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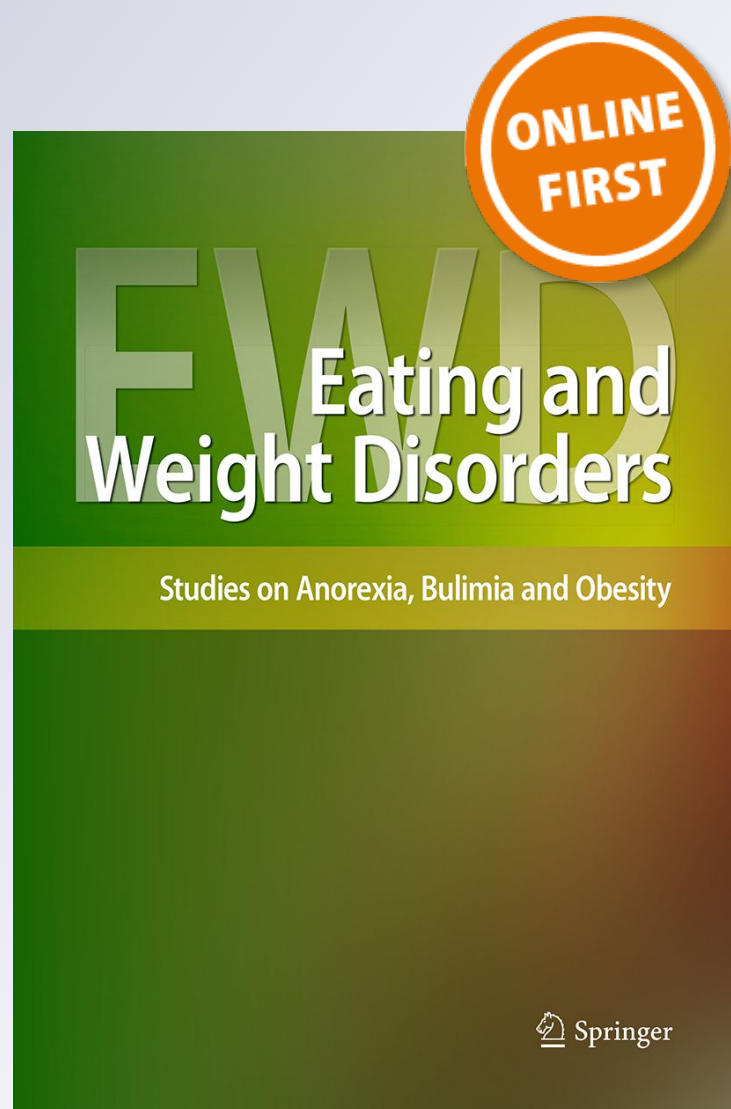
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Depression, worry, and psychosocial functioning predict eating disorder treatment outcomes in a residential and partial hospitalization setting

Laura K. Fewell¹ · Cheri A. Levinson^{2,3} · Lynn Stark¹

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Abstract This retrospective study explores depression, worry, psychosocial functioning, and change in body mass index (BMI) as predictors of eating disorder (ED) symptomatology and BMI at discharge and 1-year follow-up from a residential and partial hospitalization ED treatment center. Participants were 423 male and female patients receiving treatment at an ED treatment center. Results indicate significant improvement in ED symptomatology, psychological impairment, and change in BMI (in patients with anorexia nervosa) at treatment discharge and follow-up compared to treatment admission ($p < 0.001$). Depression and worry predicted ED symptomatology and psychological impairment at discharge ($p < 0.05$). Depression, worry, and psychosocial functioning predicted ED symptomatology and psychological impairment at 1-year follow-up ($p < 0.001$). Change in BMI was not a significant predictor of outcome. Depression, worry, and psychosocial functioning each play a role in treatment outcomes and may help clarify who might benefit from treatment. Clinicians in ED treatment centers should consider these as areas of focus for improved outcomes.

Keywords Eating disorder · Predictor · Treatment outcome · Depression · Anxiety · Worry

Introduction

Eating disorders (EDs) are serious and often chronic illnesses, with anorexia nervosa (AN) accounting for the highest mortality rate among all mental illnesses [1]. Most individuals diagnosed with an ED are also diagnosed with comorbid conditions, such as anxiety and depression [2–5]. Researchers have estimated that nearly two-thirds of ED patients have at least one anxiety disorder diagnosis [2] and approximately half have at least one depressive disorder diagnosis [4]. EDs are often treated in inpatient (IP), residential, or partial hospitalization programming (PHP) treatment settings [6]. Because these settings differ in treatment intensity, philosophy, and overall objectives [7–9], the effectiveness of these programs has been a primary focus of investigation in the ED literature [6, 10, 11]. In addition, given the high rates of depression and anxiety diagnoses accompanying EDs, it is useful to understand how these comorbid conditions affect such treatments.

Unsurprisingly, findings regarding the effectiveness of treatment centers vary. Some findings suggest that between 72% and 78% of patients have improved symptoms (ED symptomatology, depression, anxiety) after seeking treatment in either an IP or PHP setting [12, 13] and either maintain or continue to improve after discharge [10, 11, 14]. However, Fichter, Quadflieg, and Hedlund (2006) found positive outcomes in only 30% of patients with AN in their 12-year longitudinal study; 8% of patients were deceased at study end and 70% showed intermediate to poor outcome [15]. The variability in treatment efficacies may stem from the disparity of measured outcomes: for

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example, a meta-analysis by Friedman et al. [16] found that the majority of reported outcomes reflect improvement at treatment discharge rather than at post discharge. Furthermore, they demonstrated that improvement at treatment discharge did not predict post-discharge outcomes. Therefore, specific factors that predict long-term outcome, rather than overall treatment progress, should be considered.

Another possible explanation for the variability in treatment effectiveness may be due, at least in part, to the selected outcomes of focus. Most of the available literature focuses on both physical condition (i.e., weight) and psychopathology to measure outcomes in ED treatment [17–21]. For example, a recent study by Dingemans et al. [22] investigated ED symptomatology as a treatment outcome and found that high interpersonal distrust, low self-esteem, and low body image satisfaction were associated with increased ED symptomatology at treatment end. Yet psychosocial functioning and quality of life have also been considered to be important aspects of ED treatment outcomes, as individuals with EDs tend to report impaired social functioning and quality of life, and psychosocial functioning has been linked to higher mortality rates in AN [17, 23]. Based on previous research, in the current study, we focus on ED symptomatology, psychological impairment, and weight (BMI) as our outcomes of interest.

Depression has been found to play a role in the effectiveness of ED treatment and is theorized to be a potential precursor of EDs [2]. Several studies have shown that depression is associated with the presence of ED diagnostic criteria in those with both AN and bulimia nervosa (BN) [6, 24–26]. Higher levels of depression have been found to predict greater ED pathology at 12 years following inpatient treatment (though these findings may have been better accounted for by impulsivity, sexual problems, longer duration of ED prior to treatment, and longer duration of treatment) [15]. However, other researchers did not find that pre-treatment depression is related to treatment outcomes [27] but, rather, that the presence of depressive symptoms is a risk factor for an ED [28, 29]. Yet another study found that depression and eating pathology were bi-directional; that is, eating pathology predicted depression and depression predicted eating pathology [30]. Further research on depression as a predictor of ED symptomatology and weight maintenance during and after treatment, specifically in a residential and PHP setting, is warranted.

Anxiety has yet to be established as a predictor of ED symptomatology following ED treatment. In a retrospective report by Kaye et al. [2], participants with anxiety disorders, such as obsessive compulsive disorder, social phobia, specific phobia, and generalized anxiety disorder, reported that their anxiety had an earlier age of onset than their ED. However, it is unclear whether anxiety is a precursor to an ED as it has not yet been explored in relation to ED

treatment outcomes [31]. The literature that does exist finds that state anxiety is significantly higher in patients who are underweight versus weight-restored [3], yet weight-restored patients still report significant state anxiety. One aspect of anxiety that may be particularly relevant for treatment outcomes is worry. Worry, a core feature of anxiety, has been shown to be significantly higher in patients with EDs than in control groups [32]. However, no research currently exists that tests if worry predicts ED treatment outcomes.

Rate of weight gain during treatment and underweight or low weight at discharge have been proposed as potential factors contributing to ED symptomatology and weight normalization following treatment [13, 33]. Researchers found that patients with AN who gained at least 0.8 kg per week had better outcomes at 1-year follow-up than those who gained less than 0.8 kg per week [34]. Other researchers found that early weight gain in adolescents with AN seeking family-based treatment and adolescent-focused therapy predicted remission at treatment end but not at 1-year follow-up [19]. In addition, patients who had lower body mass index (BMI), worse living situations, and an inability to work demonstrated higher ED symptomatology than those with higher BMIs and better psychosocial functioning [13]. However, other reports suggest that lower percent of body fat at treatment discharge, but not BMI, is associated with poor outcome [35]. Change in BMI in patients with AN should be the focus of additional research to better understand the relationship between BMI and ED outcomes.

Hypotheses

In the current study, we tested if ED symptomatology, psychological impairment, and BMI (in patients with AN only) changed from treatment admission to treatment discharge. We also tested several potential factors that may contribute to ED treatment outcomes. Specifically, we tested if depression, worry, and general psychosocial functioning (such as difficulty in work/school, making new friends, and joining in community activities) contributed to the following outcomes: ED symptomatology, psychological impairment, and BMI (in patients with AN) both at discharge and 1 year after discharge from a residential/partial hospitalization treatment program. Finally, we tested if amount of weight gained during treatment (measured by change in BMI) in patients with AN contributed to ED symptomatology and psychological impairment at discharge and 1 year after discharge from treatment.

Based on the previous literature investigating depression and psychosocial factors as predictors of outcome [13, 28, 29], we had five primary hypotheses. First, we hypothesized that ED symptomatology and psychological

impairment would be significantly lower at treatment discharge than treatment admission, and BMI (in patients with AN) would be significantly higher at treatment discharge than treatment admission. Second, we hypothesized that higher levels of depression at treatment admission would predict higher ED and psychological impairment and lower BMI (in patients with AN) at both discharge and follow-up. Third, we hypothesized that higher levels of worry at admission would predict higher ED and psychological impairment and lower BMI (in patients with AN) at discharge and follow-up. Fourth, we hypothesized that poorer general psychosocial functioning at treatment admission would predict higher ED and psychological impairment and lower BMI (in patients with AN) at discharge and follow-up. Fifth, we hypothesized that greater change in BMI during treatment would predict lower ED and psychological impairment in patients with AN at discharge and follow-up.

Methods

Participants

Participants were 423 adults ($n=311$) and adolescents ($n=112$) receiving either residential or partial hospitalization treatment at an ED treatment facility between December 2012 and December 2015. The standard treatment (e.g., treatment as usual) provided at the treatment facility used a combination of individual, family, and group therapies which encompassed a variety of theoretical approaches, including nutrition therapy, cognitive behavioral therapy, dialectical behavior therapy, exposure therapy (specifically at meal time), psychoeducation, and the experiential therapies such as art therapy, dance/movement therapy, and music therapy. Treatments were tailored to patients based on age-appropriate methods (i.e., increased family sessions for adolescent patients). Treatment also integrated individual and group fitness sessions, and patients met with a board-certified psychiatrist at least once per week. Patients were assigned to a combined treatment approach which may or may not have included sequential treatments. Psychopharmacological treatments were also implemented based on the needs of each individual patient as determined by a psychiatrist. Length of stay (LOS) included both residential and PHP levels of care and ranged from 7 to 120 days (mean LOS = 49.47; SD = 27.11). Participants were diagnosed with AN (restricting or binge/purge subtype), BN, eating disorder not otherwise specified (EDNOS), other specified feeding or eating disorder (OSFED; a diagnosis derived from the DSM-V revision which was used during the latter course of the study), binge eating disorder (BED; also used in conjunction with the release of the DSM-V), or avoidant/restrictive food intake disorder (ARFID; used

in conjunction with the release of the DSM-V). Diagnoses were made by board-certified psychiatrists upon treatment admission. Please see Table 1 for a description of all demographic and clinical characteristics.

Procedures

This retrospective study was approved by the Institutional Review Board at Washington University in St. Louis. Participants completed the following paper and pencil assessments at the beginning ($N=423$) and end ($n=423$) of treatment: Eating Disorder Examination Questionnaire (EDE-Q) [36]; Clinical Impairment Assessment (CIA) [37]; Beck Depression Inventory-II [38]; Penn State Worry Questionnaire [39]; and World Health Disability Assessment Schedule 2.0 [40] (see descriptions below). BMI was also obtained at admission and discharge. Participants completed the EDE-Q and CIA and reported height and weight online 1 year following discharge from the treatment center ($n=65$).

Outcome measures

Eating Disorder Examination Questionnaire 16.0 (EDE-Q) [36] is a 28-item self-report questionnaire designed to assess ED behaviors and thoughts. We used the EDE-Q version 16.0, which is a data-compatible but slightly shorter update to the EDE-Q version 12.0 and includes modifications to language and a BED module [36]. We used the global score of the EDE-Q, calculated by summing the subscale totals and dividing by the number of subscales, to measure overall eating symptomatology. The EDE-Q has demonstrated excellent test-retest reliability and internal consistency [41], and acceptable to good criterion validity and concurrent validity [42]. Chronbach's α was 0.95 for the present study.

Clinical Impairment Assessment (CIA) [37] is a 16-item self-report measure designed for use in conjunction with the EDE-Q to measure psychological and social impairment related to an ED. Items ask about ED impairment on mood, cognitive functioning, interpersonal functioning, and work performance. We used the global score of the CIA, calculated by summing the item totals. The CIA has demonstrated high internal consistency and test-retest reliability, and good construct and criterion validity [43]. Chronbach's α was 0.95 for the present study.

Outcome predictor measures

Beck Depression Inventory-II (BDI-II) [38] consists of 21 self-report items measuring severity of depression. The BDI-II has demonstrated high internal consistency and concurrent validity [44]. In the current study, we used the

Table 1 Participant demographic and clinical characteristics

	Mean (SD)	Range
Age in years	23.72 (9.49)	11 to 60 years
LOS in days	49.47 (27.11)	7 to 120 days
BMI in AN	17.61 (2.60)	11.7 to 28.1
Age of ED onset	14.49 (5.48)	2 to 55 years
Duration of ED	9.21 (9.21)	0 to 44 years
	<i>n</i> (%)	
Female	402 (95%)	
European-American	389 (92%)	
AN	263 (62.2)	
BN	66 (15.6)	
EDNOS	51 (12.0)	
OSFED	28 (6.6)	
BED	10 (2.4)	
ARFID	5 (1.2)	
Anxiety disorder	206 (48.7)	
Depressive disorder	212 (50.1)	
Bipolar disorder	17 (4.0)	
AdBMI < 18.5	184 (43.5)	

BMI in AN body mass index in patients with anorexia nervosa, *LOS* length of stay in days for the total number of days in either residential and/or partial hospitalization programming, *AN* anorexia nervosa; *BN* bulimia nervosa, *EDNOS* eating disorder not otherwise specified, *OSFED* other specified feeding or eating disorder, *BED* binge eating disorder, *ARFID* avoidant restrictive food intake disorder, *AdBMI* admission body mass index

BDI-II to measure *depression*. Chronbach's α was 0.93 for the present study.

Penn State Worry Questionnaire (PSWQ) [39] is a 16-item self-report questionnaire measuring trait assessment of pathological worry. It has been shown to possess high internal consistency and test–retest reliability, and good convergent validity [45]. Chronbach's α was 0.76 for the present study.

World Health Organization Disability Assessment Schedule 2.0 (WHODAS 2.0) [40] is a 36-item, self-report measure designed to assess disability across six domains, including areas of communication and participation in society. We used the global score (scored by summing all items and dividing by the number of items) to measure *psychosocial functioning*, which has demonstrated good test–retest reliability and very good internal consistency [40]. Chronbach's α was 0.96 for the present study.

Body mass index (BMI) was assessed by an approved staff at admission and discharge from treatment. Height and weight were measured using a medical-grade Detecto precision scale and height tool. Participants were not informed of their weight and were weighed in light clothing and without shoes. At 1-year follow-up, participants self-reported their height and weight and BMI was calculated using an online BMI calculator [46].

Analyses

First, we conducted within-subjects ANOVAs for each predictor and outcome variable to look at change over time (e.g., did scores improve?) from treatment admission, discharge, and 1-year follow-up (see Table 2). Then, utilizing multiple regression, we concurrently tested if depression, worry, and psychosocial functioning predicted ED symptomatology at discharge over and above admission ED symptomatology using the BDI-II, PSWQ, WHODAS 2.0, and EDE-Q (see Table 3). Then, in a separate regression, we concurrently tested if depression, worry, and psychosocial functioning predicted ED symptomatology at 1-year follow-up over and over admission ED symptomatology (Table 3). We also tested if change in BMI, in addition to depression, worry, psychosocial functioning, and admission ED symptomatology, predicted discharge and 1-year follow-up ED symptomatology (Table 3). Next, we concurrently tested if depression, worry, and psychosocial functioning predicted psychological impairment at discharge and then again at 1-year follow-up over and above admission psychological impairment (see Table 4). We also tested if change in BMI, in addition to depression, worry, psychosocial functioning, and admission psychological impairment, predicted discharge and 1-year follow-up

Table 2 Within-subjects ANOVA: ED symptomatology, impairment, BMI, depression, worry, and psychosocial functioning (standard errors)

	Admission EDE-Q Mean (SD)	Discharge EDE-Q Mean (SD)	12mo EDE-Q Mean (SD)	F value	Sig.	N
ANOVA 1; ED Symptoms over time in the full sample	4.01 (1.51)	2.57 (1.50)	2.89 (1.37)	300.30	$p < 0.001$	423
	Admission CIA Mean (SD)	Discharge CIA Mean (SD)	12mo CIA Mean (SD)	F value	Sig.	N
ANOVA 2; Impairment over time in the full sample	33.89 (0.53)	20.64 (0.61)	21.40 (0.58)	311.11	$p < 0.001$	423
	Admission BMI Mean (SD)	Discharge BMI Mean (SD)	12mo BMI Mean (SD)	F value	Sig.	N
ANOVA 3; BMI over time in AN patients	17.65 (0.17)	20.36 (0.15)	20.61 (0.18)	316.39	$p < 0.001$	263
	Admission BDI Mean (SD)	Discharge BDI Mean (SD)		F value	Sig.	N
ANOVA 4; Depression over time in the full sample	31.36 (0.70)	19.30 (0.70)		357.61	$p < 0.001$	423
	Admission PSWQ Mean (SD)	Discharge PSWQ Mean (SD)		F value	Sig.	N
ANOVA 4; Worry over time in the full sample	63.21 (0.61)	57.43 (0.70)		102.55	$p < 0.001$	423
	Admission WHO- DAS 2.0 Mean (SD)	Discharge WHO- DAS 2.0 Mean (SD)		F value	Sig.	N
ANOVA 4; Psychosocial functioning over time in the full sample	2.30 (0.04)	1.9 (0.03)		106.37	$p < 0.001$	423

ED Symptomatology Eating Disorder Examination Questionnaire, *Impairment* Clinical Impairment Assessment, *EDE-Q* Eating Disorder Examination Questionnaire, *CIA* Clinical Impairment Assessment, *BMI* body mass index, *AN* anorexia nervosa. *BDI* Beck Depression Inventory, *PSWQ* Penn State Worry Questionnaire, *WHODAS*=World Health Disability Assessment Schedule 2.0

psychological impairment in patients with AN (Table 4). Finally, we concurrently tested if depression, worry, and psychosocial functioning predicted BMI at discharge and 1-year follow-up in patients with AN over and above admission BMI (see Table 5). We controlled for LOS and age for each analysis due to the wide range of LOS and ages in this population, which could potentially account for varying results. Missing data were accounted for using Amelia II, a statistical program that fills missing values with multiple imputations to reproduce missing data [47]. This method of imputation reduces bias from incomplete data that would otherwise be removed. Imputation diagnostics indicated that the imputation had been successful.

Results

Zero-order correlations

In the full sample, all variables were significantly and positively correlated, with the exception of the correlation between psychosocial functioning and 1-year follow-up ED symptomatology ($r=0.05$, $p=0.27$). In participants with

AN, psychosocial functioning positively correlated with BMI at discharge ($r=0.15$, $p<0.01$) and BMI at 1-year follow-up ($r=0.49$, $p<0.001$), but not with change in BMI ($r=-0.12$, $p=0.06$). In addition, depression ($r=0.56$, $p<0.001$) and worry ($r=0.26$, $p<0.001$) were positively correlated with BMI at 1-year follow-up. Depression ($r=0.09$, $p=0.15$) and worry ($r=0.06$, $p=0.65$) were not correlated with BMI at discharge, and there was no significant correlation with depression ($r=-0.05$, $p=0.41$) or worry ($r=-0.06$, $p=0.35$) and change in BMI.

Within-subjects ANOVAs

As shown in Table 2, a within-subjects analysis of variance (ANOVA) was computed for each outcome variable (e.g. ED symptomatology, psychological impairment, and BMI in AN), as well as for each predictor (depression, worry, and psychosocial functioning). Psychological impairment scores decreased from admission to discharge and admission to 1 year following discharge. Psychological impairment scores at 1-year follow-up were not significantly different than scores at discharge. ED symptomatology decreased from admission to discharge and admission to

Table 3 Multiple regression analyses predicting eating disorder symptomatology in the full sample and in patients with AN

Predictors	β	Part r	Sig.	N
Regression 1; discharge ED symptomatology in the full sample				423
Worry	-0.21**	-0.20**	$p < 0.001$	
Depression	0.26**	0.19**	$p < 0.001$	
Psychosocial functioning	-0.05	-0.05	$p = 0.34$	
Admission EDE-Q	0.58**	0.44**	$p < 0.001$	
Regression 2; discharge ED symptomatology in patients with AN				263
Worry	-0.17*	-0.16*	$p < 0.01$	
Depression	0.21*	0.15*	$p < 0.01$	
Psychosocial functioning	-0.10	-0.08	$p = 0.17$	
Change in BMI	0.08	0.10	$p = 0.11$	
Admission EDE-Q	0.61**	0.46**	$p < 0.001$	
Regression 3; 1-year follow-up ED symptomatology in the full sample				423
Worry	0.22**	0.22**	$p < 0.001$	
Depression	0.25**	0.20**	$p < 0.001$	
Psychosocial functioning	-0.55**	-0.48**	$p < 0.001$	
Admission EDE-Q	0.56**	0.48**	$p < 0.001$	
Regression 4; 1-year follow-up ED symptomatology in patients with AN				263
Worry	0.23**	0.22**	$p < 0.001$	
Depression	0.23**	0.18**	$p < 0.001$	
Psychosocial functioning	-0.56**	-0.45**	$p < 0.001$	
Change in BMI	0.00	0.00	$p = 0.95$	
Admission EDE-Q	0.58**	0.46**	$p < 0.001$	

Standardized beta and partial correlation coefficients are reported

ED Symptomatology Eating Disorder Examination Questionnaire, *Impairment* Clinical Impairment Assessment, *Admission EDE-Q* Admission Eating Disorder Examination Questionnaire, *Admission CIA* Admission Clinical Impairment Assessment, *Worry* Penn State Worry Questionnaire, *Depression* Beck Depression Inventory-II, *Psychosocial Functioning* World Health Organization Disability Assessment Schedule 2.0

* $p < .05$

** $p < .001$

1 year following discharge; however, ED symptomatology scores at 1-year follow-up were significantly higher than discharge scores (Table 2). In patients with AN, BMIs were significantly higher at discharge than at admission, and BMIs at 1-year follow-up were similar to discharge BMIs. The average change in BMI in AN was 2.69, and 79.5% of AN patients discharged with a BMI of at least 18.5 (up from 30% upon admission). BMIs at 1-year follow-up were significantly higher than at admission and did not differ significantly from discharge BMIs (see Table 2). Depression, worry, and psychosocial functioning were all significantly lower at treatment discharge compared to treatment admission ($ps < 0.001$).

There were three differences found between diagnostic groups: patients with AN had significantly better psychosocial functioning at discharge ($M = 1.85$) than patients with EDNOS ($M = 2.14$; $p < 0.01$); patients with AN had significantly longer LOS ($M = 53.6$ days) than patients with both BN ($M = 41.8$ days) and EDNOS ($M = 43.5$ days; $ps < 0.05$); and patients with BN had significantly

higher ED symptomatology at treatment admission ($M = 4.34$) than patients with AN ($M = 3.93$; $ps < 0.05$). No other differences were found between groups.

Multiple regression analyses: are there factors that predict ED treatment outcomes?

ED symptomatology at discharge

As shown in Table 3, in multiple regression, depression and worry predicted discharge ED symptomatology over and above admission ED symptomatology. Depression and worry, but not change in BMI, were also predictors of discharge ED symptomatology in patients with AN. These effects remained significant when controlling for LOS and age. There were no main effects or interactions with LOS and age ($ps \geq 0.24$), and there were no significant differences found between ED diagnoses.

Table 4 Multiple regression analyses predicting psychological impairment in the full sample and in patients with AN

	Regression 1; discharge impairment in the full sample			N
				423
Worry	-0.13*	-0.11*	$p=0.03$	
Depression	0.34**	0.23**	$p<0.001$	
Psychosocial functioning	-0.13	-0.06	$p=0.25$	
Admission CIA	0.35**	0.22**	$p<0.001$	
	Regression 3; discharge impairment in patients with AN			N
				263
Worry	-0.06	-0.05	$p=0.45$	
Depression	0.28**	0.17**	$p<0.001$	
Psychosocial functioning	-0.07	-0.05	$p=0.41$	
Change in BMI	0.02	0.02	$p=0.77$	
Admission CIA	0.30**	0.18**	$p<0.001$	
	Regression 3; 1-year follow-up impairment in the full sample			N
				423
Worry	0.31**	0.26**	$p<0.001$	
Depression	0.19*	0.13*	$p<0.01$	
Psychosocial functioning	-0.41**	-0.30**	$p<0.001$	
Admission CIA	0.32**	0.21**	$p<0.001$	
	Regression 4; 1-year follow-up impairment in patients with AN			N
				263
Worry	0.35**	0.28**	$p<0.001$	
Depression	0.15	0.10	$p=0.11$	
Psychosocial functioning	-0.34**	-0.24**	$p<0.001$	
Change in BMI	-0.06	-0.06	$p=0.30$	
Admission CIA	0.26*	0.16*	$p<0.01$	

Standardized beta and partial correlation coefficients are reported

ED Symptomatology Eating Disorder Examination Questionnaire, *Impairment Clinical Impairment Assessment*, *EDE-Q* Eating Disorder Examination Questionnaire, *CIA* Clinical Impairment Assessment, *Worry* Penn State Worry Questionnaire, *Depression* Beck Depression Inventory-II, *Psychosocial Functioning* World Health Organization Disability Assessment Schedule 2.0, *BMI* body mass index

* $p < .05$

** $p < .001$

ED symptomatology at 1-year follow-up

Depression, worry, and psychosocial functioning predicted ED symptomatology at 1-year follow-up over and above admission ED symptomatology (see Table 3). Depression, worry, and psychosocial functioning, but not change in BMI, also predicted ED symptomatology at 1-year follow-up in patients with AN. There were no main effects or interactions for age or LOS and depression, worry, or psychosocial functioning on ED symptomatology at follow-up ($ps \geq 0.33$), and there were no significant differences found between ED diagnoses.

Psychological impairment at discharge

Depression and worry predicted psychological impairment at discharge over and above admission psychological impairment in the full sample. Psychosocial functioning was not a significant predictor (see Table 4). Depression

also predicted psychological impairment at discharge in patients with AN, though worry and depression did not. Change in BMI was not found to be a significant predictor of impairment in patients with AN. There were no main effects or interactions for age or LOS and depression, worry, or psychosocial functioning on psychological impairment at discharge ($ps \geq 0.05$), and there were no significant differences found between ED diagnoses.

Psychological impairment at 1-year follow-up

Depression, worry, and psychosocial functioning predicted psychological impairment at 1-year follow-up over and above admission impairment (see Table 4). Worry and psychosocial functioning, but not depression or change in BMI, were also predictors of impairment at 1-year follow-up in patients with AN. There were no main effects or interactions for age or LOS and depression, worry, or psychosocial impairment on psychological impairment at follow-up

Table 5 Multiple regression analyses predicting BMI in patients with AN

Regression 5; discharge BMI in patients with AN				N
Worry	-0.05	-0.05	$p=0.42$	263
Depression	0.04	0.03	$p=0.61$	
Psychosocial functioning	-0.00	-0.00	$p=0.95$	
Admission BMI	0.66**	0.65**	$p<0.001$	
Regression 6; 1-year follow-up BMI in patients with AN				263
Worry	-0.01	-0.01	$p=0.83$	263
Depression	0.12	0.10	$p=0.09$	
Psychosocial functioning	0.29**	0.26**	$p<0.001$	
Admission BMI	0.51**	0.57**	$p<0.001$	

Standardized beta and partial correlation coefficients are reported

Worry Penn State Worry Questionnaire, Depression Beck Depression Inventory-II, Psychosocial Functioning World Health Organization Disability Assessment Schedule 2.0, BMI body mass index

* $p < .05$

** $p < .001$

($ps \geq 0.08$), and there were no significant differences found between ED diagnoses.

Body mass index at discharge (in AN patients only)

No significant predictors of discharge BMI were found in patients with AN (see Table 5). There were no main effects for age or LOS and depression, worry, or psychosocial functioning on discharge BMI in patients with AN. There was a significant interaction between LOS and worry on discharge BMI, such that AN patients with longer LOS and lower worry had higher BMIs at discharge than those with shorter LOS and higher worry ($r = -0.12$, $\beta = -0.98$, $p < 0.05$). There was also a significant interaction between psychosocial functioning and LOS on discharge BMI, such that AN patients with longer LOS and lower psychosocial functioning had higher discharge BMIs than those with shorter LOS and higher psychosocial functioning ($r = -0.21$, $\beta = -1.14$, $p = 0.001$). Interaction graphs are available from first author upon request.

Body mass index in AN at 1-year follow-up

Psychosocial functioning predicted BMI at 1-year follow-up over and above admission BMI in patients with AN. Depression and worry did not significantly predict 1-year follow-up BMI in patients with AN (see Table 5). There were no main effects or interactions for age or LOS, and depression, worry, or psychosocial functioning on BMI at 1-year follow-up in patients with AN ($ps > 0.62$).

Discussion

The aim of this study was to identify predictors of ED symptomatology, psychological impairment, and BMI (in patients with AN only) at the end of treatment and 1 year after discharge from treatment. Our goal was to pinpoint areas of focus in ED treatment settings, which may lead to overall improved ED outcomes. We demonstrated that patients had significant improvement after receiving treatment at an ED treatment center, and they maintained some improvement 1 year following treatment. We also found that depression, worry, and psychosocial functioning were factors which predicted outcome measures (ED symptomatology and psychological impairment, but not BMI in AN).

More specifically, we found that patients had significantly improved ED symptomatology, psychological impairment, and change in BMI (in AN patients only) upon discharge from an ED treatment center (compared to admission). Psychological impairment and change in BMI were not significantly different at 1-year follow-up than at discharge, indicating that improvement was retained. ED symptomatology was significantly higher at 1-year follow-up than at discharge, but it was significantly lower at follow-up than at admission, suggesting that at least some improvement was maintained post discharge. In line with previous research [9–11], it appears that undergoing residential and/or PHP treatment is beneficial for patients with EDs, specifically to reduce ED impairment and increase BMI in patients with AN.

Our primary aim was to identify what might predict these outcomes across time. We identified several significant predictors of ED treatment outcomes. In all patients, regardless of diagnosis, higher levels of depression predicted higher ED symptomatology and psychological impairment at treatment discharge and follow-up. This finding is consistent with other research [2, 6, 15, 28] and suggests that clinicians could focus on depression as part of the ED treatment process, which may lead to improved ED outcomes. Along the same lines, bipolar disorders may be considered as an area of future research to further specify the effects of mood on ED treatment outcomes.

Higher worry predicted higher psychological impairment at both discharge and 1-year follow-up. Previous research on the relationship between anxiety and EDs is scarce, and worry as a distinct element of anxiety had yet to be explored as a predictor of ED outcomes prior to this study. The treatment center in the present study used standard ED treatment, which included therapies that could be used for anxiety and depression but did not specifically target either. Our findings suggest that treatments could be modified to include therapies specific to anxiety and depression, such as cognitive behavior therapy with exposure and behavioral activation [48, 49]. Furthermore, our

findings indicate that worry may be a specific area of focus for clinicians in ED treatment centers to improve long-term outcomes. Future research should continue to explore the relationship between EDs and worry. In addition, the distinction between worry and specific ED fears could be an area of focus for future research, since worry itself is a broad construct encompassing multiple topics (e.g., the future, relationships) [50] and worries related to EDs may be more specific (i.e., fear of becoming fat), therefore producing differential outcomes.

Psychosocial functioning predicted ED symptomatology and psychological impairment at 1-year follow-up, but not at discharge, though the direction was negative. These findings suggest that suppression occurred, meaning that when the other predictors are included in the equation, the relationship between psychosocial functioning and ED symptomatology becomes negative. In other words, the variance that is related to ED symptomatology is accounted for by other factors. We await future replication research before making any conclusions regarding these specific findings, which could be attributed to the measurement of psychosocial functioning.

Though the importance of weight gain in treatment has been supported [13, 23, 24, 51], the present study did not find change in BMI to be a predictor of any outcome variable. In the current sample, the average change in BMI was significant, with most AN patients discharging within a healthy BMI range. Yet, despite the significant change in BMI, the outcomes we investigated did not contribute to who gained more or less weight. This finding may be accounted for by considering supplementary factors that may influence treatment outcome, such as motivation or treatment acceptance. Indeed, Espel et al. (2016) [52] found that patients in a residential ED treatment facility who demonstrated higher experiential acceptance had increased motivation, and this led to decreased ED symptoms at treatment end. Further study on weight gain in AN and the mechanisms that influence treatment outcomes, such as motivation and acceptance, is warranted.

Limitations

There were several limitations to this study. First, patients filled out self-report questionnaires as outcome measures rather than undergoing structured clinical interviews. This methodology poses the risk of limited introspective ability and/or inaccurate interpretation when responding to items. Second, patients self-reported height and weight at 1-year follow-up. It is fair to assume that not all patients had an accurate report of their weight, though some research suggests that self-reported weight is almost as accurate as measured weight [53]. A third limitation

was missing data due to a high attrition rate, though this is not an unusual phenomenon in the ED population [54]. Our sample size was still quite large, and the issue of missing data was remedied using multiple imputation [55]. A fourth limitation was use of the WHODAS 2.0 as a measure of psychosocial functioning, as it is a relatively new measure that had not been previously studied in an ED population and could possibly explain our suppression findings. A fifth limitation is the absence of residential versus PHP percentages due to patients frequently alternating between levels of care. Sixth, patients underwent clinical rather than structured diagnostic interviews as part of their admission process to the treatment center, resulting in decreased reliability and validity of the diagnoses. Finally, the population in the present study was non-homogenous, such that they comprised mixed ED diagnoses and wide ranges in age, LOS, and duration of ED (however, these were accounted for in the analyses). Such variations could impact study findings, though these variations reflect the nature of clinical treatment settings and, thus, have highly applicable clinical relevance.

Conclusions

This study supports the findings of previous research suggesting that depression predicts ED treatment outcomes. In addition, we provide novel data demonstrating that worry as a distinct feature of anxiety is a predictor of ED treatment outcomes. This study also extends the findings which suggest that psychosocial functioning predicts ED treatment outcomes by addressing specific areas of functioning, such as communication and participation in society. Furthermore, the lack of association between outcomes and change in BMI suggests that increased attention on psychosomatic therapies, in conjunction with weight restoration in AN, may be helpful in the treatment of patients with EDs since weight change in BMI alone was not found to influence outcomes in patients with AN. Overall, this research implies that focused attention on depression, worry, and psychosocial functioning during the course of ED treatment may be effective in improving ED symptomatology, psychological impairment, and BMI in patients with AN.

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Compliance with ethical standards

Conflict of interest LF was an employee and LS was the Chief Executive Officer of the eating disorder clinic where data were collected in the present study.

Ethical approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. This retrospective study was approved by the Institutional Review Board at Washington University in St. Louis.

Informed consent We submitted an IRB for this study to Washington University in St. Louis and the IRB deemed this study to not need IRB approval due to its retroactive nature.

References

- Harris EC, Barraclough B (1998) Excess mortality of mental disorder. *Br J Psych* 173:11–53. doi:10.1192/bjp.173.1.11
- Kaye WH, Bulik CM, Thornton L, Barbarich N, Masters K (2004) Comorbidity of anxiety disorders with anorexia and bulimia nervosa. *Am J Psychiatry* 161:2215–2221. doi:10.1176/appi.ajp.161.12.2215
- Pollice C, Kaye W, Greeno CG, Weltzin TE (1997) Relationship of depression, anxiety, and obsessionality to state of illness in anorexia nervosa. *Int J Eat Disord* 21:367–376
- Pearlstein T (2002) Eating disorders and comorbidity. *Arch Women Ment Hlth* 4:67–78
- Pallister E, Waller G (2008) Anxiety in the eating disorders: understanding the overlap. *Clin Psychol Rev* 28:366–386. doi:10.1016/j.cpr.2007.07.001
- Herpetz-Dalman B, Hebebrand J, Müller B, Herpetz S, Heussen N, Remschmidt H (2001) Prospective 10-year follow-up in adolescent anorexia nervosa—Course, outcome, psychiatric comorbidity, and psychosocial adaptation. *J Child Psychol Psych* 42:603–612. doi:10.1111/1469-7610.00756
- Abbate-Daga G, Gramaglia C, Preda S, Comba E, Brustolin A, Fassino S (2009) Day hospital programmes for eating disorders: a review of the similarities, differences and goals. *Eat Weight Disord* 14:e31–e41. doi:10.1007/BF03327798
- McFarlane TL, MacDonald DE, Trottier K, Olmsted MP (2015) The effectiveness of an individualized form of day hospital treatment. *Eat Disord* 23:191–205. doi:10.1080/10640266.2014.981430
- Olmsted MP, McFarlane T, Trottier K, Rockert W (2013) Efficacy and intensity of day hospital treatment for eating disorders. *Psychother Res* 23:277–286. doi:10.1080/10503307.2012.721937
- Fittig E, Jacobi C, Backmund H, Gerlinghoff M, Wittchen HU (2008) Effectiveness of day hospital treatment for anorexia nervosa and bulimia nervosa. *Eur Eat Disord Rev* 16:341–351. doi:10.1002/erv.883
- Goldstein M, Peters L, Baillie A, McVeagh P, Minshall G, Fitzjames D (2011) The effectiveness of a day program for the treatment of adolescent anorexia nervosa. *Int J Eat Disord* 44:29–38. doi:10.1002/eat.20789
- Abbate-Daga G, Marzola E, De-Bacco C, Buzzichelli S, Brustolin A, Campisi S et al (2015) Day hospital treatment for anorexia nervosa: A 12-month follow-up study. *Eur Eat Disord Rev* 23:390–398. doi:10.1002/erv.2369
- Lowe B, Zipfel S, Buchholz C, Dupont Y, Reas DL, Herzog W (2001) Long-term outcome of anorexia nervosa in a prospective 21-year follow-up study. *Psychol Med* 31:881–890. doi:10.1017/S003329170100407X
- Bégin C, Gagnon-Girouard MP, Aimé A, Ratté C (2013) Trajectories of eating and clinical symptoms over the course of a day hospital program for eating disorders. *Eat Disord* 21:249–264. doi:10.1080/10640266.2013.779188
- Fichter MM, Quadflieg N, Hedlund S (2006) Twelve-year course and outcome predictors of anorexia nervosa. *Int J Eat Disord* 39:87–100. doi:10.1002/eat.20215
- Friedman K, Ramirez AL, Murray SB, Anderson LK, Cusack A, Boutelle KN, Kaye WH (2016) A Narrative review of outcome studies for residential and partial hospital-based treatment of eating disorders. *Eur Eat Disord Rev* 24(4):263–276. doi:10.1002/erv.2449
- Franko DL, Keshaviah A, Eddy KT, Krishna M, Davis MC, Keel PK et al (2013) A longitudinal investigation of mortality in anorexia nervosa and bulimia nervosa. *Am J Psychiatry* 170:917–925. doi:10.1176/appi.ajp.2013.12070868
- Goddard E, Hibbs R, Raenker S, Salerno L, Arcelus J, Boughton N et al (2013) A multi-centre cohort study of short term outcomes of hospital treatment for anorexia nervosa in the UK. *BMC Psychiatry* 13:287. doi:10.1186/1471-244X-13-287
- Le Grange D, Accurso E, Lock J, Agras S, Bryson S (2014) Early weight gain predicts outcome in two treatments for adolescent anorexia nervosa. *Int J Eat Disord* 47:124–129. doi:10.1002/eat.22221
- Lock J, Agras WS, Le Grange D, Couturier J, Safer D, Bryson S (2013) Do end of treatment assessments predict outcome at follow-up in eating disorders? *Int J Eat Disord* 46:771–778. doi:10.1002/eat.22175
- Steinhausen HC (2002) The outcome of anorexia nervosa in the 20th century. *Am J Psychiatry* 159:1284–1293. doi:10.1176/appi.ajp.159.8.1284
- Dingemans AE, van Son GE, Aardoom JJ, Bruidegom K, Slof-Op Landt, MCT, van Furth EF (2016) Predictors of psychological outcome in patients with eating disorders: a routine outcome monitoring study. *Int J Eat Disord* 49:863–873. doi:10.1002/eat.22560
- Darcy AM, Katz S, Fitzpatrick KK, Forsberg S, Utzinger L, Lock J (2010) All better? How former anorexia nervosa patients define recovery and engaged in treatment. *Eur Eat Disord Rev* 18:260–270. doi:10.1002/erv.1020
- Fichter MM, Quadflieg N (1999) Six-year course and outcome of anorexia nervosa. *Int J Eat Disord* 26:359–385. doi:10.1002/(SICI)1098-108X(199912)26:4<359::AID-EAT2>3.0.CO;2-7
- Godart N, Radon L, Curt F, Duclos J, Perdereau D, Lang F (2015) Mood disorders in eating disorder patients: prevalence and chronology of ONSET. *J Affect Disord* 185:115–122. doi:10.1016/j.jad.2015.06.039
- Keel PK, Mitchell JE, Miller KB, Davis TL, Crow SJ (2000) Predictive validity of bulimia nervosa as a diagnostic category. *Am J Psychiatry* 157:136–138. doi:10.1176/ajp.157.1.136
- Voderholzer U, Witte S, Schlegl S, Koch S, Cuntz U, Schwartz C (2016) Association between depressive symptoms, weight and treatment outcome in a very large anorexia nervosa sample. *Eat Weight Disord* 21(1):127–131. doi:10.1007/s40519-015-0227-7
- Rossiter EM, Agras WS, Telch CF, Schneider JA (1993) Cluster B personality disorder characteristics predict outcome in the treatment of bulimia nervosa. *Int J Eat Disord* 13:349–357. doi:10.1002/1098-108X(199305)13:4<349::AID-EAT2260130403>3.0.CO;2-C
- Stice E (2002) Risk and maintenance factors for eating pathology: a meta-analytic review. *Psychol Bull* 128:825–848. doi:10.1037/0033-2909.128.5.825
- Puccio F, Fuller-Tyszkiewicz M, Ong D, Krug I (2016) A systematic review and meta-analysis on the longitudinal relationship between eating pathology and depression. *Int J Eat Disord* 49:439–454. doi:10.1002/eat.22506
- Swinbourne J, Hunt C, Abbott M, Russell J, Clare T, Touyz S (2006) The comorbidity between eating disorders and anxiety

- disorders: Prevalence in an eating disorder sample and anxiety disorder sample. *Aust N Z J Psychiatry* 46:118–131. doi:10.1177/0004867411432071
32. Kerkhof A, Hermas D, Figea A, Laeremans I, Pieters G, Aardema A (2000) The Penn State Worry Questionnaire and the Worry Domains Questionnaire: first results in Dutch and Flemish in- and outpatient groups. *Gedragstherapie* 33:135–145
 33. Baran SA, Weltzin T, Kaye E, Walter H (1995) Low discharge weight and outcome in anorexia nervosa. *Am J Psychiatry* 152:1070–1072. doi:10.1176/ajp.152.7.1070
 34. Lund BC, Hernandez ER, Yates WR, Mitchell JR, McKee PA, Johnson CL (2009) Rate of inpatient weight restoration predicts outcome in anorexia nervosa. *Int J Eat Disord* 42:301–305. doi:10.1002/eat.20634
 35. Mayer L, Walsh BT, Pierson RN Jr, Heymsfield SB, Gallagher D, Wang J et al (2005) Body fat redistribution after weight gain in women with anorexia nervosa. *Am J Clin Nutr* 81:1286–1291
 36. Fairburn CG (2008) Cognitive behavior therapy and eating disorders. The Guilford Press, New York
 37. Bohn K, Fairburn CG (2008) The Clinical Impairment Assessment questionnaire (CIA 3.0). In: Fairburn CG (ed) Cognitive behavior therapy and eating disorders. Guilford Press, New York. doi:10.1007/978-981-287-087-2_85-1
 38. Beck AT, Steer RA, Brown GK (1996) Manual for the Beck Depression Inventory-II. Psychological Corporation, San Antonio
 39. Meyer TJ, Miller ML, Metzger RL, Borkovec TD (1990) Development and validation of the Penn State Worry Questionnaire. *Behav Res Ther* 28:487–495. doi:10.1016/0005-7967(90)90135-6
 40. Ustün TB, Kostanjsek N, Chatterji S, Rehm J (eds) (2010) Measuring health and disability: Manual for WHO Disability Assessment Schedule (WHODAS 2.0). World Health Organization, Geneva
 41. Luce KH, Crowther JH (1999) The reliability of the Eating Disorder Examination Self-Report Questionnaire Version (EDE-Q). *Int J Eat Disord* 25:349–351. doi:10.1002/(SICI)1098-108X(199904)25:3<349::AID-EAT15>3.0.CO;2-M
 42. Mond JM, Hay P, Rodger B, Owen C, Beumont PJV (2004) Validity of the Eating Disorder Examination (EDE-Q) in screening for eating disorders in community samples. *Behav Res Ther* 42:551–567. doi:10.1016/S0005-7967(03)00161-X
 43. Vannucci A, Kass AE, Sinton MM, Aspen V, Weisman H, Bailey JO et al (2012) An examination of the clinical impairment assessment among women at high risk for eating disorder onset. *Behav Res Ther* 50:407–414. doi:10.1016/j.brat.2012.02.009
 44. Storch EA, Roberti JW, Roth DA (2004) Factor structure, concurrent validity, and internal consistency of the beck depression inventory—second edition in a sample of college students. *Depress Anxiety* 19:187–189. doi:10.1002/da.20002
 45. Stöber J (1998) Reliability and validity of two widely-used worry questionnaires: self-report and self-peer convergence. *Pers Individ Dif* 24:887–890. doi:10.1016/S0191-8869(97)00232-8
 46. Adult BMI Calculator (n.d.). In Centers for Disease Control and Prevention online. http://www.cdc.gov/healthyweight/assessing/bmi/adult_bmi/english_bmi_calculator/bmi_calculator.html. Accessed 20 May 2016.
 47. Honaker J, King G, Blackwell M. AMELIA II: A program for missing data (2015). Retrieved from <https://cran.r-project.org/web/packages/Amelia/vignettes/amelia.pdf>
 48. Beck AT, Emery G, Greenberg RL (2005) Anxiety disorders and phobias: a cognitive perspective. Basic Books, Cambridge
 49. Lewinsohn PM, Biglan A, Zeiss AS (1976) Behavioral treatment of depression. In: Davidson PO (ed) The behavioral management of anxiety, depression and pain. Brunner/Mazel, New York, pp 91–146
 50. Andrews G, Hobbs MJ, Borkovec TD, Beesdo K, Craske MG, Heimberg RG, Rapee RM, Ruscio AM, Stanley MA (2010) Generalized worry disorder: a review of DSM-IV generalized anxiety disorder and options for DSM-V. *Depress Anxiety* 27(2):134–147. doi:10.1002/da.20658
 51. Vall E, Wade TD (2016) Predictors and moderators of outcomes and readmission for adolescent inpatients with anorexia nervosa: a pilot study. *Clin Psych*. doi:10.1111/cp.12091 (in press)
 52. Espel HM, Goldstein SP, Manasse SM, Juarascio AS (2016) Experiential acceptance, motivation for recovery, and treatment outcome in eating disorders. *Eat Weight Disord* 21(2):205–210. doi:10.1007/s40519-015-0235-7
 53. Stunkard AJ, Albaum JM (1981) The accuracy of self-reported weights. *Am J Clin Nutr* 34:1593–1599
 54. Herzog DB, Deter HC, Fiehn W, Petzold E (1997) Medical findings and predictors of long-term physical outcome in anorexia nervosa: a prospective 12 year follow-up study. *Psychol Med* 27:269–279
 55. Sinharay S, Stern HS, Russell D (2001) The use of multiple imputation for the analysis of missing data. *Psychol Methods* 6:317. doi:10.1037/1082-989X.6.4.317