A LONGITUDINAL EXAMINATION OF THE STABILITY OF PERSONALITY CLUSTER MEMBERSHIP AND ASSOCIATIONS WITH PSYCHOPATHOLOGY

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ABSTRACT

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Objective: Women with bulimia nervosa (BN) often have very heterogeneous personality profiles. Cluster analysis has been used to identify personality profiles of women with BN. One of the clusters that has emerged most consistently is that of the dysregulated cluster, characterized largely by emotional lability and behavioral dysregulation. Despite the robustness of this cluster, previous research has been limited in that it has all been cross-sectional (and thus, stability of the clusters is unknown), and has mostly utilized clinical samples. The present study aimed to replicate the dysregulated cluster among a population-based sample and to examine the stability of cluster membership across time. **Method:** Participants included a longitudinal, convenience sample of female twins assessed at ages 17 and 25 from the Minnesota Twin Family Study. Facet scales from the Multidimensional Personality Questionnaire were used to cluster the participants based on their personality. BN symptoms and behaviors were assessed to determine the prevalence of this pathology by cluster. Additional measures (i.e., alcohol use disorder symptoms, depressive symptoms, trait anxiety, and behavioral disinhibition) were assessed to determine correlates of the clusters and whether they are stable across time. **Results:** The dysregulated cluster was identified at both time points and it emerged as the most stable profile compared to the other clusters. Examination of the correlates revealed increased levels of alcohol use disorder

symptoms, depressive symptoms, trait anxiety, and behavioral disinhibition among the dysregulated group with stability longitudinally. **Discussion:** Findings suggest that the dysregulated cluster is a relatively robust profile that is present across adolescence and into adulthood. Given the higher rates of BN symptoms in this compared to other clusters, future eating disorder research may benefit from focusing on this cluster when examining etiological and treatment features

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PREFACE

The following two papers comprise my dissertation. The first paper is entitled, "A Longitudinal Examination of the Stability of Personality Cluster Membership and Associations with Psychopathology." This paper focused on the two first aims of my dissertation proposal. Data from female twins, assessed longitudinally at ages 17 and 25, from the Minnesota Twin Family Study were utilized. Cluster analysis was used to determine personality cluster membership among the sample at both time points using the facet scales of the Multidimensional Personality Questionnaire. The main aims of this paper were to 1) replicate the dysregulated cluster, identified in several previous cluster analytic studies, among a population-based sample assessed in adolescence and young adulthood, and 2) examine the stability of cluster membership.

The second paper is an extension of the initial paper and is entitled, "Genetic and Environmental Factors Underlying Comorbid Bulimic Symptoms and Alcohol Use Disorder Symptoms: A Role for Personality?" The clusters identified at ages 17 and 25 in the first paper were utilized in this paper. Previous research has been inconsistent with regard to whether bulimia nervosa (BN) and alcohol use disorders (AUDs) share genes. This study hypothesized that the reason for these discrepancies is that previous twin studies have not considered the role of personality. Personality traits have been found to be very heterogeneous among women with BN. Therefore, studies examining whether common genetic or environmental factors underlie the co-occurrence of BN and AUDS may have been inconsistent because women with varied personality profiles (and potentially distinct etiologies) have been included in the same sample based on their eating disorder diagnosis. The goal of this second manuscript was to determine

whether the common genetic factors underlying the association between BN and AUDs are strongest among a particular cluster identified in the first paper (i.e., the Dysregulated cluster).

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INTRODUCTION

Impulsivity and related traits (e.g., sensation seeking, emotional instability) are often associated with bulimia nervosa (BN). Indeed, research has indicated that these traits are often higher among women with eating disorders who exhibit bulimic behaviors (i.e., bingeing and purging) compared to those who only restrict their intake and compared to controls (Cassin & von Ranson, 2005). However, additional studies, examining a range of personality traits among women with BN reveal a greater degree of personality heterogeneity beyond impulsivity (Vitousek & Manke, 1994). For example, in studies where individual personality traits were examined, women exhibiting bulimic behaviors have been found to report both low and high levels of negative affectivity, extraversion, and novelty-seeking (Westen, Thompson-Brenner, & Peart, 2006; Vervaet, van Heeringen, & Audenaert, 2004). Thus, although theoretically, many researchers associate impulsivity and related traits to BN, women with this diagnosis tend to vary with regard to their personality characteristics. This heterogeneity has been summarized in a review of personality studies in women with eating disorders (Westen et al., 2006) and has led some (e.g., Westen & Harnden-Fischer, 2001) to explore other approaches to investigating personality characteristics in individuals with BN.

A popular approach has been to use cluster analysis to group individuals into clusters based on similarities in personality profiles. Studies using the personality profiling approach have been relatively consistent with regard to finding clusters that are characteristically similar. Several studies found clusters that consist of groups characterized as high-functioning/perfectionistic (i.e., conscientious, perfectionistic, and anxious), constricted/overcontrolled (i.e., passive, avoidant, and emotionally constricted), and emotionally

dysregulated/undercontrolled (i.e., emotionally intense and labile, impulsive, poor negative mood regulation, and behaviorally disinhibited; see Table 1 for a summary of these studies). One of the most consistent profiles identified is that of a dysregulated cluster (e.g., emotionally dysregulated/undercontrolled cluster; Westen & Harnden-Fischer, 2001). Indeed, as shown in Table 1, every study to date has identified a cluster characterized largely by high impulsivity and behavioral dysregulation. Although these studies have focused primarily on clinical samples of individuals with eating disorders, they have consistently identified a dysregulated cluster despite differences in study measures (e.g., NEO- Five Factor Inventory, Millon Clinical Multiaxial Inventory-II, Dimensional Assessment of Personality Pathology; Claes et al., 2006; Goldner, Srikameswaran, Schroeder, Livesley, & Birmingham, 1999; Espelage, Mazzeo, Sherman, & Thompson, 2002) and developmental periods (i.e., adolescents and adults with eating disorders; Thompson-Brenner, Eddy, Satir, Boisseau, & Westen, 2008; Westen & Harnden-Fischer, 2001).

Notably, in several studies, the dysregulated cluster only included women who binge and/or purge (Claes et al., 2006; Perkins, Slane & Klump, in preparation; Thompson-Brenner et al., 2008; Westen and Harnden-Fischer, 2001), whereas other clusters included women who binge and purge as well as women who restrict their intake only. Furthermore, individuals with BN are distributed across all clusters in each of these studies, supporting the idea that despite a higher proportion of women with BN in the dysregulated group, these women represent a heterogeneous group with regard to personality profiles. Nonetheless, the higher proportion of BN in the dysregulated group suggest that this cluster may be uniquely associated with bulimic pathology and may represent a group that is categorically different from the other clusters that also include women who restrict.

Importantly, within subtyping research, the dysregulated cluster shows incremental validity over and above eating disorder diagnoses in terms of predicting disordered eating symptoms, adaptive functioning (as measured by Global Assessment of Functioning [GAF] scores), and illness severity (Thompson-Brenner & Westen, 2005; Westen & Harnden-Fischer, 2001). Specifically, in both Thompson-Brenner & Westen (2005) and Westen & Harnden-Fischer's (2001) studies, the dysregulated group exhibited a worse course than the other clusters. For example, the dysregulated cluster, had the greatest frequency of binges, the highest rates of psychiatric hospitalizations, the lowest and longest recovery rates, and received the lowest pretreatment GAF scores compared to the other clusters (Thompson-Brenner & Westen, 2005; Westen & Harnden-Fischer, 2001). These findings suggest that personality clusters may be useful in providing information on the current functioning of individuals with eating disorders and their long-term outcome.

Nonetheless, one could argue that the protracted course identified among this cluster suggests that the cluster represents symptom severity rather than a distinct personality profile. Indeed, this cluster has been associated with higher rates of substance use disorders, borderline personality disorder, depression, and anxiety (Claes et al., 2006; Espelage et al., 2002; Goldner et al., 1999; Thompson-Brenner & Westen, 2005; Thompson-Brenner et al., 2008; Wonderlich et al., 2005). Supporting this claim further, one study labeled this group as the "severe" cluster (Goldner et al., 1999).

However, there are some important distinctions that suggest that cluster membership is not just a measure of symptom severity. First, the dysregulated cluster does not score highest on all "negative" personality traits. For example, compulsivity has been associated with a longer time to recover among individuals with eating disorders (Strober, Freeman, & Morrell, 1997) and

is higher among other clusters (e.g., the rigid cluster in Goldner et al., 1999) compared to the dysregulated cluster. Second, several of the cluster studies utilized inpatient eating disorders samples, a group considered quite severe overall, given the need for intensive medical care. Yet, the inpatients with eating disorders in these samples did not all fall within the dysregulated cluster. Rather, they fell within three different clusters, suggesting again that the dysregulated group does not represent the most severe cluster. Third, as stated previously, individuals with anorexia nervosa, restricting type (AN-R) typically do not fall within dysregulated cluster (Claes et al., 2006; Thompson-Brenner et al., 2008, Westen and Harnden-Fischer, 2001). Yet women with AN-R have the highest mortality rate of any DSM-IV Axis I disorder (Sullivan, 1995). Thus, they are unlikely to be labeled as less severe than women falling in the dyregulated cluster.

In sum, cluster analyzing individuals with eating disorders based on personality appears to provide incremental validity beyond eating disorder diagnoses and offers additional information regarding associated mood symptoms and psychopathology. The dysregulated cluster has been most consistently identified and is characterized by increased impulsivity and emotional lability. Despite the robustness of previous studies with regard to replication of the dysregulated cluster, there are some limitations of past research.

First, although the dysregulated cluster has been identified among samples of adolescents and adults, due to the cross-sectional nature of previous research, it is unknown whether membership in the dysregulated cluster (and other clusters) is stable across time. As stated previously, cross-sectional research has replicated this cluster across several studies and found that bulimic behaviors are consistently higher in this cluster compared to the other clusters, suggesting that this cluster and its association with BN are robust. However, the lack of longitudinal findings makes it unclear whether the dysregulated cluster is a stable profile, present

in adolescence and young adulthood, and whether higher levels of BN are consistently found in this cluster across time. If longitudinal research supported this stability, it would suggest that the dysregulated cluster is potentially associated with the development of BN. In particular, examining this stability during the developmental transition from adolescence to young adulthood would lend strong support to this hypothesis, as BN typically emerges during these ages (American Psychiatric Association, 2000). Thus, stability of the dysregulated cluster as well as a strong association with BN during the periods of risk would suggest that the cluster of traits present in the dysregulated group may have strong etiological associations with BN, informing the development and course of this disorder.

The present study aimed to examine this stability by cluster analyzing a longitudinal sample of women assessed across two time periods spanning adolescence and young adulthood. Several correlates were examined at both time points to provide further support as to whether cluster groups and their associations with key correlates are stable across time. That is, previous research has identified significant associations by cluster with eating disorder symptoms and diagnoses, other Axis I disorders (e.g., anxiety, depression, alcohol use disorders) and personality traits, but they have not examined these correlates longitudinally. Examination of these correlates across time would provide information with regard to whether these associations have temporal stability and would therefore provide further support for the robustness of the dysregulated cluster and the potential etiologic association between this cluster and BN as well as associated psycholopathology.

Second, with the exception of one study that used a community based sample of women (Perkins et al., in preparation), all previous studies examining associations between cluster membership and eating disorders have used clinical samples of women. Indeed, even the study

using a community based sample of women only included those who endorsed eating disorder symptoms (although not full diagnoses; Perkins et al., in preparation). Although previous studies have examined personality profiles among females in the general population (e.g., Asendorpf, Borkenau, Ostendorf, & van Aken, 2001), no study to date has examined the association of clusters with eating disorders and related pathology among a population-based sample. It is important to examine these associations in a population-based sample as it will indicate that findings can be generalized and may be informative with regard to non-clinical levels of bulimic behaviors, which are far more common in the population than eating disorder diagnoses (Neumark-Sztainer & Hannan, 2000).

Given all of the above, the first aim of this study was to identify the dysregulated cluster using a longitudinal, population-based sample of women. The second aim was to examine whether cluster membership is stable from the transition from adolescence into young adulthood. Several correlates that have been examined in previous research and are most strongly associated with the dysregulated cluster (i.e., alcohol use disorder symptoms, depressive and anxiety symptoms, and levels of behavioral disinhibition) were included in the study for two main reasons. First, the present study aimed to replicate previous work showing strong associations between these correlates and the dysregulated cluster in a population-based sample. Second, given that the stability of clusters is of interest in the current study, stable associations with correlates (from adolescence to young adulthood) would provide further evidence that the clusters and their phenotypic profiles may be stable across development.

METHOD

Participants

Participants included an archival, convenience sample of female twins from the Minnesota Twin Family Study (MTFS). The MTFS is a population-based, longitudinal study of reared-together same-sex female twins and their parents. A detailed description of study recruitment and assessments can be found elsewhere (Iacono, Carlson, Taylor, Elkins, & McGue, 1999). Briefly, public databases were utilized to obtain birth records used to identify twins born in the state of Minnesota. Over 90% of twins born between 1971 and 1985 were located. An examination of several demographic variables from the US Census indicated that the MTFS is largely comparable to the Minnesota population (Holdcraft & Iacono, 2004).

The current study utilized cross-sectional and longitudinal data from two cohorts in the MTFS. Both cohorts were assessed at ages 17 (M = 17.87; SD = 0.74) and 25 (M = 25.04; SD = 0.69) years, but they began the studies at different ages and times. Cohort 1 began the study when they were 11 years old, whereas cohort 2 began the study at age 17. Due to some missing data at each time point as well as removal of outliers prior to data analysis (see Statistical Analyses), sample sizes differed across the age groups as follows: 1,264 females at age 17 and 1,184 females at age 25. Table 2 includes more information about the different cohorts including the years that they were assessed, their ages, and the sample size included in the present study.

Measures

Personality Measure for Cluster Analysis

Multidimensional Personality Questionnaire: The Multidimensional Personality Questionnaire (MPQ; Tellegen, 1982) was used to assess personality characteristics at ages 17 and 25. The MPQ is a198-item self-report, true-false questionnaire measuring personality characteristics on 11 facet scales. These scales include Well Being (e.g., optimistic, happy disposition), Social Potency (e.g., strong, persuasive), Achievement (e.g., ambitious, persistent), Social Closeness (e.g., sociable, warm), Stress Reaction (e.g.,

anxious, easily upset), Alienation (e.g., feels deceived or unlucky), Aggression (e.g., vindictive, violent), Control (e.g., cautious, rational), Harm Avoidance (e.g., avoids risks), Traditionalism (e.g., endorses religion and high moral standards), and Absorption (e.g., imaginative, able to re-experience the past, becomes absorbed in own thoughts, has episodes of heightened or altered consciousness).

Although previous research has largely focused on the MPQ higher-order factors, the individual MPQ facet scales measure several personality traits that are prominent among the personality clusters identified in previous cluster analytic studies (e.g., Westen and Harnden-Fischer, 2001). For example, the MPQ facet scales measure emotional dysregulation (i.e., MPQ alienation and stress reaction scales) and impulsivity (i.e., MPQ control scale), traits that are often increased among the dysregulated cluster (Thompson-Brenner & Westen, 2005; Westen & Harnden-Fischer, 2001; Wonderlich et al., 2005). Thus, the MPQ facet scales should provide adequate variability for replicating the dysregulated cluster as well as other clusters identified in previous research.

Internal consistency for the MPQ is very good with alphas for the 11 primary scales ranging from .76 to .90 (Tellegen, 1982). One-month test-retest reliability ranged from .82 to .92 (Tellegen, 1982). In the present sample, internal consistency ranged from .79 to .91, depending on the facet scale examined (see Table 3). Notably, the MPQ is useful for examining personality in population-based samples, as it was developed and standardized with nonclinical groups (Krueger, Caspi, & Moffitt, 2000). Further, in previous research, MPQ measured personality traits have shown moderate stability from adolescence into adulthood (Caspi & Silva, 1995; McGue, Bacon, & Lykken, 1993; Tellegen et al., 1988).

Measures for Bulimia Nervosa Symptoms

Bulimia Nervosa Symptoms: Symptom counts of bulimia nervosa (BN) were assessed using the Eating Disorders Structured Clinical Interview (EDSCI) and the Minnesota Eating Behavior Survey (MEBS; von Ranson, Klump, Iacono, & McGue, 2005)[1]. The EDSCI and MEBS were administered at both time points.

The EDSCI is a semi-structured interview based on Module H of the Structured Clinical Interview for DSM Axis I Disorders (SCID; Spitzer et al., 1987). Because the MTFS longitudinal assessments started in the early 1990s, both DSM-III-R and DSM-IV symptoms/diagnoses were assessed at both ages.

The present study utilized BN symptom counts and diagnoses in analyses. The BN symptoms that were assessed include binge eating, loss of control over eating, purging (e.g., vomiting) and nonpurging (e.g., excessive exercise) behaviors, and the undue influence of shape and weight on self-evaluation. These symptom counts allow for the examination of clinical symptomatology without having reduced power due to a potentially lower number of diagnoses, as the lifetime prevalence rate of BN is only 1-3% (American Psychiatric Association, 2000). Several studies have utilized subclinical measures of BN symptoms (Klump, McGue, & Iacono, 2000; Rowe, Pickles, Simonoff, Bulik, & Silberg, 2002; Sullivan, Bulik, & Kendler, 1998) and have supported the idea of a multiple threshold model of BN where both broad and narrow definitions of BN appear to result from a similar underlying vulnerability (Kendler et al., 1991).

BN diagnoses were coded as absent, probable, and definite based on symptoms endorsed. A definite diagnosis was given if the subject met full diagnostic criteria for BN. A probable

[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, 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.

diagnosis was given if the participant was one symptom short of a full diagnosis, but still had at least subclinical levels of binge eating (i.e., binges on an amount of food that is larger than an average meal and purging or nonpurging behaviors (e.g., excessive exercise) and/or frequency of these behaviors (e.g., twice a week for two months instead of twice per week for three months). Similar to previous research in population-based samples (e.g., Bienvenu et al., 2000), probable and definite diagnoses were combined to indicate that a diagnosis was present.

"Best estimates" of lifetime BN diagnoses and symptoms were assessed at age 17. That is, at this age, both the twin's mother and the twin herself reported on all BN symptoms, and best estimates coded a symptom as present if either the mother or twin indicated that it was present. At age 25, best estimates could not be used since the twin's mother did not report on symptoms at this time point. Thus, symptoms and diagnoses at age 25 were based on twin report only and include symptoms present over the earlier three to four years.[2] Notably, BN symptoms tend to develop in late adolescence and young adulthood and typically persist for at least several years (American Psychiatric Association, 2000). Thus, it is likely that many of the women with BN symptoms between ages 17 and 20 will have BN symptoms present between ages 20 and 5, assessed at the age 25 time point. Importantly, reliability of BN diagnoses and symptoms was good with kappa values ranging from .64 to 1.00.

The MEBS was also used to assess BN symptoms. This 30-item true/false self-report questionnaire assesses overall levels of eating pathology as well as specific disordered eating symptoms including body dissatisfaction (i.e., dissatisfaction with one's size or shape), binge

[2] Given that symptoms between ages 17 and 20 are not included in the symptom count at age 25, analyses were also conducted with variables indicating symptom counts and best estimate diagnoses between ages 17 and 25. Although the number of diagnoses and the mean symptom counts by cluster increased slightly, changes were minimal. Thus, results include the symptom count and diagnoses at age 25, which do not include symptoms and diagnoses between ages 17 and 20.

eating (i.e., thoughts about overeating or the tendency to binge eat) compensatory behaviors (i.e., the use of compensatory behaviors such as self-induced vomiting, diuretics, etc. for weight loss), and weight preoccupation (i.e., preoccupation with dieting, thinness, and weight). Examining these continuous measures of BN behaviors allowed for a more fine-tuned analysis of BN and its component symptoms. For example, examining scores on these measures within each group could reveal that personality cluster membership is differentially associated with specific bulimic symptoms (e.g., binge eating), rather than BN symptoms in general.

The internal consistency of the MEBS subscales has been shown to be adequate in samples of females ages 17 (α = .65-.89) and 20 years old (α = .68-.89) (Klump et al., 2000; von Ranson et al., 2005). The MEBS demonstrated sufficient discriminant validity through the ability to differentiate between normal control participants and individuals with eating disorders (von Ranson et al., 2005). Concurrent validity was also demonstrated through significant correlations (r = .68 - .72) between the MEBS subscales (i.e., Weight Preoccupation and Body Dissatisfaction) and similar subscales (i.e., Shape concerns and Weight Concerns subscales) from the Eating Disorders Examination Questionnaire (von Ranson et al., 2005). Notably, all subscales demonstrated sufficient discriminant validity through their ability to differentiate between normal control participants and individuals with eating disorders, including BN (von Ranson et al., 2005).

Measures of Cluster Correlates

Alcohol Use Disorder Symptoms: Alcohol abuse and dependence symptoms (i.e., alcohol use disorder [AUD] symptoms) were assessed at both ages using the Substance Abuse Module (SAM) from the Composite International Diagnostic Interview (Robins, Babor, & Cottler, 1987). These include symptoms such as recurrent alcohol-related legal problems, tolerance, and

withdrawal. The SAM is a well-established, semi-structured interview measure of these symptoms that has been used in field trials for the development of the DSM (Cottler et al, 1995; Spitzer, Williams, & Gibbon, 1987). The SAM shows excellent inter-rater reliability (Cottler, Robins, & Hezler, 1989), with an average kappa reliability of 0.92 for individual alcohol abuse and dependence symptoms. Further, kappa values from the MTFS assessment of AUD diagnoses using the SAM were excellent (all kappa's > .98).

For the purposes of this study, an AUD composite score was used in analyses. This composite score is a sum of all alcohol abuse and alcohol dependence symptoms. Combining these symptoms not only increases the variability within the sample, but also more closely resembles proposed changes to the AUD diagnosis in DSM-V, where alcohol abuse and dependence will be combined (American Psychiatric Association, 2010). Importantly, similar to BN, previous research supports the idea of a multiple threshold model of alcohol use (Heath et al., 1997; Kendler, Neale, Heath, Kessler, & Eaves, 1992, 1994), suggesting that findings for these disorders measured continuously are similar to those for full diagnoses.

Diagnoses of alcohol abuse and dependence were also included in analyses. For dependence, these included both probable (i.e., two full symptoms of dependence) and definite (i.e., three or more full symptoms of dependence) diagnoses. For abuse, these only included definite diagnoses, as only one symptom of abuse is needed to meet full diagnostic criteria. These included DSM-III-R diagnoses at age 17 and both DSM-III-R and DSM-IV diagnoses at age 25.

Depressive Symptoms: Symptoms of major depressive disorder were assessed using the SCID and included depressed mood, anhedonia, appetite disturbances, sleep problems, psychomotor agitation or retardation, fatigue, worthlessness or inappropriate guilt, difficulty concentrating or

indecisiveness, and suicidal ideation. Similar to BN and AUDs, at both time points symptom counts and diagnoses (best estimates at age 17) were included in analyses. Definite (five or more full symptoms of depression plus impairment) and probable (four full symptoms of depression, with one of the symptoms being depressed mood or anhedonia, plus impairment) and were combined to indicate the presence of a diagnosis of major depressive disorder. Notably, kappa reliabilities for the MTFS major depressive disorder diagnoses were excellent (range = .82 to .89).

Trait Anxiety: The State-Trait Anxiety Inventory (Form Y; STAI; Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983) is a 20-item self-report scale of state anxiety (i.e., current anxiety response to stressful situations) and trait anxiety (i.e., an individual's typical level of anxiety in the areas of apprehension, nervousness, and worry) that was administered to participants at both time points. For the purpose of this study, only the Trait anxiety scale was examined, as typical levels of anxiety may provide a more accurate picture of anxiety levels associated with personality cluster membership. Psychometric properties of the STAI Trait Anxiety scale are adequate in young adults. Alpha coefficients for high school and college females were .90 and .91, respectively (Spielberger et al., 1983). Test-retest reliability ranged from .65 to .77 for high school and college females over intervals between 20-104 days (Spielberger et al., 1983). Importantly, the STAI Trait Anxiety scale was not administered at age 25 for the 17-year-old cohort; thus, the sample size for these data is smaller at that age.

Behavioral Disinhibition Scale: The Behavioral Disinhibition (BD) scale is composed of 12 items from the Socialization scale of the California Personality Inventory (CPI; Cough, 1957). The Socialization scale is a 46-item self-report scale that assesses the extent to which an individual has antisocial tendencies (e.g., he/she engages in stealing, lying, truancy). Using

content and factor analysis of the CPI Socialization scale, Hicks (unpublished data) developed the BD scale that includes behavioral (e.g., "When I was going to school, I played hooky quite often") and personality (e.g., "I would do almost anything on a dare") items that more specifically measure BD than the broader Socialization scale. BD is characterized as a tendency towards difficulties with impulse control, including lack of foresight, poor negative mood regulation, and a need for instant gratification (Hicks, unpublished data). Participants completed this scale at age 17, but not at age 25. Internal consistency of the BD scale in a combined sample of 17 year old males and females is adequate (α = .68; Hicks, unpublished data).

Procedures for Diagnostic Interviews

AUD, BN, and major depressive disorder symptoms were assessed by trained bachelor's and master's level. Each symptom assigned was discussed in a clinical case conference with at least two advanced clinical psychology doctoral students. Discrepancies that emerged during the case conference were clarified by listening to interview audiotapes or re-contacting study participants. Symptoms were counted as present if they qualified for clinical significance in both frequency and severity.

Statistical Analyses

Data Preparation and Descriptive Statistics

Descriptive statistics (i.e., mean, standard deviation, and range) were computed for each personality facet at both time points. At both ages, the majority of the MPQ facet scales did not differ significantly across cohorts. Importantly, the facets that did differ across cohort had mean differences of small effect (i.e., .11-.40; Cohen, 1988) with the majority having an effect size of .24 or below suggesting minimal differences in trait levels across cohorts. Thus, the two cohorts were combined for all analyses.

Prior to cluster analysis, two steps were conducted to prepare the data. First, outliers (i.e., scores with an absolute *z*-score value of 3.29 or higher) were removed. It is important to remove outliers prior to cluster analysis because outliers can alter the true structure of the data (Hair, Black, Babin, Anderson, & Tatham, 2006) by including uncharacteristic cases that do not represent the population at large (Hair et al., 2006). Second, each of the personality variables were transformed to z-scores in order to attain means of zero and unit variance. Standardization of the variables is recommended when using continuous variables that should be considered equally (i.e., have the same case weight) in the cluster analysis (Wishart, 2006). If variables are left unstandardized prior to cluster analysis, scales with greater standard deviations will have a larger impact on the estimated proximity (i.e., similarity value; Hair et al., 2006).

Cluster Analysis

In order to determine whether the dysregulated cluster is present in our population-based sample, cluster analysis was conducted at both time points (i.e., age 17 and 25) separately using the MPQ facet scales. Cluster analysis of the personality variables was conducted using ClustanGraphics 8.06 (Wishart, 2006).

Several steps were utilized to determine the best cluster solution. First, cluster analysis was conducted using increase in sum of squares (i.e., Ward's method), a hierarchical method which minimizes the squared Euclidean distances among each item in a cluster and typically creates somewhat compact clusters (Wishart, 2006).

Second, two objective measures for determining the "best cut" or optimum number of clusters, were examined. ClustanGraphics offers the upper tail *t* test and tree validation as two methods for determining the best solution. As clusters are agglomerated, the upper tail *t* test inspects the difference in fusion value size during each step (Mojena, 1977; Mojena & Wishart,

1980; Wishart, 2006). A significantly large change as two clusters are combined suggests decreasing similarity/homogeneity of the agglomerated clusters. That is, items are being clustered together that are significantly more different/heterogeneous than those resulting from previous clusterings. By contrast, if there is not a significant increase in the fusion value size, then the clusters that are being combined are somewhat similar and the agglomeration does not contribute to an increase in cluster heterogeneity. The best solution, as indicated by the upper tail *t* test, is the cluster number prior to the initial significant increase in the fusion values (i.e., if the best fit is four clusters, moving from four to three clusters resulted in the first significant increase in heterogeneity within the agglomerated clusters).

Tree validation was also used in determining which cluster solution provides the most optimum fit to the dataset. This method compares the tree (i.e., dendogram) for the current dataset with multiple trees generated using random permutation of the data. Using the randomly permutated data, a distribution is formed and a confidence interval is estimated around the mean. The tree for the dataset is then compared with the confidence interval in order to determine whether there are significant departures from random assignment. Tree validation attempts to reject the hypothesis that the data do not have structure or are distributed randomly. This method seeks cluster solutions that represent the largest deviation from randomness, as indicated by the greatest absolute difference score. This score is determined by subtracting the randomly estimated fusion statistic from the fusion statistic that is estimated using the data (e.g., |ESSData – ESSRandom| = Absolute Difference; Wishart, 2006). Importantly, tree validation reports no significant cluster solutions when used with random data. In the present study, this bootstrap validation method was employed and 120 trials were randomly conducted without replacement (i.e., the default method for this analysis in ClustanGraphics). This random permutated data was

then compared to the proposed partitions to determine which cluster solution appears to provide the best-fit.

K-means analysis using FocalPoint was also employed as a secondary examination of optimum cluster fit. Given that there are many procedures for cluster analysis and no consensus as to which method is superior (Schmitt et al., 2007), k-means is often utilized as an additional method for examining the consistency of a cluster solution. FocalPoint attempts to replicate a predetermined cluster solution (e.g., the one indicated by Ward's method) across 1000 random permutations of the dataset. Results of the analysis indicate a percentage of exact replication of the data based on the permutations.

Lastly, in addition to statistical tests to determine the best fit of a cluster agglomeration, it is useful to determine whether a cluster solution makes conceptual sense based on theoretical assumptions (Aldenderfer & Blashfield, 1984; Hair et al., 2006; Rapkin & Luke, 1993; Wishart, 2006). Therefore, as a final assessment of fit, cluster solutions were examined based on these criteria of interpretability and subjective inspection (Aldenderfer & Blashfield, 1984; Rapkin & Luke, 1993).

Cluster Comparison

After determining the best cluster solution at each time point, multivariate analysis of variance (MANOVA) was performed to characterize clusters on MPQ facets. Significant multivariate effects were examined using univariate analysis of variance (ANOVA). Findings will provide information about which MPQ facet scales typify each of the clusters.

Cluster Membership Stability

Stability of clusters across ages 17 and 25 was examined by calculating Cohen's (1960) kappa coefficient (*k*). The *k* coefficient provides percentage agreement of group membership

between each time point corrected for what would be expected due to chance. In the present study, one *k* coefficient was calculated with time point equivalent to "rater" and cluster membership equivalent to group assignment. Suggested interpretations of these kappa coefficients have been reported by Cicchetti (1994). A *k* coefficient between .21 and .40 signifies fair stability of cluster membership, between .40 and .59 is moderate, .60 to .79 is substantial, and a kappa greater than .75 is excellent (Landis & Koch, 1977).

Cluster Correlates

Correlates of the clusters were examined at each time point to determine whether there are significant associations with particular clusters as well as to examine whether there is stability in the correlates by cluster across time. MANOVAs were computed to determine whether there are significant differences between groups on personality, mood, and symptom count variables. Significant multivariate effects were examined using ANOVA to determine which groups were significantly different on each variable. Lastly, the frequency of full diagnoses of major depressive disorder, BN, alcohol abuse, and alcohol dependence were examined within each cluster at both time points.

RESULTS

Descriptive Statistics

Means, standard deviations, ranges, and alphas for each MPQ facet scale at both time points are included in Table 3. Importantly, correlations between the same MPQ facet scales at ages 17 and 25 were all relatively high (data not shown; r's between .51 and .61) indicating overall stability of these personality characteristics within this sample.

Cluster Analysis of the MPQ

The best-cut analysis indicated that a four-cluster solution was the best fit for the sample at age 17, and a three-cluster solution was the best fit at age 25. Jumps in fusion values occurred at the four and three-cluster solution, respectively, where it was significant at $p < .05$ on an upper tail t test for the 17 (t (19) = 2.65) and 25-year-old samples (t (14) = 4.91).[3]

Tree validation, using Ward's method, was also utilized to determine the best cluster solution at ages 17 and 25 by comparing proposed data partitions with randomly permutated results. Similar to the best-cut analysis findings, tree validation results indicated that the four-cluster solution was the best fit at age 17, and the three-cluster solution was the best fit at age 25, as the absolute differences (i.e., absolute deviations between the random fusion statistic and estimated fusion statistic) were 25.50 and 38.7 at ages 17 and 25, respectively. These large absolute difference scores suggests that there is a high departure from randomness among the four cluster solution at age 17 and the three cluster solution at age 25 (i.e., clusters at each time point are created based on patterns in the data).

As noted earlier, K-means analysis using FocalPoint was used to examine the reproducibility of the four-cluster solution at age 17 and the three-cluster solution at age 25. Exact replication of the four-cluster solution at age 17 was reached 97.4% of the time with 1,000 random permutations of the data. At age 25, this percentage remained high with 75.3% exact reproducibility of the three-cluster solution. These findings suggest that cluster solutions at both ages provide good fits to the data.

MPQ Comparisons

[3] Due to the non-independence of twin data, analyses were also run separately with a random selection of twins from each pair at age 17 and at age 25. Findings for both groups of twins corresponded to those of the entire sample (data not shown.), i.e., a 4-cluster solution was best at age 17, and a 3-cluster solution was best at age 25.

In order to examine the differences among the clusters at each time point, one-way MANOVAs were conducted on all MPQ facet scales (see Table 4). There was a significant overall cluster effect at both time points (age 17 Wilks's λ =.18, F_{33}= 87.66, p<0.001; age 25 Wilks's λ = .27, F_{22} = 99.06, p<0.001). A series of one-way ANOVAs were conducted to examine whether there were significant differences between the MPQ facet scales based on group membership. Due to the large number of tests, a conservative Type I error rate of .01 was used for the univariate tests.

In addition to comparisons across cluster, it is helpful to examine whether mean levels for each cluster are significantly different from those of a normative sample. That is, differences may exist between clusters suggesting that a group is high on a particular MPQ facet, but without a normative comparison group, it is unclear if the trait is also elevated compared to the general population. Unfortunately, norms for the 198-item MPQ are not available. However, the full sample at each age is well over 1,000 participants and is comprised of a community-based sample. Therefore, comparisons were made against the means of the entire sample of participants at each time point (see Table 3 for sample means) using ANOVA. Results of these comparisons are indicated by effect sizes listed in the text for the facets that primarily characterize each cluster, whereas comparisons between clusters are identified in Table 4 and Figures 1 and 2.

Dysregulated Cluster

At ages 17 and 25, 21% (268/1264) and 37% (437/1184) of women fell within the Dysregulated cluster. At both time points, the Dysregulated cluster had the highest levels of Stress Reaction (d = .85 and .69 at ages 17 and 25, respectively), Alienation (d= .96 and .88) and Aggression (d = 1.23 and .60) compared to the full sample and other clusters (see Table 4 and

Figures 1 and 2 for comparisons between clusters). This indicates increased anxiety, feelings of betrayal, and physical and interpersonal aggressiveness among this cluster. In addition, the Dysregulated cluster had the lowest levels Achievement (d = .66 and .37) and Traditionalism (d = .64 and .24) compared to the full sample as well as the other groups, suggesting that this group has low levels of ambition and moral values. This group also had the lowest scores on Well Being (d = .71 and .80) and Social Closeness (d = .48 and .56) at both time points compared to the full sample and other clusters, suggesting that this group experiences low levels of happiness and is typically aloof and distant in social interactions. Lastly, this group had the lowest Control score at age 17 (d = .77) compared to the full sample and other clusters, and the second lowest at age 25 compared to the other groups, indicating that they may exhibit impulsive and rebellious behavior.

Importantly, this group closely represents that of the dysregulated clusters identified in previous studies (e.g., emotionally dysregulated/undercontrolled group; see Table 1). Following expectations, this cluster is characterized as more interpersonally aggressive, emotionally labile, and impulsive than the other clusters.

Resilient Cluster

Twenty percent (257/1264) of women at age 17, and 32% (375/1184) of women at age 25, fell within a Resilient cluster. The Resilient cluster largely resembled clusters identified in previous studies that were labeled “Resilients” or “High-Functioning” (Claes et al., 2006; Thompson-Brenner & Westen, 2005). This cluster is characterized predominately by high social closeness, cautiousness, and moral standards and low anxiety, feelings of alienation, and aggression, with significantly different mean levels of related traits (e.g., high Control) compared to the full sample (effect size range = .49-.99) and the other clusters (see Table 4).

Sensation Seeking Cluster

The Sensation Seeking cluster contained 32% (398/1264) of women at age 17 and 31% (372/1184) of the sample at age 25. This cluster contains women with strong, influential personalities, looking for novel, risky activities. This cluster had significantly different levels of related traits (e.g., low Harm Avoidance) compared to the full sample (effect size range = .34-.79) and the other clusters (see Table 4). Although the Sensation Seeking cluster is similar to the "undercontrolled" or "impulsive" clusters found in previous cluster analytic studies (e.g., Claes et al., 2006; Wonderlich et al., 2005) in that this cluster seeks exciting, risk activities, this group is characteristically different from the undercontrolled cluster in three specific ways. First, this group is more socially persuasive and effective interpersonally than the impulsive/undercontrolled clusters. Second, the Sensation Seeking cluster does not have the high levels of negative emotionality (i.e., Stress Reaction, Aggression, and Alienation) that are characteristic of the impulsive and undercontrolled clusters. Third, the Sensation Seeking cluster does not have the characteristic emotional instability (likely associated with high Stress Reaction in the present study) that is characteristic of the impulsive/undercontrolled groups. Therefore, of the clusters identified in the present study, the Sensation Seeking cluster is less similar to those identified in previous studies.

Inhibited Cluster

The Inhibited cluster was only present at age 17 with 27% (341/1264) of women falling within this cluster. Women in this cluster tend to opt for safe activities over risky ones, prefer being alone and not being the center of attention, and are not very imaginative. This group also demonstrated significantly different mean levels of related traits (e.g., high Harm Avoidance) compared to the full sample (effect size range = .56-.85) and the other clusters (see Table 4).

This cluster is similar to the Inhibited/Anxious and Avoidant/Depressed groups found in previous research (Thompson-Brenner et al., 2008; Wagner et al., 2006).

Alternate Cluster Solutions

As stated previously, one of the final methods to determine the best fit of cluster agglomeration is to determine whether the cluster solution makes theoretical sense (Aldenderfer & Blashfield, 1984; Hair et al., 2006; Rapkin & Luke, 1993; Wishart, 2006). In the present paper, if cluster solutions as determined by fit tests (i.e., upper tail *t* test, tree validation, and k means) were not followed, and instead, the criteria of interpretability and subjective inspection (Aldenderfer & Blashfield, 1984; Rapkin & Luke, 1993) were utilized, similar cluster solutions across both ages can be fit to the data. Indeed, when a three cluster solution is forced at age 17, it largely resembles that of the three cluster solution at age 25, with a Resilient, Sensation Seeking, and Dysregulated group emerging (see Table 5). Likewise, if a four cluster solution is fit to the data at age 25, the clusters that emerge are very similar to the Resilient, Inhibited, Sensation Seeking, and Dysregulated clusters identified by the fit tests at age 17 (see Table 5). These findings support the idea that the clusters are not randomly (or arbitrarily) assigned, but that the increased number of clusters at age 17 simply reflects greater variability in personality at this younger age rather than a different personality structure.

Cluster Membership Stability

Because the kappa statistic requires a symmetric two-way table with matching variable values at both time points, individuals falling within the fourth, Socially Inhibited cluster at age 17 were excluded from kappa analyses of cluster stability.

A Cohen's *k* coefficient of .31 indicated fair stability (i.e., a *k* coefficient between .21 and .40; Cohen, 1960) of cluster membership across the first three clusters (i.e., Sensation Seeking,

Resilient, and Dysregulated) from ages 17 to 25. As shown in Table 6, the majority of women (55%; 435/797) remained within the same cluster across time, with the Dysregulated cluster being the most stable. Roughly 53% (123/231) of women originally classified in the Resilient cluster at age 17 remained in that cluster at age 25, 48% (167/349) of women remained in the Sensation Seeking cluster, and 67% (145/217) remained in the Dysregulated cluster. The largest movement between clusters occurred between the Resilient and Sensation Seeking clusters, with 32% (73/231) of women who fell within Resilient group at age 17 moving to the Sensation Seeking cluster at age 25. The opposite movement was not quite as high with 24% (85/230) of women moving from the Sensation Seeking cluster at age 17 to the Resilient cluster at age 25. By contrast, the smallest movement was between the Dysregulated and Resilient clusters, where only 10% (22/217) of women categorized as Dysregulated at age 17 moved into the Resilient cluster at age 25. The reverse of this movement was also low with 15% (32/231) moving from Resilient at 17 to Dysregulated at 25.

Overall, cluster membership appears to be moderately stable from adolescence to young adulthood. The fourth cluster identified at age 17 (i.e., Inhibited), was not included in the stability analyses due to analytic constraints. However, among the Inhibited group, 42% (121/289) moved to the Resilient cluster at age 25, 17% (49/289) moved to the Sensation Seeking cluster, and 41% (119/289) moved to the Dysregulated cluster.

Correlates of the Personality Clusters

In order to examine correlates of the clusters at each time point and determine if associations of the correlates within the clusters are stable across time, one-way MANOVAs were conducted on mean levels of continuous BN symptoms, AUD symptoms, behavioral disinhibition, trait anxiety, and depressive symptoms (see Table 7). There was a significant

overall cluster effect at both time points (age 17 Wilks's λ =.58, F_{30}= 9.46, p<0.001; age 25 Wilks's λ =.66, F_{18} = 6.76, p<0.001). Conservative Type I error rates of .01 were used for the univariate tests.

As stated previously, probable and definite diagnoses were combined to form best estimate diagnoses for BN, alcohol abuse, alcohol dependence, and major depressive disorder. DSM-III-R and DSM-IV diagnoses were available at ages 17 and 25 for all disorders with the exception of major depressive disorder, alcohol abuse, and alcohol dependence at age 17 where only DSM-III-R diagnoses are available. Frequencies of these diagnoses as well as BN symptom counts were calculated for each cluster at both time points in order to examine differences in the prevalence of these disorders and symptoms.

Cluster Comparisons

BN symptoms and diagnoses

BN symptoms and diagnoses were observed in each of the clusters at both time points, supporting the idea of personality heterogeneity among individuals with this type of eating pathology (see Table 8). However, as expected, the Dysregulated group had the highest levels of bulimic behaviors at ages 17 and 25, suggesting that these behaviors may be more strongly associated with this type of personality profile, and that this association is stable across time. Specifically, at age 17, the univariate analyses indicated a significant cluster effect for BN symptom counts, MEBS total score (age 17: F (3, 597) = 6.55, p<.001 and binge eating, F (3, 597) = 12.18, p<.001. At age 25, this was indicated for MEBS total score, F (2, 889) = 26.42, p<.001), body dissatisfaction, F (2, 889) = 23.96, p<.001 and compensatory behavior, F (2, 890) = 6.13, p=.002. The Resilient cluster had the lowest mean levels of BN symptom counts at both time points.

BN diagnoses resulted in a more varied pattern (see Table 8); however, there was a very low rate of diagnoses overall across clusters (i.e., the largest number for any cluster at either time point was N = 5). Thus differences in BN diagnosis rates are less informative than the continuous measures of BN symptoms.

Alcohol use disorder symptoms and behavioral disinhibition

Findings corroborate expectations as the Dysregulated group had the highest mean levels of AUD symptoms at both time points, and the highest behavioral disinhibition scores at age 17. Indeed, at age 17, univariate analyses indicated significant differences among the clusters in the DSM-III-R alcohol abuse and dependence composite score, F (3, 1227) = 18.87, p<.001 and behavioral disinhibition scores from the Socialization scale of the CPI, F (3, 1191) = 83.52, p<.001. Although the mean alcohol composite score was the highest among the Dysregulated cluster at age 25, the p value in the overall MANOVA was not below .01, so the post-hoc ANOVA was not conducted. However, the p value was .021 suggesting a trend in cluster differences in the alcohol composite at age 25, indicating some stability in this correlate across time. Similar to the other correlate findings, the Sensation Seeking cluster had the second highest level of these symptoms, and the Resilient cluster had the lowest.

Trait anxiety and depression symptoms

Findings followed expectations with the Dysregulated cluster having the highest level of trait anxiety at both time points, suggesting stability in this correlate. Specifically, univariate analyses indicated a significant cluster effect for trait anxiety at age 17, F (3, 1005) = 95.12, p<.001, and 25, F (2, 490) = 81.80, p<.001. The Dysregulated cluster also had the highest mean level of depressive symptoms, F (2, 1171) = 20.49, p<.001, and diagnoses at age 25. As with the other correlates, the Resilient cluster had the lowest levels of these traits and symptoms.

DISCUSSION

The current investigation is the first to examine cluster membership in a longitudinal, population-based sample of women in an attempt to replicate the Dysregulated cluster, examine cluster membership stability across time, and determine whether correlates differ between the clusters and are stable longitudinally. Notably, the Dysregulated cluster, present in all previous cluster analytic examinations of women with eating disorders, was identified in this population-based sample during late adolescence as well as adulthood. In addition, findings indicated that the Dysregulated group was the most stable cluster. Examination of correlates of the Dysregulated cluster supported previous findings in that this group had the highest levels of bulimic behaviors, alcohol use problems, behavioral disinhibition, anxiety, and depressive symptoms. Further, the majority of these correlates remained stable across time. Overall, findings suggest that the Dysregulated cluster is a fairly stable group, not only among eating disorders samples, but also among the general population, with associated correlates that are relatively consistent from late adolescence to young adulthood.

It should be noted that although the other clusters identified in the present study largely resembled those from previous research (e.g., Resilient and Westen & Harden-Fisher's [2001] High-Functioning/Perfectionistic), the majority of previous studies found three cluster solutions. This was the case in the present study at age 25, but not at age 17. It is unclear why this discrepancy occurred. However, it may be due to three differences between the current study and those listed in Table 1. First, the present study had a significantly larger sample than the previous studies (i.e., N = 1,184-1,264 vs. N = 60-306 in studies in Table 1), and larger samples tend to result in larger numbers of clusters (Wishart, 2006). Nonetheless, three clusters were

identified at age 25, suggesting that sample size is not the only potential reason for this discrepancy.

Second, the current study used a population-based sample of females rather than individuals with clinical or subclinical eating disorder diagnoses. Although Perkins and colleagues (in preparation) used a community-based sample and also found three clusters, the sample only included women who reported symptoms of disordered eating. Therefore, that sample, although community-based, did not have the range of eating pathology (i.e., from no symptoms to full BN diagnoses) of the current, population-based sample. Thus, it is possible that there is a greater range of personality profiles among population-based samples. However, this was only true for the present sample during adolescence; therefore, identifying a four cluster solution among the sample at age 17 was likely not simply due to the fact that a population-based sample was utilized.

Third, most previous studies findings a three-cluster solution used adult samples, not adolescents. It may be that during adolescence, individuals in the general population segregate into more personality profiles than they do later in development. Indeed, other cluster analytic studies using adolescent samples have found more than three clusters (e.g., Stefurak, Calhoun, & Glaser, 2004). Therefore, it may be that there is more variability in personality traits among adolescents, leading to an increased number of personality clusters, compared to adults who generally tend to become more "functionally mature" (i.e., all adults tent to have an increase in traits that contribute to their development in important adult roles, such as parents and workers; Donnellan, Conger, & Burzette, 2007), and thus more similar. In support of this hypothesis, when a three-cluster solution was forced at age 17, cluster groups were very similar to those at age 25. Likewise, when a four-factor solution was forced at age 25, the groups largely

resembled those at age 17. Thus, similar clusters can be fit to the data at both ages, but fit statistics suggest that there are more clusters (and thus, more variability) at age 17 compared to age 25. Despite inconsistencies in the number of clusters identified, the present study provides additional support for the use of cluster analysis to inform eating disorder symptomatology and comorbidity.

The current study also expanded on extant research by being the first to use a longitudinal sample to examine stability of cluster membership across time. Findings indicated only fair stability (Cohen's *k* coefficient =.31) of clusters from ages 17 to 25. The overall fair stability may be accounted for, in part, by developmental changes in personality traits. Although studies have suggested moderate stability in MPQ personality traits across time (Caspi & Silva, 1995; McGue et al., 1993; Tellegen et al., 1988), some change in facet level traits may be expected across the lifespan (Caspi & Roberts, 2001). Indeed, personality research has indicated a "trend toward growth and maturity" (Blonigen et al., 2008; i.e., "functional maturity"; Donnellan et al., 2007) across time, with increases in "positive" traits associated with maturity (e.g., Achievement) and decreases in "negative" traits associated with immaturity (e.g., Aggression; Blonigen et al., 2008). For example, on the MPQ, mean levels of Harm Avoidance and Achievement tend to increase with age, while mean levels of Aggression and Stress Reaction tend to decrease (Blonigen et al., 2008). Indeed, in the present sample, there were increases in "positive" traits, such as Well Being and Achievement, and decreases in "negative" traits, such as Stress Reaction and Aggression, from ages 17 to 25 (see Table 3).

Examination of the cluster correlates longitudinally suggested stability in associations. Further, correlates of the Dysregulated cluster were largely similar to those of the dysregulated clusters identified in previous research. First, bulimic behaviors were present in all clusters, as

expected given the heterogeneity of the disorder, but the Dysregulated group had the highest mean levels of each symptom. Previous studies have produced similar findings with either higher rates of BN behaviors or higher rates of the diagnosis (compared to AN) in the Dysregulated group (Claes et al., 2006; Espelage et al., 2002; Westen & Harnden-Fischer, 2001) compared to the other clusters. This supports the idea that the dysregulated cluster may be more strongly associated with bulimic pathology, particularly given that women with AN-R are sometimes absent from this cluster (Westen and Harnden-Fischer, 2001). Interestingly, there were more associations between cluster membership and bulimic behaviors at age 25 (disordered eating, body dissatisfaction, and compensatory behavior) compared to age 17 (disordered eating and binge eating). It is unclear why this might be. However, eating pathology tends to increase from adolescence to young adulthood (see mean changes of disordered eating in Table 8), therefore, the increased prevalence and variability may have led to stronger associations with the clusters at age 25. Importantly, however, there was a significant association between cluster membership and disordered eating at both time points, with the highest mean levels of disordered eating among the Dysregulated cluster, suggesting stability of this correlate across time.

Second, the Dysregulated cluster had the highest level of AUD symptoms at age 17 and the most diagnoses of alcohol abuse and dependence at ages 17 and 25, suggesting that this correlate is also stable longitudinally. This finding was expected given that several studies have shown that rates of substance use disorders are significantly higher for women in the dysregulated group compared to the other clusters (Claes et al., 2006; Thompson-Brenner & Westen, 2005; Wonderlich et al, 2005). The Dysregulated cluster may be more susceptible to this particular type of comorbidity as this finding is more consistent than associations with other Axis I psychopathology (e.g., depression and anxiety).

Third, the Dysregulated group had significantly higher depressive symptoms and the most diagnoses of (major depressive disorder) MDD at age 25 as well as significantly higher trait anxiety at both time points compared to the other clusters. This corroborates some previous studies indicating the highest rates of anxiety and MDD were found among the dysregulated clusters (Claes et al., 2006; Thompson-Brenner & Westen, 2005). It must be noted, however, that this has been a less consistent finding among personality cluster studies with some finding that different clusters (e.g., Avoidant/Depressed, Thompson-Brenner et al., 2008 and Affective-Perfectionistic, Wonderlich et al., 2005) have higher levels of these internalizing symptoms. Nonetheless, associations with trait anxiety were stable across time in the present study, further supporting overall stability of the Dysregulated cluster and its correlates.

Fourth, the Dysregulated cluster had significantly higher behavioral disinhibition (e.g., impulse control difficulties and poor negative mood regulation) than the other three clusters at age 17. Stability of this association could not be examined as this measure was not included in the assessment at age 25. Yet, corroborating this finding, several other studies have found that the dysregulated cluster had the highest levels of borderline personality disorder symptoms and/or diagnoses (a disorder characterized by behavioral dyscontrol and emotional lability) compared to the other clusters (Claes et al., 2006; Espelage et al., 2002; Thompson-Brenner et al., 2005; Thompson-Brenner et al., 2008). The association between BN and borderline personality disorder is not surprising given that the most common personality disorder diagnosis among women who engage in bulimic behaviors is borderline personality disorder (Cassin & von Ranson, 2005). Further, borderline personality disorder symptoms have also been found to be significantly associated with AUDs (Trull, Waudby, & Sher, 2004). It is interesting, however,

that once again, this cluster has the highest rates of this type of symptomatology, particularly given that women with BN symptoms were present in each cluster.

The present study was the first study to replicate the Dysregulated cluster longitudinally within a population-based sample and also provide information about cluster stability. However, there are some limitations that must be noted. First, despite findings suggesting that the dysregulated cluster is a personality cluster that is categorically different from other subgroups based on traits and not on severity, there is still some evidence to suggest the contrary. Indeed, the Dysregulated cluster did have the highest mean levels of psychopathology on the measures included in the present study. Nonetheless, previous research has indicated that cluster analysis provides incremental validity beyond eating disorder diagnoses, supporting its utility (Thompson-Brenner & Westen, 2005; Westen & Harnden-Fischer, 2001). Further, the present study provided initial evidence that there is some stability in cluster membership across time, particularly in the Dysregulated cluster. Thus, even if clusters are associated with symptom severity, this method is still informative, particularly with regard to identifying distinct personality profiles (and associated correlates) among individuals with BN, who tend to have very heterogeneous personality traits (Vitousek & Manke, 1994).

Second, the present study did not include measures of external validation, such as outcome, treatment response, and course. It would be useful to confirm that these clusters are externally valid across time. As stated previously, extant research has supported the external validity of the clusters by examining pretreatment GAF scores, psychiatric hospitalizations, and recovery rates (Thompson-Brenner & Westen, 2005; Westen & Harnden-Fischer, 2001). However, these outcome measures have never been examined longitudinally so it is unclear if the clusters have predictive validity across time.

Overall, the current study indicated that the dysregulated cluster is a robust personality profile that is present in adolescence and adulthood and is relatively stable across time. Given the increased rates of bulimic behaviors and associated symptoms (e.g., AUD symptoms, depression, and trait anxiety) in the dysregulated cluster, both cross-sectionally and longitudinally, future researchers may want to focus on this group (or this cluster of personality traits) when examining the etiology of BN as well as treatments for the disorder.

Table 1

Studies utilizing cluster analysis to examine personality profiles in samples of women with eating disorders and eating pathology

Study	**Sample**	**Age**	**Measure(s)**	**Clusters**
Claes et al., 2006	AN-R = 84 AN-BP = 73 BN-NP = 38 BN-P = 99 EDNOS = 12	21.5-24.3 years (SD = 3.7-7.3)	Neuroticism, Extraversion, Openness to New Experience - Five Factor Inventory (NEO-FFI; Costa & McCrae, 1992)	1) Undercontrollers 2) Resilients 3) Overcontrollers
Espelage, Mazzeo, Sherman, & Thompson, 2002	AN = 33 BN = 91 EDNOS = 59	21.81 years (SD = 5.03)	Millon Clinical Multiaxial Inventory-II (MCMI-II; Million, 1987)	1) High-functioning 2) Undercontrolled/Dysregulated 3) Overcontrolled/Avoidant
Goldner et al., 1999	AN-R = 18 AN-BP = 19 BN = 84 EDNOS= 15	26.5 years (SD = 6.6)	Dimensional Assessment of Personality Pathology (DAPP-BQ; Livesly, Jackson, & Schroeder, 1991; Schroeder, Wormworth, & Livesly, 1992)	1) Rigid 2) Severe 3) Mild
Thompson-Brenner & Westen, 2005	BN-P = 104 BN-NP = 20 AN-BP = 9 EDNOS = 12	28.5 years (SD = 10.2)	Personality prototype ratings	1) High-functioning 2) Constricted 3) Dysregulated
Thompson-Brenner, Eddy, Satir, Boisseau, & Westen, 2008	AN = 18 BN = 44 EDNOS = 57 (1 missing)	16.5 years (SD = 1.2)	Shelder-Westen Assessment Procedure for Adolescents (SWAP-II-A; Westen, Dutra, Shedler, 2005; Westen, Shedler, Durrett, Glass, & Martens, 2003) Q-sort	1) High-functioning/Perfectionistic 2) Dysregulated 3) Avoidant/Depressed

Note: AN = anorexia nervosa; AN-BP = anorexia nervosa binge-purge type; AN-R = anorexia nervosa restricting type; BN = bulimia nervosa; BN-NP = bulimia nervosa nonpurging type; BN-P = bulimia nervosa purging type; CON = control group; EDNOS = eating disorder not otherwise specified.

Table 1 (cont'd)

Study	Sample	Age	Measure(s)	Clusters
Wagner et al., 2006	Recovered AN-R = 21 Recovered AN-BP or BN = 20 Current BN = 19	23.8-28.0 years (SD = 5.2-6.9)	Temperament and Character Inventory (TCI; Cloninger, Przybeck, Svrakic, et al., 1994); Barratt Impulsiveness Scale (BIS; Barratt & Patton, 1983)	1) Impulsive 2) Inhibited/Anxious
Westen & Harnden-Fischer, 2001	AN-R = 13 AN-BP = 35 BN = 45 (10 missing)	30.0 years (SD = 8.8)	Shelder-Westen Assessment Procedure-200 (SWAP-200; Westen & Shedler, 1999a; Westen & Shedler, 1999b) Q-sort	1) High-Functioning/Perfectionistic 2) Constricted/Overcontrolled 3) Emotionally Dysregulated/ Undercontrolled
Wonderlich et al, 2005	BN-P = 119 BN-NP = 5 EDNOS = 54 (subclinical BN)	25.56 years (SD = 8.88)	Impulsivity, perfectionism, depression, anxiety, substance abuse, obsessive-compulsive symptoms, genotype	1) Low Co-morbidity 2) Affective-Perfectionistic 3) Impulsive
Perkins, Slane, & Klump, in preparation	AN-R = 20 AN-BP = 62 CON = 109	19.19 years (*SD* = 1.32)	DAPP-BQ (Livesly et al., 1991; Schroeder et al., 1992)	1) Adaptive (Resilients) 2) Rigid (Overcontrollers) 3) Dysregulated (Undercontrollers)

Note: AN = anorexia nervosa; AN-BP = anorexia nervosa binge-purge type; AN-R = anorexia nervosa restricting type; BN = bulimia nervosa; BN-NP = bulimia nervosa nonpurging type; BN-P = bulimia nervosa purging type; CON = control group; EDNOS = eating disorder not otherwise specified.

Table 2

Assessment Years, Sample Sizes, and Ages of Participants in Cohort 1 and 2 at each Time Point

	Age 17 Assessments			Age 25 Assessments		
	Assessment Years	N	Age M (SD)	Assessment Years	N	Age M (SD)
Cohort 1	1999-2006	594	17.45 (.50)	2006-2010	573	24.97 (.71)
Cohort 2	1993-2001	670	18.28 (.70)	2000-2005	611	25.08 (.65)

Table 3

Means and Standard Deviations for the Multidimensional Personality Questionnaire (MPQ) Facet Scales at Ages 17 and 25

	Age 17 (*n* = 1190-1264)				Age 25 (*n* =1114-1184)			
Scale	M	SD	Ranges	α	M	SD	Ranges	α
Well Being	55.99	7.98	30-72	.89	56.20	7.60	31-72	.90
Social Potency	45.17	8.20	22-72	.87	43.67	8.41	20-70	.89
Achievement	48.20	8.11	23-72	.87	50.09	7.62	27-72	.87
Social Closeness	56.31	8.14	30-72	.87	56.75	7.84	30-72	.88
Stress Reaction	44.59	9.27	20-70	.88	41.72	9.04	19-69	.89
Alienation	34.37	8.48	18-62	.89	29.87	7.71	18-56	.90
Aggression	34.08	8.52	18-61	.88	29.10	6.44	18-50	.85
Control	48.36	7.47	24-71	.84	52.40	7.40	28-72	.87
Harm Avoidance	50.11	9.81	18-72	.83	54.19	9.47	24-72	.84
Traditionalism	52.67	6.49	32-71	.76	52.52	6.63	31-72	.78
Absorption	43.39	9.41	18-70	.87	39.81	9.11	19-68	.88

Note. MPQ facet scales were z-transformed prior to analysis; however, raw scores are shown for descriptive purposes. Sample sizes vary due to missing data.

Table 4

Means of Three- and Four-Cluster Solutions for Multidimensional Personality Questionnaire (MPQ) Facet Scales at Ages 17and 25

	Age 17									
	Resilient Cluster 1 (n=257, 20%)		Sensation Seeking Cluster 2 (n=398, 32%)		Dysregulated Cluster 3 (n=268, 21%)		Inhibited Cluster 4 (n=341, 27%)		*F*-value	Pairwise Contrast
	M	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*		
Well Being	**62.60**	**5.13**	59.51	6.06	50.68	6.87	51.06	6.51	278.22	1>2>3&4
Social Potency	47.45	7.62	**49.87**	**7.33**	44.07	6.19	**38.84**	**6.52**	166.47	2>1>3>4
Achievement	**52.85**	**8.05**	50.21	7.63	**43.50**	**5.86**	46.06	7.59	89.88	1>2>4>3
Social Closeness	**61.44**	**6.91**	58.37	6.99	52.49	7.67	53.04	7.75	99.41	1>2>3&4
Stress Reaction	**37.02**	**7.51**	43.30	7.97	**51.82**	**7.62**	46.11	8.26	161.74	3>4>2>1
Alienation	**27.08**	**6.00**	33.77	6.96	**42.12**	**7.67**	34.49	7.01	206.36	3>2&4>1
Aggression	**27.21**	**4.95**	34.39	7.18	**43.65**	**6.99**	31.37	6.09	314.45	3>2>4>1
Control	**53.14**	**7.56**	46.51	6.06	**43.06**	**6.29**	51.07	6.27	136.53	1>4>2>3
Harm Avoidance	52.93	9.34	**45.24**	**9.50**	48.29	9.26	**55.10**	**7.59**	87.06	4>1>3>2
Traditionalism	**56.81**	**6.53**	51.71	6.40	**49.40**	**5.29**	53.25	5.62	71.73	1>4>2>3
Absorption	41.12	9.55	**48.31**	**8.91**	44.60	7.08	**38.41**	**8.38**	90.35	2>3>1>4

Note. Sample sizes vary across age due to missing data. Boldface indicates significantly higher or lower scores on a facet scale compared to the other clusters at that time point.

Table 4 (cont'd)

	Age 25							
	Resilient Cluster 1 (n=375, 32%)		Sensation Seeking Cluster 2 (n=372, 31%)		Dysregulated Cluster 3 (n=437, 37%)		*F*-value	Pairwise Contrast
	M	*SD*	*M*	*SD*	*M*	*SD*		
Well Being	59.38	6.25	59.65	5.82	**50.53**	**6.57**	286.14	1&2>3
Social Potency	42.69	8.53	**47.14**	**8.14**	41.57	7.58	52.03	2>1&3
Achievement	50.46	7.36	**52.79**	**8.07**	**47.48**	**6.52**	53.92	2>1>3
Social Closeness	**60.90**	**6.41**	57.63	7.01	**52.45**	**7.45**	151.44	1>2>3
Stress Reaction	**36.73**	**7.62**	39.97	8.29	**47.48**	**7.48**	205.97	3>2>1
Alienation	**24.53**	**4.53**	28.03	6.86	**36.03**	**6.20**	402.25	3>2>1
Aggression	**25.06**	**3.98**	28.63	5.80	**32.97**	**6.42**	207.03	3>2>1
Control	**56.28**	**6.72**	50.37	7.98	50.79	6.04	87.02	1>2&3
Harm Avoidance	**60.02**	**6.84**	**47.12**	**8.30**	55.21	8.46	252.60	1>3>2
Traditionalism	**55.60**	**6.03**	51.16	7.32	51.03	5.53	65.98	1>2&3
Absorption	**35.34**	**8.61**	**43.60**	**9.67**	40.41	7.23	90.08	2>3>1

Note. Sample sizes vary across age due to missing data. Boldface indicates significantly higher or lower scores on a facet scale compared to the other clusters at that time point.

Table 5

Means of Forced Four- and Three-Cluster Solutions for Multidimensional Personality Questionnaire (MPQ) Facet Scales at Ages 17and 25

	Age 17							
	Resilient Cluster 1 (n=370; 23%)		Sensation Seeking Cluster 2 (n=376, 24%)		Dysregulated Cluster 3 (n=846; 53%)		*F*-value	Pairwise Contrast
	M	*SD*	*M*	*SD*	*M*	*SD*		
Well Being	60.71	5.29	**61.88**	**5.51**	**51.18**	**6.83**	531.07	2>1>3
Social Potency	45.91	7.36	**50.91**	**7.22**	**42.21**	**7.66**	178.48	2>1>3
Achievement	49.12	7.46	**52.74**	**8.48**	**45.67**	**7.76**	108.98	2>1>3
Social Closeness	**62.13**	**5.57**	58.02	7.23	**52.60**	**7.96**	236.45	1>2>3
Stress Reaction	**37.25**	**6.92**	41.32	7.82	**48.94**	**8.32**	320.51	3>2>1
Alienation	**27.31**	**5.83**	31.88	7.38	**38.70**	**8.07**	330.68	3>2>1
Aggression	**27.77**	**5.10**	34.05	8.65	**36.80**	**8.53**	168.24	3>2>1
Control	**51.56**	**7.63**	47.46	7.91	46.94	7.43	49.81	2=3>1
Harm Avoidance	**53.76**	**8.64**	**43.18**	**9.40**	51.35	9.13	147.12	1>3>2
Traditionalism	**55.83**	**6.06**	52.01	7.82	**50.63**	**5.86**	84.65	1>2>3
Absorption	**37.82**	**8.26**	49.52	8.30	**51.18**	**6.83**	174.75	3>2>1

Note. Sample sizes vary across age due to missing data. Boldface indicates significantly higher or lower scores on a facet scale compared to the other clusters at that time point.

Table 5 (cont'd)

	Age 25									
	Resilient Cluster 1 (n=277, 23%)		Sensation Seeking Cluster 2 (n=372, 31%)		Dysregulated Cluster 3 (n=437, 37%)		Inhibited Cluster 4 (n=98, 8%)		*F*-value	Pairwise Contrast
	M	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*		
Well Being	**61.88**	**4.55**	59.65	5.82	**50.53**	**6.57**	52.32	4.85	288.81	1>2>4>3
Social Potency	45.40	7.63	47.14	8.14	41.57	7.58	**35.02**	**5.86**	83.08	2=1>3>4
Achievement	51.63	7.04	52.79	8.07	47.48	6.52	47.17	7.29	45.94	2=1>3=4
Social Closeness	**62.33**	**5.43**	57.63	7.01	**52.45**	**7.45**	56.85	7.23	120.19	1>2=4>3
Stress Reaction	**35.05**	**7.02**	39.97	8.29	**47.48**	**7.48**	41.48	7.28	160.31	3>4=2>1
Alienation	24.45	4.59	28.03	6.86	**36.03**	**6.20**	24.76	4.36	268.04	3>2>4=1
Aggression	24.87	3.88	28.63	5.80	**32.97**	**6.42**	25.58	4.21	138.44	3>2>4=1
Control	55.75	6.65	50.37	7.98	50.79	6.04	**57.80**	**6.71**	60.41	4>1>3=2
Harm Avoidance	59.19	6.66	**47.12**	**8.30**	55.21	8.46	**62.36**	**6.83**	173.79	4>1>3>2
Traditionalism	**56.45**	**5.82**	51.16	7.32	51.03	5.53	53.19	5.99	51.26	1>4>2=3
Absorption	36.82	8.56	**43.60**	**9.67**	40.41	7.23	**31.16**	**7.32**	72.67	2>3>1>4

Note. Sample sizes vary across age due to missing data. Boldface indicates significantly higher or lower scores on a facet scale compared to the other clusters at that time point.

Table 6

Stability of Cluster membership (Total n = 797)

Age 17 Cluster Membership	**Age 25 Cluster Membership** Resilient (Cluster 1)	Sensation Seeking (Cluster 2)	Dysregulated (Cluster 3)	Age 17 Cluster Totals
Resilient (Cluster 1)	**123 (53%)**	73 (32%)	35 (15%)	231
Sensation Seeking (Cluster 2)	85 (24%)	**167 (48%)**	97 (28%)	349
Dysregulated (Cluster 3)	22 (10%)	50 (23%)	**145 (67%)**	217
Age 25 Cluster Totals	230	290	277	797

Note. Percentages in parentheses are out of the total number of participants included in each cluster at age 17. The bold-face numbers on the diagonal indicate individuals who remained in the same cluster across time. Off-diagonal numbers depict individuals who moved between clusters across time.

Table 7

Comparisons between significant correlates at ages 17 and 25

	Age 17				Age 25		
	Resilient Cluster 1	Sensation Seeking Cluster 2	Dysregulated Cluster 3	Inhibited Cluster 4	Resilient Cluster 1	Sensation Seeking Cluster 2	Dysregulated Cluster 3
MEBS Total Score	5.03 (4.92)$_a$	5.96 (5.03)$_{ab}$	7.93 (5.85)$_c$	6.92 (6.10)$_{bc}$	5.59 (4.69)$_a$	6.33 (5.60)$_a$	8.63 (5.81)$_b$
Binge Eating	0.64 (1.03)$_a$	0.92 (1.27)$_{ab}$	1.66 (1.61)$_c$	1.13 (1.46)$_b$	--	--	--
Body Dissatisfaction	--	--	--	--	2.17 (2.05)$_a$	2.20 (2.19)$_a$	3.24 (2.27)$_b$
Compensatory Behaviors	--	--	--	--	.13 (.46)$_a$	.27 (.72)$_b$	.30 (.70)$_b$
Trait Anxiety	30.02 (5.29)$_a$	34.40 (6.62)$_b$	41.07 (7.41)$_d$	37.20 (7.75)$_c$	29.28 (5.83)$_a$	31.94 (6.10)$_b$	38.56 (8.03)$_c$
Behavioral Disinhibition	1.40 (1.38)$_a$	2.63 (1.97)$_b$	3.81 (2.50)$_c$	1.70 (1.67)$_a$	--	--	--
Depressive Symptoms	--	--	--	--	0.56 (1.64)$_a$	0.91 (2.16)$_a$	1.56 (2.66)$_b$
Alcohol composite	0.18 (0.63)$_a$	0.46 (1.32)$_b$	0.95 (2.08)$_c$	0.23 (0.89)$_{ab}$	--	--	--

Note. Depressive Symptoms represents DSM-III-R symptoms of major depressive disorder. Alcohol composite consists of DSM-III-R alcohol abuse and dependence symptoms combined. Raw scores shown for descriptive purposes. Significant differences between clusters are identified by different subscript letters.

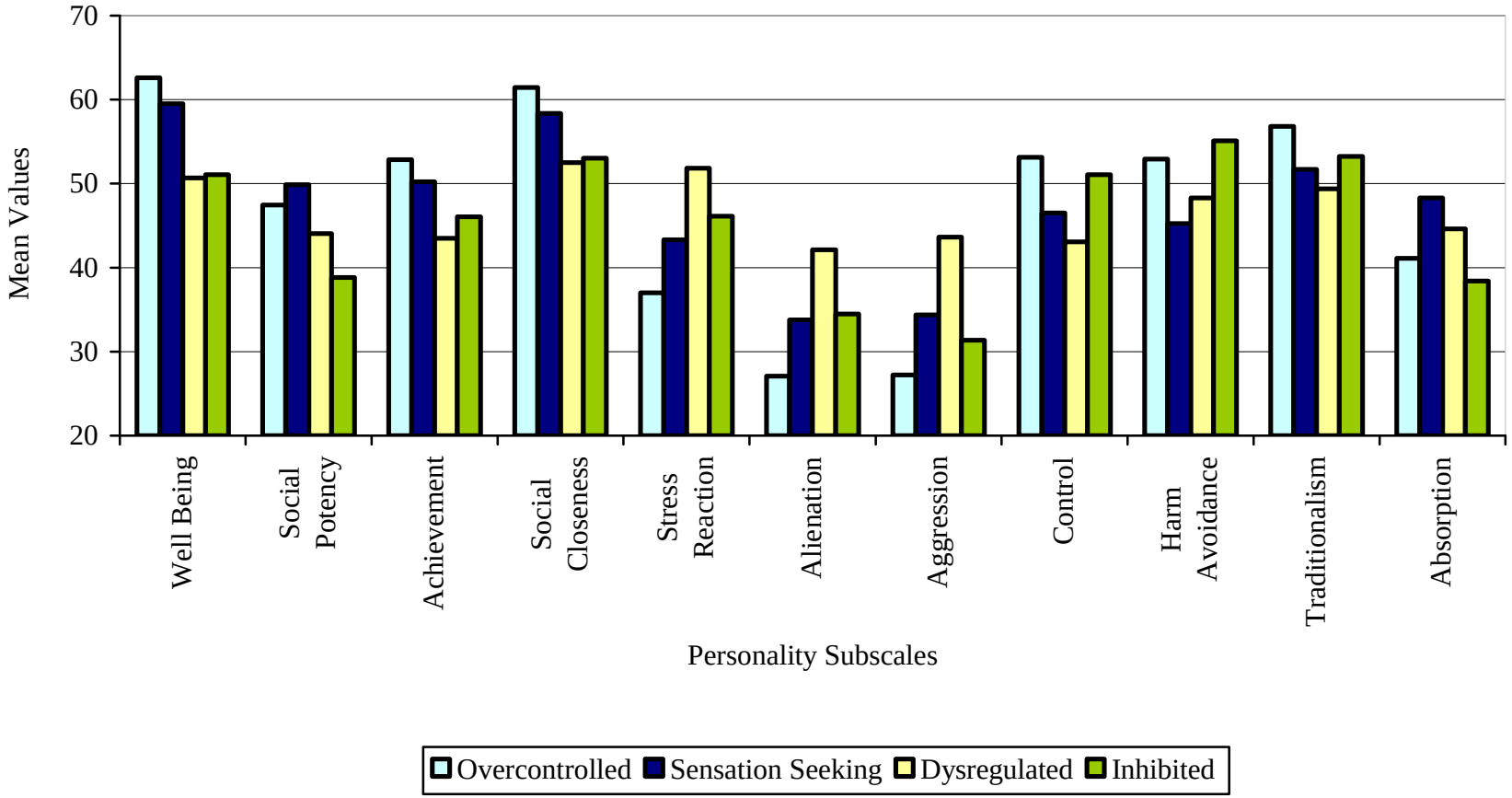
Table 8

Comparisons between Clusters on Diagnoses

	Age 17				Age 25		
	Resilient Cluster 1	Sensation Seeking Cluster 2	Dysregulated Cluster 3	Inhibited Cluster 4	Resilient Cluster 1	Sensation Seeking Cluster 2	Dysregulated Cluster 3
	Combined Number of Probable and Definite Diagnoses						
DSM-III-R Diagnoses							
Bulimia Nervosa	1 (0.4%)	3 (0.8%)	4 (1.5%)	1 (0.3%)	1 (0.3%)	2 (0.5%)	1 (0.2%)
Major Depressive D/O	24 (9%)	58 (15%)	53 (20%)	48 (14%)	24 (6%)	40 (11%)	72 (17%)
Alcohol Abuse	17 (7%)	46 (12%)	49 (18%)	17 (5%)	33 (9%)	52 (14%)	82 (19%)
Alcohol Dependence	3 (1.2%)	28 (7%)	34 (13%)	15 (4%)	15 (4%)	37 (10%)	42 (10%)
DSM-III-R BN Symptoms	.08 (.44)	.13 (.62)	.27 (.89)	.14 (.67)	.10 (.62)	.15 (.66)	.11 (.52)
DSM-IV Diagnoses							
Bulimia Nervosa	5 (1.9%)	2 (0.5%)	4 (1.5%)	0	1 (0.3%)	2 (0.5%)	1 (0.2%)
Major Depressive D/O	--	--	--	--	23 (6%)	38 (10%)	70 (16%)
Alcohol Abuse	--	--	--	--	24 (9%)	51 (14%)	81 (19%)
Alcohol Dependence	--	--	--	--	11 (3%)	24 (7%)	28 (6%)
DSM-IV BN Symptoms	.10 (.53)	.17 (.81)	.34 (1.10)	.17 (.78)	.12 (.77)	.22 (.94)	.13 (.66)

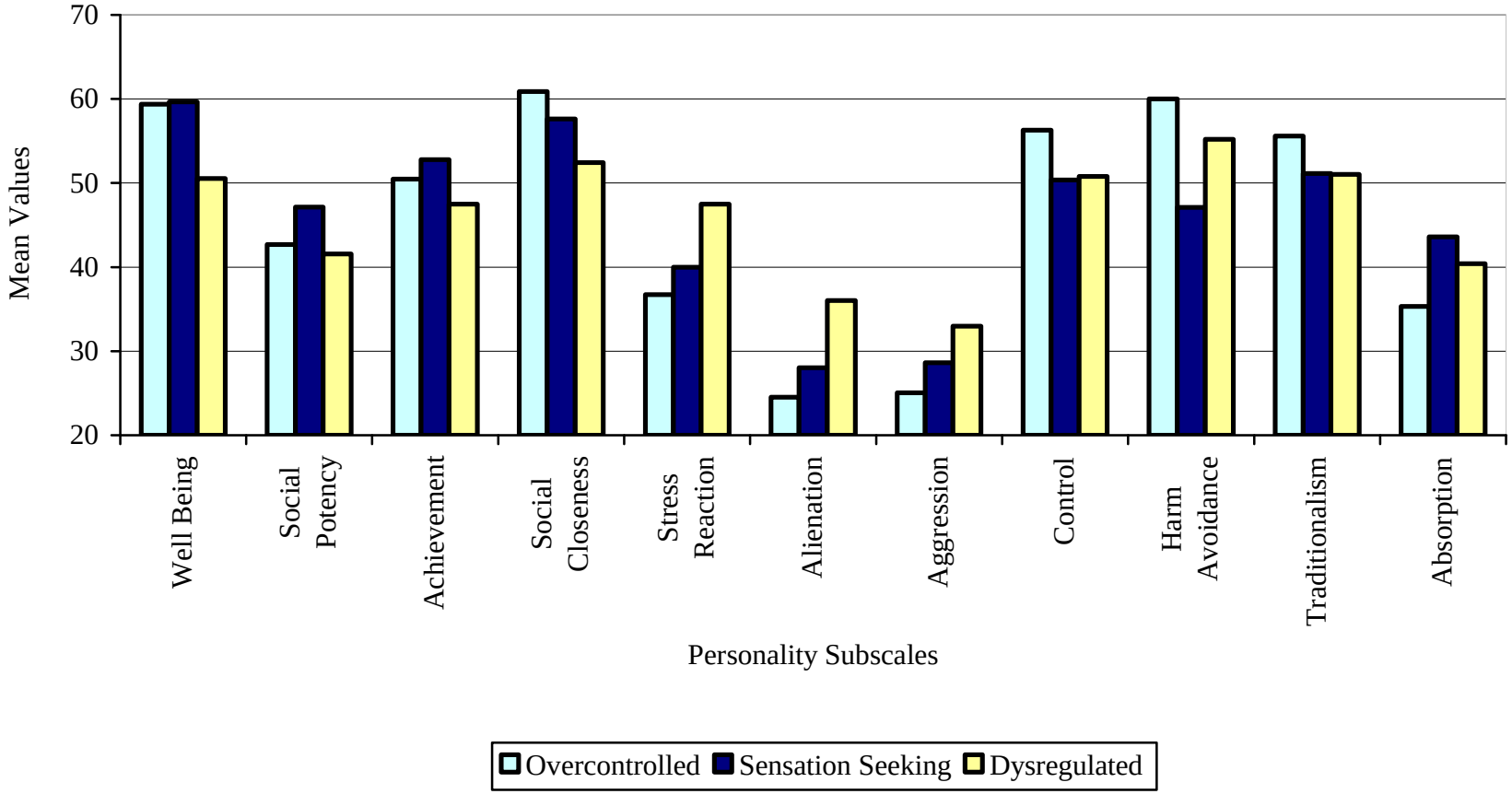
Note. D/O = Disorder; BN = Bulimia Nervosa. Percentages, shown in parentheses, indicate the number of individuals with probable and definite diagnoses divided by the total number of participants in each cluster. Standard deviations are indicated in parentheses for BN symptoms.

Figure 1. Means of MPQ Facet Scales Across Overcontrolled (*n* = 257), Sensation Seeking (*n* = 398), Dysregulated (*n* = 268), and Inhibited (*n* = 341) Clusters at 17-years-old.



Note. For interpretation of the references of color in this and all other figures, the reader is referred to the electronic version of this dissertation.

Figure 2. Means of MPQ Facet Scales Across Sensation Seeking (*n* = 372), Overcontrolled (*n* = 375), and Dysregulated (*n* = 437) Clusters at 25-years-old.



ABSTRACT

GENETIC AND ENVIRONMENTAL FACTORS UNDERLYING COMORBID BULIMIC BEHAVIORS AND ALCOHOL USE DISORDERS: A ROLE FOR PERSONALITY?

By

Jennifer Danielle Slane

Objective: Women with bulimia nervosa (BN) frequently have co-occurring alcohol use disorders (AUDs). Family and twin studies have been mixed as to whether there is shared genetic transmission of these disorders. Discrepant findings may be due to personality heterogeneity among individuals with BN. Cluster analytic studies have been used to characterize women with BN in groups based on personality profiles with the Dysregulated cluster emerging as a group that may be more closely linked etiologically to AUDs. The Dysregulated cluster is characterized largely by behavioral disinhibition and emotional dysregulation and has higher rates of AUDs compared to other clusters. The present study aimed to examine whether personality heterogeneity has contributed to mixed twin study findings by determining whether genetic associations between BN and AUDs are strongest among the Dysregulated cluster. **Method:** Participants included a longitudinal sample of female twins assessed at ages 17 and 25 from the Minnesota Twin Family Study. Symptoms of BN and AUDs were assessed using clinical interviews and self-report scales. Personality clusters were defined using scores on the Multidimensional Personality Questionnaire. **Results:** Twin moderation models suggested small-to-moderate common genetic transmission between bulimic behaviors and AUD symptoms. However, shared genetic effects did not differ by personality cluster. Nevertheless, findings did indicate in some models that genetic influences that are unique to bulimic behaviors are higher among the Dysregulated cluster compared to the Other clusters,

suggesting an influence of personality on bulimic behaviors alone, but not on their association with AUD symptoms. **Discussion:** Despite the presence of shared genetic transmission between BN and AUDs, cluster membership did not affect etiologic associations between the phenotypes. This suggests that although personality clusters may be associated with the etiology of BN, they are unlikely to account for associations between BN and AUDs and inconsistent findings in the literature regarding their shared etiology.

INTRODUCTION

Individuals with bulimia nervosa (BN) frequently have comorbid alcohol use disorders (AUDs; Dansky, Brewerton, & Kilpatrick, 2000; Holderness, Brooks-Gunn, & Warren, 1994; Wilson, 1991). Comorbidity rates in women with BN have ranged from 33-47% (Bulik, Sullivan, Carter, & Joyce, 1997; Mitchell, Hatsukami, Eckert, & Pyle, 1985), with an estimated median prevalence rate of 22.9% (Holderness et al., 1994). The co-occurrence of these disorders is associated with many maladaptive problems (Dansky et al., 2000; Duncan et al., 2006; Keel, Mitchell, Miller, Davis, & Crow, 1999). For example, women with comorbid BN and AUDs have higher rates of attempted suicide than women with BN or AUDs alone (Duncan et al., 2005, 2006). Additionally, women with co-occurring BN and AUDs are more likely to have additional diagnoses of drug dependence and major depressive disorder than women with BN or AUDs alone (Duncan et al., 2006).

Given the deleterious effects of the co-occurrence of BN and AUDs, several researchers have used twin and family studies to determine whether there is some shared etiology in the development of these disorders. Twin studies can help establish whether additive genetic (i.e., the effect of individual genes summed over loci that acts to increase twin similarity relative to the amount of genes shared), shared environmental (environmental influences common to siblings that acts to make them similar to each other), or nonshared environmental (environmental factors differentiating twins within a pair) factors contribute to this comorbidity. If there is a shared etiology between these disorders, this would inform future studies with regard to whether to examine potential common genetic (e.g., personality traits) or environmental (e.g., bullying) risk factors underlying the association between BN and AUDs.

Three family studies that have examined associations between BN and AUDs have found that these disorders co-occur in families (Bulik, 1987; Kassett et al., 1989; Lilenfeld et al., 1997). One study found higher rates of alcoholism among first-degree relatives of bulimic probands compared to healthy control participants (28% vs. 14%; Kassett et al., 1989). A second found alcoholism to be the most common disorder among first- and second-degree relatives of bulimic probands with and without comorbid AUDs compared to normal control participants (60% vs. 20%; Bulik, 1987). However, a third study had somewhat conflicting results, indicating that only family members of BN probands with comorbid AUDs had higher rates of both disorders compared to both BN probands without AUDs and controls (Lilenfeld et al., 1997). Familial transmission that occurs only among BN probands with AUDs does not necessarily imply co-aggregation of the disorders, as the disorders could simply be independently transmitted in families. Nonetheless, overall, these family studies provide important initial information suggesting familial co-aggregation of BN and AUDs.

Twin studies expand on family studies by providing estimates of genetic and environmental contributions to the covariance of phenotypes. That is, twin studies can indicate whether phenotypic and familial associations between disorders are due to a common set of genetic or environmental factors underlying risk for both phenotypes. Common genetic factors can be assessed via genetic correlations, which indicate the degree of overlap in genetic risk factors between phenotypes. Further, common shared environmental correlations indicate the magnitude of overlap in environmental effects that are shared by siblings and act to make them similar to each other. Finally, common nonshared environmental correlations indicate the degree to which the association between phenotypes is due to experiences that are not shared between siblings.

In order to determine whether genetic or environmental factors underlie the comorbidity of two disorders, it is important to first determine if they are heritable phenotypes. Genetic influences on BN and bulimic behaviors (i.e., binge eating, the use of compensatory behaviors, weight preoccupation) have ranged from 28-83% and from 41-70%, respectively, with the remaining variance due to nonshared environmental influences (Bulik, Sullivan, & Kendler, 1998; Bulik, Sullivan, & Kendler, 2003; Kendler et al., 1991, 1995; Klump, McGue, & Iacono, 2000; Kortegaard, Hoerder, Joergnesen, Gillberg, & Kyvik, 2001; Reichborn-Kjennerud et al., 2003; Reichborn-Kjennerud, Bulik, Tambs, & Harris, 2004; Rowe, Pickles, Simonoff, Bulik, & Silberg, 2002; Sullivan, Bulik, & Kendler, 1998; Wade et al., 1999; Walters et al., 1992). Large-scale twin studies of AUDs and AUD symptoms indicate heritabilities exceeding 60% and 51%, respectively, in women (Heath et al., 1997; Kendler, Neale, Heath, Kessler, & Eaves, 1992, 1994; Prescott, Aggen, & Kendler, 1999; Whitfield et al., 2004) with the remaining variance due to nonshared environmental influences.

To date, only four twin studies have examined associations between BN and AUDs to determine whether genetic and/or environmental influences underlie their comorbidity. Similar to family study findings, results have been mixed. In the first study, "problem drinking" (defined as "having had or having been considered by others as having a significant drinking problem that is not limited to single isolated incidents") and BN were found to load on two separate and independent genetic factors, suggesting distinct etiologies (Kendler et al., 1995). Further, findings indicated that shared and nonshared environmental influences were predominately disorder-specific, suggesting that there is no overlap in these factors. Mitchell et al. (2010) also found mixed evidence for shared genetic effects. Although a large genetic correlation (1.0; 95% confidence intervals [CIs]: -1.0, 1.0) and moderate nonshared environmental correlation (.53,

CIs: -.32, 1.0) was observed, both estimates overlapped with zero, indicating that they were not statistically significant effects.

By contrast, two studies suggested significant genetic overlap between BN and AUDs. Baker, Mitchell, Neale, and Kendler (2010) indicated a moderate genetic correlation between BN and AUDs (.53; CIs: .30, .80) and a small and nonsignificant nonshared environmental correlation between these phenotypes (-.03, CIs: -.24, .18). Slane, Burt, and Klump (in preparation) also found evidence for common genetic etiologies between two of the primary symptoms of BN (i.e., binge eating and compensatory behaviors) and alcohol use, with genetic correlations of .31 (CIs: .09, .53) for binge eating and .61 for compensatory behavior (CIs: .34, 1.0; Slane, Burt et al., in preparation). No overlap in shared or nonshared environmental factors were detected, as shared environmental factors were estimated close to zero and nonshared environmental influences were all disorder-specific.

Overall, results are split with two studies finding evidence for common genetic factors, and two others suggesting no shared etiology. It is unclear why results have been inconsistent. However, one hypothesis is that extant twin studies have not considered the influence of personality. Personality traits are heritable (heritability = ~50%; Bouchard & McGue, 1990; Heath, Cloninger, & Martin, 1994; Jang, Livesley, & Vernon, 1996; Jang, Livesley, Vernon, & Jackson, 1996; Klump, McGue, & Iacono, 2002; Koopmans, Boomsma, Heath, & van Doornen, 1995; Krueger, Caspi, & Moffitt, 2000) and have been studied extensively in BN with findings indicating that women with BN often show a heterogeneous mix of personality and temperamental traits (Vitousek & Manke, 1994; Westen, Thompson-Brenner, & Peart, 2006). This heterogeneity may reflect etiologic differences in the development of BN that results in distinct comorbidities (e.g., AUDs) and genetic risk profiles. Studies have begun to use a

personality profiling/cluster analytic approach to personality classification in women with BN in order to decrease heterogeneity and obtain more homogeneous groups that may reflect common etiologic factors.

Most cluster analytic studies of eating disorders have found three clusters of women that include a high-functioning/perfectionistic group (i.e., conscientious, perfectionistic, and anxious), a constricted/overcontrolled group (i.e., passive, avoidant, and emotionally constricted), and an emotionally dysregulated/undercontrolled group (i.e., emotionally intense and labile, impulsive, poor negative mood regulation, and behaviorally disinhibited; Westen & Harnden-Fischer, 2001). Among these clusters, the emotionally dysregulated/undercontrolled cluster, frequently labeled the "dysregulated" cluster (Espelage, Mazzeo, Sherman, & Thompson, 2002; Thompson-Brenner & Westen, 2005; Thompson-Brenner, Eddy, Satir, Boisseau, & Westen, 2008; Westen & Harnden-Fischer, 2001), is the most stable cluster across time (Slane, Donnellan, Klump, McGue, & Iacono, in preparation) and AUDs are more common in this group compared to other clusters (Claes et al., 2006; Thompson-Brenner & Westen, 2005; Wonderlich et al, 2005). Further, the dysregulated cluster tends to consist primarily of women who exhibit bulimic behaviors (e.g., bingeing and purging) rather than women who restrict their intake only (Claes et al., 2006; Perkins, Slane, & Klump, in preparation, Thompson-Brenner et al., 2008, Westen and Harnden-Fischer, 2001). The frequent co-occurrence of BN symptoms and AUDs in the dysregulated group suggests that this may be an ideal group to examine shared etiology between the disorders. Indeed, this group could represent a unique subtype among individuals with BN that is more closely linked etiologically to AUDs.

The overall aim of the present study was to examine whether etiologic associations between BN and AUD symptoms are strongest among individuals in the dysregulated cluster as

compared to individuals from other personality clusters. Longitudinal data from the Minnesota Twin Family Study that was previously cluster analyzed at two time points (i.e., at ages 17 and 25; Slane, Donnellan et al., in preparation) was utilized in the present study. The use of longitudinal data allowed for the examination of cross-sectional (e.g., age 17 BN with age 17 AUD) as well as longitudinal phenotypic and genetic associations between the phenotypes. Longitudinal models examined associations between age 17 BN and age 25 AUD symptoms. Given that BN typically precedes AUDs (American Psychiatric Association, 2000), temporally it makes sense to examine age 17 bulimic behaviors and age 25 AUD symptoms. It must be noted, however, that the present study hypothesizes a shared etiology, not a causal relationship, between these two disorders. Although the longitudinal aspect of these data allows for the determination of whether one of these disorders is more predictive of the other, the main goal is to examine whether common genetic or environmental influences underlie their association.

METHODS

Participants

Participants included a convenience sample of female twins from the Minnesota Twin Family Study (MTFS). The MTFS is a population-based, longitudinal study of reared-together same-sex female twins and their parents. A detailed description of study recruitment and assessments can be found elsewhere (Iacono, Carlson, Taylor, Elkins, & McGue, 1999). Briefly, public databases were utilized to obtain birth records used to identify twins born in the state of Minnesota. Over 90% of twins born between 1971 and 1985 were located.

The current study utilized cross-sectional and longitudinal data from two cohorts of MTFS female twins. Cohort 1 began the study when they were 11 years old, whereas cohort 2 began the study at age 17. Both cohorts were assessed at ages 17 (M = 17.87; SD = 0.74) and 25

(M = 25.04; SD = 0.69) years. The present study used data from both cohorts at ages 17 and 25. Overall sample sizes across the age groups were 1,264 twins at age 17 and 1,184 twins at age 25.

Zygosity Determination

Zygosity was determined using three separate methods (Iacono, Malone, & McGue, 2003). First, the twins' parents completed a physical similarity questionnaire that has been shown in previous research to be over 95% accurate in diagnosing twin zygosity (Plomin, DeFries, McClearn, & McGuffin, 2008). Second, research assistants, trained on methods for determining zygosity, evaluated the similarity of twins' eye color, hair color, ear shape, and overall physical characteristics. Third, each twin's ponderal index (i.e., a measure comparable to body mass index), cephalic index (i.e., a measure of head shape), and number of fingerprint ridges were utilized to determine zygosity using an algorithm. These three methods disagreed in their zygosity classification in 33.4% (214/641 twin pairs) of twin pairs. Disagreements among the three methods were resolved using serological analysis of 12 genetic polymorphisms. Importantly, serological analysis also confirmed the validity of the primary three zygosity methods in 50 twin pairs (McGue, Elkins, & Iacono, 2000).

Measures

Bulimia Nervosa Symptoms

Symptom counts of bulimia nervosa (BN) were assessed using the Eating Disorders Structured Clinical Interview (EDSCI) and the Minnesota Eating Behavior Survey (MEBS; von Ranson, Klump, Iacono, & McGue, 2005)[4]. The EDSCI and MEBS were administered at both time points.

[4] 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,

The EDSCI is a semi-structured interview based on Module H of the Structured Clinical Interview for the Diagnostic and Statistical Manual of Mental Disorders (DSM) Axis I Disorders (SCID; Spitzer, Williams, & Gibbon, 1987). The BN symptoms that were assessed include binge eating, loss of control over binge eating, purging (i.e., vomiting, laxatives, diuretics, diet pills, ipecac) and nonpurging (i.e., excessive exercise, such as running for two hours after a subjective or objective binge episode, fasting, strict dieting) behaviors, and the undue influence of shape and weight on self-evaluation. Due to the relatively low prevalence of BN diagnoses (Hudson, Hiripi, Harrison, & Kessler, 2007), the EDSCI BN symptom counts were used in analyses instead of diagnoses. These symptoms counts allow for the examination of clinical levels of symptomatology without having reduced power due to a small number of full diagnoses. Several studies have utilized subclinical measures of BN symptoms (Klump et al., 2000; Rowe et al., 2002; Sullivan et al., 1998) supporting the idea of a multiple threshold model of BN where both broad and narrow definitions of BN appear to result from a similar underlying vulnerability (Kendler et al., 1991).

"Best estimates" of lifetime BN symptoms were assessed at age 17. That is, at this age, both the twin's mother and the twin herself reported on all BN symptoms, and best estimates coded a symptom as present if either the mother or twin indicated that it was present. At age 25, best estimates could not be used since the twin's mother did not report on symptoms at this time point. Thus, symptoms at age 25 were based on twin report only and include symptoms present over the earlier three to four years. Importantly, reliability of BN symptoms was good with kappa values ranging from .64 to 1.00.

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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.

The MEBS was also used to assess bulimic behaviors. This 30-item true/false self-report questionnaire assesses overall levels of eating pathology as well as specific disordered eating symptoms including body dissatisfaction (i.e., dissatisfaction with one's size or shape), binge eating (i.e., thoughts about overeating or the tendency to binge eat), compensatory behaviors (i.e., the use of compensatory behaviors such as self-induced vomiting, diuretics, etc. for weight loss), and weight preoccupation (i.e., preoccupation with dieting, thinness, and weight). Examining these continuous measures of bulimic behaviors allowed for a more fine-tuned analysis of BN and its component symptoms. For example, examining scores on these measures allows for the examination of associations with specific symptoms of BN (e.g., binge eating) in addition to the full BN symptom count.

The internal consistency of the MEBS subscales has been shown to be adequate in samples of females ages 17 (α = .65-.89) and 20 years old (α = .68-.89; von Ranson et al., 2005). The MEBS demonstrated sufficient discriminant validity through the ability to differentiate between normal control participants and individuals with eating disorders (von Ranson et al., 2005). Concurrent validity was also demonstrated through significant correlations (r = .68 - .72) between the MEBS subscales (i.e., Weight Preoccupation and Body Dissatisfaction) and similar subscales (i.e., Shape concerns and Weight Concerns subscales) from the Eating Disorders Examination Questionnaire (von Ranson et al., 2005). Notably, all subscales demonstrated sufficient discriminant validity through their ability to differentiate between normal control participants and individuals with eating disorders, including BN (von Ranson et al., 2005).

AUD Symptoms

Alcohol abuse and dependence symptoms (i.e., alcohol use disorder [AUD] symptoms) were assessed at both ages using the Substance Abuse Module (SAM) from the Composite

International Diagnostic Interview (Robins, Babor, & Cottler, 1987). These include symptoms such as recurrent alcohol-related legal problems, tolerance, and withdrawal. The SAM is a well-established, semi-structured interview measure of these symptoms that has been used in field trials for the development of the DSM (Cottler et al, 1995; Spitzer et al., 1987). The SAM shows excellent inter-rater reliability (Cottler, Robins, & Hezler, 1989), with an average kappa reliability of 0.92 for individual alcohol abuse and dependence symptoms. For the purposes of this study, an AUD composite score was used in analyses. This composite score is a sum of all alcohol abuse and alcohol dependence symptoms. Combining these symptoms not only increases the variability within the sample, but also more closely resembles proposed changes to the AUD diagnosis in DSM-V, where alcohol abuse and dependence will be combined (American Psychiatric Association, 2010). Importantly, similar to BN, previous research supports the idea of a multiple threshold model of alcohol use (Heath et al., 1997; Kendler et al., 1992; Kendler et al., 1994), suggesting that findings for these disorders measured continuously are similar to those for full diagnoses.

Notably, only DSM-III-R data were available for both cohorts at age 17. However, at age 25, both DSM-III-R and DSM-IV symptoms were assessed. Analyses were conducted with DSM-III-R data at age 17 and DSM-IV data at age 25.

Procedures for Diagnostic Interviews

DSM AUD and BN symptoms were assessed by trained bachelor's and master's level. Each symptom assigned was discussed in a clinical case conference with at least two advanced clinical psychology doctoral students. Discrepancies that emerged during the case conference were clarified by listening to interview audiotapes or re-contacting study participants.

Symptoms were counted as present if they qualified for clinical significance in both frequency and severity.

Personality Clusters

The present study utilized clusters that were previously identified using the facet scales of the Multidimensional Personality Questionnaire assessed longitudinally at two time points (i.e., ages 17 and 25; Slane et al., in preparation). Data were from the Minnesota Twin Family Study and four clusters were identified at age 17, whereas three were identified at age 25. These clusters included the Resilient, Sensation Seeking, and Dysregulated clusters at both time points, and an additional Inhibited cluster at age 17. As stated previously, the Dysregulated cluster is characterized as interpersonally aggressive, emotionally labile, and impulsive. The Resilient cluster tends to be cautious, have high moral standards, and value social closeness. The Sensation Seeking cluster is characterized as decisive, persuasive, with a preference for novel, risky activities. Lastly, women in the Inhibited cluster tend to opt for safe activities over risky ones, prefer being alone and not being the center of attention, and are not very imaginative.

Statistical Analyses

Data Preparation and Descriptive Statistics

Prior to analyses, log transformations ($\log_{10} X + 1$) were performed for the Binge Eating scale, Compensatory Behaviors scale, DSM BN Symptoms, and the Alcohol Composite (i.e., AUD symptoms) to account for positive skew.

Phenotypic Associations between Bulimic Behaviors and Alcohol Use Disorder Symptoms by Cluster Membership

In order to examine initial phenotypic associations, cross-sectional Pearson correlations were calculated within cluster at each age between bulimic behaviors and AUD symptoms. Note that moderation by the Dysregulated cluster is the primary focus of the present study, as this cluster has increased levels of bulimic behaviors and AUD symptoms compared to the other clusters. Given this focus and the fact that the present study did not have specific hypotheses about the remaining clusters, the age 17 Resilient, Sensation Seeking, and Inhibited clusters and the age 25 Resilient and Sensation seeking clusters were collapsed at each time point into one cluster labeled "Other clusters" as a comparison group for the Dysregulated cluster. For correlations examining longitudinal associations, the age 25 rather than age 17 clusters were used as the grouping variable for both the age 17 and age 25 correlations since personality traits tend to be more stable in adulthood than adolescence (McGue, Bacon, & Lykken, 1993). These correlations examined associations between age 17 bulimic behaviors and age 25 AUD symptoms. Notably, however, results were nearly identical for longitudinal associations using the age 17 clusters (i.e., Dysregulated vs. Other clusters; data not shown).

Genetic and Environmental Associations between Bulimic Behaviors and Alcohol Use Disorder Symptoms by Cluster Membership

In order to examine the influence of a moderator variable (i.e., cluster membership; Dysregulated vs. Other clusters) on genetic and environmental influences on *associations* between BN and AUD symptoms, a gene-environment interaction (i.e., GxE) model was examined. Typically, GxE models are used to examine the effects of a moderator (e.g., stressful life events) on genetic and environmental influences on one trait or disorder (e.g., depression). However, a modification of this model, the GxE in the presence of r_{ge} model, can be used to examine the extent to which a moderator influences the degree of genetic and/or environmental

associations between two phenotypes (i.e., BN and AUD). In the current study, this modified model can determine whether there are differences in the extent to which genetic (and environmental) factors influence the covariation between BN and AUD across levels of the moderator (i.e., Dysregulated vs. Other clusters).

The altered GxE in the presence of r_{ge} moderator model, used in the present study, is shown in Figure 7. In this figure, there are three estimates of genetic influences. First, there is an estimate of the genetic effects specific to AUD symptoms (i.e., specific to the first phenotype in the model; a_p). Second, there are genetic factors unique to BN symptoms (i.e., specific to the second phenotype in the model, after accounting for the overlap with AUD symptoms; a_u). Third, there are genetic influences (A) common to both phenotypes (a_c; i.e., overlap in genetic effects influencing BN and AUD symptoms). It is the genetic (and environmental) influences on this covariance path and their potential moderation by personality cluster that is of key interest in the present study. Shared (C) and nonshared (E) environmental influences that are unique to the traits and common to both are also estimated, but are not included in Figure 7, due to space constraints.

Moderation effects (i.e., β_{xc} and β_{xu}) are also estimated in the model. The first β coefficient (β_{xc}) represent differences (either positive or negative) in genetic or environmental influences on the covariance between BN and AUD symptoms based on the effect of the moderator (i.e., cluster membership). The β_{xc} moderator coefficient is of particular interest in the present study as it addresses the primary aim indicating whether genetic influences on the covariance between bulimic behaviors and AUD symptoms are strongest in the Dysregulated

cluster compared to the Other clusters. Specifically, a positive and significant estimate indicates increased influence of genetic effects in the Dysregulated cluster compared to the Other clusters. By contrast, a nonsignificant estimate indicates that there is no change in genetic effects by cluster and a negative and significant estimate indicates that the genetic factors are lower in the Dysregulated cluster compared to the Other clusters.

The second β coefficient (β_{xu}) indicates whether there are changes in genetic effects that are unique to bulimic behaviors based on changes in cluster membership. It may be that there are no changes in genetic effects on the *covariance* between bulimic behaviors and AUD symptoms with different levels of cluster membership, but that there is a moderator effect on genetic influences specific to bulimic behaviors. For example, if this coefficient is significant and positive, it would indicate that genetic factors on bulimic behaviors are greater in the Dysregulated cluster compared to the Other clusters. Thus, this would suggest that women in the Dysregulated cluster have an increased genetic risk for developing bulimic behaviors. As specified in this study, these models do not provide unique estimates of genetic factors (or changes in these effects) on AUD symptoms. Reverse models, with the phenotypes entered in the opposite order, could be examined to determine the unique impact of the moderator on AUD symptoms; however, given that the present study does not have hypotheses about the influence of cluster membership on unique effects on AUD symptoms, these reverse models were not examined. It must be noted that the longitudinal models did provide this estimate, as age 17 bulimic behaviors were entered into the model first to make conceptual sense with regard to temporal order. Therefore, unique effects of the moderator on age 25 AUD symptoms were included in these models.

Three groups of models were examined in the present study: 1) cross-sectional models with age 17 cluster membership, age 17 bulimic behaviors, and age 17 AUD symptoms, 2) cross-sectional models with age 25 cluster membership, age 25 bulimic behaviors, and age 25 AUD symptoms and 3) longitudinal models that included age 25 cluster membership, age 17 bulimic behaviors and age 25 AUD symptoms. As stated previously, age 25 cluster membership was utilized in the longitudinal analyses, instead of age 17 cluster membership, as personality traits tend to be more stable in adulthood than in adolescence (McGue et al., 1993). Further, given that the age of onset for BN is typically earlier than AUDs (American Psychiatric Association, 2000), it made sense temporally to examine age 17 bulimic behaviors and age 25 AUD symptoms.

Each of the modified GxE in the presence of r_{ge} moderator models (Purcell, 2002) were fit to the raw data using full-information maximum-likelihood in Mx (Neale, 1997). Initially, a full ACE model with main and moderation effects was fit to the data. Next, a full AE submodel (with main and moderation effects) of the ACE model was examined to determine if a reduced model provides a better fit to the data given that BN and AUD symptoms are influenced primarily by genetic (A) and nonshared environmental (E) factors in late adolescence and adulthood (Bulik et al., 1998; Heath et al., 1997; Kendler et al., 1991, 1992, 1994, 1995; Kortegaard et al., 2001; Prescott et al., 1999; Rowe et al., 2002; Wade et al., 1999; Walters et al., 1992; Whitfield et al., 2004). Additional submodels (dropping the moderation coefficients) were not examined, as the remaining sources of variance would have absorbed the dropped effects and confidence intervals for other estimates in the model would be artificially narrowed (Sullivan & Eaves, 2002). Although dropping the shared environmental effects (C) can also have this influence, the AE model with full moderation was examined in the present study due to

consistent extant research indicating little to no influence of these effects on BN or AUDs in adulthood.

The fit of all models was examined using a likelihood-ratio chi-square goodness of fit (X^2) and Akaike's Information Criteria (AIC = $\chi - 2df$; Akaike, 1987). The likelihood-ratio chi-square goodness of fit (X^2) examines differences in -2lnL values and degrees of freedom between the AE submodel and the full ACE model. If the chi-square goodness of fit is non-significant, then the most parsimonious (i.e., the model containing fewer estimated parameters, and hence more degrees of freedom) model is preferred. In addition to this test, the AIC is utilized to compare model fit, with the lowest AICs indicating better fit.

Prior to conducting model fit analyses, some data preparation was necessary. First, the variables were standardized (i.e., z transformed) prior to analysis. This transformation has been conducted in previous research (Burt & Klump, 2009; Klump, Perkins, Burt, McGue, & Iacono, 2007) as it aids in interpretation of moderation effects. Second, the dichotomous cluster scores were floored at zero, otherwise, if this adjustment is not made prior to the analyses, the Mx program will arbitrarily code a group as zero, which may or may not correspond to the hypothesized moderator order.

In all tables and figures, unstandardized parameter estimates from the models are reported because standardized estimates can be misleading with regard to changes in genetic and environmental effects across levels of the moderator. For example, if the moderator causes an increase in genetic effects at different levels of the moderator, but the environmental effects stay constant, standardization of parameter estimates would make it appear as though environmental effects are decreasing as genetic effects are increasing because the standardization requires that the estimates are proportional (i.e., A, C, and E estimates add up to 1.0). Thus, the

unstandardized estimates allow for a straightforward indication of absolute changes in the genetic and environmental effects with changes in the moderator.

RESULTS

Descriptive Statistics

Means and standard deviations for the bulimic behaviors and AUD symptoms within the Dysregulated cluster and Other clusters at both time points are presented in Table 9. Importantly, at both time points, there was sufficient variability in the score ranges as well as the percent of participants scoring above the clinical cut-offs for eating disorders (i.e., mean scores among individuals with eating disorders) on the MEBS scale (age 17: 5-28%; age 25: 5-35%; see von Ranson et al., 2005).

The Dysregulated cluster had the highest levels of bulimic behaviors and AUD symptoms at both time points, with one exception. At age 25, DSM BN symptoms were higher among the Other clusters compared to the Dysregulated group.

Phenotypic Associations between Bulimic Behaviors and Alcohol Use Disorder Symptoms by Cluster Membership

Pearson correlations were calculated to examine phenotypic associations between bulimic behaviors and AUD symptoms within the Dysregulated cluster versus the Other clusters (see Table 10) cross-sectionally, at both time points, as well as longitudinally. Cross-sectionally, at age 17, nearly all of the bulimic behaviors (with the exception of weight preoccupation) were significantly and positively associated with AUD symptoms in the Dysregulated cluster (r's = .16-.29). Surprisingly, nearly all of the bulimic behaviors also were significantly and positively

associated with AUD symptoms in the Other clusters, although correlations were comparatively smaller (r's = .12-.15).

In contrast to age 17 correlations, cross-sectionally, at age 25, the only significant correlation in the Dysregulated cluster was between DSM BN symptoms and AUD symptoms (r = .15). In the Other clusters, small associations were indicated between each of the bulimic behaviors and AUD symptoms (r's = .08-.15), with the exceptions of body dissatisfaction and weight preoccupation. Hypotheses that there would be a stronger association between bulimic behaviors and AUD symptoms in the Dysregulated cluster compared to the Other clusters was not confirmed; however, this may be due, in part, to smaller sample sizes in the age 25 Dysregulated group. For example, some correlations in the Dysregulated group are of the same magnitude as those in the Other cluster group (e.g., binge eating r = .11), but the correlations are not significant.

The longitudinal associations between age 17 bulimic behaviors and age 25 AUD symptoms were somewhat similar to the age 25 cross-sectional correlations. In the Dysregulated cluster, the only age 17 bulimic behavior that was associated with age 25 AUD symptoms was compensatory behavior (r = .17). However, all of the associations were significant in the Other clusters (r's = .15-.24), with the exception of DSM BN symptoms.[5]

Genetic and Environmental Associations between Bulimic Behaviors and Alcohol Use Disorder Symptoms by Cluster Membership

[5] Although BN typically precedes AUDs, longitudinal correlations with age 17 AUD symptoms and age 25 bulimic behaviors were examined within the Dysregulated cluster and other clusters. Within both groups, there was only one significant correlation (binge eating in the other cluster group r = .09; data not shown). This provided further support for examining the association between these phenotypes in temporal order.

The Pearson correlations described above were also used to inform the twin analyses. Specifically, twin analyses were only conducted if there were significant associations between bulimic behaviors and AUD symptoms among either the Dysregulated or Other clusters. If associations were not significant across either cluster, then twin analyses were not examined for that behavior. Note that clusters were coded dichotomously as "Other clusters" (coded 0) and Dysregulated cluster (coded 1) in all of the models.[6]

Moderator Models

Fit statistics and unstandardized estimates from the cross-sectional and longitudinal moderator models are included in Table 11. Initially, a full ACE GxE in the presence of r_{ge} moderator model was examined to determine whether cluster membership moderates the genetic and environmental influences underlying the association between bulimic behaviors (e.g., binge eating) and AUD symptoms. Subsequently, an AE moderator submodel was examined (with C estimates constrained to zero). In all cases, the AE model resulted in a superior fit compared to the ACE fully uncontrained model as indicated by lower AICs and a nonsignificant change in chi-square.

Table 12 includes the unstandardized estimates of genetic and nonshared environmental variance that are unique to AUD symptoms (a_P and e_P), common to both bulimic behaviors and AUD symptoms (a_C and e_C), and unique to bulimic behaviors (a_U and e_U; after accounting for the overlap with AUD symptoms) for the AE models. In addition, the moderation effects (i.e., βa_{XC}, βa_{XU}, βe_{XC}, and βe_{XU}) are included in the table, which represent changes in the common and

[6] Twin moderation models were also examined with all clusters included and ordered by mean levels of AUD symptoms and bulimic behaviors at each time point, from lowest levels (coded 0) to the highest levels (coded 3 at age 17 and 2 at age 25; data not shown). Notably results were nearly the same for this examination including all clusters.

unique genetic and nonshared environmental variance on the covariance between bulimic behaviors and AUD symptoms and the variance in bulimic behaviors with different levels of the moderator (i.e., cluster membership). It is important to note that confidence intervals for some parameter estimates overlapped with zero (see Table 12); thus, the point estimates for these parameters are nonsignificant and not meaningful. As stated previously, submodels were not fit in these cases, as the remaining sources of variance would have absorbed the dropped effects and confidence intervals for other estimates in the model would be artificially narrowed (Sullivan & Eaves, 2002). Instead, we kept the nonsignificant parameters in the model, and indicated significant parameter estimates via boded text in the tables. Figures 3-6 include unstandardized estimates of genetic and nonshared environmental variances for models with significant moderation coefficients.

Age 17 Models

Both bulimic behaviors and AUD symptoms were heritable in all models. Among the bulimic behaviors examined, heritability estimates were higher for body dissatisfaction and MEBS total score (i.e., 55% and 51%, respectively) compared to binge eating, compensatory behavior and DSM BN symptoms (i.e., 37%, 22%, and 20%). In all models, the heritability estimate for AUD symptoms was 57%. The remaining variance for each of these phenotypes was due to nonshared environmental effects.

With the exception of associations with DSM BN symptoms, all of the moderator models indicated that there was small-to-moderate common genetic overlap (a_C = .24-.27), but no common nonshared environmental overlap (e_C = -.03-.06) between the continuous measures of bulimic behaviors (i.e., MEBS total score, body dissatisfaction, binge eating, compensatory behaviors) and AUD symptoms. Although these estimates provide information about the overlap

in genetic effects underlying the association between bulimic behaviors and AUD symptoms, the a_C estimates are not directly comparable to genetic correlations (used in three of the extant studies examining these associations, Baker et al., 2010; Mitchell et al., 2010; Slane, Burt et al., in preparation). Therefore, these estimates were computed (data not shown) for ease of comparison between the current study and previous studies that used genetic correlations to indicate associations. Age 17 cross-sectional genetic correlations ranged from .17-.27, indicating that between 3 and 7% of the variance in the association between bulimic behaviors and AUD symptoms is accounted for by common genetic factors.

Despite this genetic overlap, the estimates of interest (i.e., common genetic [βa_{XC}] and common nonshared environmental [βe_{XC}] moderation effects) were all small and nonsignificant (i.e., confidence intervals overlapped with zero), suggesting that the magnitude of the genetic and nonshared environmental effects does not differ by cluster membership. Taken together, these findings strongly suggest that the factors that underlie the association between bulimic behaviors and AUD symptoms do not differ across Dysregulated versus Other clusters.

In general, unique genetic (β_{xu}) and nonshared environmental ($\beta e_{xu)}$ moderation effects on bulimic behaviors alone also were not significant, with a few exceptions. The unique genetic moderation effect was significant for compensatory behaviors (β_{xu} = .63), suggesting that genetic influences on this phenotype are strongest in the Dysregulated cluster. In order to interpret this level of increase, the genetic effects were standardized to show differences in heritability across the clusters. These standardized estimates indicate that the genetic influences on compensatory behaviors increase from 11% to 51% moving from the Other clusters to the Dysregulated cluster. Further, the unique nonshared environmental moderation coefficient was

significant for MEBS total score and DSM BN symptoms, suggesting that there are small-to-moderate changes (βe_{xu} = .28 and .57, respectively; see Figures 3 & 4) in nonshared environmental influences on these phenotypes with levels of the moderator. Standardized estimates indicated that the nonshared environmental effects on these bulimic behaviors increase in magnitude across the Other clusters to the Dysregulated cluster from 40% to 48% for MEBS total score and from 50% to 82% for DSM BN symptoms.

Age 25 Models

At age 25, heritability estimates for bulimic behaviors were similar, but somewhat larger than at age 17 with estimates ranging from 27-62%. Specifically, estimates were 62% for MEBS total score, 46% for binge eating, 35% for compensatory behavior, and 27% for DSM BN symptoms. Heritability estimates for AUD symptoms was lower at age 25 (38%) as compared to age 17 (i.e., 57%). As before, the remaining variance for all phenotypes was accounted for by nonshared environmental effects.

In contrast to age 17 findings, there were no significant estimates of common genetic factors underlying bulimic behaviors and AUD symptoms at age 25. However, common nonshared environmental factors were indicated for associations between binge eating and AUD symptoms (e_C = .20). Age 25 cross-sectional genetic correlations ranged from .05-.14; however, these were also all nonsignificant, suggesting that none of the variance in the association between bulimic behaviors and AUD symptoms is due to common genetic effects.

Similar to the age 17 findings, the common genetic (βa_{xC}) and common nonshared environmental (βe_{xC}) moderation estimates of interest were all small and nonsignificant, indicating that personality cluster membership does not moderate common genetic or

environmental associations between these phenotypes cross-sectionally at age 25. And most of the unique genetic and nonshared environmental moderation effects on bulimic behaviors were not significant although as before, there were some exceptions. There were no significant unique genetic moderation coefficients for bulimic behaviors at age 25. However, there were significant unique nonshared environmental moderation effects on compensatory behavior (βe_{xu} = .40) and DSM BN symptoms (βe_{xu} = .16) were indicated (see Figures 5 & 6), suggesting increases in the unique nonshared environmental variance on this phenotype across cluster. This suggests that the unique nonshared environmental effects on these bulimic behaviors increase in magnitude across the Other clusters to the Dysregulated cluster from 40% to 56% for compensatory behavior and from 64% to 67% for DSM BN symptoms.

Longitudinal Models

Longitudinal models examined the influence of age 25 cluster membership on the association between age 17 bulimic behaviors and age 25 AUD symptoms.

There were a similar number of significant common genetic and nonshared environmental effects between bulimic behaviors and AUD symptoms in the longitudinal models. Significant common genetic factors were indicated for associations between age 25 AUD symptoms and age 17 MEBS total score (a_C = .17), age 17 body dissatisfaction (a_C = .16) and age 17 compensatory behavior (a_C = .36). Common nonshared environmental effects were only indicated for associations between age 17 binge eating and age 25 AUD symptoms (e_C = .17).

Similar to the cross-sectional findings, the common genetic (βa_{xc}) and common nonshared environmental (βe_{xc}) moderation estimates were all small and nonsignificant in the longitudinal models. Again, this indicates that counter to hypotheses, personality cluster memberships does not moderate the common genetic or environmental association between bulimic behaviors and AUD symptoms. Due to the ordering of these models (i.e., with age 17 bulimic behaviors entered first in the model), unique effects on bulimic behaviors were not specified in the model and thus, they are not discussed herein. Reverse models would provide these estimates; however, the models would be nonsensical temporally as age 25 AUD symptoms would be predicting age 17 bulimic behaviors.

DISCUSSION

The present study is the first to examine whether genetic factors underlying the association between bulimic behaviors and AUD symptoms are strongest in the Dysregulated cluster. Twin model findings indicated that although common genetic factors underlie some associations between bulimic behaviors and AUD symptoms, these common genetic effects did not differ between the Dysregulated cluster and Other clusters. Therefore, personality heterogeneity may not be what is accounting for discrepancies in twin studies examining factors underlying associations between BN and AUDs.

Phenotypic correlations between bulimic behaviors and AUD symptoms generally followed expectations at age 17, with significant findings for nearly all associations in the Dysregulated group. Associations were also significant in the Other clusters, although of smaller magnitude than those in the Dysregulated group. In contrast, the age 25 phenotypic correlations did not follow expectations. It was hypothesized that the Dysregulated cluster would have the strongest associations between bulimic behaviors and AUD symptoms; yet, there was only one

significant correlation between these phenotypes in the Dysregulated cluster. However, similar to age 17 correlations, there were several significant associations indicated in the Other clusters. As stated previously, this may have been due to sample size differences. That is, in some cases, the correlations were of similar magnitude in the "Other clusters" group compared to the Dysregulated cluster (e.g., MEBS total score, binge eating, compensatory behavior). However, they were significant only for "Other clusters", potentially due to the fact that the "Other clusters" group had a much higher sample size than the Dysregulated cluster (i.e., n range = 313-432 in the Other cluster group compared to 577-742 in the Dysregulated group).

Nonetheless, in general, phenotypic correlations between bulimic behaviors and AUD symptoms were smaller in magnitude at age 25 than age 17 in both clusters. Reasons for this are unclear. Findings at age 17 corroborated previous research that showed significant associations between these behaviors in a population-based sample of adolescents (Timmerman, Wells, & Chen, 1990). However, the lack of associations between several of the bulimic behaviors and AUD symptoms at age 25 was surprising given the frequent comorbidity between BN and AUDs, even in young adulthood (Krahn, Kurth, Demitrack, & Drewnowski, 1992). Notably, associations between the DSM symptom counts of AUD and BN symptoms were significant in both cluster groups at age 25. This suggests that in adults, more severe, diagnostic levels of these disorders are associated, but that continuous measures of bulimic behaviors (which are more prevalent across the sample) are not associated with AUDs. Thus, future research examining these associations in adults may want to focus on clinical samples with diagnoses of these disorders.

Longitudinally, Pearson correlations also did not follow expectations. The only significant correlation between age 17 bulimic behaviors and age 25 AUD symptoms in the

Dysregulated cluster was between age 17 compensatory behavior and age 25 AUD symptoms. Yet, all of the associations were significant in the Other clusters group, with the exception of DSM BN symptoms. It is unclear why the longitudinal findings did not follow hypotheses. As stated previously, although bulimic behaviors often precede AUDs, they are not typically seen as risk factors for AUDs. Therefore, it may be that even though there are higher rates of these symptoms and behaviors in the Dysregulated cluster, they are not associated longitudinally. Further, the unexpected significant longitudinal findings in the Other clusters group, may have been due to sample size differences between the clusters. That is, the larger sample size of the Other clusters group compared to the Dysregulated group may have contributed to finding significant associations between these phenotypes in that cluster.

Heritability estimates of AUD symptoms and bulimic behaviors roughly followed expectations from previous studies. Specifically, in the present study, AUD symptom heritabilities ranged from 38 to 57%. Previous studies have indicated heritability estimates of approximately 60% for AUDs and 51% for AUD symptoms in women (Heath et al., 1997; Kendler et al., 1992, 1994; Prescott et al., 1999; Whitfield et al., 2004) with the remaining variance due to nonshared environmental influences. For bulimic behaviors, the majority of heritability estimates (i.e., 20-55% at age 17 and 27-62% at age 25) fell within the range identified in extant research. As stated previously, many studies have indicated that genetic influences on BN and bulimic behaviors range from 28-83% and from 41-70%, respectively, with the remaining variance due to nonshared environmental influences (Bulik et al., 1998, 2003; Kendler et al., 1991, 1995; Klump et al., 2000; Kortegaard et al., 2001; Reichborn-Kjennerud et al., 2003, 2004; Rowe et al., 2002; Sullivan et al., 1998; Wade et al., 1999; Walters et al., 1992). Heritability estimates in the present study corroborate findings in extant research suggesting that

bulimic behaviors are heritable, but the magnitude differs by the symptom examined (Bulik, Sullivan, Wade, & Kendler, 2000). Indeed, previous research has indicated heritabilities are typically lower for bulimic behaviors such as binge eating, compensatory behaviors and BN symptoms (Sullivan et al., 1998). For measures of compensatory behaviors and BN symptoms, these lower estimates are often attributed to lower item endorsement (i.e., lower variability; Bulik et al., 2000), whereas for binge eating, lower heritabilities are sometimes associated with methodological issues (Field, Taylor, Celio, & Colditz, 2004). Specifically, self-report assessments of binge eating can often overestimate rates of this behavior (Field et al., 2004), thereby increasing measurement error (which is included in nonshared environmental [E] estimates).

Several of the twin models, particularly cross-sectionally at age 17, indicated that common genetic effects underlie associations between bulimic behaviors and AUD symptoms (see a_C parameter estimates in Table 12). This supports findings from two studies (Baker et al., 2010; Slane, Burt et al., in preparation), suggesting that there are common genetic influences underlying this covariance. Estimates across studies have been fairly similar in magnitude. That is, previous studies indicated significant genetic correlations between bulimic behaviors and AUD symptoms ranging from .31 to .61 (Baker et al., 2010; Slane, Burt et al., in preparation). This suggests that between 10 and 37% of the variance in the association between bulimic behaviors and AUD symptoms is accounted for by common genetic factors. Again, in the present study, genetic correlations were generally smaller than those indicated in extant research, but some were within the range of those identified previously.

In general, common nonshared environmental effects were not significant, corroborating previous research (Baker et al., 2010; Kendler et al., 1995; Mitchell et al., 2010; Slane, Burt et

al., in preparation). These findings suggest that there is little-to-no overlap in the nonshared environmental effects that influence the association between bulimic behaviors and AUD symptoms. The only exception was a significant common nonshared environmental effect underlying associations between age 25 binge eating with age 25 AUD symptoms. This suggests that for this associations, there are nonshared environmental effects (i.e., factors that act to make twins within a pair different) that similarly influence the association between binge eating and AUD symptoms. It is interesting that the bulimic behavior that was significant was a symptom that maps on most closely to BN (i.e., binge eating). Yet, common nonshared environmental effects were not indicated for associations between compensatory behavior and AUD symptoms or DSM BN symptoms and AUD symptoms; however, there may not have been enough variability of these symptoms in the sample (see limitations below).

The result of primary interest in the present study did not follow the main study hypothesis. That is, although several models indicated common genetic influences underlying the association between bulimic behaviors and AUD symptoms, these factors were not the strongest for the Dysregulated cluster compared to the Other clusters, cross-sectionally or longitudinally. In fact, findings indicated no change in the magnitude of shared genetic effects with changes in cluster membership. Results suggest that even though personality heterogeneity does exist among women with BN, there may not be different etiologic effects underlying the association between these phenotypes, based on personality. This was true for DSM BN symptoms as well as the continuous measures of bulimic behaviors examined. It must be noted, however, that the lack of a cluster effect is likely due to the lack of phenotypic associations between bulimic behaviors and AUD symptoms in the Dysregulated cluster at some ages. Indeed, if there is no phenotypic association, there cannot be an etiologic overlap.

Previous studies have indicated higher rates of AUDs in the Dysregulated cluster compared to the Other clusters. Further, the Dysregulated cluster consists primarily of individuals with bulimic pathology (rather than restriction; Claes et al., 2006; Perkins et al., in preparation; Thompson-Brenner et al., 2008; Westen and Harnden-Fischer, 2001). Thus, the present study predicted that the Dysregulated cluster might represent a unique subtype within a larger group of individuals with BN or bulimic behaviors that may be more closely linked etiologically to AUDs. Yet, associations between these disorders were small and often nonsignificant in the Dysregulated cluster. It is unclear why this was the case. One possibility was the use of AUD symptom counts, rather than a continuous measure of AUDs. It may be that more significant associations would have been identified had a continuous measure been used as variability would be increased and a wider range of problems would be present in the sample (i.e., low levels of alcohol use to problematic alcohol use). Indeed, although one of the previous studies that indicated that common genetic factors underlie the association between BN and AUDs also used a measure of AUD diagnoses (Baker et al., 2010), the other study finding a shared etiology between these disorders used a continuous measure of alcohol use (Slane, Burt et al., in preparation). Thus, the present study may have been limited in its ability to detect shared genetic transmission between BN and AUDs given that only a measure of diagnostic symptoms of AUDs was available. Although in some instances, shared genetic transmission was indicated, these estimates were small which may have been due to the use of diagnostic symptoms of AUDs rather than a continuous measure. This may have also inhibited the ability to find significant cluster effects. Future examinations of these associations within the Dysregulated may want to use a continuous measure of alcohol use.

In general, cluster membership did not moderate unique genetic or unique environmental effects on bulimic behaviors and in cases where it did, these effects were small-to-moderate in magnitude. Although changes in the unique genetic and environmental effects on bulimic behaviors by cluster membership were not hypothesized, it is not surprising given that the Dysregulated cluster typically only consists of women exhibiting bulimic behaviors (rather than restriction as in the case of anorexia nervosa, restricting type) in cluster analytic studies of women with eating disorders (Claes et al., 2006; Perkins et al., in preparation; Thompson-Brenner et al., 2008; Westen and Harnden-Fischer, 2001). Therefore, it may be that the personality traits that characterize this cluster contribute to the heritability of BN. In fact, this may aid in explaining the heritability ranges that are found in studies that examine the magnitude of genetic factors in BN and bulimic behaviors among full samples (i.e., BN ranges = 28-83% and bulimic behavior ranges = 41-70%; Bulik et al., 1998, 2003; Kendler et al., 1991, 1995; Klump et al., 2000; Kortegaard et al., 2001; Reichborn-Kjennerud et al., 2003, 2004; Rowe et al., 2002; Sullivan et al., 1998; Wade et al., 1999; Walters et al., 1992). That is, a sample consisting of women with BN will include those in the Dysregulated cluster as well as those in Other clusters so heritability may be diluted.

Despite the novelty of the present findings, some limitations must be noted. First, although the present study provided information regarding associations among both continuous measures of bulimic behaviors and DSM BN symptom counts, there may have not been enough variability in DSM BN symptom counts in the sample to find significant associations with this phenotype. Indeed, only about 6.5% of the sample at both time points had one or more BN symptom. Importantly, however, although findings with DSM BN symptoms were less consistent across analyses, there were some significant results involving these symptoms.

Further, despite this low variability, as stated in the Methods, the examination of BN symptom counts and continuous measures of bulimic behaviors was important as it allowed for the determination of whether associations with BN are driven by particular symptoms (e.g., binge eating) or symptoms of the full syndrome.

Second, as stated previously, the present study used AUD symptom counts, rather than a continuous measure of AUDs. It may be that had a continuous measure of alcohol use been used, estimates of shared genetic etiology would have been larger and significant cluster moderation would have been identified.

This study was the first to examine whether cluster membership influences the common genetic effects on the covariance between BN and AUDs as well as the unique genetic influence on BN and bulimic behaviors. Additional research is therefore needed to determine whether findings in the present study are replicable. Given the deleterious effects of the comorbidity between BN and AUDs (Dansky et al., 2000; Duncan et al., 2006; Keel et al., 1999), future studies should continue to determine what factors might be influencing the co-occurrence of these disorders. Future longitudinal studies examining the development of BN should include measures of both AUD and personality traits, as this could aid in identifying prospective risk factors.

APPENDIX

APPENDIX: STUDY SUMMARY

Findings from the two studies that comprise this dissertation are summarized below. In the first paper, the dysregulated cluster was identified at both time points and it emerged as the most stable profile compared to the other clusters. Examination of the correlates revealed increased levels of alcohol use disorder symptoms, depressive symptoms, trait anxiety, and behavioral disinhibition among the dysregulated group with stability longitudinally. Findings suggest that the dysregulated cluster is a relatively robust profile that is present across adolescence and into adulthood.

In the second paper, twin moderation models suggested small-to-moderate common genetic transmission between bulimic behaviors and AUD symptoms. However, shared genetic effects did not differ by personality cluster. Nevertheless, findings did indicate in some models that genetic influences that are unique to bulimic behaviors are higher among the Dysregulated cluster compared to the Other clusters, suggesting an influence of personality on bulimic behaviors alone, but not on their association with AUD symptoms. Despite the presence of shared genetic transmission between BN and AUDs, cluster membership did not affect etiologic associations between the phenotypes. This suggests that although personality clusters may be associated with the etiology of BN, they are unlikely to account for associations between BN and AUDs and inconsistent findings in the literature regarding their shared etiology.

Table 9

Means and Standard Deviations of Bulimic Behaviors, DSM BN symptoms, and AUD Symptoms by Cluster at Age 17 and 25

	Age 17		Age 25	
	Dysregulated	Other Clusters	Dysregulated	Other Clusters
	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)
MEBS Total Score	7.93 (5.85)	6.00 (5.43)	8.63 (5.81)	5.95 (5.16)
Body Dissatisfaction	2.47 (2.18)	2.09 (1.99)	3.24 (2.27)	2.19 (2.11)
Weight Preoccupation	2.88 (2.42)	2.34 (2.31)	3.19 (2.38)	2.33 (2.18)
Binge Eating	1.66 (1.61)	0.91 (1.28)	1.34 (1.68)	0.83 (1.25)
Compensatory Behavior	0.33 (0.68)	0.25 (0.68)	0.30 (0.70)	0.20 (0.61)
Bulimia Nervosa Symptoms	0.27 (0.89)	0.12 (0.60)	0.13 (0.66)	0.17 (0.86)
Alcohol Composite	0.95 (2.08)	0.31 (1.04)	0.98 (1.77)	0.55 (1.31)

Note. MEBS = Minnesota Eating Behavior Survey. Raw scores for Binge Eating, Compensatory Behaviors, Bulimia Nervosa Symptoms, and Alcohol Composite shown for descriptive purposes.

Table 10

Cross-Sectional and Longitudinal Phenotypic Correlations within the Dysregulated Cluster and Other Clusters at Age 17 and 25

	Cross-Sectional Associations				**Longitudinal Associations**	
	Age 17 AUD Symptoms		Age 25 AUD Symptoms		Age 17 Bulimic Behaviors and Age 25 AUD Symptoms	
Bulimic Behaviors	Age 17 Dysregulated (n=92-262)	Age 17 Other Clusters (n=509-968)	Age 25 Dysregulated (n=313-432)	Age 25 Other Clusters (n=577-742)	Age 25 Dysregulated (n=178-386)	Age 25 Other Clusters (n=342-670)
MEBS Total Score	.25*	.14**	.06	.09*	.07	.24**
Body Dissatisfaction	.24*	.12**	-.01	.05	.06	.20**
Weight Preoccupation	.12	.08	.03	.07	.07	.15*
Binge Eating	.21*	.15**	.11	.11**	.02	.22**
Compensatory Behavior	.29**	.13**	.09	.15**	.17*	.22**
DSM BN Symptoms	.16*	.05	.15**	.08*	-.01	.05

Note. AUD = Alcohol Use Disorder; MEBS = Minnesota Eating Behavior Survey; DSM BN = Diagnostic and Statistical Manual Bulimia Nervosa. Sample sizes vary due to missing data. * $p < .05$, ** $p < .01$.

Table 11

Fit Statistics for GxE in the Presence of r_{ge} Models of the Association between Bulimic Behaviors and Alcohol Use Disorder Symptoms by Cluster Membership

Model	-2lnL	*df*	X^2 (*df*)	*p*	AIC
Cross-Sectional Models at Age 17					
MEBS Total Score					
ACE moderation	4589.41	1690	--	--	1209.41
AE moderation	**4589.41**	**1695**	**0.00 (5)**	**.99**	**1199.41**
Body Dissatisfaction					
ACE moderation	4593.37	1690	--	--	1213.37
AE moderation	**4593.37**	**1695**	**0.00 (5)**	**.99**	**1203.37**
Binge Eating					
ACE moderation	4620.183	1690	--	--	1240.18
AE moderation	**4624.710**	**1695**	**4.53 (5)**	**.48**	**1234.71**
Compensatory Behavior					
ACE moderation	4640.210	1690	--	--	1260.21
AE moderation	**4641.635**	**1695**	**1.43 (5)**	**.92**	**1251.64**
DSM BN Symptoms					
ACE moderation	6293.616	2277	--	--	1739.62
AE moderation	**6293.616**	**2282**	**0.00 (5)**	**.99**	**1729.62**
Cross-Sectional Models at Age 25					
MEBS Total Score					
ACE moderation	5115.360	1878	--	--	1359.36
AE moderation	**5117.823**	**1883**	**2.46 (5)**	**.78**	**1351.82**
Binge Eating					
ACE moderation	5160.696	1878	--	--	1404.70
AE moderation	**5161.047**	**1883**	**0.35 (5)**	**.99**	**1395.05**
Compensatory Behavior					
ACE moderation	5179.732	1878	--	--	1423.73
AE moderation	**5184.600**	**1883**	**4.87 (5)**	**.43**	**1418.60**
DSM BN Symptoms					
ACE moderation	5950.401	2129	--	--	1692.40
AE moderation	**5950.404**	**2134**	**0.00 (5)**	**.99**	**1682.40**

Note. DSM BN = Diagnostic and Statistical Manual Bulimia Nervosa; MEBS = Minnesota Eating Behavior Survey. Longitudinal models include age 17 bulimic behaviors and age 25 AUD symptoms modified by age 25 cluster membership. -2lnL = -2 times log likelihood of data; *df* = degress of freedom; AIC = Akaike's information criterion; X^2 = compares the ACE fully unconstrained model and AE submodel. The best-fitting model is indicated by bold type.

Table 11 (cont'd)

Model	-2lnL	*df*	X^2 (*df*)	*p*	AIC
Longitudinal Models					
MEBS Total Score					
ACE moderation	4169.82	1538	--	--	1093.82
AE moderation	**4169.83**	**1543**	**0.01 (5)**	**.99**	**1083.83**
Body Dissatisfaction					
ACE moderation	4183.15	1538	--	--	1107.15
AE moderation	**4183.15**	**1543**	**0.00 (5)**	**.99**	**1097.15**
Weight Preoccupation					
ACE moderation	4210.68	1538	--	--	1134.68
AE moderation	**4210.76**	**1543**	**0.07 (5)**	**.99**	**1124.76**
Binge Eating					
ACE moderation	4219.10	1538	--	--	1143.10
AE moderation	**4219.10**	**1543**	**0.00 (5)**	**99**	**1133.10**
Compensatory Behavior					
ACE moderation	4239.86	1538	--	--	1163.86
AE moderation	**4240.36**	**1543**	**0.49 (5)**	**.99**	**1154.36**

Note. DSM BN = Diagnostic and Statistical Manual Bulimia Nervosa; MEBS = Minnesota Eating Behavior Survey. Longitudinal models include age 17 bulimic behaviors and age 25 AUD symptoms modified by age 25 cluster membership. -2lnL = -2 times log likelihood of data; *df* = degress of freedom; AIC = Akaike's information criterion; X^2 = compares the ACE fully unconstrained model and AE submodel. The best-fitting model is indicated by bold type.

Table 12

Unstandardized Genetic Parameter Estimates from the Best Fitting GxE in the Presence of r_{ge} Moderator Models

Model	a_p	a_c	a_u	βa_{xc}	βa_{xu}
Cross-Sectional Models at Age 17					
MEBS Total Score					
AE	**.75**	**.27**	**.71**	-.01	-.26
	(.67, .82)	**(.10, .43)**	**(.59, .82)**	(-.36, .34)	(-.66, .15)
Body Dissatisfaction					
AE	**.75**	**.24**	**.74**	-.08	-.18
	(.68, .82)	**(.08, .41)**	**(.63, .85)**	(-.41, .24)	(-.59, .18)
Binge Eating					
AE	**.74**	**.25**	**.56**	.16	-.13
	(.67, .82)	**(.09, .41)**	**(.40, .69)**	(-.24, .54)	(-.96, .59)
Compensatory Behavior					
AE	**.74**	**.17**	.28	.23	**.63**
	(.67, .81)	**(.00, .35)**	(-.22, .50)	(-.13, .57)	**(.04, 1.17)**
DSM BN Symptoms					
AE	**.75**	.04	**.60**	.10	-.29
	(.67, .82)	(-.08, .17)	**(.44, .72)**	(-.14, .35)	(-.60, .08)

Note. MEBS = Minnesota Eating Behaviors Survey; BN = Bulimia Nervosa, AUD = Alcohol Use Disorder. a_p = AUD symptoms unique genetic path (except for longitudinal models where this estimates bulimic behaviors unique genetic path); a_c = common genetic path; a_u = bulimic behaviors unique genetic path (except for longitudinal models where this estimates AUD symptoms unique genetic path); βa_{xc} = common genetic moderation effect; βa_{xu} = unique genetic moderation effect. Alcohol use disorder symptoms were assessed using DSM-III-R criteria at age 17 and DSM-IV criteria at age 25. DSM BN symptoms were assessed using DSM-IV criteria at both time points. Ninety-five percent confidence intervals are in parentheses. Significant effects are indicated by boldface.

Table 12 (cont'd)

Model	a_p	a_c	a_u	βa_{xc}	βa_{xu}
Cross-Sectional Models at Age 25					
MEBS Total Score					
AE	**.58**	.14	**.74**	.06	.09
	(.49, .67)	(-.04, .31)	**(.65, .84)**	(-.24, .37)	(-.11, .27)
Binge Eating					
AE	**.58**	.06	**.62**	.10	.16
	(.49, .67)	(-.11, .23)	**(.51, .72)**	(-.18, .38)	(-.06, .36)
Compensatory Behaviors					
AE	**.58**	.13	**.67**	.02	-.25
	(.49, .67)	(-.05, .30)	**(.56, .76)**	(-.31, .37)	(-1.25, .04)
DSM BN symptoms					
AE	**.59 (.50,**	.05	**.53**	.23	-.16
	.67)	(-.12, .21)	**(.40, .64)**	(-.01, .48)	(-.56, .10)

Note. MEBS = Minnesota Eating Behaviors Survey; BN = Bulimia Nervosa, AUD = Alcohol Use Disorder. a_p = AUD symptoms unique genetic path (except for longitudinal models where this estimates bulimic behaviors unique genetic path); a_c = common genetic path; a_u = bulimic behaviors unique genetic path (except for longitudinal models where this estimates AUD symptoms unique genetic path); βa_{xc} = common genetic moderation effect; βa_{xu} = unique genetic moderation effect. Alcohol use disorder symptoms were assessed using DSM-III-R criteria at age 17 and DSM-IV criteria at age 25. DSM BN symptoms were assessed using DSM-IV criteria at both time points. Ninety-five percent confidence intervals are in parentheses. Significant effects are indicated by boldface.

Table 12 (cont'd)

Model	a_p	a_c	a_u	βa_{xc}	βa_{xu}
Longitudinal Models with Age 17 Bulimic Behaviors and Age 25 AUD Symptoms					
Age 25 Cluster Membership and Age 17 MEBS Total Score					
AE	**.72**	**.17**	**.33**	-.03	**.51**
	(.61, .83)	**(.01, .33)**	**(.17, .47)**	(-.34, .28)	**(.24, .73)**
Age 25 Cluster Membership and Age 17 Body Dissatisfaction					
AE	**.77**	**.16**	**.35**	-.01	**.49**
	(.66, .87)	**(.00, .31)**	**(.20, .48)**	(-.30, .29)	**(.24, .71)**
Age 25 Cluster Membership and Age 17 Weight Preoccupation					
AE	**.64**	.14	**.34**	-.11	**.53**
	(.51, .76)	(-.06, .31)	**(.19, .47)**	(-.45, .24)	**(.28, .74)**
Age 25 Cluster Membership and Age 17 Binge Eating					
AE	**.61**	.08	**.35**	.07	**.48**
	(.46, .73)	(-.14, .29)	**(.20, .49)**	(-.31, .45)	**(.21, .71)**
Age 25 Cluster Membership and Age 17 Compensatory Behaviors					
AE	**.48**	**.36**	.24	-.20	**.61**
	(.26, .64)	**(.12, .54)**	(-.03, .42)	(-.54, .19)	**(.36, .85)**

Note. MEBS = Minnesota Eating Behaviors Survey; BN = Bulimia Nervosa, AUD = Alcohol Use Disorder. a_p = AUD symptoms unique genetic path (except for longitudinal models where this estimates bulimic behaviors unique genetic path); a_c = common genetic path; a_u = bulimic behaviors unique genetic path (except for longitudinal models where this estimates AUD symptoms unique genetic path); βa_{xc} = common genetic moderation effect; βa_{xu} = unique genetic moderation effect. Alcohol use disorder symptoms were assessed using DSM-III-R criteria at age 17 and DSM-IV criteria at age 25. DSM BN symptoms were assessed using DSM-IV criteria at both time points. Ninety-five percent confidence intervals are in parentheses. Significant effects are indicated by boldface.

Table 13

Unstandardized Nonshared Environmental Parameter Estimates from the Best Fitting GxE in the Presence of r_{ge} Moderator Models

Model	e_p	e_c	e_u	βe_{xc}	βe_{xu}
Cross-Sectional Models at Age 17					
MEBS Total Score					
AE	**.66**	-.03	**.63**	.18	**.28**
	(.61, .71)	(-.14, .08)	**(.56, .72)**	(-.13, .49)	**(.03, .50)**
Body Dissatisfaction					
AE	**.66**	-.03	**.62**	.22	.23
	(.61, .71)	(-.14, .08)	**(.55, .70)**	(-.07, .50)	(-.01, .46)
Binge Eating					
AE	**.66**	.05	**.76**	-.10	.22
	(.62, .71)	(-.08, .18)	**(.68, .86)**	(-.43, .28)	(-.25, .51)
Compensatory Behavior					
AE	**.66**	.06	**.92**	-.11	-.44
	(.62, .71)	(-.10, .21)	**(.83, 1.01)**	(-.38, .21)	(-.63, .04)
DSM BN Symptoms					
AE	**.66**	.03	**.71**	.10	**.57**
	(.61, .71)	(-.07, .13)	**(.63, .81)**	(-.15, .34)	**(.39, .73)**

Note. MEBS = Minnesota Eating Behaviors Survey; BN = Bulimia Nervosa, AUD = Alcohol Use Disorder. e_p = AUD symptoms unique nonshared environmental path (except for longitudinal models where this estimates bulimic behaviors unique nonshared environmental path); e_c = common nonshared environmental path; e_u = bulimic behaviors unique nonshared environmental path (except for longitudinal models where this estimates AUD symptoms unique nonshared environmental path); βe_{xc} = common nonshared moderation effect; βe_{xu} = unique nonshared moderation effect. Alcohol use disorder symptoms were assessed using DSM-III-R criteria at age 17 and DSM-IV criteria at age 25. DSM BN symptoms were assessed using DSM-IV criteria at both time points. Ninety-five percent confidence intervals are in parentheses. Significant effects are indicated by boldface.

Table 13 (cont'd)

Model	e_p	e_c	e_u	βe_{xc}	βe_{xu}
Cross-Sectional Models at Age 25					
MEBS Total Score					
AE	**.78**	.07	**.58**	-.10	.10
	(.73, .84)	(-.03, .17)	**(.51, .65)**	(-.30, .11)	(-.05, .25)
Binge Eating					
AE	**.78**	**.20**	**.67**	-.15	.11
	(.73, .84)	**(.09, .31)**	**(.60, .75)**	(-.35, .06)	(-.05, .27)
Compensatory Behavior					
AE	**.78**	.09	**.63**	-.09	**.40**
	(.73, .84)	(-.01, .20)	**(.56, .71)**	(-.34, .15)	**(.23, .56)**
DSM BN symptoms					
AE	**.78**	.07	**.80**	-.11	**.16**
	(.73, .84)	(-.05, .18)	**(.73, .88)**	(-.30, .08)	**(.03, .29)**

Note. MEBS = Minnesota Eating Behaviors Survey; BN = Bulimia Nervosa, AUD = Alcohol Use Disorder. e_p = AUD symptoms unique nonshared environmental path (except for longitudinal models where this estimates bulimic behaviors unique nonshared environmental path); e_c = common nonshared environmental path; e_u = bulimic behaviors unique nonshared environmental path (except for longitudinal models where this estimates AUD symptoms unique nonshared environmental path); βe_{xc} = common nonshared moderation effect; βe_{xu} = unique nonshared moderation effect. Alcohol use disorder symptoms were assessed using DSM-III-R criteria at age 17 and DSM-IV criteria at age 25. DSM BN symptoms were assessed using DSM-IV criteria at both time points. Ninety-five percent confidence intervals are in parentheses. Significant effects are indicated by boldface.

Table 13 (cont'd)

Model	e_p	e_c	e_u	βe_{xc}	βe_{xu}
Longitudinal Models with Age 17 Bulimic Behaviors and Age 25 AUD Symptoms					
Age 25 Cluster Membership and Age 17 MEBS Total Score					
AE	**.65**	.10	**.77**	-.11	-.01
	(.58, .74)	(-.04, .25)	**(.70, .84)**	(-.40, .17)	(-.15, .16)
Age 25 Cluster Membership and Age 17 Body Dissatisfaction					
AE	**.64**	.09	**.77**	-.23	-.02
	(.57, .72)	(-.06, .23)	**(.70, .83)**	(-.50, .04)	(-.16, .15)
Age 25 Cluster Membership and Age 17 Weight Preoccupation					
AE	**.74**	.03	**.78**	.08	-.04
	(.67, .84)	(-.10, .18)	**(.71, .84)**	(-.20, .33)	(-.18, .13)
Age 25 Cluster Membership and Age 17 Binge Eating					
AE	**.78**	**.17**	**.76**	-.17	.00
	(.70, .88)	**(.02, .31)**	**(.69, .83)**	(-.42, .09)	(-.14, .17)
Age 25 Cluster Membership and Age 17 Compensatory Behaviors					
AE	**.88**	.01	**.75**	.09	-.01
	(.79, .98)	(-.11, .14)	**(.67, .82)**	(-.13, .29)	(-.14, .15)

Note. MEBS = Minnesota Eating Behaviors Survey; BN = Bulimia Nervosa, AUD = Alcohol Use Disorder. e_p = AUD symptoms unique nonshared environmental path (except for longitudinal models where this estimates bulimic behaviors unique nonshared environmental path); e_c = common nonshared environmental path; e_u = bulimic behaviors unique nonshared environmental path (except for longitudinal models where this estimates AUD symptoms unique nonshared environmental path); βe_{xc} = common nonshared moderation effect; βe_{xu} = unique nonshared moderation effect. Alcohol use disorder symptoms were assessed using DSM-III-R criteria at age 17 and DSM-IV criteria at age 25. DSM BN symptoms were assessed using DSM-IV criteria at both time points. Ninety-five percent confidence intervals are in parentheses. Significant effects are indicated by boldface.

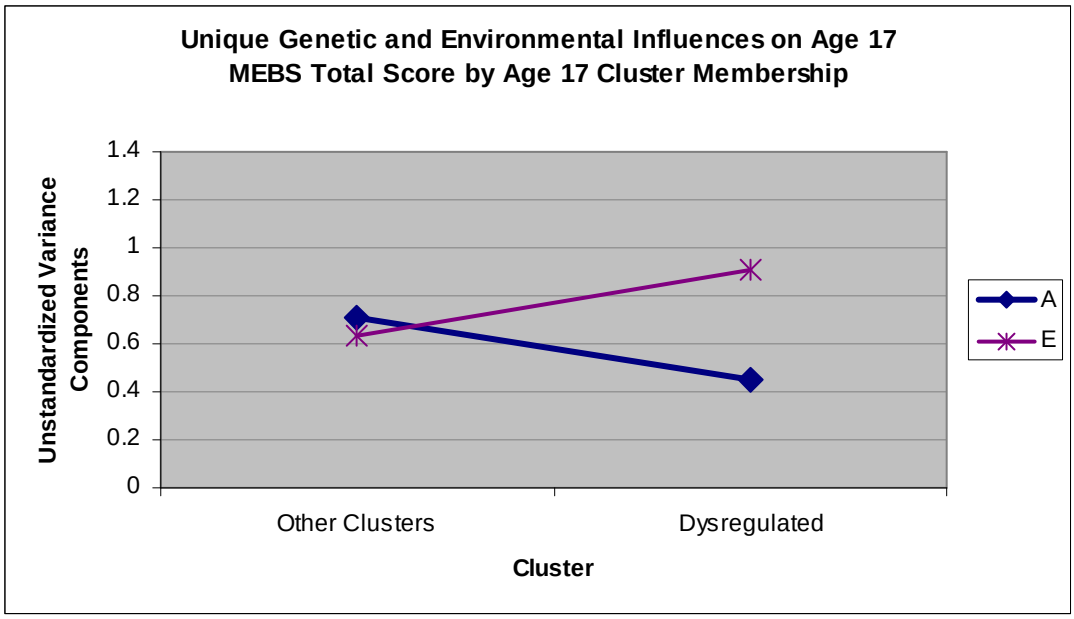

Figure 3. Unstandardized variance components of unique genetic (A) and nonshared environmental (E) influences on age 17 MEBS total score by age 17 cluster membership.

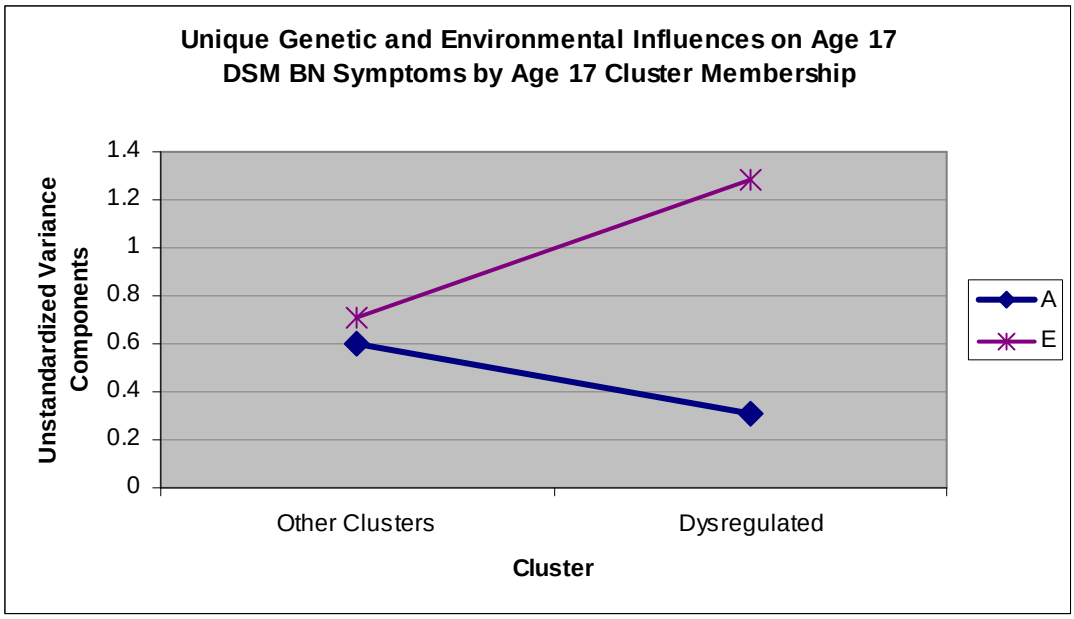

Figure 4. Unstandardized variance components of unique genetic (A) and nonshared environmental (E) influences on age 17 DSM BN symptoms by age 17 cluster membership.

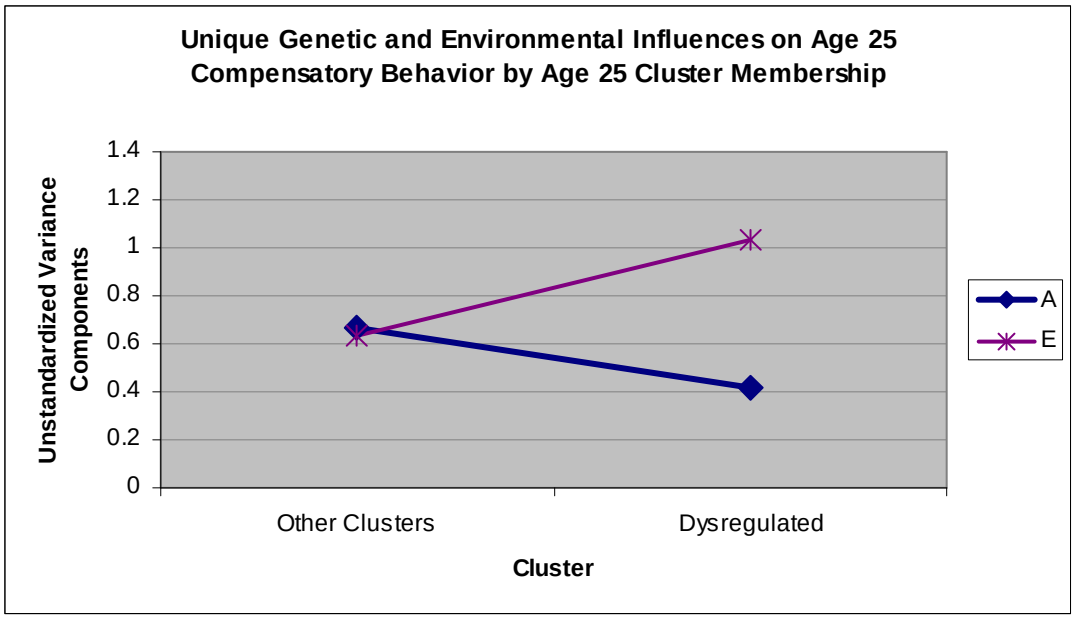

Figure 5. Unstandardized variance components of unique genetic (A) and nonshared environmental (E) influences on age 25 compensatory behavior by age 25 cluster membership.

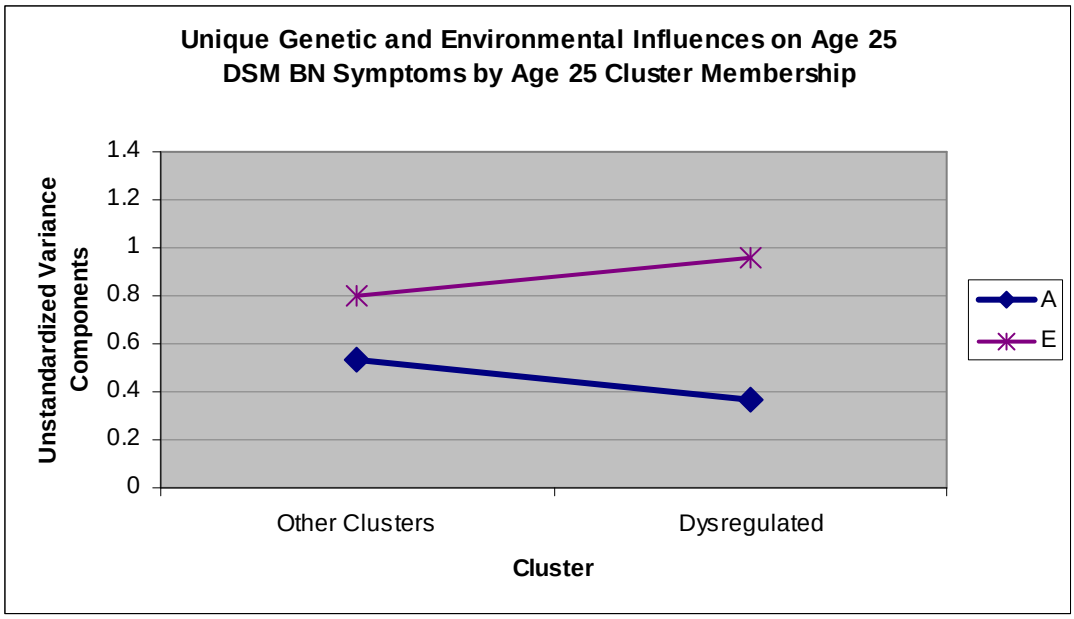

Figure 6. Unstandardized variance components of unique genetic (A) and nonshared environmental (E) influences on age 25 DSM BN symptoms by age 25 cluster membership.

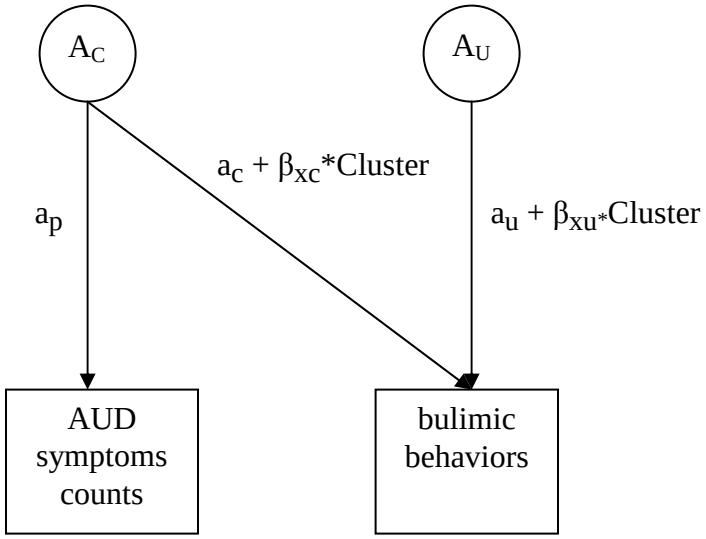

Figure 7. GxE in the Presence of r_{ge} Model. a_p = genetic effects that are specific to the first phenotype (i.e., unique) to AUD; a_c = genetic effects that are common between the phenotypes; a_u = genetic effects that are unique to bulimic behaviors; β_{xc} = coefficient representing moderation between the common path and the moderator; β_{xu} = coefficient representing moderation between the unique path and the moderator; Cluster = moderator value (coded as: Other clusters [0] and Dysregulated [1]). The unstandardized common genetic influence on the association between bulimic behaviors and AUD symptoms is indicated by the equation $a_c + \beta_{xc}$ *Cluster. The shared (C) and nonshared (E) environmental components are not shown.

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