Exposure to environmental tobacco smoke (ETS) is a major cause of morbidity and mortality among U.S. children. Despite African-American children’s having a lower reported exposure to tobacco compared to whites, they suffer disproportionately from tobacco-related illnesses and have higher levels of serum cotinine than white children. The goal of this study was to test whether African-American children have higher levels of serum and hair cotinine, after accounting for ETS exposure and various housing characteristics. We investigated the level of cotinine in both hair and serum in a sample of 222 children with asthma. Using a previously validated survey for adult smokers, we assessed each child’s exposure to ETS. We collected detailed information on the primary residence, including home volume, ventilation, and overall home configuration. Despite a lower reported ETS exposure, African-American children had higher mean levels of serum cotinine (1.41 ng/mL vs. 0.97 ng/mL; \( p = 0.03 \)) and hair cotinine (0.25 ng/mg vs. 0.07 ng/mg; \( p < 0.001 \)) compared with white children. After adjusting for ETS exposure, housing size, and other demographic characteristics, serum and hair cotinine levels remained significantly higher in African-American children (\( \beta = 0.34, p < 0.03 \)) than in white children (\( \beta = 1.06, p < 0.001 \)). Housing volume was significantly associated with both serum and hair cotinine but did not fully explain the race difference. Our results demonstrate that, despite a lower reported exposure to ETS, African-American children with asthma had significantly higher levels of both serum and hair cotinine than did white children. Identifying causes and consequences of increased cotinine may help explain the striking differences in tobacco-related illnesses.

Environmental tobacco smoke (ETS) is a major cause of morbidity and mortality among children. ETS increases the risk of sudden infant death syndrome (SIDS), otitis media, lower respiratory tract infections, and asthma (Cook and Strachan 1999; Larsson et al. 2001). Furthermore, ETS contains known carcinogens, such as polycyclic aromatic hydrocarbons and 4-aminobiphenyl, which react with DNA and proteins to form adducts (Sexton et al. 2004; Tang et al. 1999). These compounds have been associated with the development of cancer (Perera et al. 2002; Tang et al. 2001). Data from the National Health Interview Survey indicate that regular smoking occurs in 36% of homes in which children reside, an estimate that far exceeds the Healthy People 2010 goal of reducing the percentage of children exposed to ETS to ≤ 10% [Schuster et al. 2002; U.S. Department of Health and Human Services (DHHS) 2000].

There is a disparity between the reported level of tobacco use and tobacco-associated outcomes among African Americans. Despite lower levels of reported tobacco use and exposure than among whites, African-American adults and children experience significantly higher levels of tobacco-related morbidity and mortality [Centers for Disease Control and Prevention (CDC) 1998], African-American smokers experience significantly higher rates of smoking-related cancers when compared with white smokers, even though they report smoking fewer cigarettes per day (Caraballo et al. 1998, 2004; CDC 1998). African-American children experience higher rates of low birth weight, SIDS, and asthma, even though their reported exposure to ETS is less than that of white children. Although this paradox is not completely understood, many investigators hypothesize that racial differences in the metabolism of tobacco toxins may explain these striking differences in tobacco-related morbidity and mortality (Ahijevych and Garrett 2004; Ahijevych et al. 2002; Benowitz et al. 1999, 2004; Clark et al. 1996a; Mannino et al. 2001b; Perez-Stable et al. 1998; Tang et al. 1999).

Surprisingly, studies show that, despite lower levels of reported tobacco use compared with white smokers, African-American smokers have higher levels of some biologic markers of tobacco exposure. Until recently, most studies have relied on self-report to assess tobacco exposure. Increasingly, studies are incorporating biomarkers to objectively assess tobacco exposure (Al-Delaimy et al. 2001; Caraballo et al. 1998, 2004; Hecht et al. 2001; Klein and Koren 1999; Knight et al. 1996; Mannino et al. 2002, 2003). The most widely used biomarker is cotinine, which is a relatively stable product of nicotine metabolism. In laboratory experiments that controlled for tobacco smoke exposure, African Americans had serum cotinine levels that were 32–45% higher than those of whites (Benowitz et al. 1999, 2002; Perez-Stable et al. 1998). In a nationally representative sample, African-American smokers had significantly higher serum cotinine levels compared with white smokers, even though they reported smoking fewer cigarettes (Caraballo et al. 1998). However, the data for children and ETS exposure, rather than actual tobacco use, are more limited. In one Canadian study, black children had higher levels of urine and hair cotinine than did white or East Indian children, despite a lower reported home ETS exposure (Knight et al. 1996). In contrast, Mannino et al. (2001a) found no significant racial differences in serum cotinine among ETS-exposed children. Few studies involving children have systematically examined how key factors such as housing size, housing ventilation, and out-of-home exposure might influence the relationship between race, reported ETS, and cotinine (Henschel et al. 1997). Smaller housing size, for example, could be more common among African-American children and thus concentrate their exposure to ETS and increase cotinine levels.

The goal of the present study was to test whether African-American children with asthma have higher serum and hair cotinine levels compared with white children with asthma, even after accounting for reported ETS exposure both inside and outside of the home as well as important housing characteristics such as home volume and home ventilation.

**Materials and Methods**

*Study design and subjects.* Data for this study were drawn from the Cincinnati Asthma Prevention (CAP) study. The CAP study is an ongoing double-blind, placebo-controlled trial, designed to test the efficacy of reducing ETS exposure to children with asthma.

**Address correspondence to S.E. Wilson, Division of General Internal Medicine, University of Cincinnati, French-East, Suite 275, 3202 Eden Ave., Cincinnati, OH 45267-0840 USA. Telephone: (513) 558-2763. Fax: (513) 558-2744. E-mail: Stephen.Wilson@uc.edu.**

We thank R. Hornung and A. Leonard for their helpful comments.

This work was supported by funding from a National Research Service Award (T32PE10027), National Heart Lung and Blood Institute (R01 HS65731), and National Institute of Child Health and Development (K23 HD40362).

The authors declare they have no competing financial interests.

Received 2 July 2004; accepted 9 December 2004.
exposure using carbon-permanganate-zeolite (CPZ) high-efficient particulate air (HEPA) cleaners among children with asthma. We used the baseline data of the CAP study for our analysis. We identified potential subjects by using medical records and billing information from a large tertiary care center and a regional managed care organization, yielding subjects from urban, suburban, and rural communities. After notifying the child’s health care provider, we contacted the family by mail to describe the study in detail. Families that were interested in participating were contacted by telephone to determine their eligibility, and invited to participate in the trial. Eligibility criteria included physician diagnosis of asthma, exposure to five or more cigarettes per day in or around the home, electricity in the home, and no plans to move within the next 12 months. We excluded subjects who had a coexisting chronic lung disease, congenital heart disease, neuromuscular disease, or mental retardation.

**ETS reported exposure measure.** We adapted a questionnaire previously validated for adult smokers to systematically assess reported ETS exposure both in and outside the home (Coghlin et al. 1989). The primary caregiver reported the number of cigarettes smoked per day in the home by each resident of the household as well as the number of cigarettes smoked by regular visitors to the home. Using these data, we calculated the average number of cigarettes smoked per day in or around the home. For the previous day, we asked the caregiver to recall the number of hours the child spent inside the home and to estimate the level of ETS exposure in locations outside of the home. We also asked the parents whether their child had been exposed to tobacco smoke in a motor vehicle, a room, type of heating system, presence of an electronic tape measure and documented the date of each home visit and the airflow in and around the home. We determined longer-term exposure to ETS using hair cotinine. Cotinine enters the hair shaft through the hair-bulb blood supply, thus reflecting the average concentration in blood over a longer period of time (Al-Delaimy 2002; Al-Delaimy et al. 2002). Approximately 10 shafts of hair were cut at the root from the occipital region of each child. These hair samples were washed and dried with a mild detergent. Cotinine was extracted from the hair using sodium hydroxide. This solution was neutralized using hydrochloric acid. Cotinine concentrations were determined using radio-immunoassay as described by Klein and colleagues (Eliopoulos et al. 1994; Klein and Koren 1999). In this study, serum cotinine and hair cotinine were moderately correlated (r = 0.54, p < 0.001).

**Housing characteristics.** We collected detailed information about the home environment. An environmental technician visited the home, measured the total volume using an electronic tape measure and documented the age and general condition of the residence. The technician collected detailed information on the type and condition of the floors in each room, type of heating system, presence of an air conditioner or fans, and the overall design of the unit. During the home visit, we measured the level of particulate matter < 5 μm in diameter (PM5) using a Greentek-321 particle monitor (Met One Instruments, Inc., Grants Pass, OR). Particulate matter is highly correlated with air nicotine and thus can be used as an indicator of ETS (Cains et al. 2004).

**Race and sociodemographic covariates.** Primary caregivers were given a list of seven racial categories (African American or black, white, Asian or Asian American, Asian Indian, Native American, Native Hawaiian/Pacific Islander, Middle Eastern) along with Hispanic/Latino ethnicity from which to select the categories that best described their child. Parents were instructed to select as many of the categories as they deemed appropriate. Because there were few subjects in other racial and ethnic categories, only those children reported to be African American or white were included in our analysis. Children who were described as both African American and white were categorized as mixed-race subjects. We performed a sensitivity analysis with mixed-race subjects included with African-American subjects and then with white subjects to determine whether there were any differences. Additional measured covariates included insurance status, household income, parental education, parental marital status, and maternal depressive symptoms. Maternal depressive symptoms were assessed with the Beck Depression Inventory (Beck et al. 1996). We used a cutoff score of ≥ 17 to indicate moderate to severe levels of depressive symptoms.

**Statistical analysis.** Serum and hair cotinine levels were both highly skewed. Thus, we log-transformed these variables before conducting any analyses. We compared subject characteristics using t-tests for continuous variables and chi-square tests for categorical variables. All values for cotinine are presented as geometric means. Using bivariate analysis, we compared a number of environmental characteristics between the two racial groups. We examined these factors to determine their association with both serum and hair cotinine. To account for seasonal variation, we documented the date of each home visit and grouped them into a particular season. Seasons were assigned using the following definitions: winter (January–March), spring (April–June), summer (July–September), fall (October–December). We compared these means using analysis of variance. We created a series of linear regression models including those factors that were associated with race and cotinine at a p-value ≤ 0.25. The first model contained only the race variable (model 1). Subsequent models included both race and one or more covariates. From this series of models, we selected factors that changed the race estimate by at least 5–10% and these factors were included in the final model. Although age, sex, and season of the year did not meet our definition of confounding, we included them in our final analysis for face validity of our model. We tested for effect modification by introducing a race–ETS product term in our multivariable regression models. We assessed the residuals of the final models for normality. All analyses were completed in SAS version 8.2 (SAS Institute Inc., Cary, NC).

**Results**

Of the 222 children we selected for this study, 52% were described as African American, 45% were described as white, and 3% were described as African American and white. African-American children were slightly older than white children, but there were no significant differences in sex or parental education by race (Table 1). African-American children were more likely to reside in single-parent and low-income households and to have public insurance compared with white children. African-American children were reportedly exposed to fewer cigarettes per day in or around the home than white children. Despite this lower reported exposure to ETS, African-American children had significantly higher levels of both serum and hair cotinine (Table 2).
Table 1. Demographic characteristics of children in the CAP study, by race.

| Characteristic            | Total (n = 222) | White (n = 97) | African American (n = 125) | p-Valuea |
|---------------------------|-----------------|---------------|---------------------------|----------|
| Age (years, mean ± SD)    | 8.6 ± 1.8       | 8.4 ± 1.7     | 8.8 ± 1.7                 | 0.046    |
| Sex (%)                   |                 |               |                           |          |
| Female                    | 38.3            | 37.1          | 39.2                      | 0.75     |
| Parental education (%)    |                 |               |                           |          |
| Less than high school     | 18.5            | 19.6          | 17.6                      | 0.67     |
| High school graduate      | 46.9            | 42.3          | 50.4                      |          |
| Some college              | 23.0            | 24.7          | 21.6                      |          |
| College graduate          | 11.7            | 13.4          | 10.4                      |          |
| Parental married status (%)|               |               |                           |          |
| Married                   | 37.8            | 63.9          | 17.6                      | < 0.001  |
| Divorced                  | 11.7            | 18.6          | 6.4                       |          |
| Single never married      | 41.9            | 11.3          | 85.6                      |          |
| Separated/widowed         | 8.6             | 6.2           | 10.4                      |          |
| Household income (%)      |                 |               |                           |          |
| < $20,000                 | 41.4            | 27.8          | 52.0                      | < 0.001  |
| $20,000–$40,000           | 30.2            | 25.8          | 33.6                      |          |
| > $40,000                 | 24.3            | 41.2          | 11.2                      |          |
| Missing                   | 4.1             | 5.2           | 3.2                       |          |
| Insurance status (%)      |                 |               |                           |          |
| Private insurance         | 42.8            | 67.0          | 24.0                      | < 0.001  |
| Public insurance          | 51.4            | 27.8          | 69.6                      |          |
| Uninsured                 | 5.9             | 5.2           | 6.4                       |          |
| Maternal depression (%)b  | 27.5            | 23.7          | 30.4                      | 0.27     |
| Season of visit (%)       |                 |               |                           |          |
| Winter                    | 27.5            | 25.7          | 28.8                      | 0.47     |
| Spring                    | 27.5            | 26.8          | 28.0                      |          |
| Summer                    | 28.8            | 26.8          | 30.4                      |          |
| Fall                      | 16.2            | 20.6          | 12.8                      |          |

*p < 0.05.

*Comparison of African-American versus white children using chi-square or t-test as appropriate.

Table 2. ETS exposure, cotinine, and housing characteristics by race.

| Covariates | Total | White | African American | p-Valuea |
|------------|-------|-------|------------------|----------|
| Home ETS exposure (cigarettes/day) | 16.5 (14.9–18.1) | 18.7 (16.3–21.0) | 14.9 (12.8–14.9) | 0.013 |
| Serum cotinine (ng/mL)b | 1.2 (1.01–1.42) | 0.97 (0.74–1.27) | 1.41 (1.14–1.75) | 0.03 |
| Hair cotinine (mg/g)c | 0.14 (0.12–0.17) | 0.07 (0.06–0.09) | 0.25 (0.20–0.31) | < 0.001 |
| PM2.5 (µg/m³)d | 3.791 (3.328–4.318) | 4.634 (3.842–5.580) | 3.237 (2.716–3.857) | 0.007 |
| Home volume (m³)e | 226 (215–241) | 249 (225–272) | 212 (198–226) | 0.01 |
| Time at home (hr)f | 16.8 (16.2–17.4) | 16.0 (15.0–17.0) | 17.3 (16.6–18.1) | 0.04 |
| Car ETS exposure (%) | 25.7 | 34.0 | 19.2 | < 0.001 |
| Air conditioner (%) | 71.6 | 82.4 | 63.2 | 0.0016 |
| Fan in home (%) | 36.0 | 46.4 | 36.0 | 0.005 |
| Open floor plan (%) | 5.0 | 3.1 | 6.4 | 0.26 |
| Public ETS exposure (%) | 3.6 | 5.2 | 2.4 | 0.27 |
| Carpet in bedroom (%) | 95.1 | 92.8 | 79.2 | 0.0048 |
| Carpet in main room (%) | 76.6 | 82.5 | 63.2 | 0.0016 |

*p < 0.05.

*Comparison of African American versus white using chi-square or t-test as appropriate.

When stratified by reported level of ETS exposure, African-American children had higher levels of serum and hair cotinine (Figures 1 and 2). Lower levels of PM2.5 in African-American homes offered some confirmation of their lower reported exposure to ETS. Among the factors that might influence ETS exposure, we found that African-American children had smaller home volumes, were less likely to have fans or an air conditioner, and were less likely to be exposed to ETS in a car.

In bivariate analyses, total home volume, ETS exposure in motor vehicles, and the presence of an air conditioner were significantly associated with increased serum cotinine (Table 3). Maternal depression was also associated with increased serum cotinine. Home volume, the presence of an air conditioner, and were less likely to be exposed to ETS in a car.

In a multivariable analysis, African-American race was independently associated with serum cotinine (Table 4). In model 1, we included only the race term and found that African-American race was associated with increased serum cotinine. After adding reported ETS exposure as displayed in model 2, the race coefficient increased, indicating that African-American children had higher serum cotinine levels despite a lower reported exposure. In model 3, we added the variable for ETS exposure in a motor vehicle to the model. The race estimate increased, indicating that the racial differences in cotinine were increased because fewer African-American children were exposed to ETS in a motor vehicle. In contrast, after accounting for the smaller home volume among African-American children, the race estimate decreased. Still, African-American race was still significantly associated with serum cotinine. We found a similar pattern of results between African-American race and hair cotinine after adjusting for covariates (Table 5). The race–ETS interaction was not significant for either serum or hair cotinine.

**Discussion**

Our results demonstrate that, despite lower reported exposure to ETS, African-American children have significantly higher levels of both serum and hair cotinine. These findings were partially explained by smaller home sizes among African-American children. Still, the racial differences in cotinine persisted after accounting for housing characteristics and exposures that occurred outside the home. Consistent with this study, Knight et al.
(1996) similarly found that black children had significantly higher levels of cotinine compared with white children, despite lower ETS exposure. In contrast, Mannino et al. (2001a) found no differences by race in a cohort of tobacco-exposed children. However, neither study accounted for housing characteristics or ETS exposure outside the home. Our study clearly demonstrates that even after accounting for reported exposure and potential modifying environmental factors, African-American children have significantly higher levels of both serum and hair cotinine.

There are at least two possible explanations for why African Americans may have higher levels of cotinine. One explanation could be a racial difference in their metabolism of tobacco-related products. A number of recent studies have found that African-American smokers metabolize nicotine and cotinine more slowly than do white smokers. Perez-Stable and colleagues infused deuterium-labeled nicotine and cotinine into subjects and monitored for nicotine and cotinine clearances (Benowitz et al. 1999, 2004; Perez-Stable et al. 1998). They found that African-American subjects had a higher total clearance and nonrenal clearance of cotinine compared with white subjects. They also found that white and African-American women who smoked mentholated cigarettes had higher levels of serum cotinine compared than did white women who smoked nonmentholated tobacco products. Because no African-American women smoked nonmentholated products, this group could not be assessed. Although these studies demonstrate an effect of menthol on nicotine and cotinine metabolism, they were all completed in adult smokers. It is not clear whether this relationship between menthol and cotinine exists among children exposed to ETS.

Although racial differences in cotinine were present for hair and serum, the relative racial difference was greater for hair cotinine. The larger racial difference for hair cotinine

| Covariates                  | Serum cotinine (ng/mL) | p-Value | Hair cotinine (ng/mg) | p-Value |
|-----------------------------|------------------------|---------|----------------------|---------|
| Correlation coefficients    |                        |         |                      |         |
| Age                         | −0.039                 | 0.56    | 0.31                 | 0.66    |
| Home volume (m³)            | −0.370                 | < 0.0001| −0.287              | < 0.0001|
| Time at home (hr)           | −0.047                 | 0.49    | 0.192               | 0.79    |
| Geometric means for cotinine|                        |         |                      |         |
| Car ETS exposure            |                        |         |                      |         |
| No                          | 1.05                   | 0.01    | 0.14                | 0.76    |
| Yes                         | 1.75                   |         | 0.15                |         |
| Air conditioner             |                        |         |                      |         |
| No                          | 2.25                   | 0.01    | 0.21                | 0.01    |
| Yes                         | 1.04                   |         | 0.15                |         |
| Fan in home                 |                        |         |                      |         |
| No                          | 1.31                   | 0.18    | 0.15                | 0.21    |
| Yes                         | 1.03                   |         | 0.12                |         |
| Home configuration          |                        |         |                      |         |
| Open                        | 1.20                   | 0.95    | 0.14                | 0.89    |
| Closed                      | 1.17                   |         | 0.15                |         |
| Public ETS exposure         |                        |         |                      |         |
| No                          | 1.19                   | 0.70    | 0.14                | 0.44    |
| Yes                         | 1.40                   |         | 0.20                |         |
| Carpeting in activity room  |                        |         |                      |         |
| No                          | 1.25                   | 0.80    | 0.21                | 0.0149  |
| Yes                         | 1.18                   |         | 0.12                |         |
| Carpeting in bedroom        |                        |         |                      |         |
| No                          | 1.77                   | 0.06    | 0.24                | 0.013   |
| Yes                         | 1.12                   |         | 0.13                |         |
| Maternal depression         |                        |         |                      |         |
| No                          | 0.96                   | 0.0001  | 0.12                | 0.0107  |
| Yes                         | 2.13                   |         | 0.20                |         |
| Season of the year          |                        |         |                      |         |
| Winter                      | 1.63                   | 0.07    | 0.14                | 0.013   |
| Spring                      | 1.30                   |         | 0.15                |         |
| Summer                      | 0.93                   |         | 0.19                |         |
| Autumn                      | 0.99                   |         | 0.08                |         |

Pearson correlation coefficients.

| Covariates                  | Model 1 (β) | Model 2 (β) | Model 3 (β) | Model 4 (β) | Model 5 (β) |
|-----------------------------|------------|------------|------------|------------|------------|
| African American            | 0.38 ± 0.17| 0.56 ± 0.15| 0.62 ± 0.16| 0.44 ± 0.15| 0.34 ± 0.16|
| ETS exposure at home (cigarettes/day) | 0.05 ± 0.01| 0.04 ± 0.01| 0.05 ± 0.01| 0.04 ± 0.01| 0.04 ± 0.01|
| ETS exposure in the car     | 0.48 ± 0.18| 0.41 ± 0.16| 0.43 ± 0.17| 0.43 ± 0.17| 0.43 ± 0.17|
| Home volume (100 m³)        | −0.45 ± 0.07| −0.42 ± 0.07|          |            |            |

Model 5 adjusts for age, sex, fan, air conditioner, maternal depression, and season in addition to covariates listed above. Final model adjusted $r^2 = 0.35$, model $p < 0.0001$.

| Covariates                  | Model 1 (β) | Model 2 (β) | Model 3 (β) | Model 4 (β) |
|-----------------------------|------------|------------|------------|------------|
| African American            | 1.22 ± 0.16| 1.30 ± 0.16| 1.21 ± 0.16| 1.06 ± 0.17|
| ETS exposure at home (cigarettes/day) | 0.02 ± 0.01| 0.02 ± 0.01| 0.02 ± 0.01| 0.02 ± 0.01|
| Home volume (100 m³)        | −0.28 ± 0.07| −0.30 ± 0.08|          |            |            |

Final model adjusted $r^2 = 0.33$, model $p < 0.0001$.
compared with serum cotinine may be due to multiple factors. First, there may be less variability in hair cotinine. Serum cotinine measures short-term ETS exposure (3–4 days), whereas hair cotinine measures ETS exposure in the prior month (Al-Delaimy 2002a; Al-Delaimy et al. 2002). Because hair cotinine measures long-term ETS exposure, it is less vulnerable to everyday variability in ETS exposure and metabolism (Al-Delaimy 2002). Second, there may be racial differences in the use of dyes and hair treatments, which could affect hair cotinine levels. Pichini et al. (1997) found that use of dyes and hair treatments decreased levels of hair cotinine. Last, there may be differences in other unmeasured factors. Despite these relative differences, our results consistently demonstrate that African-American children have higher levels of cotinine in both hair and serum.

Racial differences in cotinine metabolism raise questions about whether cotinine could help explain the racial differences in tobacco-related morbidity and mortality. Although cotinine is often considered an inert biomarker, recent literature suggests that cotinine exhibits biologic activity. Cotinine is mitogenic on vascular smooth muscle cells in a dose-related fashion (Carty et al. 1997) and has been shown to reduce the survival of hippocampal neurons and to decrease neurite function (Audesirk and Cabell 1999). Further research is necessary to ascertain whether prolonged exposure to nicotine and cotinine contributes to the observed racial differences in childhood asthma morbidity and mortality.

Our study has some limitations. First, we measured ETS exposure using parent report. A systematic underreporting of tobacco use by African-American parents could explain the racial differences in serum and hair cotinine. However, prior studies suggest this is unlikely (Caraballo et al. 2001, 2004; Clark et al. 1996b; Wills and Cleary 1997). For example, Clark et al. (1996b) found no racial difference in the reporting of tobacco use relative to the number of cigarette butts collected. Furthermore, our study measured the level of particulate matter in the main activity room at each residence. Consistent with lower reported ETS exposure, we found that African-American residences had significantly lower levels of particulate matter. Data from other tobacco study groups indicate that particulate matter correlates well with ETS; indeed, ETS is the major contributor of indoor particulates (Cains et al. 2004; Wallace et al. 2003). Nevertheless, additional studies that objectively measure air nicotine are needed. Second, we did not collect information on cigarette type and thus cannot account for the potential effect of ETS exposure from mentholated cigarettes. As mentioned above, menthol has been associated with elevated levels of serum cotinine in active smokers. Finally, we grouped individuals into broad categories by race. Race is not a biologic construct, but an imprecise categorization that is a proxy for environmental, cultural, socioeconomic, and biologic differences. We need additional research to identify the specific factors that increase the risk of African-American individuals having higher levels of cotinine.

In summary, our study demonstrates that African-American children with asthma who were, on average, exposed to lower levels of ETS had higher levels of both serum and hair cotinine compared with white children with asthma. If African-American children are more susceptible to tobacco-induced toxicity, then we should target additional public policy initiatives toward reducing ETS exposure among this population. Further studies are needed to determine whether there are racial differences in the metabolism of other tobacco-related toxins and to assess the efficacy of interventions to reduce ETS exposure among all children.

REFERENCES

Ahijevych K, Garret BE. 2004. Menthol pharmacology and its potential impact on cigarette smoking behavior. Nicotine Tob Res Suppl 1:107–152.

Ahijevych K, Tyndale RF, Dhat RK, Weed HG, Brownking KX. 2002. Factors influencing cotinine half-life during smoking abstinence in African American and Caucasian women. Nicotine Tob Res 4:423–431.

Al-Delaimy WK. 2002. Hair as a biomarker for exposure to tobacco smoke. Tob Control 11:176–182.

Al-Delaimy WK, Crane J, Woodward A. 2002. Is the hair nicotine level a more accurate biomarker of environmental tobacco smoke exposure than urine cotinine? J Epidemiol Community Health 56:66–71.

Al-Delaimy WK, Crane J, Woodward A. 2001. Passive smoking in children: effect of avoidance strategies, at home as measured by hair nicotine levels. Arch Environ Health 56:117–122. Audesirk T, Cabell L. 1999. Nanomolar concentrations of nicotine and cotinine alter the development of cultured hippocampal neurons via non- acetylcholine receptor-mediated mechanisms. Neurotoxicology. Balbach ED, Gasior RJ, Barbeau EM. 2003. R.J. Reynolds’ targeting of African Americans: 1988–2000. Am J Public Health 93:822–827.

Beck AT, Steer RA, Brown GK. 1996. Beck Depression Inventory—II. San Antonio, TX:Harcourt Assessment, Inc.

Benowitz NL, Herrera B, Jacob IP. 1996. Mentholated cigarette smoking inhibits nicotine metabolism. J Pharmacol Exp Ther 310:1209–1215.

Benowitz NL, Perez-Stable EJ, Fong I, Modin G, Herrera B, Jacob P III. 1999. Ethnic differences in N-glucuronidation of nicotine and cotinine. J Pharmacol Exp Ther 291:1196–1203.

Benowitz NL, Perez-Stable EJ, Herrera B, Jacob P III. 2002. Slower metabolism and reduced intake of nicotine from cigarette smoking in Chinese-Americans. J Natl Cancer Inst 94:108–115.

Bennett JT Jr, McGuffey JE, Morrison MA, Pirkle JL. 2000. Comparison of serum and saliva cotinine: measurement by a sensitive high-performance liquid chromatography–tandem mass spectrometry method as an indicator of exposure to tobacco smoke among smokers and nonsmokers. J Anal Toxicol 24:333–339.

Bennett JT Jr, Turner WE, Pirkle JL, Jossoff CS, Akins JR, Waldrep MK, et al. 1997. Development and validation of sensitive method for determination of serum cotinine in smokers and nonsmokers by liquid chromatography/atmospheric pressure ionization tandem mass spectrometry. Clin Chem 43:2281–2289.

Cains T, Cannata S, Foulis R, Ferson MJ, Stewart BW. 2004. Designated “no smoking” areas provide from partial to no protection from environmental tobacco smoke. Tob Control 13:17–22.

Caraballo RS, Giovino GA, Pechacek T. 2004. Self-reported cigarette smoking vs. serum cotinine among U.S. adolescents. Nicotine Tob Res 6:19–25.

Caraballo RS, Giovino GA, Pechacek TF, Mowery PD. 2001. Factors associated with discrepancies between self-reports in cigarette smoking and measured serum cotinine levels among persons aged 17 years or older: Third National Health and Nutrition Examination Survey, 1988–1994. Am J Epidemiol 153:807–814.

Caraballo RS, Giovino GA, Pechacek TF, Mowery PD, Richter PA, Straus WJ, et al. 1998. Racial and ethnic differences in serum cotinine levels of cigarette smokers: Third National Health and Nutrition Examination Survey, 1988–1991. JAMA 280:135–139.

Carty CS, Huribal M, Marsan BU, Ricotta JJ, Dryski M. 1997. Nicotine and its metabolite cotinine are mitogenic for human vascular smooth muscle cells. J Vasc Surg 25:882–888.

CDC. 1998. Tobacco Use among U.S. Racial/Ethnic Minority Groups African Americans, American Indians and Alaska Natives, Asian Americans and Pacific Islanders, Hispanics: A Report of the Surgeon General. Atlanta, GA:Centers for Disease Control and Prevention.

CDC. 1998. Tobacco Use among U.S. Racial/Ethnic Minority Groups African Americans, American Indians and Alaska Natives, Asian Americans and Pacific Islanders, Hispanics: A Report of the Surgeon General. Atlanta, GA:Centers for Disease Control and Prevention.

Clark PI, Gautam S, Gerson LW. 1996a. Effect of menthol cigarette on biochemical markers of smoke exposure in black and white smokers. Chest 110:1194–1198.

Clark PI, Gautam SP, Hsing WM, Gerson LW. 1996b. Response error in self-reported current smoking frequency by black and white established smokers. Am J Epidemiol 143:483–490.

Cohglin J, Hammond SK, Gann PH. 1989. Development of epidemiologic tools for measuring environmental tobacco smoke exposure. Am J Epidemiol 130:696–704.

Cook DG, Strachan DP. 1999. Summary of effects of parental smoking on the respiratory health of children and implications for research. Thorax 54:357–365.

Cooper RS. 2003. Race, genes, and health—new wine in old bottles? Int J Epidemiol 32:23–25.

Cooper RS, Kaufman JS, Ward R. 2003. Race and genomics. N Engl J Med 348:1166–1170.

Elipouloos C, Klein J, Phan MK, Knie B, Greenwald M, Chitayat D, et al. 1994. Hair concentrations of nicotine and cotinine in women and their newborn infants. JAMA 271:821–823.

Federal Trade Commission. 2000. “Tar,” Nicotine and Carbon Monoxide of the Smoke of 1249 Varieties of Domestic Cigarettes for the Year 1998. Washington, DC:Federal Trade Commission.

Federal Trade Commission. 2002. Federal Trade Commission Cigarette Report for 2000. Washington, DC:Federal Trade Commission.

Hecht SS, Ye M, Carmella SG, Fredrickson A, Adgate JL, Greaves IA, et al. 2001. Metabolites of a tobacco-specific lung carcinogen in the urine of elementary school-aged children. Cancer Epidemiol Biomarkers Prev 10:1109–1116.

Henschen M, Frischer T, Pracht T, Speikerkater E, Karmus W, Meinert R, et al. 1997. The internal dose of passive smoking at home depends on the size of the dwelling. Environ Res 72:65–71.

Kabat GC, Morabia A, Wynder EL. 1991. Comparison of smoking habits of blacks and whites in a case-control study. Am J Public Health 81:1483–1486.

Klein J, Koren G. 1999. Hair analysis—a biological marker for passive smoking in pregnancy and childhood. Hum Exp Toxicol 18:279–282.

Knight JM, Elipouloos C, Klein J, Greenwald M, Koren G. 1996. Passive smoking in children. Racial differences in systemic exposure to cotinine by hair and urine analysis. Chest 109:464–466.

Larsen MS, Frisk M, Hallstrom J, Kivijorg J, Lundback B. 2001. Environmental tobacco smoke exposure during childhood is associated with increased prevalence of asthma in adults. Chest 120:711–717.

Mannino DM, Albalak R, Grosse S, Repace J. 2002. Second-hand smoke exposure and blood lead levels in U.S. children. Epidemiology 14:719–727.

Mannino DM, Caraballo R, Benowitz N, Repace J. 2001a. Predictors of cotinine levels in US children: data from the Third National Health and Nutrition Examination Survey. Chest 120:718–724.
Mannino DM, Homa DM, Redd SC. 2002. Involuntary smoking and asthma severity in children: data from the Third National Health and Nutrition Examination Survey. Chest 122:409–415.

Mannino DM, Moorman JE, Kingsley B, Rose D, Repace J. 2001b. Health effects related to environmental tobacco smoke exposure in children in the United States: data from the Third National Health and Nutrition Examination Survey. Arch Pediatr Adolesc Med 155:36–41.

Paschke T, Riefler M, Schuler-Metz A, Wolz L, Scherer G, McBride CM, et al. 2001. Comparison of cytochrome P450 2A6 polymorphism frequencies in Caucasians and African-Americans using a new one-step PCR-RFLP genotyping method. Toxicology 188:259–268.

Perera FP, Mooney LA, Stampfer M, Phillips DH, Bell DA, Rundle A, et al. 2002. Associations between carcinogen-DNA damage, glutathione S-transferase genotypes, and risk of lung cancer in the prospective Physicians’ Health Cohort Study. Carcinogenesis 23:1641–1648.

Perez-Stable EJ, Herrera B, Jacob P III, Benowitz NL. 1998. Nicotine metabolism and intake in black and white smokers. JAMA 280:152–156.

Pichini S, Alberi I, Pellegriini M, Pacifici R, Zuccaro P. 1997. Hair analysis for nicotine and cotinine: evaluation of extraction procedures, hair treatments, and development of reference material. Forensic Sci Int 84:243–252.

Schuster MA, Franke T, Pham CB. 2002. Smoking patterns of household members and visitors in homes with children in the United States. Arch Pediatr Adolesc Med 156:1094–1100.

Sexton K, Adgate JL, Church TR, Hecht SS, Ramachandran G, Greaves IA, et al. 2004. Children’s exposure to environmental tobacco smoke: using diverse exposure metrics to document ethnic/racial differences. Environ Health Perspect 112:292–297.

Tang D, Phillips DH, Stampfer M, Mooney LA, Hsu Y, Cho S, et al. 2001. Association between carcinogen-DNA adducts in white blood cells and lung cancer risk in the physicians health study. Cancer Res 61:706–712.

Tang D, Warburton D, Tannenbaum SR, Skipper P, Santella RM, Cereijido GS, et al. 1999. Molecular and genetic damage from environmental tobacco smoke in young children. Cancer Epidemiol Biomarkers Prev 8:427–431.

Tricker AR. 2003. Nicotine metabolism, human drug metabolism polymorphisms, and smoking behaviour. Toxicology 182:151–173.

U.S. DHHS. 2000. Healthy People 2010: Tracking Healthy People 2010. Washington, DC.U.S. Department of Health and Human Services.

Wallace LA, Mitchell H, O’Connor GT, Neas L, Lippmann M, Kattan M, et al. 2003. Particle concentrations in inner-city homes of children with asthma: the effect of smoking, cooking, and outdoor pollution. Environ Health Perspect 111:1265–1272.

Wills TA, Cleary SD. 1997. The validity of self-reports of smoking: analyses by race/ethnicity in a school sample of urban adolescents. Am J Public Health 87:56–61.