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Income Inequality and US Children's Secondhand Smoke Exposure: Distinct Associations by Race–Ethnicity

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Abstract

Introduction—Prior studies have found considerable racial and ethnic disparities in secondhand smoke (SHS) exposure. Although a number of individual-level determinants of this disparity have been identified, contextual determinants of racial and ethnic disparities in SHS exposure remain unexamined. The objective of this study was to examine disparities in serum cotinine in relation to area-level income inequality among 14 649 children from the National Health and Nutrition Examination Survey.

Methods—We fit log-normal regression models to examine disparities in serum cotinine in relation to Metropolitan Statistical Areas level income inequality among 14 649 nonsmoking children aged 3–15 from the National Health and Nutrition Examination Survey (1999–2012).

Result—Non-Hispanic black children had significantly lower serum cotinine than non-Hispanic white children (−0.26; 95% CI: −0.38, −0.15) in low income inequality areas, but this difference

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Declaration of Interests

None declared.

was attenuated in areas with high income inequality (0.01; 95% CI: −0.16, 0.18). Serum cotinine declined for non-Hispanic white and Mexican American children with increasing income inequality. Serum cotinine did not change as a function of the level of income inequality among non-Hispanic black children.

Conclusions—We have found evidence of differential associations between SHS exposure and income inequality by race and ethnicity. Further examination of environments which engender SHS exposure among children across various racial/ethnic subgroups can foster a better understanding of how area-level income inequality relates to health outcomes such as levels of SHS exposure and how those associations differ by race/ethnicity.

Implications—In the United States, the association between children's risk of SHS exposure and income inequality is modified by race/ethnicity in a manner that is inconsistent with theories of income inequality. In overall analysis this association appears to be as predicted by theory. However, race-specific analyses reveal that higher levels of income inequality are associated with *lower* levels of SHS exposure among white children, while levels of SHS exposure among non-Hispanic black children are largely invariant to area-level income inequality. Future examination of the link between income inequality and smoking-related health outcomes should consider differential associations across racial and ethnic subpopulations.

Introduction

Despite significant declines during the past two decades in the prevalence of US youth exposed to secondhand smoke (SHS) from burning tobacco products,[1,2] exposure to SHS remains highly prevalent. In the United States, 41% of children (aged 3–11) and 34% of adolescents (aged 12–19) were exposed to SHS (as indicated by serum cotinine levels ≥0.05 ng/mL) from 1999 to 2012.[3] This translates to approximately 15.1 million children and 9.6 million nonsmoking adolescents who are exposed to SHS. Consistency, strength, and specificity of the evidence linking SHS exposure with elevated risk of poor health outcomes,[4–9] as well as biologic plausibility of this association,[10] has led the Surgeon General to declare the SHS-disease association to be causal.[11]

Prior studies have found considerable racial and ethnic disparities in SHS exposure, with the prevalence of SHS exposure highest among black, non-Hispanics (46.8%), followed by Mexican American (23.9%) and white, non-Hispanic (21.8%) populations.[3] Racial and ethnic disparity in SHS exposure extend to serum cotinine concentrations among children and adolescents. Compared to non-Hispanic white children and nonsmoking adolescents, serum cotinine levels are significantly higher among non-Hispanic black children and nonsmoking black adolescents.[12,13] Although a number of individual-level determinants of this disparity have been identified,[14–16] contextual determinants of racial and ethnic disparities in SHS remain unexamined. However, it is generally accepted that the extent of disparities in health and health behaviors, although multifactorial, are associated with structural features of communities.[17]

Prior research has suggested that income inequality is a key structural feature of communities and an important determinant of health.[18,19] Despite some controversy,[20–22] evidence suggests income inequality is associated with poor health outcomes as well as risky

health behaviors.[23–27] In fact, Pickett and Wilkinson[28] have argued that the evidence linking income inequality and poor health meets epidemiologic criteria for causality. Experimental evidence supports the notion of a causal association between income inequality and health.[29]

Multiple pathways between income inequality and health have been described, which largely fall within two theoretical perspectives, the neo-materialist and the psychosocial.[30] According to the neo-materialist perspective, economic stratification, related to income inequality, often leads to concentration of poverty in relatively small and well defined areas[31] which then experience disinvestment in economic, social, educational, and physical resources; factors which in turn are related to poor health outcomes.[32–36] In contrast, the psychosocial perspective explains the association between income inequality and health in terms of individual-level consequences of (objective or perceived) relative deprivation and resultant stress which can negatively affect health.[37–42]

The closest line of research to the present investigation is found in the only three studies that have examined the association between smoking and income inequality as measured by the Gini index with findings generally supportive of a positive association between income inequality and prevalence of smoking within larger geographies such as US states[43] and across nations.[44] However, when examined at the county-level, a more nuanced association emerges. Among adolescents (mean age 15), higher income inequality at the county-level predicted elevated prevalence of smoking among white and Hispanic boys but not among African Americans or girls.[45] Importantly, all three studies relied on self-reported measures of smoking, and did not examine exposure to SHS.

We conducted the first investigation of the association between income inequality and racial and ethnic disparities in SHS exposure among a nationally representative sample of nonsmoking children.

Materials and Methods

Study Population

We analyzed data from the National Health and Nutrition Examination Survey (NHANES) for 1999–2012. NHANES is a nationally representative survey of the civilian, noninstitutionalized US population with data collected yearly and released in 2-year survey cycles since 1999–2000, consisting of a household interview and subsequent examination component, which includes laboratory testing.[46] Response rates for the examination component during this time period ranged from 78% to 88% for children between 1 and 15 years of age.[47] Analyses were restricted to 14 649 nonsmoking children aged 3–15 years (serum cotinine is not assessed for children younger than 3 years of age).

Individual Measures

The outcome variable of interest was serum cotinine (ng/mL), which was measured using an isotope dilution high performance liquid chromatography/atmospheric pressure chemical ionization tandem mass spectrometry.[48] Of children in the sample, 1915 (16%) had cotinine values below the limits of detection; these children were assigned a value of 0.011 ng/mL, which is the detection limit divided by the square root of two.[48] Children aged 3–11 years

were defined to be nonsmokers if their serum cotinine was below 10 ng/mL. Youth aged 12–15 years were defined to be nonsmokers if their serum cotinine was below 10 ng/mL and they reported no tobacco or nicotine use in the past 5 days; the latter question was not asked of children less than 12 years old.[48] Respondents with missing data for household income or country of birth (6%) were excluded from multivariate analyses leaving 13 706 respondents.

Additional covariates included the 2-year cycle; sex; country of birth (US born, not US born); age in years (3–5, 6–11, or 12–15); race/ethnicity, household size (number of persons) and family income-to-poverty ratio (IPR). For this analysis, we categorized race/ethnicity as Mexican American; non-Hispanic white; non-Hispanic black; and Other (the "Other" category includes "other Hispanic," "other race," and "multi-racial"; this subgroup was included in the analysis but results are not reported due to the small number of participants and racial/ethnic heterogeneity of this subgroup, consistent with NHANES analytic guidelines.[46] Family IPR is calculated as the ratio of family income to the federal poverty threshold (FPT). Household smoking is defined as an affirmative answer to the question, "does anyone smoke in the home"; these household respondents are also asked to estimate the total number of cigarettes smoked in the home per day.

Area-Level Measures

For each Metropolitan Statistical Areas (MSAs), we calculated the Gini income inequality coefficient using Census 2000 income and population data. The Gini index, G, is calculated as

$$G=1-\sum p_i\,(q_i+q_{i-1})$$

where p_i is the proportion of the population in the i'th income category, q_i is the proportion of all incomes that fall in the i'th income category or below, and the sum is over all the income categories. The Gini index ranges between 0 and 1, where 0 means complete equality and 1 means perfect inequality. Due to the non-normal distribution of the Gini index, and to avoid assuming a linear relationship with serum cotinine, the MSA-level Gini index for all the NHANES individuals surveyed, including children and adults, and smokers and nonsmokers, were grouped into three tertiles for these analyses. We used inequality at the MSA level because it is a lower-level of geography than prior studies that have used state-level inequality, while using counties as the unit of analysis may not capture the income inequality between suburbs and urban cores. By contrast, MSAs represent economically and socially integrated clusters of outlying counties around urban cores, thus representing a set of discrete regional housing and labor markets.[49,50]

To examine associations between income inequality and economic stratification, we examined the percent of residents below the FPT at the tract level, obtained from the 2000 US decennial census data. Census tracts have relatively homogeneous populations, and are commonly used to examine contextual-level exposures and health outcomes.[51,52]

Area-level measures were linked with NHANES respondents based on the nonpublic use NHANES data with geographic identifiers (eg, census tract Federal Information Processing Standard [FIPS] code).

Analyses

Two statistical models were fitted to the data. Model A regressed serum cotinine against income inequality (ie, Gini index) tertile. Informed by prior works,[12,14,16,17] we controlled for NHANES cycle, race/ethnicity (non-Hispanic white, non-Hispanic black, Mexican American and "other"), age (3–5 years, 6–11 years and 12–15 years), sex, country of birth (US born or not), household size, and family IPR. Model B added an interaction term between race/ethnicity and income inequality tertile to compare these race/ethnicity differences in serum cotinine between the income inequality tertiles for children of the same age group, sex, household income level, and NHANES cycle. Estimates and confidence intervals (CIs) for differences in serum cotinine adjusted for the NHANES cycle and included covariates were calculated using NHANES survey weights and design-based variance estimation to account for the survey design. Log-normal regression models were used to account for the skewed distribution of serum cotinine. Marginal effects were obtained following the second set of models, these estimates refer to the change in serum cotinine (on the original scale) associated with a one-unit increase in a given covariate, or in the case of race/ethnicity, the difference in serum cotinine between a given group and the reference group (non-Hispanic white). All analyses were performed using Stata SE (version 12.1) survey commands to account for the complex, stratified, multistage sample design and the applicable Mobile Examination Center person-level survey weights for each survey cycle, appropriately scaled given the combining of several survey cycles, were used to account for oversampling, noncoverage and nonresponse.

In post hoc analyses, we explored associations between income inequality and economic stratification by race/ethnicity by examining how the prevalence of tract-level poverty rates and family income level (IPR) differ by income inequality tertile, and whether these patterns vary by race/ethnicity. Additionally, we explored MSA-level income inequality in relation to level of reported household smoking (ie, number of cigarettes smoked in the home per day).

Results

Descriptive characteristics of our study sample appear in Table 1. Approximately 82% of nonsmoking children aged 3–15 reportedly resided in nonsmoking households. The median and mean levels of serum cotinine were highest for non-Hispanic black children. Overall, cotinine levels were lowest for Mexican American children.

There were significant differences in serum cotinine levels as a function of MSA-level income inequality (Table 2). Overall, serum cotinine levels "decrease" as income inequality increases. Adjusted differences in nonsmoking children's cotinine levels compared to the reference category of non-Hispanic white children are reported in Table 3. After controlling for individual- and household-level demographic measures, non-Hispanic black and Mexican American children had significantly lower average levels of serum cotinine than

non-Hispanic white children (adjusted differences: −0.22; 95% CI: −0.30, −0.13; and adjusted differences: −0.52; 95% CI: −0.61, −0.43, respectively).

Race–Ethnic Comparisons by MSA Income Inequality

Relative to non-Hispanic white children, non-Hispanic black children had lower serum cotinine levels in areas with low income inequality; however, this difference was not found in areas with moderate or high income inequality (Table 3). More specifically, within areas with low income inequality, non-Hispanic black children had significantly lower serum cotinine concentrations than non-Hispanic white children (−0.26; 95% CI: −0.38, −0.15). Within areas with moderate income inequality, this difference in serum cotinine levels between black and white children was less pronounced (−0.16; 95% CI: −0.33, 0.01). Within areas with high income inequality, average serum cotinine levels for non-Hispanic black and non-Hispanic white children were no different (0.01; 95% CI: −0.16, 0.18). In contrast, cotinine concentrations remained significantly lower among Mexican American compared to non-Hispanic white children in all areas, this difference was least pronounced within areas of high income inequality (−0.32; 95% CI −0.46, −0.18).

We further examined race-specific marginal changes in children's serum cotinine concentrations across Gini tertiles (Table 4). For both non-Hispanic white and Mexican American children we observed a pattern of "decreasing" serum cotinine levels with increasing income inequality. In contrast, serum cotinine levels of non-Hispanic black children did not significantly change as a function of the level of income inequality. When contrasting serum cotinine levels across race–ethnic groups, the decrease in average cotinine levels with increasing income inequality is approximately seven times more pronounced among white children (−0.31 ng/mL; $p < .01$) than among black children. In contrast, Mexican American children's decrease in serum cotinine levels between low-versus high-inequality areas (−0.13 ng/mL) was not significantly different from that of non-Hispanic white children (ie, Diff.: 0.18; $p = .056$).

In post hoc analyses, we explored associations between income inequality and tract-level poverty rates and family income level (IPR), and whether these patterns vary by race/ethnicity. In the United States, tract-level poverty rates declined for non-Hispanic white children from 11% (95% CI: 10%–12%) in the lowest-inequality tertile to 7% (95% CI: 6%–8%) in the highest income inequality tertile, a significant decline ($p < .001$). For non-Hispanic black children, their experience of tract-level poverty is more stable across the inequality tertiles: the decline of 3 percentage points (from 22% to 19%) in the prevalence of poverty is not significantly different from zero ($p = .09$). For Mexican American children, the experience of tract-level poverty was stable (18%) across the MSA-level income inequality tertiles. These same patterns hold for family IPR. The mean family IPR among non-Hispanic white children is 1.16 units higher among children residing in the highest inequality tertiles compared to the lowest (IPR = 3.63 vs. 2.48, $p < .001$). Among non-Hispanic black children, the mean family IPR is only 0.59 units higher in the high-inequality MSAs compared to the low (IPR = 2.00 vs. 1.42, $p < .001$). Among Mexican American children, there are no differences in the mean family IPR across the income inequality tertiles. Thus, residence in MSAs characterized by high income inequality is associated with

lower levels of tract-level poverty and higher family IPR among non-Hispanic white children. For non-Hispanic black children and Mexican American children, however, the experience of tract-level poverty is largely unassociated with MSA-level income inequality. Moreover, for non-Hispanic black children, family income levels increase to a smaller extent across the income inequality tertiles as compared to non-Hispanic white children; for Mexican American children, family income levels do not change across MSA income inequality tertiles.

Finally, while models did not include household smoking as a covariate, as this would be controlling for a mediator,[11] we did examine levels of household smoking in relation to MSA income inequality tertile and race/ethnicity in post hoc analyses. Consistent with the main findings for serum cotinine, we found that the average number of cigarettes reportedly smoked in the home declined with higher levels of MSA income inequality to a greater extent for non-Hispanic white children (4.17 cigarettes/day in the lowest income inequality tertile vs. 1.80 cigarettes/day in the highest tertile; decrease of 2.37, $p < .0001$) than for non-Hispanic black children (2.59 cigarettes/day in the lowest inequality tertile vs. 1.63 cigarettes/day in the highest tertile; decrease of 0.96, $p < .025$). Average cigarettes smoked per day in the home was 1.13 for Mexican American children in the lowest inequality tertile, decreasing to 0.20 per day in the highest inequality tertile (decrease of 0.93 $p < .001$).

Discussion

We conducted the first study of the association between MSA-level income inequality and racial and ethnic disparities in serum cotinine, an objective measure of SHS exposure among nonsmoking children. We found that In the United States, metropolitan areas characterized by low income inequality, both non-Hispanic black and Mexican American children have significantly lower levels of SHS exposure than non-Hispanic white children. This association dissipates with rising income inequality, such that in areas with high income inequality, there is no difference in the level of SHS exposure between black and white children and there is a smaller relative advantage among Mexican American children. Dissolution of this difference among blacks and the reduction in the difference among Mexican Americans as a function of rising income inequality appears to be in accord with the theory of income inequality. However, race-specific analyses revealed that dissolution of non-Hispanic black's relative advantage is not due to an increase in SHS exposure levels among this population, as would be expected by theory of income inequality. Rather, this effect is due to a decline in cotinine concentrations of non-Hispanic whites with increasing income inequality. Likewise, reduction in the difference for Mexican American children is primarily due to the decline in cotinine concentrations of white children with increasing income inequality. These serum cotinine patterns are confirmed in that the average number of cigarettes reportedly smoked in the home declined with higher levels of income inequality to a greater extent for non-Hispanic white than for non-Hispanic black or Mexican American households.

Our findings regarding differential association between area-level income inequality and SHS among racial and ethnic subgroups are in accord with two lines of extant evidence. First, our observation that Mexican American children have the lowest SHS exposure across

all tertiles of income inequality is in agreement with the literature on Hispanic paradox.[53,54] Second, in the only study of income inequality and smoking among youth conducted within the relatively small geography of counties, Mistry et al.[45] results indicated that risk of smoking among black youth were unrelated to income inequality. Furthermore, the only studies of income inequality as assessed by the Gini index, which have conducted race specific analyses, and controlled for individual-level covariates have reported that the crude association between income inequality and health outcomes for black respondents is reduced to null after adjustment for individual-level variables.[55,56] Recently, Nuru-Jeter et al.[57] noted that "a criticism, which has received relatively little attention, is that tests of the income inequality hypothesis fail to assess the role of race and/or ethnicity…" (p.436). In their study, which used three measures of income inequality, including the Gini coefficient, these authors found a positive association between income inequality and mortality among blacks but an inverse association among white children. In sum, our findings describe no association between income inequality and levels of SHS exposure among black children and a negative association among white children. Collectively, our findings and prior studies suggest that associations between income inequality and health outcomes are likely to be race-specific.

In regards to practical implications of our findings, since there are no safe levels of SHS exposure,[11] continued efforts to reduce SHS exposure remain a public health imperative.[58,59] While it is unclear how various efforts to reduce SHS exposure may have had differential impacts across various subpopulations, there is some evidence that there are differences by race/ethnicity in the coverage of comprehensive smoke-free laws.[58] Moreover, higher education and higher SES communities are more likely to adopt smoke-free laws than lower-education or lower SES communities, though there are differences in these patterns by geographic region as well.[59] To our knowledge, there have been no examinations of how the implementation and coverage of various tobacco control policies may vary by area-level income inequality. Nevertheless, regional and race/ethnicity-related differences in the implementation and coverage of tobacco control policies may be connected to area-level socioeconomic factors,[58,59] and it remains to be seen how income inequality relates to these patterns and disparities. Further examination of these complex pathways and environments as they relate to the sources of SHS exposure among children across various racial/ethnic subgroups can foster a better understanding of how area-level income inequality relates to health outcomes such as levels of SHS exposure and how those associations differ by race/ethnicity.

Our findings should be considered in the context of our study's strengths and weaknesses. This is the only study of income inequality and SHS conducted among a nationally representative sample with a wide age range of respondents as young as 3 years using an objective measure of SHS. Our work also adds to the sparse literature on race-specific associations between income inequality and health. As for shortcomings, the temporal discrepancy between when income inequality was assessed (based on the 2000 decennial census) may lead to attenuation of these associations over time as residence in a given tertile of income inequality might be misclassified, and the degree of this misclassification would be greater in the later survey cycles due to the time elapsed since the 2000 census. As with other studies that have relied on administratively defined geographic units, findings may

differ based on how geographic units are defined. Additionally, while we explored area-level income inequality consistent with a neo-materialist approach, it remains to be seen how accounting for individual-level perceptions of relative inequality, such as that described by the psychosocial theory of inequality, would influence our findings. Finally, we note that these observed epidemiologic associations do not allow causal inference, so results should be interpreted with caution.

In conclusion, we have found evidence of differential associations between income inequality and SHS by race/ethnicity. Specifically, findings suggest a stronger association between area-level socioeconomic factors such as income inequality and levels of SHS exposure among non-Hispanic white children, as compared to both non-Hispanic black and Mexican American children, whose levels of exposure to SHS are largely invariant to area-level income inequality. Future examination of the link between income inequality and smoking-related health outcomes should consider differential associations across racial and ethnic subpopulations.

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Table 1

Descriptive Characteristics for Nonsmoking[a] Children Aged 3–15 (N = 14 649) in the National Health and Nutritional Examination Survey, United States, 1999–2012

Characteristic	Unweighted *N*	Serum cotinine (ng/mL)	
		Geometric mean (95% CI)	Arithmetic mean (95% CI)
All	14 649	0.08 (0.07, 0.09)	0.46 (0.41, 0.51)
Race/ethnicity			
Non-Hispanic white	3841	0.09 (0.07, 0.10)	0.53 (0.45, 0.60)
Non-Hispanic black	4233	0.18 (0.16, 0.21)	0.64 (0.58, 0.71)
Mexican American	4588	0.04 (0.04, 0.05)	0.16 (0.14, 0.19)
Other	1987	0.06 (0.06, 0.07)	0.33 (0.27, 0.40)
Age			
3–5	2679	0.11 (0.09, 0.13)	0.62 (0.52, 0.72)
6–11	6497	0.09 (0.08, 0.10)	0.48 (0.42, 0.54)
12–15	5473	0.07 (0.06, 0.08)	0.36 (0.32, 0.40)
Sex			
Female	7231	0.08 (0.07, 0.09)	0.46 (0.41, 0.52)
Male	7418	0.08 (0.07, 0.09)	0.47 (0.41, 0.52)
Family IPR[b]			
<100% FPT	4664	0.20 (0.17, 0.24)	0.87 (0.76, 0.98)
100%–199% FPT	3836	0.12 (0.10, 0.14)	0.56 (0.49, 0.64)
200%–299% FPT	1966	0.07 (0.06, 0.08)	0.37 (0.29, 0.44)
300%–399% FPT	1252	0.06 (0.05, 0.07)	0.26 (0.19, 0.34)
≥400% FPT	2007	0.03 (0.03, 0.04)	0.13 (0.10, 0.16)

CI = confidence interval; IPR = income-to-poverty ratio; FPT = federal poverty threshold.

[a]Children aged 3–11 years were defined to be nonsmokers if their serum cotinine was below 10 ng/mL. Youth aged 12–15 years were defined to be nonsmokers if their serum cotinine was below 10 ng/mL and they reported no tobacco or nicotine use in the past 5 days.

[b]IPR missing information for 924 children.

Table 2

Unadjusted and Adjusted Differences in Nonsmoking Children's Serum Cotinine Levels (ng/mL) by MSA Income Inequality Tertile (95 % CI[a])

	Diff. (95% CI)	
	Unadjusted	**Fully adjusted**
Low inequality[a]	Ref	Ref
Moderate inequality	−0.20 (−0.33, −0.07)	−0.03 (−0.15, 0.09)
High inequality	−0.42 (−0.53, −0.31	−0.22 (−0.32, −0.12)

CI = confidence interval; IPR = income-to-poverty ratio; MSA = Metropolitan Statistical Area; NHANES = National Health and Nutrition Examination Survey.

[a]CIs estimated using design-based variance estimation; adjusted for age, sex, US born vs. foreign born, IPR, household size, and NHANES cycle.

[b]Low: MSA income inequality index in lowest 1/3 of all respondents, including children, adults, nonsmokers, and smokers. Moderate: MSA income inequality index in middle 1/3 of all respondents. High: MSA income inequality index in highest 1/3 of all respondents.

Table 3

Adjusted Differences in Nonsmoking Children's Serum Cotinine Levels (ng/mL) Compared to Reference Category (95 % CI[a]): (A) Differences by Race/Ethnicity, Controlling for Individual-Level Covariates (eg, Age, Sex); (B) Change in Serum Cotinine as a Function of MSA-Level Income Inequality by Race/Ethnicity and Age Group, Results From Fully Adjusted Models With an Interaction Term for Income Inequality

		B—Fully adjusted models with race/ethnicity by inequality–tertile interaction terms		
		Low[b]	Moderate	High
	A—Models adjusted for individual-level effects only	Diff. (95% CI)	Diff. (95% CI)	Diff. (95% CI)
Race/ethnicity				
Non-Hispanic white	Ref	Ref	Ref	Ref
Non-Hispanic black	−0.22 (−0.30, −0.13)	−0.26 (−0.38, −0.15)	−0.16 (−0.33, 0.01)	0.01 (−0.16, 0.18)
Mexican American	−0.52 (−0.61, −0.43)	−0.49 (−0.61, −0.38)	−0.49 (−0.66, −0.31)	−0.32 (−0.46, −0.18)
Other	−0.33 (−0.43, −0.22)	−0.26 (−0.38, −0.14)	−0.14 (−0.36, 0.08)	−0.27 (−0.41, −0.14)
Age				
3–5	Ref	Ref	Ref	Ref
6–11	−0.10 (−0.19, −0.00)	−0.16 (−0.32, 0.00)	−0.03 (−0.11, 0.04)	−0.03 (−0.12, 0.06)
12–15	−0.17 (−0.27, −0.08)	−0.29 (−0.43, −0.15)	−0.07 (−0.18, 0.036)	−0.02 (−0.11, 0.07)

CI = confidence interval; IPR = income-to-poverty ratio; MSA = Metropolitan Statistical Area; NHANES = National Health and Nutrition Examination Survey. Linear trends across the three inequality tertiles indicated that cotinine levels declined significantly for non-Hispanic white children (−0.12, 95% CI: −0.23, −0.01, $p = .038$), did not change for non-Hispanic black children (−0.02, 95% CI: −0.07, 0.04, $p = .508$), and declined significantly for Mexican American children (−0.11, 95% CI: −0.11, −0.03, $p < .001$).

[a] CIs estimated using design-based variance estimation; adjusted for age, sex, US born vs. foreign born, IPR, household size, and NHANES cycle.

[b] Low: MSA income inequality index in lowest 1/3 of all respondents, including children, adults, non-smokers, and smokers. Moderate: MSA income inequality index in middle 1/3 of all respondents. High: MSA income inequality index in highest 1/3 of all respondents.

Table 4

Predicted Change in Serum Cotinine Levels (ng/mL) for Each Race/Ethnicity/Income Inequality Tertile Compared to the First Tertile—Results From Models Fitted With Interactions Between Race/Ethnicity and Low, Moderate, and High Income Inequality MSAs

Tertiles of MSA income inequality	White, non-Hispanic	Black, non-Hispanic	Mexican American	*p* values for pairwise comparisons across race/ethnicity group[a]
Low	ref	ref	ref	
Moderate	−0.07 (−0.27, 0.13) a	0.04 (−0.07, 0.14) b	−0.06 (−0.13, 0.01) c	a–b: $p = .32$ a–c: $p = .95$
High	−0.31 (−0.49, −0.13) d	−0.04 (−0.15, 0.08) e	−0.13 (−0.19, −0.07) f	d–e: $p = .009$ d–f: $p = .056$

CI = confidence interval; MSA = Metropolitan Statistical Area.

[a]Wald test with sampling errors estimated using design-based variance estimation.