International Journal of Bipolar Disorders

RESEARCH Open Access

Perceived cognitive loss, symptomology, and psychological well-being with bipolar disorder

Ophir Haglili[1], Andrew Sixsmith[2], Ariel Pollock Star[3], Moshe Shmueli[4] and Norm O'Rourke[1,3,5*]

Abstract

Background Adults with bipolar disorder (BD) commonly present with cognitive deficits. Many also report subjective or perceived cognitive failures. For this study, we identified four distinct clusters of adults with BD on the basis of both BD symptoms (depression and hypo/mania) and perceived cognitive errors (i.e., forgetfulness, distractibility, false triggering). We hypothesized that participants reporting more BD symptoms and cognitive errors would report lower psychological well-being (i.e., self-efficacy, life scheme, life satisfaction). A second objective was to determine if and how clusters differed in terms of BD related factors (e.g., subtypes, sleep, medications) and sociodemographic differences such as age of participants. From the BADAS (Bipolar Affective Disorder and older Adults) Study, we identified 281adults with BD ($M = 44.27$ years of age, range 19–81), recruited via social media.

Results All clusters significantly differed across all grouping variables except symptoms of hypo/mania due to low frequency. Across clusters, perceived cognitive failures and BD symptoms increased in lockstep; that is, those reporting more cognitive errors also reported significantly higher symptoms of both depression and hypo/mania. As hypothesized, they also reported significantly lower psychological well-being.

Conclusions Age did not significantly differ across clusters in contrast to existing research in which cognitive loss is objectively measured. That is, perceived cognitive errors are significantly associated with lower psychological well-being for both young and older adults with BD.

Keywords Bipolar disorder, Depression, Hypo/mania, Mania, Perceived cognitive loss, Psychological well-being

Introduction

Adults with bipolar disorder (BD) commonly present with both objective and subjective or perceived cognitive deficits. Research indicates considerable variability in cognitive loss, with deficits most commonly observed in older adults with BD (Montejo et al. 2022a, b, c); however, associations are not strong between self-reported, subjective reports and objective cognitive deficits as measured using structured assessment batteries (Cullen et al. 2016; Miskowiak et al. 2018). That is, self-reported cognitive errors are a poor proxy for objective cognitive loss. Various explanations for this discrepancy have been

*Correspondence:
Norm O'Rourke
ORourke@bgu.ac.il
[1]Department of Psychology, Faculty of Humanities and Social Sciences, Ben-Gurion University of the Negev, Be'er Sheva, Israel
[2]STAR Institute, Simon Fraser University, Vancouver, BC, Canada
[3]School of Public Health, Faculty of Health Sciences, Ben-Gurion University of the Negev, Be'er Sheva, Israel
[4]Goldman School of Medicine, Faculty of Health Sciences, Ben-Gurion University of the Negev, Be'er Sheva, Israel
[5]Multidisciplinary Center for Research on Aging, Faculty of Health Science, Ben-Gurion University of the Negev, Be'er Sheva, Israel

suggested, yet reasons remain unclear. Irrespective, perceived cognitive errors directly and indirectly affect mental health (O'Rourke et al. 2017), and have been found to affect psychosocial functioning and quality of life for those with various neurological and psychiatric conditions (Groenman et al. 2022) including BD (O'Rourke et al. 2017).

Previous BD research has identified three naturally-occurring groups or clusters of objectively measured cognitive deficits (i.e., intact, selective impairment, & global severe impairment (Burdick et al. 2005; Lima et al. 2019; Montejo et al. 2022a, b, c; Solé et al. 2016). This study is the first to identify clusters of BD symptoms and perceived or subjectively-reported cognitive errors (i.e., forgetfulness, distractibility, false triggering). In addition, we identify differences in psychological well-being across naturally occurring groups of adults with BD (i.e., self-efficacy, life scheme, life satisfaction). And we compare BD related factors and sociodemographic features across clusters. For instance, older age was assumed to be significantly associated with severity of reported cognitive errors.

BD and cognitive functioning

BD is a chronic and episodic mental health condition characterized by mood variability. Symptoms and episodes of depression and mania or hypomania (i.e., hypo/mania) emerge across the life span, sometimes together (i.e., mixed symptom presentation (Grande et al. 2016)). Lifetime prevalence of BD is about 1–3% worldwide (Merikangas et al. 2011). And BD is highly heritable, with estimates from twin and family studies suggesting 60–85% heritability between first-degree relatives (Johansson et al. 2019; Smoller and Finn 2003).

BD is also a leading cause of disability worldwide due, in part, to early onset in most cases (i.e., late adolescence to late 20's), chronicity and impact on employment and relationships (Yerushalmi et al. 2021; Alonso et al. 2011; Tohen et al. 2000; Zarate et al. 2000; Martinez-Aran et al. 2007). Moreover, BD has the highest rate of completed suicides across psychiatric disorders (Plans et al. 2019). Fortunately, pharmacotherapy is effective for most with BD, yet subthreshold symptoms commonly persist (Fountoulakis et al. 2012). In addition to mood symptoms, cognitive loss is commonly reported (Arts et al. 2008).

Cognitive impairment and decline with BD is the focus of growing research and clinical interest (Montejo et al. 2022a, b, c; Lin et al. 2019). This includes both perceived or subjectively reported cognitive impairment and objective measurement (i.e., neurocognitive testing). Existing research suggests that primary cognitive deficits with BD include verbal memory, speed/reaction time, attention and executive functioning (Cullen et al. 2016; Latalova et al. 2011), evident during BD mood episodes and also when euthymic (Thompson et al. 2005).

However, there is high variability in cognitive loss across and within samples when cognition is objectively measured, ranging between 5 −57% for executive functions, 9 −51% for working memory, 23 −44% for speed/reaction time and 8 −42% for verbal memory (Cullen et al. 2016). Other studies suggest that euthymic persons with BD have significant cognitive impairment, yet many perform within age-adjusted normal parameters (Sparding et al. 2017).

One hypothesis that may explain these discrepancies is the *neuroprogression model of cognitive decline* which contends that recurrent psychosis (which occurs in manic states) disrupts homeostasis between inflammatory mechanisms, oxidative processes and neuroprotective mechanisms; these effects cause neural death and are cumulative (Serafini et al. 2021). Cognitive decline is associated with BD chronicity (Cardoso et al. 2015), meaning that more acute mood episodes and number of hospitalizations predict more pronounced cognitive loss. These deficits are thought to become self-evident and clinically manifest in middle age, accelerating age-related cognitive decline into later life (Szmulewicz et al. 2020). Therefore, age is likely associated with cognitive decline though findings are inconsistent (Montejo et al. 2022a, b, c; Robinson and Nicol Ferrier 2006). Some evidence supports the neuroprogression model (Serafini et al. 2021; Robinson and Nicol Ferrier 2006) but meta-analyses provide little evidence of progressive cognitive decline (Samamé et al. 2014, 2022). Nor have the effects of cognitive deficits on psychological well-being and quality of life with BD been fully examined.

Cognitive deficits are reliably measured by objective neuropsychological test batteries and brain imaging (i.e., fMRI, identified lesions) whereas perceived cognitive errors are measured by self-report. Of note, perceived and objectively measured cognitive deficits with BD are not strongly correlated (Miskowiak et al. 2018; Jensen et al. 2015). Some studies found no significant associations (Werf-Eldering et al. 2011) yet others reported that perceived cognitive deficits are moderately but not strongly correlated with objective measures of memory, attention, and executive function (Martinez-Aran et al. 2005). This discrepancy may be due to depressive symptoms, poor medication adherence, comorbid psychiatric conditions, and metacognitive capacity (Miskowiak et al. 2018).

Though perceived cognitive errors are a poor proxy for objective cognitive deficits, this does not negate the importance of the former. For instance, perceived or subjective cognitive errors may better reflect change from premorbid functioning, not fully captured by objective tests (Forcada et al. 2015). It has also been suggested that objective tests lack ecological validity relative to aspects

of cognition necessary for daily living (Mohn and Rund 2016; Elst et al. 2008). In fact, perceived cognitive deficits are strongly associated with distress and poor psychosocial functioning with various psychological and neurological conditions (Groenman et al. 2022). Perceived cognition is also significantly associated with quality of life with Alzheimer disease in contrast to cognition as objectively measured (Naglie et al. 2011). Similarly, subjective memory impairment predicts future depression among healthy older adults (Heun and Hein 2005).

Self-reported cognitive deficits with BD are associated with various psychosocial difficulties including inability to work, live independently, participate in leisure activities, and form and maintain social relationships (Demant et al. 2015). Though poor proxies of objective cognitive deficits (Burdick et al., 2005, Svendsen et al., 2012), perceived cognitive errors directly and indirectly affect mental health with BD (O'Rourke et al. 2017) as patients with cognitive complaints report more suicide ideation and attempts (i.e., self-harm (Luo et al. 2020; Lima et al. 2018)).

Perceived cognitive errors are associated with depressive symptoms while symptomatic and euthymic (O'Rourke et al. 2017). And frequency and severity of depressive mood episodes are a significant predictor of cognitive decline. It remains unclear however how various perceived cognitive errors differentially affect psychological well-being. Do effects accumulate over time (e.g., years since BD diagnosis, age), and are specific BD symptoms (i.e., depression vs. hypo/mania) associated with certain cognitive deficits?

Despite extensive research examining the neuropsychology of BD, many questions remain. One major question pertains to the considerable heterogeneity in cognitive function with BD (Montejo et al. 2022a, b, c). Using hierarchical cluster analyses performed on standardized test scores, previous BD research has identified distinct subpopulations: those cognitively intact (31-43%), those with selective cognitive impairment (28-46%) and a notable minority with global severe impairment (12-39%) (Lima et al. 2019; Montejo et al. 2022c; Millett and Burdick 2021). Intelligence (i.e., IQ), early versus late BD onset, years of educations and number of mood episodes significantly differ across clusters, but associations between age and cognitive decline with BD remain unclear (Lima et al. 2019; Montejo et al. 2022a, b, c; Millett and Burdick 2021). Nor does subjective cognitive loss significantly differ across clusters (Lima et al. 2019). Moreover, few studies address the proportion and heterogeneity of perceived cognitive failures (Lima et al. 2019; Grover et al. 2023). This underscores the need for a closer and separate look at perceived cognition in order to better understand its heterogeneity, the mechanisms accounting for it, and associations with BD symptoms and well-being.

Based on existing research, we similarly assumed that distinct clusters would form based on various types of subjective or perceived cognitive errors and BD symptoms (both depression and hypo/mania). Two to five cluster solutions were considered based on interpretability, cluster size, with statistically significant differences in cognitive errors and BD symptoms across clusters. We hypothesized that psychological well-being would significantly differ across clusters; that is, groups reporting more cognitive errors would report lower psychological well-being. We further hypothesized that those who report lower BD symptoms would report lower cognitive deficits, and greater psychological well-being.

A secondary objective was to compare BD related factors and sociodemographic variables across cluster. For instance, we assumed that those diagnosed decades ago and treated over many years would report more cognitive errors (i.e., older adults with BD). And in accord with the neuroprogression model, we assumed that cognitive errors are associated with age. That is, that those diagnosed decades ago and treated over many years would report more cognitive errors (i.e., older adults with BD).

Methods

Participant recruitment and online data collection

For this study, we identified 281 young and older adults with BD recruited for the BADAS (Bipolar Affective Disorder and older Adults) Study (Yerushalmi et al. 2021; O'Rourke et al. 2021a, b, c). Participants provided responses online to measures of cognitive loss, BD symptomology (both depression and hypo/mania) and psychological well-being. They also reported BD related factors (e.g., medications, BD subtype), sociodemographic factors such as age, and behavioral factors (i.e., sleep, alcohol consumption).

BADAS participants were recruited over 19 days using social media advertising targeted to adults with BD (Cohen et al. 2023). By clicking on advertisements appearing within Facebook newsfeeds, participants were directed to an online form that specified study inclusion criteria (King et al. 2014). Thereafter, they completed a series of counterbalanced online questionnaires hosted on a secure university https server; responses were encrypted before transmission (King et al. 2016).

Machine-generated algorithms calculated by social media platforms like Facebook are unique not so much for their *sensitivity* but *specificity* (i.e., exclusion of those who do not have BD). That is, participants recruited via social media are not representative of the population (Mühlhoff and Willem 2023); instead, they may be more symptomatic than outpatients attending mood disorder clinics (O'Rourke et al. 2021a, b, c), yet we can be

confident these are persons with BD as only Facebook users with BD received recruitment notices (King et al. 2014; Pollock Star et al. 2022). In previous research with BADAS participants, BD diagnoses were confirmed in clinical interviews (Stalman et al. 2018; O'Rourke et al. 2016) and corroborated by proxy informants (i.e., cohabiting partners (Yerushalmi et al. 2021).

Instruments

The BDS_x (Bipolar Disorder Symptom Scale) was developed to briefly measure symptoms of both depression and hypo/mania (hypomania + mania = hypo/mania: continuum where the point of transition is not always apparent (O'Rourke et al. 2016, 2018a, b). Respondents indicate the degree to which each of 20 mood adjectives corresponds to how they feel at that moment on a Likert scale ranging from *not at all* (0) to *a lot* (2). Though not designed as a screening measure, sensitivity for the depression subscale is high at 88% (6+, 76% specificity) but lower at 57% for the hypo/mania subscale (5+, 90% specificity (Osher et al. 2020).

Internal consistency of BDS_x responses has been reported as $\alpha = 0.91$ for the depression subscale (Cohen et al. 2023) but lower for the hypo/mania subscale at $\alpha = 0.76$ (Kraun et al. 2020). This 4-factor model appears stable over time (O'Rourke et al. 2016), with young and older adults with BD (O'Rourke et al. 2018a, b) and across BD subtypes (O'Rourke et al. 2021a, b, c). Construct validity is supported by research with cohabiting partners of persons with BD as affrontive symptoms of mania (e.g., furious, disgusted, argumentative) have the greatest negative impact on partners, especially when physically together (i.e., same GPS coordinates (Yerushalmi et al. 2021).

The Cognitive Failures Questionnaire (CFQ (Broadbent et al. 1982) measures subjective or perceived failures in perception, memory, and motor function (e.g., "Do you often forget people's names?"; "Do you lose your temper then regret it?"; "Do you bump into people?"). Responses to each of 17 items are reported along a Likert scale ranging from *never* (0) to *very often* (4). CFQ responses have been validated with general samples (Rast et al. 2009) and older adults with BD, $0.80 < \alpha < 0.88$ (O'Rourke et al. 2021c). Responses to the CFQ measure three distinct factors: Forgetfulness, distractibility and false triggering (i.e., interrupted processing of cognitive and motor actions (Rast et al. 2009).

The Spiritual Index of Well-Being (SiWB) was developed to measure health-related quality of life (QoL (Daaleman et al. 2004). Responses to each of 12 items are reported along a Likert scale ranging from *strongly agree* (1) to *strongly disagree* (5). The SiWB does not measure religiosity, but self-efficacy ($\alpha = 0.86$) and life scheme, $\alpha = 0.86$ (López et al. 2009). Test-retest reliability over 2-weeks has been reported as $r = .78$. Good internal consistency has been reported with various adult populations, $0.84 < \alpha < 0.90$ (Daaleman and Frey 2004).

The Satisfaction with Life Scale (SWLS (Diener et al. 1985) measures quality of life based on person-specific criteria. Participants respond to 5 questions with response alternatives ranging from *very strongly disagree* (1) to *very strongly agree* (7). Good internal consistency has been reported with older adults ($\alpha = 0.82$) (Heisel and Flett 2016), including older adults with BD, $\alpha = 0.89$ (O'Rourke et al. 2018b). Research indicates that depression is associated with lower SWLS, but unrelated to hypo/manic symptoms of BD (Meyer et al. 2004).

Pittsburgh Sleep Quality Index (PSQI) is a 19-item self-rated questionnaire that assesses perceived sleep quality (Buysse et al. 1989). Responses are reported along 4-point Likert scales. PSQI items measure sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleep medication, and daytime dysfunction. Each of these domains differs significantly between euthymic persons with BD and control participants (Saunders et al. 2013). PSQI responses are also significantly correlated with cognitive loss associated with BD (Russo et al. 2015). Construct validity of PSQI responses has been demonstrated using objective indices of sleep disturbance (i.e., actigraphy (Geoffroy et al. 2014). Prospective research suggests that the PSQI predicts BD mood episode recurrence even after controlling for residual mood symptoms (Cretu et al. 2016).

Alcohol Use Disorders Identification Test (AUDIT) is a self-report scale initially developed to measure alcohol misuse (Babor and Grant 1989). Responses are reported along a 5-point (8 items) and 3-point Likert scales (2 items). Internal consistency of AUDIT responses ranges from $0.74 < \alpha < 0.96$, and $\alpha = 0.86$ with BD (O'Rourke et al. 2018a, b). Concurrent validity has been demonstrated in comparison to the Michigan Alcoholism Screening Test, $r = .77$ (Allen et al. 1997). Research with the AUDIT demonstrates that cognitive loss is greater for BD patients with concomitant alcohol use disorders (Levy et al. 2008).

A socio-demographic questionnaire was constructed for the BADAS Study to collect descriptive and mental health information (e.g., psychotropic medication). Participants indicated their country of residence (drop-down menu), number of years of education, work or occupation, current employment, and relationship status. At recruitment, they were asked if they had BD, and subtype if known; at Time 2, they were asked if they had been diagnosed with BD by a clinician (e.g., psychiatrist), and date of BD diagnosis (month, year). We included only those who indicated that they both had BD, and had been diagnosed with BD.

Analytic strategy

Cluster analysis was performed using Ward's method, consistent with previous BD research (Lima et al. 2019; Montejo et al. 2022a, b, c; Millett and Burdick 2021). Clusters were formed based on three cognitive errors (i.e., forgetfulness, distractibility, false triggering) and four BDS_x factors (i.e., cognitive S_x depression, somatic S_x depression, elation/loss of insight, affrontive S_x hypo/mania). Analysis of Variance (ANOVA) compared clusters by age, education, number of prescribed psychotropic medications, employment, years since BD diagnosis, alcohol misuse and sleep quality.

Multivariate analyses of variance (MANOVA) were performed to confirm the distinctiveness of each cluster, compare sociodemographic features (e.g., age, medication), and to compare psychological well-being across clusters (i.e., self-efficacy, life scheme, life satisfaction). We assumed that those who reported fewer cognitive errors and lower BD symptoms would report greater psychological well-being.

Results

Our sample was composed of 199 women, 81 men and 1 person of undefined gender ($N=281$), ranging from 19 to 81 years of age. Participants were 44.27 years of age on average ($SD=13.59$), they were diagnosed with BD 10.01 years ago ($SD=8.69$, range=1–46) and completed 12.51 years of education ($SD=6.10$, range=1–26). Linear interpolation was performed to impute for missing scale responses (less than 2%, missing at random). Participants took 28.25 min on average to complete online questionnaires ($SD=25.83$).

Most participants lived in North America (38% Canada, 31% USA), with 15% from the U.K, 6% from Australia/New Zealand, and 5% from Ireland. In total, participants from 13 countries provided responses. The majority were Caucasian (91%) or mixed race (5%). Most (51%) were married or otherwise coupled, 14% were separated, 30% were single and 3.2% were widowed. Among those who specified a BD subtype, the plurality (42%) reported BD II, 29% BD NOS (not otherwise specified), 25% BD I, and 5% with cyclothymia. Most (64%) participants reported comorbid mental health conditions, with generalized anxiety disorder ($n=27$) and post-traumatic stress disorder ($n=21$) reported most often. See Table 1.

On average, participants were currently prescribed 2.35 psychotropic medications ($SD=1.66$) to treat BD and other mental health conditions. More precisely, most participants indicated that they were currently prescribed 1 or more mood stabilizer ($n=165$, 59%) and 1 or more antidepressant ($n=180$, 64%); smaller numbers listed 1 or more anxiolytic ($n=110$, 39%) and 1 or more antipsychotic ($n=107$, 38%). By category, lithium ($n=233$), bupropion ($n=131$), clonazepam ($n=168$), and quetiapine ($n=208$) were the medications most listed by participants (i.e., mood stabilizer, antidepressant, anxiolytic, and antipsychotic, respectively).

Cognition/BD symptom clusters

Hierarchical cluster analysis revealed distinct groups based on perceived cognitive errors and BD symptoms (both depression and hypo/mania). Participants appear to cluster in four distinct subgroups computed using squared Euclidean distance. MANOVA results indicate a significant multivariate effect across clusters by cognitive failures and BD symptoms, Wilk's $\lambda=0.11$, $p<.01$. With 37 participants in the smallest of four clusters, our sample is sufficient to identify medium to large effect sizes (where $\alpha=0.80$, $d=0.80$) (Cohen 2016).

Of note, symptom levels are somewhat greater than the BDS_x depressive cut-off score of 6+for both of the comparatively euthymic clusters, $M=7.57$ & $M=7.58$. In contrast, response levels were well above cut-off for both symptomatic clusters, $M=15.15$ & $M=14.60$. Hypo/

Table 1 Descriptive features and psychometric statistics for study variables ($N=281$)

	Mean (SD)	Range	Skewness	Kurtosis	alpha
Participant age in years	44.27 (13.59)	19–81	−0.21	− 0.82	
Years of education	12.51 (6.10)	1–26	− 0.48	− 0.59	
Duration of BD D_x (years)	10.01 (8.69)	1–46	1.42	2.24	0.73
PSQI Sleep quality	9.09 (3.03)	3–17	0.27	− 0.56	0.88
AUDIT Alcohol misuse	5.29 (6.32)	1–34	2.12	4.73	
BDS_x - depression	11.04 (5.44)	0–21	−0.20	−1.01	0.91
BDS_x - hypo/mania	4.37 (4.02)	0–20	1.16	1.25	0.81
CFQ - Distractibility	18.73 (6.49)	2–32	− 0.15	−0.46	0.83
CFQ - Forgetfulness	13.44 (4.06)	0–20	−0.49	− 0.28	0.76
CFQ - Triggering	6.84 (3.70)	0–16	0.29	−0.39	0.75
SiWB - Life Scheme	17.36 (6.75)	6–30	0.14	− 0.82	0.87
SiWB - Self-Esteem	19.72 (5.48)	6–30	−0.25	−0.50	0.84
SWLS Life Satisfaction	16.01 (7.57)	5–34	0.29	−0.85	0.88

Note D_x = diagnosis, PSQI = Pittsburgh Sleep Quality Index, AUDIT = Alcohol Use Disorders Identification Test, BDS_x = Bipolar Disorder Symptom Scale, CFQ = Cognitive Failures Questionnaire, SiWB = Spiritual Index of Well-Being, SWLS = Satisfaction with Life Scale

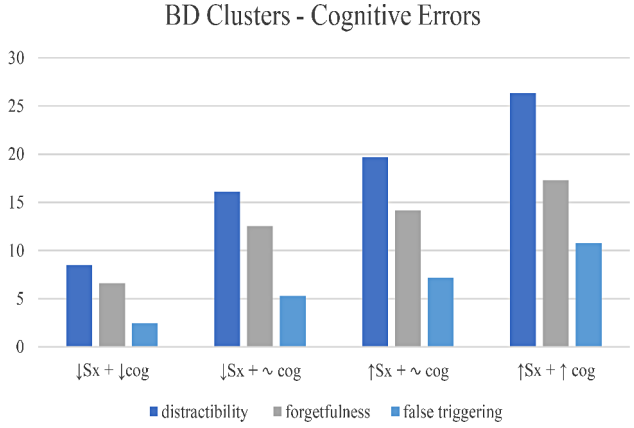

Fig. 1 Cognitive Errors Across Clusters: Distractibility, Forgetfulness and False Triggering. $\downarrow S_x + \downarrow cog$ = low BD S_x – low cognitive errors. $\downarrow S_x + \sim$ cog = low BD S_x – moderate cognitive errors. $\uparrow S_x + \sim$ cog = high BD S_x – moderate cognitive errors. $\uparrow S_x + \uparrow cog$ = high BD S_x – high cognitive errors. *Note*: y-axis = mean cognitive errors (Cognitive Failures Questionnaire), each cluster

mania response levels are elevated for both symptomatic clusters, $M=5.18$ & $M=6.36$. And hypo/mania response levels are below the 5+cut-off score for euthymic clusters, $M=2.73$ & $M=2.99$ (Osher et al. 2020).

Based on reported cognitive errors and BD symptomology, the smallest cluster was labeled **low symptoms and low cognitive errors**, $\downarrow S_x + \downarrow cog$; $n=37$, 13.1%; these participants appeared largely euthymic and reported few perceived cognitive errors. The largest cluster is characterized by **low symptoms and moderate cognitive errors**, $\downarrow S_x + \sim$ cog; $n=104$, 37.01%; these participants reported some cognitive loss though largely euthymic. The next cluster is characterized by **high symptoms and moderate cognitive errors**, $\uparrow S_x + \sim$ cog; $n=62$, 22.06%. And the final cluster is characterized by both **high symptoms and high cognitive errors**, $\uparrow S_x + \uparrow$ cog; $n=78$, 27.7%. Across clusters, perceived cognitive errors and BD symptoms largely increase in lockstep, both depression and hypo/mania. See Fig. 1.

All four clusters significantly differed for each of the three cognitive errors, Wilk's $\lambda=0.16$, $p<.01$. Moreover, post hoc analyses indicate that all clusters differed from all others for distractibility (F [3,277] = 329.96, $p<.01$), forgetfulness (F [3,277] = 173.47, $p<.01$) and false triggering, F [3,277] = 120.61, $p<.01$. Differences across clusters, however, were less pronounced for BD symptoms. See Table 2.

For instance, those in the two comparatively euthymic clusters ($\downarrow S_x + \downarrow cog$ & $\downarrow S_x + \sim$ cog) differed in neither depressive nor hypo/manic symptoms. The two symptomatic clusters ($\uparrow S_x + \sim$ cog & $\uparrow S_x + \uparrow$ cog) did not differ in depressive symptoms (neither cognitive nor somatic symptoms) nor affrontive symptoms of hypo/mania. See Fig. 2.

However, both euthymic clusters differ from both symptomatic clusters in affrontive symptoms of hypo/mania, cognitive and somatic symptoms of depression. Similarly, the two symptomatic clusters differ from both

Table 2 Cognitive errors across clusters: distractibility, forgetfulness and false triggering

	$\downarrow S_x + \downarrow cog$	$\downarrow S_x + \sim$ cog	$\uparrow S_x + \sim$ cog	$\uparrow S_x + \uparrow cog$
CFQ - Cognitive Errors				
• Distractibility	8.46 ***	16.11 ***	19.68 ***	26.36 ***
• Forgetfulness	6.62 ***	12.53 ***	14.18 ***	17.32 ***
• Triggering	2.46 ***	5.28 ***	7.16 ***	10.77 ***
BDS_x - BD Symptoms				
• Cognitive	3.73 **	3.77 **	9.00 **	8.71 **
• Somatic	3.84 **	3.79 **	6.15 **	5.89 **
• Elation/loss of insight	1.41	1.61	1.31 *	2.24 *
• Affrontive	1.32 **	1.38 **	3.87 **	4.12 **
	↑ Low/moderate ↑		↑ Moderate/high ↑	

* significantly differs from one other group,
** significantly differs from two other groups,
*** significantly differs from all other groups.

$\downarrow S_x + \downarrow cog$ = low BD S_x – low cognitive errors
$\downarrow S_x + \sim$ cog = low BD S_x – moderate cognitive errors
$\uparrow S_x + \sim$ cog = high BD S_x – moderate cognitive errors
$\uparrow S_x + \uparrow cog$ = high BD S_x – high cognitive errors

Note: S_x = BD symptoms, cog = Cognitive errors, CFQ = Cognitive Failures Questionnaire, BDS_x = Bipolar Disorder Symptom Scale

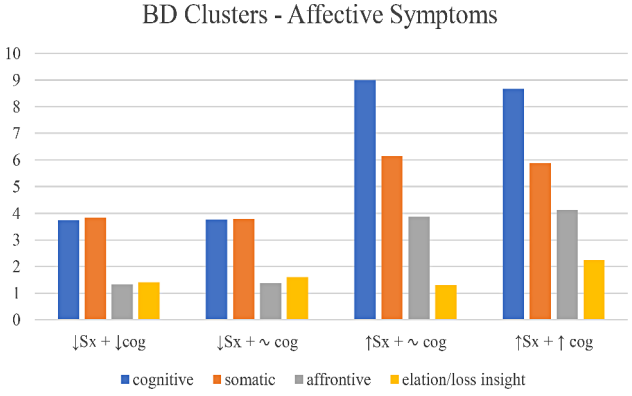

Fig. 2 Affective Symptoms Across Clusters: Depression and Hypo/mania *Note*: y-axis = mean BDS_x response levels across clusters, both depression and hypo/mania.↓S_x + ↓cog = low BD S_x – low cognitive errors.↓S_x + ∼ cog = low BD S_x – moderate cognitive errors. ↑S_x + ∼ cog = high BD S_x – moderate cognitive errors.

euthymic clusters for these three BD symptoms. Of note, elation/loss of insight differs only between the two symptomatic clusters. In other words, symptomatic clusters are high in cognitive errors and depressive symptoms (↑S_x + ∼ cog) and high in cognitive errors, depressive symptoms and hypo/mania, ↑S_x + ↑ cog.

Between cluster comparisons – sociodemographic features

There were no significant differences across clusters in education (F [3, 276] = 1.35; p = .26), relationship status (χ (Cullen et al. 2016) [df = 21] = 16.68, p = .73), gender (χ (Cullen et al. 2016) [df = 6] = 4.99, p = .55), ethnicity (χ (Cullen et al. 2016) [df = 18] = 11.01, p = .89) or country of residence, χ (Cullen et al. 2016) (df = 51) = 57.88, p = .24. Nor do clusters differ by BD subtype, χ (Cullen et al. 2016) [df = 9] = 10.58, p = .31. However, participants in the two relatively euthymic clusters were prescribed significantly fewer psychotropic medications than both symptomatic clusters, F(3,77) = 5.09, p < .01.

There were differences across clusters in reported sleep quality, F [3, 277] = 8.10, p < .01; yet post-hoc comparisons indicate that this difference is only between the high S_x – high CE cluster (↑S_x + ↑ cog) and all others; the other three do not significantly differ. Significant differences in alcohol misuse were also observed across clusters, F [3, 277] = 3.02; p = .03; here again, reported consumption was highest for the high S_x – high CE cluster (↑S_x + ↑ cog), significantly higher than both comparatively euthymic clusters (↓S_x + ↓cog & ↓S_x + ∼ cog), but not the high S_x – moderate CE cluster (↑S_x + ∼ cog).

Contrary to assumption, age did not significantly differ across clusters, (F [3, 277] = 0.30, p = .83) despite the wide age range of participants recruited for the BADAS study (19 to 81 years); those reporting more symptoms and cognitive errors were no older than those reporting few or any. And though duration of BD diagnosis significantly differed across clusters (F [3, 256] = 8.66, p < .01), those in the high S_x - high CE cluster (↑S_x + ↑ cog) were, in fact, diagnosed more recently with BD 7.43 years ago on average (SD = 6.24) in contrast to those in the low S_x - low CE cluster, ↓S_x + ↓cog, n = 260; M = 15.91 (SD = 12.09).

Neither result supports the neuroprogression model of cognitive decline, as older age and longer duration of diagnosis are generally correlated with number of BD mood episodes. Not only is age consistent across clusters, but those reporting fewer BD symptoms and cognitive errors were diagnosed twice as long ago on average as those reporting highest levels of symptoms and cognitive errors. This result is opposite than predicted by the neuroprogression model.

Comparison between clusters – psychological well-being

MANOVA revealed that all clusters significantly differed for each of the three measures of psychological well- being, Wilk's λ = 0.75, p < .01. That is, post-hoc tests indicate that all clusters significantly differ from all others in self-efficacy (F [3,277] = 23.33, p < .01), life scheme (F [3,277] = 15.20, p < .01), and life satisfaction, F [3,277] = 17.21, p < .01. The low S_x - low CE group (↓S_x + ↓cog) reported highest self-efficacy, followed by low S_x - moderate CE group (↓S_x + ∼ cog), moderate S_x - moderate CE group (∼S_x + ∼ cog) and high S_x - high CE group (↑S_x + ↑ cog). Self-efficacy significantly differed across all groups. See Table 3.

A similar inverse pattern between greater psychological well-being and BD lower symptoms/cognitive errors was observed also for life scheme and life satisfaction. However, the low to moderate clusters did not significantly differ for life scheme, and neither the low-moderate nor moderate-high clusters differed for life satisfaction. Nonetheless, as perceived cognitive loss and BD symptoms increase, psychological well-being declines. See Fig. 3.

Discussion

For this study, we categorized BD patients with varying levels of perceived cognitive failures and BD symptomology (both depression and hypo/mania). Using hierarchical cluster analyses, we identified four distinct clusters within two groupings. The comparatively euthymic clusters are: low S_x - low CE (low symptoms, few cognitive errors); and low S_x - moderate CE clusters (low symptoms, moderate cognitive errors). The symptomatic clusters are: high S_x - moderate CE (moderate cognitive errors, low hypo/mania, high depression); and high S_x – high CE clusters (high cognitive errors, high depressive symptoms, high hypo/mania). Differences between clusters are greater for cognitive errors than BD symptoms, and greater between- than within-groupings (i.e., the two

Table 3 Psychological well-being across clusters: Self-efficacy, Life Scheme and Life satisfaction

	$\downarrow S_x + \downarrow cog$	$\downarrow S_x + \sim cog$	$\uparrow S_x + \sim cog$	$\uparrow S_x + \uparrow cog$
Self-Efficacy	23.76 ***	21.26 ***	18.74 ***	16.54 ***
Life Scheme	20.57 **	19.43 **	16.26 ***	13.96 ***
Life Satisfaction	19.62 **	18.70 **	13.86 **	12.44 **
	Moderate/high		Low/moderate	

** significantly differs from two other groups,
*** significantly differs from all other groups.

$\downarrow S_x + \downarrow cog$ = low BD S_x – low cognitive errors
$\downarrow S_x + \sim cog$ = low BD S_x – moderate cognitive errors
$\uparrow S_x + \sim cog$ = high BD S_x – moderate cognitive errors
$\uparrow S_x + \uparrow cog$ = high BD S_x – high cognitive errors

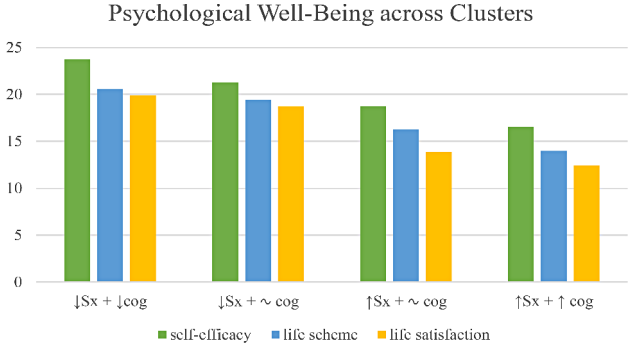

Fig. 3 Psychological well-being across clusters *Note*: y-axis = mean response levels for well-being measures, each cluster $\downarrow S_x + \downarrow cog$ = low BD S_x – low cognitive errors. $\downarrow S_x + \sim cog$ = low BD S_x – moderate cognitive errors. $\uparrow S_x + \sim cog$ = high BD S_x – moderate cognitive errors. $\uparrow S_x + \uparrow cog$ = high BD S_x – high cognitive errors

euthymic clusters more similar than both symptomatic clusters, and vice versa).

Our four cluster solution based on perceived cognitive errors (and BD symptoms) is distinct from previous BD research in which cognition was objectively measured. Each of these prior studies has proposed a three cluster solution: An intact cluster (no difference between BD and control groups); a selective deficits cluster with circumscribed deficits in specific domains; and a global deficits cluster with widespread deficits across domains (Burdick et al. 2005; Lima et al. 2019; Montejo et al. 2022a, b, c; Solé et al. 2016). Though the number of clusters differ, we similarly found that perceived cognitive errors increase along a continuum; cognitive loss appears progressive and incremental, not circumscribed to limited domains or specific BD symptoms.

Instead, perceived cognitive failures and BD symptomology increase largely in lockstep across clusters. That is, as perceived cognitive loss increases, so too BD symptoms, both depression and hypo/mania. And as we hypothesized, BD symptoms and cognitive errors are inversely associated with psychological well-being. As cognitive errors and BD symptoms increase, psychological well-being declines. Those reporting the highest symptom levels and cognitive errors were prescribed more psychotropic medications, reported poor sleep quality and alcohol misuse.

Previous research reports that depressive symptoms are closely linked to perceived cognitive loss (Demant et al. 2015; Lima et al. 2018; Miskowiak et al. 2012) suggesting that perceived cognitive loss may reflect depression and not cognition. Yet residual depressive symptoms between mood episodes do not fully explain these deficits (Luo et al. 2020; Toyoshima et al. 2019); moreover, reported that depressive symptoms do not appear to

moderate associations between subjective cognition and psychosocial functioning (Luo et al. 2020).

As our results suggest, hypo/mania along with depressive symptoms account for variance in perceived cognitive errors. Yet only patients in the final cluster reported significantly higher elation/loss of insight. Depressive symptoms were similar between clusters 3 and 4; only in the latter were classic mania symptoms elevated (e.g., talkativeness, euphoria). And BD patients in this cluster reported highest levels of all cognitive errors (i.e., forgetfulness, distractibility, false triggering). Some studies suggest that persons reporting hypo/mania tend to underestimate cognitive deficits due to increased energy and heightened productivity (Lin et al. 2019; Siegel-Ramsay et al. 2022). By contrast, our findings suggest that symptoms of hypo/mania are also associated with cognitive errors. Depressive symptoms alone do not fully account for perceived cognitive errors (Lima et al. 2018).

Contrary to our hypothesis, age did not differ across clusters. Both young and older adults with BD reported similar levels of perceived cognitive errors. According to some research and the neuroprogression hypothesis, cognitive decline with BD is associated with age; that is, older adults exhibit comparatively poor cognitive performance (Serafini et al. 2021). By contrast, we found no association between age and perceived cognitive errors consistent with other studies (Grover et al. 2023). In fact, patients who had lived with a BD diagnosis longest reported fewer cognitive errors and higher psychological well-being (Lima et al. 2018). We might speculate that those who lived longest with BD have learned to cope with their condition and learned to manage mood episodes (e.g., consistent exercise, diet, social support). This finding is important since it might indicate that intervention (e.g., psychotherapy) can be effective, especially in first years of diagnosis, when patients are learning to cope with the condition.

Limitations and directions for future study

The present study has various limitations. First, data are cross-sectional limiting our ability to make causal conclusions regarding the direction of influence between BD symptoms, cognitive errors and well-being. Associations between constructs may well be longitudinal, bidirectional and cross-lagged. For instance, medication and sleep quality examined above as dependent or outcome variables, significantly differ across clusters. Yet both are known to impact cognition and may significantly increase perceived cognitive errors. Mediation and regression analyses should be conducted in future research to better understand the strength and direction of associations over time.

Nor did we include a healthy comparison sample or employ objective measurement. BD symptoms, cognitive errors and psychological well-being were each measured by self-report alone; neither responses nor BD diagnoses were clinically corroborated. Future research should measure cognition objectively (e.g., standardized neurocognitive battery) and perceived or subjective cognitive errors along with psychological well-being.

Additionally, participants for this study were recruited online and may be more symptomatic than BD outpatients supported in the community (O'Rourke et al. 2021a, b, c). The results of this study should be replicated with BD participants recruited via more traditional measures (e.g., memory disorders clinics). This should include factors such as substance misuse (not just alcohol), known to adversely affect cognition with BD (Levy et al. 2008).

In summary, our findings suggest that adults with BD who report more perceived cognitive errors and BD symptoms also report lower psychological well-being (i.e., self-efficacy, life scheme, life satisfaction). This did not differ between young and older adults with BD. To the contrary, time since BD diagnosis appears to be inversely associated with cognitive errors. These findings underscore the importance of subjective or perceived cognition; perceived cognitive errors appear significantly and inversely associated with psychological well-being with BD.

How should clinicians understand perceived cognitive errors reported by patients? If reported while euthymic, impacting functional ability (e.g., employment) or quality of life, a neurocognitive assessment may be warranted. Yet perceived cognitive errors may be a stronger proxy for BD symptoms not cognitive decline. The results of this study suggest perceived cognitive errors are associated with symptoms of both depression and hypo/mania.

Author contributions

Conceptualization, H.G., A.S. and N.O'R.; Methodology, A.S. and NO'R.; Formal analysis, H.G. and N.O'R.; Writing—original draft, H.G., A.P.S., M.S. and N.O'R.; Writing—review & editing, H.G., A.P.S., M.S. and NO'R.; Supervision, A.S. and N.O.; Project administration, A.S. and N.O'R.

Data availability

Anonymized data are available from the corresponding author on reasonable request.

Declarations

Ethics approval

The BADAS Study conformed to the Principles of Human Rights, as adopted by the World Medical Association at the 18th WMA General Assembly, Helsinki Finland (June, 1964) and subsequently amended at the 64th WMA General Assembly, Fortaleza Brazil (October, 2013). As stated on the study *splashpage*, respondents indicated consent to participate by clicking to proceed. They were not required to provide identifying information aside from an email address if they wished. One randomly selected participant received a $500 lottery prize. The BADAS Study received approval from the Human Research Ethics Committee, Simon Fraser University, Burnaby (BC), Canada (#2014s0375). Support for the BADAS study provided by the Age-Well Network of Centres for Excellence [CRP 2015-WP6.2; O'Rourke] and Canadian Institutes of Health Research, Institute of Aging [RN 134209–259022] awarded

to Drs. O'Rourke and Sixsmith. Portions of this study were presented at the International Psychogeriatric Association Meeting (September, 2024) Buenos Aires, Argentina.

Competing interests
The authors declare no competing interests.

Received: 11 April 2024 / Accepted: 23 September 2024
Published online: 05 October 2024

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