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Racial Differences in Mechanisms Linking Childhood SES with Growth in Adult BMI: The Role of Adolescent Risk and Educational Attainment

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Abstract

Purpose: The present study examined whether risk factors during adolescence, including substance use, depression, overweight status, and young adult educational attainment, mediated the association between low childhood socioeconomic status (SES) and higher body mass index (BMI) in adulthood. We also evaluated whether the hypothesized pathways differed based on racial group status.

Methods: Participants from the Seattle Social Development Project were followed from ages 10–39 years. The present study included White (n=381), African American (n=207), and Asian American (n=171) participants. Structural equation models tested pathways linking low childhood SES to BMI from ages 24–39. Multiple group modeling was used to test potential racial differences.

Results: Analyses indicated racial differences in the pathways linking low childhood SES with adult BMI. For Whites, overweight status and educational attainment were significant mediators. For Asians, there was an unmediated and significant pathway between low childhood SES and low adult BMI. For African Americans, there were no significant mediated or unmediated pathways.

Conclusions: Results stress that the pathways that link childhood SES with adult BMI may operate differently based on race. Research is particularly needed to identify mechanisms for African Americans in order to better inform obesity prevention efforts.

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Keywords

Obesity; Body Mass Index; Race; Adolescent Substance Use; Young Adult Educational Attainment; Childhood Socioeconomic Status; Seattle Social Development Project; Structural Equation Model; Longitudinal

In the United States, obesity is a public health concern. Although there is evidence that the prevalence of obesity has remained relatively stable in 2003-2004 and 2013-2014, the prevalence remains high with 39.8% of adults being obese [1]. Co-morbid conditions associated with obesity include cardiovascular conditions, hypertension, type 2 diabetes, and major depression [2]. Effective treatments designed to reduce adult obesity are limited, despite our advancing knowledge of nutritional and physical activity factors which contribute to the development of obesity [3]. The lack of progress in addressing adult obesity may in part be due to prevention efforts being primarily focused during adulthood rather than from a life course perspective. A life course framework to chronic disease prevention suggests that exposure to factors beginning in early life and beyond determines adult obesity risk [4]. Ample research shows adult obesity has its genesis in childhood [5]. For example, nearly half of overweight children become overweight adults [6] and 90% of obese adolescents remain obese in adulthood [7]. Given the developmental nature of adult obesity, identification of prevention targets in childhood may lead to the development of effective preventive interventions [3].

Studies suggest that an early life risk factor for adult obesity is low childhood socioeconomic status (SES) [8-9]. Early economic disadvantage may affect adult obesity as a function of social gradients in the distribution of resources [8-9]. However, questions remain as to early life risk factors that influence the association between low childhood SES and adult obesity. Guided by the Social Development Model (SDM), a theory of the etiology of health-promoting and health-compromising behaviors and the effects of the social environment on those behaviors [10], we examine whether adolescent substance use, adolescent depression, adolescent overweight status, and young adult educational attainment mediate the association between low childhood SES and adult obesity.

Prior research suggests that risk factors during adolescence, including substance use, depression, overweight status, and young adult educational attainment may be potential mediators of the low childhood SES-adult obesity association. Two longitudinal studies reveal a positive association between adolescent substance use and obesity during young adulthood. Using data from the 1979 National Longitudinal Survey of Youth, Huang and colleagues found adolescent cigarette smoking and marijuana use (ages 12-18) increased obesity risk in young adulthood (20-24 years) [11]. In another study, adolescent chronic heavy drinking (13-18 years) was associated with being overweight/obese at age 24 [12]. Some studies suggest an association between adolescent depression and increased risk of elevated adult BMI [13]. In addition, being overweight/obese during adolescence is associated with adult obesity [14]. Despite these findings, we are unaware of any studies that examine these adolescent health behaviors as mediators of pathways linking low childhood SES and adult obesity in mid-life.

Young adult educational attainment might function as an additional risk source for adult obesity. Educational attainment may provide the skills to acquire health-related knowledge and implement behaviors consistent with healthy lifestyles, which in turn reduce obesity risk [15]. Lower educational attainment has been linked to increased risk for adult obesity [16]. Further, young adult's educational attainment is influenced by risk exposures during adolescence. Adolescent depressive symptoms, for example, are associated with lower educational attainment in young adulthood [17]. In the case of adolescent substance use, evidence of an association with later-life educational attainment is mixed. Recent studies report that frequent adolescent cannabis use is associated with lower educational attainment in young adulthood but there is less evidence of an association between adolescent alcohol use and educational attainment [18, 19]. Additional research is required regarding the pathways linking childhood SES and adolescent risks with educational attainment and, in turn, adult obesity.

Few studies have examined racial differences in the association between low childhood SES and adult obesity and its mediated pathways. Racial disparities are evident in childhood SES and adult obesity, as well as in the mechanisms hypothesized to link SES and obesity. African Americans report the highest prevalence of adult obesity followed by Latinos, non-Hispanic Whites, and Asians [1]. A similar pattern exists during adolescence with African Americans reporting higher rates of being overweight or obese compared with other racial groups [1]. Racial differences are present in adolescent depressive symptoms with African Americans compared to Whites and Asians as reporting higher rates [20]. Some studies report differences for substance use as well, with white adolescents having a higher prevalence of use than African American, Asian or Hispanic adolescents [21-23]. These findings suggest that race may influence the explanatory mechanisms linking childhood SES and adult obesity, highlighting a need for research in these associations over the life-course.

To address limitations of prior studies, the present study is designed to: examine whether adolescent substance use, adolescent depression, adolescent overweight status, and young adult educational attainment mediate the pathways linking low childhood SES to adult obesity risk, and determine whether these pathways differ for African Americans, Asian Americans, and Whites. No studies, to our knowledge, have assessed potential racial differences in these mediated pathways. Determining the mechanisms underlying early life exposures and adult obesity is critical for the design of effective prevention strategies [3] that can potentially ameliorate racial and economic disparities in adult obesity.

Methods

Participants

This study used data from the Seattle Social Development Project (SSDP), a sample of 808 participants interviewed between ages 10 to 39. SSDP is a longitudinal study of risk and protective factors for behavioral risk taking and health outcomes, a study description can be found elsewhere [24]. The sample was recruited in 1985 from 18 elementary schools that overrepresented high crime neighborhoods. Participants were ethnically diverse; 47% White, 26% African American, 22% Asian American, and 5% Native American. The sample was gender balanced (49% female) and 52% of participants were low-income. Respondents'

parents were interviewed when participants were aged 10-18. Interviewers received a minimum of 30 hours of training in standardized interviewing techniques including: establishing rapport, building interest, effective contact strategies, and refusal prevention techniques. Sample retention rates across waves of data collection can be found in Table 1. The Human Subjects Review Committee at the University of Washington approved all SSDP study procedures and measures.

Measures

Outcome—Adult BMI was calculated using self-reported height and weight – in the physical presence of the interviewer – at ages 24, 27, 30 and 33. At age 35, interviewers objectively measured height and weight. At age 39, height and weight were both self-reported in a self-administered survey and objectively measured in a subsequent home visit. At age 39, the correlation between self-report and objective measures of BMI was 0.96, objective measures of BMI were used and missing values were filled in with self-reports of height and weight where possible. Missing values for BMI were assigned at each interview time point for participants who reported being pregnant or possibly pregnant (see Table 1).

Predictor—Low childhood SES was assessed when participants were 10-16 years of age. Participant responses were coded as 1 if they reported both low parental education (12 years education) and low parental income (lowest quartile, adjusted for household size). All other responses were coded as 0.

Adolescent and Young Adult Mediators—Adolescent substance use was modeled as a latent factor comprised of average past month alcohol, tobacco, marijuana, and hard drug use reported at ages 13-16 and 18. Fit of the latent factor was sufficient [$\chi^2(5) = 26.27(p < .0001)$, RMSEA = .075, CFI = 0.957, TLI = 0.914]. To reduce model complexity, factor scores of the latent factor were included in the final model. Internalizing was measured using self-reports based on the CBCL items at ages 10-13. An average of internalizing items across time resulted in a scale score ($\alpha = 0.78$). Overweight status was measured at ages 10-13 using teacher reports from the Child Behavior Checklist (CBCL) [25]. Teachers rated participants as *overweight as not true* (coded as 0), *somewhat true* (coded as 1) and *very true* (coded as 2). Overweight status was modeled as a latent factor across the four available waves of data, and fit was sufficient [$\chi^2(2) = 6.81(p < .03)$, RMSEA = .056, CFI = 0.993, and TLI = 0.978]. Factor scores were included in the final model. Young adult educational attainment included years of education that participants had received by age 21. The variable was coded as follows: less than high school (coded as 8), high school (coded as 12), and more than high school (coded as 14). Gender was included as a control variable (female = 0, male = 1).

Moderator—Race was determined using self- and school-reports when students consented to the study. When participants reported their race differently across waves of data collection, they were assigned to the racial group they most consistently reported as primary. When inconsistencies could not be resolved, the self-reported race of the biological parents was considered.

Data Analytic Strategy

Structural equation modeling was used to conduct all analysis using Mplus version 7.11 [26]. The maximum likelihood estimator was used and outcome variables were continuous and roughly normally distributed. Full-information maximum likelihood estimation (FIML) was used for missing data. Table 2 shows intercorrelations among variables in the model for all participants and for race separately.

Part of the sample was exposed to a multicomponent, social developmental, preventive intervention in the elementary grades [24]. Control participants were interviewed but had no intervention exposure. To confirm that there were no differences in structural pathways by intervention group, multiple group models were conducted based on participant's intervention status, we tested for differences in model fit when pathways were constrained to be equal across intervention status. We found no differences in the covariance structure, which is consistent with prior analyses [27]; all reported models were conducted with the pooled sample of intervention and control groups.

We first modeled growth in BMI across adulthood from ages 24 to 39 and tested for differences in growth trends by race. We modeled linear growth with the intercept set at age 39 where a life course model would expect the greatest disparities to emerge. The model indicated significant linear growth in BMI. Race differences in BMI trajectories were then tested by using race dummy codes (Black, Asian) to predict both intercept and slope.

Next, we tested whether patterns of mediation also differed by race. We used a multiple group approach to test whether the relationships between a) predictors and mediators and b) mediators and outcomes, differed by race. This structural equation modeling approach provides additional power compared to traditional interaction tests. The final model had nine structural paths linking predictors, mediator, and outcomes. First, a full constrained model with all nine paths held equal was compared to a fully unconstrained model. Results indicated significant differences by race in the omnibus model ($\chi^2=107.07$, $df = 46$, $p<0.0001$), motivating further inquiry. Differences in individual paths were tested for racial group differences using the following procedure: First, path coefficients were constrained to be equal across the three racial groups in each model path examined separately. The resulting chi-square was compared to the fully unconstrained model with two degrees of freedom. Next, if there was a significant decrease in model fit, paths for two of the racial groups were constrained to be the same, and the third was free to vary. This step was repeated in order to examine all possible differences between each race. BMI intercept and slope were allowed to vary by race since significant differences were already established. The final model allowed all paths to vary by race. Paths where differences are significant are noted. Gender was regressed on all model variables.

Results

Descriptive statistics for all model variables are reported in Table 2, including average BMI for each racial group at each time point. Significant differences in the mean BMI at age 39 and slope of BMI were found (except Whites and African Americans did not have significantly different slopes). At age 39, the mean BMI for Asians was 27.6 with a mean

slope of 0.17, mean BMI for African Americans was 32.6 with mean slope of 0.24, and mean BMI for Whites was 29.7 with mean slope of 0.27 (all values controlled for gender).

The coefficients for the structural pathways by race are shown in Table 2 and the final models by race are shown in Figure 1. Results for the unconstrained model are reported separately for each race, and significant racial differences in paths are noted in Table 3. Fit of the final model was adequate ($\chi^2=255.37$, $df=121$, $RMSEA=0.07$, $CFI=0.98$, $TLI=0.96$). The models in Figure 1 show the significant pathways for each racial group.

We found overweight status in adolescence was significantly associated with increased BMI at age 39. This pathway was consistently significant across Asian Americans, African Americans, and Whites. In other respects, the models suggest many unique relationships among the constructs examined for each racial group.

Among Asian Americans, there was a statistically significant unmediated pathway linking low childhood SES with *reduced* BMI intercept. This finding suggests that the inclusion of adolescent substance use and mental and physical health risks did not fully mediate the path between childhood SES and adult obesity risk at age 39. Zero order correlations in Table 2 indicated this pathway among Asian Americans was in the opposite direction and significantly different from African Americans. No pathways other than childhood SES and adolescent overweight status predicted adult BMI intercept or slope among Asian Americans.

Among African Americans, there was no association of low childhood SES with adult BMI intercept or BMI slope in the mediated model, although zero order correlations show a robust relationship between these variables in 4 of the six time points. While pathways examined did not link SES and BMI, results suggest that both low childhood SES and substance use in adolescence reduced years of education for African Americans in young adulthood. However, for African Americans there was no significant pathway between educational attainment and BMI slope or intercept at age 39. The only significant predictor of BMI intercept was adolescent overweight status.

Among Whites, the pathway linking childhood SES and adult BMI intercept and slope was mediated by young adult educational attainment and adolescent overweight status. The path from young adult education to BMI slope was significantly different compared to both African Americans and Asian Americans, suggesting that Whites may have received a health benefit from increased education not found in other racial groups. The pathway between childhood SES and adult BMI trajectory was also mediated by overweight status in adolescence. Significant indirect effects of low childhood SES through educational attainment and overweight status on both the intercept and slope of BMI were confirmed in formal mediation tests ($\beta=0.18$, $p<.01$; $\beta=0.29$, $p<.01$; $\beta=0.16$, $p<.05$; $\beta=0.12$, $p<.05$, respectively). Internalizing and substance use were not associated with childhood SES, but they were significantly linked with BMI through reduced educational attainment.

Discussion

The models presented in this study examined life course pathways linking low childhood SES to adult obesity risk at age 39. We found differences by racial group status in the adolescent explanatory mediators linking childhood SES and adult obesity. These findings question the approach of adopting universal prevention targets in reducing racial disparities in obesity. Overall, our findings suggest that adolescent overweight status was the only variable that consistently predicted adult obesity risk across all racial groups. This finding is consistent with previous studies which found elevated childhood BMI is associated with adult obesity [5-15]. It also highlights the importance of early life weight issues as a target of prevention as elevated childhood BMI is associated not only with later obesity but also with chronic health risk (e.g., diabetes mellitus and heart disease) across the life-course. Once overweight status is established in early life, management of adult obesity is challenging and often unsuccessful [28].

We found evidence that the associations between low childhood SES, adolescent health risks, young adult educational attainment and adult obesity risk differed by race. For Whites, the path linking young adult education to adult obesity risk was significantly different compared to both African Americans and Asian Americans. Adolescent overweight status mediated the pathway between childhood SES and adult obesity risk. Specifically low childhood SES was associated with adolescent overweight status and, in turn, adolescent overweight status was associated with adult obesity risk. Consistent with previous studies, this finding suggests that early life economic disadvantage may increase the risk of being overweight during adolescence, which then may increase the risk of obesity in later life [29]. Finally, we found a mediated pathway between adolescent internalizing and adult obesity risk through low young adulthood educational attainment, which is in line with prior research [30]. However, adolescent internalizing and substance use did not function as mediators linking childhood SES to adult obesity.

Among Asian Americans, we found an unmediated pathway linking low childhood SES and *decreased* adult obesity risk, which is inconsistent with previous studies that found low childhood SES increased the risk of adult obesity [8, 9]. Over half of the Asian American families in the current sample were first and second generation immigrants, or at least one parent was an immigrant. It is possible that there may be healthier dietary patterns among Asian immigrants compared to native-born, which then leads to reduced obesity risk. Immigrant families are also more likely to be lower SES due to immigrant parents being employed in low paying jobs [31]. In other studies, healthier dietary patterns (higher fruit and vegetable consumption, lower soda consumption) were more likely to be reported among Asian immigrant youth compared to Whites [32]. Researchers also report first generation immigrant youth have significantly lower odds of obesity compared to US-born youth [33].

For African Americans, there were no significant pathways linking low childhood SES and adult obesity risk. African American adult obesity risk is not well explained by any path in our model except adolescent overweight status, and the pathway from childhood SES to adolescent overweight status failed to reach statistical significance. An explanation for the

finding may be due to the inclusion of only individual-level explanatory mediators in the model linking childhood SES and adult obesity. Extant research suggests elements of the built environment, including neighborhood-level racial/ethnic segregation, may significantly contribute to adult obesity risk among African Americans [34]. Additional research is needed to better understand other malleable obesity risk factors for African Americans.

The findings should be viewed in light of the following limitations. The sample was originally drawn from a single school district in a large west coast city and, while relatively diverse, did not well represent Latinos or Native Americans. For these reasons, caution should be used in generalizing findings to other populations. Also, most measures were based on self-reports. This was of particular concern for the assessment of height and weight (to derive BMI). However, self-reports at ages 21 through 33 were typically provided during in-home visits by an interviewer, making noticeable over- or under-reports less likely. Moreover, both self-reports and objective assessment were obtained at age 39—these two were highly correlated with each other bolstering confidence in the prior self-reports. Another limitation of our findings is that we had higher rates of missing on our BMI variable by race, though missingness in the entire sample was not related to race. In addition, the temporal order of childhood low SES overlapped with certain adolescent health risks (e.g., overweight status and internalizing).

In summary, efforts to prevent adult obesity should consider these possible differences by race when selecting program components to target specific risk factors. Failure to acknowledge prevention targets differ by race may exacerbate disparities in adult obesity. One universal message is to implement prevention efforts early in the life-course to avoid the onset of childhood overweight status. We also strongly endorse calls for further research on life course prevention targets for non-Whites.

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Implications and Contributions

Study findings suggest there are racial differences in the adolescent behavioral health and economic predictors of adult obesity among African American, Asian American, and White participants from the Seattle Social Development Project.

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Figure 1:

Multiple group structural equation models by racial group

Note: Only significant paths shown. Correlations within time and gender controls modeled but not shown. Standardized estimates reported.

^a Indicates race difference, see Table 2 for specifics.

Table 1

Waves of data collection for the Seattle Social Development Project

Wave	W1	W2	W3	W4	W5	W6	W7	W8	W9	W10	W11	W12	W13	W14*	W15
Year of survey	1985	1986	1987	1988	1989	1990	1991	1993	1996	1999	2002	2005	2008	2010	2014
Mean age	10	11	12	13	14	15	16	18	21	24	27	30	33	35	39
Participants (N)	808	703	558	654	778	783	770	757	765	752	747	719	721	641	678
Deceased (N)	0	0	1	1	2	2	4	6	11	14	15	18	23	28	37
Retention of still living (%)	--	87%	69%	81%	97%	97%	96%	94%	96%	95%	94%	91%	92%	82%	88%
Parent report (N)	605	--	623	626	742	719	728	--	--	--	--	--	--	--	--
Pregnant women excluded from analysis (N)										17	31	16	8	2	2
Teacher report (N)	--	800	510	628	675	--	--	--	--	--	--	--	--	--	--

Note: Wave 14 was focused on collection of DNA samples with significant changes in consent and procedures.

Table 2:

Correlations among model variables

Full sample correlations above diagonal; Asian American sample correlations below diagonal												
	BMI 39	BMI 35	BMI 33	BMI 30	BMI 27	BMI 24	Low Child SES	Over weight	Substa nce use	Inter naliz ing	Educa tion	Me an (S D)
BMI 39	-	0.90	0.86	0.84	0.80	0.76	0.03	0.49	0.04	0.02	-0.18	29.86(7.7)
BMI 35	0.88	-	0.91	0.86	0.83	0.79	0.07	0.49	0.03	0.01	-0.20	29.30(7.2)
BMI 33	0.85	0.92	-	0.91	0.86	0.81	0.03	0.51	0.05	0.01	-0.20	28.58(6.7)
BMI 30	0.82	0.87	0.91	-	0.89	0.81	0.00	0.49	0.01	0.01	-0.15	27.87(6.3)
BMI 27	0.76	0.85	0.82	0.88	-	0.87	0.06	0.52	0.06	-0.02	-0.17	27.10(6.0)
BMI 24	0.78	0.79	0.85	0.87	0.82	-	0.04	0.52	0.08	-0.01	-0.17	26.10(5.6)
Low child SES	-0.21	-0.19	-0.20	-0.24	-0.12	-0.19	-	0.06	0.00	0.08	-0.16	0.19(0.39)
Overweight	0.43	0.38	0.46	0.43	0.44	0.44	0.00	-	0.03	0.02	-0.10	0(0.30)
Substance use	-0.01	0.08	0.10	0.06	0.23	0.09	-0.06	-0.02	-	-0.11	-0.38	0(0.19)
Internalizing	-0.05	-0.08	-0.05	-0.10	-0.04	-0.08	0.10	0.09	-0.04	-	-0.03	0.80(0.33)
Education(years)	-0.26	-0.45	-0.25	-0.19	-0.24	-0.21	0.02	-0.17	-0.12	0.11	-	12.64(1.94)
Gender(male=1)	0.05	0.10	0.18	0.14	0.18	0.20	-0.04	0.06	0.19	-0.14	-0.08	0.50(0.50)
Mean(SD)	27.72(6.65)	27.0(5.6)	26.66(5.9)	26.50(5.3)	25.56(5.3)	25.06(5.4)	0.40(0.49)	-0.06(0.18)	-0.06(0.13)	0.84(0.30)	13(1.7)	55(0.50)

African American Sample correlations above diagonal; White sample correlations below diagonal.

	BMI 39	BMI 35	BMI 33	BMI 30	BMI 27	BMI 24	Child low SES	Over weight status	Substance use	Inter- nal- izing	Educa- tion	Male	Me- an (SD)
BMI39	-	0.87	0.83	0.82	0.78	0.73	0.14	0.50	0.06	0.03	0.04	-0.24	32.60(8.2)
BMI35	0.90	-	0.88	0.84	0.78	0.76	0.20	0.47	0.03	0.03	0.02	-0.27	31.73(8.24)
BMI33	0.87	0.92	-	0.89	0.88	0.83	0.18	0.52	0.09	0.02	-0.05	-0.23	31.21(7.3)
BMI30	0.86	0.87	0.91	-	0.90	0.81	0.08	0.51	0.05	0.04	0.01	-0.17	30.17(6.98)
BMI27	0.81	0.85	0.84	0.88	-	0.87	0.16	0.57	0.10	-0.06	-0.08	-0.23	29.87(7.0)
BMI24	0.75	0.79	0.77	0.76	0.86	-	0.14	0.55	0.16	-0.01	-0.07	-0.17	28.51(6.4)
Childhood low SES	0.16	0.13	0.10	0.08	0.09	0.13	-	0.07	0.08	0.07	-0.31	-0.22	0.25(0.43)
Overweight status	0.47	0.50	0.48	0.46	0.47	0.50	0.15	-	0.07	-0.08	0.05	-0.15	.07(0.36)
Drug use	0.00	-0.01	-0.04	-0.06	-0.04	-0.03	0.10	-0.03	-	-0.06	-0.40	0.05	.02(0.21)

African American Sample correlations above diagonal; White sample correlations below diagonal.

	BMI 39	BMI 35	BMI 33	BMI 30	BMI 27	BMI 24	Child low SES	Over weight status	Substance use	Inter- nal- izing	Educa- tion	Male	Me- an (SD)
Internalizing	0.02	0.01	-0.01	0.01	-0.01	-0.02	0.00	0.05	-0.14	-	-0.02	-0.17	0.84(0.33)
Years of education	-0.20	-0.21	-0.21	-0.17	-0.14	-0.14	-0.28	-0.15	-0.41	-0.07	-	-0.07	12.18(2.09)
Gender (male=1)	0.03	0.06	0.06	0.07	0.08	0.08	0.03	-0.01	0.12	-0.19	-0.07	-	0.51(0.50)
Mean (SD)	29.39(7.4)	28.92(6.8)	28.09(6.4)	27.24(6.0)	26.34(5.3)	25.37(4.87)	0.06(0.23)	-0.01(0.29)	0.02	0.77(0.33)	12.7(1.95)	0.51(0.50)	-

Note: Significant correlations bolded p<.05

Table 3:

Standardized structural model results and pathway racial difference tests

	Asian American			African American			White			Race Differences
	β^*	S.E	p-value	β^*	S.E	p-value	β^*	S.E	p-value	Based on significant X^2_2
Child SES→BMI Intercept	-0.19	0.07	0.01	0.08	0.07	0.26	0.01	0.05	0.83	Asian Am.<Af. Am.
Child SES→BMI Slope	-0.21	0.11	0.06	0.07	0.11	0.55	0.02	0.07	0.72	
Child SES→Drug Use	-0.05	0.07	0.49	0.10	0.07	0.16	0.09	0.05	0.07	
Child SES→Internalizing	0.10	0.08	0.20	0.04	0.07	0.63	0.01	0.05	0.85	
Child SES→Overweight	0.01	0.08	0.94	0.04	0.07	0.56	0.15	0.05	0.00	Asian Am.<White
Child SES→Education	0.00	0.08	1.00	-0.31	0.06	0.00	-0.22	0.0	0.00	Asian>Af. Am., White
Drug Use→BMI Intercept	0.02	0.07	0.77	0.06	0.07	0.45	-0.10	0.06	0.08	
Drug Use→BMI Slope	-0.09	0.11	0.44	-0.02	0.12	0.86	-0.05	0.07	0.47	
Drug Use→Education	-0.11	0.08	0.13	-0.39	0.06	0.00	-0.41	0.04	0.00	
Internalizing→BMI Intercept	-0.10	0.08	0.22	0.01	0.07	0.84	-0.03	0.05	0.53	
Internalizing→BMI Slope	-0.03	0.12	0.78	0.12	0.10	0.24	-0.01	0.07	0.92	
Internalizing→Education	0.13	0.08	0.11	-0.03	0.07	0.69	-0.14	0.05	0.00	Asian Am.<White
Overweight→BMI Intercept	0.43	0.07	0.00	0.50	0.06	0.00	0.44	0.04	0.00	
Overweight→BMI Slope	0.18	0.11	0.09	0.06	0.10	0.58	0.18	0.06	0.01	
Overweight→Education	-0.18	0.07	0.01	0.09	0.06	0.16	-0.12	-0.05	0.01	Af. Am. >White, Asian Am.
Education→BMI Intercept	-0.13	0.08	0.08	0.02	0.08	0.77	-0.18	0.06	0.00	
Education→BMI Slope	-0.14	0.12	0.24	0.19	0.12	0.13	-0.16	0.07	0.03	White< Af. Am.

Note: Af.= African; Am.= American. All paths controlled for gender, results not shown.

* Standardized estimates reported. Significant paths bolded p<.05.