



Crossover Trends in Current Cigarette Smoking Between Racial and Ethnic Groups of US Adolescents Aged 12–19 Years Old, 1999–2018

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Abstract

Objective To examine the racial difference and trends in cigarette smoking among adolescents from 1999 to 2018.

Methods We analyzed the data of 10,760 adolescents aged 12–19 who participated in the National Health and Nutrition Examination Surveys (NHANES), 1999–2018. Current tobacco smoking (CTS) was defined as participants with serum cotinine ≥ 10 ng/mL. Adjusted biennial prevalence ratios (abiPR: the ratio associated with a two-year increase in time) were estimated.

Results Diverging trends in CTS prevalence were revealed in adolescents. The steepest decrease occurred in Hispanics aged 12–17, with 15% declining every two calendar years [abiPR = 0.85(0.77, 0.94)]. The sharpest increase occurred with Blacks aged 18–19 years [abiPR = 1.06(0.99, 1.14)]. A crossover of prevalence trend between Blacks and Whites occurred in adolescents aged 18–19 years old due to the diverging trends. The average CTS prevalence was significantly higher in Whites than in Blacks in the early [(1999–2008, 13.65% (11.85%, 15.46%) vs. 8.80% (7.55%, 10.04%)], but Blacks had a higher average in recent years [(2009–2018, 8.32% (6.53%, 10.12%) vs. 7.77% (5.86%, 9.68%)]. For adolescents aged 18–19 years, the survey cycles or calendar years linearly explained 71% of the variations in the prevalence for Hispanics, 60% for Whites, but only 1% for Blacks.

Conclusions A crossover in the trend of current tobacco smoking occurred between 1999 and 2018 due to an increase in prevalence among Black adolescents and a significant decrease in prevalence among other racial groups.

Introduction

Studies have consistently concluded that African American youth and young adults have a significantly lower prevalence of cigarette smoking than Hispanics and Whites [1], and African Americans initiate smoking later than Whites [2]. Previous studies, however, have mainly relied on self-reports to assess smoking status [3, 4], including Youth Risk

Behavior Surveillance [4, 5], National Surveys on Drug Use and Health [6], National Youth Tobacco Survey [7], Monitoring the Future (MTF) Surveys [8, 9], and National Health Interview Survey [7]. Underestimation of smoking prevalence may occur by race based on self-report [10–12]. The racial differences reported [4–9] may be artefactual due to race-specific underestimations [13]. The escalated smoke-free legislation and the recent introduction of new electronic cigarette devices have altered adolescents' types of tobacco products and changed the tobacco smoking epidemic landscape [14]. The literature on ethno-racial differences in adolescent tobacco smoking has not yet been updated for years. It is desirable to re-assess the previously reported declining trend in current tobacco smoking (CTS) among adolescents with more reliable objective measurements [15].

The ongoing COVID-19 pandemic further exacerbates the devastating consequence of tobacco use [16, 17] and highlights the newfound urgency to address the related racial/ethnic disparity. More severe and prevalent multisystem

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inflammatory syndromes were reported among Black pediatric COVID-19 patients than patients of other racial/ethnic groups [18, 19]. Chronic pulmonary ailments are the most common pre-existing conditions associated with hospitalization of pediatric COVID-19 cases and ICU admission [20]. Examining the race difference in tobacco use among adolescents may offer clues to explain the race difference in COVID-19-related disease burden in pediatric populations. Using the most recent data from the National Health and Nutrition Examination Survey (NHANES), we quantified the race-specific trends in CTS using serum cotinine, a biomarker of nicotine intake or exposure.

Methods

Data Sources and Study Participants

As a continuous national survey, the NHANES is conducted by the National Center for Health Statistics to assess the health and nutritional status of the US civilian, non-institutionalized US population. NHANES uses a complex multistage probability sampling design, with some subgroups oversampled. The unweighted response rate for the interviewed sample has been reported as above 80% [21]. This analysis started with 16,750 adolescents aged 12 to 19 years. We excluded adolescents who had missing data for serum cotinine ($n = 1218$) or the number of smokers in the home ($n = 178$), resulting in 13,593 adolescents. White, Black, and Hispanic adolescents were included; adolescents from races/ethnicities other than the three major races/ethnicities ($n = 1218$) were excluded from the current analysis due to the relatively small sample size for robust trend assessment. An additional 966 adolescents were excluded due to missing information on family income, and 1937 were excluded because of missing data regarding maternal education attainment. A total of 10,760 adolescents were retained for the final analysis. The NHANES protocol was reviewed and approved by the National Center for Health Statistics Institutional Review Board (IRB). The current study was exempt from ethics review by the IRB committee of the institution with which the senior author was affiliated.

Definition of Current Smokers

The TU-11.2 objective (reduce the proportion of adolescents aged 12 to 17 years exposed to second-hand smoke) of Healthy People 2020 differs from Healthy People 2010 objective 27-10 in several ways. In Healthy People 2010, a single age group (persons aged four years and over) was monitored. Healthy People 2020 assesses exposure for persons aged 3 to 11 years, 12 to 17 years, and 18 years and over. Persons with cotinine levels greater than 10 ng/mL

were counted as exposed nonsmokers in Healthy People 2010 but considered smokers for Healthy People 2020 [22, 23]. To be consistent with the objectives of Healthy People 2020, we defined adolescents with cotinine levels greater than 10 ng/mL as current smokers. Serum cotinine measurement was performed with isotope dilution-high-performance liquid chromatography/atmospheric pressure chemical ionization tandem mass spectrometry.

Classification of Race/Ethnicity and Categorization of Age Group

Race/ethnicity was stratified in current analyses. NHANES classifies participants based on their responses as non-Hispanic Whites (hereafter, Whites), non-Hispanic Blacks (Blacks), Mexican Americans, and other Hispanics. The “Mexican American” and “other Hispanic” were combined as “Hispanic Americans.” To be consistent with the age classification of the Healthy People 2020 and the U.S. Census Bureau, we grouped adolescents into early and middle adolescence (aged 12 to 17 years) and late adolescence (aged 18 and 19 years). The “late adolescence” is a distinct group created to assess the transition from the protective environment to a young adult.

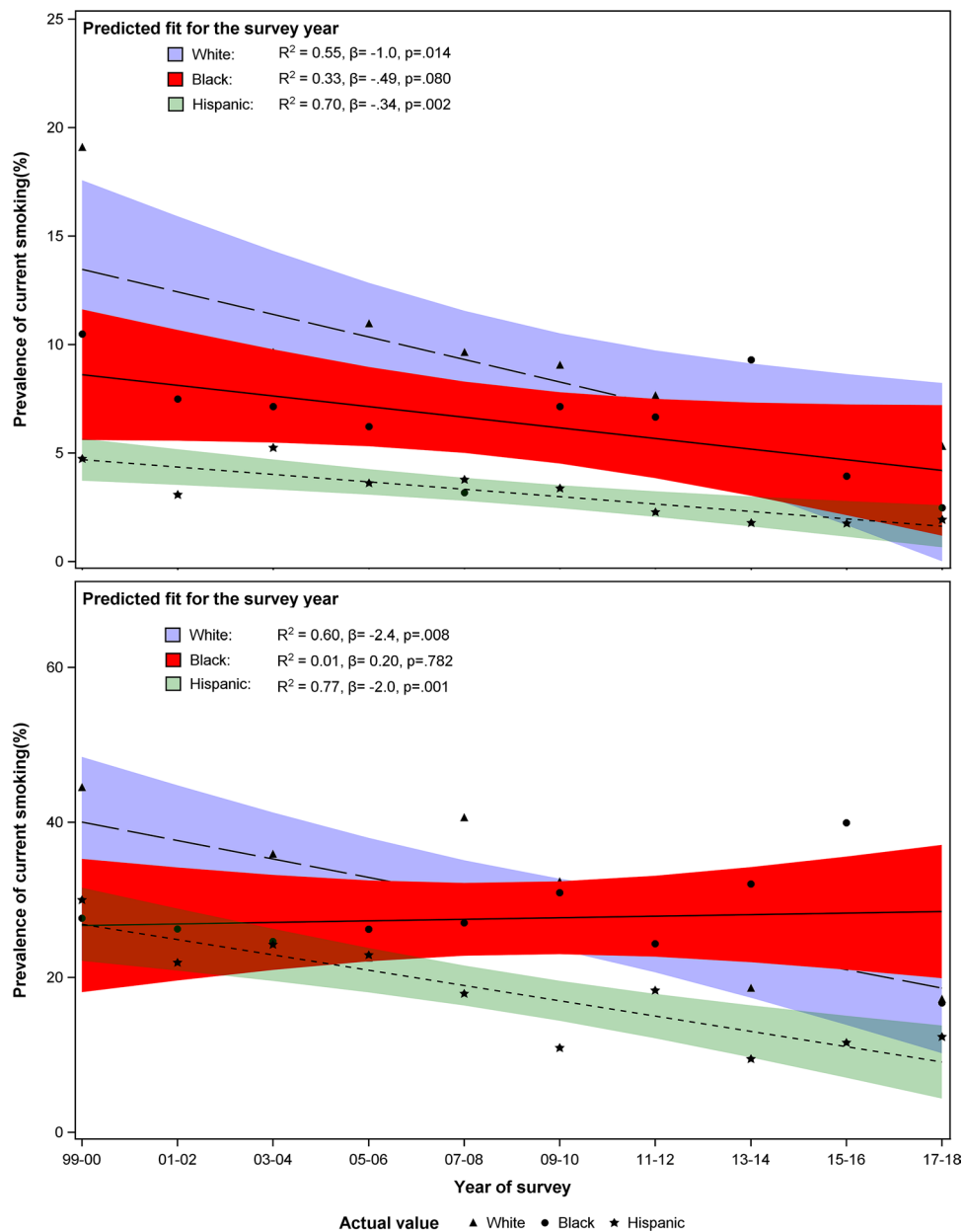
Major Covariates

Income was reported as a range for the previous calendar year. A poverty income ratio (PIR) was calculated by comparing the midpoint of the selected income range value to the appropriate poverty threshold based on family size and composition. PIR values below 1.00 were categorized as below the official poverty line. For this study, four categories of PIR were considered: poor ($PIR < 1.0$), near poor ($1 \leq PIR < 2$), middle income ($2 \leq PIR < 4$), and high income ($PIR \geq 4$). The family head is the first household member 18 years of age or older who owned or rented the residence where members of the household resided. The marital status of the family head was collapsed into three categories: never married, previously, and currently married. Trained technicians collected body measurements following a standard protocol. To control the exposure to indoor household smoking, the responses to the question of “*Total number of smokers inside the home?*” were also included.

Statistical Analysis

With appropriate weighting and nesting variables to account for the complex sampling designs, we used SAS survey procedures (version 9.4, Research Triangle Park, NC) to calculate weighted percentages for every 2-year survey cycle to illustrate the trends in the CTS prevalence. The percentage was then ecologically correlated with the survey year using simple linear regression (Fig. 1). The survey cycle was used

Fig. 1 Trends in the prevalence of current smoking, a sample of 10,760 adolescents aged 12–19 NHANES 1999–2016. Note: NHANES, National Health and Nutrition Examination Survey. (a) The top panel is for adolescents 12–17 years old, and the bottom one is for adolescents 18–19 years old. For better clarity, different scales were used for Y-axis. (b) With appropriate weighting and nesting variables, we used SAS survey procedures to calculate the weighted prevalence for every 2-year survey cycle. (c) The *p*-value was for the coefficient rather linear trend test; the *p* for the trend test can be found in Supplementary Table S1. (d) The colored bands around the prediction line are plotted confidence intervals



as the explanatory variable to estimate the change in the prevalence of CTS associated with a 2-year increase in the calendar year. The biennial change in CTS prevalence was measured by a coefficient (β) of the variable of the survey cycle in the equation: the CTS prevalence = intercept + $\beta \times$ biennial survey cycle + e (error term).

The biennial changes estimated from the ecological correlation were not adjusted for sociodemographic shifts in the study populations. In step two, multiple variable logistic regression was utilized to estimate CTS's adjusted biennial prevalence ratio (abiPR: prevalence ratio associated with a two-year increase in time) (Table 1). The multivariable regressions were run on individual-based (vs. group-based ecological correlation in step one) with the survey

cycle (every two years) as an explanatory variable. Previous studies observed that during 1997–2017, a significant linear decrease occurred in the overall CTS prevalence [4], and the linear trend was also observed by visual inspection of the prevalence trend in the preliminary analyses. Therefore, we included the survey cycle as a continuous variable rather than as pair contrasts between each cycle against the first cycle (1999–2000) for the multivariable regression to estimate the biennial change in CTS prevalence during the study period for each race/ethnicity. The quadratic and higher-order trends were not assessed. Stratified regressions were run for Whites, Blacks, and Hispanic adolescents separately as the modifying effects from races/ethnicities were detected. In addition to the estimates associated

Table 1 Changes of selected characteristics of weighted study population across survey periods, 10,760 adolescents aged 12–19 years old, NHANES 1999–2018

Characteristic	Blacks			Hispanics			Whites		
	<i>n</i>	%(95%CI)	<i>p</i> ^a	<i>n</i>	%(95%CI)	<i>p</i> ^a	<i>n</i>	%(95%CI)	<i>p</i> ^a
Living under the poverty line ^b			0.04			0.15			0.44
1999–2002	487	53.87 (48.79–58.96)		633	45.37 (40.34–50.41)		157	22.60 (15.95–29.26)	
2003–2006	528	45.35 (40.03–50.66)		537	45.27 (40.86–49.69)		200	18.41 (14.20–22.62)	
2007–2010	174	41.87 (35.44–48.31)		349	49.49 (44.02–54.96)		195	20.89 (16.65–25.12)	
2011–2014	260	49.37 (40.48–58.25)		333	54.29 (47.84–60.74)		158	18.89 (13.54–24.24)	
2015–2018	175	46.03 (40.97–51.09)		241	45.40 (37.87–52.93)		142	17.40 (12.45–22.35)	
House head is currently married			0.05			0.53			<0.01
1999–2002	330	37.53 (32.36–42.70)		823	71.42 (65.42–77.43)		531	72.05 (68.03–76.06)	
2003–2006	447	38.48 (34.60–42.36)		835	73.71 (70.08–77.35)		689	82.24 (77.84–86.63)	
2007–2010	169	43.77 (38.17–49.38)		494	73.50 (69.61–77.38)		446	80.95 (77.81–84.10)	
2011–2014	231	48.80 (42.21–55.38)		439	76.31 (70.31–82.30)		378	83.07 (79.40–86.74)	
2015–2018	145	44.58 (39.78–49.38)		354	72.12 (67.65–76.60)		403	80.97 (74.96–86.98)	
Mother smoked during the pregnancy			0.20			0.12			0.60
1999–2002	85	17.30 (12.68–21.93)		51	9.57 (6.17–12.96)	0.116	84	21.06 (15.57–26.56)	
2003–2006	101	17.08 (13.90–20.27)		50	10.10 (7.15–13.04)		110	24.53 (20.47–28.59)	
2007–2010	28	14.27 (8.94–19.61)		24	6.24 (3.84–8.64)		73	19.27 (15.26–23.28)	
2011–2014	19	6.35 (4.11–8.60)		10	3.38 (1.11–5.66)		49	16.79 (11.07–22.50)	
2015–2018	14	5.89 (2.14–9.64)		16	5.75 (2.53–8.97)		61	16.12 (9.94–22.29)	
No smoker in the home			0.09			0.04			0.43
1999–2002	634	70.34 (67.71–72.97)		1080	84.15 (78.74–89.56)		577	75.82 (71.57–80.07)	
2003–2006	860	74.14 (69.58–78.69)		1008	90.05 (87.72–92.38)		655	77.03 (71.50–82.57)	
2007–2010	325	79.16 (71.07–87.25)		642	91.77 (88.59–94.95)		466	81.68 (76.49–86.86)	
2011–2014	417	79.92 (74.86–84.97)		522	88.81 (86.14–91.47)		371	82.96 (77.25–88.67)	
2015–2018	236	70.00 (65.48–74.51)		386	75.83 (70.92–80.75)		349	71.05 (64.38–77.72)	

CI, confidence interval; NHANES, The National Health and Nutrition Examination Survey

^aThe *p* of the trend tests

^bA poverty index ratio (PIR) was calculated by comparing the midpoint of the family income category and the family size with the federal poverty line. A PIR < 1 was defined as poor

with a biennial increase in the calendar year, multivariable regression also offered an opportunity to estimate CTS prevalence ratios across sociodemographic strata and other factors and describe the adolescents at high risk of CTS. We did not use the $-2 \log$ -likelihood test to simplify the regression models; instead, saturated models were retained, including the variable of race/ethnicity, educational attainment, and family income regardless of *p*-values. A *p*-value associated with the regression coefficient that was <0.05 was considered statistically significant.

Results

Table 1 presents the race/ethnicity-specific changes in sociodemographic characteristics across survey periods. Despite a continuous increase in the percentage of children who lived with parents currently married, throughout the

entire study period, less than half of Black adolescents, in contrast to more than 70% of Hispanic and 80% of White adolescents, were with parents currently married. Black families had the lowest percentage of smoking-free households relative to Hispanic and White families. CTS was associated with various factors. Boys were more likely to smoke than girls (Table 2), and Hispanic adolescents had the lowest CTS prevalence, with family income inversely associated with CTS prevalence. The number of smokers living in the household was linearly associated with CTS; 52.91% (S.E.: 5.62%) of the CTS adolescents had >2 family members who smoked at home. A significant decline in CTS prevalence occurred during the study period, from 20.23% (2.15%) at the beginning of the study period (1999–2000) to 6.72% (1.25%) in the ending year (2016–2018).

Stratified by races/ethnicities, the CTS prevalence decreased significantly among Whites and Hispanics but

Table 2 Current tobacco smoking prevalence across the levels of characteristics, 10,760 adolescents aged 12–19, NHANES 1999–2018^{a,b}

Characteristic	Level	<i>n</i> of current smokers	% (se) of current smokers	<i>N</i>	<i>p</i>
Sex	Boys	703	13.51 (0.72)	5320	<0.01
	Girls	417	8.73 (0.55)	5440	
Age group	12–17 years old	559	7.09 (0.44)	8454	<0.01
	18–19 years old	561	26.29 (1.20)	2306	
Race/ethnicity	White	478	12.89 (0.71)	3251	<0.01
	Black	389	10.74 (0.63)	3368	
	Hispanic	253	6.01 (0.57)	4141	
Family income ^c	High income	179	8.45 (0.80)	2253	<0.01
	Middle income	204	9.19 (1.01)	2450	
	Near poor	139	11.07 (1.27)	1488	
	Poor	598	15.84 (0.90)	4569	
Body weight ^d	Normal weight	683	11.27 (0.63)	6188	0.19
	Overweight	175	10.62 (0.90)	1842	
	Obese	201	10.21 (1.00)	2309	
	Underweight	45	15.67 (3.06)	307	
Covered by insurance	No	264	18.22 (1.67)	1847	<0.01
	Yes	852	10.22 (0.50)	8876	
Mom's smoking during pregnancy	No	113	2.50 (0.29)	4882	<0.01
	Yes	70	9.98 (1.45)	775	
# Smokers at home	None	621	8.00 (0.50)	8528	<0.01
	One	241	17.13 (1.33)	1407	
	Two	175	24.33 (1.86)	659	
	More than two	83	52.91 (5.62)	166	
Survey years	1999–2000	153	20.23 (2.15)	1219	<0.01
	2001–2002	172	10.88 (1.15)	1688	
	2003–2004	189	13.52 (1.64)	1631	
	2005–2006	166	11.96 (1.00)	1514	
	2007–2008	91	13.82 (1.29)	831	
	2009–2010	97	11.67 (1.83)	882	
	2011–2012	75	9.80 (1.72)	732	
	2013–2014	77	8.59 (1.49)	868	
	2015–2016	54	6.49 (1.11)	770	
2017–2018	46	6.72 (1.25)	625		

SE, standard error; NHANES, The National Health and Nutrition Examination Survey

^aThe current cigarette smoking status was defined as serum cotinine >10 ug/mL

^bThe *p*-values of the Wald statistics were used to judge the association between current smoking status and the characteristics

^cA poverty index ratio (PIR) was calculated by comparing the midpoint for the family income category and the family size with the federal poverty line. A PIR <1 was defined as poor and 1–1.99 as close to poor

^dThe bodyweight category was based on directly measured height and weight. The BMI <85th percentile was defined as normal weight, 85–94.9th percentile as overweight, and 95th above as obese

not Black adolescents (Fig. 1). For adolescents aged 12 and 17 years old (top panel of Fig. 1), the change of biennial percentage points (β in the equations) in White adolescents was -1.0 ($p = 0.014$) and -0.34 ($p = 0.002$) for Hispanic adolescents. No sign of a significant decrease in CTS prevalence was detected among Black adolescents ($\beta = -0.49$, $p = 0.80$). For adolescents aged 18 and 19 years old, both

Whites ($\beta = -2.4$) and Hispanics ($\beta = -2.0$) experienced more than 2 percentage points decrease in CTS prevalence for every survey cycle (i.e., 2 years), whereas a non-significant increase in CTS prevalence was found among Blacks ($\beta = 0.20$, $p = 0.78$, the bottom panel of Fig. 1). A crossover of CTS prevalence between Blacks and Whites occurred during the study period in adolescents aged 18–19 years old. The

average CTS prevalence in surveys 1999–2008 was higher among Whites than Blacks, 13.65% (11.85%, 15.46%) vs. 8.80% (7.55%, 10.04%), but Blacks had a higher average prevalence compared to Whites for the surveys conducted between 2009 and 2018, 8.32% (6.53%, 10.12%) vs. 7.77% (5.86%, 9.68%). The survey cycles or calendar years linearly explained more than 70% of the variations in CTS prevalence for Hispanic adolescents, about 60% for White adolescents, but only 1% for Black adolescents aged 18–19 years (Fig. 1).

Without stratification and measured by PR, there were overall declining trends among adolescents, significantly for both young and late adolescence, declining by 12% [PR = 0.88(0.84, 0.92)] and 11% [0.89(0.85, 0.93)], respectively, for every survey cycle, i.e., 2 calendar years (Table 3). When stratified by race/ethnicity, the decreasing trends remained significant for Whites and Hispanics aged 12–17 years and 18–19 years. An increasing trend at borderline significance was revealed for Black adolescents aged 18–19 years after adjustment for other sociodemographic factors. The steepest decrease occurred among Hispanics aged 12–17 years [abiPR = 0.85(0.77, 0.94)], and the sharpest increase took place among Blacks aged 18–19 years [abiPR = 1.06(0.99, 1.14)]. The number of smokers living at home was strongly associated with the likelihood of smoking in adolescents across all races/ethnicities in both age groups.

Discussions

In nationally representative samples of US adolescents from multiple cross-sectional surveys, we observed overall declining trends in CTS among adolescents between 1999 and 2018. However, a crossover of CTS prevalence was detected between races/ethnicities after adjustment for other socio-economic factors; CTS prevalence decreased significantly for White and Hispanic adolescents, in both age groups (young and middle, and later adolescents), but increased for Black adolescents aged 18–19 years.

National surveys have consistently reported that the CTS prevalence is higher among Whites than Blacks in adolescence [5, 8, 9]. Most persuasively, with data from National Youth Tobacco Survey (2004–2013), National Survey on Drug Use and Health (2002–2013), National Health Interview Survey (2001–2013), and National Health and Nutrition Examination Survey (2001–2012), Caraballo and coworkers reported that in all self-reported surveys, Whites had a higher current smoking prevalence than Blacks [7]. Our results seemed consistent with previous reports until the 2011–2014 survey years when the crossover started to emerge. Multiple factors contributed to the decline in tobacco product use among youth, including the comprehensive implementation of population-based

strategies and continued research investments in cessation-related initiatives [9, 24, 25]. Technological advancement has changed the way adolescents socialize and project their identity, leaving shrinking opportunities for smoking to become a social exercise [26, 27]. Sociocultural explanations such as changes in the school environment [28], parenting, and general economic and labor market conditions [29] have also been postulated to have contributed to the overall declining trend of adolescent smoking.

The key finding of the current report was the crossover of CTS prevalence between races after adjustment for other socio-economic factors. CTS prevalence decreased significantly for White and Hispanic adolescents, in both young and middle and later adolescents, but increased for Black adolescents aged 18–19 years. Multiple factors may underline the increasing CTS prevalence for Black teenagers in later adolescence. Targeted advertising of cigarettes in locations with a high proportion of Black residents has been part of tobacco companies' marketing strategies. A relatively lower price and the availability of cigarettes for purchase might also contribute to the high smoking rates among Black teenagers [30]. Black men aged 26 years or older were observed to have a higher smoking prevalence than White men of the same age [7], and the percentage of smoking-free households was significantly lower among Black families than in Hispanic and White families in the current report. It is possible that increasing CTS prevalence in Black adolescents was due to increasing household exposure to tobacco smoking as the present study used serum cotinine to proxy current tobacco smoking. However, the increasing trend in Black adolescents was obtained after adjustment for the number of smokers living in the household. More importantly, the percentage of smoking-free households increased at roughly the same pace for races. Therefore, household exposure may explain only a small portion of the increasing trend of CTS in Black adolescents. Public health interventions such as the tobacco tax and smoke-free environment legislation have played a crucial role in decreasing CTS prevalence [9, 24, 25]. CTS prevalence has been declining simultaneously in developed countries with widely different regulatory contexts [31], but this is not true for Black youths aged 18–19 years old in the USA, suggesting race-specific or sociocultural factors rather than the tobacco control policies *per se* may be responsible for the increasing trend in Black adolescents.

The current study has strengths and limitations. We used the data from large surveys with representative of the national samples of adolescents over many years. Biomarkers were tested under uniform and rigorously controlled conditions. Repetitive analyses over time indicated the absence of unusual variations or drift in the analytic method [32]. Thus, the declining trend of CTS prevalence

Table 3 Adjusted prevalence ratio (95%CI) of current tobacco smoking, 10,760 adolescents aged 12–19, NHANES 1999–2018^{a,b}

	All combined			Black			Hispanic			White		
	12–17 years	18–19 years	12–17 years	18–19 years	12–17 years	18–19 years	12–17 years	18–19 years	12–17 years	18–19 years	12–17 years	18–19 years
Survey wave (continuous variable)												
Every 2 calendar years	0.88 (0.84, 0.92)	0.89 (0.85, 0.93)	0.95 (0.89, 1.01)	1.06 (0.99, 1.14)	0.85 (0.77, 0.94)	0.89 (0.81, 0.98)	0.88 (0.83, 0.93)	0.87 (0.82, 0.92)				
Child's age at interview												
One year of age	1.86 (1.74, 1.99)	1.22 (0.90, 1.66)	1.96 (1.73, 2.23)	0.89 (0.59, 1.33)	2.22 (1.86, 2.66)	1.36 (0.87, 2.15)	1.83 (1.68, 1.99)	1.25 (0.82, 1.91)				
Child's sex (reference group = boys)												
Girls	0.60 (0.45, 0.80)	0.41 (0.30, 0.56)	0.33 (0.22, 0.50)	0.29 (0.18, 0.47)	0.28 (0.16, 0.50)	0.36 (0.22, 0.60)	0.68 (0.48, 0.95)	0.45 (0.29, 0.70)				
Family income (reference group = poor)												
High income	0.47 (0.32, 0.69)	0.92 (0.59, 1.43)	0.54 (0.24, 1.18)	0.28 (0.13, 0.60)	0.74 (0.33, 1.67)	1.12 (0.44, 2.81)	0.31 (0.20, 0.48)	0.74 (0.39, 1.38)				
Middle income	0.58 (0.38, 0.90)	0.64 (0.41, 0.99)	0.52 (0.30, 0.90)	0.61 (0.31, 1.22)	0.40 (0.19, 0.82)	1.39 (0.67, 2.89)	0.48 (0.28, 0.80)	0.45 (0.23, 0.87)				
Near poor	0.64 (0.40, 1.02)	0.97 (0.64, 1.48)	0.73 (0.36, 1.49)	0.77 (0.38, 1.56)	0.43 (0.19, 1.00)	0.96 (0.47, 1.98)	0.58 (0.32, 1.07)	1.09 (0.53, 2.24)				
Directly measured body weight (reference group = healthy weights)												
Obese	0.71 (0.49, 1.02)	0.80 (0.56, 1.16)	0.47 (0.30, 0.75)	0.71 (0.42, 1.22)	1.00 (0.56, 1.78)	0.51 (0.22, 1.14)	0.81 (0.50, 1.31)	0.97 (0.57, 1.64)				
Overweight	1.02 (0.70, 1.50)	0.86 (0.56, 1.32)	0.90 (0.56, 1.43)	0.70 (0.39, 1.25)	1.21 (0.57, 2.58)	0.56 (0.29, 1.06)	1.10 (0.67, 1.80)	1.05 (0.55, 2.00)				
Underweight	1.13 (0.53, 2.41)	1.80 (0.89, 3.62)	1.05 (0.30, 3.68)	1.17 (0.52, 2.66)	1.64 (0.39, 6.89)	0.78 (0.22, 2.69)	1.03 (0.43, 2.46)	2.36 (0.93, 6.04)				
# of smokers at home (reference group = none)												
1	2.14 (1.55, 2.96)	2.50 (1.64, 3.79)	3.02 (1.94, 4.70)	2.35 (1.34, 4.13)	1.74 (0.66, 4.58)	3.13 (1.64, 5.97)	1.80 (1.15, 2.81)	2.26 (1.25, 4.08)				
2	4.17 (2.84, 6.13)	5.61 (3.70, 8.50)	6.37 (3.41, 11.9)	7.26 (3.63, 14.5)	8.25 (3.41, 19.9)	4.16 (1.90, 9.07)	2.95 (1.81, 4.80)	4.58 (2.54, 8.26)				
2+	11.8 (6.53, 21.5)	15.2 (5.65, 40.9)	7.20 (1.81, 28.6)	14.2 (1.12, 179)	10.4 (3.66, 29.8)	3.82 (1.31, 11.1)	8.24 (4.22, 16.1)	16.2 (4.77, 54.9)				
Covered by insurance (reference group = no)												
Yes	1.06 (0.70, 1.62)	0.93 (0.64, 1.36)	0.76 (0.40, 1.44)	0.73 (0.44, 1.20)	0.75 (0.38, 1.47)	0.91 (0.50, 1.65)	0.94 (0.52, 1.70)	0.78 (0.42, 1.43)				
Parental marital status (reference group = currently)												
Never	1.01 (0.66, 1.53)	2.12 (1.41, 3.19)	2.07 (1.34, 3.18)	2.45 (1.44, 4.19)	1.32 (0.44, 3.95)	2.83 (1.12, 7.16)	1.02 (0.35, 2.98)	1.99 (0.88, 4.49)				
Previously	1.79 (1.27, 2.52)	1.74 (1.27, 2.39)	2.57 (1.77, 3.73)	2.25 (1.30, 3.91)	1.95 (1.07, 3.56)	1.51 (0.81, 2.81)	1.71 (1.06, 2.78)	1.85 (1.15, 2.99)				

SE, standard error; NHANES, The National Health and Nutrition Examination Survey

^aCurrent smoking was defined based on serum cotinine (>10 ug/l), not self-reported^bSaturated logistic regressions models were applied; all variables listed in the first column of the table were included on the right side of the regression regardless of the *p*-value of the coefficients

proxied by serum cotinine and the race differences we observed over time most likely reflect corresponding epidemiological trends. However, it has been observed that pharmacokinetic differences exist between racial groups [33, 34]. Blacks have consistently higher serum cotinine concentrations per cigarette smoked than Whites [35–37], indicating that Blacks metabolize cotinine at a slower rate [23, 38]. The genetic differences in metabolism may have contributed, at least in part, to the racial differences in CTS prevalence proxied by serum cotinine [33–37]. Genetic predisposition, however, cannot explain the crossover of the trends between races within such a short time scale. There is a possibility that current smokers were misclassified as nonsmokers in our study, as infrequent smoking, common among adolescents, may have serum cotinine levels <10 ng/ml [39]. The Hispanic group is overly broad and does not consider the heterogeneity of the cultural backgrounds of people of Latin American descent. The participants of NHANES were sampled from non-institutionalized populations, excluding the adolescents held in the juvenile justice system, psychiatric hospitals, or other rehabilitation facilities. Adolescents living in these facilities typically have a higher prevalence of substance abuse than the general adolescent population [40], and Black juveniles are disproportionately detained at higher rates than Whites [41]. It must be pointed out that excluding more Black adolescents from the current analysis potentially caused underestimation rather than an overestimation of the racial differences (Supplementary Table S2).

Conclusion

There may be an overlap between the time-race crossover we observed and the race-sex-age crossover reported by Caraballo et al. [7]. Our observation cast doubts on the prevailing descriptions of the ethno-racial difference in tobacco use in the past decades. The emerging cross-over between races, if continued, will ruin overall tobacco cessation efforts and make health-related disparities run deeper since cigarette smoking begins as experimental smoking during youth and young adulthood [42]. An increase in prevalence among Black adolescents will be translated into increasing smoking among adults. Efforts are needed to harness the momentum created by the COVID-19 pandemic to scale up the effective components of policy interventions and determine the factors preventing the health gains of the population-wide policy interventions in Black communities.

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Author Contribution Zhang Jian: conceptualization, methodology, supervision. Samuel O Nwaobi and Holly L Richmond: data curation, investigation, original draft preparation. All authors: visualization, investigation, reviewing and editing.

Declarations

Ethics Approval This is an observational study. The Research Ethics Committee of the institute the senior authors affiliated with has confirmed that no ethical approval is required. Data collection agencies obtained informed consent from all participants included in the study.

Competing Interests The authors declare no competing interests.

Disclaimer The views expressed in this article are those of the authors and do not reflect the official policy of the National Center for Health Statistics, Centers for Disease Control and Prevention, which is responsible only for the initial data.

References

1. Arrazola RA, Singh T, Corey CG, Husten CG, Neff LJ, Apelberg BJ, Bunnell RE, Choiniere CJ, King BA, Cox S, McAfee T, Caraballo RS. Tobacco use among middle and high school students – United States, 2011–2014. *MMWR Morb Mortal Wkly Rep.* 2015;64:381–5.
2. U.S. Department of Health and Human Services. Tobacco Use Among U.S. Racial/Ethnic Minority Groups—African Americans, American Indians and Alaska Natives, Asian Americans and Pacific Islanders, and Hispanics: A Report of the Surgeon General. Atlanta, Georgia: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health, 1998.
3. Gentzke AS, Creamer M, Cullen KA, Ambrose BK, Willis G, Jamal A, King BA. Vital signs: tobacco product use among middle and high school students – United States, 2011–2018. *MMWR Morb Mortal Wkly Rep.* 2019;68:157–64. <https://doi.org/10.15585/mmwr.mm6806e1>.
4. Kann L, McManus T, Harris WA, Shanklin SL, Flint KH, Queen B, Lowry R, Chyen D, Whittle L, Thornton J, Lim C, Bradford D, Yamakawa Y, Leon M, Brener N, Ethier KA. Youth risk behavior surveillance – United States, 2017. *MMWR Surveill. Summ.* 2018;67:1–114. <https://doi.org/10.15585/mmwr.ss6708a1>.
5. Eaton DK, Kann L, Kinchen S, Shanklin S, Flint KH, Hawkins J, Harris WA, Lowry R, McManus T, Chyen D, Whittle L, Lim C, Wechsler H, Centers for Disease Prevention. Youth risk behavior surveillance – United States 2011. *MMWR Surveill. Summ.* 2012;61(2012):1–162.
6. Kandel D, Schaffran C, Hu MC, Thomas Y. Age-related differences in cigarette smoking among Whites and African-Americans: evidence for the crossover hypothesis. *Drug Alcohol Depend.* 2011;118:280–7. <https://doi.org/10.1016/j.drugalcdep.2011.04.008>.
7. Caraballo RS, Sharapova SR, Asman KJ. Does a race-gender-age crossover effect exist in current cigarette smoking between non-Hispanic Blacks and non-Hispanic Whites? *United States,*

- 2001–2013. *Nicotine Tob Res.* 2016;18(1):S41–48. <https://doi.org/10.1093/ntr/ntv150>.
8. Pampel FC. Racial convergence in cigarette use from adolescence to the mid-thirties. *J Health Soc Behav.* 2008;49:484–98.
 9. Pampel FC, Aguilar J. Changes in youth smoking, 1976–2002: a time-series analysis. *Youth Soc.* 2008;39:453–79. <https://doi.org/10.1177/0044118X07308070>.
 10. Connor Gorber S, Schofield-Hurwitz S, Hardt J, Levasseur G, Tremblay M. The accuracy of self-reported smoking: a systematic review of the relationship between self-reported and cotinine-assessed smoking status. *Nicotine Tob Res.* 2009;11:12–24. <https://doi.org/10.1093/ntr/ntn010>.
 11. Wills TA, Cleary SD. The validity of self-reports of smoking: analyses by race/ethnicity in a school sample of urban adolescents. *Am J Public Health.* 1997;87:56–61. <https://doi.org/10.2105/ajph.87.1.56>.
 12. Bauman KE, Ennett SE. Tobacco use by Black and White adolescents: the validity of self-reports. *Am J Public Health.* 1994;84:394–8. <https://doi.org/10.2105/ajph.84.3.394>.
 13. Caraballo RS, Giovino GA, Pechacek TF. Self-reported cigarette smoking vs. serum cotinine among U.S. adolescents. *Nicotine Tob Res.* 2004;6:19–25. <https://doi.org/10.1080/14622200310001656821>.
 14. Cantrell J, Huang J, Greenberg MS, Xiao H, Hair EC, Vallone D. Impact of e-cigarette and cigarette prices on youth and young adult e-cigarette and cigarette behaviour: evidence from a national longitudinal cohort. *Tob Control.* 2020;29:374–80. <https://doi.org/10.1136/tobaccocontrol-2018-054764>.
 15. A David, K Esson, AM Perucic, C Fitzpatrick, (2010) Tobacco use: equity and social determinants., in: E. Blas, A. Sivasankara Kurup (Eds.) *Equity, social determinants and public health programmes*, World Health Organization., Geneva 199-218.
 16. M The Lancet Respiratory. COVID-19, smoking, and cancer: a dangerous liaison. *Lancet Respir Med.* 2021;9:937. [https://doi.org/10.1016/s2213-2600\(21\)00373-8](https://doi.org/10.1016/s2213-2600(21)00373-8).
 17. Klein JD, Resnick EA, Chamberlin ME, Kress EA. Second-hand smoke surveillance and COVID-19: a missed opportunity. *Tob Control.* 2021. <https://doi.org/10.1136/tobaccocontrol-2021-056532>.
 18. Martin B, DeWitt PE, Russell S, Anand A, Bradwell KR, Bremer C, Gabriel D, Girvin AT, Hajagos JG, McMurry JA, Neumann AJ, Pfaff ER, Walden A, Wooldridge JT, Yoo YJ, Saltz J, Gersing KR, Chute CG, Haendel MA, Moffitt R, Bennett TD. Characteristics, outcomes, and severity risk factors associated with SARS-CoV-2 infection among children in the US national COVID cohort collaborative. *JAMA Netw Open.* 2022;5:e2143151. <https://doi.org/10.1001/jamanetworkopen.2021.43151>.
 19. Fernandes DM, Oliveira CR, Guerguis S, Eisenberg R, Choi J, Kim M, Abdelhemid A, Agha R, Agarwal S, Aschner JL, Avner JR, Ballance C, Bock J, Bhavsar SM, Campbell M, Clouser KN, Gesner M, Goldman DL, Hammerschlag MR, Hymes S, Howard A, Jung HJ, Kohlhoff S, Kojaoghlanian T, Lewis R, Nachman S, Naganathan S, Paintsil E, Pall H, Sy S, Wadowski S, Zirinsky E, Cabana MD, Herold BC. Severe acute respiratory syndrome coronavirus 2 clinical syndromes and predictors of disease severity in hospitalized children and youth. *J Pediatr.* 2021;230:23–31. <https://doi.org/10.1016/j.jpeds.2020.11.016>.
 20. Götzinger F, Santiago-García B, Noguera-Julián A, Lanaspá M, Lancella L, Calò Carducci FI, Gabrovská N, Velizarova S, Prunk P, Osterman V, Krivec U, Lo Vecchio A, Shingadia D, Soriano-Arandes A, Melendo S, Lanari M, Pierantoni L, Wagner N, L'Huillier AG, Heininguer U, Ritz N, Bandi S, Krájcar N, Roglič S, Santos M, Christiaens C, Creuven M, Buonsenso D, Welch SB, Bogyi M, Brinkmann F, Tebruegge M. COVID-19 in children and adolescents in Europe: a multinational, multicentre cohort study. *Lancet Child Adolesc Health.* 2020;4:653–61. [https://doi.org/10.1016/s2352-4642\(20\)30177-2](https://doi.org/10.1016/s2352-4642(20)30177-2).
 21. Johnson CL, Paulose-Ram R, Ogden CL, Carroll MD, Kruszon-Moran D, Dohrmann SM, Curtin LR. National health and nutrition examination survey: analytic guidelines, 1999–2010. *Vital Health Stat.* 2013;2:1–24.
 22. Office of Disease Prevention and Health Promotion, the U.S. Department of Health and Human Services. *Healthy People 2020*. <https://www.healthypeople.gov/2020/topics-objectives/topic/tobacco-use/objectives>. Accessed 18 Aug 2022.
 23. Hukkanen J, Jacob P 3rd, Benowitz NL. Metabolism and disposition kinetics of nicotine. *Pharmacol Rev.* 2005;57:79–115. <https://doi.org/10.1124/pr.57.1.3>.
 24. White VM, Warne CD, Spittal MJ, Durkin S, Purcell K, Wakefield MA. What impact have tobacco control policies, cigarette price and tobacco control programme funding had on Australian adolescents' smoking? Findings over a 15-year period. *Addiction.* 2011;106:1493–502. <https://doi.org/10.1111/j.1360-0443.2011.03429.x>.
 25. White VM, Durkin SJ, Coomber K, MA. Wakefield, What is the role of tobacco control advertising intensity and duration in reducing adolescent smoking prevalence? Findings from 16 years of tobacco control mass media advertising in Australia. *Tob Control.* 2015;24:198–204. <https://doi.org/10.1136/tobaccocontrol-2012-050945>.
 26. Iannotti RJ, Kogan MD, Janssen I, WF. Boyce, Patterns of adolescent physical activity, screen-based media use, and positive and negative health indicators in the U.S. and Canada. *J Adolesc Health.* 2009;44:493–9. <https://doi.org/10.1016/j.jadohealth.2008.10.142>.
 27. Charlton A, Bates C. Decline in teenage smoking with rise in mobile phone ownership: hypothesis. *BMJ.* 2000;321:1155.
 28. Cornell D, Huang F. Authoritative school climate and high school student risk behavior: a cross-sectional multi-level analysis of student self-reports. *J Youth Adolesc.* 2016;45:2246–59. <https://doi.org/10.1007/s10964-016-0424-3>.
 29. Settersten RA Jr, Ray B. What's going on with young people today? The long and twisting path to adulthood. *Future Child.* 2010;20:19–41. <https://doi.org/10.1353/foc.0.0044>.
 30. Corey CG, Dube SR, Ambrose BK, King BA, Apelberg BJ, Husten CG. Cigar smoking among U.S. students: reported use after adding brands to survey items. *Am J Prev Med.* 2014;47:S28-35. <https://doi.org/10.1016/j.amepre.2014.05.004>.
 31. Ball J, Sim D, Edwards R. Why has adolescent smoking declined dramatically? Trend analysis using repeat cross-sectional data from New Zealand 2002–2015. *BMJ Open.* 2018;8:e020320. <https://doi.org/10.1136/bmjopen-2017-020320>.
 32. Pirkle JL, Bernert JT, Caudill SP, Sosnoff CS, Pechacek TF. Trends in the exposure of nonsmokers in the U.S. population to secondhand smoke: 1988–2002. *Environ Health Perspect.* 2006;114:853–8. <https://doi.org/10.1289/ehp.8850>.
 33. Wilson SE, Kahn RS, Khoury J, Lanphear BP. Racial differences in exposure to environmental tobacco smoke among children. *Environ Health Perspect.* 2005;113:362–7. <https://doi.org/10.1289/ehp.7379>.
 34. Benowitz NL, Perez-Stable EJ, Fong I, Modin G, Herrera B, Jacob P 3rd. Ethnic differences in N-glucuronidation of nicotine and cotinine. *J Pharmacol Exp Ther.* 1999;291:1196–203.
 35. Wagenknecht LE, Manolio TA, Sidney S, Burke GL, Haley NJ. Environmental tobacco smoke exposure as determined by cotinine in Black and White young adults: the CARDIA Study. *Environ Res.* 1993;63:39–46. <https://doi.org/10.1006/enrs.1993.1124>.
 36. Wagenknecht LE, Cutter GR, Haley NJ, Sidney S, Manolio TA, Hughes GH, Jacobs DR. 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. <https://doi.org/10.2105/ajph.80.9.1053>.
37. Caraballo RS, Giovino GA, Pechacek TF, Mowery PD, Richter PA, Strauss WJ, Sharp DJ, Eriksen MP, Pirkle JL, Maurer KR. 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. <https://doi.org/10.1001/jama.280.2.135>.
 38. Perez-Stable EJ, Herrera B, Jacob P 3rd, Benowitz NL. Nicotine metabolism and intake in Black and White smokers. *JAMA*. 1998;280:152–6. <https://doi.org/10.1001/jama.280.2.152>.
 39. Kim S, Apelberg BJ, Avila-Tang E, Hepp L, Yun D, Samet JM, Breyse PN. Utility and cutoff value of hair nicotine as a biomarker of long-term tobacco smoke exposure, compared to salivary cotinine. *Int J Environ Res Public Health*. 2014;11:8368–82. <https://doi.org/10.3390/ijerph110808368>.
 40. A Committee on. Health care for youth in the juvenile justice system. *Pediatrics*. 2011;128:1219–35. <https://doi.org/10.1542/peds.2011-1757>.
 41. Robles-Ramamurthy B, Watson C. Examining racial disparities in juvenile justice. *J Am Acad Psychiatry Law*. 2019;47:48–52. <https://doi.org/10.29158/JAAPL.003828-19>.
 42. National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health. The health consequences of smoking-50 years of progress: a report of the surgeon general, Centers for Disease Control and Prevention (US), Atlanta, Georgia, USA. 2014.

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