



# Racial differences in alcohol and tobacco use in adolescence and mid-adulthood in a community-based sample

John R. Pamplin II.<sup>1</sup> · Ezra S. Susser<sup>1,2</sup> · Pam Factor-Litvak<sup>1</sup> · Bruce G. Link<sup>3</sup> · Katherine M. Keyes<sup>1,4</sup>

Received: 18 December 2018 / Accepted: 10 September 2019 / Published online: 21 September 2019  
© Springer-Verlag GmbH Germany, part of Springer Nature 2019

## Abstract

**Purpose** Smoking and alcohol use have been posited as possible contributors to racial health disparities, despite higher smoking and alcohol use among non-Hispanic White youth and young adults compared to Blacks. To further investigate this claim, we aim to assess variation in alcohol and cigarette use across two distinct points of the life course.

**Method** Data are from a subset of 559 (279 male, 280 female) self-identified Black and White participants of the Child Health and Development study. Self-report alcohol and cigarette use were collected between age 15–17 and at mean age 50. Logistic regressions were estimated; supplementary analyses adjusted for maternal age, prenatal smoking, household income, childhood SES, and education.

**Results** White participants were more likely to drink regularly (Odds ratio (OR) 2.2; 95%CI 1.2, 4.0) and be intoxicated (OR 2.0; 95%CI 1.2, 3.2) in adolescence compared with Blacks. In mid-adulthood, Whites remained more likely to currently drink (OR 2.3; 95%CI 1.6, 3.4) but among drinkers, less likely to binge drink (OR 0.4; 95%CI 0.2, 0.8). White participants were less likely to smoke in mid-adulthood (OR 0.4; 95%CI 0.3, 0.6), but among smokers, were more likely to smoke  $\geq 1/2$  a pack per day (OR 3.4; 95%CI 1.5, 7.8).

**Conclusions** Blacks were less likely to engage in drinking across the life course, but, among drinkers, more likely to binge drink in mid-adulthood. Blacks were more likely to smoke in mid-adulthood, but smoked infrequently compared with Whites. These patterns suggest that a reframing of disparities mechanisms to focus on broader structural and social factors may benefit progress in understanding and ameliorating inequities.

**Keywords** Racial differences · Alcohol use · Tobacco use · Adolescence · Adulthood

---

**Electronic supplementary material** The online version of this article (<https://doi.org/10.1007/s00127-019-01777-9>) contains supplementary material, which is available to authorized users.

---

✉ John R. Pamplin II.  
Jrp2166@cumc.columbia.edu

<sup>1</sup> Department of Epidemiology, Mailman School of Public Health, Columbia University, 722 W. 168th St., 7th Floor, 720-D, New York, NY 10032-3727, USA

<sup>2</sup> New York State Psychiatric Institute, New York, NY, USA

<sup>3</sup> Department of Sociology, University of California Riverside, Riverside, CA, USA

<sup>4</sup> Center for Research on Society and Health, Universidad Mayor, Santiago, Chile

## Introduction

Black individuals in the United States suffer disproportionately worse health than Whites across multiple health outcomes [1–5]. Much attention has been directed towards unhealthy behaviors, such as smoking, alcohol use, and poor diet, in the production of these disparities [6, 7]. However, for these behaviors to produce racial health disparities, Black individuals would either need to engage in unhealthy behaviors to a greater extent than Whites, or unhealthy behaviors would need to have differential effects on health among Blacks compared to Whites. One of the more prominent hypotheses for racially patterned substance use is that Black individuals may engage in unhealthy behaviors to a greater extent than Whites as a means of coping with race-based discriminatory stress [6, 8–10]. Substance use (cigarettes and alcohol in particular, as they are the most commonly used substances in the US) [11], has been

characterized as a coping mechanism in response to stress, primarily due to its ability to reduce negative affect, increase positive affect, and its short-term anxiolytic effects [12–14].

Evidence regarding this hypothesis has been mixed; one study demonstrated that Black participants experienced more stressors and were slightly more likely to engage in unhealthy behaviors than Whites, and that unhealthy behaviors were associated with increased odds of chronic health conditions [9]. In another study, a relationship between unhealthy behaviors and chronic health conditions was observed for Black, but not White participants, though neither stressors nor unhealthy behaviors were more prevalent for Black participants in that sample [6]. Alternatively, other studies have shown that though the prevalence of unhealthy behaviors increases with stressor exposure, it does so for both White and Black participants. In addition, these studies show that at any level of stressor exposure, only high BMI (intended as proxy for poor diet) is more prevalent among Black participants than Whites; cigarette and alcohol use are both less common among Blacks [15, 16]. Overall, the extent of the role of substance use in producing physical health disparities remains contentious.

Many studies have found that Black adolescents are less likely to use alcohol and tobacco than their White counterparts [17–24], a pattern that continues into young adulthood [19]. However, convergence in rates of substance use in adulthood has been observed, a phenomenon frequently referred to as “the crossover effect” [25]. In particular, cigarette use increases for both Blacks and Whites entering adulthood [26], but Black individuals in the US on average have a later age of smoking initiation than Whites and are less likely to desist from smoking during the transition to adulthood, resulting in the convergence of rates [19, 27–29]. Mechanisms underlying this convergence include the relative lack of material resources among Black individuals, which limits access to smoking cessation products [27]. Alcohol use also increases among both Black and White adolescence as they enter adulthood [26]. Rates of heavy drinking begin to converge for Black and White males in early adulthood [19, 30, 31]; however, Whites remain more likely to engage in alcohol use as well as heavy drinking during early to mid-adulthood [19].

A key to understanding the role of unhealthy behaviors in promoting health disparities is to examine variability over the life course [19, 27, 32], as chronic illnesses that disproportionately affect people of color, such as heart disease and stroke, are sensitive to changes in usage patterns [33]. For example, atherosclerosis risk is not just higher among smokers, but continues to increase as a product of pack-years smoked [34, 35]. Alternatively, smoking cessation has been shown to have beneficial effects on stroke risk, with the risk of stroke being approximately that of non-smokers, after about 5 years of smoking cessation [36]. In other words, to

fully capture long-term chronic disease risk, it is necessary to assess substance use dynamically across time. A number of studies have tracked substance-use patterns into early adulthood; however, few have documented usage patterns into middle adulthood. This is a critical period for racial patterns in cigarette and alcohol use as many of the chronic cardiometabolic diseases that disproportionately plague Black Americans have onsets during this point of the life course.

The present paper focuses on testing the extent and magnitude of racial differences in alcohol and tobacco use comparing Black individuals to Whites, assessed during both adolescence (age 15–17) as well as middle adulthood (mean age 50). Though this study does not assess usage in early adulthood, patterns of usage during this period of the life course have been well documented. In addition, to further test the hypothesis that racial substance-use patterns are driven by differential coping strategies, we assessed whether Black and White participants differed in their likelihood of turning to alcohol or cigarettes as a means to cope with stress. We focus on alcohol and cigarette use given their high prevalence [37, 38] and association with chronic disease [39–41]. A number of studies have examined racial differences in alcohol and smoking, but few do so across two diverse developmental periods in the life course. In this study, we intend to test the hypothesis that Black individuals will not exhibit greater substance use than White individuals, during adolescence, during mid-adulthood, or during the two time points considered conjointly.

Given that many of the health outcomes that disproportionately affect Black individuals manifest later in life, characterizing life course patterns of alcohol and cigarette use that extend beyond early adulthood is critical to advancing the literature. The data presented here are unique in that alcohol and cigarette use were assessed in the same individuals, both during adolescence as well as during mid-adulthood, allowing for insight into usage patterns across a considerably larger portion of the lifespan.

## Methods

### Study population

The Child Health and Development Study (CHDS) is a prospective study of pregnant women (and their offspring) who resided in the East Bay Area of California and were enrolled in the Kaiser Permanente Health Plan from 1959 to 1966 [42, 43]. The cohort is comprised of 19,044 live born offspring who were immediately enrolled into the health plan. Follow-up assessments of subgroups were conducted at three time points during development: 5 years of age, 9–11 years, and adolescence (15–17 years). An additional follow-up study was conducted in middle adulthood (mean age 50;

range 45–52 years) on a subset of participants sampled to study racial disparities [44]; details of the selection of the mid-adulthood sample from the original cohort including study flow diagram are provided elsewhere [44].

The mid-adulthood follow-up sample is comprised of a subset of the adult offspring who had serial assessments in childhood [44]. A 50% random sample of the non-Black and 100% of the Black participants who were assessed either in childhood or adolescence comprised the eligible sample, which was then restricted to California residents and those with a valid phone number. Of the 1073 participants from the eligible pool, 605 participated in the adult follow-up. Further details on the mid-adulthood follow-up sample have been published previously [44]. Presented analyses are restricted to participants of the adult follow-up study who self-identified as either Black or White ( $n=559$ ), of which 431 were assessed during adolescence. The final sample was 45% Black ( $n=252$ ) and 50% female ( $n=280$ ); median household income was \$87,500 (\$62,500 among Blacks and \$125,000 among Whites), and 42% had a college degree or greater (32% of Blacks and 51% of Whites). Mid-adulthood data were collected from 2010 to 2012. Participants were asked to complete a computer-assisted telephone interview (CATI), take part in a home visit, and fill out a self-administered questionnaire. Data collection and study analyses were approved by the Institutional Review Board of Columbia University, New York, NY, and the Public Health Institute, Oakland, CA.

## Outcome variables

### Alcohol consumption

**Adolescent assessment** Participants were asked how often they had consumed alcohol in the past 6 months with those who endorsed  $\geq 1$  drink being defined as current drinkers. A 3-level ordinal variable of frequency of consumption was created, operationalized as never,  $< 2$ –3 times a month, or  $\geq 2$ –3 times/month; categories were chosen to maximize sample size within each category.

Respondents were also asked how often they felt “high or tight” from drinks in the past year (categorized as ever versus never). In addition, an ordinal variable was created: never,  $< \text{once per month}$ , or  $\geq \text{once per month}$ .

**Mid-adulthood assessment** Participants were asked how often they consumed alcohol. Ever drinking was defined as ever having a period of consuming at least one drink per month for 6 consecutive months; current drinking was defined as consuming  $\geq 1$  drink per month for each of the past 6 consecutive months. Those who reported being current drinkers were also asked how many times they had consumed  $\geq 5$  drinks in a single sitting in the past 12 months (binge drink-

ing). Although current standards define binge drinking for women as  $\geq 4$  drinks in a single sitting [45], the questionnaire design did not allow for gender-based binge drinking designations; thus, we may underestimate binge drinking in women. Participants were also asked to describe how often they cope with stress by drinking alcohol along a 4-point Likert scale. The variable was dichotomized as some of the time/most of the time relative to rarely/never.

### Cigarette use

**Adolescent assessment** Participants were asked if they had ever ‘regularly’ smoked at least 1 cigarette daily and if they currently smoked cigarettes. Those who reported smoking currently were asked on average how many cigarettes they smoked per day. To remain consistent with the mid-adulthood assessment, responses were categorized as  $\geq \frac{1}{2}$  a pack/day (10 or more cigarettes) relative to  $< \frac{1}{2}$  a pack/day.

**Mid-adulthood assessment** Participants were asked if they had ever smoked regularly at least one cigarette daily, as well as if they had smoked cigarettes at all in the past 12 months, or the past 30 days. Those who reported smoking in the last 30 days were asked how many cigarettes they smoked a day on average. Among White smokers, 44%, 13%, and 2% reported 10–19 cigarettes/day, 20–29 cigarettes/day, and  $\geq 30$  cigarettes/day, respectively. Among Black smokers, 40% reported smoking 10–19 cigarettes/day; none reported 20 or more. Based on this distribution, amount smoked/day was operationalized as smoking  $\geq \frac{1}{2}$  a pack (or 10 or more cigarettes)/day relative to  $< \frac{1}{2}$  a pack/day. Participants were also asked on a 4-point Likert scale how often they smoked cigarettes in an attempt to alleviate stress; this was dichotomized as some or most of the time relative to rarely or never.

### Race

Offspring were classified by CHDS staff as either non-Black or Black, based on the self-identified race of the parents (both mother and father when available). Biracial offspring with one self-identified Black parent were categorized as Black. Mid-adulthood self-assessment of respondent race was consistent with birth race for all but one participant [44]. Of the 351 participants categorized as non-Black, 87% self-identified as White (6% as Asian, 5% as Hispanic, and 2% as another racial/ethnic group). Non-Black participants who did not self-identify as White were excluded ( $n=44$ ).

### Covariates

Socioeconomic status (SES) was operationalized using three different measures: a composite childhood SES variable, adult household income, and educational attainment.

Childhood SES was measured by reports of maternal education, paternal occupation, and family income at birth, age 9–11, and adolescence. Due to variation in assessment, income was standardized within those who received the same assessments, and SES indicators were weighted and combined using the procedure outlined by Link et al. [44].

At the mid-adulthood assessment, household income was categorized as \$2500–\$42,499, \$42,500–\$87,499, \$87,500–\$174,999, and \$175,000+ based on data distributions. Educational attainment was operationalized as high school graduate or less, some college, college degree, or post-college.

Maternal smoking status (smoker vs. non-smoker) during pregnancy and maternal age at birth ( $\geq 28$  vs.  $< 28$ ), both associated with adolescent substance use in this sample [46, 47], were also included.

### Statistical analysis

Logistic regression and multinomial logistic regression were used to estimate the associations between race and alcohol use, and race and cigarette use during adolescence and in mid-adulthood. Analyses of variables reflecting heavy alcohol or cigarette use (i.e., binge drinking/intoxication, and smoking  $\geq \frac{1}{2}$  a pack of cigarettes/day) were conducted, restricted to participants who reported current drinking or smoking, as well as among all participants.

Because Black race in the United States involves multiple manifestations of discrimination [48], many of the socioeconomic variables that are often conceptualized as confounders of associations between race and outcomes [49], are influenced by racialized group membership. As such, SES is a mediator between race and substance use, not a confounder [50, 51]. To represent the totality of the effect of race on substance use, we report results unadjusted for SES in our main analyses. We also performed supplementary analyses, adjusting for maternal characteristics and SES (childhood SES, household income during the mid-adulthood assessment, and educational attainment) to determine robustness of the model to multiple specifications.

### Missing data strategy

Three multiple imputation strategies were used, with slight variations in the predictor inputs based on whether the data were used for mid-adulthood analyses, adolescent analyses, or both assessments. All models were imputed prior to the exclusion of non-Black participants who self-identified as something other than White. Therefore, any missing data for the 605 respondents of the mid-adulthood follow-up were imputed for the mid-adulthood analyses, while only missing data for the 477 individuals who participated in the adolescent assessment were imputed

for the adolescent-only and both assessment analyses. Detailed information is provided in an online supplement. We imputed 50 data sets for each of the three analyses. Regressions for each of the imputations were estimated and recombined using Rubin's rules [52]. Sensitivity analyses were performed by comparing imputation estimates to the complete case estimates (Table S1, Table S2).

## Results

Racial differences in alcohol and cigarette consumption are shown at both the adolescent assessment as well as the mid-adulthood assessment in Table 1. As the objective of this analysis was to estimate the total effects of race on alcohol and cigarette outcomes, we are presenting results from the unadjusted models. Results from the adjusted models are provided in supplemental figures (Figs. S1–S4). We interpret these estimates as the direct effect of race on substance use, not mediated by SES and maternal characteristics. Though adjustment did result in wider confidence intervals which in some cases impacted statistical significance (this is to be expected when adding variables to a model), neither adjustment for maternal characteristics nor SES appreciably changed the point estimates of the results.

**Table 1** Racial differences in smoking and drinking during adolescence and in mid-adulthood in a sub-sample of the Child Health and Development Study ( $n=559$ )

		n(%)		
		Black	White	
<sup>a</sup> Adolescent smoking				
Never	361 (84)	100 (81)	261 (85)	$P=0.27$
Current	70 (16)	24 (19)	46 (15)	
<sup>a</sup> Adolescent drinking				
Never	131 (30)	56 (45)	75 (24)	$P<0.001$
<2–3 times/month	222 (52)	49 (40)	173 (56)	
$\geq 2$ –3 times/month	78 (18)	19 (15)	59 (20)	
Mid-adulthood smoking				
Never	109 (20)	47 (19)	62 (20)	$P<0.001$
Former	324 (58)	129 (51)	195 (64)	
Current	126 (22)	76 (30)	50 (16)	
Mid-adulthood drinking				
Never	137 (25)	85 (34)	52 (17)	$P<0.001$
Former	120 (21)	61 (24)	59 (19)	
Current	302 (54)	106 (42)	196 (64)	

<sup>a</sup>Adolescent variables were obtained only from those who participated in the adolescent assessment ( $n=431$ )

### Adolescent drinking

The unadjusted odds ratios of alcohol use at both assessments are displayed in Fig. 1. During adolescence, White participants had higher odds of being current drinkers (OR 2.6; 95%CI 1.6, 4.0) and of drinking  $\geq 2-3$  times/month (OR 2.3; 95%CI 1.3, 4.4) compared to Black participants. Among participants who reported drinking during the adolescent assessment, there were no statistically significant differences between Blacks and Whites regarding intoxication. However, among all respondents, White participants were more likely to report being intoxicated in the past year (OR 2.2; 95%CI 1.4, 3.3) and to report being intoxicated on average  $\geq$  once per month relative to none (OR 2.2; 95%CI 1.4, 3.6).

### Mid-adulthood drinking

During the mid-adulthood assessment, White participants had higher odds of being a current drinker (OR 2.6; 95%CI 1.7, 3.8). Among current drinkers, Whites had lower odds of binge drinking in the previous 6 months, (OR 0.4; 95%CI 0.2, 0.8); however, among all respondents, racial groups were not statistically significantly different in terms of binge drinking (OR 0.6; 95%CI 0.3, 1.3). Absolute differences in binge drinking were small; 6% of Black participants and 4% of White participants report binge drinking. Black

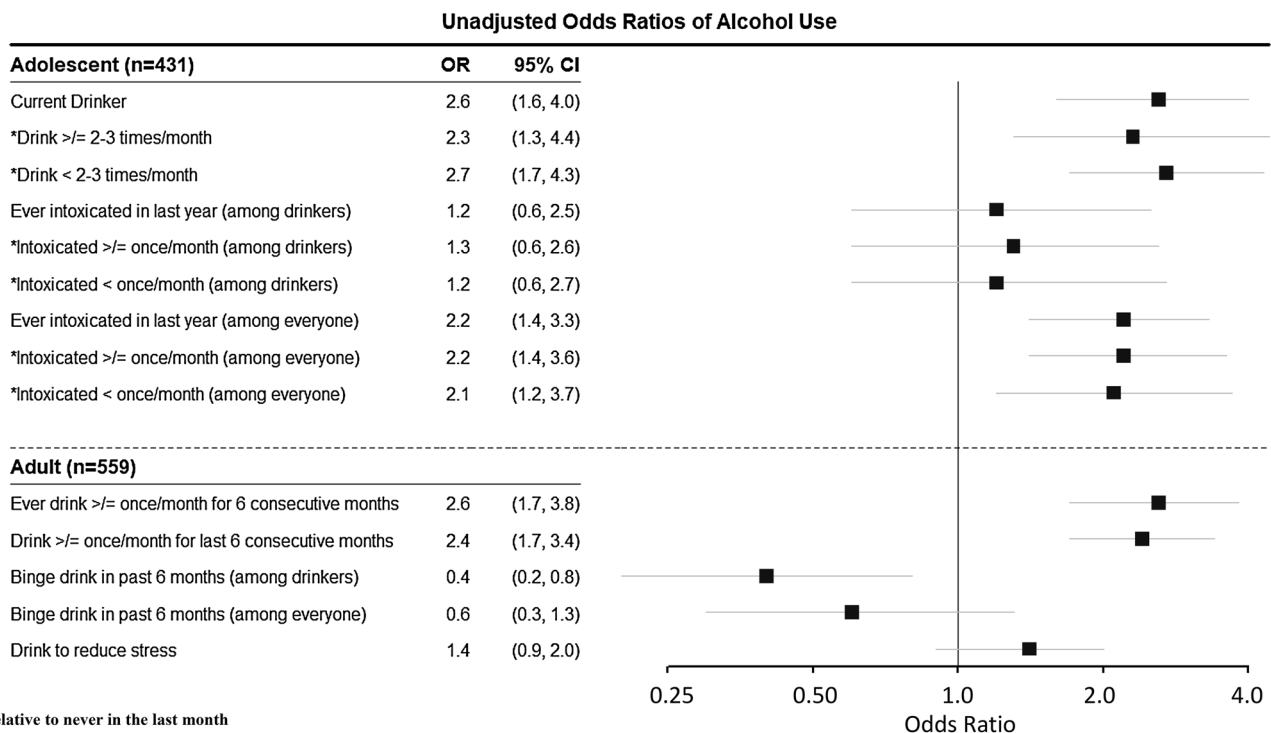
participants were not statistically significantly different from White participants in their odds of drinking to reduce stress (OR 1.4; 95%CI 0.9, 2.0).

### Adolescent cigarette use

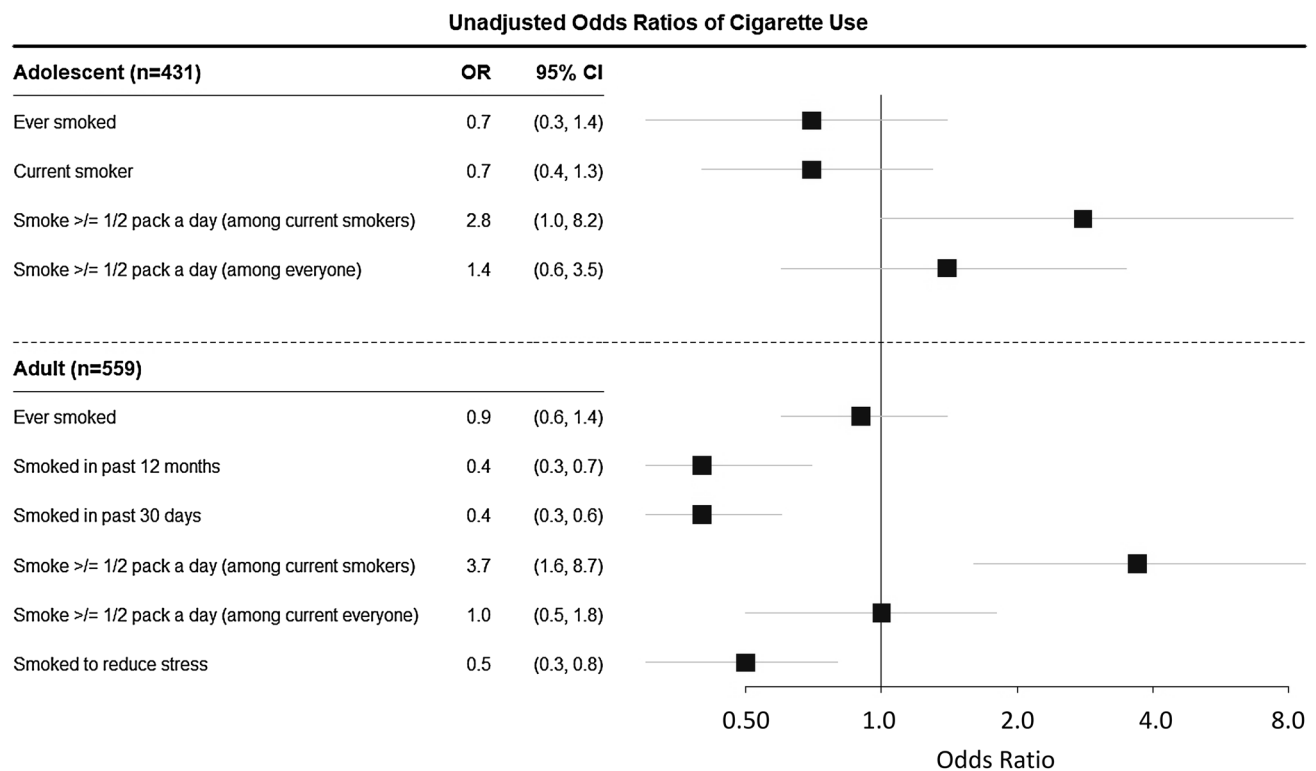
The unadjusted odds ratios of cigarette use at the adolescent and mid-adulthood assessments are displayed in Fig. 2. White and Black participants were not significantly different in their odds of ever or currently smoking cigarettes, or in the odds of smoking  $\geq \frac{1}{2}$  a pack of cigarettes/day, during the adolescent assessment. When restricting to adolescent current smokers, Whites had 2.8 times the odds of smoking  $\geq \frac{1}{2}$  a pack of cigarettes/day, relative to Black participants (95%CI 1.0, 8.2).

### Mid-adulthood cigarette use

During the mid-adulthood assessment, White participants had lower odds of being a current smoker (OR 0.4; 95%CI 0.3, 0.7) and of smoking to reduce stress (OR 0.5; 95%CI 0.3, 0.8). Among current smokers, White participants had substantially greater odds of smoking  $\geq \frac{1}{2}$  a pack of cigarettes/day (OR 3.7; 95%CI 1.6, 8.7) relative to Black participants, though among the total sample, there was no statistically significant difference.



**Fig. 1** Unadjusted odds ratios of alcohol use. Odds of alcohol use during adolescence (mean age = 17 years; n = 431) and mid-adulthood (mean age = 50 years; n = 599) in White as compared to Black (ref.) participants of a sub-sample of the Child Health and Development Study



**Fig. 2** Unadjusted odds ratios of cigarette use. Odds of cigarette use during adolescence (mean age=17 years;  $n=431$ ) and mid-adulthood (mean age=50 years;  $n=559$ ) in White as compared to Black (ref.) participants of a sub-sample of the Child Health and Development Study

### Patterns of substance use

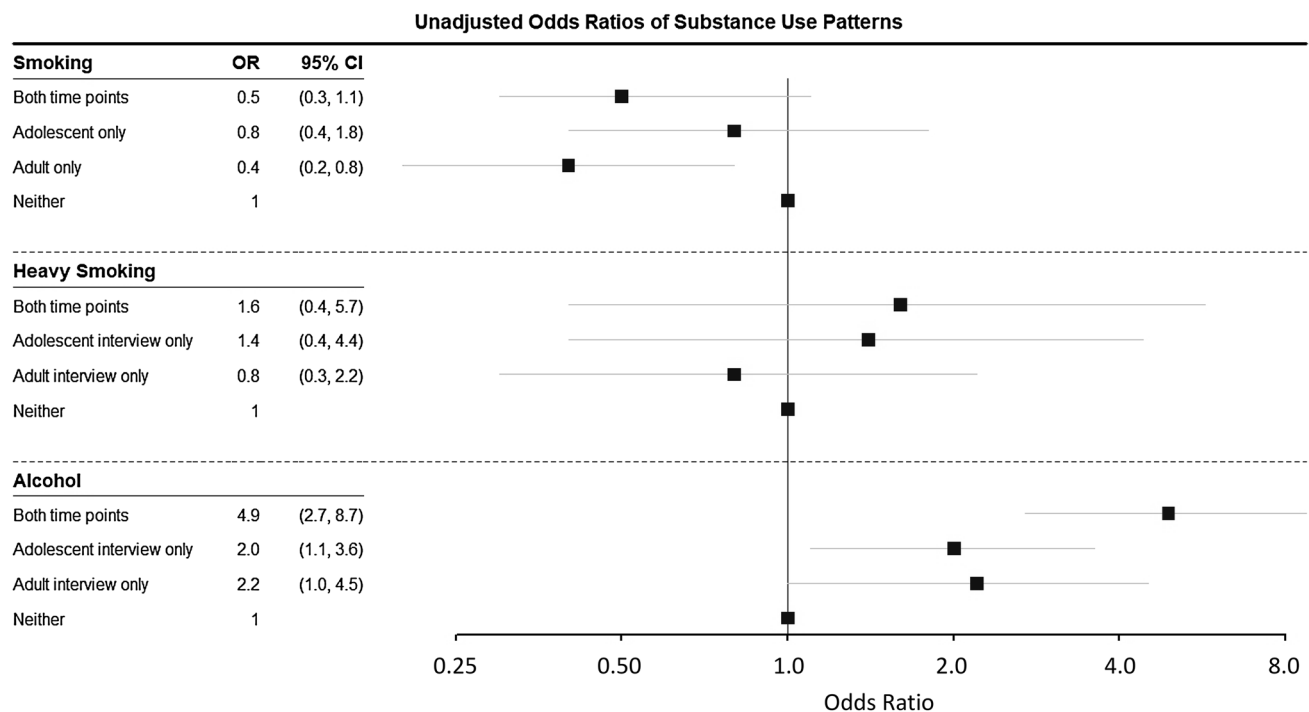
Figure 3 shows patterns of smoking, heavy smoking, and alcohol consumption by race, across the adolescent and mid-adulthood assessments considered conjointly. White participants were less likely to smoke in mid-adulthood only (OR 0.4; 95%CI 0.2, 0.8) as compared to Black participants, but were not statistically significantly different than Black participants in the odds of smoking in adolescence only, or at both assessments, all relative to neither assessment. There were no statistically significant differences between Black and White participants in terms of patterns of heavy smoking. However, in regard to alcohol consumption, White participants had greater odds of drinking in adolescence only (OR 2.0; 95%CI 1.1, 3.6), in mid-adulthood only (OR 2.2; 95%CI 1.0, 4.5), and across both assessments (OR 4.9; 95%CI 2.7, 8.7), compared with neither assessment.

### Discussion

The present study examined racial differences in alcohol and tobacco consumption at adolescence and mid-adulthood. We summarize three central findings. First, White participants in the sample report higher odds of alcohol consumption in

both adolescence and mid-adulthood, but for binge drinking, increased odds emerged for Black participants in mid-adulthood. Whites were more likely to binge drink and become intoxicated in adolescence (though differences were muted when restricted to adolescent current drinkers), but Black participants were more likely to binge drink in mid-adulthood (but only when restricting to adult current drinkers). Second, there were no significant racial differences in cigarette use in adolescence, but in mid-adulthood, White participants were less likely to use cigarettes. However, among users, Whites were heavier smokers at both assessments (though during adolescence, the finding did not meet statistical significance). Finally, in assessing the adolescent and mid-adulthood assessments conjointly, White participants were more likely to drink across both assessments (and at each assessment alone), but less likely to report smoking in mid-adulthood without also smoking in adolescence. Taken together, these results suggest that racial patterns in substance use are dynamic over the life course, which may be a product of the accumulation of racially patterned social experiences manifesting to influence health behaviors.

Consistent with prior literature [19, 22, 53–55], White participants in the sample were more likely to drink and drink heavily in adolescence, and to be drinkers in mid-adulthood. However, Whites during the mid-adulthood



**Fig. 3** Unadjusted odds ratios of substance-use patterns. Odds of smoking, heavy smoking (defined as smoking  $\geq \frac{1}{2}$  pack a day), and alcohol consumption from adolescence to mid-adulthood by race (Black = ref.) in a sub-sample of the Child Health and Development Study ( $n = 431$ )

assessment were less likely to binge drink compared with Black participants. Previous research has demonstrated that White individuals in the US experience greater rates of alcohol consumption and more problematic drinking patterns from adolescence through early adulthood [19, 21, 55–57], but few studies have assessed the same individuals in mid-adulthood, when risks for chronic health problems increase [58–60]. These results suggest that more attention may need to be directed towards reducing binge drinking among Black drinkers in adulthood. However, absolute differences were small (2%), suggesting that the contribution of heavy drinking among Black individuals towards overall health disparities is likely insufficient to explain the burden of disease, as the rates of any drinking are considerably lower in this, and other studies [19, 22, 53].

While we found that Black participants were more likely to smoke in mid-adulthood, those who smoked were less likely to smoke  $\geq \frac{1}{2}$  a pack/day compared to their White counterparts. Though there is no “safe” level of cigarette smoking, the Black participants in this study were relatively light smokers, which would not predict as many chronic health conditions as those smoking more than  $\frac{1}{2}$  a pack/day [35, 36, 61–63]. Black participants were more likely to smoke to alleviate stress, perhaps signaling a coping strategy for the cumulative effects of marginalized social status, given that smoking is anxiolytic in the short term and at low levels [64–66]. The cumulative effects of chronic stress and

discrimination have been contextualized vis a vis “weathering” [67, 68], with evidence that chronic marginalization may lead to higher inflammation and other indicators of adverse cardiometabolic risks [69, 70]. Our results suggest that cigarette use later in life may be one pathway through which the “weathering” effect of chronic discrimination may be experienced. Given the reported increased use of smoking as a coping mechanism, future studies should investigate potential racial patterns in the relationship between motivation to use substances (i.e., coping with stress) and likelihood of heavy use.

Limitations of the study should be noted. This sample is drawn from a 1960s birth cohort in the Bay Area, and thus is not representative of White or Black adults in the US. All participants were members of the Kaiser Permanente health plan and are, on average, more educated and have higher incomes than the US population; these differences are especially pronounced for the Black participants. To address this potential limitation, we compared the prevalence of tobacco and alcohol consumption in this cohort to comparably aged White and Black participants in the National Health and Nutrition Examination Surveys (Tables S3, S4). Racial patterns of substance use from our sample are consistent with those in NHANES, suggesting that in regards to substance use, our results may be more broadly generalizable to the US population, despite the sample not being representative of the US population in other respects. In addition, there

is a considerable gap of time between the two assessments in which we do not have data on substance use. Therefore, inferences based on these results to patterns of use in the intervening developmental periods are not appropriate given the lack of information of substance-use patterns between the two assessments. Furthermore, substance use was assessed via self-report, using survey questions that varied in their wording between the two assessments, resulting in adolescent substance-use measures that are not entirely consistent with mid-adulthood substance-use measures; thus, said measures should be compared with caution. In addition, it is worth noting that ethnic and cultural variation within the racial group of non-Hispanic Black (e.g., Caribbean Black vs African–American) can result in differential substance-use patterns, though this was not assessed in this study. However, none of the individuals in our sample (and none of the mothers) were born in the Caribbean, limiting concerns of sub-group differences due to ethnic and or cultural differences. Nevertheless, the unique qualities of this sample include prospective measures of substance use and rigorous controls for potential confounding that have been measured in this cohort since birth, thus mitigating concerns about measurement error.

Our findings suggest that patterns of alcohol and cigarette use for White and Black individuals vary over the life course. Absent from our findings was any strong evidence in support of individual health behaviors such as alcohol and cigarette use being a major explanation for observed Black–White differences in chronic physical illness. The emergence of binge drinking among Black participants in mid-adulthood supports the role of cumulative disadvantage and discrimination as predicting differential substance-related coping strategies among Black individuals that also vary over the life course. However, absolute differences in binge drinking were small, suggesting that said differences do not drive Black–White disparities in cardiometabolic disorders. Cigarette smoking was more common among Black adults compared with White adults, but at relatively low levels, again suggesting that smoking is unlikely to drive disparities in cardiometabolic disorders.

Overall, our data are not consistent with the idea that differential substance use between White and Black Americans is a major driver of Black–White disparities in chronic physical illness. Additional longitudinal studies that allow for further insight into substance use throughout the life course will help to further assess the validity of said hypothesis. Furthermore, SES has the potential to impact race–substance-use relationships via effect modification. Assessing these potential interactions in future studies will be important for the overall framing of the effects of race on substance use. Future work should also investigate the role of gender within these relationships as choice of coping mechanisms tend to be patterned by gender [71–73].

Despite precedence in the literature focusing on substance use within the Black community, our findings suggest what others have suggested: true progress in eliminating health disparities will require greater attention on upstream, structural drivers of health, and less focus on downstream individual behaviors [74–76]. To date, strategies to address racial health disparities have produced mixed results [77–85], possibly due to lack of certainty regarding the major drivers of said disparities. With this in mind, future work investigating the causes of racial health disparities should also assess the role of structural determinants of stress and disease that are experienced differentially by Black and White individuals in the US.

**Acknowledgments** We would like to acknowledge Barbara Cohn and Katrina Kezios for their assistance. This work was supported by the National Institutes of Health R01HD058515 (Co-PIs: Link & Cohn), and K01AA021511 (PI: Keyes). John Pamplin is a fellow in T32-MH013043 (PI: Sussner).

### Compliance with ethical standards

**Conflict of interest** On behalf of all authors, the corresponding author states that there is no conflict of interest.

### References

1. Satcher D, Fryer GE, McCann J, Troutman A, Woolf SH, Rust G (2005) What if we were equal? A comparison of the black-white mortality gap in 1960 and 2000. *Health Aff* 24(2):459–464
2. Levine RS, Foster JE, Fullilove RE, Fullilove MT, Briggs NC, Hull PC, Husaini BA, Hennekens CH (2001) Black-white inequalities in mortality and life expectancy, 1933–1999: implications for healthy people 2010. *Public Health Rep* 116(5):474
3. Davis AM, Vinci LM, Okwuosa TM, Chase AR, Huang ES (2007) Cardiovascular health disparities a systematic review of health care interventions. *Med Care Res Rev* 64(5 suppl):29S–100S
4. Gold DR, Wright R (2005) Population disparities in asthma. *Annu Rev Public Health* 26:89–113
5. Peek ME, Cargill A, Huang ES (2007) Diabetes health disparities a systematic review of health care interventions. *Med Care Res Rev* 64(5 suppl):101S–156S
6. Jackson JS, Knight KM, Rafferty JA (2010) Race and unhealthy behaviors: chronic stress, the HPA axis, and physical and mental health disparities over the life course. *Am J Public Health* 100(5):933–939
7. Roux AVD (2012) Conceptual approaches to the study of health disparities. *Annu Rev Public Health* 33:41
8. Anderson NB, Bulatao RA, Cohen B, Race P, National Research Council (2004) Racial/ethnic disparities in health behaviors: a challenge to current assumptions. National Academies Press, Washington
9. Mezuk B, Rafferty JA, Kershaw KN, Hudson D, Abdou CM, Lee H, Eaton WW, Jackson JS (2010) Reconsidering the role of social disadvantage in physical and mental health: stressful life events, health behaviors, race, and depression. *Am J Epidemiol* 172(11):1238–1249
10. Jackson JS, Knight KM (2006) Race and self-regulatory health behaviors: the role of the stress response and the HPA axis



- in physical and mental health disparities. Social structures, aging, and self-regulation in the elderly. Springer, New York, pp 189–207
11. Samhsa C (2006) Results from the 2015 national survey on drug use and health: detailed tables. SAMHSA Office of Applied Studies, Rockville
  12. Wagner EF, Myers MG, McIninch JL (1999) Stress-coping and temptation-coping as predictors of adolescent substance use. *Addict Behav* 24(6):769–779
  13. Wills TA (1986) Stress and coping in early adolescence: relationships to substance use in urban school samples. *Health Psychol* 5(6):503
  14. Wills T (1985) Stress, coping, and tobacco and alcohol use in early adolescence. In: Shiffman S, Wills TA (eds) *Coping and Substance Use*. Academic Press, San Diego
  15. Keyes KM, Barnes DM, Bates LM (2011) Stress, coping, and depression: testing a new hypothesis in a prospectively studied general population sample of US-born Whites and Blacks. *Soc Sci Med* 72(5):650–659
  16. Bates LM, Barnes D, Keyes KM (2011) Re: “Reconsidering the role of social disadvantage in physical and mental health: stressful life events, health behaviors, race, and depression”. *Am J Epidemiol* 173:1348
  17. Ellickson PL, Orlando M, Tucker JS, Klein DJ (2004) From adolescence to young adulthood: racial/ethnic disparities in smoking. *Am J Public Health* 94(2):293–299
  18. Johnston LD, O’Malley PM, Bachman JG, Schulenberg JE (2006) *Monitoring the Future: National Survey Results on Drug Use, 1975–2005. Volume 1: Secondary School Students, 2005*. NIH Publication No. 06-5883. National Institute on Drug Abuse (NIDA)
  19. Keyes KM, Vo T, Wall MM, Caetano R, Suglia SF, Martins SS, Galea S, Hasin D (2015) Racial/ethnic differences in use of alcohol, tobacco, and marijuana: is there a cross-over from adolescence to adulthood? *Soc Sci Med* 124:132–141
  20. Breslau J, Aguilar-Gaxiola S, Kessler RC, Su M, Williams D, Kessler RC (2006) Specifying race-ethnic differences in risk for psychiatric disorder in a USA national sample. *Psychol Med* 36(01):57–68
  21. Hasin DS, Stinson FS, Ogburn E, Grant BF (2007) Prevalence, correlates, disability, and comorbidity of DSM-IV alcohol abuse and dependence in the United States: results from the National Epidemiologic Survey on Alcohol and Related Conditions. *Arch Gen Psychiatry* 64(7):830–842
  22. Pacek LR, Malcolm RJ, Martins SS (2012) Race/ethnicity differences between alcohol, marijuana, and co-occurring alcohol and marijuana use disorders and their association with public health and social problems using a national sample. *Am J Addict* 21(5):435–444
  23. Swendsen J, Burstein M, Case B, Conway KP, Dierker L, He J, Merikangas KR (2012) Use and abuse of alcohol and illicit drugs in US adolescents: results of the National Comorbidity Survey–Adolescent Supplement. *Arch Gen Psychiatr* 69(4):390–398
  24. Wu L-T, Woody GE, Yang C, Pan J-J, Blazer DG (2011) Racial/ethnic variations in substance-related disorders among adolescents in the United States. *Arch Gen Psychiatr* 68(11):1176–1185
  25. Banks DE, Zapolski TC (2018) The crossover effect: a review of racial/ethnic variations in risk for substance use and substance use disorder across development. *Curr Addict Rep* 5(3):386–395
  26. Harris KM, Gordon-Larsen P, Chantala K, Udry JR (2006) Longitudinal trends in race/ethnic disparities in leading health indicators from adolescence to young adulthood. *Arch Pediatr Adolesc Med* 160(1):74–81
  27. Pampel FC (2008) Racial convergence in cigarette use from adolescence to the mid-thirties. *J Health Soc Behav* 49(4):484
  28. Geronimus AT, Neidert LJ, Bound J (1993) Age patterns of smoking in US black and white women of childbearing age. *Am J Public Health* 83(9):1258–1264
  29. Trinidad DR, Gilpin EA, Lee L, Pierce JP (2004) Has there been a delay in the age of regular smoking onset among African Americans? *Ann Behav Med* 28(3):152–157
  30. Caetano R (1984) Ethnicity and drinking in northern California: a comparison among whites, blacks and Hispanics. *Alcohol Alcohol* 19(1):31–44
  31. Finlay AK, White HR, Mun E-Y, Cronley CC, Lee C (2012) Racial differences in trajectories of heavy drinking and regular marijuana use from ages 13 to 24 among African-American and White males. *Drug Alcohol Depend* 121(1):118–123
  32. DeWit DJ, Offord DR, Wong M (1997) Patterns of onset and cessation of drug use over the early part of the life course. *Health Educ Behav* 24(6):746–758
  33. Doyle JT, Dawber TR, Kannel WB, Kinch SH, Kahn HA (1964) The relationship of cigarette smoking to coronary heart disease: the second report of the combined experience of the Albany, NY, and Framingham, Mass. studies. *JAMA* 190(10):886–890
  34. Meade T, Imeson J, Stirling Y (1987) Effects of changes in smoking and other characteristics on clotting factors and the risk of ischaemic heart disease. *Lancet* 330(8566):986–988
  35. Howard G, Wagenknecht LE, Burke GL, Diez-Roux A, Evans GW, McGovern P, Nieto FJ, Tell GS (1998) Cigarette smoking and progression of atherosclerosis: the atherosclerosis risk in communities (ARIC) study. *JAMA* 279(2):119–124
  36. Wolf PA, D’Agostino RB, Kannel WB, Bonita R, Belanger AJ (1988) Cigarette smoking as a risk factor for stroke: the Framingham study. *JAMA* 259(7):1025–1029
  37. (SAMHSA) SAaMHSa (2015) Results from the 2015 National Survey on Drug Use and Health: detailed tables. Substance Abuse and Mental Health Services Administration, Rockville, MD. <https://www.samhsa.gov/data/sites/default/files/NSDUH-DetTabs-2015/NSDUH-DetTabs-2015/NSDUH-DetTabs-2015.htm#tab2-41b>. Accessed 17 Nov 2017
  38. Jamal A (2016) Current cigarette smoking among adults—United States, 2005–2015. *MMWR Morbidity and mortality weekly report*, p. 65
  39. Shield KD, Parry C, Rehm J (2014) Chronic diseases and conditions related to alcohol use. *Alcohol Res Curr Rev* 35(2):155
  40. Rehm J, Mathers C, Popova S, Thavorncharoensap M, Teerawattananon Y, Patra J (2009) Global burden of disease and injury and economic cost attributable to alcohol use and alcohol-use disorders. *Lancet* 373(9682):2223–2233
  41. Services USDoHaH (2014) The health consequences of smoking: 50 years of Progress. A report of the surgeon general. Atlanta, GA: US 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, vol 17
  42. van den Berg BJ, Christianson RE, Oechsli FW (1988) The California child health and development studies of the School of Public Health, University of California at Berkeley. *Paediatr Perinat Epidemiol* 2(3):265–282
  43. Van den Berg B, Mednick S, Harway M, Finello K (1984) The California child health and development studies. *Handb Longitud Res* 1:166–179
  44. Link BG, Susser ES, Factor-Litvak P, March D, Kezios KL, Lovasi GS, Rundle AG, Suglia SF, Fader KM, Andrews H (2016) Disparities in self-rated health across generations and through the life course. *Soc Sci Med* 174:17
  45. Drinking Levels Defined. <https://www.niaaa.nih.gov/alcohol-health/overview-alcohol-consumption/moderate-binge-drinking>. Accessed 5 May 2017

46. Keyes KM, March D, Link BG, Chilcoat HD, Susser E (2013) Do socio-economic gradients in smoking emerge differently across time by gender? Implications for the tobacco epidemic from a pregnancy cohort in California, USA. *Soc Sci Med* 76:101–106
47. Keyes KM, Keyes MA, March D, Susser E (2011) Levels of risk: maternal-, middle childhood-, and neighborhood-level predictors of adolescent disinhibitory behaviors from a longitudinal birth cohort in the United States. *Mental Health Subst Use* 4(1):22–37
48. Williams DR, Mohammed SA (2013) Racism and health I: pathways and scientific evidence. *Am Behav Sci* 57(8):1152–1173
49. VanderWeele TJ, Robinson WR (2014) On causal interpretation of race in regressions adjusting for confounding and mediating variables. *Epidemiology (Cambridge, Mass)* 25(4):473
50. Cooper R, David R (1986) The biological concept of race and its application to public health and epidemiology. *J Health Polit Policy Law* 11(1):97–116
51. Kaufman JS, Cooper RS, McGee DL (1997) Socioeconomic status and health in blacks and whites: the problem of residual confounding and the resiliency of race. *Epidemiology* 8(6):621
52. Rubin DB (2004) Multiple imputation for nonresponse in surveys. Wiley, New Jersey
53. Zapolski TC, Pedersen SL, McCarthy DM, Smith GT (2014) Less drinking, yet more problems: understanding African American drinking and related problems. *Psychol Bull* 140(1):188
54. Bachman JG, Wallace JM Jr, O'Malley PM, Johnston LD, Kurth CL, Neighbors HW (1991) Racial/ethnic differences in smoking, drinking, and illicit drug use among American high school seniors, 1976–89. *Am J Public Health* 81(3):372–377
55. O'Malley PM, Johnston LD (2002) Epidemiology of alcohol and other drug use among American college students. *J Stud Alcohol Suppl* 14:23–39
56. Lillie-Blanton M, MacKENZIE E, Anthony JC (1991) Black-white differences in alcohol use by women: baltimore survey findings. *Public Health Rep* 106(2):124
57. Caetano R, Kaskutas LA (1995) Changes in drinking patterns among whites, blacks and Hispanics, 1984–1992. *J Stud Alcohol* 56(5):558–565
58. Freid VM, Bernstein AB, Bush MA (2012) Multiple chronic conditions among adults aged 45 and over: trends over the past 10 years. *Women* 45:64
59. Alexander CM, Landsman PB, Teutsch SM, Haffner SM (2003) NCEP-defined metabolic syndrome, diabetes, and prevalence of coronary heart disease among NHANES III participants age 50 years and older. *Diabetes* 52(5):1210–1214
60. Ford ES, Giles WH, Dietz WH (2002) Prevalence of the metabolic syndrome among US adults: findings from the third National Health and Nutrition Examination Survey. *JAMA* 287(3):356–359
61. Thun MJ, Carter BD, Feskanich D, Freedman ND, Prentice R, Lopez AD, Hartge P, Gapstur SM (2013) 50-year trends in smoking-related mortality in the United States. *N Engl J Med* 368(4):351–364
62. Kenfield SA, Stampfer MJ, Rosner BA, Colditz GA (2008) Smoking and smoking cessation in relation to mortality in women. *JAMA* 299(17):2037–2047
63. Shah RS, Cole JW (2010) Smoking and stroke: the more you smoke the more you stroke. *Expert Rev Cardiovasc Ther* 8(7):917–932
64. Gilbert DG, Robinson JH, Chamberlin CL, Spielberger CD (1989) Effects of smoking/nicotine on anxiety, heart rate, and lateralization of EEG during a stressful movie. *Psychophysiology* 26(3):311–320
65. Morissette SB, Tull MT, Gulliver SB, Kamholz BW, Zimring RT (2007) Anxiety, anxiety disorders, tobacco use, and nicotine: a critical review of interrelationships. *Psychol Bull* 133(2):245
66. Zvolensky MJ, Feldner MT, Leen-Feldner EW, McLeish AC (2005) Smoking and panic attacks, panic disorder, and agoraphobia: a review of the empirical literature. *Clin Psychol Rev* 25(6):761–789
67. Geronimus AT (1991) The weathering hypothesis and the health of African-American women and infants: evidence and speculations. *Ethn Dis* 2(3):207–221
68. Geronimus AT, Hicken M, Keene D, Bound J (2006) “Weathering” and age patterns of allostatic load scores among blacks and whites in the United States. *Am J Public Health* 96(5):826–833
69. Slopen N, Lewis TT, Gruenewald TL, Mujahid MS, Ryff CD, Albert MA, Williams DR (2010) Early life adversity and inflammation in African Americans and whites in the midlife in the United States survey. *Psychosom Med* 72(7):694
70. Dong M, Giles WH, Felitti VJ, Dube SR, Williams JE, Chapman DP, Anda RF (2004) Insights into causal pathways for ischemic heart disease adverse childhood experiences study. *Circulation* 110(13):1761–1766
71. Tamres LK, Janicki D, Helgeson VS (2002) Sex differences in coping behavior: a meta-analytic review and an examination of relative coping. *Personal Soc Psychol Rev* 6(1):2–30
72. Frone MR, Cooper ML, Russell M (1994) Stressful life events, gender, and substance use: an application of tobit regression. *Psychol Addict Behav* 8(2):59
73. Lengua LJ, Stormshak EA (2000) Gender, gender roles, and personality: gender differences in the prediction of coping and psychological symptoms. *Sex Roles* 43(11–12):787–820
74. Link BG, Phelan J (1995) Social conditions as fundamental causes of disease. *Journal Health Soc Behav* 1:80–94
75. Williams DR, Priest N, Anderson NB (2016) Understanding associations among race, socioeconomic status, and health: patterns and prospects. *Health Psychol* 35(4):407
76. Thornton RL, Glover CM, Cené CW, Glik DC, Henderson JA, Williams DR (2016) Evaluating strategies for reducing health disparities by addressing the social determinants of health. *Health Aff* 35(8):1416–1423
77. Hunt B, Whitman S (2015) Black–white health disparities in the United States and Chicago: 1990–2010. *J Racial Ethn Health Dispar* 2(1):93–100
78. Orsi JM, Margellos-Anast H, Whitman S (2010) Black–white health disparities in the United States and Chicago: a 15-year progress analysis. *Am J Public Health* 100(2):349–356
79. Phelan JC, Link BG (2005) Controlling disease and creating disparities: a fundamental cause perspective. *J Gerontol Ser B Psychol Sci Social Sci* 60(2):S27–S33
80. Hunt BR, Whitman S, Hurlbert MS (2014) Increasing black: white disparities in breast cancer mortality in the 50 largest cities in the United States. *Cancer Epidemiol* 38(2):118–123
81. Rosenstock S, Whitman S, West JF, Balkin M (2014) Racial disparities in diabetes mortality in the 50 most populous US cities. *J Urban Health* 91(5):873–885
82. Tehranifar P, Goyal A, Phelan JC, Link BG, Liao Y, Fan X, Desai M, Terry MB (2016) Age at cancer diagnosis, amenability to medical interventions, and racial/ethnic disparities in cancer mortality. *Cancer Causes Control* 27(4):553–560
83. Allgood KL, Hunt B, Rucker MG (2016) Black: white disparities in HIV mortality in the United States: 1990–2009. *J Racial Ethn Health Dispar* 3(1):168–175
84. Kelly SP, Rosenberg PS, Anderson WF, Andreotti G, Younes N, Cleary SD, Cook MB (2016) Trends in the incidence of fatal prostate cancer in the United States by race. *Eur Urol* 71:195
85. DeSantis CE, Fedewa SA, Goding Sauer A, Kramer JL, Smith RA, Jemal A (2016) Breast cancer statistics, 2015: convergence of incidence rates between black and white women. *CA Cancer J Clin* 66(1):31–42