winter) vs. NNAL + NNAL-Gluc (winter)	30	0.023	0.33

^aAverage of urine cotinine from winter and spring monitoring sessions.

Table 5. Association of measured winter cotinine with measured spring cotinine [n (%)], when both are dichotomized as urine cotinine < 5 ng/mL versus ≥ 5 ng/mL.

Winter cotinine	Spring cotinine		Total
	< 5 ng/mL	≥ 5 ng/mL	
< 5 ng/mL	38 (58.46)	1 (1.54)	39 (60.00)
≥ 5 ng/mL	7 (10.77)	19 (29.23)	26 (40.00)
Total	45 (69.23)	20 (30.77)	65 (100.00)

Misclassification error: $8/65 = 0.12$.

Table 6. Association of predicted winter cotinine (from BQ regression model) with measured spring cotinine [n (%)], when both are dichotomized as urine cotinine < 5 ng/mL versus ≥ 5 ng/mL.

Predicted winter cotinine ^a	Spring cotinine		Total
	< 5 ng/mL	≥ 5 ng/mL	
< 5 ng/mL	38 (60.32)	4 (6.35)	42 (66.67)
≥ 5 ng/mL	5 (7.94)	16 (25.40)	21 (33.33)
Total	43 (68.25)	20 (31.75)	63 (100.00)

Misclassification error: $9/63 = 0.14$.

^afrom BQ regression model.

classified as "other" (white, Southeast Asian, Native American) tended to be intermediate, and Hispanic and Somali immigrant children typically had the lowest exposure. Second, both the BQ and T-A log did a reasonably good job of predicting urine total cotinine levels, and measured urine total cotinine levels were a comparatively good predictor of urinary NNAL + NNAL-Gluc, based on analysis of a relatively small number of samples. Our results demonstrate *a*) the importance of considering differences in smoking prevalence by ethnicity and race when conducting children's ETS exposure studies, *b*) the value of measuring biomarkers of uptake for accurate assessment of children's exposure to ETS, and *c*) the potential worth of questionnaires and T-A logs as screening tools or adjunct exposure metrics.

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