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Original article

Racial Differences in Mechanisms Linking Childhood Socioeconomic Status With Growth in Adult Body Mass Index: The Role of Adolescent Risk and Educational Attainment

Amelia R. Gavin, Ph.D., M.S.W., M.P.P.[a,*], Tiffany M. Jones, Ph.C., M.S.W., M.F.T.[a], Rick Kosterman, Ph.D.[a,b], Jungeun Olivia Lee, M.S.W., Ph.D.[b], Christopher Cambron, Ph.D., M.S.W., M.P.P.[c], Marina Epstein, Ph.D.[a,b], Karl G. Hill, Ph.D.[d], and J. David Hawkins, Ph.D.[b]

[a] *School of Social Work, University of Washington, Seattle, Washington*
[b] *Social Development Research Group, School of Social Work, University of Washington, Seattle, Washington*
[c] *School of Social Work, University of Southern California, Los Angeles, California*
[d] *Center for Health Outcomes and Population Equity, Huntsman Cancer Institute, University of Utah, Salt Lake City, Utah*
[e] *University of Colorado, Boulder, CO*

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A B S T R A C T

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 to 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 to 39 years. 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 Asian Americans, 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.

IMPLICATIONS AND CONTRIBUTION

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.

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]. Comorbid conditions associated with obesity include cardiovascular conditions, hypertension, type 2 diabetes, and major depression [2]. Effective treatments designed to reduce adult obesity are

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* Address correspondence to: Amelia R. Gavin, Ph.D., M.S.W., M.P.P., School of Social Work, University of Washington, Seattle, WA 98105-6299.
E-mail address: gavina@uw.edu (A.R. Gavin).

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limited, despite our advancing knowledge of nutritional and physical activity factors that 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 the 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, 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 et al. found adolescent cigarette smoking and marijuana use (ages 12–18 years) 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 body mass index (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 midlife.

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 adults' 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 to other racial groups [1]. Racial differences are present in adolescent depressive symptoms, with African Americans compared to whites and Asian Americans 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 American, 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 and 39 years. 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 years. 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 .96, objective measures of BMI were used, and missing values were filled in with self-reports of