A Genetically Informed Study of Disordered Eating Pathology in Adolescent Twins

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Abstract

Using a multivariate twin analysis to control for non-random genetic factors, we investigated whether body dissatisfaction, binge eating, and weight preoccupation predicted dietary restraint. Data was analyzed from 190 twin pairs (110 MZ; 80 DZ) from the Michigan State University Twin Registry. Analyses suggested that the significant effects of body dissatisfaction and weight preoccupation on dietary restraint were moderated by non-shared environmental factors. The effect of binge eating on dietary restraint was moderated by genetic and non-shared environmental factors, however these findings were not statistically significant. The present findings indicate that dietary restraint may be targeted indirectly through the treatment of body dissatisfaction, binge eating, and weight preoccupation.

*Keywords:* disordered eating, body dissatisfaction, binge eating, weight preoccupation, dietary restraint, behavior genetic, twins
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In an increasingly weight and figure conscious society, the potential for women to develop disordered eating attitudes and behaviors continues to be an issue of great importance. Today, girls begin to exhibit symptoms at exceedingly younger ages, with girls as young as five years of age showing pre-symptomatic attitudes towards the importance of weight and bodily appearance (Davison, Markey & Birch, 2000). Although precursors to disordered eating may present in girls of elementary and middle school age (Hill & Pallin, 1998; Schur, Sanders, & Steiner, 2000), it is adolescent and young adult women who are at the greatest risk for developing both clinical threshold eating disorders and disordered eating attitudes and behaviors (Hoek & Hoeken, 2003). The young age at which women begin to exhibit disordered eating symptoms is alarming, due to a host of associated negative psychological and physical outcomes, including suicide and death (Keel et al., 2003). The serious outcomes and life course associated with disordered eating necessitate further research on the causes and exacerbating factors of disordered eating attitudes and behaviors.

Eating Disorder Classification

The Diagnostic and Statistical Manual of Mental Disorders IV - Text Revision (DSM-IV-TR) classifies anorexia nervosa (AN) and bulimia nervosa (BN) as two discrete eating disorders “characterized by severe disturbances in eating behavior” (4th ed., text rev.; DSM-IV-TR; American Psychiatric Association, 2000). Although both disorders are associated with an intense, persistent fear of gaining weight, a strong desire to lose weight, undue emphasis on bodily appearance in self-evaluation, and extreme dissatisfaction with body, the disorders differ according to their accompanying behaviors (4th ed., text rev.; DSM-IV-TR; American Psychiatric Association, 2000). Whereas anorexia nervosa is characterized by extreme weight restriction and
the failure to maintain 85% of expected body weight, individuals with bulimia nervosa engage in a cycle of discrete periods of excessive food consumption followed by the use of inappropriate compensatory behaviors. Compensatory behaviors include purging behaviors, such as self-induced vomiting, laxative and diuretic abuse, and restrictive behaviors, such as fasting, excessive exercise, and diet pill usage (4th ed., text rev.; DSM-IV-TR; American Psychiatric Association, 2000). Like bulimia nervosa, individuals with anorexia nervosa maintain dietary restraint through similar restrictive behaviors, and may also engage in purging behaviors to a lesser extent (4th ed., text rev.; DSM-IV-TR; American Psychiatric Association, 2000). Binge Eating Disorder (BED), which is a disorder proposed for the next edition of the DSM, is characterized by the discrete periods of binge eating and feelings of loss of control characteristic of bulimia nervosa, without accompanying compensatory behaviors (Walsh, 2011).

**Epidemiology**

Despite the severe nature of eating disorders, lifetime prevalence rates remain low in adolescent female populations. The lifetime prevalence for anorexia nervosa is 0.3% - 0.9%, 1.0% - 1.5% for bulimia nervosa, and 1.6% - 3.5% for binge eating disorder (Hoek & Hoeken, 2003; Hudson, Hiripi, Pope & Kessler, 2007; Swanson, Crow, Le Grange, Swendsen & Merikangas, 2011). The onset of eating disorders for females occurs most frequently between mid-adolescence and early adulthood. The mean ages of onset for these disorders are 18.9, 19.7, and 25.4 years, respectively (Hudson, Hiripi, Pope & Kessler, 2007).

Although the onset of full threshold eating disorders tends to occur toward young adulthood, the onset of disordered eating attitudes and behaviors often occurs during earlier adolescence. For example, Grigg, Bowman, and Redman (1996) found that for a sample of 14-16 year old girls, 33% engaged in disordered eating, 57% engaged in unhealthy dieting, and 12%
exhibited an unhealthy body image. Additionally, 27% reported binge eating, 8% reported self-induced vomiting, and 46% reported skipping meals to restrict intake over the past month. Two-thirds of the girls perceived themselves as fat, while three-quarters of those with a distorted body image were actually underweight.

**Lifetime Course**

The progression of disordered eating to clinical eating disorders may be reflected by changes in the prevalence of disordered eating behaviors from early adolescence to young adulthood. Neumark-Sztainer, Wall, Larson, Eisenberg, and Loth (2011) found that although the prevalence of dieting remains constant, the prevalence of extreme weight control behaviors, binge eating, diet pill usage, and laxative abuse increases in women from early adolescence to young adulthood. Furthermore, dieting, binge eating, and engaging in unhealthy weight control behaviors as an adolescent significantly predict the continuation of these behaviors into adulthood (Neumark-Sztainer, Wall, Larson, Eisenberg, & Loth, 2011).

The increase in disordered eating behaviors from adolescence to young adulthood is demonstrated by the high prevalence of disordered eating behaviors in college women. Mintz and Betz (1988) found that over 61% of the female college students engaged in the disordered eating behaviors of chronic dieting, binge eating, and purging. Over 38% of college women reported difficulties with binge eating, whereas only 33% of women reported normal eating habits. An additional 33% of women engaged in severe forms of weight control monthly, and over 82% of women engaged in one or more dieting behaviors daily. Disordered eating behaviors were correlated with lowered self-esteem, negative body image, desirability of culture driven thinness, obsessive thoughts concerning weight and appearance, and interference of weight and appearance in other life domains (Mintz & Betz, 1988).
In addition to an increased prevalence of disordered eating behaviors, college women also have a greater prevalence of clinical eating disorders relative to the general female population. For college populations, research supports a lifetime prevalence of approximately 3% for anorexia nervosa and 4% for bulimia nervosa (Kurth, Krahn, Nairn, and Drewnowski, 1995; Mint and Betz, 1998; Strigel-Moore, Silberstein, Freisch & Rodin, 1989). The estimated rates of anorexia nervosa and bulimia nervosa for college women are strikingly higher than the respective rates of 0.3% - 0.9% and 1.0% - 1.5% reported in the general female population.

Outcomes

The elevated rates of disordered eating and clinical eating disorders in college women are alarming considering the severe negative psychological and physical outcomes associated with them. Hudson, Hiripi, Pope, and Kessler (2007) found that 50% - 78.9% of women diagnosed with anorexia nervosa, bulimia nervosa or binge eating disorder also met criteria for at least one other DSM-IV disorder. Eating disorders are significantly comorbid with mood, anxiety, impulse-control, and substance use disorders, and over than 60% women with bulimia nervosa and binge eating disorder report associated life impairment (Hudson, Hiripi, Pope, & Kessler, 2007). Krahn, Kurth, Gomberg, and Drewnowski (2005) found that extreme dieting and dietary restriction are positively correlated with alcohol use and severity. The frequency of lifetime comorbidity of at least one anxiety disorder with a diagnosis of anorexia nervosa or bulimia nervosa is significantly higher than for controls (Godart, Flament, Perdereau & Jeammet, 2002).

Anorexia nervosa in particular is associated with an increased rate of mortality and a greater risk of suicide (Herzog et al., 2000). Significant predictors of mortality in anorexia nervosa include severity of substance abuse and worse social adjustment (Keel et al., 2003). Keel et al. found, however, that previous hospitalization for the treatment of an affective disorder may
act as a protective factor against negative outcomes in anorexia nervosa. The strong association between eating disorders and negative psychological and physical outcomes may be due in part to the moderate relapse rates seen in women. Stice, Marti, Shaw, and Jaconis (2009) found relapse rates of 33% to 41% in women diagnosed with bulimia nervosa or anorexia nervosa one year after concluding treatment. Furthermore, less than 30% of women with symptoms at or above clinical levels of anorexia nervosa, bulimia nervosa, and binge eating disorder seek treatment specifically for their disordered eating (Swanson, Crow, Le Grange, Swendsen & Merikangas, 2011).

**Disordered Eating**

Although much of the literature focuses on correlates of eating disorders within adolescent female populations, many women present with subclinical threshold levels of disordered eating symptoms. Although incidence rates of anorexia nervosa have remained stable, and incidence rates bulimia nervosa have decreased over recent decades (Currin, Schmidt, Treasure, & Jick, 2005; Heatherton, Nichols, Mahamedi, & Keel, 1995; Keel, Heatherton, Dorer, Joiner, & Zalta, 2006; van Son, van Hoeken, Bartelds, van Furth, & Hoek, 2006), the incidence of disordered eating behaviors continues to rise (Hay, Mond, Buttner, & Darby, 2008). It is therefore imperative for researchers to focus on the individual components of eating disorders, namely disordered eating attitudes and behaviors, in order to curtail the rising incidence of these behaviors in adolescent female populations.

**Body dissatisfaction.**

Body dissatisfaction concerns the belief that the parts of the body associated with shape or increased post-pubertal changes to the body are displeasing or excessively large (Garner, Olmstead, & Polivy, 1983). The negative self-appraisals that result in the development of body
dissatisfaction most often concern shape, hips, weight, and/or stomach appearance (Stice & Shaw, 2002). Body dissatisfaction is widely accepted as a predictor of further disordered eating behavior (Goldfield et al., 2010; Stice & Shaw, 2002). Up to 24% of adolescent girls may exhibit mild to severe body dissatisfaction (Stice & Whitenton, 2002), and female college populations may have an even greater incidence (Klemchuk, Hutchinson, & Frank, 1990). Perceived pressure to be thin, internalization of a thin ideal, parental teasing, and low social support act as predictors of body dissatisfaction, and may exacerbate pre-existing negative attitudes (Stice & Shaw, 2002; Stice & Whitenton, 2002; Neumark-Sztainer et al., 2011).

**Body dissatisfaction and dietary restraint.**

Body dissatisfaction is implicated in the onset and perpetuation of dietary restraint in women (Maloney, McGuire, Daniels, & Specker, 1989; Neumark-Sztainer et al. 2011; Ricciardelli, Tate, & Williams, 1997; Thompson, Coovert, Richards, Johnson, & Cattarin, 1995), especially if dietary restraint is viewed as an effective means of weight loss (Stice & Shaw, 2002). Negative affect resulting from low self-appraisal may also contribute to a relationship between body dissatisfaction and binge eating, as eating is often used as a means of assuaging negative feelings (Stice & Shaw, 2002). Goldfield et al. (2010) propose that body dissatisfaction may create a path to obesity and further disordered eating behaviors by influencing dietary restraint and binge eating.

**Binge eating.**

Binge eating is a constituent part of both bulimia nervosa and binge eating disorder, and is present in the binge/purge subtype of anorexia nervosa. An episode of binge eating is constituted by the consumption of a larger than normal amount of food in a discrete period of time, often within a duration of less than two hours (4th ed., text rev.; DSM-IV-TR; American
Psychiatric Association, 2000). Additionally, binge eating can occur to the point of extreme fullness and discomfort and is often accompanied by a feeling of loss of control (4th ed., text rev.; DSM-IV-TR; American Psychiatric Association, 2000).

**Binge eating and genetic factors.**

Genetic and twin studies have reported associations between genetic factors and the onset of binge eating. Independent of other disordered eating behaviors, Wade, Bulik, Sullivan, Neale, and Kendler (2000) found that genetic factors are partly responsible for the onset of binge eating in twins. Wade et al. also found that the transition from binge eating to bulimia nervosa is mediated by both genetic factors and non-shared environmental factors. The influence of both genes and the non-shared environment in the progression of binge eating to bulimia nervosa may indicate the presence of a gene-environment interaction, meaning that a genetic propensity toward a behavior is only expressed when an individual encounters an environmental stressor. Bulik, Sullivan, and Kendler (1998) found that genetic factors account for 50% of the individual variation in binge eating. Furthermore, Racine et al. (2011) found that dietary restraint increases the influence of non-shared environmental and genetic factors on binge eating. Both findings support the hypothesis that an individual with a genetic predisposition toward binge eating is more likely to develop the behavior after encountering a relevant stressor.

**Binge eating and dietary restraint.**

Numerous studies support a significant relationship between dietary restraint and binge eating (Woods, Racine, & Klump, 2010; Stice & Shaw, 2002). The caloric intake hypothesis proposes that reduced caloric intake due to dietary restraint may lead the body to recompense through binge eating (Stice & Shaw, 2002). Conversely, the disinhibition hypothesis argues that binge eating occurs as a result of interruptions in ongoing dietary restraint by cognitive,
emotional, or pharmacological events, which lead to loss of restrictive control and extensive food consumption (Polivy, Heatherton, & Herman, 1988; Ruderman, 1986; Stice & Shaw, 2002). The latter hypothesis is supported by findings that individuals engaging in dietary restraint are more likely to overeat, especially if they exhibit low self-esteem (Polivy, Heatherton, & Herman, 1988).

**Weight preoccupation.**

Preoccupation or concern with weight is a symptom of anorexia nervosa, bulimia nervosa (4th ed., text rev.; DSM-IV-TR; American Psychiatric Association, 2000), and potentially even binge eating disorder (Eldredge & Agras, 1996). Although weight preoccupation is a requirement for the clinical diagnosis of an eating disorder, the attitude is also present within non-clinical populations (4th ed., text rev.; DSM-IV-TR; American Psychiatric Association, 2000). Weight concern is a risk factor for disordered eating behaviors and attitudes, as well as clinical eating disorders (Jacobi, Hayward, de Zwann, Kramer, & Agras, 2004; Neumark-Sztainer, 1995; Stice, Killen, Hayward & Taylor, 1998). Girls who scored higher on measures of weight concern were more likely to develop additional disordered eating symptoms and eating disorders (Killen et al., 1994). Additionally, adolescents with high weight concern scores were more likely to develop partial syndrome eating disorders, whereas adolescents who exhibited little to no weight concern did not develop disordered eating. Furthermore, weight preoccupation and dietary restraint are strongly correlated, and weight preoccupation may even serve as a better predictor of the onset of disordered eating than the latter (Killen et al., 1994).

**Dietary restraint.**

Dietary restraint concerns mild to severe restriction of the types and/or amounts of food consumed by an individual, as a means of controlling body weight and/or shape (Goldfield et al.,
It is a main component of both anorexia nervosa and bulimia nervosa, and may be associated with binge eating disorder as well (Ricciardelli, Tate, & Williams, 1997). As previously noted, dietary restraint is associated with, and may predict, the other disordered eating behaviors and attitudes of body dissatisfaction, binge eating, and weight concern (Jacobi, Hayward, de Zwann, Kraemer, & Agras, 2004; Polivy & Herman, 1985).

**Dietary restraint in the present study.**

Because dietary restraint is an integral component of anorexia nervosa, bulimia nervosa, and potentially binge eating disorder (Ricciardelli, Tate, & Williams, 1997), the severe prognosis of these disorders indicates the need for further study on the potential causes and predictive risk factors of this behavior. Whereas numerous other studies show high correlations among dietary restraint, body dissatisfaction, weight preoccupation, and binge eating, the present study aims to examine a potential causal pathway among these disordered eating behaviors. To our knowledge, this is the first such study to determine underlying causal influences in the relationship between dietary restraint and binge eating, body dissatisfaction, and weight preoccupation.

**Behavior Genetic Design**

Simple correlational analyses lack the ability to establish causal pathways in disordered eating. Because there is the potential for unobserved third factors to confound the associations observed between two variables, these confounds obscure a researcher’s ability to draw causal conclusions. For example, potential confounds, such as nonrandom genetic and environmental selection effects may underlie the observed correlations between dietary restraint and body dissatisfaction, binge eating, and weight preoccupation. If these non-random selection effects are not taken into account, it may be impossible to understand how the cognitive and behavioral features of disordered eating causally influence dietary restraint. The current study recognizes
this deficiency in the current literature and uses behavioral genetic design to statistically adjust for nonrandom genetic and environmental selection confounds.

Behavior genetic designs allow researchers to differentiate between the potential causal effects of two observed variables and selection confounds. Also referred to as quasi-causal designs, these twin designs permit one to account for nonrandom genetic effects by using monozygotic (identical; MZ) and dizygotic (fraternal; DZ) twins (Harden, Mendle, Hill, Turkheimer, & Emery, 2008). Because the genetic material of monozygotic twins is identical, any phenotypic differences between MZ twins cannot be due to genetic factors. Therefore, to the extent that MZ twins are more concordant for a behavior than DZ twins – who share 50% of their genes, on average – one can conclude that nonrandom genetic effects influence twins’ disordered eating habits. Additionally, behavior genetic twin designs also account for the environments that co-twins share with one another, because shared environments (e.g., parental SES, parental education) influence MZ and DZ twins equally. By controlling for these non-random genetic and environmental effects, we are able to draw strong conclusions about the causality of the effects of binge eating, body dissatisfaction, and weight preoccupation on dietary restraint (Harden et al, 2008). Additionally, as noted by, Harden et al. (2008), no correlational study can provide definitively causal findings. Therefore any “causal” findings reported here are termed “quasi-causal”.

**Previous findings.**

Previous genetically informed studies of disordered eating elucidate the potential effects of non-random genetic and environmental processes on disordered eating attitudes and behaviors.

*Bulimia nervosa and anorexia nervosa.*
Bulik, Sullivan, Wade, and Kendler (2000) reported that the development of anorexia nervosa and bulimia nervosa is partially mediated by genetic and non-shared environmental factors. Whereas additive genetic factors account for the overwhelming majority of variance in anorexia nervosa (88%), the influence of additive genetic effects on bulimia nervosa is more variable (23% - 83%). Non-shared environment also significantly contributes to the variance in bulimia nervosa, with estimated effects of 17% to 68%, and less so to the variance in anorexia nervosa (12%). Shared environment does not appear particularly influential on the development of either anorexia nervosa or bulimia nervosa (AN: 0%; BN: 0% - 37%), although the effect of shared environment varies considerably across studies. Moreover, the consistent findings of both genetic and non-shared environmental influences on the development of bulimia nervosa may be indicative of a gene-environment interaction for the development of the disorder (Bulik, Sullivan, Wade, & Kendler, 2000).

**Disordered eating behaviors and attitudes.**

Previous studies have found that behavioral and attitudinal aspects of disordered eating are influenced by a combination of genetic and environmental effects. Wade, Martin, and Tiggemann (1998) found that genetic and non-shared environmental effects moderated the overall variance of dietary restraint, eating concern, and shape concern. Environmental effects, however, accounted for all variance in weight concern. Moreover, Bulik, Sullivan, Wade, and Kendler (2000) found that genetic and non-shared environments each accounted for approximately half of variance in binge eating.

**Age differences.**

Although studies of young adult women show that genes and non-shared environment best account for the variance in disordered eating attitudes and behaviors (Bulik, Sullivan, Wade,
& Kendler, 2000), these effects may differ as a function of age. Klump, McGue, and Iacono, (2003) found that genes may not be influential in the development of disordered eating before the onset of puberty. As opposed to young adult women, environmental effects account for the variance in the disordered eating of adolescent girls, whereas there is little to no indication of genetic effects (Klump, McGue, & Iacono, 2000; Klump, McGue, & Iacono, 2003; Klump et al., 2010).

**Hypotheses**

In accordance with previous findings, we propose that body dissatisfaction, binge eating, and weight preoccupation will predict dietary restraint, and that non-random genetic and environmental processes will partially account for the correlations among these variables. We also hypothesize that body dissatisfaction, binge eating, and weight preoccupation will continue to predict dietary restraint, however, once taking into account genetic and environmental selection factors.

**Methods**

**Sample**

Data for this study come from the Michigan State University Twin Registry (MSUTR), a database of phenotypic and genotypic data collected from monozygotic and dizygotic adolescent female twins beginning in 2001 (Klump & Burt, 2006). At the time of the current study, data from 190 twin pairs (110 MZ; 80 DZ) had been collected. Twin age ranged from 16.12 to 23.75 years ($M = 18.11, SD = 1.87$). Twin zygosity was measured through a comparison of questionnaires assessing physical similarity, and had a 95% accuracy rating or higher for determination of zygosity. The MSUTR data analyzed for the current study used items taken from the Minnesota Eating Behaviors Questionnaire (MEBS) and the Eating Disorder
Examination Questionnaire (EDE-Q) to measure body dissatisfaction, binge, eating, weight preoccupation, and dietary restraint (Klump & Burt, 2006).

**Measures**

**Eating disorder examination questionnaire.**

The Eating Disorder Examination Questionnaire (EDE-Q; See Appendix A; Fairburn & Bèglin, 1994) is a 36-item self-report measure of disordered eating behavior over a one-month (28-day) period, and is designed for completion in approximately 15 minutes. The questionnaire is composed of four subscales, Restraint, Shape Concern, Weight Concern, and Eating Concern, and measures such disordered eating behaviors as binge eating, purging, and laxative abuse. Behavioral frequency is calculated by the total number of days of behavioral presentation over the preceding 28-days (Fairburn & Bèglin, 1994). The questionnaire is adapted from the Eating Disorders Examination (EDE; Fairburn & Cooper, 1993), which is a semi-structured clinical interview designed to assess disordered eating in individuals for research purposes. Each item on the EDE-Q derives from a counterpart item on the EDE (Fairburn & Bèglin, 1994).

**Validity and reliability.**

The EDE is a valid measure of disordered eating behavior, showing moderate concurrent validity with additional measures of dietary restraint and overeating, and effectively distinguishing between individuals with and without eating disorders (Cooper, Cooper, & Fairburn, 1989; Rosen, Vara, Wendt, & Leitenberg, 1990). The EDE-Q and EDE are significantly correlated across the three main subscales of Restraint, Weight Concern, and Shape Concern, as well as the behaviors of self-induced vomiting, laxative abuse, binge eating, and strict dieting (Black & Wilson, 1996). The four EDE-Q subscales are internally consistent, highly stable, and show test-retest reliability (Luce & Crowther, 1999). Weight Concern and
Eating Concern may measure a single construct, however, as the items in these subscales load onto a single factor (Peterson et al., 2007).

The EDE-Q is an effective measure for disordered eating in adolescent populations, including samples with bulimia nervosa, anorexia nervosa, and sub-threshold bulimia nervosa (Binford, Le Grange, & Jellar, 2005). In community samples, however, scores may be higher on the EDE-Q than the EDE (Mond, Hay, Rodgers, Owen, & Beumont, 2004a; Mond, Hay, Rodgers, Owen, & Beumont, 2004b).

The four scales of the EDE-Q are also reliable measures of the behaviors and attitudes of Binge Eating Disorder, although there is greater variability among individual items (Reas, Grilo, & Masheb, 2006). Additionally, there may be discrepancies in the measurement of objective binge episodes (Mond et al., 2004a) and episodes of over eating not accompanied by loss of control (Reas, Grilo & Masheb, 2006).

**Minnesota eating behaviors survey.**

The Minnesota Eating Behaviors Survey (MEBS; See Appendix B) is a 30-item self-report questionnaire designed by researchers of the Minnesota Twin Family Study to measure disordered eating behaviors and cognitions consistent with Anorexia nervosa (AN), Bulimia nervosa (BN), and Binge Eating Disorder (BED; von Ranson, Klump, Iacono, & McGue, 2005). The questionnaire is composed of items from multiple sources, including items adapted from the Eating Disorders Inventory that intend to measure Body Dissatisfaction, Bulimia, Drive for Thinness, and Interoceptive Awareness (von Ranson, Klump, Iacono, & McGue, 2005). The survey also measures compensatory behaviors, including the use of vomiting, laxatives, dietary supplements, and exercise for the purposes of weight control. Subscale scores are calculated by the summation of the total number of pathologically indicative responses within each subscale.
The aggregate sum of subscale scores indicates the total MEBS score for the participant (von Ranson, Klump, Iacono, & McGue, 2005).

**Validity and reliability.**

The MEBS is a valid and reliable measure of disordered eating attitudes and behaviors (von Ranson, Klump, Iacono, & McGue, 2005). The total score, as well as Body Dissatisfaction, Weight Preoccupation, and Binge Eating subscales, display high internal consistency. The MEBS shows high concurrent validity with the EDE-Q, and the finding that girls with eating disorders tended to have significantly higher scores than controls without eating disorders supports its criterion validity (von Ranson, Klump, Iacono, & McGue, 2005).

**Subscales.**

The data from the current study is taken from the intake responses of the MSUTR. We used score responses from the body dissatisfaction, binge eating, and weight preoccupation subscales adapted from the MEBS, and the dietary restraint subscale adapted from the EDE-Q.

**Dietary Restraint.**

The Dietary Restraint subscale is composed of items measuring restricted intake of food. The items include, “Have you been deliberately trying to limit the amount of food you eat to influence your shape or weight?”; “Have you gone for long periods of time (8 hours or more) without eating anything in order to influence your shape weight?”; “Have you tried to avoid eating any foods which you like in order to influence your shape or weight?”; “Have you tried to follow definite rules regarding your eating in order to influence your shape or weight; for example, a calorie limit, a set amount of food, or rules about what or when you should eat?”; and “Have you wanted your stomach to be empty?” (Fairburn & Bèglin, 1994). The response for each item ranges from 1 (*No Days*) to 7 (*Everyday*), where 7 (*Everyday*) indicates greatest
pathology. The mean score of the five items constitutes the subscale score, which ranges from 1 to 7.

**Body Dissatisfaction.**

The Body Dissatisfaction subscale is composed of items measuring negative attitudes regarding bodily appearance. The items include: “My stomach is too big”; “My thighs are about the right size”; “The shape of my body is fine”; “My butt (behind) is too big”; “I’m always wishing I was thinner”; and “My hips are just about the right size” (von Ranson, Klump, Iacono, & McGue, 2005). For each item, respondents may endorse either a response of 1 (True) or 2 (False). For the items “My stomach is too big”, “My butt (behind) is too big”, and “I’m always wishing I was thinner”, an endorsement of True indicates greater pathology. The remaining three items in the subscale, however, are reverse coded. The mean number of responses indicating pathology constitutes the subscale score, which ranges from 0 to 6.

**Binge Eating.**

The Binge Eating subscale is composed of items measuring discrete episodes of binge eating accompanied by feelings of loss of control and shame. The items include: “I eat when I’m upset about things”; “Sometimes I stuff myself with food”; “Sometimes I eat lots and lots of food and feel like I can’t stop”; “I think a lot about overeating”; “Sometimes, when I’m with other people I won’t eat much, but later, when I’m alone, I’ll eat a lot”; “Sometimes I eat by myself so that others won’t know what I’m eating”; and “When I get upset, I’m afraid that I will start eating” (von Ranson, Klump, Iacono, & McGue, 2005). For each of the seven items, respondents may endorse a response of 1 (True) or 2 (False). For all items, an endorsement of True indicates greater pathology. The mean number of True endorsements constitutes the subscale score, which ranges from 0 to 7.
**Weight Preoccupation**

The Weight Preoccupation subscale is composed of items that measure cognitive preoccupation with body weight and/or shape. The items include: “I often diet to control my weight”; “I think a lot about dieting (or losing weight); “I feel terribly guilty if I overeat”; “I am really afraid of gaining weight”; “I feel fat or stuffed even after eating a normal meal”; “If I gain a pound, I worry that I will keep gaining more and more weight”; “I often weight myself to see if I am gaining weight”; and “My weight is very important to me” (von Ranson, Klump, Iacono, & McGue, 2005). For each of the eight items, respondents may endorse either a response of 1 (True) or 2 (False). For all items, an endorsement of True indicates greater pathology. The mean number of True endorsements constitutes the subscale score, which ranges from 0 to 8.

**Analyses**

**Descriptive Analyses**

We conducted descriptive analyses on participant subscale scores in order to differentiate between monozygotic and dizygotic twin pair responses, as well as the correlations between the subscales. We next compared the intraclass correlations of the MZ and DZ twin groups to explore the extent to which genes, shared environments, and non-shared environments influenced body dissatisfaction, binge eating, weight preoccupation, and dietary restraint. Higher correlations between monozygotic twins than dizygotic twins implicated a genetic influence, whereas statistically significant and fairly equal correlations between monozygotic and dizygotic twin pairs implicated shared environmental influences (Klump, McGue, & Iacono, 2000). To the extent that the monozygotic correlation were less than 1, there was evidence of a non-shared environment.
Biometric Regression Analyses

We next fit cross-sectional univariate structural equation models to the intraclass twin correlations to decompose the variance among the twin responses into genetic (A), shared environmental (C), and non-shared environmental (E) contributions. There are three assumptions taken into account by twin model analyses: 1) monozygotic and dizygotic twins differ in their amount of shared genetic material; 2) common environments influence monozygotic and dizygotic twins equally; 3) non-shared environments within twin pairs consist of all influences that co-twins accrue independently of one another. Whereas monozygotic twins share 100% of their genetic material, dizygotic twins share, on average, 50% of their genes. Therefore, the additive effects of genes on twin phenotypes should be greater for monozygotic twins than for dizygotic twins (Klump, McGue & Iacono, 2000). The presence of additive genetic effects was indicated in the model when the co-variance between subscales approached 1.0 for monozygotic twins and 0.50 for dizygotic twins. Shared environments include all environments and influences common to both co-twins and were assumed to be equal between MZ and DZ twins. The model indicated the presence of shared environmental effects when co-variance among subscales for monozygotic twins approximately equaled that of dizygotic twins. The non-shared environment encompasses all influences on twin phenotype not already accounted for by genetic or shared environmental effects, including measurement error. Any differences between MZ twins (i.e., a correlation of less than 1.0) indicated evidence of non-shared environmental effects (Klump, McGue, & Iacono, 2000). Furthermore, twin model analyses assume that the genetic, shared environmental, and non-shared environmental effects occur independently of one another, and are thus, uncorrelated.
We next calculated and compared cross-twin correlations between scores for dietary restraint and scores for body dissatisfaction, weight preoccupation, and binge eating. Next, we decomposed binge eating, body dissatisfaction, and weight preoccupation into their respective A, C, and E effects. We then regressed dietary restraint on the genetic, shared environmental, and phenotypic components of binge eating, body dissatisfaction, and weight preoccupation (Klump, McGue, & Iacono, 2000). Demonstrated in Figure 1, we determined the phenotypic effect \( \beta_P \) of binge eating, body dissatisfaction, and weight preoccupation on dietary restraint, holding constant their nonrandom genetic effect \( \beta_A \) and nonrandom environmental effect \( \beta_C \).

**Model Fitting**

We fit two models to the subscale scores, which differed in their constraint of ACE factors. Model 1 (unconstrained) measured the phenotypic effect of body dissatisfaction, binge eating, and weight preoccupation on dietary restraint, whereas Model 2 (constrained) took into account the potential genetic effects on the phenotypic score, eliminating a single degree of freedom. We determined the model of best fit for each subscale (body dissatisfaction, weight preoccupation, binge eating, and dietary restraint) using the chi-square goodness-of-fit statistic \( \chi^2 \). Large statistically significant chi-square values were indicative of a poor fit, and a rejection of the model. If the initial chi-square test did not eliminate a model, the chi-square difference test \( \Delta \chi^2 \) was employed to establish the model of best fit, using the difference in degrees of freedom between the constrained and unconstrained models. A non-significant \( \Delta \chi^2 \) result indicated that the constrained model was better fitting (Klump, McGue, & Iacono, 2000). We also employed Mean Squared Error of Analysis (RMSEA) as an additional measure of best fit, with RMSEA values below .05 indicative of a good fitting model.
Results

Descriptive Results

Overall, scores on the binge eating, body dissatisfaction, weight preoccupation, and dietary restraint subscales were low, indicating that severe eating pathology was not generally present in this sample. Scores on the binge eating subscale ranged from 0 to 7.0 ($M = 1.08$, $SD = 1.44$); scores on the body dissatisfaction subscale ranged from 0 to 6.0 ($M = 1.48$, $SD = 1.94$); scores on the weight preoccupation subscale ranged from 0 to 8.0 ($M = 1.99$, $SD = 2.16$); and scores on the dietary restraint subscale ranged from 0 to 5.40 ($M = 0.59$, $SD = 1.01$).

Table 1 shows the cross twin subscale correlations for the monozygotic and dizygotic twin pairs. Monozygotic twins showed higher cross twin correlations on subscale scores for body dissatisfaction, binge eating, and weight preoccupation than dizygotic twins, indicating the potential for the presence of genetic influences. Dietary restraint cross twin correlations, however, were higher for DZ twins than for MZ twins, potentially indicative of shared environmental influences.

Table 2 shows the intercorrelations among the subscales for all twins, regardless of zygosity. Across the entire sample, correlations among dietary restraint and body dissatisfaction, binge eating, and weight preoccupation were moderate, ranging from 0.45 to 0.67. The dietary restraint subscale was low to highly moderately correlated with the other subscales, ranging from 0.31 to 0.67.

Biometric Results

Body dissatisfaction.

Table 1 shows the results of the biometric regression of dietary restraint on body dissatisfaction, binge eating and weight preoccupation. We found that Model 1, the phenotypic
model, better fit the Body Dissatisfaction subscale than Model 2, the genetic effect model, as there was no significant difference in fit between the two models ($\chi^2 = 25.13$, $\Delta \chi^2 = 2.80$, $\Delta df = 1$, $p = .094$). Both genes and non-shared environment contributed significantly to the variance in body dissatisfaction across participants ($A_{BD} = 2.37$, $SE = 0.33$; $E_{BD} = 1.38$, $SE = 0.18$).

Surprisingly, shared environment, did not contribute to the variance in body dissatisfaction ($C_{BD} = 0.00$, $SE = 0.00$).

We found that non-shared environment significantly moderated the effect of body dissatisfaction on dietary restraint, such that for every unit increase in body dissatisfaction, there was a 0.26 unit increase in dietary restraint ($SE = 0.03$). We found no evidence of moderating effects of genes and shared environment on the effect of binge eating on dietary restraint.

**Binge eating.**

We found that the A regression model, Model 2, which took into account genetic effects on phenotype, fit the data better than Model 1, as there were significant differences in fit between the two models ($\chi^2 = 30.14$, $\Delta \chi^2 = 3.24$, $\Delta df = 1$, $p = .036$). Both genetic and non-shared environmental processes contributed significantly to the variance in binge eating across participants ($A_{BE} = 0.89$, $SE = 0.21$; $E_{BE} = 1.10$, $SE = 0.09$). Shared environment did not contribute to the variance in body dissatisfaction ($C_{BE} = 0.00$, $SE = 0.00$).

We found a non-significant trend for both genes and non-shared environment to moderate the effect of binge eating on dietary restraint ($b_A = 0.29$, $SE = 0.16$; $b_E = 0.11$, $SE = 0.07$). We found no evidence of a moderating effect of shared environment on the effect of binge eating on dietary restraint.
Weight preoccupation.

We found that Model 1, the phenotypic model, better fit the Weight Preoccupation subscale than Model 2 as there was no significant difference in fit between the two models ($\chi^2 = 8.65, \Delta\chi^2 = 1.43, \Delta df = 1, p = .233$). Non-shared environment significantly contributed to the variance in weight preoccupation across participants ($E_{WP} = 2.34, SE = 0.31$). Genes also contributed to the variance in weight preoccupation, but not significantly ($A_{WP} = 2.13, SE = 1.22$). Shared environment did not contribute to the variance in weight preoccupation ($C_{WP} = 0.00, SE = 0.00$).

We found that non-shared environment significantly moderated the effect of weight preoccupation on dietary restraint, such that for every unit increase in weight preoccupation, there was a 0.31 unit increase in dietary restraint ($SE = 0.02$). We found no evidence of a moderating effect of genes or shared environment on the effect of binge eating on dietary restraint.

Discussion

The results of the current study indicate that body dissatisfaction, binge eating, and weight preoccupation independently predict dietary restraint in adolescent women. Controlling for genetic and environmental selection factors, we found that non-shared environmental processes moderated the effects of body dissatisfaction and weight preoccupation on dietary restraint. Additionally, there was a trend for both non-shared environmental experiences and genetic factors to influence the effect of binge eating on dietary restraint. These findings support our hypotheses that body dissatisfaction, binge eating, and weight preoccupation would predict dietary restraint, and that body dissatisfaction, binge eating, and weight preoccupation would predict dietary restraint, controlling for non-random selection processes.
Variance Components

Our findings indicate that genes and non-shared environments account for the variance in body dissatisfaction, binge eating, and weight preoccupation observed among adolescent women. These findings support the results of previous studies, which found that genes and non-shared environments accounted for overall variance in disordered eating attitudes and behaviors (Bulik, Sullivan, Wade, & Kendler, 2000; Wade, Martin, & Tiggemann, 1998). Furthermore, we found that shared environmental processes accounted for a small, non-significant portion of the variance in weight preoccupation, which supports the previous finding that twins’ shared environment accounts for a portion of the variance in weight preoccupation (Wade, Martin, & Tiggemann, 1998).

Body Dissatisfaction, Weight Preoccupation, and Dietary Restraint

The results from the current study indicate that non-shared environments moderate the effects of weight preoccupation and body dissatisfaction on dietary restraint. It is individual life experiences, therefore, not genetic or shared environments, that influence whether an individual with body dissatisfaction and weight preoccupation will progress to the behavior of dietary restraint. We propose two potential environments that may differ between co-twins, and contribute to differential influences of body dissatisfaction and weight preoccupation.

One environment that may be unique to each twin is her choice of peer and friendship group. Regardless of twin zygosity, twins may choose disparate friend groups that deviate on a number of factors, including personality characteristics, moral values, engagement in deviant behaviors, and eating behaviors and attitudes. For example, the friend group of one twin may overemphasize the importance of physical characteristics relative to the friend group of her co-twin. Because the friend group of the first twin is much more likely to internalize a culture-
driven “thin ideal”, she and her friends greatly increase their risk of developing disordered eating (Stice & Shaw, 2002).

A second environment that may affect one twin in a positive or negative manner relative to her co-twin, is a romantic relationship. Because both twins do not share a romantic partner, this experience may be an important unshared influence that distinguishes co-twins. The presence of a romantic relationship in one twin’s life may influence the development of disordered eating because a) a woman’s romantic partner is supportive and reaffirms her self-worth, acting as a protective factor for disordered eating (Stice & Whitenton, 2002), or b) a woman’s romantic partner is abusive or critical, leading her to negative self-appraisals that contribute to the onset of disordered eating (Thompson, Coover, Richards, Johnson, & Cattarin, 1995).

**Binge Eating and Dietary Restraint**

In the current study, we found that there was a non-significant trend for both genetic and non-shared environmental processes to moderate the effect of binge eating on dietary restraint. These findings may indicate that a gene-environment interaction is responsible for the onset of dietary restraint. This means that a woman who is predisposed to engage in dietary restraint as a result of binge eating, may only develop restrictive eating behaviors if she is exposed to an environmental stressor. Such an interaction explains why some women who binge eat develop the dietary restrictive behaviors seen in bulimia nervosa, whereas others do not.

Our finding that binge eating predicts dietary restraint, however, contrasts with the finding of Racine et al. (2011) that dietary restraint predicts binge eating. The authors offer two explanations for the disparity between these two findings. First, the contrasting results may be due to differences in construct measurement or heterogeneity of samples, warranting further
research to elucidate the relationship between binge eating and body dissatisfaction. Second, binge eating and dietary restraint may constitute a cyclical pathway. For example, binge eating may influence the onset of dietary restraint as a means of compensation for excessive food consumption. The ensuing dietary restriction may then lead to further episodes of binge eating, as predicted by the caloric intake or disinhibition hypotheses described above (Polivy, Heatherton, & Herman, 1988; Ruderman, 1986; Stice & Shaw, 2002), perpetuating the cycle. Such a reciprocal relationship between dietary restraint and binge eating explains the disparity in findings between the current study and that of Racine et al. (2011), but complicates the task of determining the initial behavior of onset.

**Implications**

The current study has important implications for the treatment of disordered eating behaviors and attitudes. Because we found that weight preoccupation, binge eating, and body dissatisfaction predict dietary restraint, we propose that dietary restraint may be both prevented and treated by indirectly targeting these disordered eating attitudes and behaviors. This conjecture is of clinical importance, as current psychological therapies, including Cognitive Behavioral Therapy and Interpersonal Therapy, more effectively treat non-restrictive components of disordered eating, such as body dissatisfaction, binge eating, and weight preoccupation, than the severe dietary restraint characteristic of anorexia nervosa (Peterson & Mitchell, 1999).

**Limitations**

Our study has several limitations of note. First, our sample was composed of a non-clinical university population. As a result, mean subscale scores for the disordered eating items were fairly low, indicating that women of the sample generally lacked severe eating pathology.
The low levels of eating pathology present in the sample may have both decreased the power of our analyses, and decreased the generalizability of our results to populations with clinical levels of disordered eating.

Another limitation concerns our ability to draw strong causal conclusions about the effects of body dissatisfaction, weight preoccupation, and binge eating on dietary restraint. Although biometric regression models allow us to control for genetic and shared environmental factors, there are a number of other selection factors that may be partially responsible for these effects. For example, although we attempted to control for all shared environments, sample demographic characteristics may have partially confounded our results.

A final limitation concerns the validity and reliability of the Minnesota Eating Behavior Survey (MEBS). Although the MEBS is composed of items adapted from other well-established measures of disordered eating, the questionnaire itself has not been subjected to extensive tests of validity and reliability. It is therefore possible that the construct validity and/or reliability of the MEBS may not be as accurate as other widely used disordered eating measures, such as the EDE-Q.

**Future Directions**

Future studies should address the generalizability and validity of the non-clinical sample analyzed for the present study. Conducting similar analyses on a clinical sample will indicate whether the findings of our study hold true across populations. In addition, eating disorder treatment may benefit from further genetically informed research of compensatory behaviors other than dietary restraint, such as self-induced vomiting, excessive exercise, and laxative and diuretic abuse. Because non-shared environments are implicated in the onset of dietary restraint,
future studies may also seek to examine the specific environments that may distinguish the effects of disordered eating between twins.

Summary

For the present study, we performed genetically informed analyses on a non-clinical sample of adolescent twins, in order to measure the effects of body dissatisfaction, binge eating, and weight preoccupation on dietary restraint. Holding constant non-random genetic and environmental factors, we found that non-shared environmental processes moderated the effects of weight preoccupation and body dissatisfaction on dietary restraint. Furthermore, we found a non-significant trend for genetic and non-shared environmental processes to moderate the effect of binge eating on dietary restraint. These findings indicate that targeting body dissatisfaction, binge eating, and weight preoccupation, may indirectly treat and reduce dietary restraint.
References


Instructions

The following questions are concerned with the PAST FOUR WEEKS ONLY (28 days). Please read each question carefully and circle the appropriate number on the right. Please answer all the questions.

<table>
<thead>
<tr>
<th>ON HOW MANY DAYS OUT OF THE PAST 28 DAYS……</th>
<th>No</th>
<th>1-5 days</th>
<th>6-12 days</th>
<th>13-15 days</th>
<th>16-22 days</th>
<th>23-27 days</th>
<th>Every day</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Have you been deliberately trying to limit the amount of food you eat to influence your shape or weight?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>2. Have you gone for long periods of time (8 hours or more) without eating anything in order to influence your shape weight?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>3. Have you tried to avoid eating any foods which you like in order to influence your shape or weight?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>4. Have you tried to follow definite rules regarding your eating in order to influence your shape or weight; for example, a calorie limit, a set amount of food, or rules about what or when you should eat?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>5. Have you wanted your stomach to be empty?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>6. Has thinking about food or its calorie content made it much more difficult to concentrate on things you are interested in; for example, read, watch TV, or follow a conversation?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>7. Have you been afraid of losing control over eating?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
</tbody>
</table>
ON HOW MANY DAYS OUT OF THE PAST 28 DAYS…….

8. Have you had episodes of binge eating where you ate a lot of food and felt like you couldn’t stop? 0 1 2 3 4 5 6

9. Have you eaten in secret? (Do not count binges.) 0 1 2 3 4 5 6

10. Have you definitely wanted your stomach to be flat? 0 1 2 3 4 5 6

11. Has thinking about shape or weight made it more difficult to concentrate on things you are interested in; for example, read, watch TV or follow a conversation? 0 1 2 3 4 5 6

12. Have you had a definite fear that you might gain weight or become fat? 0 1 2 3 4 5 6

13. Have you felt fat? 0 1 2 3 4 5 6

14. Have you had a strong desire to lose weight? 0 1 2 3 4 5 6

OVER THE PAST FOUR WEEKS (28 DAYS)

15. On what proportion of times that you have eaten have you felt guilty because of the effect on your shape or weight? (Do not count binges.) (Circle the number which applies.)

<table>
<thead>
<tr>
<th></th>
<th>No days</th>
<th>1-5 days</th>
<th>6-12 days</th>
<th>13-15 days</th>
<th>16-22 days</th>
<th>23-27 days</th>
<th>Every day</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
</tbody>
</table>

16. Over the past four weeks (28 days), have there been any times when you have felt that you have eaten what other people would regard as an unusually large amount of food given the circumstances? (Please put appropriate number in box.)

<p>| | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>0- No</td>
<td>[ ]</td>
<td>[ ]</td>
</tr>
<tr>
<td>1- Yes</td>
<td>[ ]</td>
<td>[ ]</td>
</tr>
</tbody>
</table>

17. How many such episodes have you had over the past four weeks? [ ] [ ] [ ] [ ]
18. During how many of these episodes of overeating did you have a sense of having lost
control over your eating?


Continued →

19. Have you had other episodes of eating in which you have had a sense of having lost control
and eaten too much, but have not eaten an unusually large amount of food given the
circumstances?

0- No
1- Yes

20. How many such episodes have you had over the past four weeks?


21. Over the past four weeks have you made yourself sick (vomit) as a means of controlling
your shape or weight?

0- No
1- Yes

22. How many times have you done this over the past four weeks?


23. Have you taken laxatives as a means of controlling your shape or weight?

0- No
1- Yes

24. How many times have you done this over the past four weeks?


25. Have you taken diuretics (water tablets) as a means of controlling your shape or weight?

0- No
1- Yes

26. How many times have you done this over the past four weeks?


27. Have you exercised hard as a means of controlling your shape or weight?

0- No
1- Yes

28. How many times have you done this over the past four weeks?


<table>
<thead>
<tr>
<th>Question</th>
<th>NOT AT ALL</th>
<th>SLIGHTLY</th>
<th>MODERATELY</th>
<th>MARKEDLY</th>
</tr>
</thead>
<tbody>
<tr>
<td>29. Has your weight influenced how you think about (judge) yourself as a person?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>30. Has your shape influenced how you think about (judge) yourself as a person?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>31. How much would it upset you if you had to weigh yourself once a week for the next four weeks?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>32. How dissatisfied have you felt about your weight?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>33. How dissatisfied have you felt about your shape?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>34. How concerned have you been about other people seeing you eat?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>35. How uncomfortable have you felt seeing your body; for example, in the mirror, in shop window reflections, while undressing or taking a bath or shower?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>36. How uncomfortable have you felt about others seeing your body; for example, in communal changing rooms, when swimming or wearing tight clothes?</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
</tbody>
</table>
Appendix B

Minnesota Eating Behavior Survey - Female Form

1) I can eat sweets and starches (like potatoes, pasta and bread) without feeling upset or nervous.  
   T   F

2) I often diet to control my weight.  
   T   F

3) My stomach is too big.  
   T   F

4) I eat when I’m upset about things.  
   T   F

5) I have thought about throwing up (vomiting) to lose weight.  
   T   F

6) Sometimes I stuff myself with food.  
   T   F

7) I think a lot about dieting (or losing weight).  
   T   F

8) My thighs are about the right size.  
   T   F

9) Sometimes I completely stop eating for more than a day to control my weight.  
   T   F

10) I feel terribly guilty if I overeat.  
    T   F

11) I am really afraid of gaining weight.  
    T   F

12) The shape of my body is fine.  
    T   F

13) Sometimes I use laxatives (like Exlax, Correctol, Phenamint or Nature’s Remedy) to control my weight.  
    T   F

14) My weight is very important to me.  
    T   F

15) Sometimes I eat lots and lots of food and feel like I can’t stop.  
    T   F

16) My butt (behind) is too big.  
    T   F

17) I sometimes use diet pills (like Dexatrim, Dietac or Acrutrim) to control my weight.  
    T   F

18) I’m always wishing I was thinner.  
    T   F
19) I think a lot about overeating (eating a really large amount of food).  

20) Sometimes I have a hard time telling if I’m hungry or not.  

21) I exercise to control my weight more than other women my age.  

22) My hips are just the right size.  

23) Sometimes, when I’m with other people I won’t eat much, but later, when I’m alone, I’ll eat a lot.  

24) I feel fat or stuffed even after eating a normal meal.  

25) If I gain a pound, I worry that I will keep gaining more and more weight.  

26) Sometimes I make myself throw up (vomit) to control my weight.  

27) Sometimes I eat by myself so that others won’t know what I’m eating.  

28) When I get upset, I’m afraid that I will start eating.  

29) I often weight myself to see if I am gaining weight.  

30) I sometimes use medicine that makes me lose water (diuretics like Sunril, Aqua-Ban, Pamprin, or Midol PMS) to control my weight.
Table 1

*Cross Twin Correlations Among Body Dissatisfaction, Binge Eating, Weight Preoccupation, and Dietary Restraint Subscales for Monozygotic and Dizygotic Twins*

<table>
<thead>
<tr>
<th>Zygosity</th>
<th>BD Intake</th>
<th>BE Intake</th>
<th>WP Intake</th>
<th>RE Intake</th>
</tr>
</thead>
<tbody>
<tr>
<td>MZ</td>
<td>0.66</td>
<td>0.46</td>
<td>0.53</td>
<td>0.23</td>
</tr>
<tr>
<td>DZ</td>
<td>0.27</td>
<td>-0.02</td>
<td>0.24</td>
<td>0.36</td>
</tr>
</tbody>
</table>

*Note.* MZ = Monozygotic; DZ = Dizygotic; BD = Body Dissatisfaction; BE = Binge Eating; WP = Weight Preoccupation; RE = Dietary Restraint
Table 2

*Intercorrelations Among Body Dissatisfaction, Binge Eating, Weight Preoccupation, and Dietary Restraint Subscales for Combined Monozygotic and Dizygotic Twins*

<table>
<thead>
<tr>
<th>Subscale</th>
<th>BD Intake</th>
<th>BE Intake</th>
<th>WP Intake</th>
<th>RE Intake</th>
</tr>
</thead>
<tbody>
<tr>
<td>BD Intake</td>
<td>1.00</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BE Intake</td>
<td>0.42</td>
<td>1.00</td>
<td></td>
<td></td>
</tr>
<tr>
<td>WP Intake</td>
<td>0.65</td>
<td>0.45</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>RES Intake</td>
<td>0.50</td>
<td>0.31</td>
<td>0.67</td>
<td>1.00</td>
</tr>
</tbody>
</table>

*Note.* BD = Body Dissatisfaction; BE = Binge Eating; WP = Weight Preoccupation; RE = Dietary Restraint
Table 3

*Results of Biometric Regression and Model Fit*

<table>
<thead>
<tr>
<th></th>
<th>Body Dissatisfaction</th>
<th>Binge Eating</th>
<th>Weight Preoccupation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Model 1 Phenotypic</td>
<td>Model 1 Phenotypic</td>
<td>Model 2 Phenotypic</td>
</tr>
<tr>
<td></td>
<td>Model 2 Regression</td>
<td>Model 2 Regression</td>
<td>Model 2 Regression</td>
</tr>
<tr>
<td><strong>Variance Components</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A</td>
<td>2.37 (0.33)</td>
<td>2.37 (0.33)</td>
<td>0.87 (0.21)</td>
</tr>
<tr>
<td>C</td>
<td>0.00 (0.00)</td>
<td>0.00 (0.75)</td>
<td>0.00 (0.00)</td>
</tr>
<tr>
<td>E</td>
<td>1.38 (0.18)</td>
<td>1.38 (0.18)</td>
<td>1.10 (0.09)</td>
</tr>
<tr>
<td><strong>Regression Coefficients</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>b_A</td>
<td>-</td>
<td>0.15 (0.09)</td>
<td>-</td>
</tr>
<tr>
<td>b_C</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>b_P</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Residual Covariances</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>σ(DR_1,DR_2)_{MZ}</td>
<td>0.08 (0.08)</td>
<td>0.07 (0.07)</td>
<td>0.20 (0.09)</td>
</tr>
<tr>
<td>σ(DR_1,DR_2)_{DZ}</td>
<td>0.16 (0.10)</td>
<td>0.16 (0.10)</td>
<td>0.37 (0.11)</td>
</tr>
<tr>
<td><strong>Model Fit</strong></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>RMSEA</td>
<td>0.07</td>
<td>0.07</td>
<td>0.09</td>
</tr>
<tr>
<td>CH_2, df (p)</td>
<td>27.93, 19</td>
<td>25.13, 18</td>
<td>33.38, 19</td>
</tr>
<tr>
<td>ΔCH_2, Δdf (p)</td>
<td>-</td>
<td>(.094)</td>
<td>-</td>
</tr>
</tbody>
</table>

*Note:* Standard errors are in parentheses. Parameter estimates significantly different zero (p < .05) are bolded.
Figure 1. Biometric Regression of Dietary Restraint on the Genetic (b_A), Shared Environmental (b_C), and phenotypic (b_P) effect of Body Dissatisfaction, Binge Eating, and Weight Preoccupation.