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Racial and Ethnic Differences in Serum Cotinine Levels of Cigarette Smokers Third National Health and Nutrition Examination Survey, 1988-1991

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Original Contributions

Racial and Ethnic Differences in Serum Cotinine Levels of Cigarette Smokers

Third National Health and Nutrition Examination Survey, 1988-1991

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Context.—Cotinine, a metabolite of nicotine, is a marker of exposure to tobacco smoke. Previous studies suggest that non-Hispanic blacks have higher levels of serum cotinine than non-Hispanic whites who report similar levels of cigarette smoking.

Objective.—To investigate differences in levels of serum cotinine in black, white, and Mexican American cigarette smokers in the US adult population.

Design.—Third National Health and Nutrition Examination Survey, 1988-1991.

Participants.—A nationally representative sample of persons aged 17 years or older who participated in the survey.

Outcome Measures.—Serum cotinine levels by reported number of cigarettes smoked per day and by race and ethnicity.

Results.—A total of 7182 subjects were involved in the study; 2136 subjects reported smoking at least 1 cigarette in the last 5 days. Black smokers had cotinine concentrations substantially higher at all levels of cigarette smoking than did white or Mexican American smokers ($P<.001$). Serum cotinine levels for blacks were 125 nmol/L (22 ng/mL) (95% confidence interval [CI], 79-176 nmol/L [14-31 ng/mL]) to 539 nmol/L (95 ng/mL) (95% CI, 289-630 nmol/L [51-111 ng/mL]) higher than for whites and 136 nmol/L (24 ng/mL) (95% CI, 85-182 nmol/L [15-32 ng/mL]) to 641 nmol/L (113 ng/mL) (95% CI, 386-897 nmol/L [68-158 ng/mL]) higher than for Mexican Americans. These differences do not appear to be attributable to differences in environmental tobacco smoke exposure or in number of cigarettes smoked.

Conclusions.—To our knowledge, this study provides the first evidence from a national study that serum cotinine levels are higher among black smokers than among white or Mexican American smokers. If higher cotinine levels among blacks indicate higher nicotine intake or differential pharmacokinetics and possibly serve as a marker of higher exposure to cigarette carcinogenic components, they may help explain why blacks find it harder to quit and are more likely to experience higher rates of lung cancer than white smokers.

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ever.[3,5-8] Some researchers[3,6,7] have suggested that differences between levels of serum cotinine in non-Hispanic black smokers and non-Hispanic white smokers are attributable, at least in part, to racial differences in nicotine metabolism or elimination. Others[5] believe that such differences are attributable to other variables, including differences in the type of cigarette smoked (length of cigarette, menthol or nonmenthol, filter or nonfilter, and nicotine yield) and differences in how the cigarettes are smoked (blocking ventilation holes by fingers or lips, frequency and depth of inhalation, retention time of smoke in the lungs, and percentage of available tobacco smoked). Serum cotinine differences by race have also been attributed to differences in the accuracy of cigarette smoking self-reports[2,9-12] and to differences in exposure to ETS.[4,13,14]

See also pp 152 and 179.

Cotinine is widely applied as a marker of both tobacco use and exposure to environmental tobacco smoke (ETS) because it has a longer half-life (average, 18 to 20 hours) than nicotine does (average, 2 to 3 hours). On average, a cigarette smoker absorbs about 1 mg of nicotine per cigarette smoked.[15,16] The increment in blood nicotine concentration after smoking a single cigarette ranges from 0.031 to 0.185 µmol/L (5-30 ng/mL), depending on how the cigarette is smoked.[17-19] An average of 70% to 80% of the nicotine absorbed is metabolized to cotinine.[16,20-23] In adult smokers, a nicotine intake of approximately 1 mg can be estimated from a blood cotinine level of 71 nmol/L (12.5 ng/mL) using a conversion factor of 0.08 mg/24 h per nanogram per milliliter under steady-state conditions.[14] The co-

THE BIOCHEMICAL measurement of serum cotinine, the primary metabolite of nicotine, is widely applied as a marker of both tobacco use and exposure to environmental tobacco smoke (ETS). Previous studies[1-4] have suggested that non-Hispanic blacks have higher levels of serum cotinine than do non-Hispanic whites who report similar levels of cigarette smoking. The interpretation of the results in these studies has been subject to debate, how-

efficient of variation of the correction factor is 22%, however, and does not take into account possible racial or ethnic differences in the conversion of nicotine to cotinine.

Scientists at the National Center for Environmental Health of the Centers for Disease Control and Prevention recently developed a new, highly sensitive biochemical measurement method for detecting levels of serum cotinine as low as 0.3 nmol/L (0.05 ng/mL). Using the new method, we investigated racial and ethnic differences in serum cotinine levels. We obtained data on serum cotinine levels from a representative sample of adult smokers and nonsmokers of the third National Health and Nutrition Examination Survey (NHANES III), a nationwide household collection of health and nutritional information from a representative sample of the US civilian, noninstitutionalized population aged 2 months or older.

METHODS

The NHANES III, conducted from 1988 to 1994, consisted of a number of questionnaires performed in the household followed by standardized physical examinations and additional tobacco use questions administered in specially equipped mobile examination centers (MECs). We used NHANES III phase 1 data collected between October 25, 1988, and October 21, 1991, because the data for some of the key variables for the analyses were available for only this 3-year period. The nationally representative sample of the eligible population surveyed during phase 1 permitted calculation of national estimates.

Subjects and Demographics

Our study sample was limited to participants aged 17 years or older who described themselves as non-Hispanic blacks, non-Hispanic whites, or Mexican Americans, who had a serum cotinine measurement and provided tobacco use information in the MEC, and who did not use any other significant sources of nicotine in the previous 5 days. Of the 12 391 persons selected, 2271 refused the interview; 1315 were interviewed at home and did not visit the MECs; 281 did not answer the MEC tobacco questionnaire; 682 had no cotinine measurement; 434 reported using other significant sources of nicotine in the previous 5 days; and 226 were other than non-Hispanic white, non-Hispanic black, and Mexican American. Data from 2136 subjects who reported smoking 1 cigarette or more in the past 5 days were included in the analyses. One of the analyses included data from both smokers and nonsmokers (n = 7182).

Race and ethnicity based on self-report were categorized as non-Hispanic black, non-Hispanic white (henceforth referred to as blacks and whites), and Mexican American. Age at interview was categorized as 17 to 24, 25 to 44, 45 to 64, or 65 or more years. Educational level was categorized as 0 to 8, 9 to 11, 12, or 13 or more completed years of schooling. Poverty status was based on a measure developed by the US Bureau of the Census. Members of families whose incomes were equal to or greater than poverty thresholds were categorized as "at or above poverty level"; those with family incomes below the poverty threshold, as "below poverty level." Each subject's weight in kilograms, measured using a digital scale, was categorized as less than 60, 60 to 69.99, 70 to 79.99, and 80 kg or more.

Reported exposure to ETS at home was based on the following questions posed to 1 member of the household (usually the head of the family or spouse of the head): "Does anyone who lives here smoke cigarettes in the home?" When the answer was yes, the interviewee was then asked: "Who?" When any household member smoked, each member of that household was classified as being exposed to ETS at home. The number of household members who smoked was categorized as 0, 1, or 2 or more. In addition, one of the family members was asked how many rooms were in the home, excluding bathrooms. The number of rooms in the home was categorized as 1 to 4 or 5 or more. Persons aged 17 years or older who reported having a job or business were also asked how many hours per day they were close enough to tobacco smoke at work that they could smell the smoke. The number of hours exposed to ETS at work was categorized as 0, 1 to 3, or 4 or more. Data were also categorized by region as Northeast, North Central, South, or West, according to standard US Bureau of the Census definitions.

The MEC tobacco questionnaire asked participants: "How many cigarettes have you smoked in the past 5 days?" A smoker was defined as a person who reported smoking 1 cigarette or more during the previous 5 days. The average number of cigarettes smoked per day was calculated and used for the analyses.

Serum Cotinine Measurement

Biochemical determination of tobacco exposure was performed by measuring serum cotinine levels in blood specimens obtained by venipuncture in the MEC. The cotinine assay involved isotope dilution, liquid chromatography, and tandem mass spectrometry.[24] As in the study by Pirkle and colleagues,[13] we used cutoff points of higher than 85 nmol/L (15 ng/mL) and 85 nmol/L (15 ng/mL) or less of cotinine in serum to designate active tobacco users and nontobacco users, respectively.

Cotinine data were originally reported in nanograms per milliliter. A conversion factor of 5.67 was used to convert cotinine levels in nanograms per milliliter to cotinine levels in nanomoles per liter, based on a molecular weight for cotinine of 176.22. A conversion factor of 0.006 was used to convert nicotine levels previously reported in nanograms per milliliter to nicotine levels in micromoles/liter, based on a molecular weight for nicotine of 162.23.

Statistical Analysis

Determination of serum cotinine levels by race and ethnicity involved fitting a series of nonlinear exponential regression models of the form: $\ln(\text{cotinine}) = \beta_0 - \beta_1 \exp(-\beta_2 O) + error$, where $\ln(\text{cotinine})$ is the natural logarithm of serum cotinine; O is the number of self-reported cigarettes smoked per day; β_0, β_1, and β_2 are parameters that describe the exponential relationship; and $error$ is the residual error left unexplained by the model. This exponential model facilitated modeling serum cotinine as a monotonically increasing function of the number of cigarettes smoked, with cotinine increasing at a decreasing rate toward an upper asymptote. The variable β_0 represents the upper bound (maximum achievable level) of $\ln(\text{cotinine})$ at the highest levels of daily smoking, and β_2 represents the rate at which cotinine increases with consumption. The quantity $(\beta_0 - \beta_1)$ represents the expected $\ln(\text{cotinine})$ at a consumption level of 0 cigarettes per day. Prior to model fitting, we divided the self-reported number of cigarettes smoked in the last 5 days by 5 and rounded to the nearest integer. Therefore, in the model, less than 1 represents a response of 1 to 4 cigarettes smoked in the last 5 days.

We fit 3 exponential models, one for each racial or ethnic group, and used weights supplied with the NHANES III data set to make the results more applicable to the US population. After fitting the simple exponential model, a series of covariate adjusted models were fit to the data from each racial and ethnic group. These models investigated the relationship between serum cotinine and cigarettes smoked per day after adjustment for each of the following covariates: sex, number of smokers in the household, hours of exposure to ETS at work, age, and weight.

The more complicated models were fit by adding covariates one at a time to the base model in an ordered sequence determined by the results of an F test of significance for each variable when added individually to the base model. We assessed the adequacy of the fit of the exponential regression models by using plots of the studentized residuals vs the predicted values. The assumption of

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normally distributed cotinine data was assessed using normal probability plots applied to the studentized residuals.

We used the appropriate weights and computed the complex variance estimates using SUDAAN[25] in all analyses.

RESULTS

Study Population

Proportionately more black smokers than white smokers were poor and weighed 80 kg or more (Table 1). In terms of sociodemographic characteristics, Mexican American smokers differed from other racial and ethnic groups of smokers more than either their white or black counterparts.

Among self-reported smokers, both blacks and whites had serum cotinine levels consistent with their reported smoking levels (Table 2). Therefore, among blacks and whites, the ability of the biochemical measurement to detect smokers and nonsmokers was similar. Self-reported Mexican American smokers were less likely than blacks or whites to have serum cotinine levels consistent with their reported smoking levels. Self-reported nonsmokers (97.9% of blacks, 98.5% of whites, and 99.4% of Mexican Americans) had biochemically assessed cotinine threshold levels consistent with their reported smoking levels.

Self-reported and biochemically assessed smoking prevalences among blacks (34.9% and 34.7%, respectively) were similar to those for whites (33.1%, 32.1%) and higher than Mexican Americans (27.0%, 20.1%) (results not shown). Although blacks were as likely to have smoked in the past 5 days as whites, they reported smoking fewer cigarettes per day than whites (Table 3). Mexican Americans smoked fewer cigarettes per day than did either blacks or whites.

Statistics and Modeling

Fitting a nonlinear exponential unadjusted regression model (Figure) to determine the relationship between serum cotinine levels and self-reported cigarette smoking showed that cotinine concentrations were substantially higher among black smokers than among white or Mexican American smokers at all levels of cigarette smoking. Whites and Mexican Americans had similar serum cotinine levels when they smoked up to 5 cigarettes per day, but serum cotinine levels increased significantly more for whites than for Mexican Americans with each additional cigarette smoked. Serum cotinine levels for blacks were 125 nmol/L (22 ng/mL) (95% confidence interval [CI], 79-176 nmol/L [14-31 ng/mL]) to 539 nmol/L (95 ng/mL) (95% CI, 289-630 nmol/L [51-111 ng/mL]) higher than for whites and 136 nmol/L (24 ng/mL) (95% CI, 85-182 nmol/L [15-32 ng/mL]) to 641 nmol/L (113 ng/mL) (95% CI, 386-897 nmol/L [68-158 ng/mL]) higher than for Mexican Americans. Verification of the results of this analysis by unweighted analysis yielded similar results. Previous analyses had revealed that variables such as education, poverty status, time of day the blood was drawn, number of rooms in the house, and geographic region were not significantly associated with serum cotinine (data not shown). These variables were excluded from the final model in this study.

An F test comparing a full model (adjusting the relationship for the effects of race and ethnicity, age, sex, body weight,

Table 1.—Study Sample Distribution of 2136 Self-reported Smokers Aged 17 Years or Older, by Race/Ethnicity and Sociodemographic Characteristics, NHANES III, United States, 1988-1991*

Characteristic	No. (%) of Non-Hispanic Blacks (n = 665)	No. (%) of Non-Hispanic Whites (n = 900)	No. (%) of Mexican Americans (n = 571)
Sex			
Male	365 (55)	465 (52)	374 (66)
Female	300 (45)	435 (48)	197 (34)
Age, y			
17-24	104 (16)	152 (17)	148 (26)
25-44	324 (49)	358 (40)	255 (45)
45-64	181 (27)	255 (28)	120 (21)
≥65	56 (8)	135 (15)	48 (8)
Education, y			
0-8	70 (11)	79 (9)	228 (41)
9-11	189 (28)	179 (20)	131 (23)
12	263 (40)	360 (40)	131 (23)
≥13	140 (21)	275 (31)	74 (13)
Poverty status			
< Poverty level	279 (42)	173 (19)	296 (52)
≥ Poverty level	386 (58)	727 (81)	275 (48)
Weight, kg			
<60	110 (18)	200 (24)	113 (21)
60-60.99	147 (23)	167 (20)	143 (27)
70-79.99	144 (23)	221 (26)	117 (22)
≥80	224 (36)	253 (30)	159 (30)
No. of smokers in home			
0	58 (9)	99 (11)	155 (27)
1	355 (54)	371 (41)	254 (45)
≥2	249 (37)	430 (48)	161 (28)
No. of rooms in home			
1-4	204 (31)	245 (27)	261 (46)
≥5	461 (69)	655 (73)	310 (54)
Tobacco exposure at work, h			
0	414 (64)	477 (54)	382 (69)
1-3	74 (11)	124 (14)	75 (13)
≥4	163 (25)	280 (32)	100 (18)
US region			
Northeast	103 (16)	179 (20)	6 (1)
North Central	182 (27)	243 (27)	48 (8)
South	333 (50)	347 (38)	164 (29)
West	47 (7)	131 (15)	353 (62)

*NHANES III indicates third National Health and Nutrition Examination Survey.

Table 2.—Agreement Between Self-reported Smoking Status and Cotinine Levels of 7182 Smokers and Nonsmokers Aged 17 Years or Older, by Race/Ethnicity, NHANES III, United States, 1988-1991*

Race/Ethnicity and Self-reported Smoking Status	Cotinine Level		Total
	>85 nmol/L (15 ng/mL), No. (% ± 95% CI)	≤85 nmol/L (15 ng/mL), No. (% ± 95% CI)	
Blacks			
Smoker	632 (95.5 ± 2.0)	33 (4.5 ± 2.0)	665
Nonsmoker	30 (2.1 ± 0.6)	1129 (97.9 ± 0.6)	1159
Whites			
Smoker	849 (93.8 ± 1.7)	51 (6.2 ± 1.7)	900
Nonsmoker	42 (1.5 ± 0.5)	2299 (98.5 ± 0.5)	2341
Mexican Americans			
Smoker	426 (72.5 ± 7.4)	145 (27.5 ± 7.4)	571
Nonsmoker	10 (0.6 ± 0.5)	1536 (99.4 ± 0.5)	1546

*NHANES III indicates third National Health and Nutrition Examination Survey; CI, confidence interval.

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Table 3.—Percentage of 2136 Self-reported Smokers Aged 17 Years or Older, by Number of Cigarettes Smoked per Day in the Past 5 Days and by Race/Ethnicity, NHANES III, United States, 1988-1991*

Cigarettes per Day	Non-Hispanic Blacks, No. (% ± 95% CI)	Non-Hispanic Whites, No. (% ± 95% CI)	Mexican Americans, No. (% ± 95% CI)
<1	41 (6.2 ± 2.3)	45 (5.5 ± 1.7)	127 (24.3 ± 6.1)
1-7	200 (30.1 ± 3.7)	120 (12.6 ± 2.1)	240 (41.7 ± 3.9)
8-14	193 (29.3 ± 2.8)	152 (17.7 ± 3.0)	111 (17.9 ± 3.5)
15-24	186 (27.8 ± 3.8)	315 (36.5 ± 3.5)	80 (13.4 ± 2.8)
≥25	45 (6.6 ± 1.7)	268 (27.7 ± 4.5)	13 (2.7 ± 1.5)
Total	665 (100.0)	900 (100.0)	571 (100.0)

*NHANES III indicates third National Health and Nutrition Examination Survey; CI, confidence interval.

Table 4.—Sequential F Tests and Associated *P* Values for Each Covariate When Added to the Previous Weighted Nonlinear Exponential Regression Model for 2127 Smokers Aged 17 Years or Older, by Race/Ethnicity, NHANES III, United States, 1988-1991*

		Blacks, Covariates Added				
Result	Base Model	No. of Smokers in Home	Body Weight Category	Age Category	Hours of Work Exposure to Smoke	Sex
F	1258	4.65	3.75	2.36	1.16	0.59
P	<.001	<.001	<.001	.01	.33	.62
No.	662	656	647	638	632	629

		Whites, Covariates Added				
Result	Base Model	Hours of Work Exposure to Smoke	Sex	Age Category	No. of Smokers in Home	Body Weight Category
F	116	7.97	5.84	3.62	3.26	2.28
P	<.001	<.001	<.001	<.001	.004	.02
No.	897	891	888	879	873	864

		Mexican Americans, Covariates Added				
Result	Base Model	Hours of Work Exposure to Smoke	No. of Smokers in Home	Age Category	Body Weight Category	Sex
F	3794	4.26	3.36	1.72	1.64	0.45
P	<.001	<.001	.003	.08	.10	.72
No.	568	562	556	547	538	535

*NHANES III indicates third National Health and Nutrition Examination Survey.

Correlation between serum cotinine levels and number of cigarettes smoked per day, as predicted by preliminary regression models.

and ETS exposure at home and at work) with a reduced model (adjusting for only the effects of age, sex, body weight, and ETS exposure at home and at work) showed the statistical significance of persistent racial and ethnic differences in the relationship between serum cotinine and self-reported cigarette smoking (after adjustment for other covariates, *P*<.001).

For blacks, the number of smokers living in the home, body weight, and age, in that order, explained significant reductions in variability of serum cotinine levels (Table 4). For whites, the number of hours of ETS exposure at work, sex, age, the number of smokers living in the home, and body weight were of descending importance in explaining significantly reduced variability of serum cotinine levels. And for Mexican Americans, 2 covariates were responsible for reducing this variability: the number of hours of ETS exposure at work and the number of smokers living in the home.

COMMENT

This study provides the first evidence from a national study that black smokers have higher serum cotinine levels than do white or Mexican American smokers, after adjustment for the number of ciga-

rettes smoked per day, age, sex, body weight, number of smokers living in the home, and number of hours exposed at work to ETS. Our finding is consistent with the results of previous smaller studies[1-4] that have found that blacks have higher serum cotinine levels than whites do at similar levels of self-reported smoking, but differs from that of 1 previous report[12] suggesting that Mexican Americans derive more cotinine per cigarette than whites do.

The analysis in our study was limited by the lack of data on the type of cigarette smoked (eg, menthol vs regular or filter vs nonfilter), smoking topography (eg, depth of inhalation or vent blocking), or nicotine pharmacokinetics. Previous studies have assessed the contribution of some of these factors (eg, the use of mentholated or nonmentholated cigarettes) to serum cotinine levels among black smokers and white smokers. Some of these studies[1,26-28] have reported that smokers of mentholated cigarettes have higher levels of serum cotinine than smokers of nonmentholated cigarettes; others[3,29] have reported no significant differences. More blacks (76%) than whites (23%) prefer mentholated cigarettes.[30,31]

In a study of 5115 young adults,[3] Wagenknecht and colleagues found that the
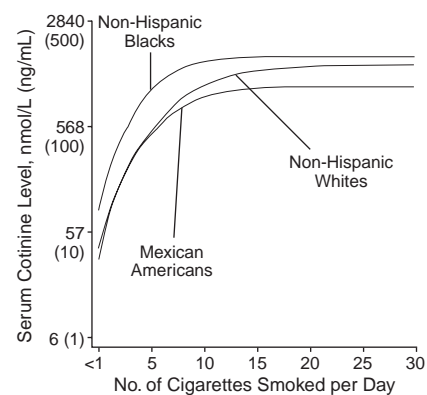
effect of mentholation on serum cotinine levels was 188 nmol/L (33.2 ng/mL) for black smokers and 20 nmol/L (3.6 ng/mL) for white smokers; neither difference was statistically significant. The authors concluded that higher serum cotinine levels in black smokers of mentholated cigarettes explained part but not all of the racial difference in serum cotinine levels. In addition to considering the mentholation of cigarettes, these authors adjusted the results for nicotine content of the cigarette, inhalation frequency, how far smokers let their cigarette burn when they smoked, and weekly exposure to sidestream smoke from the burning end of a cigarette. The serum levels of thiocyanate, a metabolite of cyanide that reflects exposure to tobacco smoke, were similar for the groups when the results were adjusted for the number of cigarettes smoked per day. This finding suggests that the higher cotinine levels found among blacks were not the result of inhaling more smoke than whites do.[7]

Serum cotinine differences between black smokers and white smokers have also been suggested to be attributable to differential reports of the number of cigarettes smoked. Although we did not measure the validity of the study participants' self-reports, we found no differences in the reliability of self-reports among blacks, whites, and Mexican Americans. Clark and colleagues[8] designed a study to determine differences in cigarette smoking reports between 66 blacks and 97 whites. They collected information about the nicotine content of the cigarette, inhalation frequency, how far smokers let their cigarettes burn when they smoked, and if the cigarettes were menthol or nonmenthol. They even measured the lengths of cigarette butts, which were collected for a week. Clark et al found no evidence that underreporting of daily cigarette consumption occurred more often in black than in white smokers and significantly higher se-

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rum cotinine levels in blacks than in whites. They concluded that the disproportionately high cotinine levels observed in their study among blacks could not be attributed to reporting error. This result is consistent with our findings. If the study by Clark et al can be generalized to the US population, it seems unlikely that reporting differences biased our results.

We also considered differences in self-reported exposure to ETS between the 3 racial and ethnic groups. Even after adjustment of the results for number of smokers living in the home, number of hours of ETS exposure at work, number of rooms in the home, and region of the country where the participants lived, the differences in the relationships between serum cotinine level and self-reported cigarette consumption for the 3 racial groups remained statistically significant. A limitation of this measure, however, is that it relies on self-reports, not a precise ETS measurement. On the other hand, chamber study results[32] suggest that the ETS exposure generated by smokers smoking 10 different brands of US cigarettes is similar, thus providing no basis for believing differential brand exposure could account for differences observed between black and white or Mexican American smokers.

Our results documenting differences between the serum cotinine levels of black smokers and white smokers are consistent with results[4,13,33-35] documenting differences in serum cotinine levels between black nonsmokers and white nonsmokers. In these reports, blacks had higher cotinine levels than whites, even after ETS exposure and other factors were taken into account. For example, after adjusting for self-reported levels of ETS exposure, Pirkle and colleagues[13] found higher cotinine levels for blacks than for whites or Mexican Americans among persons aged 17 years or older.

Results from our study suggest that differences in serum cotinine levels between blacks and whites cannot be explained by differences in reporting the number of cigarettes smoked or differences in ETS exposure. The differences may be influenced by group-specific patterns of smoking behavior and may also be influenced by differences in nicotine pharmacokinetics and brand mentholation.

Further research is needed to clarify the relationship between smoking practices and serum cotinine levels in ethnic groups in the United States. Although there is no definitive explanation why black smokers have higher serum cotinine levels than both whites and Mexican Americans, this finding is intriguing for 2 reasons: (1) black smokers are more likely to try to quit but have a lower success rate than white smokers,[36] and (2) blacks in the

United States are at higher risk than whites of developing and dying from lung cancer.[37] Whether higher serum cotinine concentrations contribute to higher rates of nicotine addiction among blacks than whites is unknown. If higher serum cotinine levels serve as a marker of higher nicotine intake and absorption, they may help explain the lower quitting success rate among black smokers compared with white smokers. If higher serum cotinine levels serve as a marker of higher exposure to other cigarette components such as carcinogenic constituents in cigarette smoke, they may help explain higher lung cancer deaths among black smokers compared with white smokers.

Future research should focus on clarifying the independent and interactive influences of race and ethnicity, nicotine intake, nicotine pharmacokinetics, and nicotine addiction, as well as the relationship between serum cotinine levels and the risk for smoking-related diseases. At present, it is not known whether the differences in serum cotinine levels have important implications for smoking prevention strategies and public health. Nonetheless, the racial or ethnic differences observed in this research provide a plausible basis for consideration of different patterns of tobacco use and related health consequences.

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