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

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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-Gile, 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...
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) and conclude that nonadditive genetic influences are 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.