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EARLY CHILDHOOD PERFECTIONISM

Katherine A. Halmi, MD,

Professor of Psychiatry, Weill Cornell Medical College, 21 Bloomingdale Rd White Plains, NY 10605

Dara Bellace, PhD,

Assistant Professor of Psychology in Psychiatry, Weill Cornell Medical College, 21 Bloomingdale Rd White Plains, NY 10605

Samantha Berthod, MA,

Research Coordinator, Eating Disorders Research Program, Weill Cornell Medical College, 21 Bloomingdale Rd White Plains, NY 10605

Samiran Ghosh, PhD,

Department of Psychiatry and Biostatistics, Weill Cornell Medical College, 21 Bloomingdale Rd White Plains, NY 10605

Wade Berrettini, MD, PhD,

Department of Psychiatry, Center of Neurobiology and Behavior, University of Pennsylvania, Philadelphia

Harry A. Brandt, MD,

Department of Psychiatry, Sheppard Pratt Health System, Towson, Maryland

Cynthia M. Bulik, PhD,

Department of Psychiatry, University of North Carolina at Chapel Hill, Chapel Hill, North Carolina

Steve Crawford, MD,

Department of Psychiatry, Sheppard Pratt Health System, Towson, Maryland

Manfred M. Fichter, MD,

Department of Psychiatry, University of Munich (LMU), Munich, Germany Roseneck Hospital for Behavioral Medicine, Prien, Germany

Craig L. Johnson, PhD,

Center for Eating Recovery, Denver, Colorado

Allan Kaplan, MD,

Center for Addiction and Mental Health, Toronto, Canada, Toronto General Hospital, Toronto, Canada and the Institute of Medical Science, University of Toronto, Toronto, Canada

Walter H. Kaye, MD,

Director, Eating Disorder Research and Treatment Program, Department of Psychiatry, University of California San Diego, La Jolla, California

Laura Thornton, PhD,

Department of Psychiatry, University of North Carolina at Chapel Hill, Chapel Hill, North Carolina

Janet Treasure, MD,

Department of Academic Psychiatry, Kings College London, Institute of Psychiatry, London, United Kingdom

A subset of this data was presented at the Eating Disorder Research Society annual meeting in 2009.

D. Blake Woodside, MD, and

Program for Eating Disorders, Toronto General Hospital, Toronto, Canada and Department of Psychiatry, University of Toronto, Toronto, Canada

Michael Strober, PhD

Department of Psychiatry, Semel Institute for Neuroscience & Human Behavior, David Geffen School of Medicine, University of California at Los Angeles, California

Abstract

Objective—To examine childhood perfectionism in anorexia nervosa (AN) restricting (RAN), purging (PAN), and binge eating with or without purging (BAN) subtypes.

Method—The EATATE, a retrospective assessment of childhood perfectionism, and the Eating Disorder Inventory (EDI-2) were administered to 728 AN participants.

Results—EATATE responses revealed General Childhood Perfectionism, 22.3% of 333 with RAN, 29.2% of 220 with PAN, and 24.8% of 116 with BAN; School Work Perfectionism, 31.2% with RAN, 30.4% with PAN, and 24.8% with BAN; Childhood Order and Symmetry, 18.7% with RAN, 21.7% with PAN, and 17.8% with BAN; and Global Childhood Rigidity, 42.6% with RAN, 48.3% with PAN and 48.1% with BAN. Perfectionism preceded the onset of AN in all subtypes. Significant associations between EDI-2 Drive for Thinness and Body Dissatisfaction were present with four EATATE subscales.

Discussion—Global Childhood Rigidity was the predominate feature that preceded all AN subtypes. This may be a risk factor for AN.

The trait of perfectionism, a multidimensional construct generally defined as the pursuit of extreme, unattainable standards of performance and intolerance of mistakes, has been implicated as a predisposing risk factor in eating disorders – anorexia nervosa (AN) in particular. Several detailed, comprehensive reviews of research in this area have now been published.¹⁻⁴ Whereas the majority of the associations described are derived from cross-sectional designs, some evidence supports the idea that higher than normative levels of perfectionism appear in advance of the onset of dieting and weight loss.^{5,6} A higher level of perfectionism has also been shown in parents of individuals with AN than in parents of non-AN controls,⁷ as well as in patients with AN compared to psychiatric controls.^{8,9}

The relationship between perfectionism and both eating and related psychopathological features in individuals with AN has also been studied, revealing positive associations with obsessive compulsive disorder (OCD) and/or obsessive compulsive personality disorder (OCPD)^{10,11} Individuals with concurrent AN and perfectionistic tendencies show an increased severity of illness as reflected in lower BMI¹², as well as an increased resistance to change,¹² and less favorable prognosis.¹³

Results of studies that have compared levels of perfectionism across the three subtypes of AN [e.g., restricting AN (RAN), purging AN (PAN), and binge-purge AN (BAN)] have been inconsistent,^{12,14} but the relationship between early childhood perfectionism and phenotypic variations within AN has not been thoroughly studied. Studies with an adequate sample size identifying childhood risk factors or correlates for developing AN have not assessed early childhood perfectionism.¹⁵ Accordingly, we report herein the prevalence of childhood perfectionism in each of the major clinical subtypes of AN. Participants studied were from a large, multi-center international collaborative study searching for disease susceptibility genes in AN and intermediate behavioral phenotypes associated with loci of potential interest.¹⁶ We hypothesized that components of premorbidly expressed perfectionism would occur more often in the PAN subtype, which in several studies^{12,17} has

been significantly correlated with greater severity and duration of illness. We hypothesized that two core features of AN psychopathology – drive for thinness and body dissatisfaction – would also be significantly related to aspects of childhood perfectionism.

Method

Participants and Recruitment

Participants were 728 women with a lifetime history of AN who participated in the NIMH funded Genetics of Anorexia Nervosa study. Complete study details are provided in an earlier report.¹⁶ Briefly, probands were male or female age 16 or older, ill or recovered. They must have met a lifetime diagnosis of DSM-IV AN, with or without amenorrhea, at least 3 years prior to study entry and prior to age 45. The amenorrhea criterion was waived because of its lack of applicability to males and the unreliability of its retrospective assessment in females. The threshold for low weight was defined as a body mass index (BMI) at or below 18 kg/m² for females and 19.6 kg/m² for males, which corresponded to the 5th percentile of BMI values from the NHANES epidemiological sample of females and males, respectively, for the average age range (27 – 29 years) of the probands in our previous studies.¹¹ Probands did not engage in regular binge eating, defined in accordance with the DSM-IV guidelines for “regular” binge episodes in bulimia nervosa (i.e., at least twice a week for at least three months). They were required to have at least one first, second, or third degree relative with AN – excluding parents and MZ twins – who was willing to participate in the study. Exclusionary criteria were a maximum lifetime BMI exceeding 30, lack of fluency in either English or German, history of severe CNS trauma, psychotic disorders, or developmental disabilities, or if they had a medical, neurological, or substance use disorder that could confound the diagnosis of AN or interfered with completion of assessments. Affected relatives also had AN and were required to meet the same inclusion and exclusion criteria as probands with the exception that regular binge eating and a diagnosis of bulimia nervosa was permitted in addition to the AN diagnosis. Although they need not have met AN criteria three years prior to the study, affected relatives were required to have had a minimal duration of at least three months at a low weight as outlined above. Data were collected from January 2003 through June 2007.

The 728 participants were subtyped as follows: RAN, n=359, which included restricting and excessive exercise only, no lifetime binge eating or purging; PAN, n=240, which included any lifetime history of vomiting, laxative and/or purgative use; and BAN, n= 129, which included lifetime binge eating with or without purging or a crossover from AN to Bulimia Nervosa (BN). Participants ranged in age from 16 to 81, with a mean age of 29.68 years (SD=11.63).

Probands provided informed consent to participate and permission to contact their willing affected relatives and parents in accordance with the institutional review board (IRB) requirements of each participating site. All probands and affected relatives gave informed consent prior to study entry.

Measures

To establish a lifetime diagnosis of AN and assess other core eating disorder symptoms, the Structured Inventory for Anorexic and Bulimic Eating Syndromes (SIAB)¹⁸ and Module H of the Structured Clinical Interview for DSM Disorders (SCID)¹⁹ were administered. Additional eating disorder symptoms and early childhood perfectionism were assessed using the Eating Disorder Inventory-2 and the EATATE Lifetime Diagnostic Interview, respectively.

Eating Disorder Inventory-2 (EDI-2)—The EDI-2 is a self-report questionnaire assessing the behavioral and psychological traits common in AN and BN.²⁰ It is comprised of eight subscales, including Drive for Thinness, Bulimia, Body Dissatisfaction, Ineffectiveness, Perfectionism, Interpersonal Distrust, Interoceptive Awareness, and Maturity Fears. Here we report on two subscales: Drive for Thinness and Body Dissatisfaction.

EATATE Lifetime Diagnostic Interview—The EATATE is a semi-structured interview for a retrospective assessment of eating disorder symptoms and childhood traits of perfectionism, obsessions and compulsions.²¹ It is comprised of thirteen subscales, which include General Childhood Perfectionism, School Work Perfectionism, Self Care Perfectionism, Order and Tidiness Perfectionism, Pet Perfectionism, Hobby Perfectionism, Other Areas of Perfectionism, Childhood Order and Symmetry, Childhood Cautiousness, Childhood Excessive Doubt, Childhood Rule Driven, Childhood Inflexibility/Stubbornness, and Global Childhood Rigidity. For each EATATE subscale (e.g., School Work Perfectionism) participants were given a score of 0, 1, or 2. A score of 0 on the EATATE indicates the absence of clinically significant symptoms. A score of 1 indicates a presence of symptoms, though not to the extent that one's life is significantly impacted; a score of 2 represents a severity great enough to impact functioning in everyday life and was used for meeting the threshold for childhood perfectionism.

Procedure

Participants were administered the EATATE interview and completed the EDI-2. EATATE interviews were conducted either in person or via telephone. Four subscales of the EATATE were examined in the current study: (1) General Childhood Perfectionism, (2) School Work Perfectionism, (3) Childhood Order and Symmetry, and (4) Global Childhood Rigidity. An individual who met the threshold for General Childhood Perfectionism reported having higher standards and or were more perfectionistic than those around them. They tended to regard other children as having unacceptable standards. These individuals reported that they tended to take longer than their peers to do certain things, which would interfere with other activities like leisure time and time with friends. In addition, other people tended to comment on their tendency to be perfectionistic. School Work Perfectionism was marked by persistence in trying to solve problems when most of one's classmates or friends had given up. These participants also reported spending much longer on their homework than they needed to. They would redo a piece of work if it had errors on it or if they made even one mistake. They were always striving for the best grade and never felt happy or content no matter how hard they had worked. Participants who received a score of 2 on the Childhood Order and Symmetry subscale reported a variety of behaviors including spending a long time doing or redoing their hair to make sure it was straight without bumps in it, and/or being particularly concerned about the symmetry of their hair, hem, or cuffs. These individuals would often spend a long time getting their room tidy and organized, making sure that everything was "just so" and in its proper place. Individuals meeting the threshold for Global Childhood Rigidity described behaviors such as feeling they always had to follow the rules and feeling ashamed if they broke a rule. They also had difficulty adjusting to change, particularly during periods of transition such as moving to a new town or changing schools. These individuals reported an inflexibility that made it difficult for them to cope with having to change their plans on short notice.

An individual's endorsement of symptoms on each of the subscales was evaluated by a doctoral-level psychologist serving as a clinical interviewer. Data were examined for participants who had a rating of 2 on any of the four EATATE subscales. Participants who met the threshold for childhood perfectionism with a score of 2 on the EATATE were asked

whether this perfectionism was present before the onset of their eating disorder. These participants were also asked for the age at which their perfectionism was first expressed by anchoring their retrospective account to developmental milestones. The EDI-2 self-report questionnaire was completed by each participant.

Data Analyses

The total sample size for this study was 728 females. Males were excluded from the current study because there were too few for meaningful comparisons. It is important to note that data for only 420 of the overall sample were utilized in regression analyses, as age at onset of perfectionism was not endorsed in 42% of the participants. This was due to the fact that this age at onset of perfectionism variable was absent if the participant did not meet the threshold for at least one of the types of childhood perfectionism assessed by the EATATE. The missingness of this variable was found to be proportionally distributed on the independent variable, such that the proportional distribution of the three AN subtypes was more or less the same with or without missing observations. We are therefore reporting the results of what is essentially a complete case analysis, which involves the assumption of “missing completely at random.”^{22,23}

Descriptive statistics were used to determine the number and percent of participants diagnosed with each of the three subtypes of AN who endorsed significant symptoms of childhood perfectionism on the four subscales of the EATATE (General Childhood Perfectionism, School Work Perfectionism, Childhood Order and Symmetry, Global Childhood Rigidity). Scores for perfectionism on the EATATE were compared with scores on the Body Dissatisfaction and Drive for Thinness subscales of the EDI-2 using Spearman correlations.

Multinomial logistic regression with polytomous nominal type response was used.²⁴ This requires generalized logit link to be used to examine the association between scores on the four subscales of the EATATE and the three subtypes of AN. The EATATE subscale scores, along with age at onset of AN, and age at onset of perfectionism served as the independent variables, while current eating disorder at time of assessment, and age at time of assessment were covariates. The dependent variable was AN subtype with one of the subtypes of AN as the referent. In order to compare all possible combinations of AN subtypes, we performed two separate regressions. In the first regression we chose RAN as the referent comparing PAN and BAN versus RAN. In the second, PAN was the referent as we compared BAN and RAN versus PAN. Explanatory variables used in the regression context were entered in their original scale (i.e., without any standardization).

We utilized SPSS software (PASW Version 18.0)²⁵ to conduct all but the regression analyses reported in this study. The regression analyses were carried out using SAS software (Version 9.2)²⁶.

Results

Features of Perfectionism and AN Subtypes

Table 1 lists the prevalence of the different subscales of perfectionism by AN subtype. There were no significant differences in the frequency of perfectionism features across the AN subtypes.

There were no differences in age at onset of AN, or in the onset of perfectionism by AN subtype (See Table 2).

Features of Perfectionism, Drive for Thinness, and Body Dissatisfaction

Of the 728 participants who completed the EATATE interview, 691 people also completed the EDI-2 Drive for Thinness and Body Dissatisfaction subscales. There were statistically significant correlations between the EDI-2 subscale scores and each EATATE score (Data available upon request). The correlations were similar in magnitude between the RAN and PAN subtypes. However, no significant associations were observed in the BAN group (see Table 3). In the BAN group, correlations for all EATATE subscales ranged in magnitude from $r = 0.07$ to $r = 0.18$ with the number of participants ranging from 113 to 114.

Predictor Variables for RAN vs PAN

For the regression analyses, initial inspection revealed that the result of the model fitting based on the likelihood ratio statistic and score test was found to be satisfactory, with the proposed model significant at the chosen alpha level of .05. As seen in Table 4, we found that age at onset of perfectionism, Global Childhood Rigidity, current eating disorder at time of assessment, and age at time of assessment were significant predictors ($p < .05$) of AN subtype when RAN was used as the reference variable. It is to be noted that each predictor variable appears twice in the Table, because the referent (RAN) was compared with PAN and BAN separately. Upon examining the odds ratios (see Table 4), the aforementioned significant predictors had a significant 95% confidence interval that did not include the number "1" in the interval. Age at onset of perfectionism had an estimate of -0.094 which yielded an odds ratio of $\exp(-0.094) = 0.91$. Thus, keeping all other predictors at a fixed value, we would expect a 9% decrease in the odds of having a diagnosis of RAN as compared to PAN for every one-year increase in age at onset of perfectionism. Similarly, age at time of assessment had an estimate of 0.039 which yielded an odds ratio of $\exp(0.039) = 1.04$. Here, keeping all other predictors at a fixed value, we would expect a 4% increase in the odds of having a diagnosis of RAN as compared to PAN for every one-year increase in age at time of assessment.

Predictor Variables for RAN vs BAN

Note that for Global Childhood Rigidity, the estimate is 0.754 when the BAN and RAN groups were compared. The corresponding odds ratio was $\exp(0.754) = 2.13$. This means that participants were 2.13 times more likely to be classified as the BAN subtype than the RAN subtype, if their score on Global Childhood Rigidity was a 2 (versus a 0). A similar interpretation can be made for the predictor variable current eating disorder at time of assessment, which yielded an odds ratio of $\exp(0.572) = 1.77$ when RAN is the referent and the comparison is with PAN. Specifically, participants were 1.77 times more likely to fall into the PAN group than the RAN group, if their score on current eating disorder at time of assessment was currently ill versus not ill.

Predictor Variables for PAN vs BAN

Table 5 reveals that age at onset of perfectionism, current eating disorder at time of assessment, and age at time of assessment were significant predictors ($p < .05$) of AN subtype when PAN was used as the reference variable. As stated earlier, the purpose of this second regression was primarily to compare the PAN versus BAN subgroups, as this comparison could not be made using the first regression described above. However, as the comparison of PAN versus RAN is exactly the inverse of comparing RAN versus PAN, we do not repeat the interpretation of those predictors here.

In this second regression analysis, we found that age at onset of perfectionism had an estimate of 0.091 which yielded an odds ratio of $\exp(0.091) = 1.09$. Thus, keeping all other predictors at a fixed value, we would expect a 9% increase in the odds of being diagnosed

with PAN as compared to BAN, for every one-year increase in an individual's age at onset of perfectionism. The predictor variable age at time of assessment had an estimate of -0.035 which yielded an odds ratio of $\exp(-0.035) = 0.96$. Thus, keeping all other predictors at a fixed value, we would expect a slight decrease in the odds of having a diagnosis of PAN as compared to BAN for a one-year increase in age at time of assessment.

Discussion

Although there were no significant differences in frequency of the aspects of perfectionism across AN subtypes, there was a hierarchy of occurrence in the different categories of perfectionism. Global Childhood Rigidity was most prominent, followed by School Work Perfectionism, General Childhood Perfectionism and, finally, Childhood Order and Symmetry. As rigidity or inflexibility is prominent in the majority of individuals with AN,¹¹ expression of perfectionism often appears many years in advance of weight loss and body image disturbance⁶. Both obsessive compulsive personality disorder and anxiety disorders aggregate in families of individuals with AN.²⁷ A plausible notion is that the confluence of childhood rigidity and anxiety proneness are transmissible factors that significantly elevate an individual's risk for developing AN.

Two of the core psychopathological features of AN – Drive for Thinness and Body Dissatisfaction – had small but significant correlations with EATATE categories of childhood perfectionism in the RAN and PAN subtypes. Drive for Thinness suggests an active energy component, which is more likely to be present in General Childhood and School Work Perfectionism. Body Dissatisfaction was related to all categories of perfectionism in these AN subtypes. On the basis of the current data set, we found that the perfectionism categories and the two EDI-2 subscales were unrelated among participants with BAN.

Regression analyses revealed several predictors of AN subtypes such that every one-year increase in age at onset of perfectionism predicted a 9% decrease in the likelihood of having RAN compared to having PAN. Why a later age onset of perfectionism is more likely to predict PAN than RAN is unclear. However, we do know that a very early onset of AN (i.e., between the ages of 10-12) and hence a presumably earlier onset of perfectionism has been associated with the RAN subtype.²⁸ It is thus possible that purging, as well as being heritable²⁹, is also a learned behavior that emerges later on during adolescence. A 4% increase in the odds of having RAN compared to PAN for every one-year increase in age at time of assessment may simply reflect unassessed population characteristics of the RAN and PAN groups.

A one-year increase in the age at onset of perfectionism predicted a 9% increase in the odds of having PAN as compared to BAN, while a one-year increase in the age at time of assessment predicted a slight decrease in the odds of having PAN compared to BAN. Since age at onset of perfectionism was determined on the EATATE by retrospective recall, it is possible that age at time of assessment might influence or bias responses on the EATATE.

Those with Global Childhood Rigidity were almost twice as likely to have BAN than RAN. This association seems counterintuitive as the binge eating and purging behaviors of BAN tend to be associated with other impulsive behaviors such as alcohol and drug abuse which seem contradictory to a childhood characteristic of rigidity. Those individuals who currently had an eating disorder at the time of assessment were 1.77 times more likely to have PAN versus RAN. Reports linking purging behaviors in AN to greater overall morbidity and worse outcome may explain this result.³⁰

The predictors of AN subtypes in this study do not provide a complete understanding of the development of the AN subtypes. There may be influences not examined here that affect the differentiation of AN subtypes. Disturbances of affect and behaviors which are often comorbid with the AN diagnosis may inform the course of a patient's illness. It is plausible to speculate that these comorbid features reflect differences in a complex interactive neurocircuitry and thus may influence the phenotypic variability within AN.^{31, 32} Therefore different aspects of childhood perfectionism may be directed toward the salient features of AN – drive for thinness and body dissatisfaction.

This study had several limitations. Age of onset for AN and perfectionism and endorsement of perfectionism symptoms were made retrospectively and are thus subject to recall biases. Individuals with current perfectionistic traits may have been more inclined to endorse symptoms in childhood, thus inflating the association between childhood perfectionism and the development of AN. In the current study, we were unable to account for the non-independence of the data due to the inclusion of affected relatives because data from the perfectionism measures were ordinal in nature. Failure to correct for correlated observations can lead to false positive findings, although such corrections tend not to dramatically influence analyses.

The EDI-2 is a self-report questionnaire and has the inherent problem of individuals denying the presence and/or severity of symptoms. This may account for the low magnitude of the correlations observed between Drive for Thinness and Body Dissatisfaction and perfectionism symptoms reported during the EATATE interviews.

This study suggests that early childhood perfectionism may be one factor influencing the development of all AN subtypes. Noteworthy are the findings that perfectionism often precedes the development of AN, and that the mean age at onset for both perfectionism and AN occurred prior to age 18 in this study population. A longitudinal study evaluating perfectionism in young children would be valuable to clarify the risk of early childhood excessive perfectionism for developing AN. To date, school-based interventions delivered via the internet or by direct interviews to assess body image and eating attitudes have had significant impacts on reducing risk factors for eating disorders.³³ If early childhood excessive perfectionism is determined to be a definite risk factor for developing AN, then creating and testing intervention techniques may be propitious.

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References

1. Jacobi C, Hayward C, de Zwaan M, Kraemer HC, Agras S. Coming to terms with risk factors for eating disorders: Application of risk terminology and suggestions for a general taxonomy. *Psych Bull.* 2004; 130(1):19–65.
2. Stice E, Whitenton K. Risk factors for body dissatisfaction in adolescent girls: A longitudinal investigation. *Dev Psych.* 2002; 38(5):669–678.
3. Lilenfeld LRR, Wonderlich S, Riso LP, Crosby R, Mitchell J. Eating disorders and personality: A methodological and empirical review. *Clin Psychol Rev.* 2006; 26(3):299–320. [PubMed: 16330138]
4. Bardone-Cone AM, Wonderlich SA, Frost RO, Bulik CM, Mitchell JE, Uppala S, Simonich H. Perfectionism and eating disorders: Current status and future directions. *Clin Psych Rev.* 2007; 27(3):384–405.

5. Fairburn CG, Cooper Z, Doll HA, Welch SL. Risk factors for anorexia nervosa: Three integrated case-control comparisons. *Arch Gen Psychiatry*. 1999; 56(5):468–476. [PubMed: 10232302]
6. Anderlueh MB, Tchanturia K, Rabe-Hesketh S, Treasure J. Childhood obsessive-compulsive personality traits in adult women with eating disorders: Defining a broader eating disorder phenotype. *Am J Psychiatry*. 2008; 160:242–247. [PubMed: 12562569]
7. Woodside DB, Bulik CM, Halmi KA, Fichter MM, Kaplan A, Berrettini WH, et al. Personality, perfectionism, and attitudes towards eating in parents of individuals with eating disorders. *Int J Eat Disord*. 2002; 31(3):290–299. [PubMed: 11920990]
8. Cockell SJ, Hewitt PL, Seal B, Sherry S, Goldner EM, Flett G, Remick RA. Trait and self-presentational dimensions of perfectionism among women with anorexia nervosa. *Cog Ther & Res*. 2002; 26(6):745–758.
9. Bulik CM, Tozzi F, Anderson C, Mazzeo SE, Aggen S, Sullivan PF. The relation between eating disorders and components of perfectionism. *Am J Psychiatry*. 2003; 160(2):366–368. [PubMed: 12562586]
10. Halmi KA, Sunday SR, Klump KL, Strober M, Leckman JF, Fichter M, et al. Obsessions and compulsions in anorexia nervosa subtypes. *Int J Eat Disord*. 2003; 33(3):308–319. [PubMed: 12655628]
11. Halmi KA, Tozzi F, Thornton LM, Crow S, Fichter M, Kaplan AS, et al. The relation among perfectionism, obsessive-compulsive disorder in individuals with eating disorders. *Int J Eat Disord*. 2005; 38(4):371–374. [PubMed: 16231356]
12. Halmi KA, Sunday SR, Strober M, Kaplan A, Woodside DB, Fichter M, et al. Perfectionism in anorexia nervosa: Variation by clinical subtype, obsessiveness, and pathological eating behavior. *Am J Psychiatry*. 2000; 157:1799–1805. [PubMed: 11058477]
13. Nilsson K, Sundbom E, Hagglof B. A longitudinal study of perfectionism in adolescent onset anorexia nervosa-restricting type. *Eur Eat Disord Rev*. 2008; 16(5):386–394. [PubMed: 18059049]
14. Reba L, Thornton L, Tozzi F, Klump KL, Brandt H, Crawford S, et al. Relationships between features associated with vomiting in purging-type eating disorders. *Int J Eat Disord*. 2005; 38(4):287–294. [PubMed: 16261604]
15. Fernandez-Aranda F, Pinheiro AP, Tozzi F, Thornton LM, Fichter MM, Halmi KA, et al. Symptom profile and temporal relation of major depressive disorder in females with eating disorders. *Aust N Z J Psychiatry*. 2007; 41(1):24–31. [PubMed: 17464678]
16. Kaye WH, Bulik CM, Plotnicov K, Thornton L, Devlin B, Fichter MM, et al. The genetics of anorexia nervosa collaborative study: Methods and sample description. *Int J Eat Disord*. 2008; 41(4):289–300. [PubMed: 18236451]
17. Eddy KT, Keel PK, Dorer DJ, Delinsky SS, Franko DL, Herzog DB. Longitudinal comparison of anorexia nervosa subtypes. *Int J Eat Disord*. 2002; 31(2):191–201. [PubMed: 11920980]
18. Fichter MM, Elton M, Engel K, Meyer AE, Mally H, Poustka F. Structured interview for anorexia and bulimia nervosa (SIAB). Development of a new instrument for the assessment of eating disorders. *Int J Eat Disord*. 1991; 10(5):571–592.
19. First, MB.; Spitzer, RL.; Gibbon, M.; Williams, JB. Structured clinical interview for DSM-IV axis I disorders SCID I: Clinician version. Washington, DC: American Psychiatric Press; 1997.
20. Garner, D. *Eating Disorders Inventory-2: Professional Manual*. Odessa, FL: Psychological Assessment Resources, Inc; 1991.
21. Anderlueh M, Tchanturia K, Rabe-Hesketh S, Collier D, Treasure J. Lifetime course of eating disorders: Design and validity testing of a new strategy to define the eating disorder phenotype. *Psychol Med*. 2009; 39(1):105–114. [PubMed: 18377676]
22. Agresti, A. *Categorical data analysis*. New York: J. Wiley & Sons; 2002.
23. Rubin, DB. *Multiple imputation for nonresponse in surveys*. New York: J. Wiley & Sons; 1987.
24. Stokes, ME.; Davis, CS.; Koch, GG. *Categorical data analysis using the SAS system*. 2. Cary NC: SAS Institute Inc.; 2000.
25. PASW Statistics, (Version 18.0). Chicago, IL: SPSS Inc.; 2009.
26. SAS Software, (Version 9.2). Cary, NC: SAS Institute Inc.; 2008.

27. Strober M, Freeman R, Lampert C, Diamond J. The association of anxiety disorders and obsessive compulsive personality disorder with anorexia nervosa: Evidence from a family study with discussion of nosological and neurodevelopmental implications. *Int J Eat Disord.* 2007; 40:S46–51. [PubMed: 17610248]
28. Mitzman SF, Slade P, Dewey ME. Preliminary development of a questionnaire designed to measure neurotic perfectionism in the eating disorders. *J Clin Psychol.* 1994; 50(4):516–522. [PubMed: 7983199]
29. Thornton, LM.; Mazzeo, SE.; Bulik, CM. The heritability of eating disorders: Methods and current findings. In: Adan, RAH.; Kaye, WH., editors. *Behavioral neurobiology of eating disorders: Current topics in behavioral neurosciences.* Vol. 6. New York: Springer-Verlag Berlin Heidelberg; 2011. p. 141-56.
30. Steinhausen HC. The outcome of anorexia nervosa in the 20th century. *Am J Psychiatry.* 2002; 159(9):1284–1293. [PubMed: 12153817]
31. Halmi KA. Perplexities and provocations of eating disorders. *J Child Psychol & Psychiat.* 2009; 50(1-2):163–9. [PubMed: 19220599]
32. Kaye, WH.; Wagner, A.; Fudge, J.; Paulus, M. Neurocircuitry of eating disorders. In: Adan, RAH.; Kaye, WH., editors. *Behavioral neurobiology of eating disorders: Current topics in behavioral neurosciences.* Vol. 6. New York: Springer-Verlag Berlin Heidelberg; 2011. p. 37-57.
33. Celio AA, Winzelberg AJ, Wilfley DE, Eppstein-Herald D, Springer EA, Dev P, et al. Reducing risk factors for eating disorders: Comparison of an internet and classroom-delivered psychoeducational program. *J Consult and Clin Psych.* 2000; 68(4):650–657.

TABLE 1

EATATE Subscales Examined

EATATE Subscale	RAN n =359	PAN n=240	BAN n=129
General Childhood Perfectionism	22.3 % of 333	29.2% of 220	24.8% of 116
School Work Perfectionism	31.2% of 333	30.4% of 220	24.8% of 116
Childhood Order and Symmetry	18.7% of 327	21.7% of 219	17.8% of 115
Global Childhood Rigidity	42.6% of 333	48.3% of 220	48.1% of 116

* Note "n" values vary slightly due to missing data

TABLE 2

Age at Onset of Perfectionism as Compared to Age at Onset of Anorexia Nervosa in Anorexia Nervosa Subtypes

AN Subtype	Age at Onset of Perfectionism (years) Mean \pm SD	Age at Onset of AN (years) Mean \pm SD	Difference between Age at Onset of AN (years) and Age at Onset of Perfectionism
RAN n = 204	8.5 \pm 3.2	17.2 \pm 4.6	8.2 \pm 4.6
PAN n = 147	7.5 \pm 3.3	17.4 \pm 5.5	9.8 \pm 6.1
BAN n = 76	8.4 \pm 3.3	16.7 \pm 4.0	8.4 \pm 4.0

TABLE 3

EDI-2 and EATATE Subscale Correlations in AN Subgroups

Anorexia Nervosa Restricting					
EATATE Subscales	n	EDI-2 Drive for Thinness (r)	p	EDI-2 Body Dissatisfaction (r)	p
General Childhood Perfectionism	326	.12	<.05	.11	NS
School Work Perfectionism	326	.25	<.01	.28	<.01
Childhood Order and Symmetry	320	.10	NS	.16	<.01
Global Childhood Rigidity	334	.11	<.05	.12	<.05

Anorexia Nervosa Purging						
EATATE Subscales	n	EDI-2 Drive for Thinness (r)	p	n	EDI-2 Body Dissatisfaction (r)	p
General Childhood Perfectionism	215	.14	<.05	212	.20	<.01
School Work Perfectionism	215	.14	<.05	212	.20	<.01
Childhood Order and Symmetry	214	.06	NS	211	.13	NS
Global Childhood Rigidity	215	.08	NS	212	.15	<.05

Anorexia Nervosa Binge/Purge					
No Significant Correlations					

* r = Spearman correlation

* NS = not significant

TABLE 4

RAN as Referent and Odds Ratio Estimates

RAN as Referent						
Variable	Logit (PAN/RAN)		p Value	Logit (BAN/RAN)		p Value
	Estimate	Standard Error		Estimate	Standard Error	
Age at Onset of Perfectionism	-0.094	0.037	0.011 *	-0.0026	0.043	0.951
Global Childhood Rigidity	0.422	0.251	0.093	0.754	0.314	0.016 *
Current Eating Disorder at Time of Assessment	0.572	0.269	0.033 *	0.455	0.333	0.172
Age at Time of Assessment	0.039	0.012	0.001 *	0.0039	0.017	0.813
Age at Onset of AN	0.0043	0.032	0.895	0.021	0.041	0.603

Odds Ratio Estimates (RAN as Referent)						
Effect	PAN		BAN			
	Point Estimate	95% Confidence Interval	Point Estimate	95% Confidence Interval	Point Estimate	95% Confidence Interval
Age at Onset of Perfectionism	0.910 *	0.846-0.979	0.997	0.917-1.085		
Global Childhood Rigidity (2 or 0)	1.525	0.932-2.495	2.127 *	1.149-3.936		
Current Eating Disorder at Time of Assessment	1.772 *	1.045-3.004	1.576	0.820-3.027		
Age at Time of Assessment	1.040 *	1.016-1.065	1.004	0.972-1.037		
Age at Onset of AN	1.004	0.942-1.070	1.021	0.943-1.106		

* significant at $p < .05$

* significant ratio

TABLE 5

PAN as Referent and Odds Ratio Estimates

PAN as Referent						
Variable	Logit (RAN/PAN)		p Value	Logit (BAN/PAN)		p Value
	Estimate	Standard Error		Standard	Standard Error	
Age at Onset of Perfectionism	0.094	0.037	0.011*	0.091	0.046	0.047*
Global Childhood Rigidity (2 or 0)	-0.422	0.251	0.093	0.333	0.331	0.314
Current Eating Disorder at Time of Assessment	-0.572	0.269	0.033*	-0.117	0.356	0.741
Age at Time of Assessment	-0.039	0.012	0.001*	-0.035	0.017	0.034*
Age at Onset of AN	-0.0043	0.032	0.895	0.017	0.038	0.661

Odds Ratio Estimates (PAN as Referent)						
Effect	RAN		BAN		95% Confidence Interval	
	Point Estimate	95% Confidence Interval	Point Estimate	95% Confidence Interval		
Age at Onset of Perfectionism	1.099*	1.021-1.181	1.096*	1.001-1.199		
Global Childhood Rigidity (2 or 0)	0.656	0.401-1.073	1.395	0.730-2.667		
Current Eating Disorder at Time of Assessment	0.564*	0.333-0.957	0.889	0.443-1.785		
Age at Time of Assessment	0.962*	0.939-0.985	0.965*	0.934-0.997		
Age at Onset of AN	0.996	0.934-1.061	1.017	0.943-1.096		

* significant at $p < .05$

* significant ratio