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Delineation of Differential Temporal Relations between Specific Eating and Anxiety Disorders

Julia D. Buckner[a,*], **Jose Silgado**[a], and **Peter M. Lewinsohn**[b]

[a] Department of Psychology, Louisiana State University, 236 Audubon Hall, Baton Rouge, LA 70803, USA

[b] Oregon Research Institute, 1715 Franklin Blvd, Eugene, OR 97401, USA

Abstract

This study examined the temporal sequencing of eating and anxiety disorders to delineate which anxiety disorders increase eating disorder risk and whether individuals with eating disorders are at greater risk for particular anxiety disorders. The sample was drawn from the Oregon Adolescent Depression Project. Temporal relations between specific eating and anxiety disorders were examined after controlling for relevant variables (e.g., mood disorders, other anxiety disorders) over 14 years. After excluding those with anorexia nervosa (AN) in adolescence (T1), OCD was the only T1 anxiety disorder to predict AN by age 30 (T4). No T1 anxiety disorder was associated with T4 bulimia nervosa (BN). Although T1 AN did not increase risk of any T4 anxiety disorder, T1 BN appeared to increase risk for social anxiety and panic disorders. Evidence that eating disorders may have differential relations to particular anxiety disorders could inform prevention and treatment efforts.

Keywords

anxiety disorders; eating disorders; temporal relations; risk factors

Introduction

There are high rates of comorbidity between anxiety and eating disorders. To illustrate, 55–60% of those with anorexia nervosa (AN) and 57–68% of people with bulimia nervosa (BN) have experienced an anxiety disorder (Bulik et al., 1997; Kaye et al., 2004). Relatives of those with AN also have a higher prevalence of anxiety disorders compared to relatives of controls (Strober et al., 2007). Further, eating disorders (ED) and anxiety disorders may share a genetic link (Keel et al., 2005; Kendler et al., 1995; Rowe et al., 2002; Silberg & Bulik, 2005). There may also be differential relations between particular anxiety disorders and particular ED. For instance, compared to individuals without ED, individuals with AN exhibit higher rates of overanxious disorder (OAD), separation anxiety, panic disorder, and

*Corresponding Author: Telephone: 1-225-578-4096, Fax: 1-225-578-4125, jbuckner@lsu.edu.

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obsessive compulsive disorder (OCD) whereas those with BN exhibit higher rates of OAD and social anxiety disorder (SAD) (Bulik et al., 1997).

Anxiety disorders are posited to be a risk factor for ED (Bulik et al., 1997). Yet, it remains unclear whether ED increase anxiety disorder vulnerability. The identification of sequelae to ED is not trivial because although ED risk may peak in adolescence and early 20's (Heatherton et al., 1997) individuals with ED may be vulnerable to developing other types of psychopathology such as anxiety disorders.

We know of no studies directly testing whether particular anxiety disorders are in fact risk factors for ED and whether particular ED increase anxiety disorder risk. Garber and Hollon (1991) outline three criteria for causal attribution that have traditionally been recognized in psychopathology risk research. First, the proposed risk factor must be correlated with the outcome. Second, the proposed risk factor must demonstrate temporal precedence. Third, the relation between the risk factor and outcome variable must be non-spurious (i.e., not due to a third variable or set of variables).

Risk Criterion 1: Review of Co-occurrence Data

Data suggest some particular anxiety disorders appear to co-occur with specific ED. Although there is some data suggesting OCD is related to BN (e.g., Hudson et al., 2007), it has more consistently been linked to AN (Bulik et al., 1997; Deep et al., 1995; Fahy et al., 1993; Godart et al., 2003; Godart et al., 2000; Rastam et al., 1995; Thornton & Russell, 1997). Concerning SAD, although some data suggest SAD can co-occur with AN (Deep et al., 1995; Godart et al., 2003; Godart et al., 2000; Kaye et al., 2004; Lilenfeld et al., 1998), SAD appears more consistently related to BN. Not only does SAD tend to be comorbid with BN (Brewerton et al., 1995; Godart et al., 2000; Schwalberg et al., 1992), lifetime prevalence of SAD occurs more frequently in BN patients than AN (restrictive subtype) patients (Iwasaki et al., 2000). Among German in-patients, patients with BN (but not AN) reported higher levels of social anxiety than patients with anxiety disorders (Grabhorn et al., 2006). Sub-clinical social anxiety in both patients with BN and nonclinical controls are associated with higher levels of bulimic behaviors than in patients with AN (Hinrichsen et al., 2003). Similarly, women with BN and non-treatment seeking women with high levels of thoughts, feelings, and behaviors associated with ED scored higher in public self-consciousness and social anxiety measures than women without ED-related thoughts, etc. (Striegel-Moore et al., 1993).

Data regarding generalized anxiety disorder (GAD), on the other hand, are less consistent. Although two studies found higher rates of GAD in individuals with AN than controls (Godart et al., 2003; Walters & Kendler, 1995), another found no such difference (Rastam et al., 1995). Regarding BN, although some have noted higher rates of GAD in BN subjects than controls (Garfinkel et al., 1995; Godart et al., 2003; Kendler et al., 1991; Schwalberg et al., 1992; Zaider et al., 2002), other studies have failed to replicate this finding (Hudson et al., 1987; Lilenfeld et al., 1998). Two clinical studies found no significant differences in rates of GAD between BN and AN patients (Iwasaki et al., 2000; Kaye et al., 2004).

Individuals with AN do not appear to demonstrate greater rates of panic disorder (Halmi et al., 1991; Hudson et al., 2007; Lilenfeld et al., 1998; Rastam et al., 1995). Yet panic disorder's relation to BN is less clear. Panic disorder seems to be significantly higher among those with BN compared to controls (Bushnell et al., 1994; Garfinkel et al., 1995; Godart et al., 2003; Kendler et al., 1991; Zaider et al., 2002), yet no differences were found between patients with BN compared to patients with SAD (Schwalberg et al., 1992) or between BN and AN patients (Iwasaki et al., 2000; Kaye et al., 2004).

Risk Criterion 2: Review of Temporal Relations Evidence

Little prospective work has examined the temporal sequencing of specific ED and anxiety disorders, with the majority of work in this area relying on retrospective accounts of age of onset. Regarding the contention that some anxiety disorders may increase ED risk, among patients with both AN and OCD, mean age of onset of OCD tends to be earlier than that of AN (Bulik et al., 1997; Kaye et al., 2004; Thornton et al., 1997), although some reports found similar age of onset of OCD and AN among patients with both disorders (Fahy et al., 1993; Godart et al., 2003). Higher social anxiety predicted higher bulimic behaviors over a 7-month period (Gilbert & Meyer, 2005). SAD age of onset tends to predate age of onset for both AN and BN among patients with SAD and an ED (Deep et al., 1995; Godart et al., 2003; Godart et al., 2000; Schwalberg et al., 1992). GAD was associated with increased risk for onset of ED 10 months later (Zaider et al., 2002) and GAD onset occurred prior to BN onset among patients with GAD and BN (Schwalberg et al., 1992).

Concerning the hypothesis that ED may increase anxiety disorder vulnerability, our group found that individuals with ED (AN and BN grouped together) had an elevated rate of nonaffective disorders (e.g., anxiety disorders, substance use disorders, conduct disorder) compared with a no disorder group 6 years later (Lewinsohn et al., 2000). Regarding particular anxiety disorders, panic disorder onset tends to follow that of both BN and AN (Bulik et al., 1997; Godart et al., 2003; Venturello et al., 2002). Yet, bulimia did not predict subsequent GAD or panic disorder 10 months later in an adolescent sample (Zaider et al., 2002).

Taken together, these data suggest differential temporal patterns based on type of anxiety disorder and type of ED under investigation. Specifically, these data indicate that: (1) OCD onset may predate AN onset, (2) SAD onset may predate AN and BN onset, and (3) GAD may predate BN onset among patients with co-occurring anxiety and eating disorders. Further, these data suggest AN and BN onset may predate panic disorder onset.

Risk Criterion 3: Review of Non-Spuriousness Evidence

Less work has examined the non-spurious criterion regarding the link between particular anxiety disorders and specific ED. When accounting for comorbidity among the anxiety disorders, the risk for AN was significantly higher among those with OAD and OCD, whereas the risk for BN was significantly increased among those with OAD and SAD (Bulik et al., 1997). After controlling for other anxiety disorders and depression, PTSD and SAD remained significantly related to disordered eating among a sample of patients from an anxiety disorders clinic (Becker et al., 2004). When controlling for the effects of depression, the strength of observed prospective relations between baseline anxiety disorders and ED 10 months later was reduced (Zaider et al., 2002). However, Zaider and colleagues only examined the temporal relations between GAD, panic disorder, and bulimia. Further work is necessary to examine temporal relations between specific ED and anxiety disorders controlling for comorbidity between eating, anxiety, and mood disorders (Hudson et al., 2007; Kessler et al., 2005b; Lewinsohn et al., 2000; Walters et al., 1995). Further, given sex differences in anxiety disorders (Lewinsohn et al., 1998) and ED rates (Hudson et al., 2007; Lewinsohn et al., 2000), it is also important to rule-out sex as a possible “third variable” that may account for the observed relations between anxiety disorders and ED. Yet, very few studies accounts for this variable (Hudson et al., 2007; Zaider et al., 2002).

Summary

Thus, when considered individually, results from the extant literature paint a somewhat unclear picture of whether particular anxiety disorders are associated with greater ED risk and vice-versa. Yet when considered in concert, these studies begin to suggest the following.

In regards to OCD, data suggest that OCD (1) is related to AN and BN, (2) tends to prior to AN onset in comorbid individuals, and (3) remains only significantly related to AN (not BN) in multivariate analyses. In regard to SAD, data suggest that SAD (1) is related to BN (but not necessarily AN), (2) onsets prior to BN in comorbid individuals, and (3) remains only significantly related to BN (not AN) in multivariate analyses. Concerning panic disorder, prior work suggests that panic disorder (1) is related to BN but not AN and (2) onsets following BN onset in comorbid individuals. GAD does not seem to be especially associated with either AN or BN.

However, knowledge of these relations is limited in several respects. First, little prospective work has been done, with the vast majority of studies relying on retrospective accounts of age on onset (Bulik et al., 1997; Deep et al., 1995; Fahy et al., 1993; Godart et al., 2003; Kaye et al., 2004; Thornton et al., 1997; Venturello et al., 2002). Second, many rely on DSM-III or III-R diagnoses (Bulik et al., 1997; Deep et al., 1995; Fahy et al., 1993; Rastam et al., 1995; Schwalberg et al., 1992; Thornton et al., 1997). Third, although some studies accounted for comorbidity among the anxiety disorders and between anxiety disorders and mood disorders (Becker et al., 2004; Bulik et al., 1997), most do not (Fahy et al., 1993; Godart et al., 2003; Kaye et al., 2004; Thornton et al., 1997). This strategy is problematic given the high rates of co-occurrence among depression with anxiety disorders (Kessler et al., 2005b) and ED (Hudson et al., 2007).

The Present Study

The overarching aim of the present study was to build upon prior work by further elucidating the temporal relations between specific ED and anxiety disorders using data from the Oregon Adolescent Depression Project (OADP; Goodwin et al., 2005; Goodwin et al., 2004; Lewinsohn et al., 1993). Although prior OADP studies have examined the relations between anxiety disorders and ED, the present study extends this work by examining particular ED and anxiety disorders versus grouping all participants with anxiety into one “anxiety disorder group” and all participants with disordered eating into one “ED group” (Lewinsohn et al., 2000; Lewinsohn et al., 1997). Our first goal was to replicate the finding that particular anxiety disorders temporally precede particular ED. As outlined above, it was predicted that among participants without an AN history in adolescence (Time 1; T1), T1 OCD would predict AN by age 30 (Time 4; T4) whereas T1 SAD would predict age 30 BN. Our second goal was to determine whether particular ED increase vulnerability for particular anxiety disorders. It was predicted that among participants without a T1 history of panic disorder, T1 BN would predict T4 panic disorder. We extended prior work by examining these relations prospectively and by testing the non-spuriousness of these relations by controlling for age, sex, other anxiety disorders and major depressive disorder (MDD).

Method

Participants and Procedures

The sample was drawn from the Oregon Adolescent Depression Project (Goodwin et al., 2005; Goodwin et al., 2004; Lewinsohn et al., 1993). Participants were randomly selected from nine senior high schools representative of urban and rural districts in western Oregon. A total of 1,709 adolescents completed T1 assessments between 1987 and 1989, with an overall participation rate of 61%. Half of the T1 sample was female (53.7%), with a mean age of 16.6 years (*SD* = 1.2). Approximately one year later, 1,507 of the participants (88%) returned for the re-administration of the interview and questionnaire (T2). As participants reached their 24th birthday, a third wave of questionnaires and diagnostic interview assessments (T3) was conducted with a selected subset of T2 participants. On the basis of

T1–T2 diagnostic information, three groups were selected for the T3 diagnostic interview: (a) 360 participants with a T2 lifetime history of major depressive disorder, (b) 284 participants with a T2 lifetime history of nonaffective Axis I disorder, and (c) 457 participants with no history of mental disorder at T2. The no-disorder comparison group was representative of the entire group of participants with no mental disorder at T2 (n = 863) in terms of age and gender within age; all participants with non-white ethnicity were invited to participate in the T3 assessment. This sampling strategy was intentional due to the expense of running this longitudinal investigation. Of the 1,101 who were sampled, 941 (85%) participated in the T3 diagnostic interview.

At age 30 (M = 30.6 years, SD = 0.6), the participants who completed the T3 interview were invited to participate in a fourth wave (T4) of data collection. Of the 941 eligible participants, 816 (86.7%) completed the T4 diagnostic interview. The T4 participants (59% women) were primarily Caucasian (59%) and married (53%). Forty-one percent had a bachelor's degree or higher. For T4, the retention rates for the three groups selected for the T3 interview were as follows: 86.5% for individuals in the major depressive disorder group, 82.5% for individuals in the nonaffective Axis I disorder group, and 89.5% for individuals in the no mental disorder group. Significantly higher attrition rates were noted for men than women (16% vs. 11%), as well as for participants with a lifetime history of alcohol use disorders (17% vs. 12% for those with no lifetime alcohol use disorder), and cannabis use disorders (18% vs. 12% for those with no lifetime history of cannabis use disorder).

Given that the age of onset for anxiety and ED peaks in adolescence (Heatherton et al., 1995; Kessler et al., 2005a; Rastam et al., 1995), this report concerns data collected concerning lifetime diagnostic histories at T1 and T4. By age 30, the adults had been assessed on four occasions. This investigation was carried out in accordance to the Declaration of Helsinki and written IRB-approved informed consent was obtained from participants (and guardians, if applicable) to conduct all assessments.

Materials

Participants were interviewed at T1 with a version of the Schedule for Affective Disorders and Schizophrenia for School-Age Children (K-SADS) that combined features of the epidemiologic version (Orvaschel et al., 1982) and the present episode version (K-SADS-P) and included additional items to derive DSM-III-R diagnoses (American Psychiatric Association, 1980). Both versions of the K-SADS have been found to demonstrate adequate psychometric properties (see Ambrosini, 2000). In the present report, lifetime histories of the following disorders at T1 were examined: SAD, panic disorder (with and without agoraphobia), GAD, OCD, specific phobia (SP), OAD, separation anxiety disorder, and major depressive disorder (MDD). Given that diagnoses at the T1 assessment periods were derived using DSM-III-R criteria, in cases where DSM-III and DSM-IV criteria differed, additional data were used to ascertain DSM-IV criteria for the diagnoses of interest (American Psychiatric Association, 1994). Lifetime history of adult T4 diagnoses were derived from a joint administration of the Longitudinal Interval Follow-up Evaluation (1987) and the Structured Clinical Interview for DSM-IV, non-patient version (SCID-I/NP) (1994) to probe for new or continuing psychiatric episodes. A dichotomous variable was created representing the absence ("0") or presence ("1") of each Axis I disorder of interest.

Diagnostic interviewers were carefully selected, trained, and supervised. Interviewers had advanced degrees in a mental health discipline and completed a 70-hour course in diagnostic interviewing. Interrater reliability was evaluated by the kappa statistic (Cohen, 1960). Kappas for the T1 disorders include: current SAD (κ = .57), current simple phobia (κ = .56), lifetime history of SAD (κ = .56), lifetime history of simple phobia (κ = .65), lifetime history of separation anxiety (κ = .76), lifetime history of overanxious (κ = .59), lifetime history of

ED (κ = .66) and lifetime history of dysthymia (κ = .58). Kappas for all other disorders were above .80 (Lewinsohn et al., 1998; Lewinsohn et al., 1997). Prior to conducting interviews, all interviewers were required to demonstrate a minimum kappa of .80 across all symptoms for at least two consecutive training interviews and on one videotaped interview of a participant with evidence of psychopathology.

Data Analysis

To examine the relations between T1 variables of interest (age, sex, and the following T1 psychiatric disorders: MDD, separation anxiety disorder, SAD, panic disorder, OAD, SP, OCD) and T4 ED (AN, BN), multivariate logistic regression analyses were conducted in which each T4 ED was a dependent variable. Separate regressions were conducted for each dependent variable. For the AN model, participants with a T1 lifetime history of AN were excluded from analyses to examine onset of new AN diagnoses. Similarly, for the BN model, participants with a T1 lifetime history of BN were excluded from analyses.

To examine whether T1 ED predicted T4 ANX, a second set of multivariate logistic regression analyses was conducted in which each T4 ANX (SAD, panic disorder, GAD, OCD, SP) was the outcome variable (separation anxiety disorder was not included as it was not assessed at T4). Separate regressions were performed for each anxiety disorder. For each model, participants with a T1 history of the ANX were excluded and the other T1 ANX, T1 MDD, and T1 age and sex were included in the model to examine the non-spuriousness of the relations between each ED and each ANX.

Results

Descriptive Information

Rates of lifetime diagnoses of interest at T1 were as follows: AN (0.3%), BN (0.5%), MDD (18.4%), PD (0.7%), overanxious (1.3%), separation anxiety disorder (4.2%), specific phobia (2.0%), OCD (0.5%), and SAD (1.5%). Rates of lifetime diagnoses of interest at T4 were as follows: AN (0.5%), BN (1.2%), MDD (28.1%), PD (3.5%), GAD (1.1%), specific phobia (2.2%), OCD (0.7%), and SAD (2.1%). Regarding the cross-sectional relationships between T1 ED with T1 depression and anxiety, T1 AN was significantly related only to T1 MDD such that participants with T1 MDD demonstrated higher rates of T1 AN (1.0%) than participants without T1 MDD (0.1%), $\chi^2(1, 1709)=5.76$, $p=.016$. T1 BN was also significantly related to MDD such that participants with T1 MDD demonstrated higher rates of T1 BN (1.9%) than participants without T1 MDD (0.1%), $\chi^2(1, 1709)=17.11$, $p<.001$. However, T1 BN was also significantly related to T1 overanxious disorder such that participants with T1 overanxious disorder demonstrated higher rates of T1 BN (4.5%) than participants without T1 overanxious disorder (0.4%), $\chi^2(1, 1709)=7.95$, $p=.005$. T1 BN was also significantly related to T1 SAD such that participants with T1 SAD demonstrated higher rates of T1 BN (4.0%) than participants without T1 SAD (0.4%), $\chi^2(1, 1709)=6.79$, $p=.009$.

Cross-sectional relations by T4, however, demonstrated a somewhat different pattern. The significant relationship between AN and MDD was reduced to a trend by T4, $\chi^2(1, 816)=3.34$, $p=.065$. T4 AN was only significantly related to T4 OCD such that participants with T4 OCD demonstrated higher rates of T4 AN (8.3%) than participants without T4 OCD (1.0%), $\chi^2(1, 816)=5.84$, $p=.016$. T4 BN remained significantly related to T4 MDD, $\chi^2(1, 816)=8.23$, $p=.004$. T4 BN also remained significantly related to T4 SAD, $\chi^2(1, 816)=5.45$, $p=.020$. However, at T4, BN was also significantly related to T4 PD such that participants with T1 PD demonstrated higher rates of T1 BN (6.8%) than participants without T1 PD (2.1%), $\chi^2(1, 816)=4.98$, $p=.026$.

Relations between T1 Anxiety Disorders and T4 ED

Among participants without a T1 history of AN, multivariate analyses suggest that T1 OCD remained the only significant predictor of T4 AN (Table 1). Among participants without a T1 history of BN, no ANX or depression was significantly related to T4 BN (Table 2).

Relations between T1 ED and T4 Anxiety Disorders

After controlling for T1 age, sex, MDD, and the other anxiety disorders, AN did not predict any T4 ANX or depression (Walds = .00, *p*'s = 1.00, *OR*'s = .00). However, BN significantly predicted panic disorder and SAD, but not GAD, OCD, or specific phobia (Table 3).

Discussion

The current study serves as one of the first known reports to systematically examine (1) whether particular anxiety disorders serve to increase ED risk and (2) whether particular ED serve as risk factors for particular anxiety disorders, using criteria considered to be the gold standard for assessing causality in psychosocial research (Garber et al., 1991). Results support the contention that anxiety disorders and ED appear related (Swinbourne & Touyz, 2007). The present study furthers knowledge regarding the relations between anxiety disorders and ED in several key ways. First, we used longitudinal data to examine temporal sequencing between anxiety disorders and ED. Prior work has often relied on retrospective accounts of age of onset (Bulik et al., 1997; Deep et al., 1995; Fahy et al., 1993; Fairburn et al., 1997; Godart et al., 2003; Godart et al., 2000; Kaye et al., 2004; Venturello et al., 2002). Second, we examined the relations between *particular* anxiety disorders and ED. Prior work tends to not examine particular anxiety disorders or particular ED, but rather uses combined anxiety disorders groups' relations to combined ED groups (Deep et al., 1995; Lewinsohn et al., 2000; Lewinsohn et al., 1997). Given the differential pattern of findings obtained, the practice of combining anxiety disorders may obfuscate the specific relations between particular anxiety disorders and ED. Third, the present study examined relations after controlling for the comorbidity among anxiety disorders and between anxiety and mood disorders.

Anxiety Disorders as Risk Factors for ED

Data from the present study support the contention that anxiety disorders in adolescence may increase ED vulnerability (Bulik et al., 1997). In line with the guidelines considered to be the gold standard for evaluating causality in psychosocial research (Garber et al., 1991), tests of non-spuriousness suggest that when controlling for the variance attributable to other anxiety disorders and depression, only OCD emerged as a unique predictor of subsequent AN. This is consistent with prior work that has consistently found higher rates of OCD amongst AN participants than BN participants (Godart et al., 2003; Godart et al., 2000; Hudson et al., 1983; Laessle et al., 1987; Lilenfeld et al., 1998; Piran et al., 1985; Speranza et al., 2001).

The question arises as to what might account for the specific risk of AN among individuals with OCD. One possibility is that these two disorders may share underlying biological risk factors. However, extant evidence does not consistently support a shared biological diathesis between AN and OCD (Serpell et al., 2002). It may be that both share a common psychological risk factor such as perfectionism. In fact, perfectionism is related to both OCD (Obsessive Compulsive Cognitions Working Group, 1997) and AN. Alternatively, people with OCD may be vulnerable to obsessive thoughts related to body weight and/or body dissatisfaction. Calorie intake may serve as a type of ritual to attempt to neutralize distressing thoughts related to body image. It has also been posited that just as compulsions

are attempts to regulate affect among those with OCD, restrictive anorexic behaviors could be similar attempts at affect regulation (Swinbourne et al., 2007). Unfortunately these theories remain necessarily speculative until empirical tests of these theories are conducted.

ED as Risk Factors for Anxiety Disorders

The present study is among the very few to examine whether ED history in adolescence places individuals at risk for development of other psychopathology. Our data suggest that the experience of BN in adolescence can place people at risk for developing SAD and/or panic disorder in adulthood. This finding is not trivial for several reasons. First, in light of data suggesting that rates of ED tend to peak in college with prevalence rates dropping by 30% 10 years post-college (Heatherton et al., 1995), the finding that these individuals remain at risk for other psychopathology provides additional rationale for the importance of understanding, preventing, and treating ED in adolescence and college. Second, both SAD and panic disorder tend to be chronic disorders associated with impairment across several domains. For instance, SAD is associated with lower educational, occupational, and economic attainment, impairment in social and romantic relationships, and restriction of and/or interference with people's plans and activities (Grant et al., 2005; Schneier et al., 1994; Stein & Kean, 2000). Individuals with SAD also report greater psychiatric and medical complaints, including high rates of suicidal ideation and suicide attempts, other anxiety and mood disorders, insomnia, and substance use disorders (Agosti et al., 2002; Buckner et al., 2008a; Buckner et al., 2008b; Davidson et al., 1993; Grant et al., 2005; Weiller et al., 1996). SAD is also associated with greater societal costs (e.g., higher rates of public assistance utilization) compared to those without SAD (Greenberg et al., 1999; Schneier et al., 1992). Similarly, panic disorder is associated with higher psychiatric comorbidity including suicide (Schmidt et al., 2001), sleep impairments (Mellman, 2006), and substance use disorders (Zvolensky et al., 2008).

How might BN serve to increase SAD risk? Perfectionism may again play a role. Perfectionism appears to interact with feeling overweight to predict bulimic behaviors among women with low self-esteem (Joiner et al., 1997). In fact, our group recently found that perfectionism moderates the relation between social anxiety and bulimic behaviors among young adults (Silgado et al., 2009). Alternatively, shame associated with bulimic behaviors may play a role. Individuals with BN report feelings of shame regarding their bulimic behaviors (Grabhorn et al., 2006). These individuals may fear scrutiny of others should they find out about their history of binging and compensatory behavior engagement. Given that fear of scrutiny is the hallmark feature of social anxiety (American Psychiatric Association, 1994), hypervigilance over being judged for bulimic behaviors may place some individuals with BN at risk for developing SAD. In fact, one report (Grabhorn et al., 2006) found shame to mediate the relation between social anxiety and both AN and BN.

How might BN increase panic disorder risk? Some individuals with BN may also be vulnerable to higher levels of anxiety sensitivity (AS), or fear of anxiety-related bodily sensations (Reiss & McNally, 1985), a well-established risk factor for panic disorder (Schmidt et al., 1997). AS (as measured by the Anxiety Sensitivity Index; Reiss et al., 1986) was significantly related to scores in the Bulimia subscale of the EDI (after controlling for depression, trait anxiety and impulsivity; Anestis et al., 2008). Along those lines, distress tolerance predicted scores in the Bulimia subscale of the EDI, and it mediated the relationship between AS and EDI-Bulimia scores (Anestis et al., 2007). It may be that among individuals with low distress tolerance and high AS, physiological hyperarousal associated with bulimic behaviors may be misinterpreted as signaling a heart attack, going crazy, or losing control, key features of panic disorder (American Psychiatric Association, 1994). However, given the dearth of research on the relations between BN and panic, future work is necessary to examine mechanisms underlying these relations.

The present study should be considered in light of limitations that suggest the need for additional work in this area. First, there were very few subjects in some diagnostic categories at T1, resulting in odds ratios with large confidence intervals. Replication in populations with higher disorder rates (e.g., treatment-seeking samples) is necessary. Second, although a psychometrically sound interview was employed to identify the presence of psychopathology, future work could benefit from the use of multiple measures to address the limitations inherent in the assessment of adolescent psychopathology (Loney et al., 2003). Third, T1 assessments were conducted using DSM-III criteria. Although sufficient information was obtained to derive DSM-IV diagnoses, future research is necessary to ascertain whether the present findings would replicate on data collected specifically using DSM-IV criteria. Fourth, given that not all participants participated in T1 and T4 assessments, generalizability to those participants not assessed at T4 is limited. Fifth, although the present study strove to test non-spuriousness by controlling for a wide range of variables, future work ruling out additional potential confounds will strengthen confidence in observed relations. Sixth, the present study examined the relations between anxiety disorder and eating disorder diagnoses. Yet it is certainly possible that elevated subclinical anxiety in adolescence could play a role in ED development and future prospective work is necessary to test this hypothesis.

The present study is the most stringent test to date of the causal relations between particular anxiety and ED. Our data suggest that OCD appears to uniquely serve as a risk for AN, whereas BN appears to serve as a specific risk for SAD and panic disorder. Identification that adolescents with OCD may be vulnerable to AN could have important treatment implications, as clinicians treating OCD may want to include strategies to prevent AN among OCD patients. Similarly, clinicians may want to include strategies to prevent SAD and/or panic disorder among adolescent BN patients.

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Table 1

Multivariate logistic regression of T1 variables predicting time 4 anorexia nervosa disorder among participants without T1 anorexia nervosa disorder.

	β	SE	Wald	P	OR	95.0% C.I. for OR	
Time 1 Variable						**Lower**	**Upper**
Age	−.95	.48	3.88	.05	.39	.15	1.00
Sex	−1.47	1.21	1.48	.22	.23	.02	2.45
Major Depressive Disorder	1.14	.93	1.53	.22	3.14	.51	19.25
Panic Disorder	−14.60	10785.55	.00	1.00	.00	.00	.00
Overanxious Disorder	−15.68	8741.50	.00	1.00	.00	.00	.00
Separation Anxiety Disorder	−16.03	5294.93	.00	1.00	.00	.00	.00
Simple Phobia	−15.67	8941.84	.00	1.00	.00	.00	.00
Obsessive-Compulsive Disorder	3.90	1.76	4.92	.03	49.26	1.57	1541.47
Social Anxiety Disorder	−15.80	7611.45	.00	1.00	.00	.00	.00

Note. $N = 1700$.

Table 2

Multivariate logistic regression of T1 variables predicting time 4 bulimia nervosa disorder among participants without T1 bulimia nervosa disorder.

	β	SE	Wald	p	OR	95.0% C.I. for OR	
Time 1 Variable						**Lower**	**Upper**
Age	.03	.23	.01	0.91	1.03	0.65	1.62
Sex	−2.25	1.04	4.66	0.03	0.10	0.01	0.81
Major Depressive Disorder	.75	.54	1.97	0.16	2.12	0.74	6.07
Panic Disorder	−16.43	12217.80	.00	1.00	0.00	0.00	0.00
Overanxious Disorder	−16.79	9491.57	.00	1.00	0.00	0.00	0.00
Separation Anxiety Disorder	−17.31	5647.23	.00	1.00	0.00	0.00	0.00
Simple Phobia	−17.23	9043.72	.00	1.00	0.00	0.00	0.00
Obsessive-Compulsive Disorder	−16.41	15087.66	.00	1.00	0.00	0.00	0.00
Social Anxiety Disorder	−17.10	8923.97	.00	1.00	0.00	0.00	0.00

Note. N = 1700. OCD=obsessive-compulsive disorder, SAD=social anxiety disorder.

Table 3

Multivariate logistic regression of T1 BN predicting T4 anxiety disorder among participants without each T1 anxiety disorder.

	β	SE	Wald	p	OR	95.0% C.I. for OR	
T4 DV						**Lower**	**Upper**
Social Anxiety Disorder	2.72	1.20	5.12	.024	15.187	1.438	160.403
Panic Disorder	2.34	1.00	5.49	.019	10.356	1.465	73.191
Generalized Anxiety Disorder	−18.01	17395.01	.00	1.00	.00	.00	.00
Major Depressive Disorder	−15.95	16845.47	.00	1.00	.00	.00	.00
Simple Phobia	−17.99	17389.06	.00	1.00	.00	.00	.00

Note. Separate multivariate analyses performed for each DV, controlling for T1 lifetime history of that DV, age, sex, mood disorders, and other ANX. *N* = 1700.