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Active and passive smoking and blood lead levels in U.S. adults: data from the Third National Health and Nutrition Examination Survey

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Lead is a component of tobacco and tobacco smoke. We examined the relationship between current, former, and passive smoking and blood lead levels in a nationally representative sample of 16,458 U.S. adults, aged 17 years or older, who participated in the Third National Health and Nutrition Examination Survey (1988–1994). We used linear and logistic regression modeling, adjusting for known covariates, to determine the relationship between smoking and blood lead levels. Geometric mean blood lead levels were 1.8 $\mu\text{g}/\text{dl}$, 2.1 $\mu\text{g}/\text{dl}$, and 2.3 $\mu\text{g}/\text{dl}$ in never-smokers with no, low, and high cotinine levels, respectively. Levels were 2.9 $\mu\text{g}/\text{dl}$ in former smokers and 3.5 $\mu\text{g}/\text{dl}$ in current smokers. The adjusted linear regression model showed that geometric mean blood lead levels were 30% higher (95% $CI=24\%–36\%$) in adults with high cotinine levels than they were in those with no detectable cotinine. Active and passive smoking is associated with increased blood lead levels in U.S. adults.

Introduction

Tobacco smoke contains over 4,000 compounds, including heavy metals such as lead (California Environmental Protection Agency, 1997; Jenkins, 1986). Active tobacco smokers have higher blood lead levels than do nonsmokers (Grasmick, Huel, Moreau, & Sarmini, 1985; Hernandez-Avila et al., 2000; Shaper et al., 1982), but evidence for similar effects in nonsmokers exposed to second-hand smoke (SHS) is limited (Berglund, Lind, Sorensen, & Vahter, 2000; Farias et al., 1998; Willers, Schutz, Attewell, & Skerfving, 1988).

Tobacco smoke exposure can be measured in different ways, ranging from the use of questionnaires to measuring tobacco tracers in air to measurement of biomarkers in individuals (Jaakkola & Jaakkola, 1997). Use of the biomarker cotinine (a metabolite of nicotine and indicator of SHS exposure) can reduce misclassification, allowing one to compare a high-exposure group with a low-exposure group (Benowitz, 1996). We had previously demonstrated that children with high cotinine levels had higher blood lead levels than did children with low cotinine levels (Mannino, Albalak, Grosse, & Repace, 2003).

The present study analyzed data among adults aged 17 years or older, from the Third National Health and Nutrition Examination Survey (NHANES III), who had blood lead levels measured. We hypothesized that adults with either active or passive exposure to tobacco smoke would have elevated blood lead levels compared with adults with no exposure. We used smoking history data and serum cotinine levels as the main basis for classifying adults into tobacco exposure groups and compared blood lead levels in adults with evidence of active or passive smoking with those who had no exposure.

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Method

NHANES III was conducted from 1988 through 1994 by the National Center for Health Statistics of the Centers for Disease Control and Prevention in Atlanta, Georgia (National Center for Health Statistics, 1994). NHANES III was approved by the National Center for Health Statistics's Institutional Review Board. In this survey, a stratified multistage clustered probability design was used to select a representative sample of the civilian, noninstitutionalized U.S. population. The final sample comprised 81 geographic sites. Survey participants completed extensive questionnaires in the household and a comprehensive physical examination at a specially equipped mobile examination center.

Subjects and demographics

For the present study, the analysis was limited to adults aged 17 years or older for whom serum cotinine levels and blood lead levels were obtained. Adults were included in the analysis if data were complete on their race/ethnicity, age, sex, year of house construction, occupation, blood lead level, whether cigarettes were smoked in the home or at the worksite, and cotinine levels. Of the 20,050 people who participated in NHANES III, 1,888 did not participate in the examination, 1,599 did not have cotinine levels obtained, 29 did not have blood lead levels, and 76 were missing data on other variables, leaving 16,458 subjects in the final dataset. The excluded subjects were similar to the 16,458 participants for all covariates except for age, poverty status, and family size.

Cotinine levels

Serum cotinine levels were determined using high-performance liquid chromatography atmospheric-pressure chemical ionization tandem mass spectrometry, as is described elsewhere (Pirkle et al., 1996). We stratified the never-smokers into three groups on the basis of cotinine levels: those below the limit of detection of 0.050 ng/ml (no cotinine), with the remainder stratified into two approximately equal groups, those with cotinine levels of 0.050–0.222 ng/ml (low cotinine) or 0.223–14.9 ng/ml (high cotinine). Subjects who reported current use of cigarettes, cigars, or pipes and those with cotinine levels of 15 ng/ml or greater were classified as current smokers. Subjects who reported previous use of cigarettes, cigars, or pipes were classified as former smokers. Some 84% of former smokers reported smoking cessation at least 1 year prior to the survey.

Blood lead measurements

A 1-ml sample of EDTA-anticoagulated whole blood was collected by venipuncture from participants

during the physical examination. Blood samples were frozen and shipped on dry ice for analysis to the NHANES Laboratory, Division of Environmental Health Laboratory Sciences, National Center for Environmental Health, Centers for Disease Control and Prevention. The blood samples remained frozen at -20°C until they were analyzed. Lead was measured by graphite furnace atomic absorption spectrophotometry (GFAAS; Miller, Paschal, Gunter, Stroud, & D'Angelo, 1987). The GFAAS method included deuterium background correction and had a limit of detection of $1.0\text{ }\mu\text{g/dl}$. The lead result is the mean of duplicate measurements. The blood lead measurements were calibrated using standards prepared from lead nitrate Standard Reference Material 928 obtained from the National Institute of Standards and Technology, Gaithersburg, Maryland.

Variable definition

Race/ethnicity was categorized as non-Hispanic White, non-Hispanic Black, Mexican American, or other and was determined by self-classification. Country of birth also was self-reported and was classified as United States, Mexico, or other. The poverty index ratio was used to divide the sample into four strata: <1 (the poorest), 1 to <2 , ≥ 2 , or unknown (National Center for Health Statistics, 1994). Year of housing construction was determined by self-report and was defined as before 1946, 1946–1973, after 1973, or don't know. Menopausal status was self-reported and classified as premenopausal, surgical menopause, postmenopausal, or unknown. Reported tobacco smoke exposure in the home was defined as "yes" if the response to the question "Does anyone who lives here smoke cigarettes in the home?" was positive and "no" if the response was negative. Reported smoke exposure at work (asked only of people aged 64 years or younger) was positive if a person reported at least 1 hr of exposure in response to the question "At work, how many hours per day are you close enough to people who smoke so that you can smell the smoke?" Occupation was determined using the most recent job and classified into one of six categories: agriculture, mining/manufacturing, transport, service, unemployed, or retail. We used six age strata in the analysis: 17–24 years, 25–44 years, 45–64 years, 65–74 years, 75–84 years, and 85+ years.

Data analyses

We performed statistical analyses using SAS and SUDAAN software packages. We calculated all estimates using the sampling weight to represent U.S. adults. The purpose of the sampling weight

calculations was to adjust for unequal probabilities of selection and to account for nonresponse. The weights were poststratified to the U.S. population as estimated by the U.S. Bureau of the Census.

Geometric mean blood lead levels were calculated as the antilog of the mean of the log of the blood lead levels. We modeled the relationship between SHS exposure and lead levels using linear and logistic regression in both univariate and multivariate models adjusting for covariates. The natural log of the blood lead levels was used as the dependent variable in the linear regressions, and the results were exponentiated to yield a percentage increase from the referent values. Blood lead levels of 5 $\mu\text{g}/\text{dl}$ or greater were the categorical dependent variable in the logistic regressions. We used cotinine levels and reported SHS exposures as our determinant of tobacco smoke exposure, but we did not include them in the models together because of collinearity. Covariates included age, poverty index, race/ethnicity, country of birth, smoking status, pack-years of smoking, menopausal status, year home built, and occupation. Multivariate models were evaluated for interaction between tobacco smoke exposure and the key covariates of age, race/ethnicity, sex, and country of birth.

Results

Our final dataset contained 16,458 adults representing an estimated 174 million adults aged 17 years or older in the United States. The distribution of the covariates and SHS exposure are presented in Table 1. As expected and reported previously, factors in adults associated with increased blood lead levels and a higher proportion of elevated blood lead levels included older age, Black race, male sex, poverty, older housing, menopausal status, country of birth, and occupation (Elmarsafawy et al., 2002; Pirkle et al., 1998).

Among never-smokers, 52% reported neither home nor work exposure to SHS. In this subgroup, however, 26% were in the high cotinine exposure group. Conversely, among the never-smokers with reported home or work SHS exposure, 72% were in the high cotinine exposure group.

Current, former, and passive smokers all had elevated blood lead levels when compared with never-smokers with no SHS exposure, when using serum cotinine as the indicator of exposure (Table 2). For example, blood lead levels were 30% higher (95% $CI=24\%-36\%$) in never-smokers with high cotinine levels, compared with those with no cotinine in their blood, in models adjusting for covariates (Table 2). By contrast, reported smoke exposure was associated with significantly higher blood lead levels in only one of the three exposure groups (those reporting exposure only at home, 11% [95% $CI=2\%-19\%$;

Table 2). The difference between reported and measured SHS exposure is also illustrated in Figure 1, which shows significantly higher blood lead levels in all age groups when cotinine was used as the metric of exposure but not with reported SHS exposure.

Overall, 18.7% of the population had blood lead levels of 5 $\mu\text{g}/\text{dl}$ or greater. Logistic regression models predicting blood lead levels of at least 5 $\mu\text{g}/\text{dl}$, adjusted for covariates, indicated that current, former, and passive smokers all had a significantly increased risk of elevated blood lead levels (Tables 3 and 4). Analysis of this relationship suggested interactions by sex and race/ethnicity. When the data were stratified by sex, we found that the association between current, former, and passive smoking and elevated blood lead levels was similar between men and women (Table 3). When the data were stratified by race/ethnicity, we found that the association between current, former, and passive smoking and elevated blood lead levels was stronger in Whites than in Blacks or Mexican Americans (Table 4).

Discussion

The present study demonstrates that both active and passive tobacco smoke exposure is associated with higher blood lead levels in U.S. adults, when compared with never-smokers with no SHS exposure. Use of the tobacco smoke biomarker cotinine showed evidence of a stronger and more consistent relationship between passive smoking and blood lead levels, compared with the use of reported tobacco smoke exposure (Table 3). Cotinine levels are a function of both exposure and absorption and provide a better indicator of recent exposure (Benowitz, 1996; Jaakkola & Jaakkola, 1997).

As noted in many previous studies, including a previous analysis of the present dataset, factors such as older age of housing, Black race, male sex, lower educational level, poverty, and occupation were associated with higher blood lead levels (Brody et al., 1994; Tumpowsky, Davis, & Rabin, 2000). One study found that mean blood levels, as related to patella bone lead levels, were higher in the wintertime than in the summertime (Oliveira, Aro, Sparrow, & Hu, 2002). Although the authors proposed decreased exposure to sunlight and enhanced bone resorption as the explanation for this phenomenon, SHS exposure—which tends to be higher in the winter when people are spending more time indoors—is a possible explanation (Murray & Morrison, 1988).

Lead is present in tobacco (Rickert & Kaiserman, 1994) and tobacco smoke (Connally, 2000; Rickert & Kaiserman, 1994). Current estimates from Canada are that each cigarette contains approximately 600–800 ng of lead but during the time NHANES III was

Table 1. Study population stratified by covariates with the weighted proportion in each stratum, the age-adjusted geometric mean blood lead level for each stratum, and the age-adjusted proportion of adults in each stratum with blood lead levels of 5 $\mu\text{g}/\text{dl}$ or greater. From the Third National Health and Nutrition Examination Survey, 1988–1994.

Variable	Number of subjects	Weighted population (millions)	Geometric mean blood lead level (geometric standard error)	Proportion with blood lead level $\geq 5 \mu\text{g}/\text{dl}$ (standard error)
Age (years)				
85+	389	1.7	3.8 (0.1)	31.2 (2.8)
75–84	1,354	7.7	3.7 (0.1)	30.5 (1.8)
65–74	2,026	16.4	3.6 (0.1)	31.1 (2.1)
45–64	3,866	43.7	3.3 (0.1)	25.0 (1.7)
25–44	6,140	77.5	2.4 (0.1)	14.4 (1.1)
17–24	2,683	27.3	1.8 (0.1)	8.6 (1.2)
Race/ethnicity				
White	6,713	132.8	2.6 (0.1)	16.9 (1.4)
Black	4,515	18.6	3.2 (0.1)	28.2 (1.1)
Mexican American	4,561	9.2	2.9 (0.1)	24.3 (1.5)
Other	669	13.7	2.8 (0.1)	21.6 (2.3)
Country of birth				
United States	12,986	150.3	2.6 (0.1)	18.1 (1.3)
Mexico	2,277	5.0	3.4 (0.1)	31.7 (1.9)
Other	1,195	19.0	2.8 (0.1)	20.0 (1.3)
Sex				
Male	7,756	84.4	3.5 (0.1)	28.4 (1.8)
Female	8,702	89.9	2.1 (0.1)	9.8 (0.8)
Menopausal status				
Premenopause	4,528	52.1	1.7 (0.1)	5.6 (1.3)
Postmenopause	1,710	16.9	2.5 (0.1)	8.1 (0.1)
Surgical menopause	1,773	15.6	3.2 (0.1)	18.3 (5.1)
Unkown	691	5.2	2.5 (0.1)	11.8 (2.2)
Poverty index ratio (income status)				
<1	3,625	21.1	2.9 (0.1)	27.5 (1.6)
1 to <2	4,145	34.4	2.7 (0.1)	22.7 (1.6)
Missing	1,516	11.0	2.8 (0.1)	18.3 (2.0)
≥ 2	7,172	107.8	2.6 (0.1)	16.1 (1.2)
Year home built				
Unknown	1,757	14.9	3.0 (0.2)	23.7 (1.6)
<1946	3,497	38.1	3.0 (0.1)	24.6 (1.1)
1946–1973	7,274	68.2	2.6 (0.1)	18.5 (0.9)
1973+	3,930	53.1	2.5 (0.1)	13.9 (0.8)
Job category				
Agriculture	693	6.4	2.8 (0.2)	20.1 (1.8)
Mining/manufacturing	2,303	29.5	3.1 (0.1)	24.9 (2.7)
Transport	527	7.4	2.8 (0.1)	29.0 (3.0)
Service	4,456	56.4	2.4 (0.1)	18.3 (1.3)
Unemployed	6,697	52.2	2.9 (0.7)	16.4 (1.0)
Retail	1,782	22.3	2.3 (0.1)	17.6 (1.6)
SHS exposure category (cotinine based)				
Current smoker	5,143	59.3	3.4 (0.1)	29.5 (1.6)
Former smoker	3,383	37.3	2.9 (0.1)	16.7 (1.6)
High cotinine	3,267	30.9	2.3 (0.1)	14.9 (1.3)
Low cotinine	3,269	33.2	2.1 (0.1)	10.0 (0.1)
No cotinine	1,396	13.6	1.8 (0.1)	4.7 (0.5)
SHS exposure category (self-reported)				
Current smoker	5,143	59.3	3.4 (0.1)	29.5 (1.6)
Former smoker	3,383	37.3	2.9 (0.1)	16.7 (1.6)
+Work, +Home	332	3.2	2.1 (0.1)	17.3 (2.7)
+Work, –Home	1,164	13.9	2.2 (0.1)	15.1 (2.3)
–Work, +Home	1,076	8.2	2.1 (0.1)	14.7 (1.7)
–Work, –Home	5,360	52.4	2.1 (0.1)	8.7 (0.7)
Total	16,458	174.3	2.6 (0.1)	18.7 (1.2)

Note. SHS, second-hand smoke.

conducted may have contained as much as 1400 ng of lead (Rickert & Kaiserman, 1994). Lead levels in tobacco have decreased with decreasing ambient air lead levels (Rickert & Kaiserman, 1994). The recently completed Massachusetts Benchmark Study estimated that mainstream tobacco smoke contains 60 ng of lead per cigarette and that sidestream smoke contains 5–10 ng of lead per cigarette

(Connally, 2000). This lead is likely to be associated with the particulate fraction of tobacco smoke and absorbed through the respiratory system. A recent study detected higher mean lead levels in the indoor air of homes in which smoking occurs, compared with levels in homes where no smoking occurs (21.8 ng/m³ vs. 7.8 ng/m³; Bonanno, Freeman, Greenberg, & Liroy, 2001). In addition, lead in the

Table 2. Percent change (corresponding to exponentiated regression coefficients) and 95% confidence intervals from linear regression analysis examining the relation of log blood lead levels and covariates in U.S. adults both in univariate and multivariate^a models. From the Third National Health and Nutrition Examination Survey, 1988–1994.

Variable	Univariate model	95% Confidence interval	Multivariate model ^b	95% Confidence interval
Age (years)				
85+	110	99, 120	132	121, 142
75–84	108	99, 116	112	104, 120
65–74	101	95, 107	95	89, 102
45–64	82	76, 88	73	67, 79
25–44	32	26, 38	32	28, 36
17–24	Referent		Referent	
Race/ethnicity				
White	Referent		Referent	
Black	15	7, 23	20	12, 28
Mexican American	7	–3, 18	1	–9, 11
Other	5	–3, 13	8	0, 16
Country of birth				
United States	Referent			
Mexico	28	18, 39	48	42, 54
Other	8	0, 16	13	5, 21
Sex				
Male	67	65, 69	73	69, 77
Female	Referent		Referent	
Menopausal status				
Premenopause	Referent			
Postmenopause	54	48, 60	17	11, 23
Surgical menopause	93	87, 100	39	33, 45
Unknown	49	41, 57	19	10, 27
Poverty index ratio (income status)				
<1	15	9, 21	12	6, 18
1 to <2	7	3, 11	1	–3, 5
Missing	11	4, 17	3	–1, 7
≥2	Referent		Referent	
Year home built				
Unknown	20	7, 32	12	1, 22
<1946	30	19, 40	20	12, 28
1946–1973	12	3, 20	3	–3, 9
1973+	Referent		Referent	
Job category				
Agriculture	23	11, 36	2	–8, 12
Mining/manufacturing	35	27, 43	13	7, 19
Transport	22	12, 32	7	–1, 15
Service	4	–4, 12	8	4, 12
Unemployed	25	19, 31	5	1, 9
Retail	Referent		Referent	
SHS exposure category (cotinine based)				
Current smoker	95	89, 102	82	76, 88
Former smoker	63	55, 71	35	29, 41
High cotinine	30	22, 38	30	24, 36
Low cotinine	20	12, 28	21	15, 27
No cotinine	Referent		Referent ^b	
SHS exposure category (self-reported)				
Current smoker	65	61, 69	51	47, 55
Former smoker	39	35, 43	13	9, 17
+Work, +Home	0	–15, 15	9	–3, 22
+Work, –Home	5	–3, 13	6	0, 12
–Work, +Home	4	–4, 12	11	2, 19
–Work, –Home	Referent		Referent	

Note. SHS, second-hand smoke.

^aMultivariate model adjusted for age group, poverty index ratio, race/ethnicity, country of birth, sex, menopausal status (for women), year home built, occupation, and tobacco exposure category (either cotinine based or self-reported).

^bReferent values range from 1.7 $\mu\text{g}/\text{dl}$ to 2.5 $\mu\text{g}/\text{dl}$ and can be found in Table 1.

Figure 1. Geometric mean blood lead levels and geometric standard errors (brackets) by age among U.S. adults aged 17 years or more, stratified by smoking status and cotinine levels (A) and smoking status and reported second-hand smoke exposure (B). All passive smoke exposure categories are restricted to never-smokers. From the Third National Health and Nutrition Examination Survey, 1988–1994.

particulate fraction could settle onto surfaces and food where it has the potential to re-expose people through either the gastrointestinal route or the respiratory system (Lanphear et al., 1996).

A puzzling finding was that the mean percentage increase in blood lead levels in passive smokers was

about 25%–40% of that seen in active smokers. The dose of smoke that passive smokers receive is much less than that of active smokers; for example, cotinine levels in passive smokers are about 1% of those in active smokers (Pirkle et al., 1996). Although some toxicants may be present in higher

Table 3. Odds ratios and 95% confidence intervals from multivariate logistic regression models predicting blood lead levels of 5 µg/dl or greater, stratified by sex and in overall group. From the Third National Health and Nutrition Examination Survey, 1988–1994.

Variable	Males, odds ratio (95% confidence interval) ^a	Females, odds ratio (95% confidence interval) ^a	All subjects, odds ratio (95% confidence interval) ^b
Tobacco exposure category (cotinine based)			
Current smoker	7.9 (5.3, 11.8)	5.1 (3.8, 6.7)	5.8 (4.6, 7.2)
Former smoker	3.3 (2.1, 5.2)	2.9 (2.1, 3.8)	3.7 (2.6, 5.1)
High cotinine	4.2 (2.7, 6.5)	1.9 (1.4, 2.7)	2.6 (1.9, 3.5)
Low cotinine	2.3 (1.5, 3.5)	2.0 (1.4, 2.8)	1.9 (1.4, 2.6)
No cotinine	Referent	Referent	Referent

^aAdjusted for age, poverty index ratio, race/ethnicity, country of origin, pack-years of smoking, menopausal status, year home built, and occupation.

^bAdjusted for age, poverty index ratio, race/ethnicity, country of origin, pack-years of smoking, sex, menopausal status, year home built, and occupation.

Table 4. Odds ratios and 95% confidence intervals from multivariate logistic regression models predicting blood lead levels of 5 µg/dl or greater, stratified by race and in overall group. From the Third National Health and Nutrition Examination Survey, 1988–1994.

Variable	White, odds ratio (95% confidence interval) ^a	Black, odds ratio (95% confidence interval) ^a	Mexican American, odds ratio (95% confidence interval) ^a	All subjects, odds ratio (95% confidence interval) ^b
Tobacco exposure category (cotinine based)				
Current smoker	6.1 (4.4, 8.5)	4.4 (2.6, 7.5)	2.4 (1.7, 3.2)	5.8 (4.6, 7.2)
Former smoker	2.9 (1.9, 4.3)	1.5 (0.9, 2.5)	1.3 (0.9, 1.9)	3.7 (2.6, 5.1)
High cotinine	3.0 (2.0, 4.6)	1.7 (1.0, 2.9)	1.5 (1.2, 1.9)	2.6 (1.9, 3.5)
Low cotinine	3.2 (1.5, 3.4)	1.1 (0.6, 2.0)	1.1 (0.8, 1.5)	1.9 (1.4, 2.6)
No cotinine	Referent	Referent	Referent	Referent

^aAdjusted for age, poverty index ratio, country of origin, pack-years of smoking, sex, menopausal status, year home built, and occupation.

^bAdjusted for age, poverty index ratio, race/ethnicity, country of origin, pack-years of smoking, sex, menopausal status, year home built, and occupation.

concentrations in sidestream smoke than in mainstream smoke (California Environmental Protection Agency, 1997), that difference is unlikely to explain the disproportionately higher lead levels in passive smokers. Other possibilities might be that tobacco-smoke-related inflammation is enhancing absorption of lead, that pulmonary absorption of lead occurs in a nonlinear fashion (with higher absorption at lower levels; Smith, Fischer, & Sears, 2000), that other undefined mechanisms of lead absorption and metabolism are playing a role, or that some unquantified confounder was present.

The present analysis is subject to several limitations. Because this was a cross-sectional study, we cannot say with certainty that SHS exposure increases blood lead levels. An unmeasured confounder or residual confounding related to socioeconomic status or other factors might explain some of these findings.

In conclusion, we found that both active and passive smoking are associated with increased blood lead levels in a nationally representative cross-section of the U.S. population. This association may be important to consider in studies examining health effects of elevated blood lead levels in the adult population.

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