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Prevalence and correlates of eating disorder co-morbidity in patients with bipolar disorder[1]

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Abstract

This study was designed to document eating disorder symptoms in a well-defined sample of patients with bipolar disorder and to evaluate the relationship of current loss of control over eating (LOC) to demographic and clinical features hypothesized to characterize bipolar patients at risk for disordered eating. Eighty-one patients enrolled in the Bipolar Disorder Center for Pennsylvanians provided demographic information and completed the Structured Clinical Interview for DSM-IV Axis I Disorders. The Eating Disorder Examination was administered by independent clinicians to evaluate current and lifetime eating disorder symptomatology. Twenty-one percent of participants met DSM-IV criteria for a lifetime eating disorder, and 44% reported a history of LOC. Patients who endorsed weekly LOC during the past six months (n = 18) were heavier, had more atypical depressive symptoms, and were more likely to have a lifetime substance use disorder compared to patients in the rest of the sample (n = 63). These findings indicate that eating disorder symptoms are prevalent in patients with bipolar disorder and are associated with obesity and other psychiatric morbidity. Screening for eating disorders in bipolar patients is warranted, as intervention may minimize distress and improve treatment outcome.

Keywords

Mood disorders; anorexia nervosa; bulimia nervosa; binge eating; obesity

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1. Introduction

There is growing recognition of the clinical significance of eating disorder co-morbidity in patients with bipolar disorder. Previous work has documented an association between eating disorder symptoms and obesity (McElroy et al., 2002), suicidal ideation (Post et al., 2003), and residual mood disorder symptoms (MacQueen et al., 2003) in bipolar patients. However, the prevalence and correlates of eating disorder co-morbidity in patients with bipolar disorder remain poorly understood. Although available data suggest that eating disordered behaviors,

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especially binge eating, may be common in this group (Kruger et al., 1996; Ramacciotti et al., 2005), few studies have used standardized measures to evaluate the full range of eating disorder psychopathology in large samples of patients with bipolar disorder (McElroy et al., 2005). For example, binge eating is defined in the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV; APA, 1994) as eating "an amount of food that is definitely larger than most people would eat" coupled with "a sense of lack of control over eating during the episode" (p. 549). However, there is widespread agreement in the eating disorders field that loss of control over eating (LOC) is more salient to psychiatric distress than is the amount of food consumed (Niego et al., 1997; Pratt et al., 1998; Keel et al., 2001; Mond et al., 2006). Clinical research has shown that individuals with bulimia nervosa (BN) and binge eating disorder (BED) have difficulty distinguishing between objectively large and subjective binge episodes, and both types of binge eating are common in these groups (Rossiter and Agras, 1990; Telch et al., 1998). However, no study to date has evaluated the prevalence and correlates of LOC in patients with bipolar disorder.

A number of demographic and clinical characteristics may be associated with LOC in patients with bipolar disorder. For example, research has demonstrated that female bipolar patients are more likely than males to have a co-morbid eating disorder (Kawa et al., 2005). There also is evidence that obese bipolar patients are more likely than are normal weight or overweight patients to have an eating disorder, particularly BED (McElroy et al., 2002), although the direction of this effect has yet to be elucidated. Atypical depressive symptoms are another potential correlate of LOC in patients with bipolar disorder. Indeed, elevated rates of both binge eating and bipolar disorder have been observed in individuals with atypical depressive symptomatology (Kendler et al., 1996; Benazzi, 1999; Angst et al., 2002). Moreover, some investigators have speculated that patients with so-called "soft bipolar spectrum" disorders may be more likely than patients with bipolar I to experience atypical depressive symptoms and eating disorders (Perugi and Akiskal, 2002). Finally, it is well documented that both eating disorders and bipolar disorder co-occur with substance use and anxiety disorders (McElroy et al., 2005). However, no study to date has evaluated the relationship between other psychiatric co-morbidity and eating disorders in patients with bipolar disorder.

Exposure to medications associated with a high degree of risk for weight gain also may be related to LOC in patients with bipolar disorder. A recent review of the relationship between pharmacotherapy and weight gain in bipolar patients hypothesized that individuals with co-morbid binge eating might be particularly vulnerable to the appetite-stimulating effects of some agents (Keck and McElroy, 2003). Many of the psychotropic medications used to treat bipolar disorder, including mood stabilizers, second-generation antipsychotics, and antidepressants have been associated with increased appetite and weight gain in psychiatric patients (Fava, 2000; Malhi et al., 2001; Keck and McElroy, 2003). Moreover, individuals with bipolar disorder frequently have medical co-morbidities (Kupfer, 2005) that may require treatment with non-psychotropic medications that are associated with weight gain such as insulin (Heller, 2004) and beta blockers (Pischon and Sharma, 2001).

Thus it appears that patients with bipolar disorder may have elevated rates of eating disorder psychopathology by virtue of the clinical characteristics of their illness and the treatments used to alleviate psychiatric symptoms and medical co-morbidities. In this study, we document current and lifetime eating disorder symptoms and diagnoses in a well-defined sample of patients seeking treatment for bipolar disorder. We then examine the relationship of weekly LOC during the past six months to demographic and clinical features hypothesized to characterize bipolar patients at risk for disordered eating.

2. Methods

2.1. Participants—This project was an ancillary study to the Bipolar Disorder Center for Pennsylvanians (BDCP), a multi-center randomized controlled trial comparing the efficacy of guideline-based pharmacotherapy alone to pharmacotherapy plus psychosocial intervention in the treatment of patients with bipolar I, bipolar II, bipolar not otherwise specified (NOS), and schizoaffective disorder bipolar type. All adults (age ≥ 18 years) enrolled at the Pittsburgh site of the BDCP between January 2005 and April 2006 were eligible to participate (N = 339). Consistent with inclusion and exclusion criteria for the parent study, subjects were required to have an IQ greater than 70, and individuals with rapid cycling bipolar disorder were excluded.

Eighty-seven BDCP patients (25.7% of eligible individuals) responded to advertisements for research study participants. After complete description of the study, 84 patients signed written informed consent forms approved by the University of Pittsburgh Institutional Review Board. Three patients were excluded from the study after informed consent was obtained because their eating disorder history could not be reliably coded, resulting in a final sample of 81.

Participants (N = 81) were 65.4% female, 72.8% white (27.2% black), and 30.9% married or cohabiting; 48.1% had a four-year college or advanced degree. Mean age was 43.1 (±10.6) years, and mean BMI was 31.1 (± 8.0) kg/m^2. Fifty-nine patients (72.8%) had bipolar I disorder, 17 (21.0%) had bipolar II, and 5 (6.2%) had bipolar NOS (n = 3) or schizoaffective disorder bipolar type. Medications at the time of the current study are presented in Table 1. There were no differences between eligible patients who did (n = 81) and did not (n = 258) participate in the current study with respect to age (P = 0.11), sex (P = 0.67), ethnicity (P = 0.99), years of education (P = 0.12), BMI (P = 0.15), or bipolar disorder diagnosis (P = 0.71). Participants and non-participants also did not differ on lifetime rates of anorexia nervosa (AN) (P = 0.34), BN (P = 0.57) or eating disorder NOS, including BED (P = 0.64) at entry into the BDCP protocol.

2.2. Measures—Patients provided demographic information and completed the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I; First et al., 1995) at entry into the BDCP protocol (310.6 ± 189.9 days prior to participation in the current study). Severity of current depressive and manic symptoms was assessed using the Bipolar Disorder Visit Form (BDVF), a clinician-rated instrument that includes the Clinical Global Impression – Severity Scale – Bipolar Version (CGI-S-BP; Spearing et al., 1997). Participants completed the BDVF 7.5 ± 7.6 days from their eating disorder assessment. Participants' heights and weights were measured after completion of the eating disorder assessment, and BMI was calculated as weight (kg)/height(m)^2.

Eating disorder symptoms and diagnoses were assessed using the 12th edition of the Eating Disorder Examination (EDE; Fairburn and Cooper, 1993), a standardized, investigator-administered interview designed to evaluate DSM-IV criteria for AN and BN. The version of the EDE employed in the present study also included items to evaluate BED, a provisional diagnostic category in DSM-IV. An extensive body of research supports the psychometric properties of the EDE, including its superiority over general semi-structured interviews in the assessment of binge eating (Fairburn and Cooper, 1993; Wade et al., 1997). The EDE distinguishes two forms of binge eating: 1) objective binge episodes (i.e., loss of control accompanied by the consumption of a large amount of food as determined by the interviewer); and 2) subjective binge episodes (i.e., loss of control without the consumption of an "objectively" large amount of food, although the participant reports excessive eating).

EDE interviews were conducted by a trained clinical psychologist (n = 1), or a psychiatric social worker (n = 1). Interviewers were blind to demographic and clinical characteristics of participants. Consistent with previous research (Tanofsky-Kraff et al., 2003; Glasofer et al.,

2007), ratings of LOC and determinations of the size of binge episodes were done by consensus at weekly rating meetings attended by all EDE interviewers in the second author's laboratory (n = 8). Threshold-level eating disorder diagnoses were confirmed using the SCID-I, which was administered independently of the current study.

2.3. Analytic plan—We used descriptive statistics to evaluate the prevalence of current and lifetime eating disorder symptoms and diagnoses. Chi square analyses were performed to determine whether rates of eating disorder psychopathology differed by sex. Next, we performed a series of chi square analyses (categorical variables) and independent samples t-tests (continuous variables) comparing participants who did and did not report weekly LOC during the past six months (Weekly LOC group) on demographic and clinical characteristics. All tests were two-tailed with alpha level set at $P < 0.05$.

Although DSM-IV criteria require an average of two objective binge episodes per week for 3–6 months depending on diagnosis, there is little data supporting the validity of these thresholds. Studies comparing individuals with once weekly versus twice weekly binge eating have found few differences on measures of general psychopathology, dieting, weight history, or body image disturbance (Garfinkel et al., 1995; Striegel-Moore et al., 1998; Striegel-Moore et al., 2000). Moreover, findings from both community and clinic samples have led several investigators to suggest that the experience of LOC may be a better index of psychiatric distress than is the amount of food consumed (Niego et al., 1997; Pratt et al., 1998; Keel et al., 2001; Mond et al., 2006). Considering only patients in the Weekly LOC group (n = 18), 7 (38.9%) reported only objective binge episodes, 1 (5.6%) reported only subjective binge episodes, and 10 (55.6%) reported both objective and subjective binge episodes. There were no differences between individuals who reported once weekly (n = 6) versus twice weekly (n = 12) LOC or individuals who reported objective (n = 7) versus subjective (n = 11) binge episodes on age, sex, BMI, or bipolar disorder diagnosis (P's ≥ 0.09).

Finally, we conducted a logistic regression to determine whether demographic and clinical features hypothesized to characterize bipolar patients at risk for disordered eating were associated with weekly LOC during the past six months. Following the guidelines of Steyerberg and Harrell (2003), we selected a set of predictors a priori rather than relying on univariate analyses or stepwise selection procedures to identify significant covariates. The dependent variable was membership in the Weekly LOC group, and the independent variables included sex, BMI, bipolar disorder diagnosis (bipolar I versus other), severity of current atypical depressive symptoms (0–1 versus 2–4 symptoms), exposure to high risk (for weight gain) medications (≥ 30 days exposure versus < 30 days or no exposure), and lifetime anxiety and substance use co-morbidity. The BMI distribution was subjected to a $\log_{10}$ transformation to correct for positive skewness and outliers. Wald's statistic was used to determine which of the predictors contributed significantly to the model. All analyses were conducted using SPSS 14.0 for Windows.

3. Results

3.1. Loss of control over eating—Eighteen patients (22.2%) reported at least one objective or subjective binge episode per week on average during the past six months (M = 68.4 ± 44.0 LOC days), and 36 patients (44.4%) endorsed a history of LOC. Table 2 presents demographic and clinical characteristics of patients in the Weekly LOC group compared to the rest of the sample. As shown, weekly LOC during the past six months was associated with increased BMI and obesity, a greater number of atypical depressive symptoms, and a higher rate of lifetime substance use co-morbidity in the univariate analyses.

3.2. Compensatory behaviors—Seven patients (8.6%) endorsed current ($n = 4$) or lifetime episodes of self-induced vomiting, and nine patients (11.1%) endorsed lifetime episodes of laxative abuse. Twelve patients (14.8%) endorsed current ($n = 2$) or lifetime episodes of recurrent fasting to lose weight. There were no gender differences in prevalence of current or lifetime fasting or self-induced vomiting (P's ≥ 0.45); women were significantly more likely than men to have a lifetime history of laxative abuse (X^2 [1] = 5.35, $P = 0.02$).

3.3. Eating disorder diagnoses—Prevalence rates for current and lifetime eating disorder diagnoses are presented in Table 3. Six patients had a threshold-level eating disorder at the time of assessment (all BED). An additional five patients met DSM-IV criteria for BED except: 1) the frequency of objective binge eating was 1/week instead of 2/week ($n = 3$), or 2) they denied distress related to binge eating ($n = 2$).

Lifetime prevalence of eating disorders was 21.0% ($n = 17$). The rates of AN and BN were particularly striking (Table 3). There were no gender differences in rates of current ($P = 0.34$) or lifetime ($P = 0.28$) eating disorder co-morbidity; however, at a trend level, women were more likely than men to have a lifetime diagnosis of AN (X^2 [1] = 3.42, $P = 0.06$).

3.4. Correlates of weekly LOC during the past six months—Table 4 presents results of a logistic regression predicting weekly LOC during the past six months from demographic and clinical characteristics hypothesized to identify bipolar patients at risk for disordered eating. As shown, BMI, atypical depressive symptoms, and lifetime substance use co-morbidity were significantly associated with current LOC in the multivariate model.

4. Discussion

This study used state-of-the-art methodology to evaluate the full range of eating disorder psychopathology in a well-defined sample of patients with bipolar disorder. Consistent with previous research (MacQueen et al., 2003; Ramacciotti et al., 2005), BN and BED were the most common eating disorders in our sample, with lifetime prevalence rates of 8.6% and 11.1%, respectively. However, the lifetime prevalence of AN also was striking (11.3% of female patients; 7.4% of the full sample), especially in comparison to general population estimates of approximately 1% in women and 0.1% in men (Hoek and van Hoeken, 2003). Few studies have evaluated rates of AN in bipolar patients using structured interviews and DSM-IV criteria, and no study has compared the prevalence in male versus female bipolar patients. However, our findings are consistent with two existing reports that found elevated rates of AN in bipolar patients relative to general population estimates (McElroy et al., 2001; MacQueen et al., 2003). The present results also converge with studies showing increased rates of bipolar disorder in patients with AN relative to normal controls (Halmi et al., 1991), as well as research demonstrating familial aggregation of eating disorders and bipolar disorder (Mangweth et al., 2003).

This study replicates and extends work suggesting that obese bipolar patients are at elevated risk for eating disorder psychopathology (McElroy et al., 2002). Indeed, patients who reported weekly LOC during the past six months were significantly heavier than were other bipolar patients and more likely to be obese. It remains to be determined whether eating disorder symptoms contribute to the onset of obesity in patients with bipolar disorder. However, given the documented association of obesity with increased medical burden and poor psychiatric outcome (Wildes et al., 2006), future studies to elucidate the relationship among eating disorders, obesity, and bipolar disorder seem warranted.

The present findings also converge with the results of studies documenting an association between atypical depressive symptoms and eating disorder co-morbidity in individuals with mood disorders (Angst et al., 2002; Angst et al., 2006). Sixty-seven percent of patients who

endorsed weekly LOC during the past six months reported two or more current atypical depressive symptoms, as compared to 30% of patients without weekly LOC. The most frequently endorsed atypical depressive symptom in the Weekly LOC group was fatigue followed by hyperphagia, psychomotor retardation, and hypersomnia. Some investigators have speculated that the observed relationship between binge eating and atypical depression is an operational artifact given that overeating is a symptom of both conditions (Angst et al., 2006). However, other researchers have documented significantly higher rates of BN in the co-twins of women with atypical depression (Kendler et al., 1996), suggesting that there may be a common vulnerability for atypical depression and binge eating.

Although some investigators have hypothesized that there may be a unique relationship between bipolar II and its variants and eating disorders (Perugi and Akiskal, 2002), bipolar disorder diagnosis was unrelated to weekly LOC in this study. The only previous empirical study to address this question also found no differences in current or lifetime eating disorder co-morbidity between patients with bipolar I and bipolar II disorder (McElroy et al., 2001). These findings may suggest that there is a non-specific relationship between bipolar illness and eating disorder psychopathology. However, future investigations with larger numbers of "soft spectrum" bipolar patients are needed to evaluate this hypothesis fully.

We also examined the association between weekly LOC and other psychiatric co-morbidity in patients with bipolar disorder. The majority of patients in this study (63.0%) had a lifetime anxiety disorder, and there was no relationship between anxiety co-morbidity and LOC. However, there was a significant association between substance use disorder co-morbidity and LOC. Both substance use and binge eating have been hypothesized to serve a functional role in regulating affective symptomatology among patients with bipolar disorder (Cassano et al., 2000; Wildes et al., 2006). It also has been suggested that substance misuse may reflect elevated levels of impulsivity, a trait thought to underlie both binge eating and bipolar disorder (McElroy et al., 2005).

This study provides limited information about the role of medication in the onset and maintenance of eating pathology in bipolar disorder. Although we found no association between pharmacotherapy and weekly LOC, there are a number of challenges inherent in coding medications as "high" versus "low" risk for weight gain that may have obscured the true relationship between medication history and disordered eating in this group. First, there are differences among pharmacological agents with respect to the characteristics of patients at greatest risk for weight gain. For example, lithium has been associated with weight gain among obese bipolar patients (Bowden et al., 2006), while olanzapine has been shown to produce weight gain in normal weight and overweight patients (Lipkovich et al., 2006). Second, the timing of weight gain appears to differ across agents such that the period of "high risk" may occur during either acute therapy or long-term maintenance (Fava, 2000; Vania et al., 2002). Collapsing medications together into a single group makes it impossible to account for these distinctions. Moreover, we had no data regarding patients' medication histories prior to enrollment in the BDCP and, thus, were unable to control for the potential effects of past medication exposure on current LOC. Third, the mechanisms responsible for medication-induced weight gain are largely unknown and may be irrelevant to LOC. For example, beta blockers have been hypothesized to produce weight change via reduction in resting energy expenditure (Pischon and Sharma, 2001), which is unlikely to influence eating disorder behavior. Finally, the amount of weight change produced by medications used to treat bipolar disorder varies from moderate to very large (Vania et al., 2002); however, there is no consensus as to the degree of weight gain required for an agent to be considered "high risk." Future research should address these limitations by examining the role of specific medications (e.g., olanzapine) in the onset and maintenance of eating disorder symptoms in bipolar disorder. Given the widespread prevalence of obesity in this population (Wildes et al., 2006), a more

detailed exploration of medication history relative to the development of eating and weight problems is needed.

The current findings must be interpreted in light of the limitations of this research. First, participants were enrolled in a clinical trial and responded to advertisements for an ancillary study; therefore, they may not be representative of the population of individuals with bipolar disorder. Although there were no differences between eligible patients who did and did not participate in the current study with respect to demographic and clinical characteristics (including lifetime rates of eating disorder co-morbidity), it is still possible that we oversampled individuals with eating problems. Second, the sample size was small, especially in the Weekly LOC group, which may limit the stability and generalizability of the findings. Third, there was no control group (e.g., patients with unipolar depression, healthy controls), which may have led to over-diagnosis of eating disorders in the present sample. Fourth, eating disorder symptoms were assessed retrospectively and thus may be subject to recall bias. Finally, the present study focused exclusively on adult bipolar patients. Future research is needed to document the prevalence and correlates of eating disorder symptomatology in children and adolescents with bipolar disorder.

In conclusion, results of the present study confirm high rates of eating disorder psychopathology in patients with bipolar disorder that are associated with obesity and other psychiatric morbidity. Screening for eating disorders in bipolar patients is warranted, as intervention may minimize distress and improve treatment outcome.

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Table 1

Pharmacotherapy at Entry into the Current Study ($N = 81$)*

	n	%
High Risk for Weight Gain		
Lithium	40	49.4
Divalproex	21	25.9
Olanzapine	12	14.8
Quetiapine	8	9.9
Risperidone	6	7.4
MAOIs (tranylcypromine)	1	1.2
Tricyclic antidepressants (desipramine)	1	1.2
Paroxetine	4	4.9
Insulin	2	2.5
Beta blockers	6	7.4
Lower Risk or No Risk for Weight Gain		
Carbamazepine, lamotrigine, topiramate, gabapentin	35	43.2
Aripiprazole, ziprasidone	26	32.1
First generation antipsychotics (perphenazine)	1	1.2
SSRIs (except paroxetine)	20	24.7
Atypical antidepressants (no subject was taking mirtzapine)	17	21.0
Benzodiazepine anxiolytics/hypnotics	28	34.6
Opiates	2	2.5
Stimulants/ADHD medications	4	4.9
Antiparkinsonian medications	5	6.2
Other non-psychotropic medications	49	60.5

MAOI = Monoamine Oxidase Inhibitor; SSRI = Serotonin-Specific Reuptake Inhibitor; ADHD = Attention-Deficit Hyperactivity Disorder.

*Classification of medications as high risk versus lower risk or no risk was based on data from the following empirical studies and literature reviews: Fava (2000); Malhi et al. (2001); Jallon and Picard (2001); Pischon and Sharma (2001); Vania et al. (2002); Keck and McElroy (2003); Heller (2004). The majority of patients (86.4%; $n = 70$) were taking ≥ 1 psychotropic medication at entry into the current study; 60.5% ($n = 49$) also were taking at least one non-psychotropic medication.

Table 2

Demographic and Clinical Characteristics of Patients Who Did and Did Not Report Weekly Loss of Control over Eating (LOC) During the Past 6 Months

	Weekly LOC (*n* = 18)	No Weekly LOC (*n* = 63)	*P*
Age (years)	*M*(*SD*) = 45.0(9.5)	*M*(*SD*) = 42.6(10.9)	0.407
BMI (kg/m^2)	*M*(*SD*) = 35.0(8.7)	*M*(*SD*) = 30.0(7.5)	0.020
Obesity			
BMI ≥ 30	72.2% (*n* = 13)	44.4% (*n* = 28)	0.038
BMI < 30	27.8% (*n* = 5)	55.6% (*n* = 35)	
Sex			
Female	77.7% (*n* = 14)	61.9% (*n* = 39)	0.212
Male	22.2% (*n* = 4)	34.8% (*n* = 24)	
Ethnicity			
Black	38.9% (*n* = 7)	23.8% (*n* = 15)	0.205
White	61.1% (*n* = 11)	76.2% (*n* = 48)	
Marital Status*			
Never married	44.4% (*n* = 8)	39.7% (*n* = 25)	
Married or Cohabiting	22.2% (*n* = 4)	33.3% (*n* = 21)	0.622
Widowed, divorced or separated	33.3% (*n* = 6)	25.4% (*n* = 16)	
Years of Education			
High school or less	5.6% (*n* = 1)	15.9% (*n* = 10)	
Some college/technical school	55.5% (*n* = 10)	33.3% (*n* = 21)	0.191
4-year college degree +	38.9% (*n* = 7)	50.8% (*n* = 32)	
Bipolar Disorder Diagnosis			
Bipolar I	66.7% (*n* = 12)	74.6% (*n* = 47)	
Bipolar II	27.8% (*n* = 5)	19.0% (*n* = 12)	0.702
Bipolar NOS	5.6% (*n* = 1)	3.2% (*n* = 2)	
Schizoaffective, bipolar type	0	3.2% (*n* = 2)	
Current Atypical Depressive Symptoms			
0–1 symptom	33.3% (*n* = 6)	69.8% (*n* = 44)	0.005
2–4 symptoms	66.7% (*n* = 12)	30.2% (*n* = 19)	
Lifetime Anxiety Disorder Co-morbidity			
Yes	72.2% (*n* = 13)	60.3% (*n* = 38)	0.356
No	27.8% (*n* = 5)	39.7% (*n* = 25)	
Lifetime Substance Use Disorder Co-morbidity			
Yes	72.2% (*n* = 13)	44.4% (*n* = 28)	0.038
No	27.8% (*n* = 5)	55.6% (*n* = 35)	

BMI = Body mass index; NOS = Not otherwise specified; CGI-S-BP = Clinical Global Impression – Severity Scale – Bipolar Version.

*Marital status data was missing for one patient in the No Weekly LOC group.

Table 3

Threshold-level Eating Disorder Co-morbidity in Patients with Bipolar Disorder

	Female (*n* = 53)		Male (*n* = 28)		Total (*N* = 81)	
Diagnosis	***n***	**%**	***n***	**%**	***n***	**%**
			Current			
BED	5	9.4	1	3.6	6	7.4
			Lifetime			
AN	6	11.3	0	0	6	7.4
BN	5	9.4	2	7.1	7	8.6
BED	7	13.2	2	7.1	9	11.1
Any Eating Disorder	13*	24.5	4	14.3	17	21.0

AN = anorexia nervosa; BN = bulimia nervosa; BED = binge eating disorder.

*Female total does not sum to 18 because 4 women had more than 1 lifetime eating disorder diagnosis as follows: AN + BN (*n* = 1); AN + BED (*n* = 1); BN + BED (*n* = 1); AN + BN + BED (*n* = 1).

Table 4

Logistic Regression Predicting Weekly Loss of Control over Eating (LOC) During the Past 6 Months in Patients with Bipolar Disorder*

Factor	B	SE (β)	Wald	*P*
Sex	1.36	0.812	2.87	0.090
BMI	6.16	2.98	4.27	0.039
Diagnosis of Bipolar I Disorder	0.120	0.717	0.028	0.867
Current Atypical Depressive Symptoms†	1.77	0.653	7.36	0.007
Exposure to High Risk Medication	−0.454	0.638	0.507	0.476
Lifetime Anxiety Disorder Co-Morbidity	−0.106	0.747	0.020	0.887
Lifetime Substance Use Disorder Co-Morbidity	1.39	0.706	3.88	0.049

BMI = Body mass index.

*Overall X^2 ($df = 7$, $N = 81$) = 20.83, $P = 0.004$.

†The most frequently endorsed atypical depressive symptom in the Weekly LOC group was fatigue (83.3%; $n = 15$) followed by hyperphagia and psychomotor retardation (both 38.9%; $n = 7$), and hypersomnia (22.2%; $n = 4$).