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Blacks' Diminished Health Return of Family Structure and Socioeconomic Status; 15 Years of Follow-up of a National Urban Sample of Youth

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Abstract The protective effect of family structure and socioeconomic status (SES) on physical and mental health is well established. There are reports, however, documenting a smaller return of SES among Blacks compared to Whites, also known as Blacks' diminished return. Using a national sample, this study investigated race by gender differences in the effects of family structure and family SES on subsequent body mass index (BMI) over a 15-year period. This 15-year longitudinal study used data from the Fragile Families and Child Wellbeing Study (FFCWS), in-home survey. This study followed 1781 youth from birth to age 15. The sample was composed of White males (n = 241, 13.5%), White females (n = 224, 12.6%), Black males (n = 667, 37.5%), and Black females (n = 649, 36.4%). Family structure and family SES (maternal education and income to need ratio) at birth were the independent variables. BMI at age 15 was the outcome. Race and gender were the moderators. Linear regression models were run in the pooled sample, in addition to race by gender groups. In the pooled sample, married parents, more maternal education, and income to need ratio were all protective against high BMI of youth at 15 years of age. Race interacted with family structure, maternal education, and income to need ratio on BMI, indicating smaller effects for Blacks compared to Whites. Gender did not interact with SES indicators on BMI. Race by gender stratified regressions showed the most consistent associations between family SES and future BMI for White females followed by White males. Family structure, maternal education, and income to need ratio were not associated with lower BMI in Black males or females. The health gain received from family economic resources over time is smaller for male and female Black youth than for male and female White youth. Equalizing access to economic resources may not be enough to eliminate health disparities in obesity. Policies should

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address qualitative differences in the lives of Whites and Blacks which result in diminished health returns with similar SES resources. Policies should address structural and societal barriers that hold Blacks against translation of their SES resources to health outcomes.

Keywords Ethnic groups · Blacks · Ethnicity · Obesity · Body mass index · Socioeconomic status · Education · Income

Background

Empirical data have shown that the health effect of socioeconomic status (SES) indicators such as education is "enduring, consistent, and growing" [1]. Long-term longitudinal studies have also documented the protective effects of high SES (e.g., education and income) against a wide range of health outcomes [2–6]. The literature has shown that distribution of health tends to follow a social gradient (based on availability of SES resources) [7–9]. Non-marital family structure, in addition to financial strain and low SES, is considered major reasons why children who grow up in poverty and particularly Black children are at higher risk for undesired outcomes [10].

While most of the mainstream literature has assumed that the health gain associated with SES is constant across groups [11–13], there are considerable variations in the degree to which SES protects populations from undesired health outcomes [14–16] including obesity [14, 17]. This is in part because populations differ in how they can translate SES indicators to health outcomes [14, 18, 19]. While one of the main mechanisms by which SES affects health is through its effects on individuals' ability to avoid risks [11–13], SES indicators do not similarly reduce exposures across all groups [20–22]. Similar increases in SES may also have differential effects on purchasing power of various social groups [23–26]. The effect of SES on access to resources and improving health behaviors [11–13, 27] also depends on race [28]. When compared to Blacks, high SES has a larger effect on access to material resources and assets [29] and human connections and social network [30] for Whites. Upward social mobility—that is expected to generate health gain [18, 21, 22, 31–34]—may be associated with burn out as well as social, psychological, and physiological costs for Black families and youth [23, 35]. Due to the lower availability of educational resources in predominantly Black communities, education may have a smaller effect on behaviors and human capital for Blacks compared to Whites [28, 36]. As a result, the effect of SES indicators may differ across populations and health outcomes [14, 37–39]. That is, health gains associated with high SES may be specific to demographic subpopulations.

Even though SES promotes health overall [36, 39–42], due to the differential opportunity structure, the health gain associated with high SES depends on a wide range of factors such as race, ethnicity, gender, and place [39, 40, 43–49]. The mechanism by which each SES indicator influences health may also differ across populations [40, 41, 50]. The ability of populations to gain from a particular SES resource depends on the context and life circumstances that are influenced by demographic factors such as race and gender [39, 44, 51–53].

Assuming that the health effects of SES are universal, most research on the link between SES and health has not explored the potential race and gender differences in how SES indicators impact health [54]. However, a recent body of research assumes that the health effects of SES depend on the availability of other resources, which in turn, vary across demographic groups [55], and the way SES increases the purchasing power and living conditions of each subpopulation [28, 56, 57].

These results document a major Black-White gap in the health effects of SES [42, 56, 58, 59]. For example, education is differentially associated with drinking patterns [41], depressive symptoms [60], suicidality [57], chronic disease [60], and mortality [42, 56, 58, 59] for Whites and Blacks. Fuller-Rowell has shown that high SES may be associated with more social costs for Black youth compared to their White counterparts [21], a phenomenon which may explain diminished health gains associated with upward social mobility among Black families [22]. Assari and Caldwell have shown a higher risk, as compared to which other populations of Major Depressive Disorder (MDD) among male Black youth in the presence of high SES [61]. Yet, there is more to be learned about Black families and Black youth.

To obtain a more nuanced understanding of heterogeneity in the association between SES and health, this study used a national sample of urban families to examine race and gender variation in the effect of family structure and family SES (i.e., maternal education and income to need ratio) at birth on body mass index (BMI) at age 15. As family structure may be one reason behind

diminished return of family SES for Blacks, we ran models with and without family structure as a control variable. A growing body of knowledge suggests psychosocial determinants of obesity depend on race [62–64], gender [65, 66], and their intersection [14, 17]. Similar findings have been reported for youth [65–68] as well as adults [62, 69–71]. Given the extensive literature on Blacks' diminished return, we expected a smaller protective effect of family SES against youth obesity in Black families. The focus of this study was on BMI for several reasons. First obesity is epidemic in USA, and youth are not exempted from this issue [51, 52, 54]. Second, obesity is a major contributor to racial health disparities [72–74]. Blacks in general, and Black women in particular, have the highest prevalence of obesity [75, 76]. Compared to non-Hispanic Whites, Blacks are 50% more likely to be obese, while, the additional risk is 80% for Black women. This figure suggests that four out of five Black women are overweight or obese. Third, because obesity is an intermediate cause of morbidity and mortality as it increases risk of cancer, diabetes, hypertension, stroke, heart disease, and among others [77].

Methods

Design and Setting

Data came from the first and sixth waves of the Fragile Families and Child Wellbeing Study (FFCWS). FFCWS is an ongoing large population-based cohort, started in 1998. This national study randomly sampled families from 20 US cities with population of 200,000 or more. More information on sampling and the interview protocol of the FFCWS is available elsewhere [78]. The Fragile Families and Child Wellbeing Study has followed a cohort of new parents and their children and provides previously unavailable information about the conditions and capabilities of new unwed parents and the well-being of their children. The study has collected data on approximately 4700 births (3600 non-marital and 1100 marital) in 75 hospitals in 20 cities across the USA [79–81].

Ethics

The FFCWS protocol was approved by the Princeton University Institutional Review Board. All adolescents' legal guardian provided informed consent. Assent was obtained from adolescents. Respondents received financial compensation for their participation.

FFCWS includes 4655 families (2407 Black, 1354 Hispanics, and 894 White). As the FFCWS has oversampled non-married couples [71, 78], it is not representative of the US population. As a result, most of the participants were in non-marital unions and had lower socioeconomic status. Data for the current analysis used baseline (wave 1) and then 15 years later (wave 6). Independent variables were family SES, measured at wave 1 (baseline). Dependent variable was youth BMI, measured at wave 6 when the youth was at age 15. The analytical sample for this study consisted of 1781 youth who were followed from birth to age 15. This number was composed of White males (n = 241, 13.5%), White females (n = 224, 12.6%), Black males (n = 667, 37.5%), and Black females (n = 649, 36.4%).

Measures

Main Independent Variables

Family structure and family SES (maternal education and income to needs ratio) at birth were the main independent variables, all measured at baseline interview (wave 1). Family structure was a dichotomous variable based on marital status of the youth's father and mother. Maternal education was measured as an ordinal variable: (1) less than high school, (2) high school, (3) some college, and (4) college completed or graduate level. Finally, income to needs ratio, an indicator of poverty status, was calculated based on household income divided by household size. Higher income to need ratio was indicative of less poverty [82–84].

Dependent Variable

Body mass index (BMI) was measured using participants' self-reported height (measured in feet and inches) and weight (measured in pounds). Height and weight were converted to meters and kilograms, respectively. Then BMI was calculated by dividing weight (kilograms) by squared height (meters). BMI based on self-report strongly correlates with BMI based on measurements [85, 86].

Missing Data

Attrition was almost exclusively due to selective attrition of participating families in the study after 15 years of follow-up. From the total number of 2923 Black and White families that were recruited in the study, only 1781 Black and White families had data at age 15. Attrition was correlated with maternal education but not family race, family structure, and income to need ratio. As a result, Black and White and poor and non-poor and married and non-married families had the same chance of being included in the current study. Although this study used data from individuals who were under follow-up for 15 years, the sample that is retained in the study is not biased based on race, family structure, or poverty status.

Statistical Analysis

We analyzed data using SPSS 22.0 (IBM Corporation, Armonk, NY, USA). Frequency and mean (SD) was reported for descriptive purposes of all variables. For bivariate analysis, we used the Pearson correlation test in the pooled sample and in race by gender groups. We ran multiple regression models, first in the pooled sample then in race by gender sub-groups. In the pooled sample, to test the separate effects of each SES indicator, we ran models that only included one main effect. Then we ran three additional models that included the following interaction terms: (1) race × SES indicator and (2) gender × SES indicator. In all models, BMI at age 15 was the dependent variable, and a family SES indicator or family structure was the independent variable. As family structure could potentially confound the effect of family SES on health, and as family structure may be one reason behind diminished return of family SES for Blacks, we ran models with (additive effects) and without (separate effects) family structure as a control variable. Adjusted unstandardized *b* (regression coefficients) and their 95% confidence interval (CI) were reported. *P* values less than 0.05 were considered statistically significant.

Results

This study followed 1781 youth from birth to age 15. The sample was composed of White males (n = 241, 13.5%), White females (n = 224, 12.6%), Black males (n = 667, 37.5%), and Black females (n = 649, 36.4%).

Table 1 describes study variables at baseline and at age 15 in the pooled sample, as well as race by gender groups. While most White youth were from families with married parents, most Black youth had unmarried parents. Maternal education was higher for White youth compared to Black youth. Income to need ratio was higher for White compared to Black youth. BMI at age 15 was highest for Black females followed by Black males. (Table 1).

Table 2 summarizes the results of bivariate correlations in the pooled sample and based on race by gender intersections. While all SES indicators were associated with BMI at age 15 in the pooled sample, these associations could only be found for White males and females (Table 2).

Table 3 summarizes the results of eight linear regressions in the pooled sample. Regression models were estimated in the absence and presence of family structure as a covariate. In models that did not include any interaction term, married parents, high maternal education and income to need ratios at birth were all protective against high BMI of youth at age 15. Models that included interaction terms showed that race interacts with all family SES (maternal education and income to need ratio) as well as family structure on BMI, with smaller effects for Blacks compared to Whites. Gender did not interact with any of the SES indicators on BMI. Interactions between race and family SES did not change in the models with (additive effects) and without (separate effects) family structure as a control variable, thus the diminished gain of family SES in Blacks is not due to different family structure of Whites and Blacks (Table 3).

Table 4 summarizes the results of stratified linear regression models in race by gender groups. Regression models were estimated in the absence and presence of family structure as a covariate. These models showed most consistent protective effects of family SES indicators at baseline on future BMI for White females followed by White males. Having married parents, high maternal education and high income to need ratio at baseline were not associated with lower BMI in Black males or Black females. As the diminished gain results were observed in models with (additive effects) and without (separate effects) family structure as a control variable, we argue that diminished gain of family SES in Blacks is not due to racial differences in family structure (Table 4).

Table 1 Descriptive statistics in the pooled sample and race by gender groups

	All (n = 1781)		White males (n = 241)		White females (n = 224)		Black males (n = 667)		Black females (n = 649)	
	n	%	n	%	n	%	n	%	n	%
Gender										
Male	908	50.98	–	–	–	–	–	–	–	–
Female	873	49.02	–	–	–	–	–	–	–	–
Race										
White	465	26.11	–	–	–	–	–	–	–	–
Black	1316	73.89	–	–	–	–	–	–	–	–
Married*										
No	1337	75.07	88	36.51	90	40.18	595	89.21	564	86.90
Yes	444	24.93	153	63.49	134	59.82	72	10.79	85	13.10
Education*										
Less than high school	492	27.64	28	11.62	30	13.39	205	30.78	229	35.29
High school	575	32.30	48	19.92	47	20.98	260	39.04	220	33.90
Some college	464	26.07	68	28.22	62	27.68	173	25.98	161	24.81
College completed or graduate level	249	13.99	97	40.25	85	37.95	28	4.20	39	6.01
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Maternal education*	2.26	1.014	2.97	1.03	2.90	1.06	2.04	0.86	2.02	0.92
Income to need ratio*	3.15	1.43	4.36	1.02	4.19	1.14	2.67	1.28	2.83	1.37
Body mass index*	23.87	5.80	22.17	4.48	22.28	4.67	23.86	5.97	25.06	6.12

*$p < 0.05$

Discussion

Our study revealed racial differences in the effect of family structure and family SES at birth on BMI at age 15, at least in the case of family structure and income to need ratio. Black youth did not show a reduction in their BMI due to SES and family structure at birth. This pattern was different from White youth, for whom all the very same SES indicators at birth were protective against high BMI 15 years later. Although we found the same trend for maternal education, the interaction was only marginally significant, indicating that differential effects of maternal education is only suggestive. These patterns were independent of controlling for family structure, as family SES continued to show smaller health gain for Blacks, even after family structure was controlled. The current paper extends the literature as most previous research is on adult and the elderly samples, and less was known about the exact developmental phase where diminished gain can be observed. In addition, less was known about diminished returns of family SES on offspring health decades later.

Our finding on the protective effect of family SES against high BMI for White youth is in line with the literature on protective effects of high SES against high BMI [87, 88]. According to the life course epidemiological approach, low SES at birth has negative long-term health consequences decades later [89, 90]. Protective effects of SES are not limited to BMI [87, 88, 91], also extending to several mental and physical health outcomes [2–6, 92]. In the Health and Retirement Study (HRS), income had a protective effect against sustained high BMI among White and Black women, but not for White and Black men. Education had a protective effect against BMI, insomnia, and physical inactivity in White men, White women, and Black women but not Black men [14]. In Americans' Changing Lives (ACL) study, education had a smaller effect on life expectancy for Blacks than for Whites [56]. Education has also shown a smaller effect on drinking of Blacks than Whites [28]. Our findings and the mentioned literature are in line with the Blacks' diminished return hypothesis, suggesting that health gain from high SES is systematically smaller for Blacks than for Whites [14, 56, 57, 65].

Table 2 Correlation matrix in the pooled sample and across race by gender groups

	1	2	3	4	5	6
Pooled sample						
1 Gender (females)	1	0.01	0.01	− 0.02	0.02	0.08**
2 Race (Blacks)		1	− 0.51**	− 0.39**	− 0.47**	0.17**
3 Married parents			1	0.50**	0.50**	− 0.14**
4 Maternal education				1	0.57**	− 0.11**
5 Income to need ratio					1	− 0.13**
6 Body mass index (BMI)						1
White males						
3 Married parents			1	0.57**	0.51**	− 0.12
4 Maternal education				1	0.51**	− 0.09
5 Income to need ratio					1	− 0.23**
6 Body mass index (BMI)						1
White females						
3 Married parents			1	0.51**	0.57**	− 0.27**
4 Maternal education				1	0.56**	− 0.22**
5 Income to need ratio					1	− 0.16*
6 Body mass index (BMI)						1
Black males						
3 Married parents			1	0.26**	0.27**	− 0.04
4 Maternal education				1	0.44**	− 0.00
5 Income to need ratio					1	− 0.00
6 Body mass index (BMI)						1
Black females						
3 Married parents			1	0.31**	0.29**	0.01
4 Maternal education				1	0.48**	− 0.03
5 Income to need ratio					1	− 0.06
6 Body mass index (BMI)						1

*$p < 0.05$, **$p < 0.01$

Our findings do not support previous theoretical and empirical work, at least for obesity as the outcome, that unmarried family structure and low family SES may at least in part explain some of increased risk of subsequent undesired health outcomes for Black youth. A number of theories suggest that low-family SES is a root cause of undesired health of offspring. Link and Phelan's (1995) fundamental cause theory attributes poor health outcomes to low SES [11–13]. Bronfenbrenner's ecological model of human development argues that family SES is a major aspect of social context of child development [93]. The family system model stresses the role of family structure and SES on child outcomes [94, 95]. According to McLoyd and colleagues, low SES and high financial stress of the family are the main mechanisms that explain poor developmental outcomes among children in Black families [96–98]. In our study, however, family structure and SES failed to protect Black youth against obesity, an effect which was present for White families.

Race [56], gender [8, 59, 99], and their intersection [31, 35, 36] alter health gains from SES [28, 100–105]. Most of these studies have documented the least health gain from their SES resources for Blacks [106]. In addition to a diminished return, there are even reports that have documented an additional risk associated with high SES among Blacks,

Table 3 Summary of linear regression models with and without interaction terms in the pooled sample (with and without family structure as a control variable)

	Separate effects				Additive effects			
	Main effect models		Interaction models		Main effect models		Interaction models	
	B	95%CI	*B*	95%CI				
Maternal education model								
Married					− 0.79*	− 1.56–− 0.03	− 0.69*	− 1.47–0.09
Maternal education	− 0.30*	− 0.58–− 0.01	− 0.63*	− 1.13–− 0.12	− 0.18	− 0.49–0.13	− 0.27	− 0.86–0.32
Female	0.91***	0.38–1.44	1.17*	0.28–2.07	0.92***	0.39–1.45	1.96**	0.67–3.26
Race (Black family)	1.95***	1.29–2.61	0.41	− 1.33–2.15	1.67***	0.95–2.38	0.48	− 1.24–2.21
Gender × Maternal education	–	–	− 0.38	− 1.40–0.64	–	–	− 0.46*	− 0.99–0.00
Race × maternal education	–	–	0.59#	− 0.02–1.21	–	–	0.47	− 0.15–1.09
Intercept	22.66***	21.64–23.68	23.65***	22.06–25.25	22.79***	21.76–23.82	23.14***	21.47–24.82
Income to need ratio model								
Married					− 0.75*	− 1.50–0.00	− 0.54	− 1.32–0.24
Income to need ratio	− 0.26*	− 0.46–− 0.05	− 0.66*	− 1.17–− 0.14	− 0.18	− 0.40–0.04	− 0.53*	− 1.07–0.00
Female	0.94***	0.41–1.47	1.79**	0.51–3.06	0.94***	0.41–1.47	1.78**	0.51–3.06
Race (Black family)	1.83***	1.14–2.51	− 0.83	− 3.06–1.41	1.57***	0.84–2.30	− 0.63	− 2.88–1.62
Gender × poverty index	–	–	− 0.28	− 0.65–0.09	–	–	− 0.28	− 0.65–0.09
Race × poverty index	–	–	0.67*	0.14–1.21	–	–	0.58*	0.03–1.13
Intercept	22.86***	21.80–23.92	24.74***	22.51–26.96	23.00***	21.93–24.06	24.51***	22.26–26.76

Outcome: *BMI* body mass inde, *CI* confidence interval

#$p < 0.1$, *$p < 0.05$, **$p < 0.01$, ***$p < 0.001$

Table 4 Summary of linear regression models based on the intersection of ethnicity and gender (with and without family structure as a control variable)

	White males (n = 241)		White females (n = 224)		Black males (n = 667)		Black females (n = 649)	
	B	95%CI	*B*	95%CI	*B*	95%CI	*B*	95%CI
Separate effects								
Maternal education model								
Maternal education	− 0.38	− 0.93–0.17	− 0.99***	− 1.56–− 0.42	− 0.02	− 0.55–0.51	− 0.20	− 0.71–0.32
Intercept	23.30***	21.57–25.03	25.15***	23.40–26.91	23.91***	22.74–25.08	25.46***	24.32–26.60
Income to need ratio model								
Income to need ratio	− 1.04***	− 1.58–− 0.49	− 0.65*	− 1.19–− 0.12	0.00	− 0.36–− 0.35	− 0.25	− 0.60–0.09
Intercept	26.69***	24.25–29.14	25.02***	22.70–27.34	23.87***	22.81–24.92	25.78***	24.69–26.86
Marital status model								
Married	− 1.09#	− 2.26–0.09	− 2.55***	− 3.76–1.34	− 0.83	− 2.30–0.63	0.11	− 1.29–1.51
Intercept	22.86***	21.92–23.79	23.80***	22.87–24.74	23.95***	23.46–24.43	25.05***	24.54–25.55
Additive effects								
Maternal education model								
Married	− 0.92	− 2.36–0.51	− 1.97**	− 3.38–− 0.57	− 0.88	− 2.40–0.63	0.31	− 1.17–1.78
Maternal education	− 0.14	− 0.80–0.53	− 0.52	− 1.17–0.13	0.06	− 0.49–0.61	− 0.23	−0.78–0.31
Intercept	23.15***	21.41–24.89	24.98***	23.24–26.71	23.84***	22.66–25.02	25.49***	24.34–26.64
Income to need ratio model								
Married	0.03	− 1.31–1.37	− 2.49	− 3.96–− 1.01	− 0.89	− 2.41–0.63	0.45	− 1.02–1.91
Income to need ratio	− 1.05	− 1.68–− 0.41	− 0.04	− 0.68–0.59	0.05	− 0.31–0.42	− 0.29	− 0.65–0.08
Intercept	26.70	24.19–29.22	23.95	21.60–26.31	23.81	22.75–24.87	25.81	24.72–26.90

Outcome: *BMI* body mass index, *CI* confidence interval

#$p < 0.1$, *$p < 0.05$, **$p < 0.01$, ***$p < 0.001$

particularly Black men [57, 60]. High SES is a risk factor for an increase in depressive symptoms over time among Black men [60]. High income is also positively associated with risk of MDD for Black boys [61].

According to a CDC report [91], the effects of SES indicators on obesity vary based on race, ethnicity, and gender. Among White men, income was protective against obesity for non-Hispanic Black and Mexican American but not White men. Among women, income was protective against obesity. There was no effect of education against obesity among men; however, for women, those with a college degree were at a lower risk of obesity compared to those without such education credentials [91].

Due to residential segregation [24, 107], high SES may be a proxy of living far from Black communities among Black families. High SES Blacks may be more distant from the rest of the Black community. In the absence of close contact with other Blacks, high SES Black families may lose social support, one of major determinants of their health and well-being [108]. Social isolation may result in vulnerability of high-SES Blacks as they experience a decline in social support from other Blacks [109, 110]. Low-social support is particularly detrimental to the health of Blacks who gain more health from social support than Whites [83, 84, 111]. Social support is also shown to be a major determinant of obesity for Black families [66, 68]. Low social support can be even more detrimental when racial discrimination is high [25, 26].

Research should test if social isolation from the rest of the Black community explains low health gain associated with high SES among Black families. Homophily of social networks is an important determinant of availability of social support, and Blacks are not an exception to this rule [112]. For high-SES Black families, social networks may be composed of Whites, which may also increase their exposure to discrimination [113–115]. This explanation is in line with the studies on positive association between SES and discrimination among Blacks [48, 115, 116]. Discrimination may confine the health gains that are expected to follow high SES in Black families [18, 48, 115, 116]. Hudson's finding on smaller effect of SES on health in the presence of discrimination supports the above argument [18].

Upward social mobility is associated with social, psychological, and physiological costs for Blacks [117] who have a higher tendency than Whites to apply effortful behavioral coping mechanisms for upward social mobility [118]. James and colleagues have introduced the concept of John Henryism, a coping mechanism associated with higher aspirations but worse health outcomes in Blacks [115]. Research has shown how goal striving stress—which is closely linked to upward social mobility—causes distress for high-achieving Blacks [119–122]. Other researchers have also shown the high-psychological costs of upward social mobility for Blacks [19, 21, 22, 123].

Culture may also have a role in explaining the differential effect of SES on childhood obesity. Race may alter how a stressed mother feeds her son or daughter as an infant, as a toddler, as a child, and as an adolescent. As a result, early social determinants and family SES are expected to have group-specific rather than universal effects on the future risk of obesity of youth [124, 125]. Different and more tolerant and liberal thresholds for defining obesity, and over-eating under stress, might explain why racial groups differ in the effect of SES on obesity [70]. The contribution of eating and nutritional habits as a mechanism behind differential gains of SES on youth BMI should also be explored in further research.

Another risk to the health of Black youth is when they become aware of the structural barriers that hold them back. Such awareness may reduce their future orientation, hope, self-efficacy, and control over life among Black youth [126, 127]. One structural barrier in the life of Blacks is labor market preferences (preferences of employers) that reduce odds of employment of Black youth [128–132] and their wage if hired [133–139]. Male Black youth have the lowest chance for employment and receive the lowest compensation if hired [129, 130]; racial income inequalities may also be larger for the highest SES groups [140–143]. This is in addition to considerable discrimination on a daily basis [25, 26]. Dangerous neighborhoods which are limited in resources may further increase undesired health outcomes [144–148]. Fear of violence in the neighborhood increases the obesity risk of Black females [144]. These conditions cumulatively reduce the health of Blacks, regardless of their SES.

In addition to structural factors such as racial residential segregation, social norms around eating and their intersections with social support may have a role in smaller health gain of high SES on obesity among Black families. It is plausible that upward social mobility does not erase their socialization with the rest of their lower SES family members and relatives and their connections

with family and friends through food. Food is a major part of the Black church, and food options may not be very healthy. In addition, family relations are extended and go beyond nuclear family for Blacks, which may adversely influence eating habits of high-SES Black households. This finding is supported by recent findings on the diminished effects of education on diet [149] and drinking [28] of Blacks compared to Whites. Physical activity, another behavior that impacts obesity risk, also varies by the intersection of race, gender, and SES. There is a need for future research on the role of health behaviors such as diet and physical activity on the diminished gain from SES against obesity in Black families.

Our findings do not blame the victim by suggesting that Blacks are unable to turn their available SES resources to health outcomes. We believe that societal and structural barriers operate as major barriers against Black families' ability to translate their SES resources to health gain. In contrast, we argue that it may be the system which fails Black families who climb the social hierarchy as they should pay extra costs for their upward social mobility. We believe that American society operates in a way that maximizes the gain of Whites and minimizes those of Blacks. Black families with high SES face more thwarted opportunities than their White counterparts. While high SES increases expectations and aspirations of Black families, they often face systematic barriers that increase stress, such as discrimination, and reduce the expected health gain associated with high SES [60]. We argue that in a race-and-color-aware society, in the presence of discrimination and systematically thwarted opportunities, high aspirations may be detrimental to the health of Blacks. Some research has also shown that high SES may be a vulnerability factor for Black families [18, 61]. Hudson and others have shown that discrimination is most costly in the presence of high SES [18], a finding which has been replicated for Black youth [61].

Our findings have implications for future research as well as public policy. Additional research should explore whether racial socialization, racial identity, discrimination, social support, culture, social norms, environmental context (neighborhood and school racial composition), and expectations explain group differences in the health gains associated with high SES. Research should also seek higher level structural factors that increase the vulnerability of high-SES Blacks to undesired outcomes.

Conceptually, race and gender operate more than as merely control variables [150], as they alter social determinants of obesity [14, 65–67, 69, 151] and other health outcomes [54, 100]. As a result, researchers may conceptualize race, ethnicity, and gender as contextual factors that alter not only exposures, but also vulnerability to risk and protective factors (i.e., effect modifiers) [28].

Our findings should be interpreted in the light of a few study limitations. We focused on the race and gender differences, and other potential moderators such as ethnicity and location were not considered. The SES indicators studied were not a comprehensive list, and other factors such as household size, employment, and wealth were not considered. We only studied baseline SES indicators and did not conceptualize SES as time-varying covariates. All our study constructs were measured at the individual level, and the effect of contextual factors was not investigated. Finally, we did not study some of the mechanisms or confounders such as eating habits. There is a need for replication of these findings using a longitudinal design [82, 152–154]. As this study was only limited to Whites and Blacks, there is a need for future research to replicate these findings across other marginalized groups such as Native Americans and American Indians, Hispanics, Arab Americans, and immigrants. Despite these limitations, this is one of the very few studies using a national sample to explore the heterogeneities in the association between SES and BMI over 15 years.

To conclude, our findings showed racial differences in the effects of family SES at birth on youth BMI at age 15. Racial groups do not similarly benefit from SES resources, with a lower gain for Black youth compare to White youth, possibly due to societal barriers and life conditions. Future research should study particular barriers that hinder Blacks from translating their SES resources to health outcomes. The more important remaining question is what policies can eliminate or at least reduce racial differences in the health gain of SES indicator? There is also a need for consideration of these heterogeneities in public health program planning and delivery.

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Authors Contribution Ronald B. Mincy was the co-principle investigator of the FFCWS. So, he contributed to the design and data collection of the original FFCWS. Shervin Assari designed the current work, analyzed the data, and drafted the manuscript.

Cleopatra Caldwell, Alvin Thomas, and Ronald B. Mincy all contributed to interpretation of the findings and drafting and revision of this paper.

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Compliance with Ethical Standards

Ethical Standard All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. Parental informed consent and assent were obtained from all adolescent participants included in the study.

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