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Research report

Differentiating between Bipolar Disorder Types I and II: Results from the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC)

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ABSTRACT

Objective: Bipolar Disorder I (BD I) and Bipolar Disorder II (BD II) vary considerably, with differences in symptomatology, management and prognosis. For patients with depression, the distinction between BD I and BD II is not always apparent, and hinges on the differentiation between manic/mixed and hypomanic episodes. Other putative differences between patients with BD I and II exist and may assist in distinguishing between these two conditions.
Methods: Data were obtained from the National Epidemiological Survey on Alcohol and Related Conditions. A total of 1429 subjects were included in our analysis based on DSM-IV criteria, 935 with BD I and 494 with BD II. We examined for differences in a number of variables including demographics, clinical features, depressive symptoms, and co-morbid conditions using t-tests and chi-square analyses for a comparison of means as well as a logistic regression for variables found to be significant.
Results: Key differences between BD I and BD II were identified in all categories in our comparison of means. In the regression analysis, a number of variables were determined to be predictors of BD I, including unemployment (OR = 0.6), taking medications for depression (OR = 1.7), a history of a suicide attempt (OR = 1.8), depressive symptoms such as weight gain (OR = 1.7), fidgeting (OR = 1.5), feelings of worthlessness (OR = 1.6) and difficulties with responsibilities (OR = 2.2), as well as the presence of specific phobias (OR = 1.8) and Cluster C traits (OR = 1.4).
Conclusions: Our results indicate that in addition to the differences between manic/mixed and hypomanic episodes, other significant differences exist that may be used to help differentiate BD I from BD II.

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1. Introduction

When a patient presents with a current or past depression, a diagnosis of Bipolar Disorder (BD) is established based on the lifetime occurrence of hypomanic, manic, or mixed episodes (American Psychiatric Association. Task Force on DSM-IV and American Psychiatric Association, 2000; Hantouche et al., 2010; Sani et al., 2011; Tondo et al., 2007; Valtonen et al., 2005). BD is divided into its principal subtypes, I and II, based on the differentiation of manic/mixed episodes from hypomanic episodes. The lifetime prevalence rates of BD range from 0.1 to 3.3% for BD I and 0.5–2.0% for BD II (Angst, 1998; Angst et al., 2002; Grant et al., 2005b; Kessler et al., 1997; Szadoczky et al., 1998; ten Have et al., 2002; Weinstock et al., 2010). These illnesses are highly prevalent in clinical samples of depressed patients, with BD I being diagnosed in 18–47.1% and BD II in 7.7–45% (Akiskal et al., 2000; Benazzi, 1997, 1999; Serretti et al., 2002).

BD types I and II can be difficult to distinguish from one another given the retrospective and often subjective nature of identifying manic or hypomanic episodes. High rates of misdiagnosis and under-diagnosis have been reported which affect both management and prognosis (Akiskal

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et al., 1995, 2000; Awad et al., 2007; Matza et al., 2005; Perlis, 2005; Yatham et al., 2009; Zimmerman et al., 2010). It is generally recommended that a diagnosis of BD subtype be based on multiple evaluations (Akiskal et al., 2000), however, this may not be practical in general practitioner's office or for patients seen by psychiatrists one time in consultation. One potential strategy to address this issue involves the identification of distinguishers of BD subtypes other than a history of manic or hypomanic episodes (Mitchell et al., 2008). These differences might then be incorporated into a diagnostic assessment of BD.

Previous studies have demonstrated several features specifically associated with BD I compared to BD II. These include a greater number of hospitalizations for depression with longer time spent in hospital (Judd et al., 2003a; Serretti et al., 2002; Vieta et al., 1997), greater prevalence of psychotic symptoms during a depressive episode (Benazzi, 1999; Judd et al., 2003a; Serretti et al., 2002; Vieta et al., 1997), greater number of annual shifts in symptom polarity (Judd et al., 2003b), and greater number of atypical symptoms reported (Weinstock et al., 2010) in patients with BD I. BD I patients are also prescribed more somatic treatment during episodes as well as between episodes (Judd et al., 2003a), however, they show poorer inter-episode recovery (Benazzi, 1999). As well, they show higher rates of both alcohol and drug abuse or dependence (Regier et al., 1990) and higher rates of generalized anxiety disorder (GAD) (Grant et al., 2005b).

This is in contrast to BD II which displays a greater number of affective episodes (Judd et al., 2003a; Serretti et al., 2002; Vieta et al., 1997), longer duration of depressive episodes (Judd et al., 2003b), greater co-morbid anxiety disorders (Judd et al., 2003a), poorer return to baseline psychosocial function (Judd et al., 2003a) with poorer health related quality of life (Maina et al., 2007) compared to BD I. This is despite no differences between BD I and BD II in age of onset of first major depressive episode (Benazzi, 1999; Judd et al., 2003b; Serretti et al., 2002). There appears to be decreased prescription of mood-stabilizing medications in BD II compared to BD I during both symptomatic and asymptomatic periods (Judd et al., 2003a), despite the fact that patients with BD II are more frequently symptomatic than those with BD I (Judd et al., 2003b; Kupka et al., 2007). There have also been differential associations of suicide ideation and attempts within BD subtypes, with some suggesting higher rates in BD I and others with higher rates in BD II (Ghaemi et al., 2008; Hantouche et al., 2010; Rihmer and Pestality, 1999; Sani et al., 2011; Serretti et al., 2002; Tondo et al., 2007; Valtonen et al., 2005; Weinstock et al., 2010).

Furthermore, differences in neurobiological markers have emerged that potentially allow for some characterization of disorder subtype based on physiological findings (Chou et al., 2010; Liu et al., 2010). These data suggest that BD I and BD II may represent distinct clinical entities.

The above examples highlight some of the differences between BD subtypes, however, previous studies have generally utilized specialized clinical samples which limit the generalizability of the findings. Often, patients are recruited following presentation at a hospital clinic or inpatient unit (Brugue et al., 2008; Judd et al., 2003a; Serretti et al., 2002) thus the results may not be representative of the broader population of people with BD in the community. This is often cited as Berkson's Bias, which is a type of selection bias that results from a greater probability of admission to hospital for people with a greater number of conditions than for people with only one condition (Schwartzbaum et al., 2003). As well, diagnoses that are based on repeated clinical evaluations using specialized diagnostic tools may not be representative of common clinical practices.

To address these limitations, an epidemiological approach involving a large, more representative population would be important to add to the existing literature identifying distinguishing characteristics between BD I and BD II. Furthermore, such an approach should be based on clearly defined criteria from the DSM-IV, which has not always been the case with prior epidemiological studies, in order to permit the results to be more applicable and more widely utilized. In the present study, we examined for differences between BD subtypes in a large representative sample of the US population. The goal of this study was to report differences in demographic variables, clinical characteristics, individual depressive symptoms, and co-morbid conditions between subjects with BD I and BD II.

2. Methods

2.1. Data source

Data were obtained from the first wave of the National Epidemiological Survey on Alcohol and Related Conditions (NESARC), sponsored by the U.S. Department of Health and Human Services, National Institutes of Health, and National Institute on Alcohol Abuse and Alcoholism (NIAAA). This survey took place from August 2001 to May 2002 and represented a sample of non-institutionalized US population 18 years of age and older. Data were collected by 1800 lay interviewers from the US Census Bureau with an average 5 years of experience (Goldstein and Levitt, 2006). Interviews were conducted face-to-face, and were computer-assisted (Goldstein and Levitt, 2006). The response rate was 81% (Goldstein and Levitt, 2006). The interview obtained information on demographics, clinical variables, depressive symptoms, and certain other DSM-IV disorders. Diagnoses were generated using the NIAAA Alcohol Use Disorder and Associated Disabilities Interview Schedule-DSM-IV Version (AUDADIS-IV). Reliability and validity for the AUDADIS-IV diagnoses have been published elsewhere, but in short, BD I had a reliability of $\kappa = 0.59$ with other mood and anxiety disorders ranging from $\kappa = 0.40$–0.65, alcohol use disorders had a reliability of $\kappa = 0.74$, drug use disorders of $\kappa = 0.79$, and personality disorders from $\kappa = 0.40$–0.67 (Grant et al., 2003a, 2003b, 2004a,2004b, 2005a,2005b).

The total number of respondents was 43,093 (response rate of 81%). Methodological details of this survey have been published elsewhere (Grant BF, 2003b; Grant et al., 2004a). Variables of interest were identified based on prior literature (Benazzi, 2003; Benazzi, 2006; Akiskal et al., 1995; Calabrese et al., 2006; Hirschfeld et al., 2003; Mitchell et al., 2008; Mitchell et al., 2001; Parker et al., 2000; Perlis et al., 2006; Serretti et al., 2002; Schaffer et al., 2010) and/or clinical relevance.

2.2. Subjects

A total of 1429 subjects with a history of mania or hypomania and at least one lifetime major depressive episode (MDE) were included in the analysis. Subjects without a history of a MDE were excluded due to our specific focus on differences in depressive symptomatology. Subjects with a history of mood episodes due to substance use or general medical condition were excluded. There were 935 subjects with BD I and 494 subjects with BD II included in the analysis.

2.3. Demographic variables

Demographic variables included age, sex, immigrant status, marital status (married or common-law), completion of high school, employment status (currently employed full time, part time or employed but away for illness/vacation/absent without pay), disability (unemployed and permanently disabled), and Medicare or Medicaid usage.

2.4. Clinical variables

Clinical variables included age of onset of first MDE, number of lifetime MDEs, length of longest MDE, having been on medication for depression, having been hospitalized overnight for depression, having a family history of depression, and total number of first degree relatives with depression. Family history of BD was not available.

2.5. Depressive symptomatology

Symptoms experienced during the worst episode of depression were collected, including anhedonia, suicidal ideation, suicide attempt, weight loss or gain, initial insomnia, terminal insomnia, hypersomnia, fatigue, psychomotor retardation psychomotor agitation, restlessness, worthlessness, guilt, trouble concentrating, indecisiveness, argumentativeness, difficulties with responsibilities, spending more time alone, decreased motivation and decreased productivity.

2.6. Co-morbid conditions

Co-morbid conditions included panic disorder (PD) with and without agoraphobia, agoraphobia without PD, social phobia, specific phobia, generalized anxiety disorder (GAD), the presence of any anxiety disorder, pathological gambling, some Cluster A personality disorders (paranoid and schizoid), some Cluster B personality disorders (antisocial and histrionic), Cluster C personality disorders (avoidant, dependent, obsessive–compulsive), the presence of any of the above personality disorders, history of alcohol abuse or dependence, and history of drug abuse or dependence (including amphetamines, opioids, sedatives, tranquilizers, cocaine, inhalant/solvent, hallucinogen, cannabis, heroin and other drugs).

2.7. Data analysis

Data for this study was obtained from the NESARC publicly available database (2001). This study was approved by the research ethics board at Sunnybrook Health Sciences Centre, University of Toronto.

Analyses were conducted using SPSS Statistics version 18 (IBM, 2010). Demographics, clinical variables, depressive symptomatology, and co-morbid conditions were compared between BD I and II using t-tests for continuous variables and chi-square tests for categorical variables. Variables significant at the $p<0.05$ level were entered into a single stepped forward-selection logistic regression without adjustment. A receiver operating characteristic (ROC) curve was plotted and area under the curve calculated to determine model fit.

3. Results

Prevalence of BD I in our population was 2.2%, and that for BD II was 1.1%.

There were a number of significant differences identified between subjects with BD I ($n=935$) and subjects with BD II ($n=494$) using a comparison of means or frequencies (shown in Table 1). Employment rates were higher in the BD II group whereas the BD I subjects showed greater rates of disability and reliance on Medicare/Medicaid. No differences were seen in age, sex, or immigration status. Differences in marital status approached significance at $p=0.053$.

Subjects with BD I demonstrated a more severe clinical course as evidenced by greater number of episodes ($p<0.001$), longer episodes ($p<0.001$), greater suicidal ideation ($p<0.001$) with a greater proportion of suicide attempts ($p<0.001$), greater hospitalizations for depression ($p<0.001$), increased likelihood of co-morbid conditions including anxiety disorders ($p<0.001$) and personality disorders ($p<0.001$), and poorer psychosocial functioning such as lower employment rates ($p<0.001$). BD I was also associated with greater use of medications for depression ($p<0.001$), and greater incidence of family history of a major depressive episode ($p<0.001$). Interestingly, higher impulsivity traits reflected by pathological gambling and alcohol abuse/dependence were not significantly different between subjects with BD I and BD II ($p=0.153$ and $p=0.307$, respectively).

Nearly all depressive symptoms were more commonly reported in the BD I group as demonstrated by comparison of frequencies. Subjects with BD I were more likely to endorse atypical symptoms such as weight gain ($p<0.001$) and hypersomnia ($p=0.034$). BD I subjects were also more likely to report melancholic features of anhedonia ($p=0.002$), psychomotor agitation (fidgeting/pacing, $p<0.001$) or retardation (moved/talked more slowly, $p=0.022$), terminal insomnia ($p=0.003$), and feelings of guilt ($p<0.001$). Weight loss was the only reported symptom not significantly different between subjects with BD I and BD II ($p=0.190$).

A stepped forward-selection logistic regression including all significant variables found several independent correlates with a BD I diagnosis (see Table 2). All four categories – sociodemographic, clinical variables, depressive symptomatology, and co-morbid conditions – were represented in the final regression model. Subjects with BD I were less likely to be employed, more likely to be treated with medications for depression, and were more likely to have made a suicide attempt. Of note, indicators of severity such as length of episodes and hospitalization rates did not emerge as associated variables with BD I despite there being a significant difference

Table 1
Comparison of sociodemographic and clinical variables between BD I and BD II in a large epidemiological sample.

		BD I N = 935	BD II N = 494	P-values
Demographic variables	Age (mean)	38.7	35.5	0.052
	Sex (male)	35.2%	38.2%	0.251
	Born in USA	89.3%	88.6%	0.717
	Married	41.5%	36.2%	0.053
	Completed high school	80.1%	83.0%	0.185
	Employed	55.9%	72.9%	<0.001*
	Disabled	12.7%	4.9%	<0.001*
	Medicare or Medicaid	28.8%	18.4%	<0.001*
Clinical variables	Age of onset of first depressive episode in years (mean, (median))	24.3 (22)	24.5 (22)	0.227
	Number of depressive episodes (mean, (median))	10.1 (3)	5.7 (2)	<0.001*
	Any past medication use for depression (% yes)	58.4%	35.6%	<0.001*
	Any past hospitalization for depression (% yes)	27.5%	9.7%	<0.001*
	Longest depressive episode (weeks; mean (median))	145.2 (26)	87.3 (17)	<0.001*
	Family history of depression (% yes)	78.8%	67.8%	<0.001*
	Number of family members with depression (mean, (median))	2.4 (2)	1.8 (1)	0.083
Depressive symptoms (during worst MDE)	Anhedonia	93.8%	89.3%	0.002*
	Suicidal ideation	59.5%	44.1%	<0.001*
	Suicide attempt	28.9%	14.6%	<0.001*
	Weight loss	54.6%	49.4%	0.062
	Weight gain	34.6%	27.1%	0.004*
	Initial insomnia	76.9%	71.1%	0.016*
	Terminal insomnia	62.5%	54.4%	0.003*
	Hypersomnia	56.9%	51.0%	0.034*
	Easily fatigued	87.6%	83.5%	0.034*
	Moved/talked more slowly	53.7%	47.2%	0.022*
	Fidgeted/paced	59.6%	44.6%	<0.001*
	Restless	68.9%	53.0%	<0.001*
	Worthless	80.8%	65.2%	<0.001*
	Guilt	75.3%	65.8%	<0.001*
	Trouble concentrating	93.1%	88.8%	0.005*
	Indecisive	86.0%	80.9%	0.011*
	Argumentative	70.0%	62.3%	0.003*
	Trouble with responsibilities	84.0%	65.4%	<0.001*
	Spent more time alone	87.1%	80.8%	0.001*
	Decreased motivation	82.4%	72.3%	<0.001*
	Decreased productivity	89.5%	84.6%	0.007*
Co-morbid conditions	Panic disorder with and without agoraphobia	32.6%	18.6%	<0.001*
	Agoraphobia without panic	1.1%	0.20%	0.074
	Social phobia	26.1%	16.8%	<0.001*
	Specific phobia	34.1%	21.1%	<0.001*
	Generalized anxiety disorder	31.9%	18.4%	<0.001*
	Any anxiety disorder	64.9%	45.3%	<0.001*
	Gambling	3.1%	1.8%	0.153
	Cluster A personality traits	43.3%	30.0%	<0.001*
	Cluster B personality traits	30.5%	23.5%	0.005*
	Cluster C personality traits	48.0%	32.4%	<0.001*
	Any personality disorder	68.2%	52.2%	<0.001*
	Alcohol Abuse or Dependence	54.6%	51.8%	0.307
	Any drug abuse or dependence	36.6%	26.7%	<0.001*

* indicates significance at $p<0.05$ level.

in the reported frequencies between the two subject groups. Depressive symptoms associated with a diagnosis of BD I included weight gain, psychomotor agitation (fidgeting), feelings of worthlessness, and difficulties with responsibilities. Finally, co-morbidities associated with BD I included specific phobias and Cluster C personality disorders, however, the variables 'any anxiety disorder' and 'any personality disorder', more generalized descriptors, were not predictive of either group. This suggests that specific phobias and Cluster C personality disorders were highly specific in their association with BD I.

Diagnostic predictions were estimated by fitting a Receiver Operating Curve (ROC), as shown in Fig. 1. The associated area under the curve was 0.726 indicating a fair fit of the model to the data.

4. Discussion

The primary aim of this study was to characterize and distinguish BD I from BD II in a population-based sample. Our study highlights several important differences between BD

Table 2
Forward selection logistic regression between BD I and BD II. OR > 1 are more likely with BD I and OR < 1 are more likely with BD II.

BD I vs. BD II		P-value	OR	95% C.I. for OR	
				Lower	Upper
Demographics	Employed*	0.003*	0.625	0.458	0.852
Clinical variables	Any past medication use for depression*	0.001*	1.668	1.236	2.251
Depressive symptoms	Suicide attempt*	0.006*	1.757	1.179	2.619
(during worst MDE)	Weight gain*	0.001*	1.669	1.226	2.272
	Fidgeted/paced*	0.006*	1.499	1.122	2.001
	Worthless*	0.007*	1.559	1.126	2.158
	Trouble with responsibilities*	<0.001*	2.243	1.598	3.147
Co-morbid conditions	Specific phobia*	0.001*	1.757	1.256	2.457
	Cluster C personality traits*	0.018*	1.440	1.064	1.949

* indicates significance at $p<0.05$ level.

subtypes in demographics, clinical variables, depressive symptoms, and co-morbid conditions. The majority of variables emerged as statistically different between the two groups in a comparison of frequencies. The regression analysis demonstrated important predictors of BD I with a fair fit of the model to the data.

4.1. Demographic variables

Employment status differed between the two populations and emerged as a significant predictor of BD II in our regression, suggesting better occupational functioning in BD II and correlating with the lower levels of disability and reliance on governmental assistance that were shown in our sample.

Most other sociodemographic variables did not consistently differ between BD subtypes similar to results previously shown in clinical samples (Judd et al., 2003a,2003b; Vieta et al., 1997).

In terms of prevalence rates, the prevalence of BD I was shown to be double that of BD II. This reflects what was previously shown in this sample by Grant et al. (Grant et al., 2004a, 2005b), but is in contrast to what was shown in a similar epidemiological sample (National Co-Morbidity Survey—Replication). Reports from that study demonstrate similar prevalence rates for BD II, but lower rates for BD I (Angst et al., 2010; Merikangas et al., 2007). Differences between these two populations have not been closely studied but may reflect differences in study design, administration of questionnaires, and diagnostic criteria used.

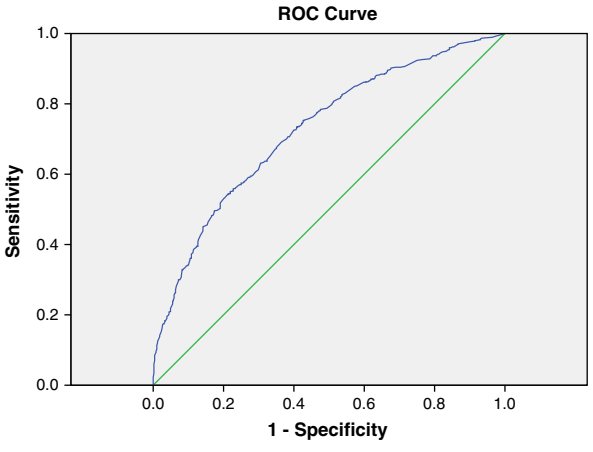

Fig. 1. ROC for BD I and BD II.

4.2. Clinical variables

Course of illness of BD I and BD II differed in several important ways in our subject population. For example, individuals with BD I suffered more depressive episodes (mean of 10 compared to 6 by BD II). This contrasts with previously published reports showing that patients with BD II suffer more mood episodes and spend a longer time in minor depression/dysthymic states than those with BD I (Judd et al., 2003a,2003b; Serretti et al., 2002; Vieta et al., 1997). Differences between our study and those reports may reflect the differences in the populations studied. In a large community sample, individuals with BD II may go unrecognized which leads to fewer diagnosed depressive episodes. For example, clinicians may have a low index of suspicion for mood episodes given that the disorder itself has not been properly diagnosed. This is supported by our findings of decreased prescription of medications for depression compared to those with BD I. The finding of lower rates of medication use among subjects with BD II reflects the results of Judd et al. (2003a, 2003b) showing that patients with BD II were less likely to receive treatment for a mood episode as well as in between episodes (Judd et al., 2003a). However, it is important to take into account that in our sample, the episodes of depression are based on self report as opposed to those clinically diagnosed in the Judd et al. studies (Judd et al., 2003a,2003b). Both the Judd studies and this current study identified BD patients as having a history of mania as well as a history of depression. This is important to note as it stands out from standard diagnosis of BD I which only requires the presence of a manic/mixed episode. In our study, we chose to incorporate a history of MDE because we wanted to examine the differences between depressive episode symptomatology in those with BD I compared to those with BD II.

Another factor that may contribute to under-recognition of BD II in the community is the decreased frequency of endorsed symptoms that was shown in our sample. Subjects presenting to a clinician with fewer reported symptoms may escape detection of a mood episode which further hinders appropriate diagnosis.

In terms of age of onset of first depressive episode, our results demonstrated no significant differences between the BD subtypes with a median age of 22 for both. This result is similar to previous publications (Benazzi, 1999; Judd et al.,

2003b; Serretti et al., 2002), but is in contrast to findings of Vieta et al. (1997) which showed BD II as having a 10 year later onset compared to those with BD I despite no significant difference in type of mood episode experienced at onset. Despite similarities between our study and that of Vieta in age of the sample, Vieta's results may reflect their relatively small clinical sample size (n = 60) (Vieta et al., 1997).

Self-reported family history of depression was highly prevalent in both subject groups (78.8% BD I and 67.8% BD II). The nature of the interview question did not specify whether family members had a diagnosis of unipolar depression or bipolar disorder, only whether they have suffered from a 2 week period of low mood (possible diagnosis of MDE). Therefore, the results do not address diagnosis-specific heritability, (Coryell et al., 1984; McMahon et al., 2001), but do highlight the fact that the significant majority of BD I and BD II subjects have had a family member with a potential history of MDE.

4.3. Depressive symptomatology

We examined specific symptoms as potential distinguishers of BD I and II, and demonstrated in our regression analysis that weight gain, psychomotor agitation, and feelings of worthlessness were significantly correlated with a BD I diagnosis. In our comparison of frequencies, we found higher rates of nearly all reported depressive symptoms collected, including those associated with atypical and melancholic type depressive episodes. This may lead to the assumption that subjects reporting a greater number of symptoms are more likely to have BD I. Our results are consistent with those of Weinstock et al. (2010) who demonstrated that mean depression severity in BD I was 0.56 standard deviation units higher than in BD II using Item Response Theory analysis (Weinstock et al., 2010), and Judd et al. (2003a, 2003b) who demonstrated greater severity of intake episode in BD I (Judd et al., 2003a,b).

Of great importance in BD are the high rates of suicide attempts that have been reported. Results in the literature have been mixed with some studies showing a greater number of suicide attempts in BD II compared to BD I (Ghaemi et al., 2008; Rihmer and Pestality, 1999; Serretti et al., 2002), while others report greater attempts in BD I compared to BD II (Tondo et al., 2007; Weinstock et al., 2010), or no differences in number of suicide attempts between the two groups (Vieta et al., 1997). Our results demonstrate greater frequency of both suicidal ideation (59.5% compared to 44.1%) and suicide attempts (28.9% compared to 14.6%) in subjects with BD I. Of note, our results represent attempts and not completed suicide. Risk factors for a suicide attempt may be different from those of completed suicides; however, a previous attempt is a strong predictor of a completed suicide. History of a suicide attempt remained an important correlate with BD I in our regression analysis with an OR of 1.8 ($p = 0.006$), indicating that subjects with BD I were significantly more likely to have attempted suicide.

4.4. Co-morbidities

In terms of substance use, subjects with BD I demonstrated similar rates of alcohol abuse/dependence compared to those with BD II (54.6% BD I and 51.8% BD II). However, rates of drug abuse/dependence were significantly higher in individuals with BD I (36.6% BD I compared to 26.7% BD II). These results are similar to those shown by Chengappa et al. (2000) which demonstrated non significant differences for alcohol dependence (28.2% BD I and 27.8% BD II), and significantly different rates of 57.8% for BD I and 38.9% for BD II for any drug or alcohol abuse/dependence. Results from the Epidemiologic Catchment Area (ECA) study, a large epidemiologic study conducted between 1980 and 1985, demonstrated rates of alcohol abuse or dependence as 46.2% in BD I and 39.2% in BD II (Regier et al., 1990). The study also showed the rates of drug abuse or dependence at 40.7% for BD I and 21.0% for BD II, which are similar to our findings (Regier et al., 1990). That study however, did not compare the rates of substance disorders between groups, only between those with mental disorder and those without.

In terms of anxiety disorders, our comparison of means demonstrated that all anxiety disorders (GAD, PD, social and specific phobias, and any anxiety disorder) were more commonly reported in BD I, with specific phobia emerging as being independently associated with BD I in our regression analysis. Similar findings from NESARC published by Grant et al. (2005a, 2005b) demonstrated that GAD was almost twice as prevalent in subjects with BD I than BD II, however, these results were dependent on classic DSM-IV primary diagnoses which differed from the population studied in this report (Grant et al., 2005a,2005b). In contrast, the patient population studied by Judd et al. (2003a, 2003b) demonstrated that GAD, phobic disorders, and the presence of any anxiety disorder were more prevalent in BD II compared to BD I, again reflecting the differences between clinical populations and community samples (Judd et al., 2003a). A population based study by Rihmer et al. (2001) found numerically higher rates of anxiety disorder comorbidity in BD II patients, however, the number of subjects was quite low and findings between groups were not statistically different (Rihmer et al., 2001).

Our data showed that BD I patients were more likely to express any of the personality disorders (Cluster A, B or C) and the presence of Cluster C personality disorders was a significant predictor of BD I in the regression analysis. Studies which have examined the prevalence of personality disorders in BD demonstrated correlations between BD and Cluster B and Cluster C personality disorders (George et al., 2003; Grant et al., 2004b; Rossi et al., 2001). However, the majority did not make direct comparisons between BD subtypes (George et al., 2003; Grant et al., 2004b; Rossi et al., 2001). One study did compare BD subtypes and identified differences in Axis II co-morbidities between BD I and BD II (Serretti et al., 2002). That study recruited patients from a hospital-based Mood Disorder Centre with each subject being independently assessed by two psychiatrists. Their results showed no difference in the prevalence of personality disorders in BD I and BD II (Serretti et al., 2002). However, the prevalence of personality disorders in that study (66.5% BD I and 56.5% BD II) (Serretti et al., 2002) is very similar to the prevalence we report (68.% BD I and 52.2% BD II).

4.5. Limitations

The limitations of this study include the lack of available information on several other potential differences between

BD I and II patients, for example depression with psychotic features, family history of BD, and post-partum mood symptoms which were not addressed in NESARC. In a comprehensive assessment of differences between BD I and BD II, these additional variables can be incorporated into the list of distinguishers we have identified.

Secondly, the retrospective nature of data collection affects the accuracy of recollection of symptomatology. This is a common limitation when using epidemiological datasets, however the reliability of the AUDADIS-IV for a BD diagnosis was good (Grant et al., 2005b). To date, there is no published reliability or validity data for the BD II diagnosis in NESARC. Demonstrating the differences in BD subtypes despite this recollection bias is precisely the goal of this study.

Finally, categorization of subjects as BD I or BD II was based on a subjective recollection of hypomanic/manic episodes which may confound the distinctions. However, this represents a clinical reality as well, affecting the gold standard of clinical diagnosis. The emergence of distinguishers despite possible contamination of groups speaks to the strength of the findings.

5. Conclusions

Distinguishing between BD I and BD II poses a significant challenge for clinicians and affects both management and prognosis. In our sample, subjects with BD I had a more severe clinical course as demonstrated by greater disability, greater frequency of reported depressive symptoms, and greater proportion of co-morbid conditions. This translates into greater use of medications and healthcare services such as hospitalizations, and indicates a poorer prognosis. The predictors that have emerged as significant in our comparison of BD I to BD II show the importance of addressing multiple aspects of the disorder when establishing a diagnosis, such as demographics, clinical variables, depressive symptom expression, and co-morbid conditions.

The majority of studies comparing BD I to BD II examine clinical samples which limits generalizability to the broader set of people with these illnesses. Our results indicate that a number of sociodemographic and clinical variables are differentially associated with each condition. Identifying differences between BD subtypes in a generalizable community sample can be used toward the development of enhanced diagnostic approaches that correctly identify the presence specific BD subtypes.

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Conflict of interest

Author Goldstein has received grant/research support from Pfizer as well as an Honoraria from Purdue Pharma. Author Schaffer has received grant/research support from Astra-Zeneca, Pfizer and CIHR, as well as served as speaker or on advisory boards for Astra-Zeneca, Eli Lilly, and Bristol-Myers Squibb, Lundbeck. All other authors declare that they have no conflicts of interest.

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