Behavior Genetics, Vol. 28, No. 4, 1998

Genetic and Shared Environmental Influences on Adolescent BMI: Interactions with Race and Sex

Kristen C. Jacobson[1,3] and David C. Rowe[2]

Received 14 Oct. 1997—Final 15 May 1998

The present study uses a behavioral genetic design to investigate the genetic and environmental influences on variation in adolescent body mass index (BMI) and to determine whether the relative influences of genetic and environmental factors on variation in BMI are similar across racial groups and sexes. Data for the present study come from the National Longitudinal Study on Adolescent Health (Add Health), a large, nationally representative study of adolescent health and health-related behaviors. The Add Health sample contains a subset of sibling pairs that differs in levels of genetic relatedness, making it well suited for behavioral genetics analyses. The present study examines whether genetic and environmental influences on adolescent BMI are the same for males and females and for Black and White adolescents. Results indicate that genetic factors contribute substantially to individual differences in adolescent BMI, explaining between 45 and 85% of the variance in BMI. Furthermore, based on an analysis of opposite-sex sibling pairs, the genes that influence variation in adolescent BMI are similar for males and females. However, the relative importance of genetic and environmental influences on variation in BMI differs for males and females and for Blacks and Whites. Although parameter estimates could be constrained to be equal for Black and White males, they could not be constrained to be equal for Black and White females. Moreover, the best-fitting model for Black females was an ADE model, for White females it was an ACE model, and for males it was an AE model. Thus, shared environmental influences are significant for White female adolescents, but not for Black females or males. Likewise, nonadditive genetic influences are indicated for Black females, but not for White females or males. Implications of these results are discussed.

KEY WORDS: Genetics; body mass index; adolescents; race; sex.

INTRODUCTION

A considerable amount of research has investigated the role of genetics and shared and nonshared environment in the variability of relative weight as defined by body mass index (BMI; see reviews by Grilo and Pogue-Geile, 1991; Maes *et al.*, 1997; Meyer, 1995). Results from these studies indicate that relative weight is heritable, with heritability estimates obtained from twin studies typically ranging from .50 to .90 (e.g., Allison *et al.*, 1994a, b; Bodurtha *et al.*, 1990; Hunt *et al.*, 1989). For example, the correlations among MZ twins raised apart average between .60 and .74 for BMI (Price and Gottesman, 1991; Stunkard *et al.*, 1990) and .86 and .73 for height and weight, respectively (Bouchard *et al.*, 1990). Moreover, results from

[1] Department of Human Development and Family Studies, Pennsylvania State University, University Park, Pennsylvania 16802.
[2] Department of Family Studies, University of Arizona, Tucson, Arizona 85721.
[3] To whom correspondence should be addressed at 210 FCR Building, P.O. Box 210033, University of Arizona, Tucson, Arizona 85721-0033. Fax:520-621-3401. e-mail: kjacobso@u.arizona.edu.

0001-8244/98/0700-0265$15.00/0

adoption studies also find strong heritabilities for relative weight, although the estimates tend to be lower (e.g., Sorensen and Stunkard, 1993; Stunkard *et al.*, 1986; Vogler *et al.*, 1995; for a review also see Maes *et al.*, 1997). Furthermore, the behavioral genetic results indicate that shared environment has little, if any, influence on the variability of relative weight. Specifically, shared environmental influences have had little impact on the variation in relative weight in both twin samples (e.g., Allison *et al.*, 1994a, b; Herskind *et al.*, 1996) and adoptive samples (e.g., Vogler *et al.*, 1995). Only one study has revealed a significant estimate of shared environment, with shared environmental factors accounting for between 14 and 18% of the variation in adult BMI among male twins (Hunt *et al.*, 1989).

However, current research has a number of important limitations. First, a majority of twin and adoption studies of BMI and relative weight has focused on adult samples or spanned a broad age range. It is possible that shared environment exerts a stronger influence on relative weight during adolescence, when siblings reared together are experiencing shared environment directly (Grilo and Pogue-Geile, 1991). For example, Khoury *et al.* (1983) report greater familial resemblance for relative weight among their pediatric sample compared to their adult sample. However, the Khoury *et al.* study could not separate out genetic effects from shared environmental effects, thus the greater resemblance could be due to either greater heritability among the pediatric sample and/or a greater effect of shared environmental factors. Although studies of personality and intelligence typically find that heritability estimates increase across the life span (McGue *et al.*, 1993; Plomin, 1986, 1990), it appears that the heritability of relative weight might actually *decrease* with age. Specifically, a number of both cross-sectional and longitudinal studies have found lower heritabilities for relative weight for older adults (e.g., Carmichael and McGue, 1995; Herskind *et al.*, 1996, for males only). However, it should be noted that these lower heritabilities may be a reflection of the greater relative influence of nonshared environmental factors, rather than an absolute decrease in genetic effects (Carmichael and McGue, 1995; Hewitt, 1997).

Although data are scarce, results from the handful of published studies of BMI and obesity in adolescence do indicate that genetics play an important role in the variation in adolescent BMI and that shared environment effects are negligible (e.g., Allison *et al.*, 1994a; Bodurtha *et al.*, 1990; Kaprio *et al.*, 1995). Moreover, results from three additional unpublished twin studies of adolescents (presented by Maes *et al.*, 1997) also demonstrate heritabilities in the .67 to .93 range. Similarly, these studies also provide no evidence for shared environmental effects (for a review see Maes, *et al.*, 1997). Nonetheless, more studies of genetic and environmental influences on variation in *adolescent* BMI are needed.

A second limitation of the previous research is that only a few studies have had sample sizes large enough to detect nonadditive genetic effects (Maes *et al.*, 1997). Of the studies that have been able to examine the relative contributions of both additive and nonadditive genetic effects, some have determined that nonadditive genetic effects play a sizable role in explaining individual differences in BMI (Allison *et al.*, 1994b; Martin, cited by Neale and Cardon, 1992; Stunkard *et al.*, 1990), whereas others have found that the more parsimonious model that includes only additive genetic influences and nonshared environmental influences (the AE model) fit better than one that also includes nonadditive genetic influences (the ADE model; e.g., Allison *et al.*, 1994a; Carmichael and McGue, 1995; Fabsitz *et al.*, 1994; Herskind *et al.*, 1996; Kaprio *et al.*, 1995). An additional study of Finnish adult twins found that dominance effects were important for female twins but not for male twins (Korkeila *et al.*, 1995). Thus, the debate over the importance of nonadditive genetic influences remains unresolved.

In a related vein, the possible presence of nonadditive genetic effects may mask smaller effects of shared environment. Specifically, the classic twin study is unable to assess the effects of nonadditive genetic influences and shared environmental influences simultaneously. Although most twin studies do compare goodness-of-fit statistics between models that include shared environmental influences but not nonadditive genetic influences (the ACE model) and models that include nonadditive genetic influences but not shared environmental influences (the ADE model) and conclude that nonadditive genetic factors are more important than shared environmental factors, multiple sibling group studies are needed to definitively address the issue of *concurrent* nonadditive genetic and shared environmental influences on variation in BMI.

Furthermore, the debate over sex differences in the relative influences of genetic and environmental influences on variation in BMI is still undecided. To date, some studies have revealed gender differences in the heritability of relative weight, but results are mixed. Specifically, some studies have found heritability estimates to be higher for males (e.g., Korkeila *et al.*, 1995; Stunkard *et al.*, 1990). Other studies have found heritability to be higher for females (Allison *et al.*, 1994b). Still others find no gender differences (e.g., Allison *et al.*, 1994a; Bodurtha *et al.*, 1990; Martin, cited by Neale and Cardon, 1992). Similarly, not all twin studies include male–female DZ twins (Bodurtha *et al.*, 1990). Male–female sibling pairs are needed in order to determine whether the same set of genes influences variation in BMI. Most studies that include opposite-sex twin pairs do find that the correlations among opposite-sex DZ twins are lower than the correlations between same-sex DZ twins (e.g., Allison *et al.*, 1994b; Meyer, cited by Neale and Cardon, 1992), indicating that genetic factors and/or shared environmental factors are different for males and females. Thus, research that includes both same-sex and opposite-sex sibling pairs is needed.

Finally, few behavior genetic studies of relative weight have been conducted using non-Caucasian samples. Black women, in particular, are typically heavier than White women and are overrepresented in the proportion of obese adults (Dawson, 1988; Greenlund *et al.*, 1996; Hsu, 1996; le Grange *et al.*, 1998). Moreover, a greater incidence of obesity-related disorders such as diabetes, hypertension, and heart disease is found among Blacks (for a review see Kumanyika, 1987). Thus, an examination of the factors related to variation in relative weight for Blacks, especially Black women, is needed. On the one hand, it has been suggested that the greater incidence of obesity among Black women may be due to environmental factors, such as cultural differences in standards of beauty or differences between Whites and Blacks in social class (Kumanyika, 1987). However, empirical support for the cultural differences hypothesis is lacking (e.g., Allison, 1992). Likewise, race differences in BMI remain even after controlling for social class (Dawson; 1988; Rand and Kuldau, 1990), suggesting that other factors are contributing to the greater mean levels of BMI among Black women. On the other hand, researchers typically acknowledge that genetic differences in Blacks and Whites and/or racial differences in the relative influences of genes and environment on variation in BMI may also be partly responsible for the mean level differences found among racial groups (Guillame and Bjorntorp, 1996; Kumanyika, 1987). Thus, behavioral genetic studies that include both Blacks and Whites are needed.

We know of only one study that has directly examined racial differences in the heritability of BMI (Allison *et al.*, 1994a). This study found greater genetic variation in relative weight for Black adolescents than for White adolescents. However, the *heritabilities* of relative weight were about the same because the total variance in relative weight was also greater for Blacks than for Whites.[4] In a similar vein, segregation analyses revealed that the mode of genetic inheritance of overweight was similar among Black and White adults (Ness *et al.*, 1991). However, in the Ness *et al.* (1991) study, shared environmental influences were considered to be part of the estimate of polygenic heritability. Thus, more research is needed in order to determine whether racial differences in heritabilities and shared environmental influences exist.

In summary, the present study intends to expand on previous research in the following ways: First, the present study allows us to assess genetic and environmental influences on variation in BMI in a sample of adolescents that is broadly representative of all adolescents in the United States. Second, by using multiple sibling groups (i.e., twins, full siblings, half-siblings, and unrelated siblings living together), it is possible to examine the effects of *both* nonadditive genetic factors and shared environmental factors on variation in BMI concurrently. Third, the present study also examines the moderating effects of sex on the relative influences of genes and environment on variation in adolescent BMI. Moreover, the genetic correlation between opposite-sex pairs is estimated, in order to determine whether the same genes contribute to variation in BMI for both males and females. Finally, we also examine how race moderates the effects of genetic and environmental influences on the variation in adolescent BMI. Due to our relatively large sample size, the potential moderating

[4] Heritability is defined as V_g/V_p, where V_g is the genetic variation and V_p is the phenotypic variation. Thus, if *both* V_g and V_p change between racial groups, their ratio can *still* remain constant.

effects of both race and sex can be examined simultaneously.

METHODS

The Add Health Sample

The present study uses data from the National Longitudinal Study on Adolescent Health (Add Health), a nationally representative, longitudinal study of adolescent health and health-related behaviors and the causes and consequences of these behaviors. Add Health consists of a total sample of over 90,000 adolescents surveyed in school. Almost one-quarter of the total respondents (N = 20,745) were interviewed at home to obtain more detailed information regarding their health and health-related behaviors. Likewise, over 17,000 adolescents from the initial home interview sample were recontacted 1 year later for a second home interview. Data are also available from parent interviews and school administrator interviews. Furthermore, information on community characteristics and adolescent peer networks is also available. The present study uses data from the Wave 1 home interview only. Additional details on sample selection and procedures are given by Resnick *et al.* (1997).

The Add Health sample contains a wide range of socioeconomic statuses and a variety of racial and ethnic groups. Specifically, a number of typically understudied groups were oversampled, including, for example, adolescents from well-educated Black families, Chinese adolescents, Japanese adolescents, and Puerto Rican adolescents. Included in the Add Health sample is a sub-sample of sibling pairs, used in the present analyses. More detailed information concerning the Add Health sample and study design can be found on the Add Health Web-site: http://www.cpc.unc.edu/addhealth/.

The Sibling Pairs Sample

The sibling pairs sample was selected using information from school rosters. Specifically, all adolescents who reported a twin, half-sibling, or unrelated sibling aged 11–20 residing in the same household were selected for inclusion in the home interview subsample. Home interview data were obtained from both the target adolescent and his/her sibling. These sibling pairs were selected regardless of whether they were present on the day of the school interview, and regardless of whether the siblings attended the same school. An additional probability sample of full sibling pairs was also selected for the home interview. Thus, the Add Health pairs sample consists of 783 twin pairs, 1252 full sibling pairs, 442 half-sibling pairs, and 662 unrelated sibling pairs, resulting in a total sample size of 3139 pairs, or 7278 adolescents. Final determination of pair type was based on household roster information from the in-home questionnaire.

Twins' zygosity was diagnosed primarily on the basis of both self-report of zygosity and self-report of confusability of physical appearance. Eighty-nine twin pairs of uncertain diagnosis were classified on the basis of molecular genetic markers. Twins were diagnosed as monozygotic (MZ) if they were the same genotype for five or more genetic makers (error rate about 4/1000 or less) and dizygotic (DZ) if they were different at one or more markers. These two methods (i.e., self-report and genetic markers) resulted in a determination of 289 MZ twin pairs and 451 DZ twin pairs. We were unable to determine the zygosity of an additional 43 twin pairs. These undecided twin pairs (UD) reported that they were DZ but were classified as MZ according to self-report of confusability of appearance. These twin pairs (1.4% of the total sibling pairs sample) were deleted from the present analyses, as combining them with the DZ pairs would most likely overestimate the influence of shared environment.

The present study also includes only those sibling pairs who indicated that they were either White (1576 pairs; 50.2% of the total sibling pairs sample) or Black (754 pairs; 24.0% of the total sibling pairs sample). Although the Add Health sibling pairs sample contains a number of adolescents from less common ethnic and racial groups (e.g., Chinese, Japanese, Hispanic, Puerto Rican, and Native American), the number of pairs for any one given ethnic or minority group was too small to permit separate analyses. Because racial and ethnic differences have been found in mean levels of adolescent body mass index (Dawson, 1988), the inclusion of these adolescents would contribute substantially to variance heterogeneity in the sample. Thus, these adolescents (809 pairs; 25.8% of the total sample) were deleted from the present analyses.

We also deleted 57 pairs (1.8% of the total sample) who we identified as outliers based on the

Table I. Sibling Correlations for Adolescent BMI

Group	Female–female pairs		Male–male pairs		Male–female pairs	
	N pairs	*r*	*N* pairs	*r*	*N* pairs	*r*
Whites						
MZ Twins	66	.73****	74	.85****		
DZ Twins	56	.54****	76	.39****	100	.18*
Full Siblings	199	.48****	184	.37****	279	.36****
Half-Siblings	43	.52****	44	.21	84	.05
Unrelated Siblings	62	.31**	47	.19	94	−.07
Blacks						
MZ Twins	25	.88****	29	.79****		
DZ Twins	31	.27	26	.39**	49	.21
Full Siblings	58	.36**	44	.08	86	.26**
Half-Siblings	40	.26	31	.40**	71	.09
Unrelated Siblings	20	.14	24	.02	23	−.09

Note. *$p < .10$; **$p < .05$; ***$p < .01$; ****$p < .001$.

frequency distributions of adolescent BMI. Specifically, sibling pairs in which one or more adolescent reported a BMI of less than 15 or greater than 39 were deleted from the present analyses. Likewise, missing data for adolescent BMI were found for 77 pairs; naturally these adolescents were also deleted. Finally, the unrelated sibling group contained a subset of sibling pairs whose relationships were not exactly *sibling* relationships. For example, we identified 53 ''sibling pairs'' who were actually aunt/uncle–nephew/niece pairs, boyfriends–girlfriends living together, or unrelated adolescents residing in a group home for delinquent children. Moreover, we identified 201 cousin pairs residing in the same household. All of these 254 pairs were deleted from the present analyses. Although we could have included the cousin pairs as a separate class of siblings (with a coefficient of genetic relatedness = 0.125), the numbers of White cousin pairs were quite small (i.e., nine female–female cousin pairs, eight male–male cousin pairs, eight male–female cousin pairs). Moreover, it is unclear why cousins would be residing in the same household, and it is likely that the time in which they lived together was of a shorter duration compared to, for example, adoptive siblings or step siblings.

In conclusion, the sibling pairs sample used in the present analyses consists of 1965 sibling pairs (62.6% of the total sibling sample; 84.3% of the Black and White adolescent siblings). The actual number of pairs in each sibling group is shown in Table I (with numbers presented separately by race and sex-type). In general, the Add Health pairs sample is similar to the full Add Health sample with respect to certain demographic characteristics. For example, both the full sample and the pairs sample are evenly divided across gender (percentage male = 50). The mean age of both the full sample and the pairs sample is approximately 16. Finally, the percentages of the three largest racial groups in the Add Health Study are also similar for the full sample and the pairs sample (50% White, 21–23% Black, and 9% Hispanic).

Procedure

Data for the present study are from the Wave 1 home interviews, conducted between April 1995 and December 1995. Trained assistants visited adolescents in their homes to administer the interview via laptop computer. Interviews typically took 1 to 2 h. Providing for greater confidentiality and accuracy of response, the adolescents entered their responses on the laptop computers and, for a portion of the interview, could hear the questions read through headphones. All adolescents received the same interview. Whenever possible, siblings were interviewed on the same day.

Measures

Body Mass Index. Adolescents reported their current weight and height. Previous research has shown that self-reports of height and weight are strongly correlated with objective measures (e.g., Stunkard and Albaum, 1981). Reports of adolescent

weight and height were then transformed into kilograms and meters, respectively. Using the traditional formula (kg/m^2), BMI was calculated. Because the distribution for BMI was positively skewed, a log-transformation was used. Because age similarity among MZ and DZ twins might artificially inflate twin correlations, the log-transformed BMI scores were then regressed onto age and age.[2] These regressions were performed separately within racial and sex groups. The transformed, residualized BMI scores were then used in all analyses.

Race. Sibling pairs were coded as either White or Black, based on self-reports.

Sex. Sex was determined by the interviewer. For model-fitting analyses, male–female sibling pairs were reordered so that sibling 1 was always male.

RESULTS

Sibling Correlations for Adolescent BMI

To begin with, within pair correlations were computed for the residualized, log-transformed BMI scores. Table I shows these within-pair correlations for each sibling group, by race and sex-type. The first item of note is that genetic influences on sibling resemblance for BMI are indicated because, in general, correlations between siblings increase as the level of genetic relatedness increases. Second, it appears that either genetic and/or shared environmental influences differ for males and females, in that sibling correlations for opposite-sex siblings are lower than those for same sex siblings. Third, there is some evidence for nonadditive genetic influences, particularly among Black female adolescents, as the MZ twin correlation is more than twice as great as the DZ and full sibling correlations. Finally, the sibling correlations also indicate some shared environmental influences, especially for White female adolescents, because the correlation between unrelated siblings is significant.

Model-Fitting Analyses

The Importance of Genetic and Environmental Influences. Next, data were analyzed using structural equation modeling (Neale and Cardon, 1992) through the statistical package *Mx* (Neale, 1997). To investigate the relative importance of genetic and environmental factors in the variation of adolescent BMI, we ran a series of hierarchically nested models. We started with the ACDE model, with the latent variables A, C, D, and E representing additive genetic factors, shared environmental factors, nonadditive genetic factors, and nonshared environmental factors, respectively. The parameter estimates for additive genetic influences, shared environmental influences, nonadditive genetic influences, and nonshared environmental influences (*a*, *c*, *d*, and *e*, respectively) were estimated separately for each of the four racial and sex groups. Moreover, the coefficient of genetic relatedness for additive genetic influences (r_g) was estimated for male–female DZ twins and full siblings (within the boundaries of 0 and 0.5), in order to allow for different genes affecting BMI for males and females. Likewise, the coefficient of genetic relatedness for nonadditive genetic influences (r_{dg}) was allowed to vary between 0 and 0.25 for male–female DZ twins and full siblings.[5] Finally, because *Mx* uses maximum likelihood to estimate parameters, all parameters were constrained to be equal to or greater than 0. This boundary constraint ensured that the parameter estimates for both males and females would be positive (Neale, 1997).

In order to determine the importance of additive genetic, nonadditive genetic, and shared environmental influences, we tested whether systematically dropping the *a*, *c*, or *d* parameters resulted in a poorer fitting model. All models, were fit to variance–covariance matrices. Table II shows the goodness of fit statistics for each of the six models tested, as well as the change in chi-square relative to the full ACDE model. Not surprisingly, given the results from previous research, models that did not include genetic variance (the CE and E models) fit the data quite poorly. Moreover, the change in chi-square for each of these models was highly significant, indicating that genetic factors cannot be dropped from the model. Somewhat

[5] The coefficient of genetic relatedness for additive genetic influences (r_g) among male–female half-siblings and unrelated siblings was constrained to be a fraction of the r_g estimated for male–female DZ twins and full siblings. Specifically, the r_g for male–female half-siblings was set to equal one-half of the estimated r_g for DZ twins and full siblings. The estimate of r_g for unrelated siblings, of course, was constrained to equal 0. Likewise, the coefficient of genetic relatedness for nonadditive genetic influences (r_{dg}) was constrained to equal 0.0 for half-siblings and unrelated siblings.

Table II. Results from Hierarchically Nested Models

Model	χ^2	df	AIC	RMSEA	$\Delta\chi^2$	df
ACDE	83.2	64	−44.8	.064	—	—
ACE	85.7	70	−54.3	.063	2.5	6
ADE	98.3***	68	−37.7	.075	15.1***	4
AE	100.4**	74	−47.6	.077	17.2*	10
CE	229.7****	76	77.7	.136	146.5****	12
E	477.0****	80	317.0	.219	393.8****	16

Note. AIC, Akaike's information criterion; RMSEA, root mean square error approximation. *$p < .10$; **$p < .05$; ***$p < .01$; ****$p < .001$.

more surprising was the finding that the models that did not include shared environmental variance (the ADE and AE models) also fit the data quite poorly. Furthermore, the change in chi-square for the ADE model was significant, and the change in chi-square for the AE model approached significance, suggesting that shared environmental factors are important for variation in adolescent BMI. The comparison of the ACE model to the ACDE model suggests that nonadditive genetic factors may not have a significant influence on variation in adolescent BMI, given that the change in chi-square was not significant. Generally, we would retain the ACE model, as it is the best-fitting, most parsimonious model [this can be seen from the more negative AIC value, which is an estimate of both goodness of fit and parsimony (Akaike, 1987; Neale, 1997)]. However, given that the primary purpose of the present study was to investigate whether genetic and environmental influences could be constrained across sexes and races, we decided to retain the more complex ACDE model until racial and gender similarities and differences could be ascertained.

Same Genes or Different Genes? As mentioned above, the estimated coefficient of genetic relatedness for additive genetic influences and nonadditive genetic influences for male–female pairs (r_g and r_{dg}, respectively) is an indication of whether the same genes contribute to variation in adolescent BMI for males and females (Bodurtha *et al.*, 1990). If r_g differs significantly from 0.5 for male–female DZ twins and full siblings and/or r_{dg} differs significantly from 0.25 for male–female DZ twins and full siblings, then the hypothesis that the same genes operate in male and female adolescents can be rejected. The estimated r_g for male–female pairs in the full ACDE model was 0.5 and the estimated r_{dg} for male–female pairs was 0.25 for both Black and White adolescents, suggesting that the genes that influence adolescent BMI are not different for males and females. Fixing the opposite-sex r_g parameter to equal 0.5 and the opposite-sex r_{dg} parameter to equal 0.25 for both Blacks and Whites did not result in a poorer-fitting model ($\Delta\chi^2 = 0.61$, df = 4, $p < .999$). Thus, the r_g parameter for opposite-sex pairs was constrained to equal 0.5 and the r_{dg} parameter for opposite-sex pairs was constrained to equal 0.25 for all subsequent analyses.

Race and Sex Differences. The next series of analyses investigated whether genetic and environmental components could be constrained to be equal across sexes and/or races. Table III shows the goodness-of-fit statistics and changes in chi-square (relative to the full model) for each of the models tested. The ACDE model with separate parameters for males, females, Blacks, and Whites was used as the base model for comparison. The first model (Model 1) examined whether the same set of parameters could be used to explain variance in BMI for all four racial and sex groups. This model fit the data quite poorly, and the significant change in chi-square suggests that genetic and environmental contributions to BMI do, in fact, vary across races and sexes.

The next model (Model 2) investigated whether parameters could be constrained to be equal across sexes, but not races (i.e., Black females = Black males, and White females = White males). This model also fit the data poorly, moreover, the significant change in chi-square suggests that this model did not fit the data as well as the full model. Submodels of Model 2 determined whether parameters could be constrained to be equal across sexes for White adolescents only and for Black adolescents only (Models 2a and 2b, respectively). These models also fit the data poorly and resulted in significant changes in chi-squares, indicating that sex differences exist for both Black and White adolescents.

Model 3 tested whether parameters could be constrained across races, but not sexes (i.e., Black females = White females, and Black males = White males). This model did not fit the data well, as indexed by both the significant chi-square and the significant change in chi-square. Submodels of Model 3 examined whether parameters could be constrained across races for females only and for males only. Although the model constraining

Table III. Results from Hierarchically Nested Models Constraining Parameters to be Equal Across Race and Sex

Model	χ^2	df	AIC	RMSEA	$\Delta\chi^2$	df
Base model (all varies) (BF ≠ WF ≠ BM ≠ WM)	83.84	68	−52.2	.064	—	—
1. All equal (BF = BM = WF = WM)	125.57***	80	−34.4	.085	41.7***	12
2. Sexes equal (BF = BM vs. WF = WM)	114.98**	76	−37.0	.082	31.1***	8
2a. Sexes equal, Whites only (WF = WM vs. BF vs. BM)	103.59**	72	−40.4	.076	19.8***	4
2b. Sexes equal, Blacks only (BF = BM vs. WF vs. WM)	95.22*	72	−48.7	.072	11.4*	4
3. Races equal (BF = WF vs. BM = WM)	105.42**	76	−46.6	.074	21.6**	8
3a. Races equal, females only (BF = WF vs. BM vs. WM)	100.80*	72	−43.2	.071	17.0***	4
3b. Races equal, males only (BM = WM vs. BF vs. WF)	**86.72**	**72**	**−57.3**	**.065**	**2.9**	**4**

Note. The best-fitting model is indicated in boldface. BF, black females; WF, white females; BM, black males; WM, white males; AIC, Akaike's information criterion; RMSEA, root mean square error approximation. *$p < .05$; **$p < .01$; ***$p < .001$.

parameters to be equal across races for females also fit the data poorly and resulted in a significant change in chi-square (Model 3a), the model that constrained parameters to be equal for Black and White male adolescents did fit the data well (Model 3b). Moreover, the change in chi-square for this model compared to the full model was not significant, indicating that the "males-equal" model fit the data as well as the model which allowed parameters to differ across Black and White male adolescents. Thus, the most parsimonious model was the model that allowed parameter estimates to vary for Black females, White females, and Black and White males, combined.

Race and Sex Differences in the Importance of Genetic and Environmental Factors. Results from the previous series of analyses indicate that genetic and environmental influences on adolescent BMI are moderated by both race and sex. Thus, our final series of analyses tested the importance of additive genetic factors, nonadditive genetic factors and shared environmental factors on variation in BMI separately for Black females, White females, and males. Table IV presents the goodness-of-fit statistics and standardized variance components (and their 95% confidence intervals) for each of the nested models tested. Model 1 (the Base Model) is the best-fitting model from the previous set of analyses, i.e., the ACDE model that constrained parameters to be equal across Black and White males but to be different from those estimated for Black females and White females.

As shown in Table IV, there appear to be racial and sex differences in the relative importance of additive genetic, nonadditive genetic, shared environmental, and nonshared environmental factors (see Model 1). First, although variance due to shared environmental factors appeared to be significant for White females, the variance component for shared environment was estimated at 0 for males, and the confidence interval surrounding this estimate for Black females also contained 0. Therefore, we tested whether systematically dropping the shared environmental parameter, c, for males (Model 2) and for Black females (Model 3) resulted in a significantly poorer fit. Dropping the c parameter for neither males nor Black females resulted in a significant change in chi-square ($\Delta\chi^2 = 0.1$, df $= 1$, $p < .999$, for Model 2; $\Delta\chi^2 = 2.5$, df $= 1$, $p < .10$, for Model 3). Thus, shared environmental factors did not contribute significantly to variation in BMI for males or for Black females.

We next investigated whether nonadditive genetic factors, d, contributed significantly to variance in adolescent BMI. Models 4–6 examined whether d could be dropped from the model for males, White females, and Black females, respectively (see Table IV). Dropping d from the model

Table IV. Goodness-of-Fit Statistics and Standardized Variance Components for Race- and Sex-Specific Models

	Goodness-of-Fit Statistics			Black females				White females				Males (Whites = Blacks)			
Model	χ^2	df	AIC	A	C	D	E	A	C	D	E	A	C	D	E
1. Base model	86.7	72	−57.3	.13	.17	.54	.16	.39	.29	.06	.26	.65	.00	.16	.19
Lower 95% CI				.00	.00	.11	.09	.02	.14	.00	.19	.25	.00	.00	.14
Upper 95% CI				.62	.36	.81	.29	.63	.43	.38	.37	.84	.07	.53	.25
2. Drop C for males	86.8	73	−59.2	.15	.17	.52	.16	.42	.28	.04	.26	.67	—	.14	.19
Lower 95% CI				.00	.00	.11	.09	.16	.14	.00	.19	.38		.00	.14
Upper 95% CI				.63	.34	.80	.29	.63	.40	.29	.36	.84		.44	.25
3. Drop C for BF	89.3	74	−58.7	.27	—	.57	.15	.42	.28	.04	.26	.69	—	.12	.19
Lower 95% CI				.01		.00	.09	.16	.14	.00	.19	.34		.00	.14
Upper 95% CI				.89		.86	.28	.63	.40	.29	.36	.85		.48	.26
4. Drop D for males	89.6	75	−60.4	.35	—	.50	.15	.45	.28	.00	.27	.80	—	—	.20
Lower 95% CI				.07		.11	.10	.23	.16	.00	.19	.74			.15
Upper 95% CI				.72		.79	.28	.61	.39	.22	.36	.85			.26
5. Drop D for WF	**89.6**	**76**	**−62.4**	**.35**	—	**.50**	**.15**	**.45**	**.28**	—	**.27**	**.80**	—	—	**.20**
Lower 95% CI				**.07**		**.11**	**.09**	**.29**	**.16**		**.20**	**.74**			**.15**
Upper 95% CI				**.72**		**.79**	**.28**	**.61**	**.39**		**.36**	**.85**			**.26**
6. Drop D for BF	95.8#	77	−58.2	.81	—	—	.19	.45	.28	—	.27	.80	—	—	.20
Lower 95% CI				.63			.10	.29	.16		.20	.73			.15
Upper 95% CI				.90			.37	.61	.39		.36	.85			.27

Note. The best-fitting model is indicated in bold face. BF, black females; WF, white females; AIC, Akaike's information criterion. *$p < .10$.

for males and for White females did not result in a significant change in chi-square ($\Delta\chi^2 = 0.3$, df = 1, $p < .999$, for Model 4; $\Delta\chi^2 = 0.0$, df = 1, $p < .999$, for Model 5). However, dropping the nonadditive genetic influence among Black females did result in a significant change in chi-square ($\Delta\chi^2 = 6.2$, df = 1, $p < .02$, Model 6). Thus, nonadditive genetic factors were significant for Black females, but not for White females or males.

In conclusion, results from the best-fitting, most parsimonious model indicate that the model that best explains variation in BMI for males is the AE model, for White females it is the ACE model, and for Black females it is the ADE model. Although the confidence intervals surrounding additive genetic variance and shared environmental variance for White females did not contain 0, for consistency, we also investigated whether *a* or *c* could be dropped from the model for White females (not shown). Both models resulted in significant changes in chi-square ($\chi^2 = 107.7$, df = 77, $p < .05$, and $\Delta\chi^2 = 18.2$, df = 1, $p < .001$, for the AE model; $\chi^2 = 141.0$, df = 77, $p < .001$, and $\Delta\chi^2 = 51.5$, df = 1, $p < .001$, for the CE model). Thus, both genetic and shared environmental factors make significant contributions to variation in adolescent BMI among White females.

DISCUSSION

The present study investigated racial and sex differences in the relative influences of additive genetic factors, nonadditive genetic factors, shared environmental factors, and nonshared environmental factors for variation in adolescent BMI. Overall, the genetic contributions to variation in adolescent BMI were substantial, with broad heritability estimates ranging from .45 (for White females) to .85 (for Black females). This finding is consistent with previous research done on adult samples, as well as with the few studies done exclusively on adolescent samples. However, results from the analyses presented here suggest that the factors that contribute to variance in BMI do in fact differ across races and sexes. Specifically, shared environmental factors contribute significantly to variation in BMI for White females but not for Black females or males. Likewise, there is evidence for nonadditive genetic influences for Black females, but not for White females or males.

Similar to other studies, the present study supports the notion that genetic factors are implicated in the etiology of individual differences in adolescent BMI. Although other studies have found both significant additive genetic and nonadditive genetic influences on BMI (e.g., Allison *et al.*, 1994b; Martin, cited by Neale and Cardon, 1992; Stunkard *et al.*, 1990), results from the present study are somewhat different, in that nonadditive genetic factors were only important for Black females. However, it should be noted that Korkeila *et al.* (1995) also found evidence for nonadditive genetic effects only among their female–female twin pairs. Nonetheless, caution should be used in interpreting this particular result from the present study. Specifically, the 95% confidence interval surrounding the estimate of nonadditive genetic variance for Black females was quite large (CI = .11–.79). Thus the finding that nonadditive genetic factors account for 50% of the variance in BMI for Black females may be an overestimate. Likewise, although dropping the nonadditive genetic parameter for males and for White females did not result in a poorer-fitting model, the ADE models for males and the ACDE model for White females did include 95% confidence intervals surrounding the estimate of nonadditive genetic variance that overlapped with the confidence interval for Black females (CI = .00–.29 for White females, CI = .00–.48 for males).

Although more research is needed to determine whether nonadditive genetic factors are more important for variation in Black female BMI, estimates of broad heritability (i.e., the combination of additive genetic and nonadditive genetic variance) are similar for Black females and for males (h^2 = .85 for Black females, h^2 = .80 for males). Moreover, the 95% confidence intervals surrounding the estimate of heritability for these two groups are relatively narrow (CI = .72–.91 for Black females, CI = .74–.85 for males). Thus, the present study suggests that, overall, genetic influences on variation in BMI are (1) considerable and (2) similar for males and females, at least among Black adolescents. Furthermore, the present study offers no support for the hypothesis that different genes influence BMI for males and females, because the estimated coefficients of genetic relatedness for additive genetic influences (r_g) and nonadditive genetic influences (r_{dg}) among male–female DZ and full sibling pairs did not differ from .5 and .25, respectively.

Perhaps the most intriguing finding from the present study is the *lower* heritability (h^2 = .45, 95% CI = .29–.61) and relatively substantial contribution of *shared environmental* factors for explaining variation in adolescent BMI among White females. Although shared environmental factors did not contribute significantly to variation in BMI for Black females or males, they explained 28% of the variation in BMI for White females (95% CI = .16–.39). At first glance, the fact that shared environment is important for only one of the four racial and sex groups suggests that this finding may be an artifact of our data set and, therefore, bears replication. However, upon reflection, this finding may be consistent with other research done on racial and sex differences in body image and dieting and exercising. Specifically, it is well documented that White females are more concerned about body image than Black females or males (Dawson, 1988; Rand and Kuldau, 1990). Furthermore, White females are more likely than Black females or males to engage in weight control strategies (Hsu, 1996) and are overrepresented in eating disorders such as anorexia nervosa and bulimia (Hsu, 1996; Wakeling, 1996). Thus, to the extent that White female siblings are more similar in diet and exercise behaviors than Black females or males, this may explain the greater influence of shared environment found among this particular group. Moreover, based on the 95% confidence intervals surrounding the estimates of nonshared environmental variance, it appears that *nonshared* environmental factors may also be more important for White females than for Black females or males (e^2 = .27, CI = .20–.36, for White females, vs. e^2 = .15, CI = .09–.28, for Black females, and e^2 = .20, CI = .15–.26 for males). Thus, even if the correlation among White female siblings for diet and exercise is not strong, racial and sex differences in diet and exercise may still explain the lower heritability of BMI among White females. In other words, the greater likelihood of White females vs. Black females or males to engage in weight control behaviors may result in stronger environmental influences on variation in BMI, be they through shared or nonshared processes. Future research should examine whether diet and exercise are important environmental influences on adolescent BMI, particularly among White females.

On the other hand, it has been suggested that examining the relative influences of genetic and en-

Table V. Comparisons of Raw Parameter Estimates and Standardized Variance Components

	Raw parameter estimate			
	a	*c*	*d*	*e*
Black females	.06	.07	.13	.07
White females	.10	.09	.04	.08
Males	.13	.00	.06	.07
	Standardized variance component			
	A	C	D	E
Black females	.13	.17	.54	.16
White females	.39	.29	.06	.26
Males	.65	.00	.16	.19

vironmental factors (i.e., the standardized estimates of variance presented in Table IV) may overstate racial and/or sex differences in the genetic and environmental contributions to variance in phenotypes (Allison *et al.*, 1994b). Specifically, these authors argue that the actual parameter estimates between groups may be similar, but different relative estimates of variance components might still be found, due to differences in overall phenotypic variation. Similar to other studies investigating racial differences in relative weight (e.g., Dawson, 1988; Greenlund *et al.*, 1996; Ness *et al.*, 1991; Rand and Kuldau, 1990), the present study does find small differences in overall (observed) phenotypic variance among racial and sex groups, with variance in BMI greatest for Black females (σ^2 = .0328, for Black females, σ^2 = .0270 for White females, σ^2 = .0268 for Black males, and σ^2 = .0256 for White males). Therefore, we also examined the raw parameter estimates of *a*, *c*, *d*, and *e* for Black females, White females, and Black and White males, combined. Table V presents the comparisons of the raw parameter estimates and the standardized variance components for the full ACDE model, unconstrained by race or sex.

As shown in Table V, the raw parameter estimate for *d* is largest for Black females. Thus, the greater effect of nonadditive genetic factors revealed in the standardized variance components is a reflection of the larger absolute contribution of nonadditive genetic factors for variation in Black female BMI. Furthermore, unstandardized estimates of the total genetic variation (i.e., V_g, the sum of the a^2 and d^2 parameters) also suggest that total genetic variance is greater for Black females and males than for White females (V_g = .0205 for Black females, V_g = .0205 for males, and V_g = .0116 for White females), similar to what was concluded using the standardized variance components. Regarding the absolute influence of shared environmental factors, however, although the raw parameter estimates for *c* were lower for males (*c* = .00), they were similar for Black and White females (*c* = .07 and .09, respectively). Estimates of *e* were similar for all three groups. Thus, the greater shared environmental influence on variation in BMI for White females relative to Black females is a function of the larger estimate for *d* found among Black females, which contributes to the larger overall phenotypic variation. Nonetheless, results from our hierarchically nested models (see Table IV) do suggest that the *c* parameter can be set to 0 for Black females without a significant worsening of fit, whereas setting this parameter to 0 for White females resulted in a poorer-fitting model.

Strengths and Limitations

The present study expands upon previous research in a number of important ways. First, this study uses a sample of adolescents, a developmental period that is relatively understudied with regard to genetic and environmental influences on variation in BMI. Furthermore, the sample used in this study is a population-based sample, therefore results should generalize to Black and White American adolescents. Third, the present sample contains a variety of sibling groups. Thus, the relative contributions of nonadditive genetic factors and shared environmental factors could be assessed simultaneously, a strength not found among the more common twin study designs. Likewise, the use of multiple sibling groups avoids potential confounds of special twin environments and age matching. In general, the full sibling correlations were as strong as the DZ twin correlations (see Table I), suggesting that neither special twin environments nor age matching is contributing to greater similarity among twins. Fourth, the sample was large enough to examine both racial and sex differences simultaneously. The fact that both sex and race moderated the relative influence of both genetic and shared environmental factors suggests that studies which combine across sexes and/or races may mask potentially significant effects (particularly for

shared environment) common to a particular sex or race. Finally, the inclusion of opposite-sex sibling pairs allowed for a direct test of whether the genes that influence variation in adolescent BMI are similar for males and females.

As with most research, this study is not without limitations. Specifically, one important limitation is that relative weight was calculated using self-reports of height and weight, which were then transformed into BMI units. Although this method is commonly used in large scale studies, it is possible that more accurate estimates of body fatness might yield different results. In a related vein, it has not been tested whether adolescents (particularly female adolescents) are more likely than adults to lie about their weight. Thus, studies that use objective measures of body fatness are warranted.

Second, although we hypothesize post hoc that racial and sex differences in dieting behaviors may be responsible for the lower heritability estimate and greater shared environmental estimates found among the White female group, we do not test this hypothesis directly. It is possible that the stronger estimate of shared environment is due to some artifact of our data. For example, examination of the sibling correlations suggests that the significant estimate of shared environment is due, at least in part, to the significant correlation between the White female unrelated siblings. A more detailed analysis of our data revealed that unrelated White females living in the same house are more likely to be adoptive or step siblings than unrelated Black females living in the same house (18.8% adoptive and 45.3% step-siblings for White females vs. 10.0% adoptive and 25.0% step-siblings for Black females).[6] Thus, factors such as selective placement or assortative mating might be contributing to the significant correlation between White female unrelated siblings. On the other hand, similar differences in proportions exist among White and Black male unrelated siblings (12.7% adoptive and 57.4% step-siblings for White males vs. 4.2% adoptive and 16.7% step-siblings, for Black males), yet estimates of shared environmental influences are 0 for both White and Black males. Nonetheless, efforts to determine the exact processes that make White female unrelated siblings more similar than Black female unrelated siblings should be made.

Third, as with most ACDE models of phenotypic variation, the present study makes the assumption of no gene × environment interactions. However, this assumption should be tested explicitly. For example, the strong inverse relationship between social class and weight, particularly among women (Dawson, 1988; Hsu, 1996; Rand and Kuldau, 1990), may suggest that relative influences of genetic and environmental factors might differ for women at varying levels of social class. Although G × E interactions are rare, another study using the Add Health sample found a G × E interaction for verbal IQ, with heritability estimates greater for adolescents whose parents were better educated and shared environmental influences greater for adolescents whose parents were less well educated (Rowe *et al.*, 1998). Thus, future research should explore the potential moderating effects of social class for genetic and environmental influences on variation in BMI.

The present study also operates under the equal-environments assumption. Although there are data to suggest that the equal environments assumption is a valid one in twin studies (Hettema *et al.*, 1995; Rowe, 1994), it is less clear whether this assumption is sound in studies of multiple sibling groups. Finally, although the Add Health sample is representative of all adolescents living in the United States, the numbers of non-White and non-Black adolescents were too small to permit separate analyses. Thus, the results here might not generalize to adolescents of other ethnic and racial groups. Future research should continue to explore the genetic and environmental contributions to variance in adolescent BMI in other populations.

ACKNOWLEDGMENTS

This research is based on data from the Add Health project, a program project designed by J. Richard Udry (PI) and Peter Bearman and funded by Grant P01-HD31921 from the National Institute of Child Health and Human Development to the Carolina Population Center, University of North Carolina at Chapel Hill, with cooperative funding participation by the National Cancer Institute; the National Institute of Alcohol Abuse and Alcoholism; the National Institute on Deafness and Other

[6] Numbers do not add up to 100% because the unrelated sibling group also contained sibling pairs who were not adoptive or step-siblings. These "other" unrelated siblings included foster siblings as well as siblings for whom no definite relationship could be established.

Communication Disorders; the National Institute of Drug Abuse; the National Institute of General Medical Sciences; the National Institute of Mental Health; the National Institute of Nursing Research; the Office of AIDS Research, NIH; the Office of Behavior and Social Science Research, NIH; the Office of the Director, NIH; the Office of Research on Women's Health, NIH; the Office of Population Affairs, HHS; the National Center for Health Statistics, Centers for Disease Control and Prevention, HHS; the Office of Minority Health, Centers for Disease Control and Prevention, HHS; the Office of Minority Health, Office of the Assistant Secretary for Health, HHS; the Office of the Assistant Secretary for Planning and Evaluation, HHS; and the National Science Foundation. Persons interested in obtaining data files from the National Longitudinal Study of Adolescent Health should contact Jo Jones, Carolina Population Center, 123 West Franklin Street, Chapel Hill, North Carolina 27516-3997 (e-mail: jo_jones@unc.edu).

REFERENCES

Akaike, H. (1987). Factor analysis and AIC. *Psychometrika* **52**:317–332.

Allison, D. B. (1992). A field study of ethnic differences in preferences for women's body shapes. *Int. J. Obes.* **16** (Suppl.):21.

Allison, D. B., Heshka, S., Neale, M. C., and Heymsfield, S. B. (1994a). Race effects in the genetics of adolescents' Body Mass Index. *Int. J. Obes.* **18**:363–368.

Allison, D. B., Heshka, S., Neale, M. C., Lykken, D. T., and Heymsfield, S. B. (1994b). A genetic analysis of relative weight among 4,020 twin pairs, with an emphasis on sex effects. *Health Psychol.* **13**:362–365.

Bodurtha, J. N., Mosteller, M., Hewitt, J. K., Nance, W. E., Eaves, L. J., Moskowitz, W. B., Katz, S., and Schieken, R. M. (1990). Genetic analysis of anthropometric measures in 11-year-old-twins: The Medical College of Virginia Twin Study. *Pediatr. Res.* **28**:1–4.

Bouchard, T. J., Lykken, D. T., McGue, M., Segal, N. L., and Tellegen, A. (1990). Sources of human psychological differences: The Minnesota Study of Twins Reared Apart. *Science* **250**:223–228.

Carmichael, C. M., and McGue, M. (1995). A cross-sectional examination of height, weight, and Body Mass Index in adult twins. *J. Gerontol.* **50A**:B237–B244.

Dawson, D. A. (1988). Ethnic differences in female overweight: Data from the 1985 National Health Interview Survey. *Am. J. Public Health* **78**:1326–1329.

Fabsitz, R. R., Sholinsky, P., and Carmelli, D. (1994). Genetic influences on adult weight gain and maximum Body Mass Index in male twins. *Am. J. Epidemiol.* **140**:711–720.

Greenlund, K. J., Liu, K., Dyer, A. R., Kiefe, C. I., Burke, G. L., and Yunis, C. (1996). Body mass index in young adults: Associations with parental body size and education in the CARDIA study. *Am. J. Public Health* **86**:480–485.

Grilo, C. M., and Pogue-Geile, M. F. (1991). The nature of environmental influences on weight and obesity: A behavior genetic analysis. *Psychol. Bull.* **110**:520–537.

Guillaume, M., and Bjorntorp, P. (1996). Obesity in children: Environmental and genetic aspects. *Horm. Metab. Res.* **28**:573–581.

Herskind, A. M., McGue, M., Sorensen, T. I. A., and Harvald, B. (1996). Sex and age specific assessment of genetic and environmental influences on body mass index in twins. *Int. J. Obes.* **20**:106–113.

Hettema, J. M., Neale, M. C., and Kendler, K. S. (1995). Physical similarity and the equal-environments assumption of twin studies of psychiatric disorders. *Behav. Genet.* **25**: 327–335.

Hewitt, J. K. (1997). The genetics of obesity: What genetic studies have told us about the environment. *Behav. Genet.* **27**:353–358.

Hsu, L. K. (1996). Epidemiology of the eating disorders. *Psych. Clin. N. Am.* **19**:681–700.

Hunt, S. C., Hasstedt, S. J., Kuida, H., Stults, B. M., Hopkins, P. N., and Williams, R. R. (1989). Genetic heritability and common environmental components of resting and stressed blood pressures, lipids, and Body Mass Index in Utah pedigrees and twins. *Am. J. Epidemiol.* **129**:625–638.

Kaprio, J., Rimpela, A., Winter, T., Viken, R. J., Rimpela, M., and Rose, R. J. (1995). Common genetic influence on BMI and age at menarche. *Hum. Biol.* **67**:739–753.

Khoury, P., Morrison, J. A., Laskarzewski, P. M., and Glueck, C. J. (1983). Parent-offspring and sibling Body Mass Index associations during and after sharing of common household environments: The Princeton School District Family Study. *Metabolism* **32**:82–89.

Korkeila, M., Kaprio, J., Rissanen, A., and Koskenvuo, M. (1995). Consistency and change of body mass index and weight. A study on 5967 adult Finnish twin pairs. *Int. J. Obes.* **19**:310–317.

Kumanyika, S. (1987). Obesity in Black women. *Epidemiol. Rev.* **9**:31–50.

le Grange, D., Telch, C. F., and Tibbs, J. (1998). Eating attitudes and behaviors in 1,435 South African Caucasian and non-Caucasian college students. *Am. J. Psych.* **155**:250–254.

Maes, H. H. M., Neale, M. C., and Eaves, L. J. (1997). Genetic and environmental factors in relative body weight and human adiposity. *Behav. Genet.* **27**:325–351.

McGue, M., Bouchard, T. J., Iacono, W. G., and Lykken, D. T. (1993). Behavioral genetics of cognitive ability: A life span perspective. In Plomin, R., and McClearn, G. E. (eds.), *Nature, Nurture, and Psychology*, American Psychological Association, Washington, DC.

Meyer, J. M. (1995). Genetic studies of obesity across the lifespan. In Turner, J. R., Cardon, L. R., and Hewitt, J. K. (eds.), *Behavior Genetic Approaches in Behavioral Medicine*, Plenum Press, New York, pp. 145–166.

Neale, M. C. (1997). *Mx: Statistical Modeling*, 4th ed., Department of Psychiatry, Box 126 MCV, Richmond, VA 23298.

Neale, M. C., and Cardon, L. R. (1992). *Methodology for Genetic Studies of Twins and Families*, Kluwer Academic, Dordrecht, The Netherlands.

Ness, R., Laskarzewski, P., and Price, R. A. (1991). Inheritance of extreme overweight in Black families. *Hum. Biol.* **63**:39–52.

Plomin, R. (1986). *Development, Genetics, and Psychology*, Lawrence Erlbaum, Hillsdale, NJ.

Plomin, R. (1990). *Nature and Nurture*, Brooks/Cole, Pacific Grove, CA.

Price, R. A., and Gottesman, I. I. (1991). Body fat index in identical twins reared apart: Roles for genes and environment. *Behav. Genet.* **21**:1–7.

Rand, C. S. W., and Kuldau, J. M. (1990). The epidemiology of obesity and self-defined weight problem in the general

population: Gender, race, age, and social class. *Int. J. Eat. Dis.* **9**:329–343.

Resnick, M. D., Bearman, P. S., Blum, R. W., Bauman, K. E., Harris, K. M., Jones, J., Tabor; J.,Beuhrin, T., Sieving, R. E., Shew, M., Ireland, M., Bearinger, L. H., and Udry, J. R. (1997). Protecting adolescents from harm: Findings from the National Longitudinal Study on Adolescent Health. *JAMA* **278**:823–832.

Rowe, D. C. (1994). *The Limits of Family Influence*, Guilford Press, New York.

Rowe, D. C., Jacobson, K. C., and van den Oord, E. J. C. G. (1998). Parental education level and Peabody Picture Vocabulary IQ: A genotype × environment interaction. (submitted for publication).

Sorensen, T. I. A., and Stunkard, A. J. (1993). Does obesity run in families because of genes? An adoption study using silhouettes as a measure of obesity. *Acta Psychiatry Scand.* **370**:67–72.

Stunkard, A. J., and Albaum, J. M. (1981). The accuracy of self-reported weights. *Am. J. Clin. Nutr.* **34**:1593–1600.

Stunkard, A. J., Sorensen, T. I. A., Hanis, C., Teasdale, T. W., Chakraborty, R., Schull, W. J., and Schulsinger, F. (1986). An adoption study of human obesity. *N. Engl. J. Med.* **314**:193–198.

Stunkard, A. J., Harris, J. R., Pedersen, N. L., and McClearn, G. E. (1990). The Body Mass Index of twins who have been reared apart. *N. Engl. J. Med.* **322**:1483–1487.

Vogler, G. P., Sorensen, T. I. A., Stunkard, A. J., Srinivasan, M. R., and Rao, D. C. (1995). Influence of genes and shared family environment on adult body mass index assessed in an adoption study by a comprehensive path model. *Int. J. Obes.* **19**:40–45.

Wakeling, A. (1996). Epidemiology of anorexia nervosa. *Psych. Res.* **62**:3–9.

Edited by Stacey Cherny