Influence of Overanxious Disorder of Childhood on the Expression of Anorexia Nervosa

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ABSTRACT

Objective: Childhood anxiety often precedes the onset of anorexia nervosa (AN) and may mark a liability to the emergence of an eating disorder for some women. This study investigates the prevalence of overanxious disorder (OAD) among women with AN and explores how OAD impacts AN symptoms and personality traits.

Method: Participants were 637 women with AN who completed an eating disorders history, the Structured Clinical Interview for DSM-IV Axis I Disorders, and assessments for childhood anxiety, eating disorder attitudes, and associated personality traits.

Results: Of 249 women (39.1%) reporting a history of OAD, 235 (94.4%) met criteria for OAD before meeting criteria for AN. In comparison to those without OAD, women with AN and OAD self-reported more extreme personality traits and attitudes and they engaged in more compensatory behaviors.

Conclusion: Among individuals with AN, those entering AN on a pathway via OAD present with more severe eating disorder pathology.

Keywords: anorexia nervosa; overanxious disorder; genetic; childhood anxiety; social phobia; comorbid anxiety disorders; panic disorder; anxiety disorder; twin studies; agoraphobia

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Introduction

Clinical and epidemiological studies indicate that a substantial portion of women with anorexia nervosa (AN) report lifetime comorbid anxiety disorders. Across studies, the lifetime prevalence of at least one anxiety disorder among patients with AN ranged between 23 and 54%. Comorbid anxiety disorders occur across AN subtypes with approximately equal frequency. Generalized anxiety disorder, obsessive compulsive disorder, and social phobia are the most common comorbid anxiety disorders reported, and they occur significantly more frequently in individuals with AN than in controls.

Anxiety disorders commonly precede the onset of the eating disorder and persist even after long-term weight restoration suggesting a more fundamental underlying anxious trait that is independent of nutritional state. Twin studies have established a substantial contribution of genetic factors to anxiety disorders with heritability estimates for both broadly defined generalized anxiety disorder and panic disorder around 40%. Similarly, twin studies examining the contribution of genetic factors in the development of eating disorders report moderate contributions of additive genetic factors. The heritability of AN has been estimated to be between 33 and 84%, and the heritability of BN between 28 and 83% with the remaining variance in liability to both disorders attributable to individual specific environmental factors, and with negligible impact of shared environmental
factors. In short, there is considerable evidence that risk for eating disorders is influenced by genetic factors.

Twin studies also suggest that eating disorders and anxiety disorders may have shared genetic transmission. Keel et al.\textsuperscript{14} examined the comorbidity of anxiety and eating disorders in monozygotic twins who were discordant for eating disorder symptoms. They found that twins with eating disorder symptoms were significantly more likely to have an unaffected cotwin with an anxiety disorder, suggesting a shared transmission for anxiety and eating disorders. Silberg and Bulik\textsuperscript{15} examined longitudinally reported data in teenage twin girls to identify common genetic liability among anxiety, depression, and eating disorder symptoms in early and late adolescence. Using multivariate genetic modeling, they identified a common genetic factor that influences the liability of childhood anxiety, separation anxiety, depression, and eating problems which remains influential throughout development. They also identified a genetic factor associated with early onset eating problems, but not associated with later eating problems or any mood disturbance. These findings suggest that anxiety and eating disturbances may share a common genetically influenced liability. However, that particular genetic liability may be only one of a variety of pathways to developing an eating disorder.

Although shared liability between anxiety disorders and eating disorders has been suggested, and the onset of anxiety disorders often predates the onset of eating disorders, very little attention has been directed to understanding the impact that childhood anxiety disorders have on the subsequent expression and development of AN. Therefore, in a large phenotypically well-characterized sample of individuals with AN we (1) report the prevalence of overanxious disorder (OAD), (2) explore the impact of OAD on the expression and severity of eating disorder symptoms and personality traits, and (3) explore the relation between OAD and other anxiety disorders in a sample of women with AN.

Method
Participants

Participants were recruited for a multisite international Price Foundation Genetic Study of Eating Disorders focusing on individuals with AN and their parents (“AN Trios”). This study was designed to identify susceptibility loci involved in the risk for eating disorders. Informed consent was obtained from all study participants, and all sites received approval from their local institutional review board.

Male and female probands affected with AN were recruited from nine sites in North America and Europe including Pittsburgh (W.H.K), New York (K.A.H.), Los Angeles (M.S.), Toronto (A.S.K., D.B.W.), Munich (M.M.F.), Pisa (A.R.), Fargo (J.M.), Baltimore (H.B., S. C.), and Tulsa (C.J.), between 2000 and 2003. Probands were required to meet the following criteria: (a) modified DSM-IV (APA, 1994) lifetime diagnosis of AN, with or without amenorrhea; (b) low weight that is/was less than the fifth percentile of body mass index (BMI) for age and gender on the chart of a National Health and Nutrition Examination Survey epidemiologic sample\textsuperscript{16}; (c) onset before the age of 25 years; (d) weight that is/was controlled through restricting and/or purging, which includes vomiting, use of laxatives, diuretics, ememas, suppositories, or ipecac; (e) age between 13 years and 65 years; (f) Caucasian (one grandparent from another racial or ethnic group was acceptable); and (g) study diagnostic criteria were met at least 3 years before study entry. This last inclusion criterion ensured that AN individuals who were unlikely to develop binge eating were appropriately classified, as research has shown that most binge eating develops within the first 3 years of illness in AN.\textsuperscript{17–21} Potential participants were excluded if they reported a maximum BMI since puberty, $>27$ kg/m$^2$ for females and $>27.8$ kg/m$^2$ for males. This exclusion of individuals who were overweight or obese was designed to increase sample homogeneity.

Measures and Interviews
Demographic and Clinical Variables. Data relative to current age, age at onset, and current, minimum, and maximum BMI were included in the analyses.

Eating Disorder Diagnoses and Associated Features. Along the lines of Lilenfeld et al.,\textsuperscript{22} Kaye et al.,\textsuperscript{2} and Godart et al.,\textsuperscript{3} this study used lifetime histories of eating disorder behaviors in the categorization of eating disorder subtypes to capture subtype crossover for more accurate classification. Lifetime histories of eating disorders and the presence or absence of eating disorder behaviors (e.g., dieting, binge eating, purging) in probands were assessed with the Structured Inventory for Anorexia Nervosa and Bulimic Disorders (SIAB);\textsuperscript{23} and with an expanded version of Module H of the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID).\textsuperscript{24} The training procedures for the SIAB and SCID have been described in detail elsewhere.\textsuperscript{25} Interviewers were mental health professionals with at least a master-level degree (most current interviewers were Ph.D. psychologists) who underwent centralized training and certification on all study interviews.

On the basis of detailed clinical interview data, participants were subtyped as follows: AN, restricting subtype
The Structured Clinical Interview for DSM-IV (SCID I).24 Other anxiety disorders were assessed with algorithms constructed to ensure compatibility across instruments. Other anxiety disorders were assessed with the Structured Clinical Interview for DSM-IV (SCID I).24 OAD was chosen as our target disorder for two reasons. First, the SCID only assessed GAD in the past 6 months and we were most interested in childhood onset anxiety. Second, the prevalence of SAD was too low to yield meaningful comparisons.

Personality and Character Traits. Temperament and character dimensions were measured with the Temperament and Character Inventory (TCI).29 Perfectionism was assessed with the Frost Multidimensional Perfectionism Scale (MPS),30 and trait levels of anxiety were assessed with the State-Trait Anxiety Inventory (STAI Form Y-1).31 OCD was assessed using the Schedule for Affective Disorders and Schizophrenia - Lifetime version (SADS-L, as modified by Merikangas et al.27 for DSM-III-R diagnoses) in 90% of the sample and by the K-SADS28 (modified for retrospective reporting) in 10% of the sample. Diagnostic algorithms were constructed to ensure compatibility across instruments. Other anxiety disorders were assessed with the YBOCS32 and the Yale-Brown-Cornell Eating Disorder Scale (YBC-EDS).33 The Y-BOCS is a semistructured interview designed to assess presence and severity of obsessive thoughts and compulsive behaviors typically found among individuals with obsessive compulsive disorder. The YBC-EDS assesses obsessive-compulsive features specific to eating disorders (e.g., those related to food, eating, weight, and exercise). The Revised NEO Personality Inventory (NEO PI-R)34 is an assessment of personality based on the five-factor model of personality, and it measures the interpersonal, motivational, emotional, and attitudinal styles of adults and adolescents.

Statistical Analyses

All statistical analyses were conducted using SAS/STAT® 9.1 software.35 To determine the prevalence of OAD in individuals with eating disorders across subtypes, chi-square tests were used to test between-group differences. χ² tests were also used to determine differences between the OAD and no OAD group in prevalence of anxiety disorders. In order to assess the effects of OAD independent of any other anxiety disorder, the presence or absence of any other anxiety disorder was entered as a covariate in all of the following analyses. Differences between the OAD and no OAD groups in prevalence of the various eating disorder related behaviors was assessed using logistic regressions. Differences between the groups in all continuous measures were determined using analysis of variance with eating disorder subtype entered as an additional covariate. All p-values were corrected for multiple tests using the false discovery rate (FDR).36

Results

A total of 744 participants met criteria for a lifetime diagnosis of AN using the SCID and SIAB. Of those, 13 (1.7%) were males and were excluded from analyses due to small sample size. Seventy participants who were not assessed for OAD or had missing anxiety disorders data were removed from the analysis. An additional 24, who were determined to have a lifetime diagnosis of separation anxiety disorder (SAD), but not OAD were also not included. The resulting sample size was 637.

Prevalence of Overanxious Disorder

Two hundred forty-nine women (39.1%) met criteria for OAD. Significant differences emerged in the prevalence of OAD across AN subtypes (RAN = 32.0%, PAN = 43.8%, BAN = 40.8%, ANBN = 49.0%; χ² = 11.5, df = 3, p = .009). Specifically, the PAN and ANBN groups had significantly higher prevalence of OAD than the RAN group. Of the women with OAD, 235 (94.4%) met criteria for OAD before they met criteria for AN. Six (2.4%) had AN at least 1 year prior to the onset of OAD and eight (3.2%) had onset of both AN and OAD in the same year. There was no difference in the age of onset of AN between those with OAD and those without OAD. For those with OAD, there was no difference in the age of onset of eating disorders between those with other anxiety disorders and those without.

Eating Disorder Symptoms and Features

As shown in Table 1, women with a history of OAD more often reported the use of appetite suppressants (χ² = 16.60, p < .001), vomiting (χ² = 20.13, p < .001), enemas (χ² = 8.55, p = .008), ipecac (χ² = 10.45, p = .004), diuretics (χ² = 8.30, p = .009), and laxatives (χ² = 9.97, p = .004) to control weight. In addition, women with histories of OAD reported longer total duration of eating disorder symptoms than those without OAD (F = –7.90, p = .01). They also reported greater EDI body dissatisfaction (F = 11.42, p = .003), drive for thinness (F = 10.22, p = .004), bulimia (F = 11.31,
p = .003), and YBC-EDS food preoccupations (F = 5.65, p = .03) (see Table 2).

**Personality and Temperament**

In comparison to AN women without OAD, women with OAD reported greater TCI harm avoidance (F = 17.23, p < .001) and persistence (F = 9.36, p = .006), STAI trait anxiety (F = 28.84, p < .001), and lower TCI self-directedness (F = 7.81, p = .01) and novelty seeking, (F = 5.68, p = .03) (Table 2).

In comparison to AN women without OAD, those with OAD also reported more perceived MPS parental criticism (F = 36.24, p < .001), higher parental expectations (F = 11.61, p = .003), higher personal standards (F = 11.93, p = .003), greater concern over mistakes (F = 32.27, p < .001), and greater doubts about actions (F = 25.56, p < .001) (Table 2). Also, women with AN and OAD reported lower NEO-PI R extraversion (F = 5.34, p = .04) and higher neuroticism (F = 10.13, p < .001) than those without a history of OAD.

**OAD and Other Anxiety Disorders**

Individuals with OAD were more likely than those without OAD to develop additional anxiety disorders (Table 3). More participants with OAD were also diagnosed with generalized anxiety disorder (χ² = 42.58, p < .001), panic disorder (χ² = 9.33, p = .002), obsessive compulsive disorder (χ² = 40.74, p < .001), social phobia (χ² = 37.11, p < .001), or specific phobia (χ² = 17.84, p < .001). The prevalence of agoraphobia was not related to OAD history; however, agoraphobia was relatively uncommon in this sample. For the participants who developed another anxiety disorder, the presence of OAD was also associated with earlier age of onset for generalized anxiety disorder (t = 2.79, p = .01) and obsessive compulsive disorder (t = 3.78, p < .001), but not for the other anxiety disorders.

**Conclusion**

To our knowledge, this is the first study to report estimates of the prevalence of overanxious disorder of childhood among women with AN. Although OAD no longer exists in the DSM-IV, we have benefited from the inclusion of that diagnosis in our assessment in order to explore more carefully patterns of early onset of anxiety disorders and AN.

Of 637 women with AN, 39% met criteria for OAD. In this study, the prevalence of OAD varied across the diagnostic subtypes of AN, with OAD more common among PAN and ANBN. This finding is inconsistent with some studies of comorbid anxiety which did not find differences across eating disorder subtypes, but consistent with others. Godart et al. found that individuals with RAN and ANBN were more likely than those with BN to have a lifetime diagnosis of OCD, and RAN reported significantly higher prevalences of current OCD and GAD than those with BN. However, in that study, PAN was not included in the comparisons and prevalence of childhood anxiety disorders was not given.

Our findings suggest that women with AN who engage in purging behaviors are more likely than those who do not purge to have had OAD in childhood. Strober theorized that women with anxiety disorders and AN may have a similar liability that manifests as a heightened sensitivity to fear conditioning with resistance to extinction. Applying that theory, those women with AN who had preexisting OAD may simply be more vulnerable to this
excessive fear conditioning, have more intense anxiety responses, and turn to purging as an immediate intervention to reduce the extreme emotional distress. An alternative explanation is that those women with OAD may have a generalized heightened sensitivity to fear conditioning that, with the emergence of AN, is complicated by the addition of a “weight based” specific fear. The additive impact of the general and specific foci for the extreme fear conditioning may result in a more intense perceived need to reduce anxiety through purging. Another possible explanation is that anxious symptoms may be related to altered striatal dopamine function in AN. A contributing component of anxiety in AN individuals is a height-

**TABLE 2.** Means and results from analysis of variance assessing differences in personality variables and eating disorder attitudes between those with OAD and those without*

<table>
<thead>
<tr>
<th>Variable</th>
<th>No OAD, (N = 388) Mean (SD)</th>
<th>OAD, (N = 249) Mean (SD)</th>
<th>F-Value (p-Value)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Eating disorder features</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ED age of onset</td>
<td>16.1 (2.7)</td>
<td>16.1 (3.0)</td>
<td>0.19 (.68)</td>
</tr>
<tr>
<td>Lowest preferred weight</td>
<td>37.3 (8.1)</td>
<td>36.2 (8.0)</td>
<td>0.48 (.51)</td>
</tr>
<tr>
<td>Highest BMI</td>
<td>21.3 (2.4)</td>
<td>21.4 (2.4)</td>
<td>0.91 (.39)</td>
</tr>
<tr>
<td>Lowest BMI</td>
<td>13.8 (1.9)</td>
<td>13.7 (1.9)</td>
<td>0.00 (.95)</td>
</tr>
<tr>
<td>ED duration</td>
<td>8.8 (7.4)</td>
<td>10.9 (7.6)</td>
<td>7.90 (.01)</td>
</tr>
<tr>
<td>Lowest preferred daily caloric intake (kcal)</td>
<td>519.0 (328.9)</td>
<td>413.6 (290.0)</td>
<td>4.21 (.06)</td>
</tr>
<tr>
<td>Temperament and Character Inventory</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cooperativeness</td>
<td>34.5 (5.6)</td>
<td>35.1 (6.1)</td>
<td>2.18 (.82)</td>
</tr>
<tr>
<td>Harm avoidance</td>
<td>20.0 (7.9)</td>
<td>24.2 (6.6)</td>
<td>17.23 (&lt;.001)</td>
</tr>
<tr>
<td>Novelty seeking</td>
<td>16.4 (7.1)</td>
<td>15.3 (7.0)</td>
<td>5.86 (.03)</td>
</tr>
<tr>
<td>Reward dependence</td>
<td>16.3 (3.9)</td>
<td>16.9 (4.0)</td>
<td>3.24 (.10)</td>
</tr>
<tr>
<td>Persistence</td>
<td>6.0 (1.9)</td>
<td>6.5 (1.6)</td>
<td>9.36 (.006)</td>
</tr>
<tr>
<td>Self-directedness</td>
<td>28.4 (8.9)</td>
<td>24.9 (9.0)</td>
<td>7.81 (.01)</td>
</tr>
<tr>
<td>Self-transcendence</td>
<td>13.7 (6.8)</td>
<td>14.7 (6.0)</td>
<td>0.60 (.48)</td>
</tr>
<tr>
<td>Yale-Brown-Cornell Eating Disorder Scale</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Worst motivation to change</td>
<td>18.6 (5.3)</td>
<td>19.1 (5.5)</td>
<td>0.18 (.68)</td>
</tr>
<tr>
<td>Worst rituals</td>
<td>12.3 (2.6)</td>
<td>13.6 (2.3)</td>
<td>4.33 (.06)</td>
</tr>
<tr>
<td>Worst preoccupations</td>
<td>12.8 (2.5)</td>
<td>13.8 (2.2)</td>
<td>5.65 (.03)</td>
</tr>
<tr>
<td>Yale-Brown-Obsessive Compulsive Scale</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Obsessions</td>
<td>6.6 (6.5)</td>
<td>10.1 (5.8)</td>
<td>2.02 (.19)</td>
</tr>
<tr>
<td>Compulsions</td>
<td>7.2 (6.7)</td>
<td>10.7 (6.2)</td>
<td>.056 (.49)</td>
</tr>
<tr>
<td>Multidimensional Perfectionism Scale</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Concern over mistakes</td>
<td>32.2 (9.1)</td>
<td>38.1 (6.7)</td>
<td>32.27 (&lt;.001)</td>
</tr>
<tr>
<td>Doubts about actions</td>
<td>12.5 (3.8)</td>
<td>15.0 (3.4)</td>
<td>25.56 (&lt;.001)</td>
</tr>
<tr>
<td>Organization</td>
<td>25.1 (5.5)</td>
<td>25.9 (4.9)</td>
<td>2.59 (.15)</td>
</tr>
<tr>
<td>Parental criticism</td>
<td>9.9 (4.4)</td>
<td>13.0 (4.9)</td>
<td>36.24 (&lt;.001)</td>
</tr>
<tr>
<td>Parental expectations</td>
<td>13.9 (5.7)</td>
<td>16.4 (6.0)</td>
<td>11.61 (.003)</td>
</tr>
<tr>
<td>Personal standards</td>
<td>28.1 (5.6)</td>
<td>30.2 (4.4)</td>
<td>11.93 (.003)</td>
</tr>
<tr>
<td>Eating Disorders Inventory-2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Body dissatisfaction</td>
<td>17.1 (7.0)</td>
<td>20.3 (6.8)</td>
<td>11.42 (.003)</td>
</tr>
<tr>
<td>Bulimia</td>
<td>2.6 (4.4)</td>
<td>4.3 (5.5)</td>
<td>11.61 (.003)</td>
</tr>
<tr>
<td>Drive for thinness</td>
<td>14.6 (5.9)</td>
<td>16.9 (5.1)</td>
<td>10.22 (.004)</td>
</tr>
<tr>
<td>State-Trait Anxiety Inventory Form Y-1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Trait anxiety</td>
<td>50.4 (13.0)</td>
<td>58.3 (12.8)</td>
<td>28.74 (&lt;.001)</td>
</tr>
<tr>
<td>The Revised NEO Personality Inventory</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Extraversion</td>
<td>105.7 (22.1)</td>
<td>98.9 (22.7)</td>
<td>5.34 (.04)</td>
</tr>
<tr>
<td>Neuroticism</td>
<td>105.7 (25.3)</td>
<td>120.8 (21.7)</td>
<td>10.13 (&lt;.001)</td>
</tr>
</tbody>
</table>

*The presence or absence of any other anxiety disorder was entered into all models as a covariate. Eating disorder subtype was also entered as a covariate for all models except those predicting eating disorder features.

**TABLE 3.** Frequency of anxiety disorders by presence or absence of a history of OAD: % (N)*

<table>
<thead>
<tr>
<th>Prevalence of Anxiety Disorders</th>
<th>Means (std) Age of Onset of Anxiety Disorders</th>
</tr>
</thead>
<tbody>
<tr>
<td>No OAD OAD</td>
<td>No OAD OAD t-Score (p-Value)</td>
</tr>
<tr>
<td>Agoraphobia</td>
<td>2.3 (9) 3.7 (11) 0.94 (.33) 15.8 (8.8) 21.3 (8.3) −1.30 (.25)</td>
</tr>
<tr>
<td>Generalized anxiety Disorder</td>
<td>7.2 (28) 25.9 (64) 42.58 (&lt;.001) 15.6 (6.7) 11.3 (6.7) 2.79 (.01)</td>
</tr>
<tr>
<td>Panic disorder</td>
<td>10.1 (39) 18.6 (46) 9.33 (.002) 20.6 (6.8) 19.4 (8.8) 0.70 (.49)</td>
</tr>
<tr>
<td>Obsessive compulsive Disorder</td>
<td>42.9 (165) 68.8 (170) 40.74 (&lt;.001) 13.6 (5.6) 11.1 (5.9) 3.78 (&lt;.001)</td>
</tr>
<tr>
<td>Social phobia</td>
<td>13.5 (52) 33.9 (83) 37.11 (&lt;.001) 12.5 (4.8) 10.8 (4.8) 1.95 (.07)</td>
</tr>
<tr>
<td>Specific phobia</td>
<td>5.4 (21) 15.5 (38) 17.84 (&lt;.001) 8.7 (6.9) 9.3 (7.2) −0.28 (.80)</td>
</tr>
</tbody>
</table>

*Comparison made by chi-square. Also, means (std) of age of onset of anxiety disorders by presence or absence of a history of OAD.
ened sensitivity to uncertainty, resulting in extreme distress reduction strategies such as purging.

The presence of OAD in this sample was associated with a more severe and complex clinical presentation. Previous research has not consistently demonstrated a relationship between anxiety disorders and increased severity of eating disorder symptoms. In contrast, our explicit focus on OAD with onset by definition in childhood identified a consistent and robust association between anxiety and severity of eating disorder symptoms and personality traits. Although all women with AN displayed the characteristic personality profiles expected in this population (e.g., harm avoidance, persistence, perfectionism, and lower self-directedness), those with a history of OAD displayed a profile that was magnified. The same pattern held for eating disorder behaviors, with those with a history of OAD engaging in more problematic weight control practices and displaying greater body dissatisfaction, higher drive for thinness, more eating preoccupation, and longer duration of illness.

OAD was also associated with the presence of additional anxiety disorders. As may be expected, those individuals with a history of OAD were significantly more likely to have GAD, OCD, specific phobia, social phobia, and panic disorder. Agoraphobia was infrequently endorsed by participants and did not differ significantly between women with or without OAD. The presence of OAD was also associated with younger ages of onset for GAD and OCD than those without OAD who developed these conditions. Collectively, these observations suggest that childhood OAD is a pernicious disorder that is associated with greater severity and longer duration of eating disorder.

The results of this investigation should be viewed within the context of several limitations. The OAD diagnoses and age at onset estimates were made retrospectively and subject to recall biases inherent in that approach. Individuals with anxiety disorders persisting into adulthood or who developed additional anxiety disorders later in life may have been more sensitized to recognize anxiety symptoms in childhood which could inflate the association between childhood onset and other anxiety disorders. Another limitation is that the SCID assesses only current GAD, so many cases of remitted GAD are missed. Therefore, many participants may, at an earlier time, have met criteria for GAD and would have gone undetected if the GAD had remitted at the time of assessment. Furthermore, the SCID screen for GAD is limited in that people who are always anxious may not report having been more anxious in the past 6 months. One of the strengths of studying OAD is that it captures those individuals with an early pervasive anxiety, even if the symptoms remitted at the time of assessment.

In addition, the distinction of OAD as a diagnosis of childhood is somewhat artificial as many of the patients reported the onset of other anxiety disorders close in time to their childhood anxiety disorder. In the DSM-IV the diagnosis of OAD was subsumed by the diagnosis of GAD, with the only remaining distinction being that children can present with fewer symptoms to meet diagnostic criteria. Also noteworthy, this study is part of multicenter genetic study, so the participants were recruited from populations of primarily European descent to ensure sample homogeneity that is critical in genetic studies. This homogeneity limits the generalizability of these findings to non-European groups.

An OAD pathway may represent one entrée into AN, which portends persistent and pervasive anxious symptoms, obsessive and perfectionistic personality styles, and later anxiety disorders. Although these observations are based on retrospective recall, they highlight the importance of early detection of OAD as a potential means of averting the emergence of both later anxiety and eating disorders symptoms.

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