THE SAME BEAST OR DIFFERENT ANIMALS? EXAMINING DIFFERENTIAL ETIOLOGIC ASSOCIATIONS BETWEEN BINGE EATING AND COMPENSATORY BEHAVIOR WITH IMPULSIVITY AND PERFECTIONISM

By

Alexia Spanos, M.A.

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ABSTRACT

THE SAME BEAST OR DIFFERENT ANIMALS? EXAMINING DIFFERENTIAL ETIOLOGIC ASSOCIATIONS BETWEEN BINGE EATING AND COMPENSATORY BEHAVIOR WITH IMPULSIVITY AND PERFECTIONISM

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Despite the use of binge eating and compensatory behavior in distinguishing diagnostic categories and subtypes of eating disorders, little research has examined the etiologic validity of distinguishing among disorders/subtypes based on these phenotypes. The current project used cross-sectional and longitudinal data to examine etiologic overlap between binge eating and compensatory behavior and explore impulsivity as a differentiating factor in the relationship between these variables. Participants included 1,434 female twins from two twin registries. Binge eating and compensatory behavior were assessed with the Minnesota Eating Behavior Survey. Impulsivity was assessed with the control subscale of the Multidimensional Personality Questionnaire. Pearson correlations were used to examine phenotypic associations and trivariate Cholesky decompositions were used to explore etiologic associations between impulsivity, binge eating and compensatory behaviors (cross-sectionally and across-time). Cross-sectional and longitudinal findings indicated a small-to-moderate degree of overlap between binge eating and compensatory behavior. In addition, although overlap with impulsivity was generally small for both phenotypes, greater phenotypic and etiologic associations were found between impulsivity and compensatory behavior. Genetic relatedness appears to account for more overlap between the three variables than nonshared environmental influences, with compensatory behavior sharing greater overlap with impulsivity than binge eating. However, residual estimates are substantial, indicating most of the etiology of binge eating and compensatory behavior is

unaccounted for by impulsivity. Substantial etiologic uniqueness in binge eating and compensatory behavior suggests merit in the addition of diagnostic categories that focus on one behavior in the absence of the other (e.g., purging disorder). Findings also indicate impulsivity may be a differentiating factor between these disordered eating phenotypes.

DEDICATION

I dedicate this project to my husband, Jesse Bledsoe for your unwavering support and infinite patience throughout graduate school and during the completion of this dissertation. You are my best friend, my strongest ally and my fiercest advocate. Thank you for pulling me through when I wanted to give up, and for believing in me when I lost faith in myself. I promise to spend our lifetime trying to match the same love and support you have shown me. You made this all worth it.

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I am forever grateful to my family for their unconditional love and faith in my abilities. Their support and cheerleading during the hardest of times was immeasurable. I owe my parents, Aris and Evie for instilling in me a work ethic and a desire to achieve that has propelled me through 10+ years of school! I am also grateful for the patience and understanding of my sisters, Stella and Marina. Thank you for always answering the hysterical and panic-stricken calls, I promise, they will lessen now.

PREFACE

My dissertation is entitled "Same Beast or Different Animals? Examining Differential Etiologic Associations between Binge Eating and Compensatory Behavior with Impulsivity and Perfectionism" and is comprised of two papers. The first is entitled "Impulsivity as a Potential Differentiating Factor between Binge Eating and Compensatory Behavior." The aim of this paper was to explore overlap between binge eating and compensatory behavior at the phenotypic and etiologic level. Further, once the degree of overlap was established, my goal was to examine whether the personality trait of impulsivity is differentially related to binge eating and compensatory behavior. I also aimed to establish whether impulsivity acts as a differential risk factor for binge eating and compensatory behavior using longitudinal data.

Once a fair amount of unique variance in binge eating and compensatory behavior was established, the second paper further explored personality as a potential factor that could account for some of this unique variance. Specifically, I examined perfectionism as a variable that may account for some of the variance in binge eating or compensatory behavior that is independent of the other. The second paper is entitled "A Multivariate Twin Model of Perfectionism and its Relationship to Binge Eating and Compensatory Behavior."

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PAPER 1 INTRODUCTION

Two key defining features of eating disorders are binge eating (i.e., the consumption of an unusually large amount of food in a short period of time with a sense of loss of control) and the use of inappropriate compensatory behavior (American Psychiatric Association, 2000). Compensatory behaviors include both purging (i.e., self-induced vomiting, diet pill abuse or the abuse of laxatives, enemas or diuretics) and non-purging (i.e., fasting, strict dieting) behaviors for weight loss or to prevent weight gain. The current diagnostic system distinguishes between diagnostic categories and subtypes of eating disorders based on the presence or absence of binge eating and compensatory behavior. For example, while both bulimia nervosa and binge eating disorder involve recurrent binge eating episodes, only bulimia nervosa includes compensatory mechanisms to counteract these episodes of overeating. The Diagnostic and Statistical manual of Mental Disorders Fourth Edition (DSM IV) also uses binge eating and purging behaviors to subtype individuals within the same diagnostic category. For example, individuals with bulimia nervosa are categorized as either having the purging (i.e., regularly engaging in self-induced vomiting, misuse of laxatives, diet pills, diuretics or enemas) or non-purging (i.e., use of inappropriate compensatory behavior without the inclusion of methods of purging) form of the disorder. Similarly, individuals with anorexia nervosa are frequently categorized as having restrictor type (i.e., no binge eating or purging) or binge/purge type (binge eating *and/or* purging) (American Psychiatric Association, 2000).

The distinction made between binge eating and compensatory behavior in the DSM is supported by data from studies comparing the course and treatment of disorders characterized by binge eating and compensatory behavior (Raymond, Mussell, Mitchell, De Zwaan, & Crosby, 1995; Wilfley, Wilson, & Agras, 2003). Studies investigating differences between bulimia

nervosa (i.e., a diagnosis that includes binge eating and compensatory behavior together) and binge eating disorder (i.e., a diagnosis that excludes compensatory behavior) find evidence for greater symptom severity (e.g., more frequent binge eating) and greater treatment resistance in bulimia nervosa (Raymond, et al., 1995; Wilfley, et al., 2003). Similarly, studies comparing purging disorder (i.e., the presence of regular episodes of purging in the absence of low weight and binge eating) to bulimia nervosa found differences in levels of anxiety (i.e., higher in those with purging disorder) and gastric function (i.e., greater dysfunction in those with bulimia nervosa in response to a test meal) (Keel, Haedt, & Edler, 2005; Keel, Wolfe, Gravener, & Jimerson, 2008; Keel, Wolfe, Liddle, De Young, & Jimerson, 2007). Importantly, differences between diagnostic categories do not seem to be an artifact of the presence of more disordered eating behaviors in one disorder versus the other (e.g., greater pathology in bulimia nervosa than binge eating disorder). Indeed, despite a better prognosis, individuals with binge eating disorder tend to report an earlier onset of binge eating behaviors and a longer mean course of illness (i.e., 14.4 versus 5.8 years) than individuals with bulimia nervosa (Pope et al., 2006; Stunkard & Allison, 2003).

Despite differences in course and treatment of binge eating and compensatory behavior, little research has examined etiologic associations between these phenotypes. Etiologic associations would provide one more piece of evidence to support the validity of using binge eating and compensatory behavior to differentiate diagnostic schemas. Exploring this validity may be particularly important given the upcoming fifth edition of the DSM. Specifically, researchers and clinicians are struggling with whether or not to continue the current subtyping for anorexia nervosa (i.e., restricting versus purging) and bulimia nervosa (i.e., purging versus non-purging), and whether binge eating disorder (i.e., regular binge eating without any

compensatory behavior) should be included as a separate and distinct disorder. In addition, researchers and clinicians are considering adding purging disorder (i.e., a diagnosis where purging occurs in the absence of binge eating) as a separate diagnosis. Exploring the etiologic overlap between binge eating and compensatory behavior could provide additional data to consider in answering these diagnostic questions. Specifically, if these two phenotypes share substantial etiologic overlap, it might not be appropriate to separate disorders by binge eating-only or purging-only behaviors, since they may be differing expressions of the same underlying phenotype. Instead, in these cases, it might be more appropriate to have the disorder defined as featuring binge eating and/or compensatory behavior, but not requiring both or neither. If, on the other hand, there is little overlap between the two phenotypes, we have additional data to support the validity of creating separate diagnostic categories based on the presence of binge eating (i.e., binge eating disorder) or purging (i.e., purging disorder). One way to explore this validity would be to examine phenotypic and etiologic overlap between binge eating and compensatory behaviors (Robins & Guze, 1970).

Phenotypic and Etiologic Associations between Binge Eating and Compensatory Behavior

Phenotypic associations provide initial estimates of the degree of overlap between binge eating and compensatory behavior. Intercorrelations among binge eating and compensatory behavior subscales on common self-report questionnaires (e.g., Minnesota Eating Behavior Survey; (von Ranson, Klump, Iacono, & McGue, 2005)) suggest a moderate association between these phenotypes (i.e., $r = .29$-$.36$) in cross-sectional, community samples of females between the ages of 17 and 20 (von Ranson, et al., 2005). Moderately high estimates of the phenotypic relatedness of lifetime binge eating (i.e., ever engaged in binge eating) and lifetime self-induced vomiting (i.e., ever used vomiting to control weight) were found in a population-based sample of

adult (i.e., mean age 35.1) female twins (i.e., odds ratio=8.78, $p< 0.0001$) (Sullivan, Bulik, & Kendler, 1998). Finally, studies exploring phenotypic associations in clinical samples also find evidence for overlap in these behaviors. For example, Fairburn et al. (2003) found earlier compensatory behavior significantly predicted binge eating behaviors five years later (relative risk ratio=2.6, $p<.0001$), and vice versa (i.e., earlier binge eating predicted compensatory behavior five years later; relative risk ratio=3.0, $p<.0001$) (Fairburn, et al., 2003).

Taken together, studies thus far suggest a moderate-to-high degree of phenotypic association between binge eating and compensatory behavior. However, comparisons across studies are difficult because of the varied statistics used (e.g., correlations versus odd ratios). In addition, the number of studies that have investigated this issue is small, suggesting further research on the phenotypic relatedness of binge eating and compensatory behavior is warranted. In addition to investigating phenotypic associations, examining etiologic associations between binge eating and compensatory behavior would be important in determining their organization within diagnostic schemas. Twin studies can be particularly useful in this regard as they provide estimates of the degree to which genetic and environmental factors are shared between these behaviors, as well as "residual" estimates that index the extent of unique risk that are specific to that phenotype (i.e., independent of other variables in a twin model). If residual estimates are large, this would indicate a substantial amount of unique variance in each phenotype, and lend credence to the opinion that by adding separate diagnostic categories for binge-only or purge-only behaviors, we may be "cleaving nature at its joints" a bit more closely. If, on the other hand, residual estimates are small, this would suggest more etiologic overlap between the two behaviors, and provide one piece of data that disorders with one behavior only or both behaviors are presenting different expressions of the same underlying syndrome.

Looking at both behaviors separately, most population-based twin studies provide evidence for significant genetic influences on both binge eating (i.e., 50-82%) and compensatory behavior (i.e., 46-50%), with nonshared environmental factors (i.e., experiences that are unique to each twin in a pair and that make twins dissimilar) accounting for the remaining variance (Bulik, Sullivan, & Kendler, 1998, 2003; Klump, McGue, & Iacono, 2000; Reichborn-Kjennerud et al., 2003; Reichborn-Kjennerud, Bulik, Tambs, & Harris, 2004; Root et al., 2010; Sullivan, et al., 1998; Wade, Bulik, Sullivan, Neale, & Kendler, 2000). These findings are broadly representative of the magnitude and type of genetic/environmental influences that have been found for other types of disordered eating symptoms in adulthood (Klump, McGue, et al., 2000; Rutherford, McGuffin, Katz, & Murray, 1993; Wade, Martin, & Tiggemann, 1998).

Unfortunately, very few twin studies have investigated the extent to which genetic and nonshared environmental influences may overlap between binge eating and compensatory behavior. To date, only two studies have investigated this issue for binge eating and compensatory behavior (Sullivan, et al., 1998; Wade, Treloar, & Martin, 2008). Both Sullivan et al. (1998) and Wade et al. (2008) found substantial genetic correlations (r_g =.74-1.00) between the two phenotypes, suggesting that genetic influences that increase the liability to binge eating and compensatory behavior overlap substantially. The presence of moderate nonshared environmental correlations (r_e =.29-.48) also suggests nonshared environmental influences that increase liability to binge eating and purging (i.e., self-induced vomiting) overlap more modestly. Shared environmental factors (i.e., experiences that are shared among twins in a pair that make them more similar) accounted for none of the variance or overlap in these behaviors.

Overall, etiologic correlations illustrate there is etiologic overlap between the two phenotypes, particularly with regard to genetic influences, suggesting perhaps binge eating and

compensatory behavior may be differing expressions of the same syndrome. However, given nonshared environmental correlations were more modest, it appears there may also be some etiologic uniqueness in each behavior, supporting the opinion that these behaviors can be used to differentiate distinct syndromes where binge eating and compensatory behavior co-occur (e.g., as in bulimia nervosa) and where they do not (e.g., as in binge eating disorder).

Given that only these two studies have investigated this issue, additional research may be helpful in clarifying the degree of etiologic association between binge eating and compensatory behavior. Importantly, despite similarities in etiologic correlations between Sullivan et al. and Wade et al.'s investigations, neither study included residual estimates in their reports. Residual estimates would provide useful information about the degree of etiologic distinctiveness between the two behaviors.

Phenotypic and Etiologic Associations with Impulsivity

In addition to exploring overlap between binge eating and compensatory behavior, it may also be helpful to explore potential *differentiating factors* that may distinguish binge eating and compensatory behavior at the phenotypic and etiologic level. Identifying these differentiating factors at both the phenotypic and etiologic level may help gain a better understanding of the degree of overlap between the two phenotypes.

One potential differentiating factor to explore is the personality trait of impulsivity, given the well-established link between disordered eating and this trait in the literature (Fahy & Eisler, 1993; Vitousek & Manke, 1994; Waxman, 2009; Wonderlich & Mitchell, 1997). Indeed, impulsivity has been linked to both binge eating and compensatory behaviors, although there is some debate as to whether impulsivity is more strongly associated with one behavior or the other. For example, previous personality theories suggest that impulsivity may be more strongly

linked to binge eating than compensatory behavior, as impulsive individuals are generally more reward seeking (i.e., seek out rewards from their environment) than non-impulsive individuals (Nederkoorn, Braet, Van Eijs, Tanghe, & Jansen, 2006), and binge eating is considered one of many rewarding behaviors impulsive individuals may engage in (particularly as a coping strategy in response to negative emotions, see Anestis, Smith, Fink, & Joiner, 2009; Cyders & Smith, 2008; Fischer, Smith, & Cyders, 2008). Empirical data is limited, but provide some support for these theories (Grucza, Przybeck, & Cloninger, 2007; Keel, Holm-Denoma, & Crosby, 2009; Nasser, Gluck, & Geliebter, 2004; Steiger, Puentes-Neuman, & Leung, 1991). For example, a study assessing binge eating and compensatory behavior in a community sample of women found significantly higher mean impulsivity scores for individuals who engaged in binge eating (with or without purging) compared to those who only engaged in compensatory behaviors without binge eating (i.e., $F(4, 500) = 19.81$, $p < .0001$) (Steiger, et al., 1991). Similarly, in comparisons across diagnostic categories, individuals who engaged only in binge eating (e.g., binge eating disorder) were more impulsive than those who engaged in binge eating and compensatory behaviors (e.g., bulimia nervosa) or purging alone (Keel, et al., 2009; Klump et al., 2000; Nasser, et al., 2004; Raymond, et al., 1995). Finally, studies using Q-sort procedures found evidence for significant associations between undercontrolled personality traits (characterized by impulsivity) and binge eating (Wade, Crosby, & Martin, 2006; Westen & Harnden-Fischer, 2001), as undercontrolled/impulsive Q-scores are significant predictors of binge frequency but not purge frequency.

Nonetheless, other studies find less support for the link between binge eating and impulsivity (Favaro et al., 2005; Fink, Smith, Gordon, Holm-Denoma, & Joiner, 2009; Klump, McGue, & Iacono, 2002). Instead, compensatory behavior emerged as more strongly associated

with impulsivity, possibly due to a link between anxiety and impulsivity. It has been theorized that individuals who use compensatory behavior to lose weight or prevent weight gain are doing so to alleviate the anxiety associated with eating and the subsequent fear of weight gain (Byrne & McLean, 2002). However, the act of engaging in compensatory behaviors is by its very nature an impulsive response that suggests an inability to inhibit action and lack of evaluation of consequences (Ho, Mobini, Chiang, Bradshaw, & Szabadi, 1999). Thus, it follows that the impulsive act of engaging in compensatory behaviors to lose weight or prevent weight gain is a direct response to the anxiety associated with eating in those with an eating disorder. Indeed, despite the more common belief that anxiety results in inhibitory responses and cautious action, others have hypothesized a positive relationship between anxiety and impulsivity (i.e., the presence of anxiety may actually lead to impulsive action) (Taylor et al., 2008). This link has been supported in a wide range of diagnoses including bipolar disorder, major depressive disorder and anxiety disorders (Bellani et al., 2012; Del Carlo et al., 2012; Taylor, et al., 2008).

Studies have found evidence for the proposed link between impulsivity and compensatory behavior. For example, in a study by Favaro et al. (2005) purging emerged as the only factor to significantly predict impulsivity. Similarly, Fink, Smith, Gordon, Holm-Denoma and Joiner et al. (2009) found higher levels of impulsivity among individuals with purging disorder as compared to those with binge eating disorder. Finally, Klump et al. (2002) found stronger phenotypic and etiologic associations between impulsivity and compensatory behavior than impulsivity and binge eating. Binge eating showed no significant phenotypic correlations with impulsivity (i.e., r=-.07, ns; measured using the inverse score on the Multidimensional Personality Questionnaire (MPQ) Constraint factor), and thus, twin analyses examining etiologic associations were not conducted (Klump, et al., 2002). By contrast, compensatory behavior

showed significant, albeit modest, phenotypic (i.e., r= -.15, p < .001) correlations with Constraint. Although this association was of small effect (Cohen, 1988), bivariate twin analyses revealed a small but significant degree of genetic overlap between compensatory behavior and Constraint (i.e., r=-.23). The nonshared environmental correlation between Constraint and compensatory behavior was negligible (Klump, et al., 2002). Critically, residual estimates indicated that the majority of the genetic influences on compensatory behavior were independent of Constraint (i.e., total heritability=.56 and residual heritability=.53). In other words, the relationship between compensatory behavior and Constraint was primarily mediated through genetic influences, but the actual proportion of genetic variance in compensatory behavior that was accounted for by Constraint was small (h^2_a=.03). Instead, other genetic influences that are unique to compensatory behavior accounted for the majority of genetic variance in this phenotype.

Overall, studies examining relationships between impulsivity, binge eating, and compensatory behavior are inconclusive. While phenotypic associations for most studies indicate greater overlap between impulsivity and binge eating, the only twin study to examine etiologic associations suggests no association with binge eating, and greater overlap with compensatory behavior. Thus, more research examining both the phenotypic and etiologic relationship between these variables may help clarify both the relationship between binge eating and compensatory behavior and the role of impulsivity in this relationship.

Given all of the above, the present study sought to extend previous research by examining phenotypic and etiologic associations between binge eating and compensatory behavior and investigating impulsivity as a differentiating factor in these associations. Using twins from the Minnesota Twin Family Study (MTFS) and the Michigan State University Twin

Registry (MSUTR), these associations were explored in cross-sectional and longitudinal analyses. The inclusion of longitudinal data in addition to cross-sectional analyses significantly extends previous work by exploring whether impulsivity is a pre-existing factor that differentiates the two behaviors across time. To date, no studies have examined this possibility, despite the potential importance of these data for understanding the extent to which impulsivity is a differentiating *risk factor* for binge eating and compensatory behaviors.

PAPER 1 METHOD

Participants

Participants included an archival sample of 1,434 female twins drawn from the Minnesota Twin Family Study (MTFS; *N*=1112) and the Michigan State University Twin Registry (MSUTR; *N*=322). The MTFS is a population-based, longitudinal study that includes two cohorts of reared-together, female twins and their parents. The 11-year-old twin cohort includes same-sex female twins who were approximately 11 years of age (*M*=11.78, *SD*=.46) at the time of the intake assessment. The 17-year-old cohort includes same-sex female twins who were approximately 17 years old (*M*=17.46, *SD*=.50) during the intake assessment. Follow-up assessments for both cohorts have subsequently occurred every three years since baseline, with data available up to ages 25 and 28, respectively. For longitudinal analyses, data from both cohorts at ages 17 and 25 (see Statistical Analyses section below for additional details) were used (*N*=1540). For cross-sectional analyses, the present study used participants from the 11-year-old cohort at follow-up four, and from the 17-year-old cohort at follow-up two, when both groups of participants are approximately 25 years old (i.e., 11-year-old cohort *M*=25.15, *SD*=.66 and 17-year-old cohort *M*=25.00, *SD*= .72). This age group was chosen for both cohorts because research suggests the peak period of risk for disordered eating increases through young adulthood and then plateaus by age 25 (Stice, Killen, Hayward, & Taylor, 1998; Woodside & Garfinkel, 1992). If participants are at a mean age of 25, it is likely that most disordered eating symptoms have manifested by the time of the assessment. Similarly, given personality development continues to change during adolescence and into young adulthood (i.e., increasing in stability during this time and remaining relatively stable closer to age 30), conducting cross-sectional analyses at age 25 ensures greater stability of personality traits as well (Caspi, Roberts

and Shiner, 2005). Finally, because binge eating and compensatory behavior are lower prevalence disordered eating behaviors (particularly compensatory behavior), combining samples at the maximum point of risk will also maximize sample size.

MTFS twins were identified through birth records. Recruitment efforts resulted in successfully contacting 90% of all twins born in Minnesota between 1971-1985, with 82.7% of those contacted agreeing to participate. The twins and their parents came to the University of Minnesota laboratory for a full day of assessments. Families were excluded from participation if they were adopted, lived further than a day's worth of driving away from the University of Minnesota, one or both twins were deceased, or had mental or physical handicaps that would preclude them from carrying out the assessments. Retention rates were excellent, with 88% of the original sample retained over a six-year period.

Importantly, there are no significant differences in rates of parental psychopathology or self-reported socioeconomic status between MTFS participants and other families in Minnesota with similar- aged adolescents (Iacono, Carlson, Taylor, Elkins, & McGue, 1999). MTFS twins are representative of the ethnic composition of the state at the time (i.e., over 95% are Caucasian). For more complete details on sampling strategy, recruitment and the demographics of this sample, see Iacono et al. (1999).

Participants from the MSUTR are reared together, same-sex female twins between the ages of 16 and 30. Although the pilot sample of MSUTR twins was recruited through several different mediums (university registrar's offices, advertisements, flyers, birth records), recruitment for the MSUTR is now entirely done through birth records. Approximately 70% of twins included in the current sample are from the pilot sample of twins, while the remaining 30% were recruited through birth records in collaboration with the Michigan Department of

Community Health (MDCH). Importantly, the MSUTR sample included in this study has been shown to be highly representative of the ethnic composition of Michigan (i.e., 82.8% Caucasian, 11.4% African-American, 1.7% Hispanic, 1.4% Asian/Asian-American, and 2.8% "Other") (Culbert, Burt, McGue, Iacono, & Klump, 2009). For more complete details on sampling strategy, recruitment and the demographics of this sample, see Klump and Burt (2006).

Zygosity Determination

Both the MTFS and the MSUTR used the same standard physical similarity questionnaire to determine twin zygosity. Responses to the questionnaire are scored for the probability that the twins are monozygotic (MZ) versus dizygotic (DZ) based on the degree of physical similarity between them. This questionnaire was completed by trained research staff in both samples and has an accuracy rate of over 95% (Lykken, Bouchard, McGue, & Tellegen, 1990). Both twin registries used additional methods to confirm zygosity. The MSUTR had twins complete a self-report version of the physical similarity questionnaire, and responses between twins and staff were compared for accuracy. In addition, one of the MSUTR directors' (KLK or SAB) evaluated questionnaire information and photographs of the twins (if available) for added accuracy. The MTFS used an algorithm of aggregated data from DNA markers to assess twin zygosity (see Iacono et al., 1999 for further details).

Measures

Disordered eating symptoms.

Binge eating and compensatory behavior. Binge eating and inappropriate compensatory behavior were measured using the Minnesota Eating Behavior Survey (MEBS) [1]. The MEBS is a

[1] The Minnesota Eating Behavior Survey (MEBS; previously known as the Minnesota Eating Disorder Inventory (M-EDI)) was adapted and reproduced by special permission of Psychological Assessment Resources, Inc., 16204 North Florida Avenue, Lutz, Florida 33549,

30-item true/false self-report questionnaire assessing current disordered eating attitudes and behaviors in individuals as young as 10. Exploratory factor analysis of MEBS resulted in four factors (Klump, McGue, et al., 2000; von Ranson, et al., 2005) in addition to the total score: body dissatisfaction (i.e., dissatisfaction with size and/or shape of body), weight preoccupation (i.e., preoccupation with dieting or weight), binge eating (i.e., thinking about or engaging in binge eating), and compensatory behavior (i.e., thinking about or engaging in inappropriate compensatory behavior for weight control).

The present study focused on the MEBS binge eating (7 items) and compensatory behavior (6 items) subscales. The binge eating and compensatory behavior subscales from the MEBS exhibit adequate psychometric properties for assessing disordered eating in community-based samples (von Ranson, et al., 2005). Discriminant validity for the compensatory behavior scale is excellent, as females with an eating disorder scored significantly higher on this subscale compared to controls (Klump, McGue, et al., 2000; von Ranson, et al., 2005). In addition, the binge eating subscale also discriminates between specific eating disorder diagnoses, as it differentiates individuals with bulimia nervosa from controls as well as individuals with other eating disorder diagnoses that do not include binge eating (von Ranson, et al., 2005). Three-year test-retest reliability statistics for binge eating and compensatory behavior are adequate, ranging from .30 to .50 (all significant at $p < .01$). These reliability estimates are impressive given the length of time between assessments (i.e., three years). Internal consistency for the binge eating and compensatory behavior scales have been adequate in previous studies (i.e., $\alpha = .65$-$.69$) (Klump, McGue, et al., 2000; von Ranson, et al., 2005).

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from the Eating Disorder Inventory (collectively, EDI and EDI-2) by Garner, Olmstead, Polivy, Copyright 1983 by Psychological Assessment Resources, Inc. Further reproduction of the MEBS is prohibited without prior permission from Psychological Assessment Resources, Inc.

In the present study, internal consistency estimates were good for binge eating (α= .71-72) but lower for compensatory behavior than in previous studies (α= .53-.60). Internal consistency estimates tend to be lower for compensatory behavior because it is a low base-rate event, with low item endorsement among community samples. In an attempt to improve internal consistency estimates, alphas were run excluding one or more items from analyses. Unfortunately, excluding items did not improve the internal consistency estimates, and thus, the full scale was used in the present study. However, the decision to use twins in young adulthood was, in part, determined by slightly improved internal consistency estimates at this age in the present sample.

Impulsivity.

Multidimensional personality questionnaire. The control facet scale of the Multidimensional Personality Questionnaire (MPQ) (Tellegen & Waller, 2008) was used to assess the personality characteristic of impulsivity. Different versions of the MPQ were administered in the MTFS and the MSUTR. In the MTFS, a shortened 198-item version of the full MPQ was used. The MSUTR used both the full MPQ (i.e., 300 item) and the MPQ-Brief (i.e., 155 item; (Patrick, Curtin, & Tellegen, 2002)). Fortunately, the 13 items that comprise the control facet scale from the MPQ-Brief are also included in the other versions of the MPQ. Thus, only these 13 items that are identical across all versions of the MPQ will be used to calculate a final score on the control facet scale.

The control subscale assesses one's level of cautiousness, planning and level-headedness (Tellegen & Waller, 2008). High scores on this scale indicate rational planning, while low scores on this scale indicate lack of planning and impulsivity (Tellegen & Waller, 2008). In the present study, the control subscale was reverse scored so that higher scores reflect greater impulsivity.

Previous studies support the use of the control scale from the MPQ as a measure of impulsivity (Caspi & Silva, 1995; Hur & Bouchard, 1997; Slutske, Caspi, Moffitt, & Poulton, 2005; Tellegen & Waller, 2008), as individuals typically characterized by greater levels of impulsivity (i.e., excessive gambling and substance use disorders) score high on this measure. Previous studies have found good internal consistency estimates for the control scale (α=.74-.83; (Patrick, et al., 2002)). Estimates from the present study corroborate previous estimates (α=.78-.80). In addition, concurrent validity of the control scale is good, as it shows a significant inverse correlation with Buss and Plomin's (1975) Impulsivity scale (r=-.48, p<.05; (Patrick, et al., 2002)). Importantly, studies examining personality traits in eating pathology have previously used control from the MPQ as a measure of impulsivity (Lilenfeld et al., 2000; Pryor & Wiederman, 1996),

Statistical Analyses

Cross-sectional analyses.

Pearson correlations were computed to examine within-person, phenotypic associations between binge eating and compensatory behavior and between impulsivity, binge eating and compensatory behavior. Intraclass twin correlations were computed for MZ and DZ twins to provide preliminary evidence for the extent to which genetic and environmental factors influence binge eating, compensatory behavior, and impulsivity separately. Importantly, intraclass twin correlations help identify the most important genetic and environmental (i.e., additive genetic (A), shared environmental (C) and nonshared environmental influences (E)) estimates for multivariate models. Twin correlations are based on the assumption that because MZ twins share 100% of their genetic material and DZ twins share, on average, 50% of their genetic material (i.e., similar to non-twin siblings), MZ and DZ twin correlations can be compared to reveal the presence of genetic and/or environmental variance in that trait. If MZ twin correlations are

approximately twice that of DZ twin correlations, this suggests the presence of additive genetic influences. If MZ twin correlations are approximately equal to those of DZ twin correlations, the presence of shared environmental influences is supported. Finally, if MZ twin correlations are less than 1.00, this suggests the presence of nonshared environmental influences (and measurement error).

Cross-twin, cross-trait correlations (e.g., Twin 1's binge eating with Twin 2's compensatory behavior) were then computed between binge eating and compensatory behavior (e.g., Twin 1's binge eating with Twin 2's compensatory behavior) and between disordered eating variables (i.e., binge eating and compensatory behavior) and impulsivity (e.g., Twin 1's binge eating with Twin 2's impulsivity). If MZ cross-twin, cross-trait correlations are significantly higher than those of DZ twins, then shared genetic effects likely contribute to the phenotypic associations between binge eating and compensatory behavior and between disordered eating and impulsivity. If, on the other hand, MZ cross-twin, cross-trait correlations are similar to those of DZ twins, shared environmental factors primarily contribute to these phenotypic associations. Finally, if within-twin correlations (i.e., the phenotypic associations for each participant in the sample) are greater than the cross-twin, cross-trait correlations, nonshared environmental influences are likely significant in the relationship between binge eating and compensatory behavior and disordered eating and impulsivity.

Prior to any biometric modeling on the full, combined sample of MTFS and MSUTR twins, constraint models were run in order to verify that additive genetic and environmental (i.e., shared and nonshared) estimates do not differ across samples. The comparability of samples was determined by running both a fully constrained model (i.e., constraining genetic and environmental estimates across the MTFS and MSUTR samples to be equal) and a fully

unconstrained model (i.e., allowing genetic and environmental estimates across the MTFS and MSUTR samples to freely vary) and then determining the best-fitting model. Results indicated the best-fitting model was one that constrained all of the genetic and environmental estimates to be equal across samples (data now shown). These findings corroborate those from previous studies of MSUTR and MTFS samples and provided support for aggregating the two samples in remaining analyses.

Cholesky decompositions.

A trivariate (i.e., three-factor), Cholesky decomposition was used to examine genetic and environmental variance in, and covariance between, binge eating, compensatory behavior and impulsivity. Cholesky decomposition is a multivariate data-analysis technique that is based on the principles of factor analysis. The Cholesky decomposition provides estimates of the relative additive genetic (A), shared environmental (C) and nonshared environmental (E) influences on the variance in, and the covariance between, impulsivity, binge eating and compensatory behavior. The ACE trivariate, Cholesky decomposition is depicted in Figure 1. This model decomposes the genetic and environmental influences between binge eating and compensatory behavior, in order to determine what proportion of genetic and environmental factors are unique to each phenotype and what portion is shared. In addition, a trivariate Cholesky allows the genetic and environmental variance in binge eating and compensatory behavior to be decomposed into components accounted for by impulsivity and components that are independent of impulsivity. In this way, one can determine if impulsivity differentially contributes to the genetic and environmental variance in binge eating and compensatory behavior.

In Figure 1, the extent to which the genetic variance in compensatory behavior is accounted for by genetic influences on binge eating is represented by the path a_{32} (i.e.,

representing genetic overlap with binge eating). In addition, the genetic variance in compensatory behavior is also represented by paths that overlap with impulsivity (i.e., a_{31}). Importantly, the genetic influences that do not share any variance with either binge eating or impulsivity (i.e., are entirely unique to compensatory behavior) are the residual genetic estimates, represented by path a_{33}. Similar to the genetic variance, the environmental variance in compensatory behavior is also decomposed into components that overlap with binge eating (i.e., $c_{32,}$ e_{32}) and impulsivity (i.e., $c_{31,}$ e_{31}), and residual components that are independent of impulsivity and binge eating (i.e., $c_{33,}$ e_{33}).

Figure 1 also illustrates unique and shared genetic and environmental influences on binge eating. Specifically, the genetic variance in binge eating is represented by paths that illustrate genetic overlap between both binge eating and impulsivity (i.e., a_{21}) and genetic influences on binge eating that are independent of impulsivity (i.e., the residual genetic estimates represented by path a_{22}). Similarly, paths that illustrate environmental overlap between impulsivity and binge eating are denoted by c_{21}, e_{21}, and those that are independent and unique to binge eating (i.e., residual environmental estimates) are denoted by c_{22}, e_{22}. Importantly, the paths depicting genetic and environmental overlap between impulsivity and binge eating and impulsivity and compensatory behavior will help determine whether impulsivity is a differential correlate of these phenotypes (i.e., whether it shares greater etiologic overlap with binge eating than compensatory behavior). Finally, unlike the path estimates for binge eating and compensatory behavior, genetic and environmental factors influencing impulsivity (i.e., $a_{11,}$ c_{11} and e_{11}) are not

decomposed, but instead provide estimates of genetic and environmental influences that are unique to that personality trait. In Figure 1, genetic and environmental contributions to the variance in impulsivity, binge eating and compensatory behavior are obtained by simply squaring the depicted path estimates.

The ordering of the variables in the model is important (i.e., from left to right), as the second variable in the model includes genetic and environmental paths that overlap with the first variable (i.e., $a_{21,}$ $c_{21,}$ e_{21}), and the third variable includes genetic and environmental paths that overlap with both the first (i.e., $a_{31,}$ $c_{31,}$ e_{31}) and second variable (i.e., $a_{32,}$ $c_{32,}$ e_{32}). Because I was interested in determining if genetic and environmental influences on impulsivity account for genetic and environmental influences on binge eating and compensatory behavior, priority in the model was accorded to impulsivity (i.e., impulsivity will be placed first in the model). However, given that I was also interested in examining overlap between binge eating *and* compensatory behavior, all models were run twice. First, models were run placing binge eating before compensatory behavior (i.e., according priority to binge eating). A second set of models were run placing compensatory behavior before binge eating (i.e., according priority to compensatory behavior). In this way, the degree of residual (i.e., unique) genetic and environmental influences present for both binge eating and compensatory behavior can be determined. In other words, I can examine the extent to which the variance in binge eating or compensatory behavior is accounted for by genetic and/or environmental influences on the other variable, and to what extent it is unique to each phenotype.

Parameter estimates obtained from trivariate Cholesky decompositions also allow for the calculation of several useful statistics. First, the total heritability and environmentality of impulsivity (i.e., $h^2_{1,}$ c^2_1 and e^2_1), binge eating (i.e., $h^2_{2,}$ c^2_2 and e^2_2) and compensatory behavior

(i.e., h^2_3, c^2_3 and e^2_3) can be obtained. Second, the total heritability of each phenotype can be divided into portions that are due to other variables, and portions that are specific to that phenotype. For example, binge eating (i.e., h^2_2) is divided into a portion accounted for by the genetic influences on impulsivity (i.e., h^2_a) and a residual component (i.e., a portion not attributed to the genetic effects of impulsivity; h^2_r). The shared and nonshared environmental variance in binge eating is also decomposed into an overall estimate (i.e., c^2_2 and e^2_2), an estimate that is decomposed into a portion accounted for by the shared and nonshared environmental influences on impulsivity (i.e., c^2_a and e^2_a) and a residual (i.e., a portion not attributed to the environmental influences of impulsivity; c^2_r and e^2_r). The total heritability of compensatory behavior (i.e., h^2_3; the third variable in the model) is divided into a portion accounted for by the genetic influences on impulsivity (i.e., h^2_a), a portion accounted for by the genetic influences on binge eating (i.e., h^2_b) and a residual component (i.e., a portion not attributed to the genetic effects of impulsivity or binge eating; h^2_r). Similarly, the shared and nonshared environmental variance in compensatory behavior is also decomposed into an overall estimate (i.e., c^2_3 and e^2_3), a portion accounted for by the shared and nonshared environmental influences on impulsivity (i.e., c^2_a and e^2_a), a portion accounted for by the shared and nonshared environmental influences on binge eating (i.e., c^2_b and e^2_b) and a residual (i.e., a portion not attributed to the environmental influences on impulsivity; c^2_r and e^2_r).

Third, the degree of overlap in genetic and environmental influences on impulsivity, binge eating and compensatory behavior can be estimated using genetic correlations (i.e., r_g), shared environmental correlations (i.e., r_c), and nonshared environmental correlations (i.e., r_e). These correlations indicate how much overlap in genetic and environmental factors exist between phenotypes. Three sets of correlations were calculated: 1) between impulsivity and binge eating 2) between impulsivity and compensatory behavior and 3) between binge eating and compensatory behavior. As an example, the genetic and environmental correlations between impulsivity and binge eating provide an estimate of the degree to which genetic and environmental influences on impulsivity overlap with the genetic and environmental influences on binge eating.

Comparisons of model fit.

The full ACE model was examined, as well as several nested models (i.e., AE, and CE) in order to determine the best-fitting model. In order to compare the fit of these nested models, a baseline model was fit first for each phenotype to estimate the variances, covariances and means by minimizing twice the log-likelihood (-2lnL). The obtained -2lnL of the baseline model was then used to calculate a likelihood ratio chi-square test for each nested model. The fit of these nested models was then compared to that of the full ACE model using the chi-square difference test (i.e., the -2lnL of the full ACE model was subtracted from the -2lnL of the nested models), resulting in chi-square goodness of fit test for each model. Non-significant changes in chi-square are indicative of an improved fit of the model to the data. In addition to the chi-square difference test, Akaike's information criterion (AIC; χ^2-2*df*; (Akaike, 1987)), a measure of model fit versus

model parsimony, was also used to compare models, where lower and/or more negative Akaike's Information Criterion values indicate a better-fitting model.

Longitudinal analyses.

In addition to the cross-sectional analyses described above, exploratory longitudinal analyses were conducted using only twins from the MTFS (*N*=1112). These analyses permitted examining whether impulsivity is a pre-existing factor that differentiates binge eating and compensatory behavior across time.

Phenotypic and etiologic associations were explored by conducting longitudinal analyses on the relationship between age 17 impulsivity and age 25 binge eating and compensatory behavior. Specifically, these analyses explored if genetic or environmental influences on impulsivity at age 17 were differentially associated with genetic or environmental influences on binge eating and compensatory behavior at age 25.

Prior to biometric twin modeling with longitudinal data, Pearson correlations were used to examine initial, phenotypic across-time associations between variables. Following phenotypic correlations, longitudinal twin models were then fit to the data. These analyses were identical to the trivariate cross-sectional analyses described above, except that the impulsivity scores were from age 17 rather than age 25. Similar to phenotypic associations, I expected the across-time relationship between impulsivity at age 17 and binge eating at age 25 to be stronger than that of impulsivity and compensatory behavior at age 25.

PAPER 1 RESULTS

The range in scores for binge eating (i.e., 0-7), compensatory behavior (i.e., 0-6) and impulsivity (i.e., 0-13) across MTFS and MSUTR samples indicates adequate variability in scores (see Table 1). Means and standard deviations for impulsivity are generally consistent across ages and samples (i.e., means ranging from 2.77-4.69; see Table 1), and are on par with levels found in other community-based samples (Patrick, et al., 2002). Means and standard deviations for binge eating and compensatory behavior are consistent with previous mean values for these MEBS subscales in participants aged 11-17 (Spanos, Klump, Burt, McGue, & Iacono, 2010), and the percentage of participants scoring above the MEBS clinical cutoffs (i.e., binge eating = 2.5 and compensatory behavior = 1.5) ranged from 4-16%, suggesting our findings are consistent with population-based estimates of the prevalence of these behaviors (Favaro, Ferrara, & Santonastaso, 2003; Stice, Marti, Shaw, & Jaconis, 2009).

Cross-Sectional Analyses

The within-person, phenotypic correlation between binge eating and compensatory behavior is statistically significant and indicates a moderate (i.e., r=.36**), positive relationship between the two phenotypes. Phenotypic correlations between impulsivity and both binge eating (i.e., r= .12**) and compensatory behavior (i.e., r=.12**) illustrate a small but significant degree of association. Importantly, the relationship between binge eating and impulsivity is almost identical in magnitude to that of compensatory behavior and impulsivity at the phenotypic level.

The MZ intraclass twin correlations (i.e., r=.42** for binge eating; r=.26** for compensatory behavior; r=.28** for impulsivity) for all three variables are more than twice as large as DZ intraclass twin correlations (i.e., r=.16** for binge eating; r=.10* for compensatory behavior; r=-.06 for impulsivity), indicating the presence of genetic influences on all three

variables (see Table 2). In addition, MZ twin correlations are less than 1.00, indicating the presence of nonshared environmental influences on each phenotype. Shared environmental influences appear to be negligible, given the significant differences between MZ and DZ intraclass twin correlations.

Cholesky decompositions.

Cross-twin, cross-trait correlations were also used to examine initial etiologic associations (see Table 2). The pattern of results remained consistent across variables. Specifically, the MZ cross-twin, cross-trait correlation between binge eating and compensatory behavior and between impulsivity and these phenotypes was roughly two times the DZ twin correlation, suggesting that genetic factors likely contribute to the covariance between the two behaviors. In addition, the MZ cross-twin, cross-trait correlation was similar to the within-twin correlation, suggesting that nonshared environmental influences are likely significant in the relationship between all three variables as well.

Trivariate model-fitting analyses were used to further examine genetic and environmental overlap between the three variables of interest. As a reminder, the model was run twice, once according priority in the model to binge eating (i.e., variable order was impulsivity, binge eating and compensatory behavior) and once according priority to compensatory behavior (i.e., variable order was impulsivity, compensatory behavior and binge eating; see Statistical Analyses Section). However, only the model according priority to binge eating is depicted (see Figure 3), for simplicity of presentation. The AE model was best-fitting for both models, as evidenced by the lowest AIC values of all models and the non-significant changes in chi-square from the full ACE model (see Table 3). This indicates that the relationship between variables was attributed to

additive genetic and nonshared environmental influences, with little shared environmental influences.

Path estimates depicting unique and overlapping paths for impulsivity, binge eating and compensatory behavior are illustrated in Figure 3. In terms of overlap between binge eating and compensatory behavior, the genetic and nonshared environmental correlations suggest a significant and moderate degree of etiologic association between the two phenotypes. In the model according priority to binge eating, genetic and nonshared environmental correlations were both statistically significant (i.e., r_a=.53 and r_e=.27), although the genetic correlation was larger, suggesting a larger degree of overlap in genetic versus nonshared environmental factors. However, both genetic and nonshared environmental attributable path estimates (i.e., h_a^2=6%; e_a^2=5%) indicate that only a small percentage of the total variance in compensatory behavior is attributable to binge eating (see Figure 3), while a much larger percentage is unique to compensatory behavior (i.e., residual estimates are h_r^2=18%; e_r^2=67%). These findings indicate that although genetic and nonshared environmental factors contribute to phenotypic associations between binge eating and compensatory behavior, most of the etiologic variance in compensatory behavior is independent of genetic and environmental factors operating in binge eating. Results were highly similar for the model according priority to compensatory behavior, (i.e., attributable estimates were much smaller than residual estimates), with the exception that a somewhat larger proportion of genetic influences on binge eating are shared with compensatory behavior (i.e., 12%) as compared to the genetic influences on compensatory behavior that are shared with binge eating (i.e., 6%).

In terms of impulsivity, the twin models showed somewhat different effects for binge eating versus compensatory behavior. Etiologic overlap between impulsivity and binge eating appears to be minimal, as genetic and nonshared environmental correlations were small (albeit significant) (i.e., r_a=.17 and r_e=.11). Genetic and nonshared environmental attributable estimates indicated that only a small percentage of the variance in binge eating is attributable to genetic and environmental factors in impulsivity (i.e., h_a^2=1%; e_a^2=1%). In contrast, residual estimates were quite large (i.e., h_r^2 = 46% and e_r^2 = 52%), suggesting that almost all of the variance in binge eating is accounted for by factors that are independent of impulsivity.

Etiologic overlap between impulsivity and compensatory behavior was also somewhat modest, but overlap was larger than that for impulsivity and binge eating. Specifically, the genetic correlation was statistically significant and larger (i.e., r_a=.37) than the nonshared environmental correlation (i.e., $r_{e=}$.04, ns), suggesting a greater degree of overlap in genetic versus nonshared environmental factors. Importantly, this genetic correlation is greater than the genetic correlation found between impulsivity and binge eating. However, genetic and nonshared environmental attributable path estimates (i.e., h_a^2=4%; e_a^2=0%) were again small, while residual estimates were more substantial (i.e., h_r^2=18%; e_r^2=67%). These findings indicate that, once again, most of the etiologic variance in compensatory behavior is independent of genetic and environmental factors operating in impulsivity.

Longitudinal Analyses

Phenotypic correlations between impulsivity at age 17 and binge eating and compensatory behavior at age 25 are similar to cross-sectional analyses, showing a small but

significant degree of association (i.e., r=.10** and r=.08*, respectively). Again, binge eating did not show a substantially stronger association with impulsivity compared to compensatory behavior.

Model-fitting results (see Table 3) corroborate cross-sectional findings indicating that the AE model provides the best fit to the data. Findings for etiologic overlap between binge eating and compensatory behavior are highly similar to what was observed in the cross-sectional models; this is not surprising, as both models measured these variables at age 25, and so slight variations in results are likely due to the absence of MSUTR twins in the longitudinal analyses. In examining the differential etiologic relationships between impulsivity (measured at 17) and binge eating and compensatory behavior (measured at age 25), findings largely replicated those from the cross-sectional data. The genetic correlation was larger for impulsivity and compensatory behavior (i.e., r_a=.40) than impulsivity and binge eating (i.e., r_a=.10), while the nonshared environmental correlations were low and non-significant. Attributable path estimates indicated that only a small percentage of the variance in both phenotypes is attributable to impulsivity (i.e., h_a^2=0-5%%; e_a^2=0-1%) (see Figure 4), while residual path estimates indicate most of the variance in binge eating (i.e., h_r^2=37%; e_r^2=62%) and compensatory behavior (i.e., h_r^2=23%; e_r^2=64%) at age 25 are independent of impulsivity at age 17.

PAPER 1 DISCUSSION

The aim of the present study was to examine associations between binge eating and compensatory behavior and explore whether the personality trait of impulsivity can differentiate between these behaviors at the phenotypic and etiologic level. Overall, phenotypic and etiologic findings suggest moderate overlap between binge eating and compensatory behavior, with large residual estimates that indicate substantial uniqueness among the two phenotypes. In addition, differences emerged in the degree of association between these phenotypes and impulsivity (i.e., greater overlap with compensatory behavior than binge eating), further supporting the distinctiveness of binge eating and compensatory behavior at the etiologic level. Importantly, findings were nearly identical for cross-sectional and longitudinal models, indicating that results are robust and reflect associations across time.

The moderate degree of phenotypic and etiologic overlap between binge eating and compensatory behavior (Cohen, 1988) replicates previous work. All studies to date have shown that phenotypic associations between these behaviors are due largely to genetic factors, although the degree of genetic overlap has varied somewhat across studies, with our estimates ($r_{a=}$ .53) coming in slightly lower than those previously ($r_{a=}$ .74 and 1.00) (Sullivan, et al., 1998; Wade, et al., 2008). Discrepancies could be due to slight differences in the constructs examined (i.e., purging and non-purging behaviors in our study versus purging only in previous work), although the 95% confidence intervals for the genetic and nonshared environmental correlations overlap considerably across all studies, suggesting minimal differences in overall results.

Despite the presence of shared genetic factors between binge eating and compensatory behavior, it is important to note that there was a substantial degree of unique genetic and

environmental influences on both phenotypes, such that the majority of genetic and environmental influences on each phenotype were independent of each other. The present study is the first to highlight this uniqueness by providing residual estimates that illustrate binge eating and compensatory behavior are not interchangeable at the etiologic level. Our results for impulsivity further suggest that impulsive personality traits may be one set of factors that contributes to this etiologic differentiation. Specifically, despite identical (albeit, modest) phenotypic associations between impulsivity, compensatory behavior, and binge eating, genetic correlations revealed that impulsivity overlaps with compensatory behavior to a greater extent (i.e., $r_{a=}$ .37) than binge eating (i.e., $r_{a=}$ .17). Similar differential associations have been found in previous research (Klump, et al., 2002) where compensatory behavior and Constraint showed significant phenotypic and genetic associations (e.g., r_a = -.23), in contrast to binge eating and Constraint which revealed no significant relationships. These data provide support for the idea that compensatory behavior is more strongly related to impulsivity, possibly because of the link between anxiety (i.e., the fear of weight gain associated with eating) and impulsive action. Nonetheless, it is important to note that overall overlap with impulsivity was relatively small for both compensatory behavior and binge eating, suggesting that other factors (e.g., other personality traits, such as perfectionism) (Forbush, Heatherton, & Keel, 2007) likely contribute to the variance in binge eating and compensatory behavior and their differentiation as well. Future research should directly explore this possibility by examining a range of personality traits and their relationships with compensatory behavior and binge eating.

A few limitations of my study should be noted. First, I may have been limited by the use of the MPQ Control subscale as a measure of impulsivity. The Control subscale from the MPQ captures cautiousness, planning and level-headedness (Tellegen & Waller, 2008). While previous

studies have used this scale to examine impulsivity within eating pathology (Lilenfeld, et al., 2000; Pryor & Wiederman, 1996), others have argued for more nuanced measures of impulsivity in eating disorders (e.g., negative urgency or the disposition to engage in rash action when experiencing negative affect), as research suggests impulsivity in eating disorders occurs in the context of negative affect (Anestis, et al., 2009; Cyders & Smith, 2008; Fischer, Smith, & Anderson, 2003; Fischer, et al., 2008; Waxman, 2009). Thus, the measure of impulsivity used in the present study may have attenuated the estimates of overlap between impulsivity and binge eating and compensatory behavior. Indeed, the phenotypic correlations in the present study (i.e., r=.12) and previous studies (i.e., r=-.15) (Klump, et al., 2002) that have used the MPQ subscales are lower than those for other measures of impulsivity (i.e., r=.29-.48) (Anestis, et al., 2009; Fischer, et al., 2003). Future studies should attempt to replicate the present study using other measures of impulsivity to ensure that results reflect the full range and type of impulsivity that might be important for eating disorders.

Finally, the present study was unable to distinguish between purging and non-purging behaviors, given that the compensatory behavior scale includes both types of behaviors, and sample sizes were too small to subtype the sample by the presence/absence of these behaviors. Given some evidence of greater etiologic overlap between binge eating and purging behaviors specifically (Sullivan, et al., 1998; Wade, et al., 2008), future research may benefit from attempting to differentiate between purging and non-purging types of compensatory behavior when examining overlap with binge eating and impulsivity.

The findings of my study have some implications for the diagnostic questions being considered in DSM-5. Results suggest that phenotypically, the two behaviors do not frequently co-occur in the general population and etiologically, there is considerable distinctiveness in the

etiologic factors contributing to each phenotype. These findings broadly support current diagnostic categories that classify eating disorders and their subtypes by the presence or absence of binge eating and purging behaviors. The cross-sectional and longitudinal findings clearly suggest that these behaviors are not simply differing expressions of the same underlying etiology, but are instead etiologically distinct behaviors that can either co-occur *or* present independently of each other.

Table 1

Descriptive statistics for Binge Eating, Compensatory Behavior, and Impulsivity (Cross-sectional and Longitudinal Analyses)

			Cross-sectional		
MSUTR (*N*=317-325)	Age		BE	CB	Impulsivity
Item #	--		7	6	13
M	18.32		1.17	.16	3.27
SD	1.90		1.43	.50	2.86
Min	16.12		0	0	0
Max	25.92		6	4	13
% ≥ clinical cutoff	--		16%	4%	--
α	--		.67	.50	.78
MTFS (*N*=891-1037)					
M	25.06		1.02	.23	2.97
SD	.71		1.43	.63	2.77
Min	23.80		0	0	0
Max	29.30		7	6	13
% ≥ clinical cutoff	--		11%	5%	--
α	--		.56	.72	.78
MSUTR and MTFS (*N*=1251-1356)					
M	23.42		1.06	.21	3.27
SD	3.10		1.43	.60	2.86
Min	16.12		0	0	0
Max	29.30		7	6	13
% ≥ clinical cutoff	--		13%	5%	--
α	--		.71	.53	.78
			Longitudinal		
MTFS (*N*=1527-1540)	Age 17	Age 25	BE 25	CB 25	Impulsivity 17
M	17.88	25.05	1.02	.23	4.69
SD	.69	.71	1.42	.63	3.23
Min	16.55	23.80	0	0	0
Max	20.30	29.30	7	6	13
% ≥ clinical cutoff	--	--	11%	5%	--
α	--	--	.72	.56	.80

Note. BE= Binge Eating from the Minnesota Eating Behavior Survey; CB= Compensatory Behavior from the Minnesota Eating Behavior Survey; MSUTR = Michigan State University Twin Registry; MTFS = Minnesota Twin Family Study; *N* = number of twins; Min = minimum; Max = maximum. Clinical cutoff scores for the MEBS binge eating and compensatory behavior subscales were determined using mean MEBS scores for young women with anorexia nervosa and bulimia nervosa and are as follows: BE = 2.5, CB = 1.5.

Table 2

Intraclass and Cross-Twin, Cross-Trait Correlations between Binge Eating, Compensatory Behavior and Impulsivity

	Cross-sectional					
	Binge Eating		Compensatory Behavior		Impulsivity	
	MZ	DZ	MZ	DZ	MZ	DZ
Binge Eating	**.42**†**	**.16****	--	--	--	--
Compensatory Behavior	.15**	.09	**.26**†**	**.10***	--	--
Impulsivity	.07	.01	.12**†	-.04	**.28**†**	**-.06**
	Longitudinal					
	Binge Eating 25		Compensatory Behavior 25		Impulsivity 17	
	MZ	DZ	MZ	DZ	MZ	DZ
Binge Eating 25	**.36**†**	**.17****	--	--	--	--
Compensatory Behavior 25	.13**	.11	**.31**†**	**.13***	--	--
Impulsivity 17	.05	-.01	.13**	.08	**.35**†**	**.06**

Note. Binge Eating= subscale from the Minnesota Eating Behavior Survey; Compensatory Behavior= subscale from the Minnesota Eating Behavior Survey; Impulsivity= control scale reverse scored from the Multidimensional Personality Questionnaire; Intraclass twin correlations are denoted in bold-face.

** $p < .01$; * $p < .05$ (2-tailed) indicating correlations are significant.

† The MZ correlation is significantly different from the DZ correlation, $p < .01$ (1-tailed).

Table 3

Model Fit Indices for Trivariate Cholesky Decompositions

Model	Fit Statistics			
	-2ln*L*(df)	-2lnL_{Δ}(df)	*p*	AIC
	Cross-Sectional			
Trivariate IMP-BE-CB				
Saturated	22841.80 (7641)	--	--	--
ACE	22958.19 (7674)	116.39 (33)	<.001	50.39
AE	**22958.19 (7680)**	**116.39 (39)**	**1.00**	**38.39**
CE	23035.46 (7680)	193.66 (39)	<.001	115.66
	Longitudinal			
Trivariate IMP17-BE25-CB25				
Saturated	12930.87 (5355)	--	--	--
ACE	12956.22 (5388)	25.35 (33)	<.001	-40.65
AE	**12956.22 (5394)**	**25.35 (39)**	**1.00**	**-52.65**
CE	12994.52 (5394)	63.65 (78)	<.001	-14.35

Note. A= genetic effects; C= shared environmental effects; E= nonshared environmental effects; -2ln*L* = -2 times the log likelihood; -2lnL_{Δ}= differences in -2ln*L* values between the saturated model and subsequent models; AIC= Akaike's Information Criteria; BE = Binge Eating subscale from the Minnesota Eating Behavior Survey; CB = Compensatory Behavior subscale from the Minnesota Eating Behavior Survey; IMP = Impulsivity (control scale reverse scored) from the Multidimensional Personality Questionnaire. Best-fitting model for each trivariate model is noted in bold text. The fit statistics for the cross-sectional and longitudinal models where compensatory behavior was accorded priority are not depicted here because the fit is identical to the models where binge eating was accorded priority.

Figure 1. Path diagram of trivariate Cholesky ACE model for impulsivity, binge eating and compensatory behavior.The variance for each variable is accounted for by additive genetic effects (i.e., A_1, A_2, A_3), shared environmental effects (i.e., C_1, C_2, C_3) and nonshared environmental effects (and measurement error) (i.e., E_1, E_2, E_3). The genetic, shared and nonshared environmental variance in BE is decomposed into components that are accounted for by genetic and environmental effects on impulsivity (i.e., a_{21}, c_{21}, e_{21}) and residual components that are independent of impulsivity (i.e., a_{22}, c_{22}, e_{22}). The genetic, shared and nonshared environmental variance in CB is decomposed into components that are accounted for by genetic and environmental effects on impulsivity (i.e., a_{31}, c_{31}, e_{31}), components that are accounted for by genetic and environmental effects on BE (i.e., a_{32}, c_{32}, e_{32}) and residual components that are independent of impulsivity (i.e., a_{33}, c_{33}, e_{33}). The genetic correlations (r_a), shared environmental correlations (r_c), and nonshared environmental correlations (r_e) are depicted with curved arrows.

Figure 1 (cont'd).

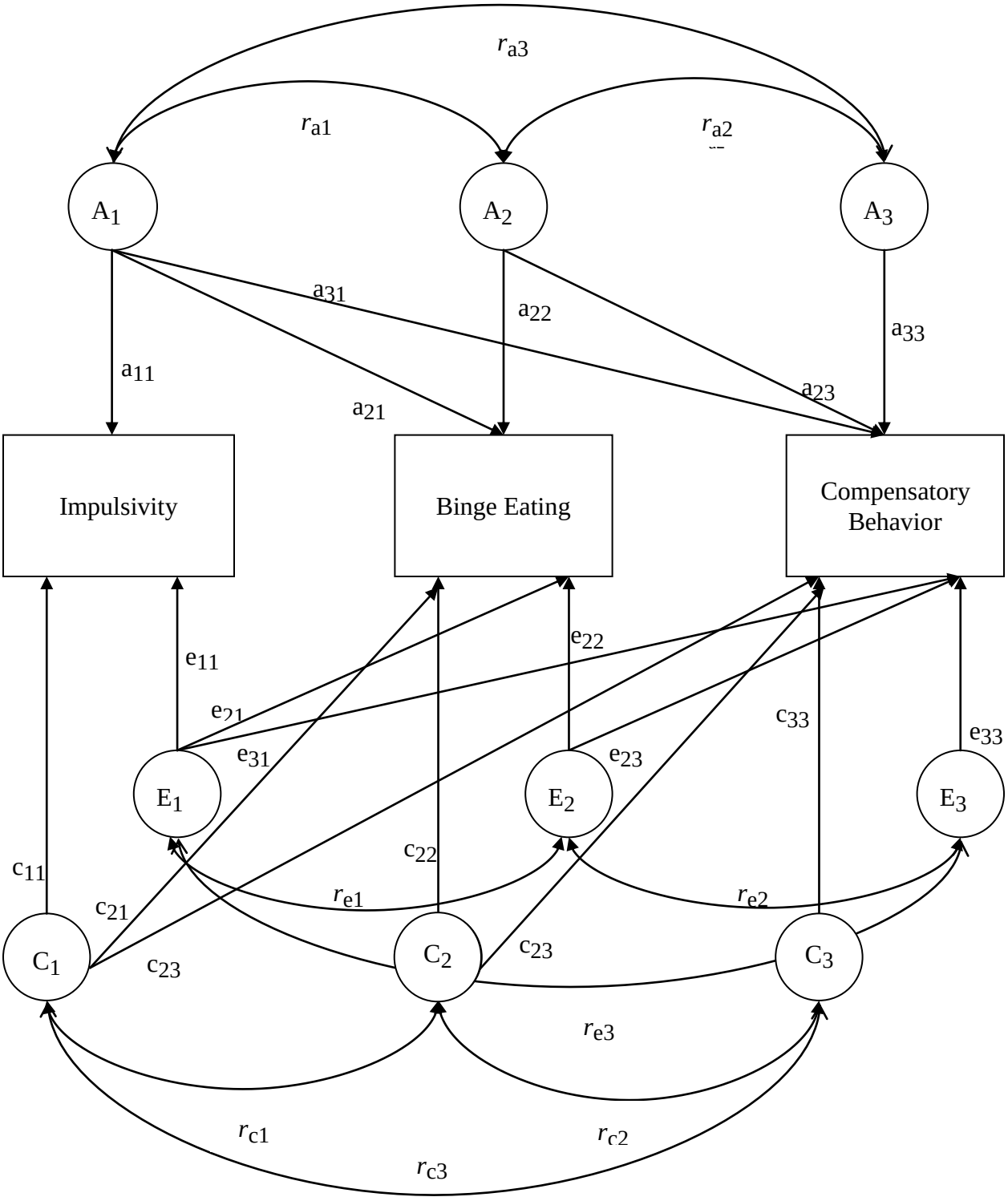


Figure 2. Path diagram of longitudinal, trivariate Cholesky ACE model for impulsivity at age 17 and binge eating (BE) and compensatory behavior (CB) at age 25. The variance for each variable is accounted for by additive genetic effects (i.e., A_1, A_2, A_3), shared environmental effects (i.e., C_1, C_2, C_3) and nonshared environmental effects (and measurement error) (i.e., E_1, E_2, E_3). The genetic, shared and nonshared environmental variance in BE at age 25 is decomposed into components that are accounted for by genetic and environmental effects on impulsivity at age 17 (i.e., a_{21}, c_{21}, e_{21}) and residual components that are independent of impulsivity at age 17 (i.e., a_{22}, c_{22}, e_{22}). The genetic, shared and nonshared environmental variance in CB at age 25 is decomposed into components that are accounted for by genetic and environmental effects on impulsivity at age 17 (i.e., a_{31}, c_{31}, e_{31}), components that are accounted for by genetic and environmental effects on BE at age 25 (i.e., a_{32}, c_{32}, e_{32}) and residual components that are independent of impulsivity and binge eating (i.e., a_{33}, c_{33}, e_{33}). The genetic correlation (r_a), shared environmental correlation (r_c), and nonshared environmental correlation (r_e) are depicted with curved arrows.

Figure 2 (cont'd).

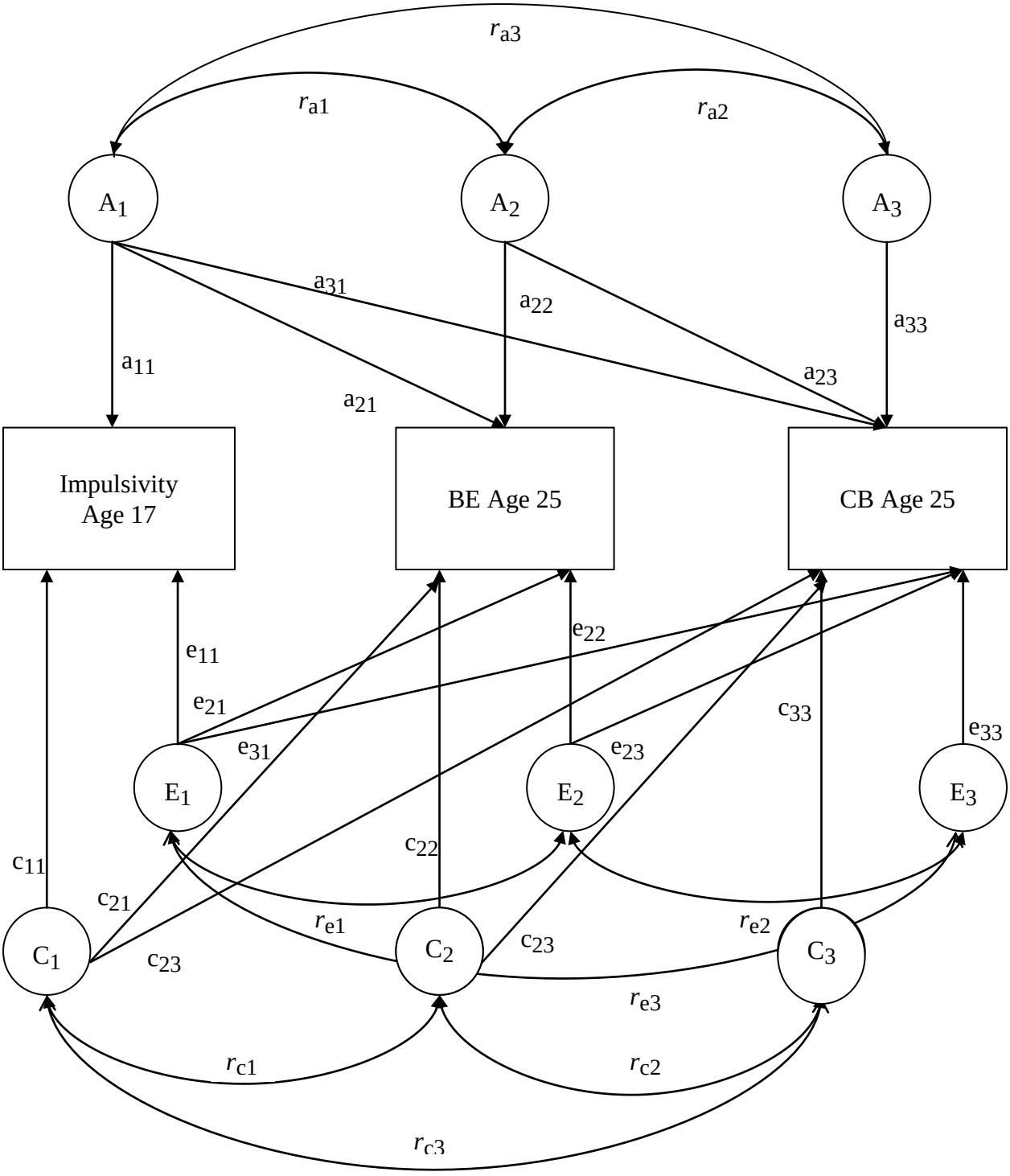
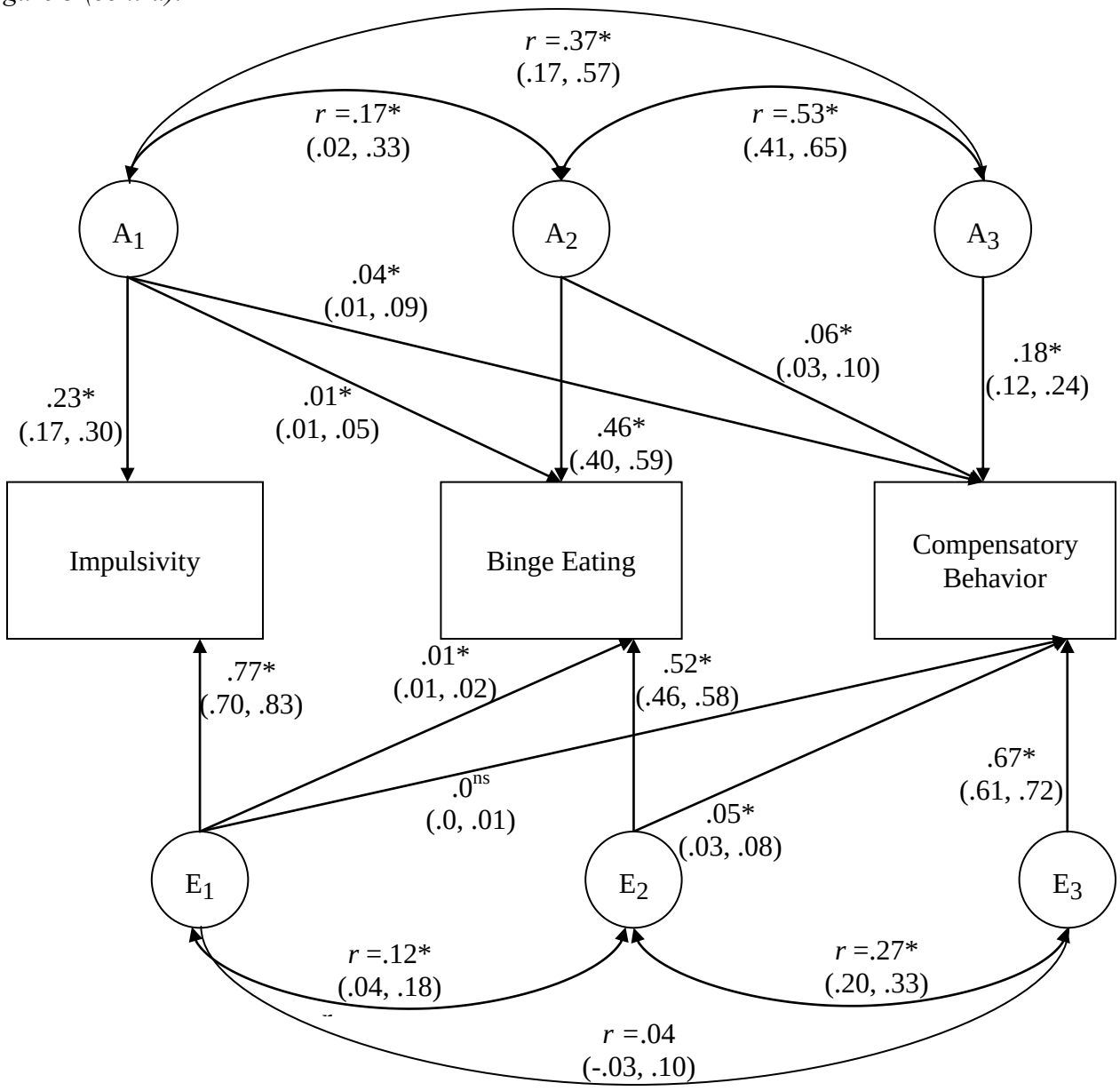


Figure 3. Path diagram of the best-fitting, trivariate Cholesky AE model of impulsivity, binge eating and compensatory behavior. The path estimates depicted illustrate the genetic and nonshared environmental contributions to the variance within and the covariance between the variables. The percentage of variance accounted for by each path is obtained by multiplying the path estimate by 100. The genetic correlations (r_a) and nonshared environmental correlations (r_e) are depicted with curved arrows.
*$p < .05$

Figure 3 (cont'd).



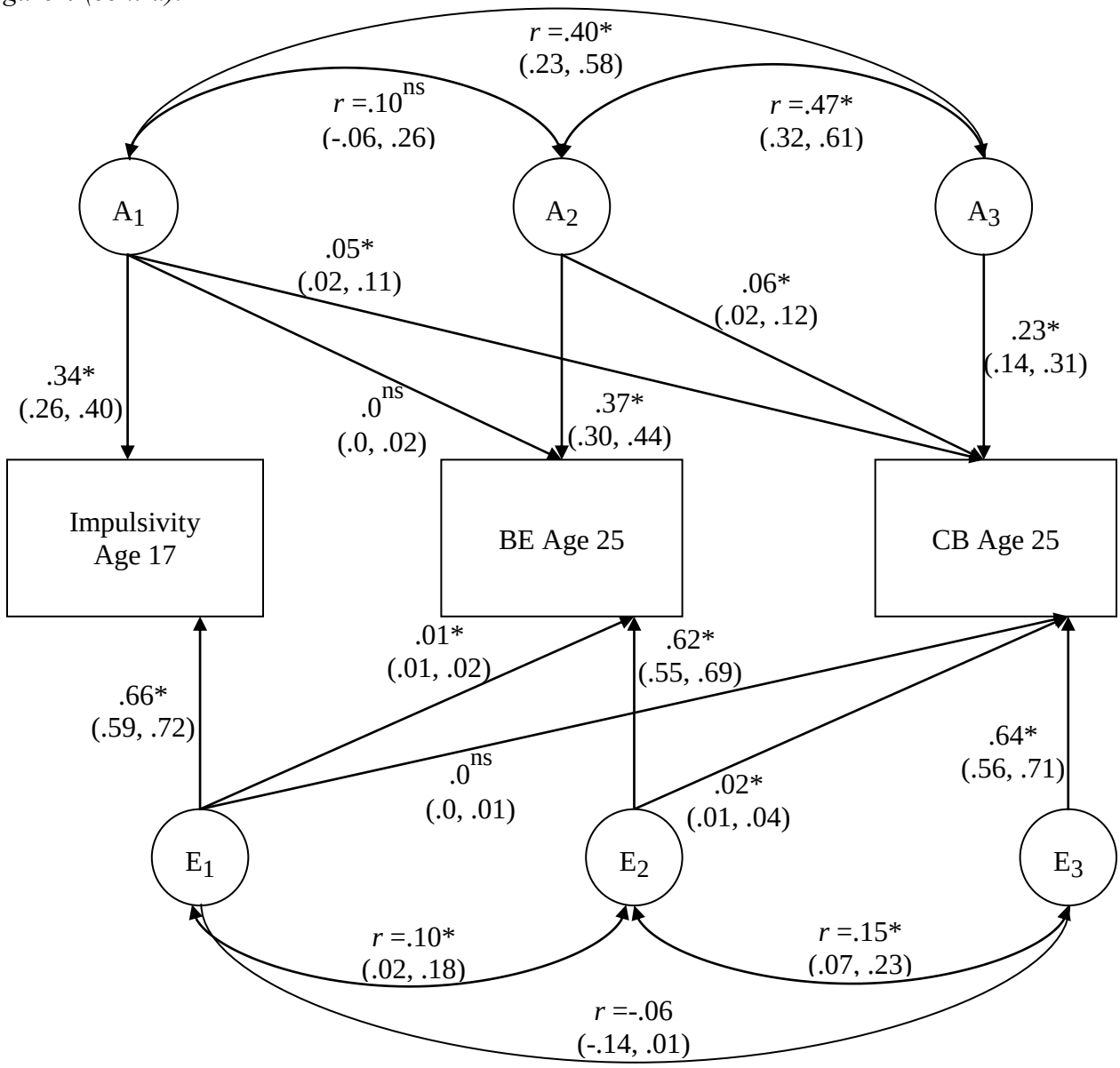
Total Variance Estimates Impulsivity:
A=23% (.17, .30)
E=77% (.70, .83)

Binge Eating:
A=47% (.42, .53)
E=53% (.47, .58)

Compensatory Behavior:
A=28% (.21, .35)
E= 72% (.65, .79)

Figure 4. Path diagram of the best-fitting, longitudinal Cholesky AE model of impulsivity, binge eating (BE) and compensatory behavior (CB). Standardized path estimates of the genetic and nonshared environmental contributions to the variance within and the covariance between the variables are illustrated. The percentage of variance accounted for by each path is obtained by multiplying the path estimate by 100. The genetic correlations (r_a) and nonshared environmental correlations (r_e) are depicted with curved arrows.
*$p < .05$

Figure 4 (cont'd).



Total Variance Estimates Impulsivity Age 17:
A=34% (.26, .40)
E=66% (.59, .72)

Binge Eating Age 25:
A=37% (.30, .44)
E=61% (.57, .68)

Compensatory Behavior Age 25:
A=34% (.18, .54)
E= 65% (.54, .75)

A MULTIVARIATE TWIN MODEL OF PERFECTIONISM AND ITS RELATIONSHIP TO BINGE EATING AND COMPENSATORY BEHAVIOR

By

Alexia Spanos, M.A.

ABSTRACT

A MULTIVARIATE TWIN MODEL OF PERFECIONISM AND ITS RELATIONSHIP TO BINGE EATING AND COMPENSATORY BEHAVIOR

By

Alexia Spanos, M.A.

Previous studies have explored the overlap in genetic and environmental variance between binge eating and compensatory behavior. This overlap is important to consider given the two phenotypes are intimately linked in diagnostic schemas, and this may provide additional information to help researchers and clinicians determine if disorders that are characterized by the presence of only one of these behaviors are valid and distinct disorders. Studies thus far find evidence for a significant amount of unique etiologic variance between the two phenotypes. Given the presence of this unique variance in each phenotype, it is important to understand factors that may contribute to this uniqueness. One avenue to explore is personality, and perfectionism specifically, given it has been closely linked to eating pathology. Thus, the aim of the present study was to determine whether perfectionism contributes to the unique variance between these closely-linked disordered eating behaviors, and to examine if it is more associated with binge eating versus compensatory behavior. Study hypotheses predicted perfectionism would account for more of the unique variance in binge eating compared to compensatory behavior, at the phenotypic and etiologic level. Using a population-based sample of same-sex female twins (N=425), binge eating and compensatory behavior were assessed with the Minnesota Eating Behavior Survey and perfectionism was assessed with three scales from the Multidimensional Perfectionism Questionnaire (i.e., Total Perfectionism, Concern over Mistakes and Doubts about Actions). Phenotypic correlations revealed a moderate and significant degree of association between all three perfectionism variables and binge eating and compensatory

behavior, with little differences between the disordered eating phenotypes. Twin models also indicated near identical associations between two perfectionism variables (i.e., Total Perfectionism and Concern over Mistakes) and both binge eating and compensatory behavior. In these models, genetic influences accounted for the overlap between these variables, although residual estimates indicated that most of the etiologic variance in binge eating and compensatory behavior is independent of genetic and environmental factors operating in Total Perfectionism and Concern over Mistakes. In contrast, Doubts about Actions exhibited stronger etiologic associations with binge eating than compensatory behavior, where genetic effects accounted for most overlap between these variables, and residual estimates indicated more overlap than uniqueness between binge eating and Doubts about Actions. Findings suggest the Doubts about Actions scale of perfectionism does differentially account for some of the unique variance between binge eating and compensatory behavior, thus providing evidence for considerable distinctiveness in the genetic factors contributing to binge eating and compensatory behavior. These findings have implications for diagnostic schemas, lending support to distinct categories that are primarily characterized by either binge eating or compensatory behavior.

PAPER 2 INTRODUCTION

Previous studies have explored phenotypic and etiologic overlap between binge eating and compensatory behavior. It is important to consider the degree of overlap between these two disordered eating phenotypes given they are intimately linked in diagnostic schemas. More importantly, this may provide additional information to help researchers and clinicians determine if disorders that are characterized by the presence of only one of these behaviors are valid and distinct disorders. One way to explore this question is to examine phenotypic and etiologic associations between binge eating and compensatory behavior. Indeed, the few studies which have explored this research question find evidence for a moderate-to-substantial degree of overlap between these phenotypes (Spanos, Klump, Burt, McGue, & Iacono, in preparation; Sullivan, et al., 1998; Wade, et al., 2008). Specifically, phenotypic estimates were r=.36 (Spanos, et al., in preparation) with genetic (i.e.,. r=.53-1.00) and environmental correlations (i.e., r=.27-.48) ranging from small to substantial.

However, the most recent investigation improved upon previous studies by also providing estimates of the percentage of variance in each phenotype that is not accounted for by the other variable. These estimates indicated a fair proportion of the genetic (i.e., 46% in binge eating and 18% in compensatory behavior) and environmental (i.e., 52% in binge eating and 67% in compensatory behavior) variance in binge eating and compensatory behavior is independent of the other (Spanos, et al., in preparation). Given the presence of this unique variance in each phenotype, it is important to understand factors that may contribute to this uniqueness as this information may contribute to knowledge regarding their etiology and possible future diagnostic schemas.

Personality factors have been previously explored in this regard based on substantial evidence that certain personality traits may differentially contribute to the development and maintenance of different eating disorders (Vitousek & Manke, 1994). Impulsivity has been the primary personality characteristic explored, likely because there is good theoretical and empirical evidence to suggest that impulsivity might be differentially related to both binge eating and compensatory behavior (Fahy & Eisler, 1993; Fischer, et al., 2003). Indeed, in behavior genetics analyses, impulsivity was found to account for some of the differentiation, as it was more closely linked etiologically to compensatory behavior than binge eating (i.e., genetic correlations of r=.37 and r=.17, respectively) (Spanos, et al., in preparation). However, impulsivity only accounted for a small proportion of the total variance in both binge eating (i.e., 1%) and compensatory behavior (i.e., 4%), suggesting that a large proportion of unique variance in each phenotype remains unexplained.

Perfectionism may be another personality trait to explore, as existing evidence suggests it may also be involved in the initiation and maintenance of disordered eating behavior (Bardone-Cone et al., 2007; Stice, 2002). Importantly, some theories link perfectionism to specific disordered eating behaviors like compensatory behavior, because of the belief that being perfectionistic may make one more likely to fear and avoid perceived personal failures like weight gain (Taranis & Meyer, 2010). Thus, in the presence of overeating or indeed, any eating, perfectionistic individuals may be more likely to attempt to alleviate anxiety regarding potential weight gain by compensating for food eaten. Indeed, there is some evidence to support these claims. For example, Forbush et al. (2007) compared the strength of the relationship between perfectionism and binge eating to the relationship between perfectionism and purging, finding evidence for a stronger association between purging and perfectionism. Similarly, individuals

with an eating disorder (i.e., anorexia nervosa or bulimia nervosa) who had a history of laxative abuse (i.e., ever used laxatives to control weight or compensate for food eaten) scored higher on a measure of perfectionism (i.e., the Eating Disorder Inventory-Perfectionism subscale) than individuals with an eating disorder who had no history of laxative abuse (Pryor, Wiederman, & McGilley, 1996)).

In contrast, other studies provide evidence for stronger associations between perfectionism and binge eating. One hypothesis is that perfectionistic individuals with significant body dissatisfaction who feel unable to change their appearance (i.e., low self-efficacy) may experience negative reactions to their perceived failure and binge eat as a way to modulate their mood (Bardone-Cone, Abramson, Vohs, Heatherton, & Joiner, 2006; Bardone-Cone, Vohs, Abramson, Heatherton, & Joiner, 2000). In support of this theory, Bardone-Cone et al. (2008) found perfectionism only predicted binge eating, not compensatory behavior, in a non-clinical sample of women (Bardone-Cone, et al., 2006). Similarly, Fink et al. (2009) compared levels of perfectionism among individuals with diagnosed eating disorders (i.e., anorexia nervosa, bulimia nervosa, binge eating disorder and purging disorder) and controls. Perfectionism scores were significantly higher for diagnostic groups than for controls, but only for diagnoses where binge eating is the primary feature (i.e., bulimia nervosa and binge eating disorder) (Fink, et al., 2009).

Despite emerging theories of the link between perfectionism and both binge eating and compensatory behavior, studies are few in number and findings are mixed. Discrepancies could be due to differences in the populations or constructs examined, and/or the measures used. Specifically, while Bardone-Cone et al. (2006) and Fink et al. (2009) used nonclinical samples of undergraduate students, participants in Pryor et al.'s (1996) study were treatment-seeking clinical samples. Thus, differences in the severity of pathology could account for differences in the

results that emerged, such that perfectionism may be more strongly linked to compensatory behavior in clinical samples versus binge eating in non-clinical samples. This is possibly due to higher range restriction for compensatory behavior compared to binge eating. In other words, given compensatory behaviors are lower base rate behaviors, the prevalence of compensatory behavior would be lower in non-clinical samples, thus making it more difficult to detect significant associations. In contrast, because binge eating is a higher base rate behavior, the range would be much higher than for compensatory behavior in non-clinical samples, thus making significant associations easier to detect. In addition, Pryor et al.'s (1996) investigation focused on the relationship between perfectionism and one specific compensatory behavior (i.e., laxative abuse), rather than a variety of compensatory behaviors examined in other studies (Bardone-Cone, et al., 2006; Fink, et al., 2009). Slight differences in the constructs used may account for discrepancies across studies because relationships may differ based on the specific disordered eating behaviors examined (e.g., laxative use may be more strongly associated with perfectionism than diet pill use). Given these discrepancies in methodology and results, additional research exploring the relative associations between perfectionism with binge eating and compensatory behavior may be helpful.

In addition, studies thus far are limited in their focus on only phenotypic associations without any consideration for genetic or environmental factors that may affect these relationships (i.e., none of the studies thus far have utilized twin populations to be able to explore these associations at the etiologic level). Thus, the aim of the present study was to determine if perfectionism accounts for some of the unique variance in binge eating and compensatory behavior. In addition, I explored whether perfectionism is more strongly associated with one phenotype versus the other at the phenotypic and etiologic level using twins from the Michigan

State University Twin Registry (MSUTR). The present study hypothesized that binge eating would be more strongly associated with perfectionism than compensatory behavior. This hypothesis was based on similarities (e.g., the age of participants, non-clinical sample) between community-based samples used in the current study and those in previous (albeit non-twin) studies that found stronger relationships between perfectionism and binge eating, compared to compensatory behavior (Bardone-Cone, et al., 2006; Fink, et al., 2009).

PAPER 2 METHOD

Participants

Participants included an archival sample of female twins drawn from the Michigan State University Twin Registry (MSUTR; *N*=425). Participants from the MSUTR are reared together, same-sex female twins between the ages of 16 and 30 (*M*= 20.46, *SD*=2.41). Although the pilot sample of MSUTR twins was recruited through several different mediums (university registrar's offices, advertisements, flyers, birth records), recruitment for the MSUTR is now entirely done through birth records. Approximately 70% of twins included in the current sample are from the pilot sample of twins, while the remaining 30% were recruited through birth records in collaboration with the Michigan Department of Community Health (MDCH). Importantly, the MSUTR sample included in this study has been shown to be highly representative of the ethnic composition of Michigan (i.e., 82.8% Caucasian, 11.4% African-American, 1.7% Hispanic, 1.4% Asian/Asian-American, and 2.8% "Other") (Culbert, et al., 2009). For more complete details on sampling strategy, recruitment and the demographics of this sample, see Klump and Burt (2006).

Zygosity Determination

The MSUTR used a standard physical similarity questionnaire to determine twin zygosity. Responses to the questionnaire are scored for the probability that the twins are monozygotic (MZ) versus dizygotic (DZ) based on the degree of physical similarity between them. This questionnaire was completed by trained research staff in both samples and has an accuracy rate of over 95% (Lykken, et al., 1990). For additional accuracy, the MSUTR had twins complete a self-report version of the physical similarity questionnaire, and responses between twins and staff were compared for accuracy. In addition, one of the MSUTR directors' (KLK or

SAB) evaluated questionnaire information and photographs of the twins (if available) for added accuracy.

Measures

Disordered eating symptoms.

Binge eating and compensatory behavior. Binge eating and inappropriate compensatory behavior were measured using the Minnesota Eating Behavior Survey (MEBS)[2]. The MEBS is a 30-item true/false self-report questionnaire assessing current disordered eating attitudes and behaviors in individuals as young as 10. Exploratory factor analysis of MEBS resulted in four factors (Klump, McGue, et al., 2000; von Ranson, et al., 2005) in addition to the total score: body dissatisfaction (i.e., dissatisfaction with size and/or shape of body), weight preoccupation (i.e., preoccupation with dieting or weight), binge eating (i.e., thinking about or engaging in binge eating), and compensatory behavior (i.e., thinking about or engaging in inappropriate compensatory behavior for weight control).

The present study focused on the MEBS binge eating (7 items) and compensatory behavior (6 items) subscales. The binge eating and compensatory behavior subscales from the MEBS exhibit adequate psychometric properties for assessing disordered eating in community-based samples (von Ranson, et al., 2005). Discriminant validity for the compensatory behavior scale is excellent, as females with an eating disorder scored significantly higher on this subscale compared to controls (Klump, McGue, et al., 2000; von Ranson, et al., 2005). In addition, the binge eating subscale also discriminates between specific eating disorder diagnoses, as it

[2] The Minnesota Eating Behavior Survey (MEBS; previously known as the Minnesota Eating Disorder Inventory (M-EDI)) was adapted and reproduced by special permission of Psychological Assessment Resources, Inc., 16204 North Florida Avenue, Lutz, Florida 33549, from the Eating Disorder Inventory (collectively, EDI and EDI-2) by Garner, Olmstead, Polivy, Copyright 1983 by Psychological Assessment Resources, Inc. Further reproduction of the MEBS is prohibited without prior permission from Psychological Assessment Resources, Inc.

differentiates individuals with bulimia nervosa from controls as well as individuals with other eating disorder diagnoses that do not include binge eating (von Ranson, et al., 2005). Three-year test-retest reliability statistics for binge eating and compensatory behavior are adequate, ranging from .30 to .50 (all significant at $p < .01$). These reliability estimates are impressive given the length of time between assessments (i.e., three years). Internal consistency for the binge eating and compensatory behavior scales have been adequate in previous studies (i.e., $\alpha = .65$-$.69$) (Klump, McGue, et al., 2000; von Ranson, et al., 2005). In the present study, internal consistency estimates were good for the binge eating scale (i.e., $\alpha = .73$) and adequate for the compensatory behavior scale (i.e., $\alpha = .61$).

Perfectionism.

Multidimensional perfectionism scale. The Multidimensional Perfectionism Scale (Frost, Marten, Lahart, & Rosenblate, 1990) was used to assess perfectionism. The MPS is a 35-item scale designed to measure overall perfectionism (i.e., Total Perfectionism) and six facets of perfectionism: Concern over Mistakes, Personal Standards, Parental Expectations, Parental Criticism, Doubts about Actions, and Organization. The present study focused on the Total Perfectionism scale (i.e., an aggregate of all six subscales, excluding the Organization subscale, as this subscale shows the weakest intercorrelations with the other MPS subscales you need a cite here) and the Concern over Mistakes (i.e., intolerance for one's own mistakes) and Doubts about Actions (i.e., tendency to feel dissatisfied with one's own work) subscales (Enns & Cox, 1999). The focus on these scales is appropriate given all three show stronger associations with disordered eating behaviors (Bastiani, Rao, Weltzin, & Kaye, 1995; Minarik & Ahrens, 1996; Srinivasagam et al., 1995) than the other MPS scales and show greater associations with eating pathology than other psychiatric disorders (i.e., depression, and alcoholism) (Bulik et al., 2003).

Internal consistency for TP, CM and DA in past studies is excellent, with alpha coefficients of .90, .77 and .88, respectively (Frost, et al., 1990). The present study confirmed these excellent internal constancy estimates for all three scales with alphas between .81-.92. Test-retest reliability for the TP scale is high with correlations of .69, indicating good stability over time (Hewitt, Flett, Turnbull-Donovan, & Mikail, 1991). Convergent validity for the TP and CM scales is also good. Specifically, the TP scale is highly correlated with other measures of perfectionism (i.e., the Burns Perfectionism Scale, r=.85), including a measure used specifically for examining perfectionistic traits in eating disorders (i.e., Eating Disorders Inventory-Perfectionism, r=.59) (Enns & Cox, 1999). Similarly, the CM subscale exhibits significant intercorrelations with the Eating Disorders Inventory-Perfectionism (r=.46, $p < .01$; (Minarik & Ahrens, 1996)). Finally, discriminant validity for TP in eating pathology is also good as women recovered from an eating disorder scored higher than never-ill women on overall perfectionism (Srinivasagam, et al., 1995).

Statistical Analyses

Phenotypic relationships.

Pearson correlations were computed first to examine within-person, phenotypic associations between perfectionism, binge eating and compensatory behavior. Intraclass twin correlations were then computed for MZ and DZ twins to identify the most important genetic and environmental (i.e., additive genetic (A), shared environmental (C) and nonshared environmental influences (E)) estimates for multivariate models. If MZ twin correlations are approximately twice that of DZ twin correlations, this suggests the presence of additive genetic influences (i.e., the sum of two or more genes across multiple loci). If MZ twin correlations are *more* than twice the DZ twin correlations, this suggests the presence of dominant genetic influences (i.e., the

expression of one allele over another on the same locus). If MZ twin correlations are approximately equal to those of DZ twin correlations, the presence of shared environmental influences is supported. Finally, if MZ twin correlations are less than 1.00, this suggests the presence of nonshared environmental influences (and measurement error).

Cross-twin, cross-trait correlations were then computed between binge eating and compensatory behavior (e.g., Twin 1's binge eating with Twin 2's compensatory behavior) and between disordered eating variables (i.e., binge eating and compensatory behavior) and perfectionism (e.g., Twin 1's binge eating with Twin 2's perfectionism) in order to provide preliminary indications of genetic and environmental associations that underlie phenotypic relationships. If MZ cross-twin, cross-trait correlations are significantly higher than those of DZ twins, then shared genetic effects are important in phenotypic associations. If, on the other hand, MZ cross-twin, cross-trait correlations are similar to those of DZ twins, shared environmental factors primarily contribute to these phenotypic associations. Finally, if within-twin correlations (i.e., the phenotypic associations for each participant in the sample) are greater than the cross-twin, cross-trait correlations, nonshared environmental influences are likely significant in the phenotypic relationships.

Etiologic relationships.

Etiologic relationships were explored with trivariate, biometric models (i.e., Cholesky decomposition) which provide estimates of the relative additive genetic (A), shared environmental (C) and nonshared environmental (E) influences on the variance in, and the covariance among, perfectionism, binge eating and compensatory behavior. Given that three measures of perfectionism were used, three separate Cholesky models were run (i.e., TP, binge eating and compensatory behavior; CM, binge eating and compensatory behavior; DA, binge

eating and compensatory behavior). Cholesky decomposition is a multivariate data-analysis technique that is based on the principles of factor analysis. The ACE trivariate, Cholesky decomposition is depicted in Figure 1. This model allows the genetic and environmental variance in binge eating and compensatory behavior to be decomposed into components that overlap with one another, components that are accounted for by perfectionism, and components that are independent of perfectionism. In this way, one can determine if perfectionism differentially contributes to the genetic and environmental variance in binge eating and compensatory behavior.

Specifically, the genetic variance in compensatory behavior is decomposed into genetic influences that overlap with perfectionism (i.e., a_{31}) and binge eating (i.e., $a_{32;}$ attributable variance), and genetic influences (i.e., residual variance) that are unique to compensatory behavior (i.e., not overlapping with the other two variables in the model). Similar to the genetic variance, the environmental variance in compensatory behavior is also decomposed into components that overlap with binge eating (i.e., $c_{32,}$ e_{32}) and perfectionism (i.e., $c_{31,}$ e_{31}), and residual components that are independent of perfectionism and binge eating (i.e., $c_{33,}$ e_{33}). Finally, the genetic and environmental variance in binge eating can be decomposed into that which overlaps with perfectionism (i.e., $a_{21,}$ c_{21}, e_{21}) and those that are independent of perfectionism (i.e., the residual genetic estimates represented by path $a_{22,}$ c_{22}, e_{22}). Unlike the path estimates for binge eating and compensatory behavior, genetic and environmental factors influencing perfectionism (i.e., $a_{11,}$ c_{11} and e_{11}) are not decomposed, but instead provide estimates of genetic and environmental influences that are unique to that personality trait.

Parameter estimates obtained from trivariate Cholesky decompositions also allow for the calculation of several useful statistics. First, the total heritability (i.e., h^2) and environmentality (i.e., e^2) of all three perfectionism variables, binge eating and compensatory behavior can be obtained. Second, the total heritability and environmentality of each phenotype can be divided into portions that are due to other variables, and portions that are specific to that phenotype (e.g., the total heritability of binge eating is divided into a portion accounted for by the genetic influences on perfectionism, and a residual component). Third, the degree of overlap in genetic and environmental influences on perfectionism, binge eating and compensatory behavior can be estimated using genetic correlations (i.e., r_g), shared environmental correlations (i.e., r_c), and nonshared environmental correlations (i.e., r_e). These correlations indicate how much overlap in genetic and environmental factors exist between phenotypes. Three sets of correlations were calculated for each of the three perfectionism models: 1) between perfectionism and binge eating 2) between perfectionism and compensatory behavior and 3) between binge eating and compensatory behavior. As an example, the genetic and environmental correlations between TP and binge eating provide an estimate of the degree to which genetic and environmental influences on TP overlap with the genetic and environmental influences on binge eating.

Comparisons of model fit.

The full ACE and ADE models were examined, as well as several nested models (i.e., AE, and CE) in order to determine the best-fitting model. Reduced models are fit in order to test the significance of each parameter in the model. In order to compare the fit of these nested models, a baseline model was fit first for each phenotype to estimate the variances, covariances and means by minimizing twice the log-likelihood (-2lnL). The obtained -2lnL of the baseline

model was then used to calculate a likelihood ratio chi-square test for each nested model. The fit of these nested models was then compared to that of the full model using the chi-square difference test (i.e., the -2lnL of the full model was subtracted from the -2lnL of the nested models), resulting in chi-square goodness of fit test for each model. Non-significant changes in chi-square are indicative of an improved fit of the model to the data. In addition to the chi-square difference test, Akaike's information criterion (AIC; χ^2-2*df*; (Akaike, 1987)), a measure of model fit versus model parsimony, was also used to compare models, where lower and/or more negative Akaike's Information Criterion values indicate a better-fitting model.

PAPER 2 RESULTS

The range in scores for binge eating (i.e., 0-7), compensatory behavior (i.e., 0-6) and all three perfectionism measures (i.e., TP= 34-127; CM= 9-45; DA= 4-19) indicate adequate variability in scores (see Table 1). Means and standard deviations for binge eating and compensatory behavior (see Table 1) are all consistent with previous mean values in similar-aged participants (Spanos, et al., 2010), and the percentage of participants scoring above the MEBS clinical cutoffs (i.e., binge eating = 2.5 and compensatory behavior = 1.5) was 21% for binge eating and 9% for compensatory behavior, suggesting our findings are similar to population-based estimates of the prevalence of these behaviors (Favaro, et al., 2003; Stice, et al., 2009).

The within-person, phenotypic correlation between binge eating and compensatory behavior (i.e., r=.39**) indicate a moderate, positive relationship between the two phenotypes. Phenotypic correlations between all three perfectionism variables and both binge eating (i.e., range r= .26-.36**) and compensatory behavior (i.e., range r=.24-.29**) also illustrate a moderate and significant degree of association. Importantly, the phenotypic relationship between binge eating and perfectionism is very similar in magnitude to that of compensatory behavior and perfectionism at the phenotypic level.

The MZ intraclass twin correlations for all variables are more than twice as large as DZ intraclass twin correlations, indicating the presence of genetic influences on all measures used in the present study (see Table 4). In addition, MZ twin correlations are all less than 1.00, indicating the presence of nonshared environmental influences on each phenotype. Shared environmental influences appear to be negligible, given the significant differences between MZ and DZ intraclass twin correlations.

Cross-twin, cross-trait correlations were also used to examine initial etiologic associations (see Table 5). The pattern of results remained consistent across variables. Specifically, the MZ cross-twin, cross-trait correlation between binge eating and compensatory behavior and between TP, CM and DA and these phenotypes was approximately two times the DZ twin correlation, suggesting that genetic factors likely contribute to the covariance between the two behaviors. In addition, all the MZ cross-twin, cross-trait correlations were similar in magnitude to the within-twin correlations, suggesting that nonshared environmental influences are likely significant in the relationship between the variables as well.

Trivariate model-fitting analyses were used to further examine genetic and environmental overlap between the variables of interest. The AE model was best-fitting for all three models, as evidenced by the lowest AIC values and the non-significant changes in chi-square from the full model (see Table 6). This indicates that the relationship between variables was attributed to additive genetic and nonshared environmental influences, with little shared environmental influences.

Unique and overlapping genetic and environmental estimates between perfectionism variables and disordered eating variables are presented in Table 7. Similar to previous analyses, etiologic associations revealed a moderate degree of overlap between binge eating and compensatory behavior. These associations were largely attributed to genetic influences, with substantial residual variance that suggests a large proportion of the variance in each phenotype is unique. In terms of overlap with perfectionism, findings were nearly identical for the TP and CM scales and both binge eating and compensatory behavior. Specifically, these perfectionism variables showed more genetic (r_a = .47-.62*) than nonshared environmental overlap (i.e., r_e = .05-.13), for both binge eating and compensatory behavior. Nonetheless, residual estimates were

larger (i.e., h_r^2=13-27%; e_r^2=64-66%; see Table 4) than the attributable estimates (i.e., h_a^2=6-13%; e_a^2=0-1%), suggesting that most of the etiologic variance in binge eating and compensatory behavior is independent of genetic and environmental factors operating in TP and CM.

In contrast, substantial differences were observed between binge eating and compensatory behavior in the DA model. Specifically, while nonshared environmental correlations were small, nonsignificant and similar for both binge eating and compensatory behavior (i.e., r_e=.05-.08), the genetic correlation between DA and binge eating (i.e., r_a=.80*) was substantial and almost twice that of the genetic correlation between DA and compensatory behavior (i.e., r_a=.46*). Similarly, the attributable genetic variance between DA and binge eating (i.e., h_a^2= .23) was nearly four times larger than the attributable genetic variance between DA and compensatory behavior (i.e., h_a^2=.06), suggesting greater overlap in genetic factors for binge eating. Indeed, the attributable genetic overlap between binge eating and DA (i.e., h_a^2= .23) accounted for more of the total variance in binge eating than unique factors (i.e., h_r^2= .13). In contrast, the residual genetic variance in compensatory behavior (i.e., h_r^2= .20) was larger than the overlap with DA (i.e., h_a^2= .06), suggesting most of the etiologic variance in compensatory behavior is independent of genetic factors operating in this measure of perfectionism.

PAPER 2 DISCUSSION

The aim of the present study was to explore whether perfectionism accounts for more unique variance in binge eating than compensatory behavior at the phenotypic and etiologic level. Perfectionism was examined using three different constructs of perfectionism (i.e., TP, CM and DA). With regard to TP and CM, no differences emerged in the degree of etiologic association between these variables and binge eating and compensatory behavior. Although most of the variance in binge eating and compensatory behavior was independent of these measures of perfectionism, the small degree of overlap was attributable almost entirely to shared genetic influences. This was evidenced by the moderate-to-large genetic correlations and small nonshared environmental correlations (Cohen, 1988).

In contrast, DA did show differences in the degree of etiologic overlap with binge eating and compensatory behavior (i.e., greater associations with binge eating). This suggests that perfectionism differentially accounted for more of the unique variance in binge eating than compensatory behavior. Etiologic associations were primarily driven by genetic factors as both the genetic correlations and attributable genetic variance between DA and binge eating were substantially greater than for DA and compensatory behavior. What, therefore, could account for these significant differences between binge eating and compensatory behavior and their relative genetic associations with DA?

Recent integrative models of binge eating focus on perfectionism and the role perfectionism might play in binge eating. Specifically, Bardone-Cone and colleagues (2006; 2000) proposed a three-factor interactive model whereby individuals with high perfectionistic standards, high body dissatisfaction and low self-efficacy or self-esteem experience negative affect when unable to achieve their desired weight/body image goals. This negative affect then

confers risk to binge eat in order to escape or obtain relief from these emotions. The three-factor model is specific to binge eating, but when the authors tested whether this model would extend to compensatory behavior as well, they found it predicted only binge eating behaviors in a nonclinical sample (Bardone-Cone, et al., 2006). Given the use of a nonclinical sample in the present study, our findings provide further support that this integrative model might apply to binge eating more than to compensatory behavior and can account for why the present study found differences between binge eating and compensatory behavior. The present study may also provide additional information pertinent to the three-factor theory by suggesting Doubts about Actions may be particularly linked to binge eating, and that this association may be due to a set of shared genetic risk factors. While the specific genetic risk factors are unknown, knowing that this association is primarily genetic sets up new pathways for understanding the link between perfectionism and binge eating (i.e., examining whether other parts of the three-factor model act as psychological and environmental "triggers" for shared genetic effects).

Future studies should attempt to address limitations of the current work, including the present study's reliance on self-report measures as there is currently some debate in the literature as to which assessment methods capture pathology most accurately. Specifically, over-reporting of binge eating behaviors may occur when relying on self-reports, as compared to interview methods (Johnson, Grieve, Adams, & Sandy, 1999). In contrast, other research suggests the use of interview methods in twin studies may result in higher nonshared environmental estimates and lower genetic and shared environmental estimates (Burt, 2009). Although the exact reason for this is unclear, one hypothesis suggests slight increases in measurement error, which loads onto nonshared environmental estimates, associated with interviews relative to questionnaire methods (Burt, 2009). Thus, future studies should attempt to use both self-report and interview-based

measures of binge eating and compensatory behavior to ensure findings replicate across assessment methods. In addition, the present study focused on a community sample rather than clinical cases. It remains unclear whether these results extend to individuals with clinical eating disorders, although all twin studies to date suggest similar levels of genetic and environmental influences on eating disorders and eating disorder behaviors (including binge eating and compensatory behavior) in clinical and non-clinical populations.

Despite these limitations, the present study is the first to my knowledge to explore whether perfectionism differentially accounts for some of the unique variance in binge eating versus compensatory behavior at the etiologic level. Knowing that a specific dimension of perfectionism (i.e., DA) differentiates these disordered eating phenotypes at the genetic level has important implications. Specifically, these findings have implications for the diagnostic questions being considered in DSM-5, as etiologically, there is evidence for considerable distinctiveness in the genetic factors contributing to binge eating and compensatory behavior. Similar to our previous study (Spanos, et al., in preparation), results lend support to current diagnostic categories that classify eating disorders and their subtypes by the presence or absence of binge eating and compensatory/purging behaviors. In addition, findings from the current study suggest disorders primarily characterized by binge eating may show more perfectionistic tendencies than those primarily characterized by compensatory behavior, with important implications for etiology and treatment. For example, intervention and prevention efforts should specifically target the types of perfectionistic attitudes present in the Doubts about Actions dimension of perfectionism (e.g., the need to repeat actions until they are "right") when treating binge eating, as this type of perfectionism may be a particularly potent risk factor.

Table 4
Intraclass Twin Correlations for MPS Scales with Binge Eating and Compensatory Behavior

Scale	MZ	DZ
Binge Eating (*N*= 423) *M* = 1.47 *SD* = 1.68 Min = 0 Max = 7 % ≥ clinical cutoff = 20.8	.38**†	.06
Compensatory Behavior (*N*= 423) *M* = .41 *SD* = .88 Min = 0 Max = 6 % ≥ clinical cutoff = 9.2	.30**†	.07
Total Perfectionism (*N*= 415) *M*= 72.22 *SD* = 18.14 Min = 34 Max = 127	.55**†	.26**
Concern over Mistakes (*N*= 411) *M* = 19.51 *SD* = 7.66 Min = 9 Max = 45	.58**†	.24**
Doubts about Actions (*N*=415) *M* = 8.56 *SD* = 3.53 Min = 4 Max = 19	.51**†	.23**

Note. MZ= monozygotic twins; DZ= dizygotic twins. Intraclass twin correlations are denoted in bold-face. Clinical cutoff scores for the MEBS binge eating and compensatory behavior subscales were determined using mean MEBS scores for young women with anorexia nervosa and bulimia nervosa and are as follows: BE = 2.5, CB = 1.5.

** $p < .01$ (2-tailed) indicating correlations are significant

† The MZ correlation is significantly different from the DZ correlation, $p < .001$ (1-tailed).

Table 5

Cross-Twin, Cross-Trait Correlations between Binge Eating, Compensatory Behavior with Perfectionism

	Binge Eating		Compensatory Behavior	
MPS Scales	MZ	DZ	MZ	DZ
Total Perfectionism	.26**†	.08	.17*	.08
Concern over Mistakes	.32**†	.14	.23**	.13
Doubts about Actions	.38**†	.09	.21**†	.01

Note. MPS= Multidimensional Perfectionism Scale; MZ= monozygotic twins; DZ= dizygotic twins.

** $p < .01$; * $p < .05$ (2-tailed) indicating correlations are significant.

† The MZ correlation is significantly different from the DZ correlation, $p < .05$ (1-tailed).

Table 6

Model Fit Indices for Trivariate Cholesky Decompositions

Model	Fit Statistics			
	-2ln*L*(df)	-2lnL_{Δ}(df)	*p*	AIC
Total Perfectionism				
Trivariate TP-BE-CB				
Saturated	6201.12 (1579)	--	--	--
ACE	6249.69 (1612)	48.57 (33)	<.001	-17.43
ADE	6246.95 (1612)	45.83 (33)	<.001	-20.17
AE	**6249.76 (1618)**	**48.64 (39)**	**1.00**	**-29.36**
CE	6264.67 (1618)	63.35 (39)	.02	-14.65
Concern Over Mistakes				
Trivariate CM-BE-CB				
Saturated	5427.22 (1575)	--	--	--
ACE	5481.94 (1608)	54.72 (33)	<.001	-11.28
ADE	5479.48 (1608)	52.26 (33)	<.001	-13.74
AE	**5481.98 (1614)**	**54.76 (39)**	**1.00**	**-23.24**
CE	5494.90 (1614)	67.68 (39)	.04	-10.32
Doubts About Actions				
Trivariate DA-BE-CB				
Saturated	4846.48 (1579)	--	--	--
ACE	4886.90 (1612)	40.42 (33)	<.001	-25.58
ADE	4884.29 (1612)	37.81 (33)	<.001	-28.19
AE	**4887.16 (1618)**	**40.68 (39)**	**1.00**	**-37.32**
CE	4904.74 (1618)	58.26 (39)	.01	-19.38

Note. A= additive genetic effects; D= dominant genetic effects; C= shared environmental effects; E= nonshared environmental effects; -2ln*L* = -2 times the log likelihood; -2lnL_{Δ}= differences in -2ln*L* values between the saturated model and subsequent models; AIC= Akaike's Information Criteria; BE = Binge Eating subscale from the Minnesota Eating Behavior Survey; CB = Compensatory Behavior subscale from the Minnesota Eating Behavior Survey; TP = Total Perfectionism from the Multidimensional Perfectionism Scale; CM=Concern Over Mistakes scale from the Multidimensional Perfectionism Scale; DA= Doubts About Actions scale from the Multidimensional Perfectionism Scale. Best-fitting model for each trivariate model is noted in bold text.

Table 7
Genetic and environmental correlations, total variance in and covariance between variables in the best-fitting AE models

Estimate	Binge Eating	Compensatory Behavior
	Total Perfectionism	
Heritability		
Total Perfectionism scale		
Total (h^2)	.52* (.40, .63)	.52* (.40, .63)
MEBS scale		
Total (h^2)	.35* (.22, .47)	.27* (.13, .39)
Attributable (h_a^2)	.08* (.02, .19)	.06* (.01, .14)
Residual (h_r^2)	.27* (.12, .40)	.18* (.07, .30)
Nonshared Environmentality		
Total Perfectionism scale		
Total (e^2)	.48* (.37, .60)	.48* (.37, .60)
MEBS scale		
Total (e^2)	.65* (.53, .78)	.73* (.61, .87)
Attributable (e_a^2)	$.00^{ns}$ (-.01, .03)	$.01^{ns}$ (.00, .05)
Residual (e_r^2)	.64* (.53, .77)	.66* (.53, .77)
Correlation		
Genetic (r_a)	.50* (.25, .76)	.47* (.19, .76)
Nonshared environmental (r_e)	$.05^{ns}$ (-.12, .21)	$.12^{ns}$ (-.03, .26)
	Concern over Mistakes	
Heritability		
Concern over Mistakes scale		
Total (h^2)	.52* (.40, .63)	.52* (.40, .63)
MEBS scale		
Total (h^2)	.34* (.21, .46)	.26* (.13, .39)
Attributable (h_a^2)	.13* (.05, .25)	.09* (.03, .18)
Residual (h_r^2)	.21* (.07, .36)	.17* (.03, .28)
Nonshared Environmentality		
Concern over Mistakes scale		
Total (e^2)	.48* (.37, .60)	.48* (.37, .60)

Table 7 (cont'd)

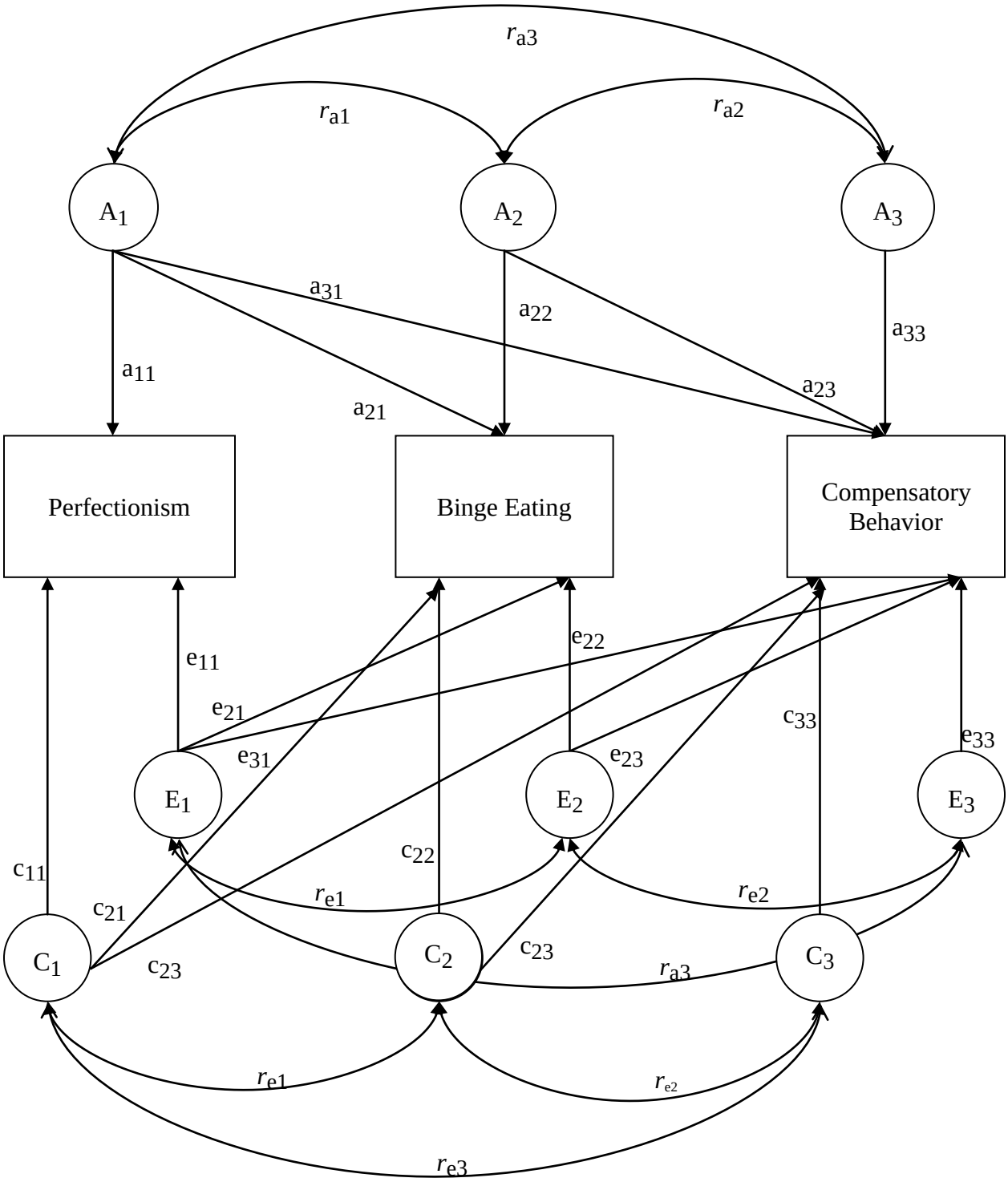
MEBS scale		
Total (e^2)	.66* (.54, .79)	.72* (.61, .87)
Attributable (e_a^2)	$.01^{ns}$ (.00, .05)	$.00^{ns}$ (.00, .04)
Residual (e_r^2)	.64* (.53, .77)	.66* (.55, .77)
Correlation		
Genetic (r_a)	.62* (.38, .87)	.58* (.32, .89)
Nonshared environmental (r_e)	$.13^{ns}$ (-.04, .28)	$.07^{ns}$ (-.08, .22)
	Doubts about Actions	
Heritability		
Doubts about Actions scale		
Total (h^2)	.48* (.35, .59)	.48* (.35, .59)
MEBS scale		
Total (h^2)	.35* (.22, .47)	.27* (.13, .40)
Attributable (h_a^2)	.22* (.10, .38)	.06* (.01, .14)
Residual (h_r^2)	.13* (.05, .25)	.20* (.05, .30)
Nonshared Environmentality		
Doubts about Actions scale		
Total (e^2)	.52* (.41, .65)	.52* (.41, .65)
MEBS scale		
Total (e^2)	.65* (.53, 78)	.73* (.60, .87)
Attributable (e_a^2)	$.00^{ns}$ (.01, .03)	$.00^{ns}$ (.00, .04)
Residual (e_r^2)	.64* (.53, .77)	.64* (.53, .77)
Correlation		
Genetic (r_a)	.80* (.55, 1.00)	.46* (.17, .77)
Nonshared environmental (r_e)	$.05^{ns}$ (-.21, .11)	$.08^{ns}$ (-.07, .23)

Note. MEBS= Minnesota Eating Behavior Survey; h^2= heritability; e^2= nonshared environmentality; r_a= genetic correlation; r_e= nonshared environmental correlation; ^{ns} = non-significant. The 95% confidence intervals for correlations are presented in parentheses. Correlations whose confidence intervals overlap with zero are non-significant.

* $p < .05$ (2-tailed)

Figure 5. Path Diagram of Trivariate, Cholesky ACE Model for Perfectionism, Binge Eating and Compensatory Behavior.The variance for each variable is accounted for by additive genetic effects (i.e., A_1, A_2, A_3), shared environmental effects (i.e., C_1, C_2, C_3) and nonshared environmental effects (and measurement error) (i.e., E_1, E_2, E_3). The genetic, shared and nonshared environmental variance in binge eating is decomposed into components that are accounted for by genetic and environmental effects on perfectionism (i.e., a_{21}, c_{21}, e_{21}) and residual components that are independent of perfectionism (i.e., a_{22}, c_{22}, e_{22}). The genetic, shared and nonshared environmental variance in compensatory behavior is decomposed into components that are accounted for by genetic and environmental effects on perfectionism (i.e., a_{31}, c_{31}, e_{31}), components that are accounted for by genetic and environmental effects on binge eating (i.e., a_{32}, c_{32}, e_{32}) and residual components that are independent of perfectionism (i.e., a_{33}, c_{33}, e_{33}). The genetic correlations (r_a), shared environmental correlations (r_c), and nonshared environmental correlations (r_e) are depicted with curved arrows.

Figure 5 (cont'd).



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