Title

Eating Disorders with and without Comorbid Depression and Anxiety: Similarities and Differences in a Clinical Sample of Children and Adolescents

Journal

European Eating Disorders Review, 21(5)

ISSN

1072-4133

Authors

Hughes, Elizabeth K
Goldschmidt, Andrea B
Labuschagne, Zandre
et al.

Publication Date

2013-09-01

DOI

10.1002/erv.2234

Peer reviewed

RESEARCH ARTICLE

Eating Disorders with and without Comorbid Depression and Anxiety: Similarities and Differences in a Clinical Sample of Children and Adolescents

Elizabeth K. Hughes[1,2,3]*, Andrea B. Goldschmidt[4], Zandre Labuschagne[4,5], Katharine L. Loeb[6,7], Susan M. Sawyer[1,2,3] & Daniel Le Grange[4]

[1]Department of Paediatrics, The University of Melbourne, Vic., Australia
[2]Centre for Adolescent Health, The Royal Children's Hospital, Vic., Australia
[3]Murdoch Childrens Research Institute, Vic., Australia
[4]Department of Psychiatry and Behavioral Neuroscience, The University of Chicago, IL, USA
[5]University of Missouri, Columbia, MO, USA
[6]School of Psychology, Fairleigh Dickinson University, NJ, USA
[7]Department of Psychiatry, Mount Sinai School of Medicine, NY, USA

Abstract

Objective: This study aimed to describe and compare the demographic and clinical characteristics of children and adolescents with an eating disorder (ED) and comorbid depression or anxiety.
Method: Data were drawn from intake assessments of children and adolescents at a specialist ED clinic. Demographic characteristics (e.g. age and gender) and clinical characteristics (e.g. body mass, binge eating and purging) were compared between 217 ED participants without comorbidity, 32 with comorbid anxiety, 86 with comorbid depression and 36 with comorbid anxiety and depression.
Results: The groups with comorbid depression had more complex and severe presentations compared with those with an ED and no comorbid disorder and those with comorbid anxiety alone, especially in regard to binge eating, purging, dietary restraint and weight/shape concerns.
Discussion: Depression and anxiety were differentially related to clinical characteristics of EDs. The findings have implications for understanding the relations between these disorders and their potential to impact outcome of ED treatments.

Keywords
eating disorders; anorexia nervosa; bulimia nervosa; depression; anxiety; adolescents; children

***Correspondence**
Elizabeth K. Hughes, The Royal Children's Hospital, 50 Flemington Road, Parkville, Vic. 3052 Australia. Tel.: +61 3 9345 4738; Fax: +61 3 9345 6273.
Email: Libby.Hughes@rch.org.au

Published online 16 May 2013 in Wiley Online Library (wileyonlinelibrary.com) DOI: 10.1002/erv.2234

Psychiatric comorbidity is common among individuals with eating disorders (ED). Clinical and community studies have reported that between 55% and 98% of individuals with anorexia nervosa (AN) and between 88% and 97% of individuals with bulimia nervosa (BN) have another Axis I disorder (Blinder, Cumella, & Sanathara, 2006; Brewerton, Lydiard, Herzog, Brotman, et al., 1995; Swanson, Crow, Le Grange, Swendsen, & Merikangas, 2011). Although prevalence rates for specific disorders vary widely across studies, the most common co-occurring conditions are consistently mood and anxiety disorders, particularly major depressive disorder and obsessive–compulsive disorder (Blinder et al., 2006; Brewerton et al., 1995; Swanson et al., 2011). Comorbidity may complicate the diagnostic picture and treatment process (Zerbe, Marsh, & Coyne, 1993) either directly or as a function of ED severity or chronicity; therefore, a growing body of research has attempted to investigate comorbid conditions in ED patients.

There is considerable variation in estimated prevalence rates of depression and anxiety in children and adolescents with EDs (Herpertz-Dahlmann, 2009; Hughes, 2012). These variations are likely due to differences in sample selection and methodology, as well as diagnostic difficulties resulting from the considerable overlap in symptoms between disorders (Godart et al., 2007). For example, lack of appetite, lethargy, poor concentration, emotional lability, self-hatred and guilt are characteristics of both EDs and depression (American Psychiatric Association, 2000). Further, the effects of starvation may contribute to a depressive-like state or even to major depressive disorder. Indeed, studies of the temporal onset of EDs and depression suggest that EDs tend to precede the development of depression (Ivarsson, Råstam, Wentz, Gillberg, & Gillberg, 2000; Lucka, 2006) and that depressed mood improves with weight restoration (Meehan, Loeb, Roberto, & Attia, 2006). Similarly, there is diagnostic overlap between EDs and anxiety disorders. For example, common signs of AN and BN

may include intense anxiety around food and eating, fear of weight gain and anxiety about social evaluation (American Psychiatric Association, 2000). The effects of starvation and other methods employed to lose weight can also contribute to obsessive–compulsive behaviours related to food, eating and body checking (Keys, Brozek, Henschel, Mickelsen, & Taylor, 1950; Mazure, Halmi, Sunday, Romano, & Einhorn, 1994). Like depression, anxiety has been reported to improve with weight restoration (Pollice, Kaye, Greeno, & Weltzin, 1997). However, in contrast with depression, anxiety tends to precede the development of AN and BN (Bulik, Sullivan, Fear, & Joyce, 1997; Godart, Flament, Lecrubier, & Jeammet, 2000).

Given the high rates of comorbid conditions in the EDs, several studies have examined their association with outcome. Studies have reported that individuals with EDs and comorbid depression or anxiety are more likely to have persisting ED symptoms, poorer health and financial functioning, and higher rates of mortality than those without comorbidity at up to 14 years follow-up (Button, Chadalavada, & Palmer, 2010; Hjern, Lindberg, & Lindblad, 2006; Milos, Spindler, Ruggiero, Klaghofer, & Schnyder, 2002). Other studies, however, have failed to find a relationship between depression or anxiety and poorer outcome for EDs (Brewerton et al., 1995; Thiel, Zuger, Jacoby, & Schussler, 1998; Wentz, Gillberg, Anckarsater, Gillber, & Rastam, 2009). In treatment studies for adolescents with EDs, results are also mixed. One study reported that baseline comorbid psychiatric disorders predicted lower rates of remission following family-based treatment (FBT) for AN (Lock, Couturier, & Agras, 2006). Similarly, baseline comorbid major depressive disorder was reported to predict lower rates of remission in FBT for BN (Le Grange, Crosby, & Lock, 2008). However, a recent study comparing FBT and adolescent-focused therapy did not find a relationship between comorbidity and outcome (Le Grange et al., 2012). These conflicting findings may be attributable to differences in sample characteristics (e.g. age and temporal onset) and methodology (e.g. definition of outcome). Of significance, these studies investigated the effects of any Axis I disorder and did not examine depression or anxiety independently.

One possible explanation for the potential effects of comorbidity on outcome is that comorbidity hampers recovery efforts. For example, social anxiety may prevent engagement in treatment because of fear of negative evaluation and avoidance of interactions with others (Goodwin & Fitzgibbon, 2002). Alternatively, comorbidity may be a marker of greater severity of illness or complexity in presentation, which may independently predict poorer outcome (Le Grange et al., 2012). If so, it would be expected that there would be observable differences in the clinical features or sociodemographic characteristics of individuals with comorbid conditions and those without. It is this latter proposition that is the focus of the current research.

Very few studies have compared characteristics of individuals with EDs with and without comorbid depression or anxiety. In the most comprehensive to date, Bulik, Sullivan, Carter, and Joyce (1996) studied 17- to 45-year-old women with BN and comorbid mood or anxiety disorder. They reported no difference in age, age at onset, body mass index (BMI) or frequency of objective binge eating, vomiting or laxative use between women with a comorbid mood disorder, anxiety disorder, both mood or anxiety disorders and those with no comorbid mood or anxiety disorder. However, women with a comorbid mood disorders had greater body dissatisfaction and were more likely to report a history of childhood externalising disorders, whereas women with comorbid anxiety disorders were more likely to have a history of AN and an earlier age of onset of substance use or dependence. In addition, women with both comorbid mood and anxiety disorders had greater food restriction and were more likely to exhibit personality disorders. Similarly, Hatsukami, Mitchell, Eckert, and Pyle (1986) reported that adult women with BN and comorbid mood disorders had an older age of BN onset, greater disruption to their social life, more suicide attempts and more previous treatment, compared with women with BN who had no comorbid mood disorder. Contrary to expectations, however, the comorbid group reported less frequent vomiting. In a community sample, Bodell, Brown, and Keel (2012) found that adults experiencing BN syndrome (i.e. binge eating or purging behaviours) had greater psychosocial impairment when combined with anxiety disorder, but that although mood disorder was associated with high levels of impairment, the presence of BN syndrome did not increase impairment further. This finding contrasts with that of Padierna, Quintana, Arostegui, Gonzalez, and Horcajo (2000) who reported that both elevated depressive and anxiety symptoms were associated with poor health-related quality of life in 14- to 65-year-old individuals with AN, BN or binge eating disorder (BED).

Notably, the aforementioned studies were restricted to individuals with BN or BN syndromes (Bodell et al., 2012; Bulik et al., 1996; Hatsukami et al., 1986), comprised relatively small samples (Bodell et al., 2012; Bulik et al., 1996), only included women (Bulik et al., 1996; Hatsukami et al., 1986) or adults (Bodell et al., 2012; Bulik et al., 1996; Hatsukami et al., 1986), or examined a very restricted range of characteristics (Bodell et al., 2012; Padierna et al., 2000). No study was found that compared a range of demographic and clinical characteristics in women and men with ED and comorbid depression and anxiety. Further, no study was found that compared characteristics across comorbidity groups in children and adolescents. This is particularly important given that EDs typically start during adolescence (Hoek & van Hoeken, 2003; Lewinsohn, Striegel-Moore, & Seeley, 2000).

This study aimed to further our understanding of comorbid depression and anxiety in the eating disorders by comparing the demographic and clinical characteristics of children and adolescents with an eating disorder and either or both comorbid depression and anxiety to those without comorbidity. In doing so, the study aimed to identify differences and similarities in characteristics between these groups and test the assumption that comorbidity equates to greater severity or complexity of illness. To our knowledge, this study is the first to provide a detailed profile of the demographic and clinical characteristics of these clinical groups in a relatively large sample of male and female children and adolescents. Because of limited access to large and diverse samples, studies of comorbidity have often analysed depression and anxiety in isolation from each other or have often combined them with other psychiatric disorders. However, this may mask important differences between the two most common disorders. Indeed, there is evidence that depression and anxiety differ in their neurobiology (Nutt & Stein, 2006) and in their temporal onset to EDs (Bulik et al., 1997; Ivarsson et al., 2000; Lucka, 2006). Our study allowed for comparisons between depressed and anxious groups.

We hypothesised that compared with those without comorbidity, those with comorbid depression and/or anxiety would have a more severe and complex presentation as indicated by more frequent bingeing and purging, and greater dietary restraint, eating concerns, and weight and shape concerns. As depression tends to have its onset subsequent to EDs, we hypothesised that, compared with those without depression, those with depression (with or without anxiety) would be older and have longer duration of illness. Because of the lack of previous research, no specific hypotheses were formulated regarding expected differences in ED diagnoses, BMI or demographic features (i.e. gender, ethnicity and family structure).

Method

Participants

Data were drawn from intake assessments of patients who presented to the Eating Disorders Program at The University of Chicago between 2001 and 2012 ($N = 800$). Included in this study were children and adolescents 18 years and younger with a DSM-IV (American Psychiatric Association, 2000) diagnosis of AN, BN or eating disorder not otherwise specified (EDNOS) and either no comorbidity or one or more comorbid mood or anxiety disorder ($n = 461$). Patients with comorbidities that did not include a mood or anxiety disorder were excluded ($n = 17$), as were participants who were missing height or weight measurements, Eating Disorder Examination results or comorbidity status ($n = 73$).

The final sample comprised 371 children and adolescents aged between 7 and 18 years ($M = 15.4$, $SD = 2.2$). Ninety-one per cent were female ($n = 339$), mean BMI was 19.8 kg/m^2 ($SD = 5.2$) and mean per cent of expected body weight (EBW) was 99.8% ($SD = 27.2$). Seventy-four per cent were Caucasian, 13% Hispanic, 7% African American, 2% Asian and 3% were of other ethnicity. The study was approved by the Institutional Review Board at The University of Chicago.

Measures

Participants in this study completed the following structured interviews and paper-and-pencil measures:

Eating Disorder Examination (*EDE*): The EDE (Cooper & Fairburn, 1987) is a semi-structured, investigator-based instrument used to diagnose EDs and measure eating-related attitudes, cognitions and behaviours. The EDE yields four subscale scores (Restraint, Eating Concern, Weight Concern and Shape Concern) and a global score measuring the overall severity of ED psychopathology, all of which range from 0 to 6, with higher scores indicating greater psychopathology. Behavioural symptoms of EDs (e.g. binge eating and purging) are assessed through a series of diagnostic questions, with higher scores indicating greater frequency of behaviours in the 3–6 months prior to assessment. The EDE has good reliability and validity (Berg, Peterson, Frazier, & Crow, 2011) and has been used in several studies of paediatric EDs (Eddy, Doyle, Hoste, Herzog, & Le Grange, 2008; Hoste & Le Grange, 2008; Stiles-Shields, Goldschmidt, Boepple, Glunz, & Le Grange, 2011). Internal consistency for the current sample was .79 (Restraint), .76 (Eating Concerns), .90 (Shape Concern) and .81 (Weight Concern).

Schedule for Affective Disorders and Schizophrenia for School-Age Children (*K-SADS*, 4th version): The K-SADS (Orvaschel & Puig-Antioch, 1987) is a structured clinical interview that was used to assess the presence or absence of current comorbid psychiatric disorders. The K-SADS has good reliability and validity (Ambrosini, 2000).

The Beck Depression Inventory (*BDI*): The BDI (Beck, 1987) is a self-report measure of depression symptoms. BDI scores range from 0 to 63, with higher scores indicating more severe depression symptoms. The BDI has good reliability and validity in adolescent outpatient samples (Ambrosini, Metz, Bianchi, Rabinovich, & Undie, 1991). Internal consistency of the BDI for the current sample was .92.

Rosenberg Self-esteem Scale (*RSE*): The RSE (Rosenberg, 1965) is a self-reported measure of global self-esteem. Scores range from 0 to 30, with higher scores indicating higher self-esteem. The RSE has good reliability and validity in youth samples (Hagborg, 1993, 1996). Internal consistency of the RSE for the current sample was .84.

Treatment history: Demographic information and duration of illness, weight history, menstrual status and psychiatric/psychological treatment history were based on the clinical interview and self-report and/or parent report at baseline.

Procedure

A clinical team member conducted the intake assessments for all patients presenting to The University of Chicago Eating Disorders Program, after appropriate consent procedures had been completed. Structured interviews were conducted by bachelors-level research assistants who completed at least 20 hours of training and received weekly assessment supervision to ensure proper calibration. Current ED diagnosis for this study was determined from the EDE and per cent EBW at the time of assessment. Per cent EBW was calculated from the 50th percentile BMI for age and gender according to the Centers for Disease Control and Prevention clinical growth charts (Centers for Disease Control and Prevention, 2000). A diagnostic algorithm was developed in accordance with classifications employed in previous studies (Berg, Stiles-Shields, et al., 2011; Eddy et al., 2008). ED diagnoses were collapsed into four categories: AN, BN, EDNOS-subthreshold AN (EDNOS-AN) and EDNOS-subthreshold BN/BED/Purging Disorder (EDNOS-BN/BED/PD). Comorbidity diagnosis for the past 6 months was confirmed via the K-SADS in combination with a psychiatric evaluation that was conducted by the clinical team member.

Results

Of the total sample ($N = 371$), 217 had no comorbid diagnosis (Group 'ED Only'), 32 had a comorbid anxiety disorder (Group 'ED + A'), 86 had a comorbid mood disorder (Group 'ED + M') and 36 had both comorbid anxiety and mood disorders (Group 'ED + AM'). The frequencies of anxiety and mood disorders in each comorbid group are shown in Table 1. In the ED + A group, generalised anxiety disorder was the most common, followed by obsessive–compulsive disorder. In the ED + M group, major depressive disorder was the most common followed by depressive

Table 1 Frequencies of comorbid disorders in each diagnostic subgroup

	Comorbid anxiety	Comorbid mood	Comorbid mood and anxiety
N	32	86	36
GAD	13 (40%)	—	20 (56%)
OCD	7 (22%)	—	6 (17%)
Social phobia	4 (13%)	—	4 (11%)
Specific phobia	2 (6%)	—	2 (6%)
PTSD	2 (6%)	—	3 (8%)
Panic disorder	1 (3%)	—	6 (17%)
Separation anxiety	1 (3%)	—	3 (8%)
Anxiety NOS	6 (19%)	—	5 (14%)
MDD	—	58 (67%)	26 (72%)
Dysthymia	—	8 (9%)	3 (9%)
Depression NOS	—	15 (17%)	6 (17%)
Bipolar I	—	1 (1%)	2 (6%)
Bipolar II	—	3 (3%)	0 (0%)
Cyclothymia	—	3 (3%)	0 (0%)

Note:
Some participant received more than one comorbid diagnosis; hence, summed percentages are greater than 100.
MDD, major depressive disorder; GAD, generalised anxiety disorder; NOS, not otherwise specified; OCD, obsessive–compulsive disorder; PTSD, post traumatic stress disorder.

disorder NOS. In the ED + AM group, major depressive disorder was the most common followed by generalised anxiety disorder.

The characteristics of each comorbid group were compared using analysis of variance and Chi-square (χ^2). First, the groups were compared on demographic characteristics including age, gender, ethnicity and family structure. Second, they were compared on clinical characteristics including diagnosis, weight history, binge eating and purging behaviours, and medication and treatment history. Finally, scores on the psychometric measures (i.e. EDE, BDI and RSE) were examined for differences across groups.

Comparisons of demographic characteristics across comorbid subgroups

As can be seen in Table 2, the ED + AM group was the oldest at assessment ($M = 16.2$ years), followed by ED + M ($M = 15.6$ years), ED Only ($M = 15.2$ years) and ED + A ($M = 15.1$ years). Although the univariate test for age was significant ($p = .048$), follow-up comparisons revealed no significant differences between the groups. There was also a significant difference between groups for family structure ($p = .019$). Follow-up tests indicated that the ED + M and ED + AM groups were more likely to come from non-intact families (35% and 36%) than ED Only (21%) and ED + A (16%) groups. No significant differences between groups were observed for gender or ethnicity.

Comparisons of clinical characteristics across comorbid subgroups

Table 3 compares the clinical characteristics of each group at presentation. There were significant between-group differences for ED diagnosis, current %EBW, presence of binge eating/purging, number of objective binge episodes, number of vomiting episodes, current medication use and history of psychiatric admission(s). There were no group differences for lowest %EBW, age at onset of symptoms, duration of illness, menstrual status, subjective binge eating, driven exercise, past outpatient treatment or past medical admissions.

Diagnosis

The ED Only and ED + A groups were most frequently diagnosed with EDNOS-AN, whereas the ED + M and ED + AM groups were most frequently diagnosed with EDNOS-BN/BED/PD. Specifically, 52% of the ED Only group and 78% the ED + A group were diagnosed with EDNOS-AN, whereas 49% of the ED + M group and 47% of the ED + AM group were diagnosed with EDNOS-BN/BED/PD. In total, 29 participants had AN, 43 had BN, 171 had EDNOS-AN and 128 had EDNOS-BN/BED/PD.

Weight history

Current BMI and current %EBW were highest in the ED + AM group and lowest in the ED + A group. The ED + M and ED + AM groups were similar on these indices (i.e. both above 100%EBW), as were the ED Only and ED + A groups (i.e. both below 100% EBW). However, only the ED Only and ED + M groups were significantly different with the ED + M group being significantly heavier than the ED Only group.

Table 2 Comparisons of demographic characteristics across diagnostic subgroups

		Eating disorder only	Comorbid anxiety disorder	Comorbid mood disorder	Comorbid mood and anxiety disorder	*p*	Effect size
		217	32	86	36		
Age, years		15.2 (2.2)[a]	15.1 (2.7) [a]	15.6 (1.8) [a]	16.2 (2.1) [a]	.048	.021
Gender	Female	199 (92%)	28 (88%)	79 (92%)	33 (92%)		
	Male	18 (8%)	4 (13%)	7 (8%)	3 (8%)	.880	.042
Ethnicity	Caucasian	161 (75%)	26 (81%)	62 (73%)	26 (72%)		
	Other	53 (25%)	6 (19%)	23 (27%)	10 (28%)	.540	.172
Family	Intact	165 (79%)[a]	27 (84%)[a]	56 (65%)[b]	23 (64%)[b]		
	Non-intact	44 (21%)	5 (16%)	30 (35%)	13 (36%)	.019	.166

Note:
For continuous variables, mean (standard deviation) is reported and differences tested with one-way analysis of variance (effect size: partial Eta-squared). For categorical variables, *n* (%) is reported and differences tested with χ^2 (effect size: Phi).
Values with differing superscripts are significantly different at $p < .05$ or less.

Table 3 Comparisons of clinical characteristics across diagnostic subgroups

		Eating disorder only	Comorbid anxiety disorder	Comorbid mood disorder	Comorbid mood and anxiety disorder	*p*	Effect Size
Diagnosis	AN	16 (7%)[a]	2 (6%)[c]	6 (7%)[b]	5 (14%)[b]		
	BN	22 (10%)	2 (6%)	13 (15%)	6 (17%)		
	EDNOS-AN	113 (52%)	25 (78%)	25 (29%)	8 (22%)		
	EDNOS-BN/BED/PD	66 (30%)	3 (9%)	42 (49%)	17 (47%)	.000	.317
Current BMI		19.1 (4.8)[a]	18.6 (4.8)[a,b]	21.2 (5.6)[b]	21.6 (6.4)[a,b]	.001	.044
Current %EBW		96.6 (23.3)[a]	95.3 (28.4)[a,b]	106.3 (29.4)[b]	107.8 (37.1)[a,b]	.007	.032
Lowest % EBW		89.0 (22.7)	84.8 (14.9)	93.2 (17.8)	83.4 (12.7)	.216	.021
Age at onset (years)		13.8 (2.6)	14.3 (2.5)	13.9 (2.6)	14.3 (2.1)	.692	.005
Duration of illness (months)		16.5 (19.4)[a]	14.6 (11.0)[a]	21.6 (28.5)[a]	26.3 (20.0)[a]	.049	.025
Menstruation (3 months)	Present	47 (26%)	2 (10%)	23 (33%)	10 (38%)		
	Absent	132 (74%)	19 (90%)	47 (67%)	16 (62%)	.108	.143
Binge/purge (3 months)	Present	113 (52%)[a]	11 (34%)[a]	61 (71%)[b]	26 (72%)[b]		
	Absent	104 (48%)	21 (66%)	25 (29%)	10 (28%)	.000	.226
Objective binge episodes (past month)		5.1 (14.5)[a, b]	1.8 (4.6) [a]	7.8 (17.3) [a, b]	10.8 (18.7)[b]	.047	.022
Subjective binge episodes (past month)		6.2 (17.2)	4.3 (10.7)	5.3 (9.6)	12.6 (19.6)	.094	.018
Vomiting episodes (past month)		11.5 (24.2)[a, b]	1.8 (5.0)[a]	19.8 (31.0)[b]	15.7 (23.0)[a, b]	.003	.037
Driven exercise episodes (past month)		7.7 (12.6)	8.5 (9.8)	8.6 (13.0)	6.9 (9.2)	.874	.002
Medication	Yes	44 (20%)[a]	13 (41%)[b]	39 (45%)[b]	16 (44%)[b]		
	No	173 (80%)	19 (59%)	47 (55%)	20 (56%)	.000	.258
Past outpatient treatment	Yes	126 (60%)	22 (69%)	55 (66%)	29 (81%)		
	No	83 (40%)	10 (31%)	28 (34%)	7 (19%)	.110	.129
Past medical admission	Yes	29 (14%)	4 (13%)	12 (15%)	7 (19%)		
	No	177 (86%)	28 (87%)	69 (85%)	29 (81%)	.841	.049
Past psychiatric admission	Yes	30 (15%)[a]	26 (19%)[a, b]	57 (29%)[b]	26 (28%)[a, b]		
	No	204 (85%)	32 (81%)	80 (71%)	36 (72%)	.031	.159

Note:
For continuous variables, mean (standard deviation) is reported and differences tested with one-way analysis of variance (effect size: partial Eta-squared). For categorical variables, *n* (%) is reported and differences tested with χ^2 (effect size: Phi).
Values with differing superscripts are significantly different at $p < .05$ or less.
AN, anorexia nervosa; BN, bulimia nervosa; EDNOS, eating disorder not otherwise specified; BED, binge eating disorder; PD, purging disorder; EBW, expected body weight.

Binge eating and purging

The ED Only and ED + A groups reported similar rates of binge eating and purging to each other and less frequent rates compared with the ED + M and ED + AM groups. In regard to any occurrence of either or both binge eating or purging in the past 3 months, there was no significant difference between the ED Only (53%) and ED + A groups (37%), but these rates were significantly less than in the ED + M (71%) and ED + AM (72%) groups, with no significant difference between these latter two groups. In regard to the number of objective binge episodes in the past month, the ED + AM group reported the greatest number of episodes ($M = 10.8$) and significantly more than the ED + A group who reported the least ($M = 1.8$). The ED Only ($M = 5.1$) and ED + AM ($M = 7.8$) groups did not differ from any group in number of objective binge episodes. In regard to the number of vomiting episodes in the past month, the ED + M group reported the greatest number of episodes ($M = 19.8$) and significantly more than the ED + A group who reported the least ($M = 1.8$). These groups did not differ in number of vomiting episodes to the ED Only ($M = 11.5$) and ED + AM ($M = 15.7$) groups. There were no significant group differences for number of subjective binge episodes or episodes of driven exercise.

Medication and treatment history

The three comorbid groups had significantly higher rates of prescribed medication than the ED Only group with 41% of the ED + A, 45% of the ED + M and 44% of the ED + AM groups being on psychotropic medication compared with 20% of the ED Only group. Antidepressants were the most common class of psychotropic medication used with 17% of the ED Only, 34% of the ED + A, 40% of the ED + M and 31% of the ED + AM groups taking antidepressants. The ED + M and ED + AM groups had the highest occurrence of previous psychiatric hospital admissions with 29% and 28% having had at least one previous admission, respectively. These rates were significantly greater than the ED Only group (15%) but not the ED + A group (19%).

Comparisons of psychometric measures across comorbid subgroups

Table 4 compares scores on standardised psychometric tests of each group at presentation. There were significant between-group differences for all measures. On the EDE, the ED + M and ED + AM groups scored significantly higher than the ED Only and ED + A groups on almost all subscales. Exceptions were the Dietary Restraint scale for which only the ED + M group scored

Table 4 Comparisons of psychometric measures across diagnostic subgroups

		Eating disorder only	Comorbid anxiety disorder	Comorbid mood disorder	Comorbid mood and anxiety disorder	*p*	Effect size
		M (*SD*)	*M* (*SD*)	*M* (*SD*)	*M* (*SD*)		
EDE	Global score	2.12 (1.45)[a]	2.09 (1.39) [a]	3.22 (1.41)[b]	3.51 (1.41)[b]	.000	.136
	Dietary restraint	2.29 (1.73) [a]	2.20 (1.76) [a]	3.30 (1.71)[b]	3.14 (1.76)[a,b]	.000	.067
	Weight concern	2.21 (1.72)[a]	2.15 (1.46)[a]	3.44 (1.63)[b]	3.90 (1.59)[b]	.000	.137
	Shape concern	2.44 (1.80)[a]	2.30 (1.65)[a]	3.69 (1.66)[b]	4.01 (1.58)[b]	.000	.124
	Eating concerns	1.55 (1.42)[a]	1.74 (1.47)[a,b]	2.49 (1.44)[b,c]	2.98 (1.73)[c]	.000	.112
BDI total		15.1 (11.0)[a]	22.1 (15.0)[a,b]	24.9 (12.4)[b]	31.6 (11.6)[b]	.000	.188
RSE total		22.9 (7.6)[a]	23.7 (7.2)[a,b]	27.4 (5.8)[b]	27.7 (5.5)[b]	.000	.087

Note:
Values with differing superscripts are significantly different at $p < .05$ or less.
EDE, Eating Disorder Examination; BDI, Beck Depression Inventory; RSE, Rosenberg Self-esteem Scale (lower = better self-esteem).

significantly higher than the ED Only and ED + A groups, and the Eating Concerns scale for which only the ED + AM group scored significantly higher than the ED Only and ED + A groups. There were no significant differences between the ED + M and ED + AM groups or between the ED Only and ED + A groups.

Discussion

This study highlights several important similarities and differences in the demographic and clinical characteristics of children and adolescents who present with EDs with and without comorbid depression and/or anxiety. Of interest, children and adolescents with an ED and comorbid anxiety disorders were similar in presentation to those without a comorbid psychiatric disorder. Likewise, children and adolescents who presented with an ED and comorbid depression were similar to those who presented with both comorbid depression and anxiety. Notably, the latter two depressed groups tended to have more complex and severe presentations compared with the former groups, especially in regard to binge eating, purging, dietary restraint and concerns about weight and shape. Collectively, these results suggest that depression carries more explanatory power than anxiety in understanding the associations with comorbidity in EDs. Overall, the hypotheses were partially supported and revealed additional information about aspects of presentation not previously researched in children and adolescents with EDs.

Of note, there were few demographic differences between children and adolescents with and without comorbid depression and/or anxiety, with all groups being of similar age, gender and ethnicity. Individuals in the depressed groups, however, were more likely to come from non-intact families than those with either no comorbid condition or only comorbid anxiety disorder. It may be that there is greater propensity towards psychopathology in these families, which contributes to more unstable parental relationships. Alternatively, disruptions in the family may contribute to the manifestation of depression in children, although the reverse may also be true (Hughes & Gullone, 2008). Examination of the timing of illness onset relative to family breakdowns was not possible in this study but would be an important aspect for future research to aid in disentangling these relationships. It is possible that non-intact families are more likely to struggle in treatment programmes for EDs. However, a study of family-based treatment for adolescent BN failed to find any differences in ED symptoms between single-parent and two-parent families at baseline, end of treatment or 6 months post treatment (Doyle, McLean, Washington, Le Grange, & Hoste, 2009).

In regard to clinical features, the depressed groups tended to present with a binge/purge disorder profile compared with the ED Only and ED + A groups. The depressed groups exhibited higher BMI and %EBW, reported the highest frequencies of objective binge eating and vomiting and were most frequently diagnosed with EDNOS-BN, BED or PD. By comparison, the ED Only and comorbid anxiety groups had lower BMI and % EBW, higher rates of amenorrhoea and were most frequently diagnosed with EDNOS-AN. These findings are in line with previous research indicating high rates of mood disorders in individuals with BN (Brewerton et al., 1995) and associations of emotion dysregulation and low mood with binge/purge behaviours (Crosby et al., 2009; Whiteside et al., 2007). Emotion regulation is becoming an important aspect of aetiological and treatment research for EDs (Clyne & Blampied, 2004; Hughes & Gullone, 2011) and in the future may be a critical factor in better understanding the relationships between mood and EDs and the ways in which we classify and treat co-occurring symptomatology.

Not surprisingly, psychotropic medication use was greater in the groups with comorbid conditions. These groups were also most likely to report a past inpatient admission to a psychiatric facility. In contrast, past outpatient treatment for EDs and inpatient admissions to medical facilities did not differ significantly between the groups. Taken together, these findings suggest that although children and adolescents with EDs and comorbid conditions receive more psychiatric treatment, they are not necessarily more frequently medically unwell. However, limited information was available on the timing and duration of treatments. Prospective monitoring of larger samples is needed to draw firm conclusions.

In regard to the standardised psychometric measures, the comorbid depression groups reported more severe symptoms. Specifically, they reported the highest levels of dietary restriction and concerns about eating, shape and weight. They also reported

the highest levels of depressive symptoms and the poorest self-esteem. Symptom scores for the comorbid anxiety (without depression) group neared those of the comorbid depression groups on some scales but overall were similar to the ED Only group.

Overall, the pattern of results suggests that the comorbid depression groups experience greater symptom severity than the ED Only and ED + A groups. Although it is difficult to compare this finding with those of previous studies because of differences in samples and methodologies, some general comments can be made albeit with caution. On the one hand, the results differ from previous studies of adults, which reported no difference in the frequency of binge eating and vomiting, or lower frequency of vomiting, for individuals with BN and comorbid depression (Bulik et al., 1996; Hatsukami et al., 1986). The results of the current study also differ from a study of adults, which reported that comorbid anxiety, but not comorbid depression, conferred greater psychosocial impairment for individuals with BN syndrome (Bodell et al., 2012). In this latter study, however, depression was associated with high levels of impairment, but the presence of BN syndrome did not increase impairment further. On the other hand, the results are consistent with previous research findings suggesting that individuals with EDs and comorbid mood disorders have greater body dissatisfaction and food restriction, and poorer social functioning and quality of life (Bulik et al., 1996; Hatsukami et al., 1986; Padierna et al., 2000). The current results may suggest that depression exacerbates ED-related symptoms in children and adolescents or that more severe ED symptoms contribute to the manifestation of depression. It is also possible that depressed individuals are more likely to experience their ED symptoms as distressing or to more readily communicate their distress. Conversely, individuals in the ED Only group may have personality traits that lead them to minimise symptomatology, and this may apply to both their ED symptoms and other psychiatric symptoms.

The hypothesis that those with depression (with or without anxiety) would be older and have longer duration of illness was not supported. Although the results were in the expected direction and the univariate tests were significant at $p < .05$, the between-group contrasts were not significant. The effect sizes were small, however, and it may be that the smaller number of participants in the ED + A and ED + AM groups and wide distribution in duration of illness impeded detection of significant differences in these variables. Although comorbid depression might predict a longer course of illness, a more chronic course could also contribute to the development of depression and the intensification of ED symptoms. Clearly, there are likely to be complex interactions at play for which a larger longitudinal sample would help to further explore and determine their importance.

The study improves upon previous research by reporting on a sample of male and female children and adolescents presenting to a specialist EDs clinic at which standardised clinical assessments are administered. Previous studies of comorbidity have focused on adults (Bodell et al., 2012; Bulik et al., 1996; Deter & Herzog, 1994; Hatsukami et al., 1986; Milos et al., 2002), despite the mean age of onset of EDs being during adolescence. Some have only included women (Bulik et al., 1996; Hatsukami et al., 1986), and some studies of clinical samples have relied on retrospective analysis of medical files (Button et al., 2010; Hjern et al., 2006; Papadopoulos, Ekbom, Brandt, & Ekselius, 2009). Although this study features a large clinical sample of children and adolescents with EDs assessed using standardised clinical measures, these analyses could have benefited from larger numbers, particularly in the ED + A and ED + AM groups. Correction for Type I error was not made in this study as this was likely to increase Type II error given the small sample size of some groups. Replication of this study with a larger sample size would allow confirmation of the current findings and possibly identify significant differences not detectable in this study. It might also allow for comparisons across ED classifications, for example, comparison of individuals with AN and depression to those with BN and depression, and for more complex analysis such as interactions effects between variables and control of potential confounding variables. This could include differences in the demographic features (e.g. family structure), which may partly contribute to differences in clinical features. A larger sample would improve cell counts and enable future analysis of interaction patterns.

Aside from sample size, other features of the current study limit its findings. The assessments relied on self-report and retrospective accounts, which may have implications for reliability of the data. In addition, the temporal relationships between onset of the disorders in question was not ascertained. The use of medical records and parent-report measures could help to confirm adolescent-sourced reports and bolster reliability. The cross-sectional design is also a limiting factor, and as noted earlier, prospective studies would be of great benefit for observing the temporal onset of comorbidity and trajectories of illness and treatment provision over time. Control groups of children and adolescents with depression or anxiety but no ED would also be helpful in teasing out the relationships between the characteristics examined and each of these disorders.

There is a clear need for research into this area, and this study represents an important step in identifying clinical and demographic features that differentiate children and adolescents presenting with EDs with and without comorbid depression and anxiety. Comparisons between depressed and anxious groups was a particular strength of this study as previous research has shown that depression and anxiety differ in their neurobiology (Nutt & Stein, 2006) and in their temporal onset in relation to EDs (Bulik et al., 1997; Ivarsson et al., 2000; Lucka, 2006). Thus, studies that combine comorbid diagnoses may mask important differences between these conditions.

Of significance, this study highlighted that depression and anxiety may be differentially related to EDs. Specifically, those with anxiety (and no depression) were similar in many respects to those without comorbidity. In contrast, children and adolescents with depression (with or without anxiety) were distinct in many respects from those without any comorbid condition. This overall finding fits with current theories on the intrinsic relations between EDs and anxiety. For example, Hildebrandt, Bacow, Markella, and Loeb (2012) argued that anxiety disorders and EDs share similar temperamental, cognitive and behavioural characteristics and that effective treatments for EDs, such as FBT, utilise strategies akin to those used to treat anxiety (e.g. exposure). Indeed, anxiety appears to pose significant risk for the development of EDs in that many individuals with AN and BN present with a comorbid anxiety disorders that commenced prior to the

development of the ED (Bulik et al., 1997; Godart et al., 2000). Ultimately, it will be important to integrate such theories and findings into complex models of ED prognosis, as well as in the development of more successful treatment models.

Acknowledgements

This study is supported by The Baker Foundation and the Victorian Government's Operational Infrastructure Support Program.

REFERENCES

Ambrosini, P. J. (2000). Historical development and present status of the schedule for affective disorders and schizophrenia for school-age children (K-SADS). *Journal of the American Academy of Child and Adolescent Psychiatry*, *39*(1), 49–58.

Ambrosini, P. J., Metz, C., Bianchi, M. D., Rabinovich, H., & Undie, A. (1991). Concurrent validity and psychometric properties of the Beck Depression Inventory in outpatient adolescents. *Journal of the American Academy of Child and Adolescent Psychiatry*, *30*(1), 51–57.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text revision). Washington DC: American Psychiatric Press.

Beck, A. T. (1987). Cognitive models of depression. *Journal of Cognitive Psychotherapy*, *1*(1), 5–37.

Berg, K. C., Peterson, C. B., Frazier, P., & Crow, S. J. (2011). Psychometric evaluation of the eating disorder examination and eating disorder examination-questionnaire: A systematic review of the literature. *International Journal of Eating Disorders*, *45*(3), 428–438. doi: 10.1002/eat.20931

Berg, K. C., Stiles-Shields, E. C., Swanson, S. A., Peterson, C. B., Lebow, J., & Le Grange, D. (2011). Diagnostic concordance of the interview and questionnaire versions of the eating disorder examination. *International Journal of Eating Disorders*, n/a-n/a. doi: 10.1002/eat.20948

Blinder, B. J., Cumella, E. J., & Sanathara, V. A. (2006). Psychiatric comorbidities of female inpatients with eating disorders. *Psychosomatic Medicine*, *68*(3), 454–462. doi: 10.1097/01.psy.0000221254.77675.f5

Bodell, L. P., Brown, T. A., & Keel, P. K. (2012). The impact of bulimic syndromes, mood and anxiety disorders and their comorbidity on psychosocial impairment: What drives impairment in comorbidity? *European Eating Disorders Review*, *20*(1), 74–79. doi: 10.1002/erv.1139

Brewerton, T. D., Lydiard, R., Herzog, D. B., Brotman, A. W., O'Neill, P.M., & Ballenger, J. C. (1995). Comorbidity of Axis I psychiatric disorders in bulimia nervosa. *The Journal of Clinical Psychiatry*, *56*(2), 7780.

Bulik, C. M., Sullivan, P. F., Carter, F. A., & Joyce, P. R. (1996). Lifetime anxiety disorders in women with bulimia nervosa. *Comprehensive Psychiatry*, *37*(5), 368–374. doi: 10.1016/S0010-440X%2896%2990019-X

Bulik, C. M., Sullivan, P., Fear, J., & Joyce, P. (1997). Eating disorders and antecedent anxiety disorders: A controlled study. *Acta Psychiatrica Scandinavica*, *96*(2), 101–107. doi: 10.1111/j.1600-0447.1997.tb09913.x

Button, E. J., Chadalavada, B., & Palmer, R. L. (2010). Mortality and predictors of death in a cohort of patients presenting to an eating disorders service. *International Journal of Eating Disorders*, *43*(5), 387–392. doi: 10.1002/eat.20715

Centers for Disease Control and Prevention. (2000). Clinical growth charts, from www.cdc.gov

Clyne, C., & Blampied, N. M. (2004). Training in emotion regulation as a treatment for binge eating: A preliminary study. *Behaviour Change*, *21*(4), 269–281.

Cooper, Z., & Fairburn, C. (1987). The Eating Disorder Examination: A semi-structured interview for the assessment of the specific psychopathology of eating disorders. *International Journal of Eating Disorders*, *6*(1), 1–8.

Crosby, R. D., Wonderlich, S. A., Engel, S. G., Simonich, H., Smyth, J., & Mitchell, J. E. (2009). Daily mood patterns and bulimic behaviors in the natural environment. *Behaviour Research and Therapy*, *47*(3), 181–188.

Deter, H., & Herzog, W. (1994). Anorexia nervosa in a long-term perspective: Results of the Heidelberg-Mannheim Study. *Psychosomatic Medicine*, *56*(1), 20–27.

Doyle, A. C., McLean, C., Washington, B. N., Le Grange, D., & Hoste, R. R. (2009). Are single-parent families different from two-parent families in the treatment of adolescent bulimia nervosa using family-based treatment? *International Journal of Eating Disorders*, *42*(2), 153–157.

Eddy, K. T., Doyle, A. C., Hoste, R. R., Herzog, D. B., & Le Grange, D. (2008). Eating disorder not otherwise specified in adolescents. *Journal of the American Academy of Child and Adolescent Psychiatry*, *47*(2), 156–164.

Godart, N. T., Flament, M., Lecrubier, Y., & Jeammet, P. (2000). Anxiety disorders in anorexia nervosa and bulimia nervosa: Comorbidity and chronology of appearance. *European Psychiatry*, *15*(1), 38–45. doi: 10.1016/S0924-9338%2800%2900212-1

Godart, N. T., Perdereau, F., Rein, Z., Berthoz, S., Wallier, J., Jeammet, P., et al. (2007). Comorbidity studies of eating disorders and mood disorders. Critical review of the literature. *Journal of Affective Disorders*, *97*(1-3), 37–49. doi: 10.1016/j.jad.2006.06.023

Goodwin, R. D., & Fitzgibbon, M. L. (2002). Social anxiety as a barrier to treatment for eating disorders. *International Journal of Eating Disorders*, *32*(1), 103–106. doi: 10.1002/eat.10051

Hagborg, W. J. (1993). Middle-school student satisfaction with group counseling: An initial study. *The Journal for Specialists in Group Work*, *18*(2), 80–85. doi: 10.1080/01933929308413740

Hagborg, W. J. (1996). Self-concept and middle school students with learning disabilities: A comparison of scholastic competence subgroups. *Learning Disability Quarterly*, *19*(2), 117–126.

Hatsukami, D. K., Mitchell, J. E., Eckert, E. D., & Pyle, R. L. (1986). Characteristics of patients with bulimia only, bulimia with affective disorder, and bulimia with substance abuse problems. *Addictive Behaviors*, *11*(4), 399–406. doi: 10.1016/0306-4603%2886%2990018-3

Herpertz-Dahlmann, B. (2009). Adolescent eating disorders: Definitions, symptomatology, epidemiology and comorbidity. *Child and Adolescent Psychiatric Clinics of North America*, *18*(1), 31–47. doi: 10.1016/j.chc.2008.07.005

Hildebrandt, T., Bacow, T., Markella, M., & Loeb, K. L. (2012). Anxiety in anorexia nervosa and its management using family-based treatment. *European Eating Disorders Review*, *20*(1), e1–e16. doi: 10.1002/erv.1071

Hjern, A. M. D. P., Lindberg, L. P., & Lindblad, F. M. D. P. (2006). Outcome and prognostic factors for adolescent female inpatients with anorexia nervosa: 9- to 14-year follow-up. *British Journal of Psychiatry November*, *189*(5), 428–432.

Hoek, H. W., & van Hoeken, D. (2003). Review of the prevalence and incidence of eating disorders. *International Journal of Eating Disorders*, *34*(4), 383–396. doi: 10.1002/eat.10222

Hoste, R. R., & Le Grange, D. (2008). Expressed emotion among white and ethnic minority families of adolescents with bulimia nervosa. *European Eating Disorders Review*, *16*(5), 395–400. doi: 10.1002/erv.856

Hughes, E. K. (2012). Comorbid depression and anxiety in childhood and adolescent anorexia nervosa: Prevalence and implications for outcome. *Clinical Psychologist*, *16*(1), 15–24.

Hughes, E. K., & Gullone, E. (2008). Internalizing symptoms and disorders in families of adolescents: A family systems review. *Clinical Psychology Review*, *28*(1), 92–117. doi: 10.1016/j.cpr.2007.04.002

Hughes, E. K., & Gullone, E. (2011). Emotion regulation moderates relationships between body image concerns and psychological symptomatology. *Body Image: An International Journal of Research*, *8*(3), 224–231. doi: 10.1016/j.bodyim.2011.04.001

Ivarsson, T., Råstam, M., Wentz, E., Gillberg, I., & Gillberg, C. (2000). Depressive disorders in teenage-onset anorexia nervosa: A controlled longitudinal, partly community-based study. *Comprehensive Psychiatry*, *41*(5), 398–403. doi: 10.1053/comp.2000.9001

Keys, A., Brozek, J., Henschel, A., Mickelsen, O., & Taylor, H. L. (1950). *The biology of human starvation*. Minneapolis: University of Minnesota Press.

Le Grange, D., Crosby, R. D., & Lock, J. (2008). Predictors and moderators of outcome in family-based treatment for adolescent bulimia nervosa. *Journal of the American Academy of Child and Adolescent Psychiatry*, *47*(4), 464–470.

Le Grange, D., Lock, J., Agras, W. S., Moye, A., Bryson, S. W., Jo, B., et al. (2012). Moderators and mediators of remission in family-based treatment and adolescent focused therapy for anorexia nervosa. *Behaviour Research and Therapy*, *50*(2), 85–92. doi: 10.1016/j.brat.2011.11.003

Lewinsohn, P. M., Striegel-Moore, R. H., & Seeley, J. R. (2000). Epidemiology and natural course of eating disorders in young women from adolescence to young adulthood. *Journal of the American Academy of Child and Adolescent Psychiatry*, *39*(10), 1284–1292. doi: 10.1097/00004583-200010000-00016

Lock, J., Couturier, J., & Agras, W. S. (2006). Comparison of long-term outcomes in adolescents with anorexia nervosa treated with family therapy. *Journal of the American Academy of Child and Adolescent Psychiatry*, *45*(6), 666–672. doi: 10.1097/01.chi.0000215152.61400.ca

Lucka, I. (2006). Depressive disorders in patients suffering from anorexia nervosa. *Archives of Psychiatry and Psychotherapy*, *8*(2), 55–61.

Mazure, C. M., Halmi, K. A., Sunday, S. R., Romano, S. J., & Einhorn, A. M. (1994). The Yale-Brown-Cornell eating disorder scale: Development, use, reliability and validity. *Journal of Psychiatric Research*, *28*(5), 425–445.

Meehan, K. G., Loeb, K. L., Roberto, C. A., & Attia, E. (2006). Mood change during weight restoration in patients with anorexia nervosa. *International Journal of Eating Disorders*, *39*(7), 587–589. doi: 10.1002/eat.20337

Milos, G., Spindler, A., Ruggiero, G., Klaghofer, R., & Schnyder, U. (2002). Comorbidity of obsessive-compulsive disorders and duration of eating disorders. *International Journal of Eating Disorders*, *31*(3), 284–289. doi: 10.1002/eat.10013

Nutt, D. J., & Stein, D. J. (2006). Understanding the neurobiology of comorbidity in anxiety disorders. *CNS Spectrums*, *11*(10(Suppl 12)), 13–20.

Orvaschel, E., & Puig-Antioch, J. (1987). *Schedule for Affective Disorders and Schizophrenia for School-Age Children-Epidemiologic Version (K-SADS-E)*. Philadelphia: Medical College of Pennsylvania.

Padierna, A., Quintana, J., Arostegui, I., Gonzalez, N., & Horcajo, M. (2000). The health-related quality of life in eating disorders. *Quality of Life Research: An International Journal of Quality of Life Aspects of Treatment, Care & Rehabilitation*, *9*(6), 667–674. doi: 10.1023/A:1008973106611

Papadopoulos, F. C., Ekbom, A., Brandt, L., & Ekselius, L. (2009). Excess mortality, causes of death and prognostic factors in anorexia nervosa. *The British Journal of Psychiatry*, *194*(1), 10–17. doi: 10.1192/bjp.bp.108.054742

Pollice, C., Kaye, W. H., Greeno, C. G., & Weltzin, T. E. (1997). Relationship of depression, anxiety, and obsessionality to state of illness in anorexia nervosa. *International Journal of Eating Disorders*, *21*(4), 367–376. doi: 10.1002/%28SICI%291098-108X%281997%2921:4%3C367::AID-EAT10%3E3.0.CO;2-W

Rosenberg, M. (1965). *Society and the adolescent self-image*. Princeton, NJ: Princeton University Press.

Stiles-Shields, C., Goldschmidt, A. B., Boepple, L., Glunz, C., & Le Grange, D. (2011). Driven exercise among treatment-seeking youth with eating disorders. *Eating Behaviors*, *12*(4), 328–331. doi: 10.1016/j.eatbeh.2011.09.002

Swanson, S. A., Crow, S. J., Le Grange, D., Swendsen, J., & Merikangas, K. R. (2011). Prevalence and correlates of eating disorders in adolescents: Results from the national comorbidity survey replication adolescent supplement. *Archives of General Psychiatry*, *68*(7), 714–723. doi: 10.1001/archgenpsychiatry.2011.22

Thiel, A., Zuger, M., Jacoby, G. E., & Schussler, G. (1998). Thirty-month outcome in patients with anorexia or bulimia nervosa and concomitant obsessive-compulsive disorder. *The American Journal of Psychiatry*, *155*(2), 244–249.

Wentz, E., Gillberg, I., Anckarsater, H., Gillber, C., & Rastam, M. (2009). Adolescent-onset anorexia nervosa: 18-year outcome. *The British Journal of Psychiatry*, *194*(2), 168–174. doi: 10.1192/bjp.bp.107.048686

Whiteside, U., Chen, E., Neighbors, C., Hunter, D., Lo, T., & Larimer, M. (2007). Difficulties regulating emotions: Do binge eaters have fewer strategies to modulate and tolerate negative affect? *Eating Behaviors*, *8*(2), 162–169. doi: 10.1016/j.eatbeh.2006.04.001

Zerbe, K. J., Marsh, S. R., & Coyne, L. (1993). Comorbidity in an inpatient eating disordered population: clinical characteristics and treatment implications. *The Psychiatric Hospital*, *24*(1-2), 3–8.

