American Journal of Epidemiology

Vol. 153, No. 8
Printed in U.S.A.

PRACTICE OF EPIDEMIOLOGY

Factors Associated with Discrepancies between Self-Reports on Cigarette Smoking and Measured Serum Cotinine Levels among Persons Aged 17 Years or Older

Third National Health and Nutrition Examination Survey, 1988–1994

Ralph S. Caraballo,[1] Gary A. Giovino,[2] Terry F. Pechacek,[1] and Paul D. Mowery[3]

The discrepancy between cigarette smoking status reported during an interview and measured level of serum cotinine, a nicotine biomarker, was investigated in a representative sample of the US population aged ≥17 years (N = 15,357). Data were collected from participants in the Third National Health and Nutrition Examination Survey (1988–1994). Among self-reported smokers, 7.5% (95% confidence interval: 6.3, 8.7) had a serum cotinine level less than or equal to 15.0 ng/ml, the selected cutoff point for identifying nonsmokers. Age (p < 0.01), race/ethnicity (p < 0.01), and average number of cigarettes smoked per day (p < 0.01) were associated with these discrepant findings. Among self-reported nonsmokers, 1.4% (95% confidence interval: 1.1, 1.7) had a serum cotinine level greater than 15.0 ng/ml, the selected cutoff point for identifying smokers. Race/ethnicity (p < 0.01), education (p < 0.01), number of household members who smoked in the home (p = 0.03), and self-reported smoking status from an earlier home interview (p < 0.01) were associated with these discrepant findings. Differences in smoking patterns, including the extent of nicotine dosing, may explain most of the discrepancy observed among self-reported smokers, whereas deception regarding smoking status may explain most of the discrepancy among self-reported nonsmokers. This study provides evidence that self-reported smoking status among adult respondents to a population-based survey conducted in a private medical setting is accurate. *Am J Epidemiol* 2001;153:807–14.

adult; cotinine; data collection; epidemiologic methods; smoking

Studies collecting data on cigarette smoking typically rely on participants' reports of their smoking status. Such self-reports may be suspect, however, because participants may be unwilling to admit to a health or social behavior that many perceive to be undesirable. Studies comparing self-reported smoking status with concentrations of biochemical markers of tobacco consumption (e.g., nicotine, cotinine, carbon monoxide, carboxyhemoglobin, and thiocyanate) have generally found self-reports to be good indicators of actual smoking status (1–5), although in some populations self-reports may underestimate the actual prevalence of cigarette smoking by up to 4 percent (1, 2, 6). Factors that may affect the accuracy of self-reports include age (7), race/ethnicity (2, 3, 8), education (2, 3), smoking history (2), and the presence or absence of a disease known to be linked with smoking (9). Identifying factors that contribute to misclassification of self-reported smokers and nonsmokers could allow more accurate estimates of smoking prevalence.

Biochemical markers of tobacco consumption vary in sensitivity (biochemical measures lose sensitivity at lower levels of smoking), specificity, and difficulty of analysis (3–5, 10–12). Overall, studies comparing nonsmokers (exposed or unexposed to environmental tobacco smoke) with active smokers (13–19) have consistently found that measurement of cotinine in serum can distinguish active smokers from nonsmokers. However, the concentration distributions for occasional smokers and nonsmokers exposed to environmental tobacco smoke have been found to overlap (20), exposing one limitation of the use of serum cotinine concentration as a gold standard. Nicotine is the best marker of tobacco exposure, because this biomarker is relatively unique to tobacco, but its half-life is short (2–3 hours) (21). Cotinine is a primary metabolite of nicotine, and it has an average half-life of 18–20 hours (21); thus, it can be used to accurately assess a person's exposure to tobacco—whether through passive exposure (i.e., to environmental tobacco smoke) or through active use. Indeed, serum cotinine concentration has been widely used as

Received for publication December 8, 1999, and accepted for publication August 8, 2000.

Abbreviations: MEC, mobile examination center; NHANES III, Third National Health and Nutrition Examination Survey.

[1] National Center for Chronic Disease Prevention and Health Promotion, Centers for Disease Control and Prevention, Atlanta, GA.
[2] Roswell Park Cancer Institute, Buffalo, NY.
[3] Research Triangle Institute, Research Triangle Park, NC.
Reprint requests to Dr. Ralph Caraballo, Mailstop K-50, Office on Smoking and Health, National Center for Chronic Disease Prevention and Health Promotion, 4770 Buford Highway NE, Atlanta, GA 30341-3724 (e-mail: rfc8@cdc.gov).

a biomarker in studies assessing exposure to tobacco (20, 22–27). The National Center for Environmental Health (Centers for Disease Control and Prevention) recently developed a highly sensitive measurement method for detecting serum cotinine concentrations as low as 0.05 ng/ml (28).

We explored in depth discrepancies between self-reported smoking status and measured serum cotinine concentrations. We used data collected from persons aged 17 years or older in the United States who participated in the Third National Health and Nutrition Examination Survey (NHANES III).

MATERIALS AND METHODS

NHANES III was a nationwide household survey that collected health and nutritional information from a representative sample of the US civilian, noninstitutionalized population aged 2 months or older (29). The survey, which was conducted from 1988 to 1994, consisted of an interview carried out in the participant's home (including questions on tobacco use and exposure), followed by a standardized physical examination and an additional questionnaire on tobacco use administered in specially equipped mobile examination centers (MECs).

Subject selection

In NHANES III, 24,230 persons aged 17 years or older were selected to participate in the survey; 4,180 refused the interview, and 2,345 were interviewed at home but did not visit the MEC. For our study, we excluded 2,348 persons because they either did not answer the questionnaire on tobacco use, had no cotinine measurement, or reported having had exposure to significant sources of nicotine other than cigarettes in the previous 5 days. Thus, our study consisted of 15,357 persons aged 17 years or older. Children and adolescents aged 8–16 years were excluded because of methodological issues in using biomarkers to assess active smoking among persons initiating or experimenting with cigarette use (7).

Demographic classification

Demographic information was collected during the in-home interview. We classified our study participants by sex, age (17–24, 25–44, 45–64, or ≥65 years), race/ethnicity (non-Hispanic Black, non-Hispanic White, or Mexican-American), marital status (married or not married), and education (0–8, 9–11, 12, or ≥13 completed years of schooling). Poverty status was based on a measure developed by the US Census Bureau (30) in which members of families having annual incomes equal to or greater than the poverty threshold were categorized as "at or above poverty level" and those having annual incomes below the poverty threshold were categorized as "below poverty level." Body mass index, which is commonly used to assess obesity, was computed as body weight (in kilograms) divided by the square of height (in meters). Obesity was defined as a body mass index of 30.0 or higher (31, 32).

For determination of environmental tobacco smoke exposure in the home, one member of the household (usually the head of the family or his/her spouse) was asked, "Does anyone who lives here smoke cigarettes in the home?" If the answer was yes, the interviewer asked who it was. When that household member was identified, every other member of the household was classified as being exposed to environmental tobacco smoke at home. We categorized the number of household members who smoked cigarettes at home as zero, one, or two or more. One of the family members was asked how many rooms were in the home, excluding bathrooms. We categorized the number of rooms as 1–4 rooms and five or more rooms.

We created a variable to capture the presence of a tobacco-related disease. Participants who reported that they had ever been told by a doctor that they had a stroke, a heart attack, congestive heart failure, emphysema, chronic bronchitis, or a smoking-related cancer were categorized as having a smoking-related disease. All others were categorized as not having such a disease.

Participants who reported having a job or business were asked how many hours per day they were close enough to tobacco smoke at work that they could smell the smoke. We classified the number of hours exposed to environmental tobacco smoke at work as 0, 1–3, and 4 or more hours.

An average of 2 weeks elapsed from the time of the in-home interview to the time of physical examination and administration of the additional tobacco-use questionnaire. To take into account potential changes in smoking status between the household interview and the medical examination, we created a variable in which participants were identified as never, former, or current smokers by their smoking status in the household interview. In the household questionnaire, respondents were asked if they had smoked at least 100 cigarettes during their lifetime. Those who answered "no" were classified as never smokers. Those who answered "yes" were then asked if they were currently smoking cigarettes. Respondents who answered "no" to this question were classified as former smokers, and respondents who answered "yes" were classified as current smokers.

Cigarette smoking status

The tobacco-use questionnaire, administered by an interviewer in the MEC, asked participants, "How many cigarettes have you smoked in the past 5 days?" In our study, we defined smokers as persons who reported smoking at least one cigarette in the previous 5 days. We calculated the average number of cigarettes smoked per day for those who had smoked one cigarette or more in the previous 5 days. In this study, we refer to nonsmokers as persons who reported that they had not smoked during the 5 days prior to the MEC interview. This study did not use a "bogus pipeline" strategy to increase the accuracy of self-reports; however, given that the NHANES III survey was not tobacco-specific, each participant was informed that his or her blood specimen was going to be tested for numerous components (i.e., hematologic assessments, dietary intake, biochemistry profile, cotinine level, and other factors).

Serum cotinine measurement

Biochemical determination of tobacco exposure was performed by measuring serum cotinine level in blood speci-

mens obtained by venipuncture in the MEC. The biochemical assay involved isotope dilution, liquid chromatography, and tandem mass spectrometry (28).

In a previous analysis of NHANES III data, Pirkle et al. (20) found that for respondents aged 4 years or older, serum cotinine level was bimodally distributed for tobacco users and nonusers, with little overlap. This separation occurred at a level of approximately 10.0–15.0 ng/ml. Thus, we decided to use a cotinine cutoff point of >15.0 ng/ml to designate smokers and ≤15.0 ng/ml to designate nonsmokers.

Statistical analysis

Logistic regression analysis was used to examine the relations between serum cotinine level and self-reported smoking status. Two types of models based on different subsets of the NHANES III data were estimated. We fitted a model using data from all respondents who indicated in the MEC interview that they were smokers—i.e., that they had smoked at least one cigarette during the previous 5 days. The binary outcome for this model was set equal to 1 if a respondent was biochemically classified as a nonsmoker (serum cotinine level ≤15.0 ng/ml). The second model was based on data from all respondents who indicated in the MEC interview that they were nonsmokers—i.e., that they had not smoked during the previous 5 days. The binary outcome for this second model was set equal to 1 if a respondent was biochemically classified as a smoker (serum cotinine level >15.0 ng/ml).

We included in the logistic regression models variables to account for the stratification and multistage design of NHANES III. All logistic model parameters were estimated using SUDAAN (33). Survey weights were used to account for different probabilities of selection within strata.

The covariates included those shown in tables 4 and 5. Additional covariates that we examined and rejected included poverty status, obesity, exposure to environmental tobacco smoke at work, and number of rooms in the house. For self-reported smokers, number of cigarettes smoked per day was categorized in various ways. Our final categorization was an attempt to make the analysis as sensitive as possible to persons who smoked on some days but not every day. The final categories were <1, 1–<2, 2–<3, 3–<5, and ≥5 cigarettes per day, on average, during the 5 days preceding the MEC examination. These cutpoints corresponded to NHANES III responses of 1–4, 5–9, 10–14, 15–24, and ≥25 cigarettes smoked during the previous 5 days.

Bivariate tables were used to examine the strength of the association between each covariate and disagreement of biochemical and self-reported smoking status. We fitted logistic regression models that included all possible combinations of the covariates. Covariates were added and deleted from the models until we found models that best characterized the disagreement patterns and produced stable log odds ratios. We did not include interactions between covariates in any of the models, because of small sample sizes. Finally, self-reported smoking status ascertained during the household interview was used only as a covariate. It was not used to calculate misclassification.

RESULTS

Study population

Compared with self-reported smokers, self-reported nonsmokers comprised a higher proportion of persons who were female, were aged 65 years or older, had 13 or more years of education, and did not live below the poverty level (table 1).

Agreement analysis

Among self-reported smokers, 92.5 percent were also biochemically classified as smokers (serum cotinine level >15.0

TABLE 1. Characteristics of participants aged 17 years or older, by smoking status,* Third National Health and Nutrition Examination Survey, United States, 1988–1994 (*n* = 15,357)

	Self-reported smokers		Self-reported nonsmokers	
	No.	%	No.	%
Sex				
Male	2,320	54	4,652	42
Female	1,954	46	6,431	58
Age (years)				
17–24	759	18	1,783	16
25–44	2,006	47	3,790	34
45–64	1,056	25	2,570	23
≥65	453	10	2,940	27
Race/ethnicity				
White	1,603	38	4,640	42
Black	1,405	33	2,714	25
Mexican-American	1,117	26	3,252	29
Other	149	3	477	4
Marital status				
Married	2,375	56	6,631	60
Not married	1,890	44	4,432	40
Unknown	9	0	20	0
Education (years)				
0–8	883	21	2,548	23
9–11	1,044	24	1,812	16
12	1,468	34	3,250	29
≥13	848	20	3,410	31
Unknown	31	1	63	1
Below federal poverty level				
Yes	1,147	27	2,126	19
No	2,726	64	7,787	70
Unknown	401	9	1,170	11
Average no. of cigarettes smoked per day				
≥5	2,915	68		
3–<5	482	11		
2–<3	215	5		
1–<2	243	6		
<1	419	10		
Total	4,274	100	11,083	100

* Self-reported in a mobile examination center (use of cigarettes during the past 5 days).

ng/ml) (table 2). Among self-reported nonsmokers, 98.6 percent were also biochemically classified as nonsmokers (serum cotinine level ≤15.0 ng/ml). According to self-reports, the prevalence of cigarette smoking in the previous 5 days was 30.2 percent (95 percent confidence interval: 28.6, 31.8), and according to serum cotinine concentrations, it was 29.0 percent (95 percent confidence interval: 27.4, 30.6).

Statistics and modeling

When we assessed agreements and discrepancies between self-report and biochemical analysis by selected sociodemographic characteristics of the participants, we found an average discrepancy of 7.5 percent among self-reported smokers (range: from 0.5 percent for persons who reported smoking an average of five or more cigarettes per day to 71.5 percent for persons who reported smoking less than one cigarette per day) (table 3). Of the self-reported smokers with serum cotinine levels less than or equal to 15.0 ng/ml, 87.5 percent smoked an average of fewer than five cigarettes per day (data not shown).

We found an average discrepancy of 1.4 percent among self-reported nonsmokers (range: from 0.6 percent for Mexican Americans to 3.1 percent for persons with 0–8 years of education) (table 3). Persons aged 65 years or older, Blacks, and persons with 0–8 years of education had a discrepancy of 2.0 percent or higher.

In the final model for self-reported smokers, after simultaneous adjustment for several selected characteristics, we found that younger persons were more likely than persons aged ≥65 years, and Blacks were less likely than Whites, to be in discrepancy with results from biochemical assessment (table 4). The average number of cigarettes smoked per day in the past 5 days was inversely and highly associated with the probability of discrepancy. The presence or absence of a smoking-related disease was not associated with the probability of discrepancy in this study.

In the final model for self-reported nonsmokers, after simultaneous adjustment for several selected characteristics, we found that discrepancy with the results of biochemical assessment was more likely among Blacks than among Whites, less likely among Mexican Americans than among Whites, less likely among persons with ≥12 years of education than among persons with 0–8 years of education, and more likely among persons who reported two or more smokers living in the home than among persons who reported no smokers living in the home (table 5). Persons who were classified as former smokers or current smokers (according to the household interview) and who reported not smoking in the previous 5 days (during the MEC visit) were more likely to be in discrepancy than those who were classified as never smokers.

DISCUSSION

The results of our study indicate that self-report smoking and biochemical measurement of serum cotinine concentration give approximately the same overall estimates of smoking prevalence. Most validity studies regarding smoking have concentrated on deception among self- reported nonsmokers (i.e., cessation studies), but we found a higher overall discrepancy between self-reports and serum cotinine levels among self-reported smokers (7.5 percent) than among self-reported nonsmokers (1.4 percent).

We can postulate several explanations for the observed discrepancy between self-reports and the biochemical measures among self-reported smokers. First, because the half-life of cotinine in the blood is 18–20 hours, the serum cotinine levels of smokers who do not smoke every day or who smoke only a few cigarettes per day will be more likely to drop to ≤15.0 ng/ml in a shorter period of time than those of smokers who smoke a greater amount of cigarettes. Approximately 34.7 percent of smokers who reported smoking an average of fewer than five cigarettes per day in the previous 5 days had a serum cotinine level less than or equal to 15.0 ng/ml. About one third of those who smoked fewer than five cigarettes per day can be considered occasional smokers (less than one cigarette per day). Occasional smokers, in particular, did not smoke daily; they smoked just a few cigarettes per day when they did smoke, and the number of cigarettes they smoked per day probably varied greatly. It is possible that for occasional smokers, serum cotinine concentration either never reached levels of >15.0 ng/ml or reached low levels (≤15.0 ng/ml) by the time the blood was drawn during the physical examination. Unfortunately, no data were collected about when the last cigarette was smoked. Recency of use is now measured in the NHANES.

TABLE 2. Agreement between self-reported smoking status* and serum cotinine concentration among participants aged 17 years or older, Third National Health and Nutrition Examination Survey, United States, 1988–1994 (*n* = 15,357)

Self-reported smoking status	Serum cotinine concentration (ng/ml)						Total no.
	>15.0			≤15.0			
	No.	%†	95% confidence interval	No.	%†	95% confidence interval	
Smoker	3,825	92.5	91.3, 93.7	449	7.5	6.3, 8.7	4,274
Nonsmoker	166	1.4	1.1, 1.7	10,917	98.6	98.3, 98.9	11,083

* Self-reported in a mobile examination center (use of cigarettes during the past 5 days).
† Weighted for the US population aged 17 years or older.

TABLE 3. Unadjusted rates* of agreement and discrepancy between self-reported† and biochemically assessed‡ cigarette smoking status among participants aged 17 years or older, Third National Health and Nutrition Examination Survey, United States, 1988–1994 (*n* = 15,357)

Characteristic	Self-reported smokers			Self-reported nonsmokers		
	Agreement (%)	95% confidence interval	Discrepancy (%)	Agreement (%)	95% confidence interval	Discrepancy (%)
Sex						
Male	92.1	90.4, 93.8	7.9	98.5	98.0, 99.0	1.5
Female	93.0	91.3, 94.7	7.0	98.7	98.4, 99.0	1.3
Age (years)						
17–24	87.0	84.0, 90.0	13.0	98.9	98.1, 99.7	1.1
25–44	92.2	90.4, 94.0	7.8	98.9	98.4, 99.4	1.1
45–64	96.1	94.4, 97.8	3.9	98.6	98.0, 99.2	1.4
≥65	98.1	96.8, 99.4	1.9	97.8	97.1, 98.5	2.2
Race/ethnicity						
White	94.1	92.8, 95.4	5.9	98.6	98.3, 98.9	1.4
Black	95.7	94.5, 96.9	4.3	98.0	97.6, 98.4	2.0
Mexican-American	72.4	69.3, 75.5	27.6	99.4	99.1, 99.7	0.6
Marital status						
Married	90.9	89.3, 92.5	9.1	98.6	98.1, 99.1	1.4
Not married	93.5	92.0, 95.0	6.5	98.5	98.1, 98.9	1.4
Education (years)						
0–8	91.2	88.0, 94.4	8.8	96.9	95.6, 98.2	3.1
9–11	93.5	91.1, 95.9	6.5	98.2	97.6, 98.8	1.8
12	94.5	92.7, 96.3	5.5	98.5	97.9, 99.1	1.5
≥13	89.2	86.6, 91.8	10.8	99.1	98.8, 99.4	0.9
Below federal poverty level						
Yes	91.7	88.9, 94.5	8.3	98.4	97.6, 99.2	1.6
No	92.8	91.3, 94.3	7.2	98.7	98.4, 99.0	1.3
Average no. of cigarettes smoked per day						
≥5	99.5	99.1, 99.9	0.5			
3–<5	88.0	83.2, 92.8	12.0			
2–<3	93.3	90.1, 96.5	6.7			
1–<2	58.8	43.8, 73.8	41.2			
<1	28.5	21.2, 35.8	71.5			
Total	92.5	91.3, 93.7	7.5	98.6	98.3, 98.9	1.4

* Percentages are weighted for the US population aged 17 years or older.
† Self-reported in a mobile examination center (use of cigarettes during the past 5 days).
‡ Serum cotinine concentration: >15.0 ng/ml indicated a smoker and ≤15.0 ng/ml indicated a nonsmoker.

Second, persons who reported smoking fewer than five cigarettes per day may in fact have been nonsmokers who provided inaccurate information about their smoking status. However, we observed a pattern at very low levels of cigarette smoking: the lower the number of cigarettes smoked per day, the greater the likelihood of discrepancy. Thus, deception seems unlikely.

Third, despite the observations of Pirkle et al. (20), the selected cotinine cutoff point of >15.0 ng/ml may be too high to identify all smokers. When we reanalyzed the data using a cutoff of >10.0 ng/ml for active smokers and ≤10.0 ng/ml for nonsmokers, the discrepancy for self-reported smokers dropped from 7.5 percent to 6.3 percent but the overall results for predictors of discrepancy did not change. Previous studies that have measured cotinine levels in the blood have used different cutoff points, usually 10.0–15.0 ng/ml, to detect active smoking (15, 34–38). We did not calculate a receiver operating characteristic curve to choose the cutoff that maximized sensitivity and specificity, because the receiver operating characteristic curve may be affected by inaccurate self-reported smoking information. A receiver operating characteristic curve is a graph of sensitivity (the true-positive fraction) against the complement of specificity (the false-positive fraction) for all possible cutoff points (39).

Previous studies have found that, in the United States, Blacks have higher serum cotinine levels than Whites do at similar levels of smoking (34, 40–44). Thus, self-reported

TABLE 4. Correlates of discrepancy* among self-reported smokers† aged 17 years or older, Third National Health and Nutrition Examination Survey, United States, 1988–1994 (*n* = 3,615)

Variable	Odds ratio	95% confidence interval	*p* value
Sex			
Male‡			
Female	0.76	0.48, 1.19	0.23
Age (years)			
17–24	2.68	1.23, 5.86	0.01
25–44	3.28	1.60, 6.74	0.00
45–64	3.03	1.04, 8.82	0.04
≥65‡			
Race/ethnicity			
White‡			
Black	0.25	0.14, 0.42	0.00
Mexican-American	1.30	0.84, 2.01	0.24
Marital status			
Married‡			
Not married	1.23	0.67, 2.25	0.49
Education (years)			
0–8‡			
9–11	1.10	0.61, 2.01	0.74
12	1.32	0.73, 2.39	0.36
≥13	1.34	0.79, 2.29	0.27
No. of cigarettes smoked per day			
≥5‡			
3–<5	36.58	15.55, 86.08	0.00
2–<3	14.79	4.83, 45.29	0.00
1–<2	165.45	50.34, 543.74	0.00
<1	567.35	248.86, 1,293.46	0.00

* Self-reported smoking of one cigarette or more during the 5 days preceding the tobacco use questionnaire interview and a serum cotinine concentration of ≤15.0 ng/ml. Blood samples were drawn on the same day as the interviews were performed.

† Self-reported in a mobile examination center (use of cigarettes during the past 5 days).

‡ Referent.

TABLE 5. Correlates of discrepancy* among self-reported nonsmokers† aged 17 years or older, Third National Health and Nutrition Examination Survey, United States, 1988–1994 (*n* = 11,083)

Variable	Odds ratio	95% confidence interval	*p* value
Sex			
Male‡			
Female	1.20	0.77, 1.87	0.43
Age (years)			
17–24	0.72	0.34, 1.56	0.42
25–44	0.85	0.43, 1.68	0.64
45–64	0.72	0.41, 1.29	0.26
≥65‡			
Race/ethnicity			
White‡			
Black	1.73	1.17, 2.54	0.01
Mexican-American	0.34	0.17, 0.68	0.00
Education (years)			
0–8‡			
9–11	0.61	0.34, 1.10	0.11
12	0.44	0.25, 0.78	0.01
≥13	0.31	0.16, 0.62	0.00
Tobacco-related disease			
No‡			
Yes	1.04	0.65, 1.65	0.88
No. of persons in household who smoked in the home			
0‡			
1	1.50	0.80, 2.82	0.22
≥2	2.31	1.08, 4.96	0.03
Smoking status in the household interview			
Never smoker‡			
Former smoker	4.78	2.91, 7.85	0.00
Current smoker	12.18	4.00, 37.1	0.00

* Participant self-reported in a mobile examination center that he or she did not smoke cigarettes during the 5 days preceding the tobacco use questionnaire interview and had a serum cotinine concentration of >15.0 ng/ml. Blood samples were drawn on the same day as the interviews were performed.

† Self-reported in a mobile examination center (use of cigarettes during the past 5 days).

‡ Referent.

smokers who have higher serum cotinine concentrations at similar levels of cigarette smoking will be less likely to be in discrepancy with the selected biochemical cutoff. Among self-reported smokers, our finding of lower discrepancy among Blacks than among Whites is consistent with these reports. It is possible that different cutoff points for cotinine concentration may be needed for Blacks and Whites. This issue should be studied further; differences in the characteristics of the cigarettes smoked, smoking topography, or the pharmacokinetics of nicotine should be considered (43). Information about differences in serum cotinine concentrations between Whites or Blacks and Mexican Americans is scarce.

We can also postulate a number of explanations for the discrepancy between self-reports and the results of biochemical assessment among self-reported nonsmokers. First, Blacks, as compared with Whites, may be highly exposed to environmental tobacco smoke or may differ in terms of nicotine pharmacokinetics. These two possibilities would help to explain why Blacks who reported themselves to be nonsmokers were more likely to have a cotinine level greater than 15.0 ng/ml. Indeed, several studies have found differences in serum cotinine concentrations between Black and White nonsmokers (20, 42, 45–48). In these studies, Blacks had higher cotinine levels than did Whites, even after environmental tobacco smoke exposure and other factors were taken into account. Because racial differences in nicotine pharmacokinetics (44) and genetic polymorphisms involved (49) exist, different cutoff points are probably

needed for each racial group. Second, among all self-reported nonsmokers who lived with two or more smokers who smoked inside the home, the high concentration of serum cotinine may have been the result of high exposure to environmental tobacco smoke. Third, among respondents who reported being current smokers (during the household interview) but reported not smoking in the 5 days before their blood was drawn (in the MEC interview), the presumably high serum cotinine level (on average, a regular smoker has a serum cotinine concentration of ≥250 ng/ml) may have not dropped to ≤15.0 ng/ml by the time of the MEC interview. Fourth, respondents may have provided accurate information in the household interview but not in the MEC interview. Fifth, perhaps most of the persons who reported not smoking in the previous 5 days and had serum cotinine levels greater than 15.0 ng/ml provided inaccurate information about their smoking status. Eighty-three percent of self-reported nonsmokers who were determined by the biochemical measure to be smokers had a serum cotinine level greater than or equal to 25.0 ng/ml (the median cotinine level for non-tobacco users in NHANES III was <2.0 ng/ml (20)), and 38 percent had a serum cotinine level greater than or equal to 100.0 ng/ml (data not shown). A value greater than 100.0 ng/ml is inconsistent with either high exposure to environmental tobacco smoke or occasional smoking. Sensitivity to the social stigma associated with smoking has been cited as one reason why people might underreport their smoking status (48).

Overall, in this study there were higher levels of discrepancy among self-reported smokers than among self-reported nonsmokers. Smoking patterns, including the extent of nicotine dosing, may be the main explanation for the 7.5 percent discrepancy among self-reported smokers, and social stigma may be the main explanation for the 1.4 percent discrepancy among self-reported nonsmokers. The private setting used to collect information on smoking status in NHANES III may have contributed to the low levels of discrepancy observed among self-reported nonsmokers.

To our knowledge, this study is the first to provide quantification of the discrepancy between self-reported smoking information and data from a biochemical measure for the US population aged 17 years or older. Although biochemical assessment is generally believed to be more reliable than self-reports in assessing smoking status, its validity at very low levels of cigarette smoking may be questioned. Specifically, the validity of serum cotinine concentration as an objective measure for detecting true smokers should be carefully considered when trying to assess smoking status among groups with a substantial proportion of persons who smoke fewer than five cigarettes per day (i.e., Mexican Americans). In general, however, self-reports appear to be a very good indicator of actual smoking status.

REFERENCES

1. Patrick DL, Cheadle A, Thompson DC, et al. The validity of self-reported smoking: a review and meta-analysis. Am J Public Health 1994;84:1086–93.
2. Wagenknecht LE, Burke GL, Perkins LL, et al. Misclassification of smoking status in the CARDIA Study: a comparison of self-report with serum cotinine levels. Am J Public Health 1992; 82:33–6.
3. Klesges LM, Klesges RC, Cigrang JA. Discrepancies between self-reported smoking and carboxyhemoglobin: an analysis of the second National Health and Nutrition Survey. Am J Public Health 1992;82:1026–9.
4. Velicer WF, Prochaska JO, Rossi JS, et al. Assessing outcome in smoking cessation studies. Psychol Bull 1992;111:23–41.
5. Perez-Stable EJ, Marin G, Marin BV, et al. Misclassification of smoking status by self-reported cigarette consumption. Am Rev Respir Dis 1992;145:53–7.
6. Klebanoff MA, Levine RJ, Clemens JD, et al. Serum cotinine concentration and self-reported smoking status during pregnancy. Am J Epidemiol 1998;148:259–62.
7. Pechacek TF, Murray DM, Luepker RV, et al. Measurement of adolescent smoking behavior: rationale and methods. Behav Med 1984;7:123–40.
8. Ahijevych KL, Wewers ME. Patterns of cigarette consumption and cotinine levels among African American women smokers. Am J Respir Crit Care Med 1994;150:1229–33.
9. Slattery ML, Hunt SC, French TK, et al. Validity of cigarette smoking habits in three epidemiologic studies in Utah. Prev Med 1989;18:11–19.
10. Benowitz NL. The use of biologic fluid samples in assessing tobacco smoke consumption. NIDA Res Monogr 1983;48: 6–26.
11. Muranaka H, Higashi E, Itani S, et al. Evaluation of nicotine, cotinine, thiocyanate, carboxyhemoglobin, and expired carbon monoxide as biochemical tobacco smoke uptake parameters. Int Arch Occup Environ Health 1988;60:37–41.
12. Waage H, Silsand T, Urdal P, et al. Discrimination of smoking status by thiocyanate and cotinine in serum, and carbon monoxide in expired air. Int J Epidemiol 1992;21:488–93.
13. Matsukura S, Sakamoto N, Seino Y, et al. Cotinine excretion and daily cigarette smoking in habituated smokers. Clin Pharmacol Ther 1979;25:555–61.
14. Wilcox RG, Hughes J, Roland J. Verification of smoking history in patients after infarction using urinary nicotine and cotinine measurements. BMJ 1979;2:1026–8.
15. Williams CL, Eng A, Botwin GJ, et al. Validation of students' self-reported cigarette smoking levels with plasma cotinine levels. Am J Public Health 1979;69:1272–4.
16. Haley NJ, Axelrad CM, Tilton KA. Validation of self-reported smoking behavior: biochemical analyses of cotinine and thiocyanate. Am J Public Health 1983;73:1204–7.
17. Hill P, Haley NJ, Wynder EL. Cigarette smoking: carboxyhemoglobin, plasma nicotine, cotinine, and thiocyanate versus self-reported smoking data and cardiovascular disease. J Chronic Dis 1983;36:439–49.
18. Jarvis MJ, Russell MA. Measurement and estimation of smoke dosage to non-smokers from environmental tobacco smoke. Eur J Respir Dis Suppl 1984;133:68–75.
19. Wall MA, Johnson J, Jacob P, et al. Cotinine in the serum, saliva, and urine of nonsmokers, passive smokers, and active smokers. Am J Public Health 1988;78:699–701.
20. Pirkle JL, Flegal KM, Bernert JT, et al. Exposure of the US population to environmental tobacco smoke: The Third National and Nutrition Examination Survey, 1988 to 1991. JAMA 1996;275:1233–40.
21. National Center for Chronic Disease Prevention and Health Promotion. Nicotine addiction: a report of the Surgeon General. Rockville, MD: Office on Smoking and Health, National Center for Chronic Disease Prevention and Health Promotion, 1988. (DHHS publication no. 88-8406).
22. Jarvis MJ, Russell MA, Benowitz NL, et al. Elimination of cotinine from body fluids: implications for noninvasive measurement of tobacco smoke exposure. Am J Public Health 1988;78:696–8.
23. Haley NJ, Axelrod CM, Tilton KA. Validation of self- reported smoking behavior: biochemical analyses of cotinine and thiocyanate. Am J Public Health 1988;73:1204–7.
24. Jarvis MJ, Tunstall-Pedoe H, Feyerabend C, et al. Comparison

of tests used to distinguish smokers from nonsmokers. Am J Public Health 1987;77:1435–8.
25. Patrick DL, Cheadle A, Thomson DC, et al. The validity of self-reported smoking: a review and meta-analysis. Am J Public Health 1994;84:1086–93.
26. Benowitz NL, Perez-Stable E, Herrera B, et al. African American-Caucasian differences in nicotine and cotinine metabolism. Clin Pharmacol Ther 1995;57:159.
27. Benowitz NL, Kuyt F, Jacob P, et al. Cotinine disposition and effects. Clin Pharmacol Ther 1983;34:604–11.
28. Bernert JT, Sosnoff C, Turner WE, et al. Development of a rapid and sensitive method for serum cotinine analysis as a marker of exposure to environmental tobacco smoke. Clin Chem 1994;40:1075.
29. National Center for Health Statistics. Plan and operation of the Third National Health and Nutrition Examination Survey, 1988–1994. Hyattsville, MD: National Center for Health Statistics, 1994. (Vital and health statistics, series 1, no. 32) (DHHS publication no. 94-1308).
30. US Census Bureau. Poverty thresholds: poverty thresholds by size of family and number of children [1985–2000]. Current Population Survey (revised January 26, 2001). Washington, DC: US Census Bureau, 2001. (http://www.census.gov/hhes/poverty/threshld.html).
31. World Health Organization. Obesity: preventing and managing the global epidemic. Report of a WHO consultation on obesity, Geneva, 3–5 June 1997. Geneva, Switzerland: World Health Organization, 1998.
32. National Heart, Lung, and Blood Institute. Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults: the evidence report. Bethesda, MD: National Heart, Lung, and Blood Institute, 1998. (NIH publication no. 98-4083).
33. Shah BV. Software for survey data analysis (SUDAAN), version 6.40. Research Triangle Park, NC: Research Triangle Institute, 1995.
34. Wagenknecht LE, Cutter GR, Haley NJ, et al. Racial differences in serum cotinine levels among smokers in the Coronary Artery Risk Development in (Young) Adults Study. Am J Public Health 1990;80:1053–6.
35. Haddow JE, Palomakii GE, Knight GJ. Use of serum cotinine to assess the accuracy of self-reported non-smoking. (Letter). BMJ 1986; 293:1306.
36. Pojer T, Whitefield JB, Poulos V, et al. Carboxyhemoglobin, cotinine, and thiocyanate assay compared for distinguishing smokers from non-smokers. Clin Chem 1984;30:1377–80.
37. Slattery ML, Hunt SC, French TK, et al. Validity of cigarette smoking habits in three epidemiologic studies in Utah. Prev Med 1989;18:11–19.
38. van Vunakis H, Tashkin DP, Rigas B, et al. Relative sensitivity and specificity of salivary and serum cotinine in identifying tobacco-smoking status of self-reported nonsmokers and smokers of tobacco and/or marijuana. Arch Environ Health 1989;44:53–8.
39. Etzel RA. A review of the use of saliva cotinine as a marker of tobacco smoke exposure. Prev Med 1990;19:190–7.
40. Clark PI, Gautman S, Gerson L. Effect of menthol cigarettes on biochemical markers of smoke exposure among Black and White smokers. Chest 1996;110:1194–8.
41. English PB, Eskenazi B, Christianson RE. Black-white differences in serum cotinine levels among pregnant women and subsequent effects on infant birthweight. Am J Public Health 1994;84:1439–43.
42. Pattishall EN, Strope GL, Etzel RA, et al. Serum cotinine as a measure of tobacco smoke exposure in children. Am J Dis Child 1985;139:1101–4.
43. Caraballo RS, Giovino GA, Pechacek TF, et al. Racial and ethnic differences in serum cotinine levels of cigarette smokers: Third National Health and Nutrition Examination Survey, 1988–1991. JAMA 1998;280:135–9.
44. Pérez-Stable EJ, Herrera B, Jacob P III, et al. Nicotine metabolism and intake in black and white smokers. JAMA 1998; 280:152–6.
45. Knight JM, Eliopoulus C, Klein J, et al. Passive smoking in children. Chest 1996;109:446–50.
46. Wagenknecht LE, Manolio TA, Sidnet S, et al. Environmental tobacco smoke exposure as determined by cotinine in black and white young adults. Environ Res 1993;63:39–46.
47. Crawford FG, Mayer J, Santella RM, et al. Biomarkers of environmental tobacco smoke in pre-school children and their mothers. J Natl Cancer Inst 1994;86:1398–402.
48. National Center for Chronic Disease Prevention and Health Promotion. Reducing the health consequences of smoking: 25 years of progress. Rockville, MD: Office on Smoking and Health, National Center for Chronic Disease Prevention and Health Promotion, 1989. (DHHS publication no. 89-8411).
49. Fernandez-Salguero P, Hoffman SM, Cholerton S, et al. A genetic polymorphism in coumarin 7-hydroxylation: sequence of the human *CYP2A* genes and identification of variant *CYP2A* alleles. Am J Hum Genet 1995;57:651–60.