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Associations of Family and Neighborhood Socioeconomic Characteristics with Longitudinal Adiposity Patterns in a Biracial Cohort of Adolescent Girls

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Although many studies have examined the relationship of adiposity with neighborhood socioeconomic context in adults, few studies have investigated this relationship during adolescence. Using 10-year annual measurements of body mass index, expressed as z-scores (BMI_z), obtained from 775 black and white participants of the National Heart, Lung, and Blood Institute Growth and Health Study, a prospective cohort study of girls from pre- to postadolescence, we used multilevel modeling to investigate whether family socioeconomic status (SES) and neighborhood socioeconomic characteristics (measured by census-tract median family income) explain variation in BMI_z trajectory parameters. Analyses controlled for pubertal maturation. We found that lower SES was associated with higher overall levels of BMI_z for both white and black girls. Additionally, lower-SES black girls had a more sustained increase in BMI_z during early adolescence and reached a higher peak compared to higher-SES black girls and to white girls. Neighborhood income was associated with BMI_z trajectory for black girls only. Unexpectedly, among black girls, living in higher-income neighborhoods was associated with higher overall levels of BMI_z, controlling for SES. Our findings suggest that neighborhood socioeconomic characteristics may affect adolescent BMI_z trajectories differently in different racial/ethnic groups.

Introduction

Overweight and obesity in children and adolescents are major risk factors for chronic disease and are associated with increased adult obesity risk (Whitaker et al. 1997) and premature mortality (Engeland et al. 2003; Engeland et al. 2004). The prevalence of overweight and obesity in children and adolescents exceeds 25 percent in many countries

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(World Health Organization 2009). In 2007–08, 34 percent of U.S. adolescents aged 12 to 19 were overweight or obese (Ogden et al. 2010).

There are large disparities in obesity prevalence among population groups in the United States, with prevalence varying by socioeconomic status (SES) (Ogden et al. 2014; Wang and Beydoun 2007). In the United States and other high-income countries, children from families with lower SES, as measured by household income or parental education, often have a higher risk of obesity (e.g., Strauss and Knight 1999; Stamatakis et al. 2005; Lamerz et al. 2005; Romon et al. 2005; Howe et al. 2011; Shrewsbury and Wardle 2008). However, the relationship between family SES and obesity has been found to vary by race and gender. In a study of children and adolescents using data from the National Health and Nutrition Examination Survey, Wang et al. (2007) and Wang and Zhang (2006) found that SES was inversely related to obesity risk among whites but not among blacks or Hispanics; in fact, high-SES black girls were at increased risk compared to their lower-SES counterparts. Using data from the National Longitudinal Study of Adolescent Health (Add Health), Gordon-Larsen, Adair, and Popkin (2003) reported similar findings, while Robinson et al. (2009) found that the lowest parental education stratum had the highest prevalences of obesity for black females, white females, and white males, and the lowest prevalence for black males. Scharoun-Lee, Kaufman, and Popkin (2009) examined racial/ethnic differences in the influence of life-course SES on obesity risk from adolescence to adulthood, and found no differences. Using data from the National Longitudinal Study of Youth, Hernandez and Pressler (2014) found that childhood poverty was positively related to overweight in young adulthood among white, black, and Hispanic women, but inversely or not related to obesity risk among white, black, and Hispanic men. Studies among U.S. adults show similarly complex variation in the relationship between SES and obesity across gender, age, and racial/ethnic groups (see, e.g., Zhang and Wang 2004; Wang and Beydoun 2007).

Obesity is related not only to individual SES, but also to the socioeconomic characteristics (SEC) of a person's environment (Robert and Reither 2004; Wang et al. 2007). In higher-income countries, neighborhood SEC such as aggregated income, education, and employment levels has been consistently associated with increased obesity risk (e.g., Kinra, Baumer, and Davey 2005; Kinra, Nelder, and Lewendon 2000; Black and Macinko 2008). Neighborhood SEC has been reported to be associated with features of an obesogenic environment, such as a lack of healthy food options and safe facilities for physical activity (Gordon-Larsen et al. 2006; Morland and Filomena 2007; Wang et al. 2007). Most of these studies have been of adults; few studies have focused on children or adolescents specifically. Furthermore, most studies of children and adolescents have been cross-sectional (e.g., Chen and Paterson 2006; Janssen et al. 2006; Nelson et al. 2006), with only a few having a longitudinal design (Burdette and Needham 2012; Nicholson and Browning 2012).

A life-course approach suggests that an insult to the optimal development of adipose tissue during youth may have enduring effects on obesity and obesity-related conditions later in life (Darnton-Hill, Nishida, and James 2004; Daniels et al. 2005). Adolescence has been identified as a particularly critical period for the development of obesity, as it is characterized by pubertal development and considerable changes in body composition (Dietz 1994; Adair 2008). From a biological perspective, it is considered a critical period for the differentiation of adipocytes, cells that specialize in storing energy as fat (Daniels et al. 2005). From a psychosocial perspective, it is considered a period of increasing independence and transition into adulthood. During this life stage, adolescents become increasingly exposed to the neighborhood environment (Leventhal, Dupere, and Brooks-Gunn 2009).

As they get older, adolescents may feel more empowered to purchase foods on their own as a result of increasing independence and money obtained from their allowances and/or jobs. Furthermore, peer influences become more important with age (Taylor et al. 1998; Beal, Ausiello, and Perrin 2001). These factors suggest that the effects of neighborhoods on adolescents become more evident with age.

Studies have also reported that the relationship between neighborhoods and health outcomes varies by race. Several cross-sectional studies have reported that the association of neighborhood poverty with obesity risk is stronger among whites than among blacks (Wickrama, Wickrama, and Bryant 2006; Lovasi, Hutson, et al. 2009; Lovasi, Neckerman, et al. 2009). A longitudinal study using three waves of data from the Add Health study found that the association of neighborhood deprivation with body mass index (BMI) was weaker among black and Latino youth than among white youth (Burdette and Needham 2012). These attenuated effects, it is argued, may be explained because minorities are less apt to reap the benefits of community resources compared to whites (Burdette and Needham 2012; Lovasi, Neckerman, et al. 2009). It is also possible that these attenuated effects result from compressed variation in neighborhood SEC among minority populations. In addition, trajectories of adiposity appear to differ between black and white women. Specifically, a study reported that black women had higher baseline BMI and a greater rate of increase in BMI over 16 years compared to white women (Ruel et al. 2010).

Given these observations from the literature, we investigate the following hypotheses with regard to the longitudinal effects of family SES and neighborhood SEC on adiposity, as measured by sex- and age-specific body mass index z-scores (BMIz):

1. Lower family SES is associated with higher BMIz and steeper trajectories of BMIz increase during adolescence in both black and white girls.
2. Independent of family SES, neighborhood SEC is associated with BMIz trajectories; specifically, girls living in low-SES neighborhoods experience greater increases in BMIz during adolescence compared to girls living in high-SES neighborhoods.
3. Race moderates these associations such that the effects of neighborhoods on BMIz trajectories are attenuated among black girls compared to white girls.

We test these hypotheses using 10 years of prospective data from the National Heart, Lung, and Blood Institute Growth and Health Study (NGHS; National Heart, Lung, and Blood Institute Growth and Health Study [NGHS] Research Group 1992). Taking advantage of the comparatively long period of observation and high frequency of anthropometric measurements (collected annually), we focus on examining longitudinal patterns of adiposity and their association with family SES and neighborhood SEC. Longitudinal *patterns* (or trajectories) of adiposity, rather than simply changes in weight status (obese/nonobese), are important to study, given findings such as those of Barker et al. (2005), which suggested that a pattern of low adiposity followed by rapid weight gain during early childhood is associated with insulin resistance and coronary events in later life.

Methods

Study Design and Sample

Data analyzed for this study are from the NGHS, a 10-year prospective cohort study of 2,379 girls, enrolled at age 9 to 10 years in 1987–88, designed to identify factors associated with the onset and development of obesity in black and white preadolescent girls (NGHS

Research Group 1992). At each annual examination (visit), height and weight were measured and all residential addresses of the participants in the preceding year were recorded. Pubertal development stage was assessed by physical examination using a modification of Tanner staging (Tanner 1992; NGHS Study Group 1992). Information on education and income was obtained from parents during the first year of the study. The NGHS had three recruitment sites. We used data from participants recruited by the University of California at Berkeley site ($N = 887$), which had residential addresses geocoded. These participants resided in Contra Costa County, California, at study entry. Only participants retained at Visit 9 and/or 10 were included, resulting in an analytic sample of 775 participants (87% of original sample). Participants experiencing pregnancy during the study were excluded from our analyses.

Measures

The dependent variable was sex- and age-specific BMIZ, which indicates BMI relative to other individuals of the same age and sex on a standard deviation scale. BMIZ was calculated using Centers for Disease Control and Prevention (CDC) 2000 Growth Charts (Kuczmarski et al. 2002) and software developed by the CDC (<http://www.cdc.gov/nccdphp/dnpao/growthcharts/resources/sas.htm>).

To determine neighborhood SEC, participant addresses were geocoded using ArcGIS, version 10.0 (Esri, Redlands, CA) to determine the census tracts in which they had lived. We considered several measures of neighborhood SEC available from the U.S. Census Bureau, including median family income (referred to as median income herein), percentage of families living in poverty, and percentage of college graduates, as well as composite measures such as the neighborhood disadvantage index (Ricketts and Sawhill 1988; Sampson, Raudenbusch, and Earls 1997), all of which were highly correlated (Pearson $r = 0.64$ – 0.98). Neighborhood disadvantage index is the average of the percent of female-headed households, the percent of households in poverty, the percent of individuals on public assistance, and the percent of males unemployed in a community (Ricketts and Sawhill 1988; Sampson, Raudenbusch, and Earls 1997). Because its distribution was less skewed and its ranges for blacks and whites had greater overlap compared to other measures, census-tract median income from the 1990 U.S. Census was selected to characterize neighborhood SEC. When a participant had lived in multiple census tracts during a preceding year, the mean of the median income values over all reported census tracts of residence was used. A single average value was computed for each year, and these yearly values were used as a time-varying covariate, which changed when participants moved. Time points at which participants resided outside Contra Costa County or adjacent Alameda County were not included in analyses. We repeated the analyses using neighborhood disadvantage index.

Family-level SES was assessed by family annual income and the educational attainment level of the parent or guardian with the higher level of education (maximal parental education). These two variables were combined into a single composite family SES index by coding each variable on an ordinal scale with the same range and then taking the mean. This composite score was dichotomized at the midpoint to classify the participants as having high or low family SES. A continuous version of this index was not used in analyses since this variable did not have a proper numeric scale. Because these variables were collected only in Year 1, they were modeled as time-invariant. As a sensitivity analysis, we repeated the analysis using three-level maximal parental education (less than high school, high school or some college, college degree or higher) as the family SES measure and including both family income and maximal parental education together in the model.

Pubertal development stage may confound associations with adolescent adiposity (Kaplowitz et al. 2001). Pubertal stage was considered as a time-varying covariate, using a four-level pubertal stage variable based on Tanner staging (Tanner 1992)—prepuberty, early maturity, midpuberty, maturity—and also as a time-fixed covariate for which participants were classified as early maturers or late maturers depending on whether they reached maturity before or after age 15; this age was selected because the median age at which the participants reached pubertal maturity (Tanner stage 5) was 15.4 years.

Statistical Methods

The longitudinal trajectories of BMI_z of the participants were modeled using multi-level polynomial growth curve models (Fitzmaurice, Laird, and Ware 2011; Hedeker and Gibbons 2006). Such models include: (1) a level-1 within-subjects model for change over time in the outcome variable, represented as

$$y_{it} = b_{0i} + b_{1i}a_{it} + b_{2i}a_{it}^2 + \dots + b_{pi}a_{it}^p + \varepsilon_{it}$$

where y_{it} is the observed value of the outcome variable at time t for individual i , a_{it} is the age at time t for individual i , and b_{pi} is the growth trajectory parameter for individual i associated with the polynomial term of degree p , $p = 0, 1, \dots, P$; and (2) a level-2 between-subjects model to represent variation in growth trajectory parameters between individuals, including dependence of growth parameter values on covariates, represented as

$$b_{pi} = \beta_{pi} + \sum_{q=1}^Q \beta_{pq}X_{qi} + r_{pi}$$

where X_{qi} is a covariate, β_{pq} represents the effect of X_{qi} on the p th growth parameter, and r_{pi} is a random effect with mean of 0.

Development of the multilevel growth curve models was conducted separately for white and black participants. The first step was to determine the best-fitting growth curve model for each race group, absent any covariate effects. For each race, we began with a full model including fixed and random effects for intercept, linear, quadratic, and cubic terms for age (centered at age 15 years). We conducted likelihood ratio tests for the random effects, beginning with the cubic term, to determine which to retain in the models, comparing the test statistic to an equal mixture of chi-squares with one and two degrees of freedom, as recommended by Verbeke and Molenberghs (2000). We then conducted likelihood ratio tests for the fixed age polynomial terms to arrive at the best-fitting basic growth curve model for each race group. Models for the within-subject error covariance structure were compared using the Akaike Information Criterion (AIC; lower AIC indicates better model fit), and a first-order autoregressive process (AR-1) was selected.

We next determined the best manner in which to enter pubertal maturation into the models. We examined models incorporating pubertal stage as a time-varying covariate and as a time-fixed early maturer/late maturer dichotomous variable. Results from both were similar; hence, we used the simpler model in which pubertal maturation was entered as a dichotomous variable. The resulting model was termed Model 1, or the base model.

Next, we built upon the base models to incorporate family SES and neighborhood SEC in the level-2 models. Family SES was entered dichotomously as high or low, and neighborhood SEC was entered as a time-varying continuous covariate. Models were constructed in

steps. Model 2 consisted of Model 1 plus the family SES binary indicator. Model 3 added neighborhood median income. Model 4 added terms for interactions between family SES and neighborhood median income. We compared the models using AIC and likelihood ratio tests (Fitzmaurice, Laird, and Ware 2011; Hedeker and Gibbons 2006).

In longitudinal studies, it is possible to distinguish two different effects of time-varying covariates: (1) a population-average or between-subject effect corresponding to the overall effect of the covariate on the outcome variable, as measured by the association of the mean of the covariate with the outcome; and (2) an individual-specific or within-individual effect, corresponding to the effects of deviations from the average of the covariate on the series of responses within an individual (Neuhaus and Kalbfleisch 1998). Thus, we also tested for a difference in population-average and individual-specific effects for neighborhood SEC. In our application, this corresponded to testing whether the difference in growth parameters between individuals who differ in mean neighborhood SEC (averaged over the 10-year observation period) by one unit was the same as the difference in growth parameters associated with a one-unit change in neighborhood SEC within an individual.

Analyses were conducted using SAS version 9.3 (SAS Institute Inc., Cary, NC).

Results

The analytic dataset consisted of 367 white and 408 black participants (Table 1). At baseline, black and white girls were of similar age (10 years) but differed with respect to many other characteristics. Black girls had higher adiposity compared to white girls at both baseline and at Visit 10. For example, at Visit 10, 24 percent of black girls were obese compared to 12 percent of white girls. A higher proportion of black girls had reached pubertal maturity before age 15 compared to white girls. Also, black girls had lower family and neighborhood socioeconomic resources than white girls. For instance, 20 percent of black girls had parents with a college degree compared to 43 percent of white girls. Similarly, the average median income of the census tracts in which they had lived (time-weighted) was \$29,667 for black girls compared to \$47,979 for white girls.

Figure 1 shows the trajectories of BMI_z for a representative sample of participants. There is considerable variation in initial BMI_z at the first visit, as well as in trajectory shape over the observation period, with some participants following linear trajectories and others following curvilinear trajectories.

Model building for the growth curve model (first conducted without covariate effects) revealed that the BMI_z trajectories for white participants were best represented by a quadratic polynomial and random effects for the intercept, linear, and quadratic terms, while the trajectories for black participants were best represented by a cubic polynomial, also with random effects for the intercept, linear, and quadratic terms (results available from authors). The effect of pubertal maturity was found to be adequately represented by including an effect of early versus late maturity (maturity before or after age 15 years) on the intercept.

In Table 2, we compare the fit of multilevel growth curve models by progressively adding effects of family SES, neighborhood median income, and interactions between family SES and neighborhood median income. For white participants, adding family SES as a covariate explained a significant amount of variation in growth parameters ($p = .01$). The lack of improvement in model fit after adding neighborhood median income as a covariate indicated that this variable did not explain substantial variation in growth parameters after accounting for family SES (Model 3). Further adding interactions between family SES and neighborhood median income also did not improve the model fit. For black participants,

Table 1
 Characteristics of the National Heart, Lung, and Blood Institute Growth and Health Study participants ($N = 775$)

Characteristic	White ($n = 367$) mean \pm SD (min, max) or N (%)	Black ($n = 408$) mean \pm SD (min, max) or N (%)
Age at Visit 1, years	10.0 \pm 0.6 (9, 11)	10.0 \pm 0.6 (9, 11)
BMI z-score, Visit 1	0.3 \pm 1.0 (-2.5, 2.7)	0.5 \pm 1.1 (-2.6, 2.6)
Underweight (<5th percentile)	9 (2%)	9 (2%)
Normal (5th to <85th percentile)	255 (69%)	258 (63%)
Overweight (85th to <95th percentile)	65 (18%)	69 (17%)
Obese (\geq 95th percentile)	38 (10%)	72 (18%)
BMI z-score, Visit 10 ^a	0.4 \pm 1.0 (-3.1, 2.6)	0.7 \pm 1.1 (-3.0, 2.5)
Underweight (<5th percentile)	9 (2%)	8 (2%)
Normal (5th to <85th percentile)	260 (71%)	224 (55%)
Overweight (85th to <95th percentile)	55 (15%)	79 (19%)
Obese (\geq 95th percentile)	43 (12%)	97 (24%)
Age of pubertal maturity		
Age 15 or younger	240 (65%)	287 (70%)
Older than age 15	127 (35%)	121 (30%)
Parental educational attainment		
Less than high school	73 (20%)	104 (25%)
Some college	136 (37%)	223 (55%)
College degree or higher	157 (43%)	80 (20%)
Missing	1 (0.3%)	1 (0.3%)
Family annual income		
<\$10,000	23 (6%)	130 (32%)
\$10,000 to \$19,000	41 (11%)	89 (22%)
\$20,000 to \$39,999	117 (32%)	100 (25%)
\$40,000 and over	171 (47%)	61 (15%)
Missing	15 (4%)	28 (7%)
Family SES composite index		
Low	111 (30%)	253 (62%)
High	256 (70%)	155 (38%)
Census-tract median family income in \$1,000 (mean over period of observation)	48 \pm 11 (18, 73)	30 \pm 12 (12, 60)

Note: ^a BMI z-score at Visit 9 was used if Visit 10 was missing.

adding family SES significantly improved the model fit compared to the base model ($p = .002$), and adding neighborhood median income further improved the model fit ($p = .002$), indicating that both family SES and neighborhood SEC help to explain individual variation in growth parameters. The addition of interaction terms did not improve the model fit.

Tests for a difference in population-average (between-subject) and individual-specific (within-subject) effects of neighborhood median income did not find evidence of a

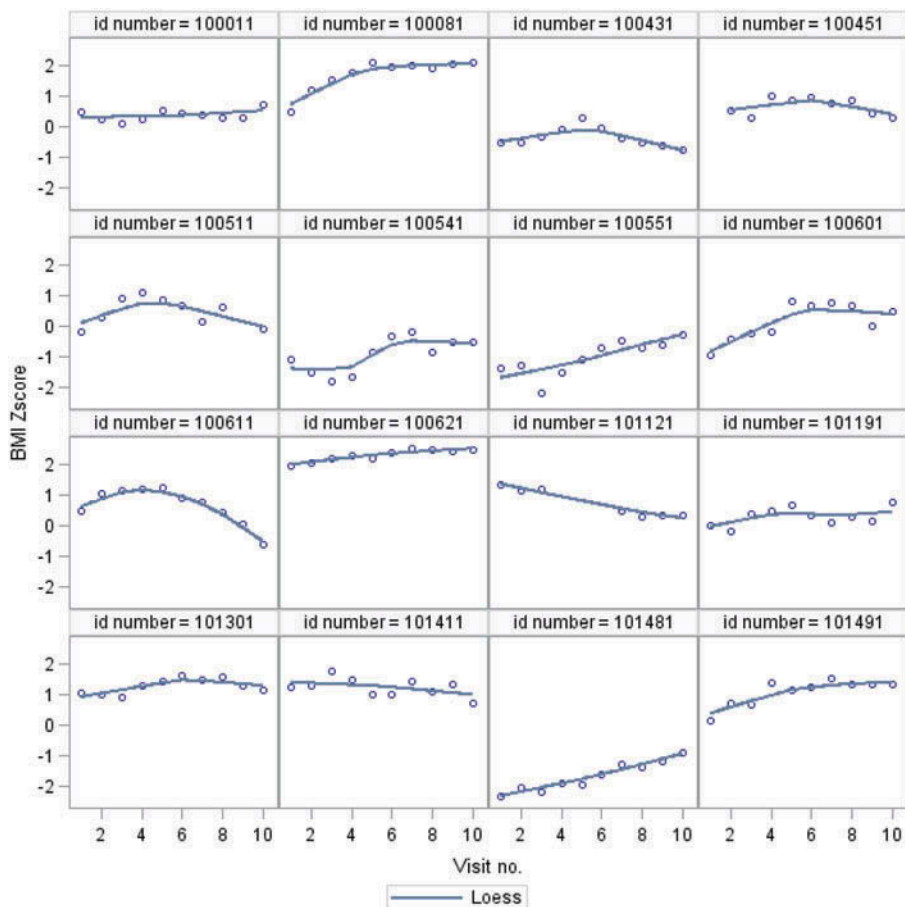


Figure 1. Sample of 16 BMI z-score trajectories of participants in the NGHS study. Scatterplots of BMI z-score versus visit number are overlaid with loess (locally weighted scatterplot smoothing) curves.

difference (all p -values $> .05$). Thus, in the models presented, we assume that the population-average and individual-specific effects are the same. This implies that a one-unit change in neighborhood median income (\$1,000) has the same effect on growth parameters whether we consider individuals who differ in neighborhood median income by \$1,000 or an individual who moves between neighborhoods that differ in median income by \$1,000.

Fitted model parameters are provided in Table 3. Because we centered age at 15 years, the low family SES indicator can be interpreted as indicating that the difference in mean BMIz between girls with low versus high family SES at age 15 was 0.33 among whites and 0.16 among blacks. Other estimated parameters of polynomial growth curve models are difficult to interpret directly. To facilitate interpretation, we provide effect displays that show the mean BMIz growth curves for white and black participants with specific values of covariates (Figure 2). We provide plots of the mean growth curves for participants with low individual SES and low neighborhood SEC (defined as census-tract level median income of \$25,000), high individual SES and low neighborhood SEC, low individual SES and high neighborhood SEC (defined as census-tract level median income of \$50,000), and

Table 2
Comparison of fit of models for BMI z-score trajectories, white and black participants,
National Heart, Lung, and Blood Institute Growth and Health Study

Model	AIC	-2 log likelihood	Df	Likelihood ratio χ^2	<i>p</i> -value
White participants					
1. Base model	2750.6	2726.6			
2. Base model + family SES	2745.9 ^a	2715.9 ^b	3	10.7	0.01
3. Base model + family SES + neighborhood median income	2750.0	2714.0	3	1.9	0.59
4. Base model + family SES + neighborhood median income + family SES-by-neighborhood median income interactions	2752.0	2710.0	3	4.0	0.26
Black participants					
1. Base model	2649.4	2623.4			
2. Base model + family SES	2640.8	2606.8	4	16.6	0.002
3. Base model + family SES + neighborhood median income	2632.2 ^a	2590.2 ^b	4	16.5	0.002
4. Base model + family SES + neighborhood median income + family SES-by-neighborhood median income interactions	2639.5	2589.5	4	0.7	0.95

Notes: AIC = Akaike Information Criterion; SES = socioeconomic status. Base model included basic polynomial growth model terms and pubertal stage. ^aBest model by AIC; ^bbest model by likelihood ratio tests.

high individual SES and high neighborhood SEC. The plots shown are for participants with early pubertal maturity; plots for participants with late pubertal maturity exhibited the same shape but were translated down the vertical axis by an increment (0.006 standard deviation units for whites and 0.12 for blacks). To increase comparability of the growth curves for white and black participants, plots for Model 3, including both family SES and neighborhood median income, are provided for both race groups, even though neighborhood median income was not found to be statistically significant for whites.

The plots show that for white participants, family SES had a large effect on overall BMIz level, with low-family SES participants having higher overall BMIz than high-family SES participants over the entire observation period. There appeared to be a more rapid increase in BMIz among low-family SES participants between ages 9 and 14. Within groups defined by low or high family SES, there was little difference between those residing in higher- versus lower-income neighborhoods, other than a suggestion that girls in higher-income neighborhoods reached a peak BMIz at a slightly older age.

For black participants, at age 9, neighborhood median income was more strongly associated with BMIz than was family SES; an increase in census-tract median income of \$25,000 was associated with a BMIz that was about 0.1 standard deviation units higher. All four groups had a similar steep increase in BMIz between ages 9 and 11, after which BMIz for low-family SES participants continued to increase strongly to a peak around age

Table 3
Estimates from best-fit polynomial growth curve models for longitudinal measurements of BMI z-scores

Variable	White			Black		
	Coef	SE	p-value	Coef	SE	p-value
Intercept	0.424	0.088	<.0001	0.460	0.121	0.0002
Early maturity	0.0042	0.0999	0.97	0.117	0.104	0.26
Low family SES	0.330	0.103	0.001	0.162	0.100	0.105
Neighborhood median family income (\$1,000 increase)	—	—	—	0.0048	0.0014	0.0008
Age	-0.0014	0.0064	0.83	-0.037	0.022	0.09
Age × low family SES	-0.0055	0.0116	0.63	0.029	0.014	0.03
Age × neighborhood median family income	—	—	—	0.00005	0.00051	0.99
Age ²	-0.0073	0.0015	< 0.0001	-0.0045	0.0037	0.22
Age ² × low family SES	-0.0032	0.0027	0.24	-0.0072	0.0022	0.001
Age ² × neighborhood median family income	—	—	—	0.000033	0.000092	0.72
Age ³	—	—	—	0.0025	0.0010	0.01
Age ³ × low family SES	—	—	—	-0.0010	0.0006	0.08
Age ³ × neighborhood median family income	—	—	—	-0.00001	0.000024	0.56
Random effect variance parameter estimates	Estimate	SE	p-value	Estimate	SE	p-value
Intercept	0.68	0.06	< 0.0001	0.84	0.07	< 0.0001
Age	0.0050	0.0008	< 0.0001	0.004	0.0007	< 0.0001
Age ²	0.00011	0.00004	0.006	0.00004	0.00003	0.11
AR-1	0.47	0.06	< 0.0001	0.56	0.07	< 0.0001
Residual	0.13	0.02	< 0.0001	0.12	0.02	< 0.0001

Notes: AR-1 = first-order autoregressive process correlation parameter; SE = standard error; SES = socioeconomic status.

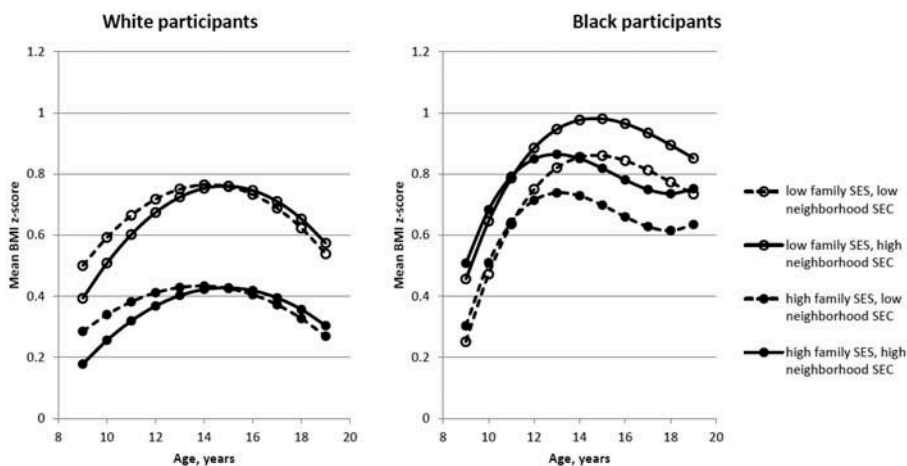


Figure 2. Effect displays showing mean BMI z-score growth curves for white and black participants by low vs. high family socioeconomic status (SES) and low vs. high neighborhood socioeconomic characteristics (SEC; \$25,000 vs. \$50,000 census-tract median family income).

15, while the rate of increase among high-family SES participants decelerated and reached lower peaks at earlier ages (about age 13). Within groups defined by low or high family SES, participants living in higher-income neighborhoods had higher overall BMIz levels.

Repeating the analyses using the neighborhood disadvantage index gave similar results as using neighborhood median income: neighborhood effects were significant for blacks but not whites, and lower disadvantage index (i.e., higher neighborhood SEC) was associated with higher BMIz for blacks. The findings were again replicated when including both family income and parental education as distinct predictors rather than using the family SES composite, and when using three-level maximal parental education as the family SES measure (results available from authors).

Discussion

Few studies have examined trajectories of adiposity among adolescents in relation to neighborhood characteristics. Yet understanding these trajectories is critically important, particularly for youth who are rapidly developing both biologically and socially. Our study, using 10 years of prospective data, investigated how BMIz trajectories varied as a function of family and neighborhood socioeconomic context and race. The data yielded several findings that were not fully consistent with a priori hypotheses based on the current sparse literature for adolescents and the assumption that relationships observed for adults would also hold for adolescents.

Consistent with our first hypothesis, we found that low family SES was related to higher overall BMIz in this sample of adolescent girls from Contra Costa County. The mean difference in BMIz at age 15, between low- and high-family SES girls, was estimated to be 0.33 for white and 0.16 for black girls. Although there is no established threshold for a clinically significant change or difference in BMI z-score, some investigators have used a change of 0.10 units as a benchmark (Dorsey et al. 2010). Patterns of change in BMIz over time, however, were more complex. Among blacks, low family SES was associated with a

more sustained increase in BMIZ during early adolescence and a higher peak; this pattern was not repeated among whites.

Our second hypothesis, that neighborhood SEC is associated with BMIZ trajectories independent of family SES, was only partially supported; neighborhood median income was related to BMIZ trajectories for blacks but not whites. Our third hypothesis, that the associations between neighborhood SEC and BMIZ trajectories would be attenuated among black girls compared to white girls, was not supported. Instead, we found no significant association among white girls, and we unexpectedly found that among black girls, residing in neighborhoods with more advantaged socioeconomic characteristics was associated with higher overall levels of BMIZ, controlling for family SES. This finding was robust, being replicated using various alternative predictor sets for family SES and for neighborhood SEC. We further found a different longitudinal pattern in BMIZ among blacks according to family SES, such that girls from low- and high-SES families diverged in BMIZ trajectory at about age 12. Specifically, the trajectories of girls from low-SES families continued to climb upward, while the trajectories of girls from high-SES families slowed and reached a lower peak at an earlier age. We are unable to empirically account for the mechanisms that drive these patterns, but we speculate that black adolescent girls from high-SES families may be exposed to different psychosocial or biological stressors during early childhood than black girls from low-SES families. For example, black girls may experience psychosocial stresses related to their ethnic minority status that are less buffered by supportive social networks (Jarrett 1995). They may also experience biological stresses. In particular, there is emerging evidence to suggest that exposure to high levels of endocrine-disrupting chemicals (EDCs) found in household products such as plastic food containers, personal care products, and household cleaners and deodorizers is associated with early pubertal maturation (Buttke, Sircar, and Martin 2012), which was observed more frequently in the NGHHS black girls (Braithwaite et al. 2009). While only race but not income has been found to be associated with exposure to EDCs, interactions between race and income have not been explored (Buttke, Sircar, and Martin 2012).

It is curious that neighborhood SEC was associated with BMI trajectory variation only among the black girls in our sample. Some studies have found that the effects of neighborhoods vary by race (e.g., Kirby et al. 2012), while other studies have not (e.g. Kimbro and Denney 2012). Our results differ from other longitudinal studies examining the relation between adolescent adiposity and neighborhood SEC in black and white girls. Nicholson and Browning (2012) found that higher neighborhood disadvantage increased the odds of becoming obese for adolescent girls similarly among whites, blacks, and Hispanics. Burdette and Needham (2012) found no association between neighborhood characteristics and initial BMI for girls in grades 7 through 11. For white but not black or Latina girls, they observed a positive association between neighborhood disadvantage and rate of change in BMI. In comparison, we found that white girls living in more disadvantaged neighborhoods had somewhat higher BMIZ at age 9–10 years, but these differences were no longer evident by age 19. For black girls, living in higher-income neighborhoods was associated with higher BMIZ at age 9–10, and this difference was essentially sustained over the 10-year observation period. These differences among studies may be due to differences in outcomes and age ranges. Nicholson and Browning (2012) examined adolescents who were ages 11 through 21 at the first wave and ages 18 through 25 at the second wave (two waves of data were analyzed), and used obesity as the outcome. The study by Burdette and Needham (2012) examined young persons who were aged 13 at the first wave and 32 at the last wave (three waves of data were analyzed), and their outcome was BMI. In contrast, our study focused on girls starting at age 9 or 10 and ending at age 19 or 20 and examined BMIZ

trajectories. Differences could be explained by our greater level of detail of BMI trajectory (10 annual measurements over a span of 10 years), in which we had more intermediate time points and thus could more fully resolve the longitudinal patterns. Furthermore, we used gender- and age-specific z-scores, which are appropriate because youth are rapidly growing in both weight and height, whereas in adults, the assumption is that BMI changes mainly as a function of weight (with height generally unchanging for most adults). In addition, our study controlled for biological maturation (pubertal stage) based on physical examination, which was either not included in prior neighborhood studies or was based on self-report (Nicholson and Browning 2012). This control is critically important because the nutritional requirements of youth change as a function of maturation, and also because there is some evidence to suggest that early-life deprivation and stress can hasten biological maturation (Mishra et al. 2009). In addition, race differences in average age of maturation have been reported. In an earlier study using data from all three centers of the NGHS, Braithwaite et al. (2009) reported similar findings with regard to the difference in average age of maturation between blacks and whites.

Alternatively, the different findings may be due to the use of different neighborhood disadvantage measures. Burdette and Needham (2012) used parental perceptions of neighborhood disorder, adolescents' perception of neighborhood safety, and a composite index of neighborhood disadvantage based on census-tract measures together in the same models, which makes direct comparisons with our results difficult. Nicholson and Browning (2012) used a composite of census-tract items. We reached similar conclusions whether using census-tract median income or a composite measure. Disentangling the various aspects of the neighborhood environment that act as barriers to or facilitators of obesogenic behaviors is a major methodological challenge (Black and Macinko 2008; Lake, Townshend, and Alvanides 2010; Wall et al. 2012). There is not yet a consensus on which characteristics of a neighborhood may be most strongly related to health outcomes and how best to measure these characteristics. Additionally, it is well known that SES measures can lack comparability across racial groups (Williams and Collins 1995). Further research is needed to better identify which specific features of the neighborhood environment are most important for different population segments.

Our study has limitations. First, we used observational data, which has well-known limitations for causal inference. Self-selection of families into certain neighborhoods could account for the findings and lead to erroneous conclusions about neighborhood effects (Oakes 2004). Second, the generalizability of the findings is limited. The NGHS studied only black and white girls, so our findings cannot be extended to boys or to other racial/ethnic groups. Also, the sample was not designed to be nationally representative; rather, the sampling frame was selected to minimize the disparity in education and income between blacks and whites. Further, it should be noted that our study used data from only one of the three NGHS sites because of the unavailability of geocoded addresses of participants from the other two sites. Our sample was from Contra Costa County only. Some participants were dropped because of missing data, and some did not have data for all 10 visits, which could have resulted in further biases. Nonetheless, the NGHS retained over 90 percent of participants over the course of a decade, and our study used complete data from 87 percent of the participants. Third, we assumed that effects of family SES and neighborhood SEC were the same over all ages, whereas their influence may vary with age. Since family SES was assessed only in Year 1, we were unable to explore its potentially time-varying impact.

These limitations are balanced by several strengths, including a fairly long period of observation with many repeated measures, the use of clinical assessments of height

and weight (not self-reported), and controls for pubertal stage determined by physical examination, which is critically important for persons in the age range studied.

To our knowledge, this is the first report of a difference in the relative contributions of family and neighborhood SEC to BMI trajectories during adolescence between black and white girls. Our findings suggest, as others have noted (Cummins and MacIntyre 2006), that context is important in understanding the contributions of the environment to obesity risk. Further work is needed to elucidate the mechanisms that connect neighborhood context to obesity development among adolescents from varying sociocultural groups. Understanding such mechanisms has implications for the design of effective interventions to address adolescent obesity risk.

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