Bipolar Disord. Author manuscript; available in PMC 2013 January 14.

Published in final edited form as:
Bipolar Disord. 2009 September ; 11(6): 650–656. doi:10.1111/j.1399-5618.2009.00730.x.

Can body mass index help predict outcome in patients with bipolar disorder?

Cynthia Calkin[a], **Caroline van de Velde**[b], **Martina Růžičková**[a], **Claire Slaney**[c], **Julie Garnham**[c], **Tomas Hajek**[a,d], **Claire O'Donovan**[a], and **Martin Alda**[a,d]

[a]Department of Psychiatry, Dalhousie University, Halifax, Nova Scotia, Canada

[b]Department of Psychology, Concordia University, Montreal, Quebec, Canada

[c]Capital District Health Authority, Halifax, Nova Scotia, Canada

[d]Department of Psychiatry, Third Faculty of Medicine, Charles University, Prague, Czech Republic

Abstract

Objective—Several studies have reported higher prevalence of obesity in patients suffering from bipolar disorder (BD). To study the relation of elevated body mass index (BMI) in patients with BD more closely, we investigated differences in sociodemographic, clinical, and medical characteristics with respect to BMI, with the hypothesis that BMI is related to prognosis and outcome.

Methods—We measured the BMI of 276 subjects of a tertiary care sample from the Maritime Bipolar Registry. Subjects were 16 to 83 years old, with psychiatric diagnoses of bipolar I disorder (n = 186), bipolar II disorder (n = 85), and BD not otherwise specified (n = 5). The registry included basic demographic data and details of the clinical presentation. We first examined the variables showing a significant association with BMI; subsequently, we modeled the relationship between BMI and psychiatric outcome using structural equation analysis.

Results—The prevalence of obesity in our sample was 39.1%. We found higher BMI in subjects with a chronic course ($p < 0.001$) and longer duration of illness ($p = 0.02$), lower scores on the Global Assessment of Functioning Scale ($p = 0.02$), and on disability ($p = 0.002$). Overweight patients had more frequent comorbid subthreshold social ($p = 0.02$) and generalized anxiety disorders ($p = 0.05$), diabetes mellitus type II ($p < 0.001$), and hypertension ($p = 0.001$). Subjects who achieved complete remission of symptoms on lithium showed significantly lower BMI ($p = 0.01$).

Conclusions—Our findings suggest that BMI is associated with the prognosis and outcome of BD. Whether this association is causal remains to be determined.

Corresponding author: Martin Alda, MD, FRCPC, Department of Psychiatry, Dalhousie University, 5909 Veterans Memorial Lane, Halifax, Nova Scotia B3H 2E2, Canada, Fax: 902-473-4877, malda@dal.ca.

Results of this study were presented at the annual conference of the American Psychiatric Association, May 3–8, 2008, Washington DC, USA.

None of the authors declares any conflict of interest with respect to the treatments investigated by this study.

Keywords

bipolar disorder; body mass index; BMI; clinical characteristics; clinical correlates; clinical course; comorbidity; obesity

Obesity is common in patients with bipolar disorder (BD), with a prevalence of 20% to 49% (1–3), compared to 18% in the general US population (4). There are a number of possible causes of obesity in BD, including lifestyle, medication exposure, binge-eating comorbidity, neuroendocrine and neurotransmitter dysfunctions, comorbid metabolic syndrome, and genetic predisposition. The growing evidence of obesity in patients with BD and its correlation with increased morbidity and mortality led us to study the association between the two disorders more systematically, with the hypothesis that body mass index (BMI) is related to prognosis and outcome. In this paper, we examine differences in sociodemographic, clinical, and medical characteristics of BD patients with elevated BMI.

Obesity appears to be correlated with important clinical features of BD, resulting in a poorer prognosis and outcome. Fagiolini et al. (3) found that obese bipolar patients experienced a greater number of lifetime depressive and manic episodes; presented with more severe and difficult-to-treat index affective episodes; and were more likely to develop an affective recurrence, particularly depression, following acute treatment. Two recent studies have also suggested a relationship between obesity and suicidal ideation and behaviour in BD patients (5, 6), specifically, a positive correlation between BMI and history of suicide attempts.

Not only is this of great concern in treating BD, but obesity is the most important predictor of metabolic syndrome (7). In a later study, Fagiolini et al. (6) found that 30% of their sample of BD patients met the National Cholesterol Education Program Expert Panel on Detection, Evaluation and Treatment of High Blood Cholesterol in Adults (NCEP ATP III) criteria for metabolic syndrome. It has been increasingly recognized that individuals with BD have a higher prevalence of modifiable risk factors for metabolic syndrome and cardiovascular disease (6, 7), as well as higher mortality rates due to natural causes (8, 9), compared to the general population.

Methods

Participants

All subjects were recruited through the Maritime Bipolar Registry, a community-based project carried out throughout the Maritime Provinces of Canada, including New Brunswick, Nova Scotia, and Prince Edward Island (10). At the time of the analysis, the registry included data from 276 patients with BD, comprising 104 men and 172 women in the age range of 16 to 83 years. Of these, 186 participants were diagnosed with bipolar I disorder (BD I), 85 with bipolar II disorder (BD II), and 5 with BD not otherwise specified (NOS). All consenting participants were interviewed in person and met the DSM-IV criteria for BD. Interviews were carried out by experienced psychiatrists or trained research nurses. For each pro-band, the following information was collected by interview and chart review: basic demographic characteristics, height and weight, details of the clinical presentation (including age of onset of mania and depression, psychiatric comorbidity, history of suicidal

behaviour, and psychosis during illness episodes), response to lithium, and specific details of medical comorbidity [including hypertension, thyroid disorder, and diabetes mellitus type II (DM II)]. Medical comorbidity was based on previous diagnosis and evidence of treatment for each selected medical condition. Ethnically, most participants were of Irish, Scottish, or French Acadian origin.

Statistical analysis

We used *t*-tests and analysis of variance (ANOVA) to analyze BMI in categories defined by independent variables; for continuous measures, we used Pearson correlation coefficients. Subsequently, we carried out an exploratory multivariate analysis using the structural equation method implemented in LISREL (11).

Results

Correlates of obesity in the sample

The average BMI in our sample was 29.9 ± 5.2 for men and 29.2 ± 7.4 for women. The rates of overweight and obesity in this group of patients with BD were high, with 36.6% (n = 101) being overweight and 39.1% (n = 108) obese. Only 22.8% (n = 63) of patients were of normal weight, and 1.4% (n = 4) underweight. For obese patients, mean height was 167.8 ± 10.3 cm and mean weight was 101.1 ± 14.9 kg. For patients of healthy weight, mean height was 168.2 ± 9.1 cm and mean weight was 62.9 ± 8.3 kg.

Table 1 shows the demographic characteristics of the patients in our sample and their relation to BMI. The average age was 44.0 ± 12.6 years, with no significant association with BMI ($r = 0.10$, p = 0.12). There was no significant correlation between age of mania ($r = -0.05$, p = 0.41) or depression onset ($r = -0.05$, p = 0.46) and BMI, with mean ages of 29.6 ± 11.6 years and 25.0 ± 11.6 years, respectively. BMI was comparable across the patients' sex and marital status, but it was significantly higher in those on disability compared to patients who worked full time. This is consistent with a negative correlation between BMI and Global Assessment of Functioning (GAF) scores ($r = -0.14$, p = 0.02), with a mean GAF score of 67.4 ± 16.7.

Table 2 shows the clinical characteristics of this group of bipolar patients. BMI was not significantly different across BD subtypes (BD I versus BD II). In terms of the clinical course of BD, subjects with a chronic illness showed significantly higher BMI than those with an episodic course. The duration of illness in this sample was 19.5 ± 12.1 years on average, and it correlated with BMI ($r = 0.14$, p = 0.02). Patients with a history of rapid cycling showed a trend toward higher BMI compared to those without. When comparing subjects with and without a history of psychotic episodes and suicidal behavior, there was no significant difference in BMI between these two subgroups. There was a trend toward higher BMI in patients with full-criteria generalized anxiety disorder (GAD) and social anxiety disorder (SAD), but when we tested for the entire spectrum of anxiety symptoms, including patients with subsyndromal GAD and SAD, BMI was clearly significantly higher than in those without anxiety symptoms. Finally, there was a positive trend toward higher BMI in subjects with comorbid personality disorders.

Table 3 shows the medical characteristics of the sample. Patients with comorbid DM II and hypertension had significantly higher BMI, as expected, given this well-documented association in the medical literature. The mean age of DM II onset was 48.1 ± 9.6 years, with a negative trend between BMI and age of onset ($r = -0.31$, p = 0.06). There was a negative correlation between duration of DM II and BMI ($r = -0.35$, p = 0.04), with mean duration 6.5 ± 6.7 years. Patients with comorbid thyroid disorder only exhibited a trend toward higher BMI.

Table 4 shows therapeutic effect of lithium in relation to BMI. Patients who achieved complete remission of psychiatric symptoms on lithium had a significantly lower BMI compared to those who showed partial or no therapeutic benefit (p = 0.01). These patients had a BMI in the healthy weight range. Patients who had a partial response to lithium had a BMI in the overweight range, while those showing no response to lithium had a BMI in the obese range.

Structural equation modeling

We hypothesized that patients with rapid cycling, anxiety disorders, and chronic course of illness, as well as with medical comorbidities (DM II, thyroid disorder, and elevated BMI) have poorer outcome as measured by GAF scores. In order to obtain a parsimonious model, the analysis was restricted to variables that showed a relationship with BMI at the significance level of $p \leq 0.10$. From this initial set we removed additional variables to reduce the impact of incomplete data (therapeutic effectiveness of lithium, antipsychotic use at interview), small number of observations (personality disorders), nominal data (socioeconomic status), and variable overlap (hypertension). GAD and SAD were combined into a single variable of anxiety. Presented in Figure 1 is a latent variable path model, with one exogenous variable, severity of illness, and an endogenous single indicator of patient outcome, GAF score. All variables in the model were ordinal, with the exception of BMI and GAF scores, which were continuous variables. Course of illness was coded ‘1’ for single episode, ‘2’ for completely episodic, ‘3’ for episodic residual, ‘4’for chronic fluctuating, and ‘5’ for chronic course of illness. The variables rapid cycling, diabetes mellitus, and thyroid disorder were coded ‘1’ for absence and ‘2’ for presence of the feature or disorder, whereas the variable anxiety disorder was coded ‘1’ for absence, ‘2’ for subsyndromal, and ‘3’ for meeting criteria of an anxiety disorder. The proposed model was assessed using structural equation modeling, by means of the maximum likelihood estimation procedure in LISREL 8.8 (11). The fit of the model was satisfactory ($\chi^2 = 10.77$, df = 12, p = 0.55). In addition, the root mean square error of approximation (RMSEA) was 0.0 (95% confidence interval: 0.0–0.056), with a p value of 0.92 (for test of close fit, RMSEA < 0.05), also indicative of an excellent fit. Since our sample contained missing data (1.92%), however, additional goodness-of-fit indices could not be calculated. The squared multiple correlations (R^2) for the latent variable severity of illness were 0.22 for rapid cycling, 0.69 for course of illness, 0.095 for anxiety disorder, 0.027 for diabetes mellitus, 0.026 for thyroid disorder, and 0.080 for BMI. The latent variable severity of illness accounted for 27% of the variance in the GAF scores. The path coefficient estimates in this model were moderate, ranging from 0.07 to 10.69, but were all statistically significant (a *t*-value of 1.96 or higher for the significance level of 0.05) with *t*-values ranging from 2.26 to 5.58.

Discussion

The prevalence of obesity (39.1%) in our sample was similar to the high rates reported in other studies (1–3). Further, we found that patients with BD with higher BMI differed in their basic clinical characteristics. Patients with higher BMI were not responsive to lithium and had a more chronic, fluctuating course. They had more comorbid subthreshold anxiety disorders and longer duration of illness. Interpreting the BMI of patients who met full criteria for GAD and SAD was difficult due to small sample sizes (n = 22 and n = 10, respectively). Patients with higher BMI showed a trend toward more rapid cycling and comorbid personality disorders.

Similar to Fagiolini et al. (3), we found that there was a negative correlation between BMI and outcome. Patients with BD with elevated BMI demonstrated lower GAF scores and higher rates of disability. Moreover, higher BMI was associated with greater medical comorbidity, with an increased risk of DM II and hypertension. Due to the lack of prospective research, however, it remains unclear whether these biological dysfunctions developed before the onset of obesity and BD or were the result of these conditions and/or the medications used to treat them.

Patients with elevated BMI were not lithium responsive and therefore required treatment with other medications, with the potential side effect of weight gain. The metabolic consequences of psychotropic medications (weight gain, insulin resistance/glucose intolerance, DM II, and dyslipidemia) may play a significant role in some of the positive correlations between elevated BMI and DM II that we have found. Initially, insulin resistance makes weight loss more difficult, leading to a cycle of increasing insulin resistance and further weight gain. As insulin resistance progresses to glucose intolerance and frank DM II, patients begin to lose weight as their ability to metabolize glucose is diminished. This is reflected in the negative correlation between duration of DM II and BMI that we observed. Further to the risks of obesity in BD are hypertension and the development of metabolic syndrome, increasing the risk of cardiovascular disease, the most frequent cause of death in BD patients (9).

The co-occurrence of obesity and BD, however, predates the advent of current medications (12). Other contributing factors such as genetic predisposition, biological dysfunction, phenomenology of depressive symptoms, psychiatric comorbidity, family environment, and socioeconomic status all likely interact to influence activity and diet, leading to the development of obesity (13). Several potential candidate genes for a common genetic diathesis between obesity and BD have been identified (14–16). Biological abnormalities such as dysregulation of the central serotonin and dopamine system (17–21) and increased norepinephrine turnover (17, 22) may also contribute to the correlation between BD and obesity. Hyperactivity of the hypothalamic-pituitary-adrenal axis, leading to hypercortisolemia, has been found to accompany depressive states. Cortisol has a strong effect on gluconeogenesis, and persistently elevated cortisol levels are associated with abdominal obesity (23), weight gain (24–26), insulin resistance (27), and likely the development of DM II.

Patients with elevated BMI have a greater number of lifetime episodes of illness and are more likely to develop rapid recurrence (3). Similarly, lithium nonresponders have poorer quality of remissions, with a more chronic, fluctuating course. These patients spend more time requiring acute treatment, which has been shown to increase risk of weight gain. Fagiolini et al. (4) demonstrated a relationship between acute depressive episodes and weight gain, with most weight gain occurring during the acute phase of treatment and little during the maintenance phase. Depressive episodes and subsyndromal and minor affective symptoms predominate in BD (28) and are associated with significant impairment (29). Patients gain more weight during this time, perhaps in part due to inactivity and difficulty following a healthy diet and perhaps due to greater exposure to medications that may contribute to weight gain. There may also be a metabolic relationship with the quality of remission.

Most mood stabilizers, including lithium, have been reported to cause weight gain (30–35). However, patients who demonstrated complete remission of symptoms with lithium showed significantly lower BMI compared to those reporting no therapeutic effect. We speculate that lithium-responsive patients spend more time in a state of wellness and are better able to exercise and follow a healthy diet. They spend less time in a depressed state, during which atypical symptoms may influence their activity level and appetite, and they require less acute treatment, which is related to the greatest weight gain. These patients may also be different metabolically, which may account for their lithium responsiveness and quality of remissions. Conversely, patients who respond to lithium may be affected at a metabolic level, resulting in lower BMI. The insulin-like effect of lithium may mimic the hyperinsulinemic state that pre-empts insulin resistance in an attempt to stave off its development, resulting in less insulin resistance and DM II in lithium-responsive patients.

In summary, BD patients with elevated BMI appear to suffer from a more severe course of BD in terms of chronicity and overall functioning, and are at increased risk of medical comorbidity. Our study has further identified this subgroup as lithium nonresponders, who may represent a particular phenotype of BD patients with a distinct pathophysiology and less favourable outcome. This subgroup with poor prognosis may have impaired metabolism that could underlie obesity, DM II, and response to lithium. Alternatively, elevated BMI and its associated metabolic consequences, such as insulin resistance, may be independent complicating factors affecting the course or lithium responsiveness of patients with BD.

This study cannot determine whether metabolic dysfunction leads to lithium nonresponsiveness, with its associated clinical features, or whether lithium responsiveness determines subsequent metabolic sequelae. The two may be genetically related. Until a prospective study can be done to assess causality, perhaps the most practical outcome is to demonstrate the compelling need to provide nutrition and exercise counseling, and to screen for and treat metabolic syndrome and risk factors for cardiovascular disease. Ideally, this would be part of every BD patient’s overall care. This is particularly important for patients who are already at risk for cardiovascular morbidity, have pre-existing cardiovascular disease, DM II, or hypertension, or fit into the lithium nonresponsive subgroup (i.e., elevated BMI, more chronic course, comorbid subthreshold anxiety disorders, trend toward rapid cycling and comorbid personality disorders, and increased DM II and hypertension). In turn,

these behavioral and medical interventions could possibly improve the clinical course of bipolar illness, reduce morbidity and mortality related to physical illness, and most importantly, enhance the quality of life of BD patients.

Acknowledgments

This study was funded by a grant from the Neuroscience Research Foundation (Eli Lilly Canada) and grant 64410 from the Canadian Institutes of Health Research to MA.

References

1. Elmslie JL, Silverstone JT, Mann JI, Williams SM, Romans SE. Prevalence of overweight and obesity in bipolar patients. J Clin Psychiatry. 2000; 61:179–184. [PubMed: 10817102]
2. McElroy SL, Frye MA, Suppes T, et al. Correlates of overweight and obesity in 644 patients with bipolar disorder. J Clin Psychiatry. 2002; 63:207–213. [PubMed: 11926719]
3. Fagiolini A, Kupfer DJ, Houck PR, Novick DM, Frank E. Obesity as a correlate of outcome in patients with bipolar I disorder. Am J Psychiatry. 2003; 160:112–117. [PubMed: 12505809]
4. Fagiolini A, Frank E, Houck PR, et al. Prevalence of obesity and weight change during treatment in patients with bipolar I disorder. J Clin Psychiatry. 2002; 63:528–533. [PubMed: 12088166]
5. Fagiolini A, Kupfer DJ, Rucci P, Scott JA, Novick DM, Frank E. Suicide attempts and ideation in patients with bipolar I disorder. J Clin Psychiatry. 2004; 65:509–514. [PubMed: 15119913]
6. Fagiolini A, Frank E, Scott JA, Turkin S, Kupfer DJ. Metabolic syndrome in bipolar disorder: findings from the Bipolar Disorder Center for Pennsylvanians. Bipolar Disord. 2005; 7:424–430. [PubMed: 16176435]
7. Newcomer JW. Medical risk in patients with bipolar disorder and schizophrenia. J Clin Psychiatry. 2006; 67(Suppl 9):25–30. [PubMed: 16965186]
8. Colton CW, Manderscheid RW. Congruencies in increased mortality rates, years of potential life lost, and causes of death among public mental health clients in eight states. Prev Chronic Dis. 2006; 3:A42. [PubMed: 16539783]
9. Osby U, Brandt L, Correia N, Ekbom A, Sparen P. Excess mortality in bipolar and unipolar disorder in Sweden. Arch Gen Psychiatry. 2001; 58:844–850. [PubMed: 11545667]
10. Růžičková M, Slaney C, Garnham J, Alda M. Clinical features of bipolar disorder with and without comorbid diabetes mellitus. Can J Psychiatry. 2003; 48:458–461. [PubMed: 12971015]
11. Joreskog, K., Sorbom, D. LISREL 8: User's Reference Guide. 2. Lincolnwood, IL: Scientific Software International, Inc; 2006.
12. Kretschmer, E. Physique and Character. London: Kegan, Paul, Trench, Trubner; 1936.
13. Wildes JE, Marcus MD, Fagiolini A. Obesity in patients with bipolar disorder: a biopsychosocial-behavioral model. J Clin Psychiatry. 2006; 67:904–915. [PubMed: 16848650]
14. Philibert R, Caspers K, Langbehn D, et al. The association of a HOPA polymorphism with major depression and phobia. Compr Psychiatry. 2002; 43:404–410. [PubMed: 12216017]
15. Comings DE, Gade R, MacMurray JP, Muhleman D, Peters WR. Genetic variants of the human obesity (OB) gene: association with body mass index in young women, psychiatric symptoms, and interaction with the dopamine D2 receptor (DRD2) gene. Mol Psychiatry. 1996; 1:325–335. [PubMed: 9118359]
16. Chagnon YC, Merette C, Bouchard RH, Emond C, Roy MA, Maziade M. A genome wide linkage study of obesity as secondary effect of antipsychotics in multigenerational families of eastern Quebec affected by psychoses. Mol Psychiatry. 2004; 9:1067–1074. [PubMed: 15224101]
17. Ericsson M, Poston WS, Foreyt JP. Common biological pathways in eating disorders and obesity. Addict Behav. 1996; 21:733–743. [PubMed: 8904939]
18. Mahmood T, Silverstone T. Serotonin and bipolar disorder. J Affect Disord. 2001; 66:1–11. [PubMed: 11532527]

19. Sobczak S, Honig A, van Duinen MA, Riedel WJ. Serotonergic dysregulation in bipolar disorders: a literature review of serotonergic challenge studies. Bipolar Disord. 2002; 4:347–356. [PubMed: 12519094]
20. Vogel M, Pfeifer S, Schaub RT, et al. Decreased levels of dopamine D3 receptor mRNA in schizophrenic and bipolar patients. Neuropsychobiology. 2004; 50:305–310. [PubMed: 15539862]
21. Wang GJ, Volkow ND, Logan J, et al. Brain dopamine and obesity. Lancet. 2001; 357:354–357. [PubMed: 11210998]
22. Young LT, Warsh JJ, Kish SJ, Shannak K, Hornykiewcz O. Reduced brain 5-HT and elevated NE turnover in bipolar affective disorder. Biol Psychiatry. 1994; 35:121–127. [PubMed: 7513191]
23. Thakore JH, Richards PJ, Reznek RH, Martin A, Dinan TG. Increased intra-abdominal fat deposition in patients with major depressive illness as measured by computed tomography. Biol Psychiatry. 1997; 41:1140–1142. [PubMed: 9146826]
24. Bjorntorp P, Rosmond R. Obesity and cortisol. Nutrition. 2000; 16:924–936. [PubMed: 11054598]
25. Vicennati V, Pasquali R. Abnormalities of the hypothalamic-pituitary-adrenal axis in nondepressed women with abdominal obesity and relations with insulin resistance: evidence for a central and a peripheral alteration. J Clin Endocrinol Metab. 2000; 85:4093–4098. [PubMed: 11095438]
26. Watson S, Gallagher P, Ritchie JC, Ferrier IN, Young AH. Hypothalamic-pituitary-adrenal axis function in patients with bipolar disorder. Br J Psychiatry. 2004; 184:496–502. [PubMed: 15172943]
27. Weber-Hamann B, Hentschel F, Kniest A, et al. Hypercortisolemic depression is associated with increased intra-abdominal fat. Psychosom Med. 2002; 64:274–277. [PubMed: 11914443]
28. Judd LL, Akiskal HS, Schettler PJ, et al. The long-term natural history of the weekly symptomatic status of bipolar I disorder. Arch Gen Psychiatry. 2002; 59:530–537. [PubMed: 12044195]
29. Judd LL, Akiskal HS, Schettler PJ, et al. Psychosocial disability in the course of bipolar I and II disorders: a prospective, comparative, longitudinal study. Arch Gen Psychiatry. 2005; 62:1322–1330. [PubMed: 16330720]
30. Ruetsch O, Viala A, Bardou H, Martin P, Vacheron MN. Psychotropic drugs induced weight gain: a review of the literature concerning epidemiological data, mechanisms and management. Encephale. 2005; 31:507–516. [PubMed: 16389718]
31. Keck PE, McElroy SL. Bipolar disorder, obesity, and pharmacotherapy-associated weight gain. J Clin Psychiatry. 2003; 64:1426–1435. [PubMed: 14728103]
32. Hennen J, Perlis RH, Sachs G, Tohen M, Baldessarini RJ. Weight gain during treatment of bipolar I patients with olanzapine. J Clin Psychiatry. 2004; 65:1679–1687. [PubMed: 15641874]
33. Bowden CL, Calabrese JR, Ketter TA, Sachs GS, White RL, Thompson TR. Impact of lamotrigine and lithium on weight in obese and nonobese patients with bipolar I disorder. Am J Psychiatry. 2006; 163:1199–1201. [PubMed: 16816224]
34. Vestergaard P, Poulstrup I, Schou M. Prospective studies on a lithium cohort. 3. Tremor, weight gain, diarrhea, psychological complaints. Acta Psychiatr Scand. 1988; 78:434–441. [PubMed: 3227963]
35. Lipkovich I, Citrome L, Perlis R, et al. Early predictors of substantial weight gain in bipolar patients treated with olanzapine. J Clin Psychopharmacol. 2006; 26:316–320. [PubMed: 16702898]

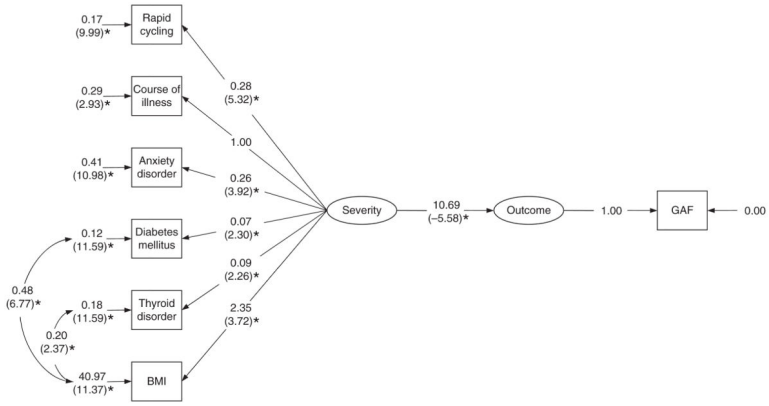

Fig. 1.

LISREL model. The patient outcome model of severity of illness [$\chi^2 = 10.77$, df = 12, p = 0.5487, root mean square error of approximation (RMSEA) = 0.00, n = 276]. Values above are estimated path coefficients, and below in parentheses are the *t*-values, where * indicates statistical significance at the 0.05 level. BMI = body mass index; GAF = Global Assessment of Functioning.

Table 1

Demographic characteristics of patients with bipolar disorder and their relation to body mass index (BMI)

		BMI			
Characteristic (number for whom data available)	**%**	**Mean**	**SD**	**Statistic**	**p**
Sex (276)				$t(274) = 0.10$	0.32
Men	37.7	29.9	5.20		
Women	62.3	29.2	7.44		
Marital status (276)				$F(3,272) = 0.82$	0.48
Single	23.6	29.7	7.38		
Married	53.6	28.9	5.67		
Divorced	19.2	30.4	8.37		
Widowed	3.6	30.8	6.01		
Socioeconomic status (271)				$F(7,263) = 3.43$	0.002
Working full time	23.2	27.9[a]	5.64		
Working part time	13.7	27.8	4.25		
Unemployment insurance	4.4	31.0	4.98		
Social assistance	3.7	32.3	10.40		
Disability	35.1	31.5[b]	7.98		
Other	8.1	27.8	5.76		
Retired	7.4	28.1	2.37		
Student	4.4	25.7	5.87		

Means in the same row that do not share superscripts differ at $p < 0.05$ in the Tukey HSD comparison.

Bipolar Disord. Author manuscript; available in PMC 2013 January 14.

Table 2

Clinical characteristics of patients with bipolar disorder and their relation to body mass index (BMI)

	BMI				
Variable (number for whom data available)	**%**	**Mean**	**SD**	**Statistic**	**p**
Diagnosis (276)				$F(2,273) = 0.78$	0.46
Bipolar I	67.4	29.7	6.93		
Bipolar II	30.8	28.9	6.13		
Bipolar NOS	1.8	26.9	6.55		
Course of Illness (273)				$F(4,272) = 5.79$	< 0.001
Single episode	2.6	30.3	5.02		
Completely episodic	20.1	26.9[a]	3.95		
Episodic with residual symptoms	31.5	28.2[a]	5.94		
Chronic fluctuating	37.0	31.4[b]	7.96		
Chronic	8.8	31.2	6.12		
Rapid cycling (264)				$t(262) = 1.73$	0.09
Present	34.1	30.4	8.02		
Absent	65.9	28.8	5.91		
Psychosis (253)				$t(251) = 0.27$	0.78
Present	57.7	29.3	6.97		
Absent	42.3	29.0	5.75		
History of suicide attempts (271)				$t(269) = 1.65$	0.10
Present	29.5	30.5	7.88		
Absent	70.5	29.0	6.13		
Generalized anxiety disorder (263)				$F(2,260) = 3.07$	0.05
Subsyndromal	34.6	30.7[a]	8.12		
Present	8.4	30.1	5.37		
Absent	57.0	28.6[b]	7.10		
Social anxiety disorder (266)				$F(2,263) = 3.97$	0.02
Subsyndromal	19.5	31.4	8.30		
Present	2.6	25.4	4.67		
Absent	77.9	29.1	6.13		

	BMI				
Variable (number for whom data available)	**%**	**Mean**	**SD**	**Statistic**	**p**
Panic disorder (266)				$F(2,263) = 0.08$	0.92
Subsyndromal	21.5	29.3	6.95		
Present	3.7	29.2	5.34		
Absent	74.8	29.6	6.65		
Substance abuse (273)				$F(2,270) = 1.50$	0.23
Subsyndromal	11.0	31.4	9.25		
Present	24.2	29.6	6.49		
Absent	64.8	29.2	6.24		
Personality disorder (252)				$F(2,249) = 2.93$	0.06
Subsyndromal	2.7	29.6	6.92		
Present	2.0	36.2^{a}	16.60		
Absent	95.3	29.1^{b}	6.16		

Means in the same row that do not share superscripts differ at $p < 0.05$ in the Tukey HSD comparison. NOS = not otherwise specified.

Bipolar Disord. Author manuscript; available in PMC 2013 January 14.

Table 3

Medical characteristics of patients with bipolar disorder and their relation to body mass index (BMI)

		BMI			
Characteristic (number for whom data available)	**%**	**Mean**	**SD**	**Statistic**	**p**
Diabetes mellitus type II (274)				$t(272) = 4.27$	< 0.001
Present	14.6	33.5	5.98		
Absent	85.4	28.7	6.59		
Hypertension (273)				$t(271) = 3.31$	0.001
Present	15.8	32.5	6.59		
Absent	84.2	28.9	6.59		
Thyroid disease (274)				$t(272) = 1.75$	0.08
Present	24.8	30.9	7.91		
Absent	75.2	29.0	6.17		

Bipolar Disord. Author manuscript; available in PMC 2013 January 14.

Table 4

Lithium therapeutic effectiveness in relation to body mass index (BMI)

		BMI			
Characteristic (number for whom data available)	**%**	**Mean**	**SD**	**Statistic**	**p**
Lithium therapeutic effectiveness (159)				F(2,156) = 4.91	0.01
No effect	13.9	32.4[a]	7.45		
Partial	71.0	30.0[a]	6.73		
Complete	15.1	26.5[b]	3.57		

Means in the same row that do not share superscripts differ at $p < 0.05$ in the Tukey HSD comparison.