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Family Background and Racial and Ethnic Differences in Adolescent Weight

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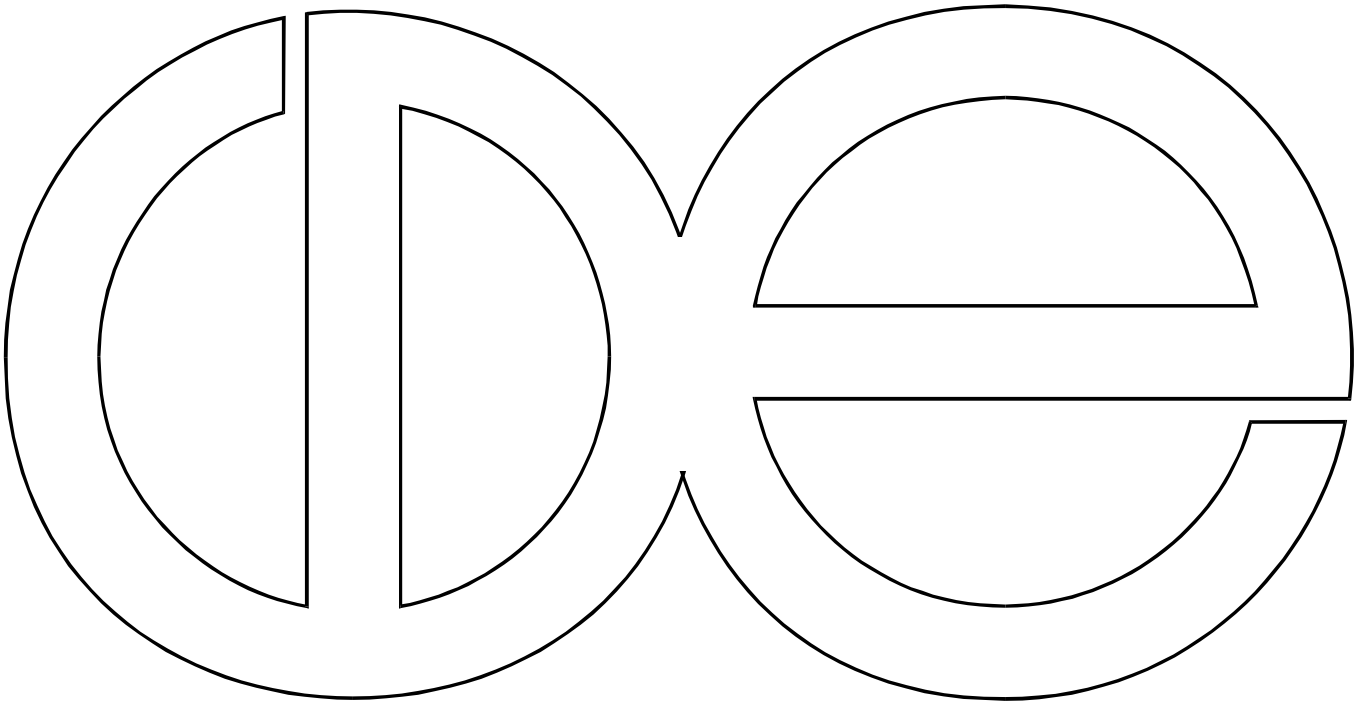

FAMILY BACKGROUND AND RACIAL AND ETHNIC DIFFERENCES IN WEIGHT*

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ABSTRACT

Medical and social research has found racial and ethnic differences in weight relative to stature. While previous research has controlled to varying degrees for family socioeconomic status, other observed and unobserved differences across families could help explain the observed racial and ethnic differences in weight. The present research seeks to better analyze the social factors associated with adolescent weight. We model adolescents' body-mass index (BMI) utilizing sibling resemblance models of sibling pairs identified in the National Longitudinal Study of Adolescent Health. By conducting sibling model analysis in the structural equation framework, we are able to account for unmeasured family background characteristics and parcel out what proportion of the variation in adolescent weight is due to variation between families and variation within families. After accounting for both observed and unobserved characteristics of families, we find that blacks and Hispanics have significantly greater weights relative to whites, though the differences between these two racial groups are smaller than found in previous research. Pairs with other racial identities or mixed racial/ethnic identities show no significant differences in weight relative to whites once the observed and unobserved characteristics of families are accounted for.

Medical and social research has established a negative relationship between body weight and other health outcomes. For example, Calle and colleagues (1999) find that increases in an adult's weight are related to an increased risk of dying from many causes, including cardiovascular disease and cancer. As one might expect, the risk of obesity in adulthood is greater for persons who were overweight as adolescents (Andersen et al., 1998; Whitaker et al., 1997). Research demonstrates that there is a strong relationship between adolescent and adult obesity, especially for those classified as obese according to the National Center for Health Statistics calculations of body mass index (BMI) percentile curves by age and sex (Barlow and Dietz, 1998; Guo et al, 1994; Mossberg, 1989).

Although obesity-associated deaths occur more frequently among adults, obese adolescents suffer significant consequences as well. Excess weight in adolescents is a risk factor for coronary heart disease morbidity and mortality. Obesity may precede the early onset of other coronary heart disease risk factors, such as hypertension and diabetes (Winkleby et al., 1999). Obese adolescents are more likely to be diagnosed with diabetes, hypertension, orthopedic disorders, sleep disorders, increased cholesterol levels, and gall bladder disease (Barlow and Dietz, 1998; Dietz, 1998).

Physical health problems are not the only consequences of being overweight as an adolescent. One physician asserts that the "most widespread consequences of childhood obesity are psychosocial" (Dietz, 1998, p.518). The well-documented stereotypes among children about overweight persons lead to discrimination against obese adolescents (Dietz, 1998; Gortmaker, et al., 1993). Discrimination against overweight adolescents is a proposed explanation for the finding that young women who were obese as adolescents

completed fewer years of education, were less likely to be married and more likely to have lower household income and live in poverty. Similarly, young men who were overweight as adolescents are less likely to be married (Gortmaker et al., 1993).

Psychological consequences of being overweight are also significant. Adolescents frequently perceive their weight as worse than it actually is. Research demonstrates that adolescents' body images are lower than their actual measurements and lower than physician and paternal assessments would suggest (Levinson, Powell, and Steelman, 1986). In addition, early maturation is associated with an increase in body fatness and children who mature early tend to have lower self-esteem. Adolescents who mature early and who are overweight are at a greater risk for adults treating the adolescents as if they are more developmentally advanced than they are. The difference between the treatment these adolescents receive and the treatment that is appropriate could result in their feeling frustrated and having socialization problems (Dietz, 1998). Finally, the relationship between obesity and psychological disorders is complex. Psychological disorders may cause or be related to obesity (Barlow and Dietz, 1998). It is important to note, however, that evidence to date has not found a significant relationship between adolescent obesity and lower self-esteem (Gortmaker et al., 1993). Together, the social and medical consequences related to overweight are substantial, especially when considering the implications for adolescents' later social, psychological, and physical development.

The public health concern over adolescent obesity has heightened in recent years as the prevalence of adolescent obesity has increased dramatically since the 1960's. The greatest increase in prevalence of adolescent obesity occurred during the 1980s and early

1990s (Troiano and Flegal, 1998). In the period between 1988 and 1994, approximately 11 percent of adolescents were overweight (Troiano and Flegal, 1998). Research has begun to document how the patterns of obesity vary by race, ethnicity, age and sex. Several physicians consider the rapid increase in the prevalence of overweight children and adolescents to be a manifestation of societal effects, primarily increases in television viewing (Troiano and Flegal, 1998; Dietz, 1990).

The present research seeks to better analyze the social factors associated with adolescent weight. By taking race and ethnicity, family socioeconomic status, family structure, and residence into account and by including important individual characteristics, we model adolescents' weight relative to their height using the National Longitudinal Study of Adolescent Health (Add Health). We assert that families play an important role in facilitating or inhibiting unhealthy weight. As such, we analyze sibling resemblance models with the family as the unit of analysis. By conducting sibling model analysis in the structural equation framework, we are able to account for unmeasured family background characteristics and parcel out what proportion of the variation in adolescent weight is due to variation between families and variation within families. In the following section we will review the previous literature on the social factors associated with adolescent overweight status. Then we will outline the overall strategy of the analysis and discuss our data and methods in detail. Our results and conclusions follow.

PREVIOUS RESEARCH

Research has documented distinct patterns in overweight by gender. Young women of all racial and ethnic groups, except for blacks, are less likely to be obese than

are young men (Troiano and Flegal, 1998; Popkin and Udry, 1998). Despite the fact that more young men are obese than are young women, other research shows that young women are more likely to perceive themselves as overweight, and more likely to be attempting to lose weight (Kilpatrick et al., 1999; Levinson, Powell, and Steelman, 1986). Black and white adolescent males and black adolescent females are more likely to perceive themselves as underweight than are white adolescent females (Levinson, Powell, and Steelman, 1986). Given that black adolescent females seem to have different perceptions of their weight, the psychological consequences for being overweight may not be as significant for them relative to adolescent females from other racial and ethnic groups.

Several researchers have examined racial and ethnic differences in weight, though most previous research concentrates on black-white differences (Calle et al., 1999; Jacobson and Rowe, 1998; Winkleby et al., 1999). Research has documented significant differences in weight by race/ethnicity and gender variations in the pattern of differences. Blacks and Mexican Americans are more likely to be overweight than are non-Hispanic whites (Popkin and Udry, 1998; Gordon-Larsen et al., 1999; Troiano and Flegal, 1998; Winkleby, et al., 1999). Racial differences in obesity are much less pronounced among young men than among young women. Young black women are almost twice as likely to be obese as young white women, for example, but prevalence differences between young black men and young white men are much smaller (Troiano and Flegal, 1998).

Less research exists on other racial and ethnic groups. Popkin and Udry (1998), however, have shown that all racial and ethnic groups, with the exception of Chinese and Filipinos have higher levels of obesity than non-Hispanic whites using data from the

Add-Health Survey. Generation in the United States also plays a role. Asian-American and Hispanic adolescents who are second or higher generation Americans are more than twice as likely to be obese than their first generation counterparts.

One explanation for the racial and ethnic differences in obesity among adolescents relates to differential activity levels. Boys and girls who watch more than four hours of television each day have greater body fat and greater body-mass index (Andersen et al., 1998). Non-Hispanic blacks have the highest rates of watching more than four hours of television a day and both non-Hispanic blacks and Mexican American youth have lower levels of physical activity than non-Hispanic white youth (Andersen et al., 1998). Vigorous activity levels are lower among females and minority males than for non-Hispanic white males (Gordon-Larsen et al., 1999; Andersen et al., 1998).

Among adult women, research has consistently found an inverse relationship between socioeconomic status and overweight, but this pattern has not been consistently established among children and adolescents (Troiano and Flegal, 1998). Using the third National Health and Nutrition Evaluation Survey (NHANES) data, Troiano and Flegal (1998) find that overweight prevalence for Mexican American and non-Hispanic black youths is not related to family income. For non-Hispanic whites, however, they do find an inverse relationship between family income and overweight (Troiano and Flegal, 1998). Troiano and Flegal (1998) find no discernable pattern in their bivariate analysis of adolescent overweight and parental education. In predicting weight among adolescents, Winkleby et al. (1999) find socioeconomic status is a significant predictor of weight when it is measured either as the household head's educational attainment or as the household's poverty-income ratio.

Due to heritability and socialization, body composition similarities within families are generally expected. Whitaker and colleagues (1997) matched medical records of parents and children residing in Washington state and found that the risk of adult obesity was significantly greater if either the mother or father was obese. At every age interval, both obese and nonobese children were at greater risk for obesity as adults if at least one parent was obese. They found no significant differences in these risk estimates between boys and girls. As children age, the child's own weight supercedes their parents' as the most significant predictor of adult obesity.

To investigate family resemblance in body composition, one can also utilize information on sibling pairs. Little research has yet taken advantage of the twin and sibling data available in the Add-Health study. Jacobson and Rowe (1998) employed the techniques of behavioral genetics to examine the role of genetic and environmental influences on obesity using the twin data in the Add-Health Study. After making the conventional assumptions required for behavioral genetics models, they concluded that genetic factors explained between 45 and 85 percent of the variance in BMI and that genetic factors had similar roles for men and women. Jacobson and Rowe's heritability estimates match heritability estimates found in earlier research (Comuzzie and Allison, 1998).

Our research builds upon previous research by examining racial and ethnic differences in body-mass index (BMI) while accounting for family similarity in body composition. This research provides two major contributions. First, we further develop models of racial and ethnic differences in BMI by using insights from sociological models of the relationship between race/ethnicity and adolescent well-being. Second, we

take advantage of the availability of twins and other siblings in the National Longitudinal Study of Adolescent Health (Add-Health) to estimate sibling models of the effects of race/ethnicity and other aspects of family background on BMI. Our research considers differences among families who are white, black, Hispanic, "other" races, and who are comprised of persons with different racial and ethnic identities.

Our approach to using the twin and sibling data differs from the approach of behavioral geneticists in that we are not concerned about the source of family similarity. We do not try to estimate what proportion of the association in weight among siblings is due to genetics and what proportion is due to the environment. In addition, we control for other factors that relate to weight whereas behavioral genetics models analyze differences in correlations of weight across different types of siblings. At this juncture, we have not analyzed the correlations in BMI according to the relatedness of the siblings. We plan to do this in future research.

OVERVIEW OF THE ANALYSIS

This paper seeks to better model the association of race and ethnicity with adolescent weight. As mentioned above, previous research has found associations between race/ethnicity and weight (Gordon-Larsen, McMurray, and Popkin, 1999; Jacobson and Rowe, 1998; Popkin and Udry, 1998; Troiano and Flegal, 1998). We utilize a sibling model to better analyze these racial/ethnic differences in adolescent weight, as measured by BMI.

Models of sibling resemblance are structural equation models using data from two or more siblings to control for unobserved family background characteristics (Hauser, 1991). A sibling model decomposes a basic regression into (1) a between-family

regression of common family factors (or characteristics similar across siblings) and (2) a pair of within-family regressions of factors varying across siblings (Hauser, 1988).

Sibling models offer several advantages over individual-level models. First, sibling models allow researchers to provide a global indication of the strength of family background characteristics on different outcomes. Generally, the effects of family background variables are underestimated in individual-level models (Hauser, Sheridan, and Warren, 1999). Second, sibling models offer a criterion for proposed theoretical explanations of familial and individual differences in various outcomes. By knowing what proportion of the variance in siblings' weight is explained by measured, family background variables, one can then ask what unobserved, shared family variables explain the rest of the sibling similarity (Hauser, Sheridan, and Warren, 1999). Individual-level models cannot offer such a criterion and therefore can provide only limited answers on the role of family in influencing adolescents' outcomes. Third, sibling models control for the global effects of family background, and therefore give better estimates of the effects that vary between and within families (Hauser, Sheridan, and Warren, 1999). In individual-level models, the within- and between-family variances operate as variation across people, so the sources of this variation cannot be identified. This third advantage is especially important for the current research. By parceling the variation in adolescents' weight into their between- and within-family components, we can better estimate the effects of key variables that have been shown to be associated with weight.

Structural equation modeling can better represent and analyze causal models of effects. The present paper uses LISREL software to test whether race/ethnicity has direct effects on weight after controlling for both observed and unobserved family

characteristics. The model is expressed in LIRSEL notation (see Sörbom and Jöreskog, 1981) by the following equations:

$$\eta = \beta\eta + \gamma\xi + \zeta \quad (1)$$

$$X = \lambda_x\xi + \delta \quad (2)$$

$$Y = \lambda_y\eta + \varepsilon \quad (3)$$

Equation 1 represents the structural model in which η is a vector of latent endogenous variables, β is a matrix of the effects of η on η, ξ is a vector of latent exogenous variables, γ is a matrix of the effects of ξ on η, and ζ is a vector of structural disturbances with a covariance matrix Ψ. The structural disturbances in the endogenous latent variables are specified to be uncorrelated with one another and uncorrelated with the exogenous latent variables. The exogenous latent variables are allowed to be freely correlated with one another.

Equations 2 and 3 are the measurement models for the latent variables. X is a vector containing measured variables related to the exogenous latent variables. λ_x is a vector of factor loadings of X on ξ, and δ is a vector of measurement errors in X, with a variance-covariance matrix of θ_δ. Y is a vector of measured individual weight variables. λ_y is a vector of factor loadings of Y on η, and ε is a vector of measurement errors in Y, with a variance-covariance matrix θ_ε.

Racial differences in BMI could be due to a host of conditions, including socioeconomic, family structure, residential patterns, dietary, activity levels and behavioral, and cultural differences. To different degrees, the conditions influencing these racial and ethnic differences can be measured and included in standard analyses. Our modeling strategy explicitly controls for the causal effects of socioeconomic status,

family structure and residential patterns and for the unobserved factors related to family characteristics. With the sibling model approach we can arrive at better estimates of the relative weight differences by race/ethnicity. Using this social clustering of respondents, we can best estimate the effects of race/ethnicity on weight. Models not recognizing the socially clustered nature of the data miss out on the advantages of sibling models.

Figures 1 and 2 provide graphical displays of our sibling model framework for studying the effect of race/ethnicity on adolescent weight. For help in reading the figure, those elements contained in squares represent measured variables and those elements in the labeled circles are the latent constructs in the structural model. The small, unlabelled circles affecting the "x" and "y" variables indicate measurement error in their respective variables. The arrows represent causal paths from latent constructs to other latent constructs and their measures. Curved, double-sided arrows represent unanalyzed correlation in measurement error.

The top of the figure displays the family-level latent constructs and their measures that influence a common family factor, labeled "Family BMI." The path coefficients from the family-level latent constructs to the common family factor derive from differences across families. The small oval labeled "Z1" represents all other causes of the variation across families not included in the present model. The common family factor, then affects each sibling's underlying BMI. Each sibling's BMI is measured with three separate indicators.

In Figure 2, several individual-level variables are allowed to affect only the adolescent's own BMI. These individual-level constructs appear on the left and right sides of the figure. The left side of the figure represents the causal paths from these

characteristics for Sibling 1's BMI, while the right side of the figure models the characteristics of Sibling 2. Since there is only one variable per each individual-level construct, the paths to the measured variables all equal 1.0 and there is no estimated measurement error. This implies a 100% correlation between the latent constructs and their measures. The path coefficients from the individual-level constructs to each sibling's latent BMI derive from variation within the family. The latent constructs labeled "Z2" and "Z3" capture each sibling's characteristics that are (1) not derived from the family and (2) different from the other sibling.

DATA AND MEASURES

Data

This paper uses data from the National Longitudinal Study of Adolescent Health (Add Health). The Add Health is a study of a nationally representative sample of adolescents in grades 7-12 in the United States. The study was designed to help explain the underlying conditions of adolescent health and health behavior with a special emphasis on the effects of multiple contexts of adolescent life. The Add Health study is longitudinal - adolescents were interviewed between April and December of 1995 (wave one) and again between April and December of 1996 (wave two).

A list of all high schools in the United States served as the primary sampling frame from which a stratified sample of 80 high schools was chosen with probability of selection proportional to size. All students from these high schools who were present on the day of the survey completed an in-school survey. Each school that was part of the in-school component provided a roster of all students enrolled. From the rosters and the pool of participants in the in-school survey, adolescents in grades 7-12 were sampled to

participate in the in-home interview. Approximately 14,738 students participated in both survey waves.

The Add Health dataset is well suited for sibling resemblance research since a substantial number of sibling or sibling-like pairs were identified and interviewed separately as part of a genetic supplement to the in-home survey. The genetic supplement identifies monozygotic and dizygotic twins, other siblings of twins, other full siblings, half-siblings, and non-related sibling-like pairs (i.e., adolescents living in the same household who did not share the same biological mother or father). Both of the adolescents in the sibling pair had to be in grades 7-12 to be included.

Siblings were identified in the in-school survey. Any student who identified him or herself as a sibling to another adolescent in grades 7-12 on the in-school questionnaire was included in the in-home genetic supplement. In addition, previously unreported twins in grades 7-12 who were discovered during the in-home interview were added at that time. These survey procedures lead to a sample of 2,631 unique sibling pairs from 2,213 families.

We then restricted our sample in a few ways. First, our analysis only contains pairs comprised of monozygotic and dizygotic twins, full siblings, half-siblings, step-siblings, and adopted siblings. All other sibling-like pairs have been excluded from our analysis. Second, we removed pairs where one or both of the siblings reported outlier values on our dependent variable for weight, BMI.[1] Third, we eliminated all sibling pairs in which one sibling was pregnant preceding the wave 1 survey (anytime in 1994 and 1995). The exclusion of these pairs is based on the individual weight fluctuations before and after pregnancy. In modeling weight, we did not want to simultaneously model

having a teenage birth. Finally, we eliminated pairs with any missing data on the variables included in the analysis. Persons without valid information on the variables were deleted from the data, as were all the pairs in which they appeared. These restrictions resulted in a sample of 1,404 unique sibling pairs and 1,228 families.[2]

For modeling purposes, we make additional adjustments to our sample of sibling pairs. Since we are not concerned with the particular ordering of siblings within a family or a pair and since we do not know the decision rules for ordering the siblings within the pair, we have double-entered the data so that the data for Sibling 1 and Sibling 2 are symmetric. In calculating the variance-covariance matrix needed for the modeling exercise, we have weighted the sibling pair data so that each family has a weight of 1.

Measures

The dependent variable in our analysis, weight, is measured as the continuous measure of body mass index (BMI). BMI is defined as weight in kilograms divided by the square of height in meters (kg/m^2). Researchers prefer BMI as the standard means to measure adolescent obesity (see Cole 1991, Himes and Dietz, 1994, WHO, 1995). Some researchers categorize BMI to define obesity according to an age-sex standard suggested by the Maternal and Child Health Bureau. That is, obesity is defined as having a BMI measure greater than or equal to 95% for age and gender standards (Barlow and Dietz, 1998, CDC 2000). In this paper, however, BMI is treated as a continuous measure because we are interested in the full scale of weight rather than whether or not an adolescent can be defined as overweight.

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[1] BMI values of less than 15 or greater than 40 are considered outliers (Jacobson and Rowe, 1998).

[2] While we lose 162 unique sibling pairs due to missing data on the dependent variables, we lose an additional 551 unique sibling pairs due to missing data on parental education and family income.

In both wave one and wave two of Add Health, respondents were asked to report their height and weight, which we use to calculate BMI. In wave two, the Add Health study also took physical measures of respondents' height and weight. Adolescents were weighed with clothes but not shoes on a spring scale brought by the interviewer. The interviewer measured adolescents' height in feet and inches using a seamstress-type tape measure. Interviewers were trained in the methods of obtaining both height and weight according to the protocol developed by Add Health staff (Goodman, Hinden and Khandelwal, 2000). Recent research by Goodman and colleagues (2000) using the Add Health survey indicates that while reported weight and height is not a perfect indicator of true height and weight, it is generally quite close to measured height and weight. The authors examine obesity status (obese or not according to an age-sex standard) as reported and measured. They find that BMI based on self-reported height and weight correctly classified 96 percent with regard to true obesity status.

We transform the three measures of BMI for the models since our modeling strategy requires that the dependent variables have a normal distribution. The transformation results in a variable whose distribution is not statistically significantly different than a normal curve using the Shapiro-Wilk *W* test (Shapiro and Wilk, 1965).

There are several indicators of family background used in our analysis. For the family background variables that are continuous measures, we use both siblings' reports to capture the underlying, true value for these variables. For the family background variables that are dichotomous, we use Sibling 1's report of the variable in the model. Given the symmetry in the data, all siblings contribute information on these variables for their family.

Race and Ethnicity are key variables in the present analysis. To capture the race and ethnicity of the family, we categorize sibling pairs as non-Hispanic white, non-Hispanic black, Hispanic, non-Hispanic "other" race (including Asians and Native Americans), and whether the sibling pair is comprised of persons with different racial or ethnic identity. In the models, the omitted category is non-Hispanic whites.

To measure socioeconomic status, we include two variables. First, we include a measure of each sibling's report of their parents' education. For those adolescents with reports for both maternal and paternal education, we average these values to arrive at our final measure of their parents' education. For those persons with only one report of parental education, we use that one report for each variable. Second, we incorporate the parental report of their family income measured in thousands of dollars. We transformed the final values of parental education and family income so that their distributions across individuals in the final sample would approximate a normal curve.

We include family structure indicators for stepparent families, single-parent families and other family structures. The omitted category is for two-parent biological families. We also incorporate indicators for urbanicity measured as suburban (outside a central city but still in a SMSA) and rural (not in a SMSA). The omitted residential category is for urban (central city in a SMSA) residences.

Finally, we also include parental reports of the adolescent's biological parents overweight status. The parental respondent was asked if the biological mother has obesity and if the biological father has obesity. We created three indicators from the categorical weight information for the biological parents: both parents are obese, one parent is obese, and there is missing data on one or both parents' obesity. The omitted

category is both parents are not obese. This variable captures the genetic, dietary, and other behavioral aspects of parental weight that could influence the adolescent's own weight.

Unlike the aforementioned family background variables, we expect the other variables in our analyses to vary within a sibling pair. Since one would expect smoking behavior and disability to be correlated with weight, we include variables for each sibling to represent these two conditions. Our measure for is an indicator for whether the adolescent identified as a regular smoker. Disability is an indicator of whether the adolescent herself or her parents identified her as disabled in the wave 1 in-home survey. Because we know that there are gender and age differences in weight, we include variables for age and gender for each sibling. To facilitate LISREL's calculation of reasonable parameter estimates for the variable age, we have rescaled the variable by dividing by 10.

Research has found that moderate to vigorous activity and inactivity are important proximate determinants of an individual's weight (Andersen et al., 1998). To capture moderate to vigorous physical activity, we have calculated the total number of times a week that the adolescent participated in active sports, bicycling, rollerblading, skating, martial arts, dance and other exercise (Gordon-Larsen, McMurray, and Popkin, 1999)[3]. We then categorize this participation frequency into low, moderate and high based on the distribution of the summary frequency. In the models, the omitted category is for high frequency of moderate to vigorous activity. To capture inactivity, we calculate the

[3] The frequency of participation in these activities is captured in three variables. Unfortunately, the values for these three variables are not continuous, but categorical measures of the frequency of activity during the last week: value of 0 for 0 times, value of 1 for 1-2 times, value of 2 for 3-4 times, and a value of 3 for 5 or more times. Our summation across these three variables is therefore not as clean as one would like.

number of hours spent watching television, watching videos, and playing video or computer games. As in previous research we categorize the hours of inactivity into low (0-10 hours/week), medium (11-24 hours/week), and high (25+ hours/week) (Gordon-Larsen, McMurray, and Popkin, 1999). For the models, the omitted category is low inactivity.

Table 1 provides the labels, descriptions, transformations, means and standard deviations for the variables included in the analyses.

RESULTS

Goodness-of-Fit Testing and Model Selection

The main goal of our structural equation model analysis is to assess the effects of membership in racial and ethnic minority groups on adolescents' weight after controlling for measured and unmeasured sources of variation common to both siblings. We developed several models in which we made incremental additional specifications to our original model. Table 2 contains the goodness-of-fit statistics for alternative models.

We began with the model depicted in Figure 1 and listed in Table 2 as Model 1. In Model 1, the race and ethnicity indicators affect the common family factor of BMI labeled "Family BMI, E1." This common family factor equally affects each sibling's latent BMI. The equality restraint in the effect of the latent family BMI on Sibling 1's latent BMI and the effect of the latent family BMI on Sibling 2's latent BMI reflects the symmetry of the data. The siblings' latent BMI factors are normalized on the wave 2 physical measurements of BMI, as indicated by the parameter path equaling one. Under this sibling model, we recognize that family members resemble each other in

race/ethnicity and other unmeasured characteristics. Model 1 fits poorly according to the bic statistics (Raftery, 1995).

Model 2 adds the other family background variables to the model. These variables include parental education, family income, and indicators for family structure, residence, and parental obesity. For the continuous variables, parental education and family income, Sibling 1 and Sibling 2's responses to survey questions identify these latent common family background variables, but these latent variables are normalized on Sibling 1's responses. For the family background characteristics with dichotomous indicators, Sibling 1's responses identify these factors. Given that there is only one measurement for these dichotomous family background factors, there is no measurement model.[4] The addition of these measured family background variables significantly improves the fit of the model.

In Model 3, the individual-level factors are added to the model. These include the sibling's gender, age, smoking and disability status, activity levels and inactivity levels. This model is represented in Figure 2. The common family factor and these individual-level factors affect the sibling's own latent BMI. These individual variables have perfect measurement because there is only one measure for each latent individual variable. Model 3 fits quite well according to the bic statistics (Raftery, 1995).

In Model 4, we allow all wave 2 measurement errors for BMI to be freely correlated as an occurrence-specific correlation in error. With this additional specification, there is a significant improvement in model fit for the additional 6 degrees

[4] We use only Sibling 1's responses for the dichotomous family background factors even though we also have information from Sibling 2 because the measurement model in LISREL assumes a linear relationship among the latent factor, its measure and its measurement error. See formula 2. A dichotomous measured

of freedom used relative to Model 3. Finally, Model 5 tests whether the paths from the sibling latent BMI variables to both of their wave 2 measures could be set equal to 1.0. In almost all models tested, the estimates of these paths were close to 1.0. This equalization does not lead to a significant loss of fit (with 4 degrees of freedom). Therefore, Model 5 is our preferred model and is represented in Figure 3.

Another means of demonstrating the goodness-of-fit of the model is to calculate the amount of variance in the dependent variable explained under different model specifications. To compare R^2's for the latent BMI explained by a sibling model versus an individual model, we took the following steps. First, using only the individuals that comprise the sibling pairs in our data, we estimate a structural equation model in LISREL where only the race/ethnicity indicators affect an individual's latent BMI, which is measured with the three calculations of BMI. Second, again using only the individuals in the sibling pairs in our final data, we estimate another individual-level structural equation model in LISREL where all the explanatory variables affect an individual's latent BMI. These two specifications ignore the relatedness of the individuals in the data.

Table 3 documents the R^2 for the latent factor of the individual's BMI to indicate the amount of variance in BMI explained under these different formulations. The individual-level structural equation model that only includes the race/ethnicity indicators as independent variables has an R^2 of only 0.01, while the individual model that includes all of the explanatory variables has an R^2 of 0.11.

A sibling model with only race/ethnicity as explanatory variables greatly improves the explained variance of the latent sibling BMI, with a reported R^2 of 0.39.

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variable would violate the model assumption. Given the symmetry of the data, or double-entry, all siblings contribute to the family background latent constructs' measures.

This, taking into account the relatedness of the individuals, on race/ethnicity and other unmeasured factors, helps to explain more of the variance in BMI. With the additional specifications, especially in Models 3 and 5, the R^2 improves. Our final sibling model (Model 5 in Table 2) greatly improves the amount of variation in the latent sibling BMI explained by the model. Our final model results in an R^2 of 0.45 for the sibling variance in BMI.

Parameter Estimates

Table 4 presents the parameter estimates from our preferred and final model, Model 5. The indicators for race/ethnicity prove interesting. After controlling for both the observed and unobserved family background characteristics, blacks do have significantly greater weights than whites, but this difference is only marginally statistically significant. Blacks, on average, have BMI values that are 0.04 points greater than whites. Said differently, being black results in a 0.05 standard deviation increase in BMI. This difference is smaller than estimates found in research that does not control for family background differences. After controlling for unobserved and observed family background characteristics, Hispanics have weights greater than blacks and whites. A Hispanic adolescent, on average, has a BMI that is 0.08 greater than whites, or a 0.11 standard deviation in BMI, controlling for other factors. All other racial groups and those in mixed racial families show no significant differences compared to non-Hispanic whites.

Parental education significantly decreases family-level BMI. Parental education has the largest coefficient of any in the model, even large than gender, age or parental weight, but the size of this coefficient is related to the transformed metrics of BMI and

parental education. Substantively the effects are small. A one-year change in parents' education results in an average 0.0001 decrease in BMI.[5] Despite the small effects, the significance of parental education indicates an important behavioral or informational importance of family characteristics on adolescent weight. Family socio-economic status, as measured by parental education, matters for adolescent weight. On the other hand, family income, as it has been measured in this analysis, does not have a significant impact on adolescent weight, nor does family structure and residence.

Parental obesity significantly impacts adolescent weight. In fact, parental obesity indicators have the greatest substantive effects on BMI of any in the models. Whether the parental obesity indicators capture genetic potential or behavioral similarities, the present research cannot determine, but the impacts of parental weight are clear. If both parents are obese, the adolescent's weight is 0.28 points greater than the adolescent whose parents are not obese. If one parent is obese, the increase in BMI is 0.21 points. Interestingly, if either parent's obesity is unknown or not reported by the parental respondent, then the adolescent has an increase in BMI of 0.11 points. This last difference could reflect the social consequences of family formation histories or adoption.

The individual factors also have important effects on each sibling's BMI. As expected, females have significantly lower BMIs and older adolescents have greater weights than younger adolescents. For each additional year, adolescent BMI increases by approximately 0.13 points.[6] After controlling for other factors, being a smoker or being

[5] Recall that for the analysis, both parental education and BMI are transformed using natural logs. Given the use of logs, the relationship is not linear between BMI and education. The interpretation of the results uses the original metrics of these variables.

[6] Recall that age has been divided by 10 and BMI has been transformed in this analysis. Our interpretation of the results uses the original metrics of the variables. Since BMI has been transformed using the natural log, the relationship between age and BMI is no longer perfectly linear.

disabled is not significantly related to BMI. Moderate to vigorous activity is not significantly related to BMI in this analysis, but levels of inactivity are. Relative to those with low measures of inactivity (0-10 hours/week), those with a moderate amount of time spent inactive (10-24 hours/week) have approximately 0.05 point greater BMIs and those the highest levels of inactivity (25+ hours/week) have BMIs of 0.06 points greater.

The bottom of Table 4 shows estimates of the between- and within-family components of variance in BMI. The between-family component of variance of sibling pairs is 0.027. The within-family variance in BMI is 0.070. Thus, the within-family variance in BMI for each sibling is 72 percent [100*0.070/(0.070+0.027)] of the total variance and only 28 percent of the variance in BMI lies between families.

The key advantage of our analysis is our ability to control for observed and unobserved differences between families that could account for observed racial differences in BMI. Our model explains only 15 percent of the total variation in BMI between families. On the other hand, our individual variables explain approximately 46 percent of the variation in BMI within families. Together these results further imply that this analysis is greatly benefited by the sibling model framework. Although we cannot explicitly account for half of the variation within-families and most of the between-family variation, this unexplained variation is accounted for in the model estimates.

CONCLUSIONS

We employed a sibling resemblance model in an attempt to assess the effects of racial and ethnic membership on adolescent weight after controlling for both observed and unobserved characteristics of families. The importance of this model improvement

lies in the fact that racially different families often experience different social and health-related experiences. Researchers are able to measure and control for these different experiences to differing degrees. Previous research has only relied on the direct measures of some of these differential experiences.

We sought to discover if observed differences in racial and ethnic groups' weight would still be significant after controlling for both observed and unobserved characteristics of families. Our research finds that after controlling for both observed and unobserved family characteristics, black and Hispanic adolescents have significantly greater weights than whites, though the difference is modest. Other minorities do not show statistically significant differences in weight relative to whites. In addition, children from families with higher educated parents have significantly lower weights. As one would expect, parents' obesity is an important family background factor for adolescent's own weight. Age and gender also have strong measured effects on an individual's BMI. Finally, inactivity leads to greater weights among adolescents.

Although this model provides a significant improvement on past research, we propose future research that goes beyond the present framework. First, we propose to more explicitly address the concerns of behavioral geneticists. Our present model combines both genetic and environmental common family factors. In additional analyses, we could perform a multiple groups model with each relationship type as a separate group. Therefore, we could test whether there are significant differences in the effects of social factors, including racial membership, across sibling relationship types. Second, most of the research on racial and ethnic differences in BMI finds interactions in the effects of race/ethnicity with gender. Our present model treats race and gender

additively. In future analyses, we propose to do a multiple groups model of different gender pairs of siblings to test for gender interaction in the effects of racial and ethnic membership on weight.

We look forward to future research in this area. With the rapid increase in the prevalence in childhood and adolescent obesity in recent decades, we need research that helps to explain the social conditions that account for this increase. Furthermore, analyses recognizing the social relationships within data sources will allow us to better model and understand how families and social contexts facilitate or hinder healthy characteristics in children, including weight.

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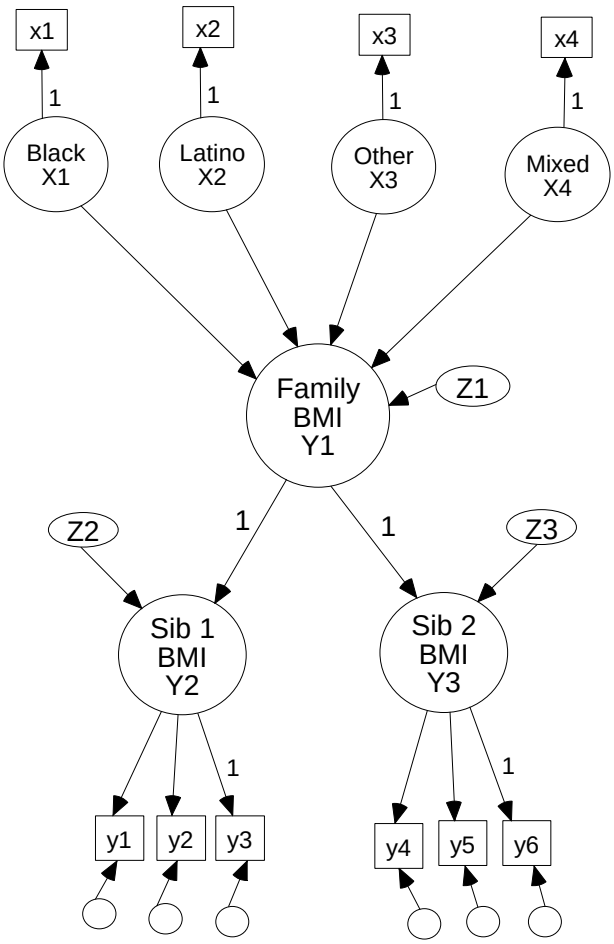
Figure 1. Sibling Structural Equation Model for Adolescent BMI:

Only Race/Ethnicity Explanatory Variables



Note: X1-X4 are intercorrelated

Figure 2. Sibling Structural Equation Model for Adolescent BMI:

Original Model with all Explanatory Variables

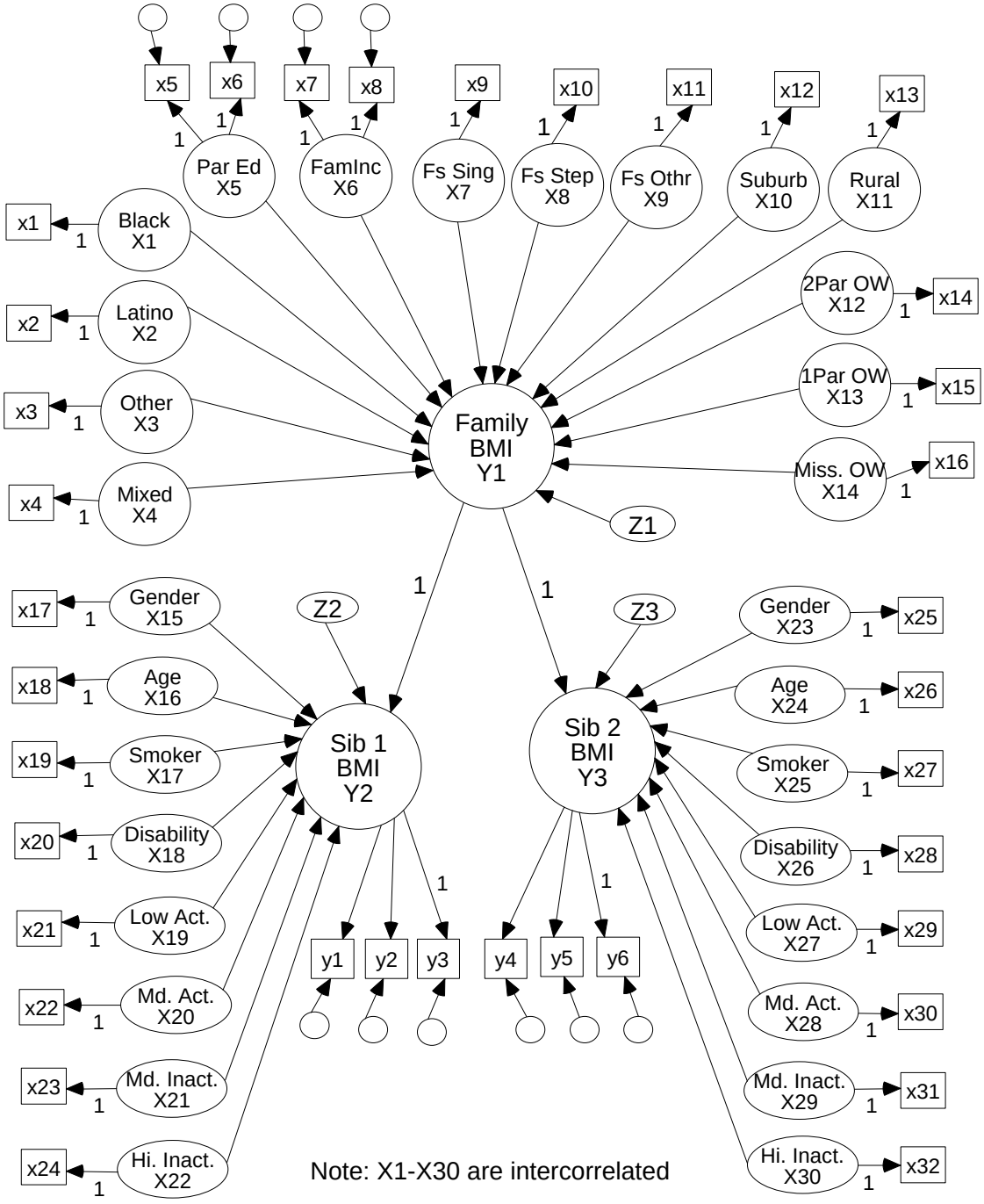


Figure 3. Sibling Structural Equation Model for Adolescent BMI: Final Model

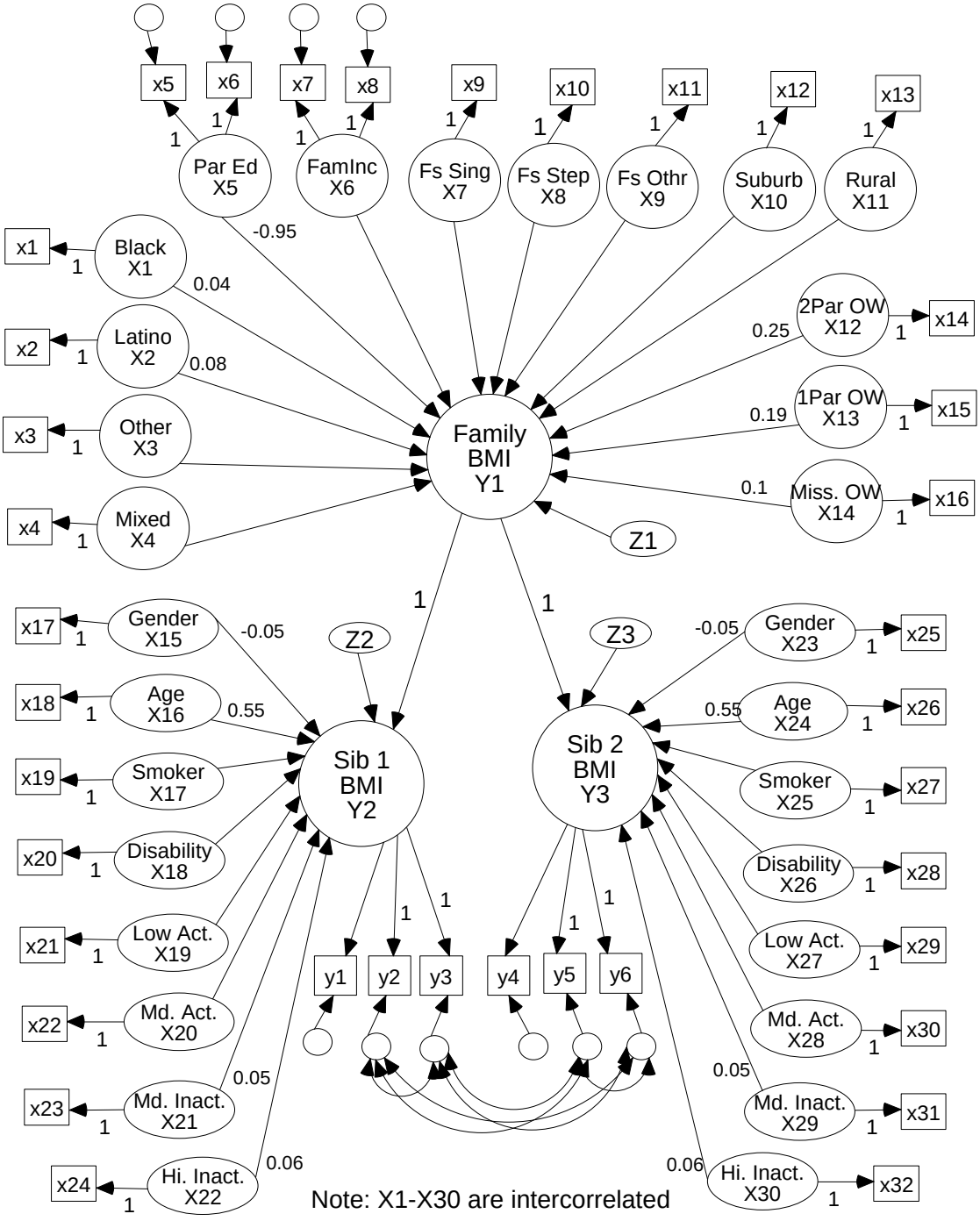


Table 1. Variable Descriptions, Transformations, Means, and Standard Deviations

Sibling 1	Sibling 2	Original Variable Description	Variable Transformation	Original metric Weighted[a] Mean	Original metric Weighted[a] St.Dev.	Model metric Unweighted Mean	Model metric Unweighted St.Dev.
Dependent Variables for BMI Factors							
y1	y4	BMI (weight/height^2) from sibling reports, wave 1	ln(BMI - 13.48)	22.42	151.99	2.07	0.49
y2	y5	BMI (weight/height^2) from sibling reports, wave 2	ln(BMI - 13.48)	23.02	156.48	2.13	0.47
y3	y6	BMI (weight/height^2)from physical measurements, wave 2	ln(BMI - 13.48)	22.98	164.14	2.12	0.49
Independent Variables for Family Background Factors							
x1	---	Pair is non-Hispanic Black (= 1)	---	0.15		0.20	
x2	---	Pair is Hispanic (= 1)	---	0.08		0.13	
x3	---	Pair is non-Hispanic, "other" race (= 1)	---	0.06		0.06	
x4	---	Pair is mixed race/ethnicity (= 1)	---	0.03		0.05	
x5	x6	Average parents' education in years	ln(educ. + 84.33)	13.31	74.33	4.58	0.02
x7	x8	Family Income in 1995 thousands of dollars	ln(inc - 7.19)	44.21	1511.72	3.75	0.64
x9	---	Family is a single parent household (= 1)	---	0.22		0.27	
x10	---	Family is a step-parent household (= 1)	---	0.14		0.16	
x11	---	Family structure is another, non-traditional family type (= 1)	---	0.04		0.04	
x12	---	Residence is suburban (= 1)	---	0.38		0.38	
x13	---	Residence is rural (= 1)	---	0.33		0.26	
x14	---	Both biological parents are obese (= 1)	---	0.06		0.05	
x15	---	One biological parent is obese (= 1)	---	0.14		0.15	
x16	---	Missing data for either parents' obesity (= 1)	---	0.15		0.18	
Independent Variables for Individual Factors							
x17	x25	Sibing is female (= 1)	---	0.49		0.50	
x18	x26	Age of sibling at wave 1	age/10	15.44	5.87	1.55	0.17
x19	x27	Sibling identifies as a regular smoker (= 1)	---	0.21		0.18	
x20	x28	Sibling has a physical disability (= 1)	---	0.02		0.03	
x21	x29	Sibling performs a low level of vigorous activity (= 1)	---	0.27		0.28	
x22	x30	Sibling performs a medium level of vigorous activity (= 1)	---	0.36		0.36	
x23	x31	Sibling has medium levels of inactivity (= 1)	---	0.33		0.33	
x24	x32	Sibling has high levels of inactivity (= 1)	---	0.35		0.33	

Source: Authors' computations with unweighted and weighted data from the National Survey of Adolescent Health, waves 1 and 2.
a: Weighted statistics are calculated using wave 2 sample weights.

Table 2: Model Fit Statistics for Sibling Model

	Model	N	df	Minimum Fit L^2	bic
1	Only Race/Ethnicity Variables	2808	28	101.744	-120.58
2	Only Family Background Variables	2808	115	182.131	-731.00
3	All variables: BE(2,1)=BE(3,1)=1, all LX's=1	2808	227	467.427	-1,335.00
4	3 + corr error in all wave 2 BMI	2808	221	326.730	-1,428.06
5	4 + LY's for wave 2 all equal to 1	2808	225	369.415	-1,417.14

Source: Authors' computations using the National Survey of Adolescent Health

Table 3: Explanatory Power of Individual and Sibling Models for the Latent BMI Factor

	R^2
Individual Models	
Race/Ethnicity Variables Only	0.009
All Variables	0.111
Sibling Model	
Race/Ethnicity Variables Only (Model 1)	0.391
Family Background Variables Only (Model 2)	0.390
All Variables (Model 3)	0.432
Final Model (Model 5)	0.452

Source: Authors' computations using the National Survey of Adolescent Health

Note: The R^2 reported for the Sibling Models is for the 2nd order factor, the latent sibling BMI.

Table 4. Selected Parameters of Final Model: Model 5

	LISREL Labels	Estimate	St. Error	Standardized Estimate
Effects of Family Background on Common Family Factor				
Race/Ethnicity (Omitted: White Pair)				
Black Pair	GA(1,1)	0.035	0.019 [c]	0.052
Hispanic Pair	GA(1,2)	0.077	0.021 [a]	0.102
Other, Same-group Pair	GA(1,3)	-0.050	0.031	-0.040
Mixed Pair	GA(1,4)	0.017	0.030	0.014
Socioeconomic Status				
Parental Education	GA(1,5)	-0.953	0.335 [a]	-0.087
Family Income	GA(1,6)	-0.004	0.013	-0.011
Family Structure (Omitted: Two-Parent, Biological)				
Single Parents	GA(1,7)	0.005	0.017	0.008
Step Parents	GA(1,8)	0.009	0.551	0.014
Other Family Type	GA(1,9)	0.015	0.403	0.010
Urbanicity (Omitted: Urban)				
Suburban	GA(1,10)	-0.016	0.015	-0.033
Rural	GA(1,11)	-0.007	0.017	-0.012
Parental Obesity (Omitted: Neither Obese)				
Both Obese	GA(1,12)	0.249	0.028 [a]	0.217
One Obese	GA(1,13)	0.193	0.017 [a]	0.286
Missing Data Flag	GA(1,14)	0.103	0.022 [a]	0.116
Effect of Latent Family BMI on each Sibling's Latent BMI	BE(2,1) & BE(3,1)	1.000	-	0.625
Effects of Individual Characteristics on each Sibling's Latent BMI				
Gender (1=Female)	GA(2,15) & GA(3,23)	-0.054	0.014 [a]	-0.067
Age	GA(2,16) & GA(3,24)	0.545	0.043 [a]	0.229
Smoker	GA(2,17) & GA(3,25)	0.003	0.018	0.003
Disabled	GA(2,18) & GA(3,26)	0.050	0.042	0.020
Moderate to Vigorous Activity (Omitted: High Frequency)				
Low Frequency	GA(2,19) & GA(3,27)	0.002	0.018	0.003
Medium Frequency	GA(2,20) & GA(3,28)	0.007	0.016	0.008
Hours of Inactivity (Omitted: Low)				
Medium	GA(2,21) & GA(3,29)	0.050	0.017 [a]	0.059
High	GA(2,22) & GA(3,30)	0.059	0.018 [a]	0.069
Total Between-Family Variance		0.027		1.000
Unmeasured Between-Family Variance	PS(1)	0.023	0.001 [a]	0.851
Total Within-Family Variances		0.070		1.000
Unmeasured Within-Family Variances	PS(2) & PS(3)	0.038	0.002 [a]	0.544

Source: Authors' computations using symmetric sibling pairs, National Survey of Adolescent Health

P-value significance: a: $p < 0.01$, b: $p < 0.05$, c: $p < 0.10$

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