Transdiagnostic Comparison of Visual Working Memory Capacity in Bipolar Disorder and Schizophrenia

Running Title: WM Capacity in the Schizo-Bipolar Spectrum

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35

36

37 **Word Count**

38 Total number of words in abstract: 245/350

39 Total number of words in text: 4112
ABSTRACT

Background

Impaired working memory is a core cognitive deficit in both bipolar disorder and schizophrenia. Its study might yield crucial insights into the underpinnings of both disorders on the cognitive and neurophysiological level. Visual working memory capacity is a particularly promising construct for such translational studies. However, it has not yet been investigated across the full spectrum of both disorders. The aim of our study was to compare the degree of reductions of visual working memory capacity in patients with bipolar disorder (PBD) and patients with schizophrenia (PSZ) using a paradigm well established in cognitive neuroscience.

Methods

62 PBD, 64 PSZ, and 70 healthy controls (HC) completed a canonical visual change detection task. Participants had to encode the color of four circles and indicate after a short delay whether the color of one of the circles had changed or not. We estimated working memory capacity using Pashler’s K.

Results

Working memory capacity was significantly reduced in both PBD and PSZ compared to HC. Working memory capacity in PSZ was also significantly reduced compared to PBD. Thus, PBD showed an intermediate level of impairment.

Conclusions

These findings provide evidence for a gradient of reduced working memory capacity in bipolar disorder and schizophrenia, with PSZ showing the strongest degree of impairment. This underscores the relevance of disturbed information processing for
both bipolar disorder and schizophrenia. Our results are also compatible with the cognitive manifestation of a neurodevelopmental gradient affecting bipolar disorder to a lesser degree than schizophrenia.

KEYWORDS

Bipolar disorder, schizophrenia, cognitive dysfunction, working memory capacity, attention
BACKGROUND

Cognitive impairment across a wide range of domains is a central common characteristic of both bipolar disorder and schizophrenia (Martínez-Arán, Vieta et al. 2004; Kahn and Keefe 2013; Vöhringer, Barroilhet et al. 2013; Bora and Pantelis 2015; Miskowiak, Burdick et al. 2018). Consequently, schizophrenia and more recently bipolar disorder have been conceptualized as information processing disorders (Kahn and Keefe 2013; Bortolato, Miskowiak et al. 2015). This development underscores the importance of transdiagnostic comparisons of crucial cognitive constructs, which might improve the precision of psychiatric classification systems (Insel 2014). So far, most of these studies have reported a gradient of cognitive impairment with patients with schizophrenia generally more affected than patients with bipolar disorder (Goldberg 1999; Schretlen, Cascella et al. 2007; Ivleva, Morris et al. 2010; Lewandowski, Cohen et al. 2011; Hill, Reilly et al. 2013; Reilly and Sweeney 2014).

Bipolar disorder and schizophrenia overlap substantially on both the phenomenological and pathophysiological level (Ivleva, Morris et al. 2010; Pearlson 2015). They have the highest amount of shared heritability among neuropsychiatric disorders (Anttila, Bulik-Sullivan et al. 2018) and are both regarded to different degrees as neurodevelopmental disorders (Bortolato, Miskowiak et al. 2015; Pearlson 2015; Owen and O'Donovan 2017). Given these similarities, studying specific cognitive impairments across both diagnostic categories is particularly relevant, because it should inform us about shared and distinct disturbances of the underlying brain systems (Insel 2014).

Working memory dysfunction is a central cognitive deficit in both bipolar disorder and schizophrenia (Glahn, Bearden et al. 2006; Barch and Smith 2008). Working memory is a crucial determinant of essential cognitive functions such as language.
comprehension and reasoning (Baddeley 1992), as well as an important mediator of
cognitive development and learning (Baddeley and Hitch 1974; Cowan 2014).

Working memory dysfunction has been reported in a large number of behavioral
studies in schizophrenia across all stages of illness (Lee and Park 2005; Barch and
Smith 2008; Luck and Gold 2008; Fuller, Luck et al. 2009; Hahn, Robinson et al.
2010; Anticevic, Repovs et al. 2011; Leonard, Robinson et al. 2017; Mayer, Stäblein
et al. 2018). Working memory impairment has also been demonstrated in bipolar
disorder (Adler, Holland et al. 2004; Glahn, Bearden et al. 2006; Thompson, Gray et
al. 2007; Mayer and Park 2012; Jensen, Knorr et al. 2016). While working memory
deficits appear to be particularly pronounced in manic or depressive phases
(Townsend, Bookheimer et al. 2010), they persist during euthymic phases of the
illness (Xu, Lin et al. 2012), at least in a sizable number of patients (Volkert, Kopf et
al. 2015). Direct comparisons between patients with bipolar I (BP-I) and bipolar II
(BP-II) disorder indicate overall a similar degree of working memory impairment
(Bora, Yücel et al. 2011; Bora 2018). Additionally, there is evidence for a modestly
greater degree of impairment in bipolar patients with a history of psychosis,
compared to bipolar patients without a history of psychosis (Bora 2018).

Based on the extensive body of work in the field of cognitive neuroscience (Luck and
Vogel 2013), working memory capacity has been proposed as a particularly suitable
construct for the translational study of working memory deficits in schizophrenia and
related neuropsychiatric disorders (Barch, Moore et al. 2011). Working memory
capacity is an easily measurable construct, with strong links to general levels of
cognitive functioning (Cowan 2014).

Visual working memory capacity has been studied most commonly using change
detection paradigms. Here, subjects have to remember one or more features such as
color, location or orientation of an array of simple visual items. Subsequently, after a
short period of time they are shown a test array and have to make a judgment, whether the test array is identical or if a single item had changed. Previous findings show that healthy individuals are able to store information of about four objects at one time as integrated features (Luck and Vogel 1997; Wheeler and Treisman 2002). Subjects are able to remember 3-4 items when required to encode a single feature such as color, or even two features of each item such as color and location. Variations of the ‘canonical’ change detection paradigm have also been implemented (Feuerstahler, Luck et al. 2019). In change localization paradigms, subjects need to specify which item has changed. In partial-report change detection paradigms, the change decision during the test array is limited to a single item. In multiple change detection paradigms, more than one item might change during the test array.

Reduced capacity of working memory has been observed in schizophrenia (Gold, Hahn et al. 2010; Mayer, Fukuda et al. 2012; Hahn, Robinson et al. 2018) and in bipolar disorder I with a history of psychosis (Gold, Barch et al. 2018). However, to our knowledge, no study has compared visual working memory capacity in cohorts of PSZ and PBD representing the full spectrum of both disorders. The main goal of our study was to assess working memory capacity in patients with bipolar disorder (PBD) of all illness subtypes, as well as patients with schizophrenia and schizoaffective disorder (PSZ) using a canonical change detection paradigm. We expected to observe a gradient of reduced working memory capacity with greater impairment in PSZ than in PBD.

**METHODS**

**Participants:**

We recruited 62 PBD (42 female, mean age 42.05, range: 20 - 61), and 64 PSZ (26 female, mean age 38.56, range: 20 – 57, n = 41 with schizophrenia and n = 23 with
schizoaffective disorder) from psychiatric outpatient clinics in and around Frankfurt am Main, Germany. Diagnoses of all patients were established according to DSM-5 criteria based on a clinical interview and careful chart review at a consensus diagnosis meeting chaired by one of the authors (R.A.B.). We pooled both patients diagnosed with schizophrenia and schizoaffective disorder because long-term diagnostic stability and inter-rater reliability of schizoaffective disorder is relatively poor (Maj, Pirozzi et al. 2000).

The Positive and Negative Syndrome Scale (PANSS) was used to assess current psychopathology in PSZ (Kay, Fiszbein et al. 1987). In order to establish euthymic mood state in BPD, participants were evaluated with the Young Mania Rating Scale (YMRS) and Montgomery-Åsberg Depression Rating Scale (MADRS) (Young, Biggs et al. 1978; Montgomery and Åsberg 1979). Participants with YMRS values of ≥ 11 or MADRS values of ≥ 11 were excluded from our analysis.

70 matched healthy control subjects (HC), (44 female, mean age 38.61, range: 21 – 61) also participated. HC had no reported history of psychiatric illness, as well as no history in first-degree family members. They were recruited from the Frankfurt University campus and surrounding areas, as well as by online and printed advertisements. Current and past symptoms of psychiatric illness were ruled out using the German version of the Structural Clinical Interview SCID-I, from the Diagnostic and Statistical Manual, Version IV (Saß, Wittchen et al. 2003).

All participants reported no history of neurological illness and no drug use (excluding nicotine) within the past six months. All participants ranged in age from 20-61 years old. We matched subject groups based on age, handedness, and participants’ years of education as well as parental years of education. The German Mehrfachwahl-Wortschatz-Intelligenz Test (MWT-B) (Lehrl, Merz et al. 2005) was administered to assess premorbid verbal intelligence. Handedness was determined with the
Edinburgh Handedness Inventory (Oldfield 1971). Further socio-demographic information for all cohorts can be found in Table 1. Prior to signing the informed consent form, participants were informed of its contents by the investigator and what to do in the case of experiencing distress and/or how to end participation in the study. The ethics committee of the University Hospital Frankfurt approved all study procedures.

Medication Scores:
Patients were on stable medication for at least one month at the time of study. One PSZ and two PBD did not receive medication. Details of medication can be found in Table 2. We calculated olanzapine equivalence scores (Gardner, Murphy et al. 2010) and daily lithium doses in both patient groups.

Change Detection Task:
We implemented a ‘canonical’ color change detection task (Figure 1) on a personal computer using Presentation software in Version14.9 (www.neurobs.com). Stimuli were presented on a grey background (RGB values: 191, 191, 191) in a dimly lit room with a viewing distance of approximately 60 cm. Throughout the experiment, a black fixation cross was displayed at the center of the screen. Each trial began with the alert phase, during which the fixation cross turned to red for 500 ms. This was followed by a preparation phase of 500 ms. During the encoding phase a sample array of four colored circles was presented for 200 ms. Each circle had a visual angle of approximately 0.95°. These circles were spaced equally apart on an imaginary circle with 12 possible locations around the black fixation cross covering a visual angle of approximately 5.25°, and the minimum distance between two circles was 0.29°. Each circle had one of seven easily discriminable possible colors with the
following RGB values: black (0, 0, 0), red (255, 0, 0), white (255, 255, 255), blue (0, 0, 255), green (0, 255, 0), yellow (255, 255, 0), and magenta (255, 0, 255), with no repetitions of colors within a trial. During the delay phase, the black fixation cross remained on the screen for 1800 ms. A whole-display recognition test array followed, in which participants had a maximum duration of 3000 ms to decide if the test array was identical to the sample array presented in the encoding phase, or if one of the circles had changed color. Half of the trials were ‘change’ trials (right mouse button), the other half ‘no change’ trials (left mouse button). In ‘change’ trials, a randomly chosen circle changed its color. The total duration of each trial was 6000 ms followed by an inter-trial interval of 3000 ms. All participants received the same instructions prior to the beginning the task, and were asked to perform as accurately as possible, and to keep their eyes fixated constantly on the center of the screen. A total of 60 trials were tested in each participant, which required approximately nine minutes of testing time.

Data Analysis:

In order to quantify the amount of information participants stored in working memory by each participant, we calculated Pashler’s $K$: $K = \frac{(\text{hit rate} - \text{false alarm rate})}{(1 - \text{false alarm rate}) \times N}$ (N = memory set size). (Pashler 1988) Our whole-display recognition paradigm required the use of Pashler’s $K$ as the appropriate estimate of working memory capacity. (Rouder, Morey et al. 2011)

A Shapiro–Wilk test across all three groups revealed that our working memory capacity estimates did not follow a normal distribution ($W(196) = 0.91, p < 0.01$). Consequently, we used the non-parametric Kruskal-Wallis test for our primary planned analysis in order to detect working memory capacity differences across all
three groups including pairwise comparisons. We calculated the effect sizes of the pairwise group comparisons within the Kruskal-Wallis test with $r = z / \sqrt{N}$.

We also performed a one-way ANCOVA to control for the possible influence of the covariates IQ and gender on working memory capacity. Possible group differences in demographic details including age, years of education, parental years of education, handedness and IQ were investigated with Kruskal-Wallis tests. Additionally, we performed separate bivariate correlations in PBD and PSZ to examine the relationship between duration of illness, olanzapine equivalence scores, daily lithium doses, as well as YMRS, MADRS, and PANSS scores with $K$.

We conducted a Mann-Whitney U test in order to investigate possible group differences in olanzapine equivalence scores for both patient groups. We conducted Spearman’s r bivariate correlations ($r_s$) to examine the relationship between working memory capacity and olanzapine equivalence scores in PSZ. We conducted another Spearman’s r bivariate correlation to examine the relationship between working memory capacity and daily lithium dose only in PBD because only three patients in the PSZ group received lithium treatment.

In order to evaluate possible working memory capacity differences in patient subgroups, we reviewed the pairwise comparisons within the Kruskal-Wallis tests of $K$ in patients with a) schizophrenia (SZ) versus schizoaffective disorder (SZAFF), b) BP-I versus BP-II, and c) bipolar with a history of psychotic symptoms (BP HPS+) versus bipolar without a history of psychotic symptoms (BP HPS-).

RESULTS

Our primary analysis revealed that $K$ was significantly different across all three groups ($H(2) = 17.34$, $p < .001$). Pairwise group comparisons revealed a significant difference of $K$ between PSZ and HC ($p < .001$, $r = .30$) and between PBD and HC ($p$
= .048, \( r = .14 \)). There was also a significant reduction of working memory capacity in PSZ compared to PBD (\( p = .035, \ r = .15 \)) (Table 3).

Nominally higher, but not significantly different IQ values were recorded in PBD (mean = 117.84) than HC (mean = 116.03, \( p = .307, \ r = .07 \)). Lower IQ was recorded in PSZ (mean = 110.34) compared to PBD (\( p < .001, \ r = .26 \) and HC (\( p = .006, \ r = .20 \)). Gender was also not matched across participant groups (\( \chi^2(2,196) = 10.91, \ p = .004 \)). However, in a one-way ANCOVA there was no significant relation of IQ, the covariate, to K across all groups (\( F(1, 191) = 0.31, \ p = .860, \ r = .01 \)). There was also no significant relation of gender, the covariate to K across all groups (\( F(1, 191) = 0.60, \ p = .440, \ r = .17 \)). A significant effect of group on K was still observed after controlling for the effects of IQ and gender (\( F(2, 191) = 6.70, \ p = .002, \ partial \eta^2 = 0.07 \). We recorded higher olanzapine equivalence scores in PSZ (\( Mdn = 13.41 \)) compared to PBD (\( Mdn = 2.50 \)) (\( U = 626.50, \ z = -6.60, \ p < .001, \ r = .67 \)). Yet, we observed no significant correlation of K with olanzapine equivalence scores in either PSZ (\( r_s = .09, \ p = .502, \ n = 62 \)), or PBD (\( r_s = .15, \ p = .242, \ n = 35 \)). Similarly, we observed no significant correlation between K and daily lithium dose in PBD (\( r_s = .08, \ p = .692, \ n = 29 \)).

There was also no significant correlation between K and years of illness in either PBD (\( r_s = .14, \ p = .291, \ n = 62 \)), or PSZ (\( r_s = .04, \ p = .782, \ n = 64 \)). There was also no significant correlation between K and scores on the YMRS (\( r_s = .03, \ p = .812 \)), or MADRS (\( r_s = .18, \ p = .151 \)) in PBD, and K and PANSS scores in PSZ (\( r_s = .12, \ p = .361 \)).

Regarding our exploratory patient subgroup analyses, we observed no significant differences of K in any of our pairwise Kruskal-Wallis tests. This included the comparison of a) schizophrenia (SZ) versus schizoaffective disorder (SZAFF) (\( p = .440, \ r = .06 \)), b) bipolar I (BP-I) versus bipolar II (BP-II) (\( p = .646, \ r = .03 \)), and c)
bipolar with a history of psychotic symptoms (BP HPS+) versus bipolar without a
history of psychotic symptoms (BP HPS-) ($p = .690$, $r = .03$), (Table 3).

**DISCUSSION**

We investigated working memory capacity in bipolar disorder and schizophrenia
compared to healthy controls in order to elucidate the degree of working memory
impairment in these major psychiatric disorders. We observed a significant reduction
in working memory capacity in PBD compared to HC and in PSZ compared to HC.
Working memory capacity of PBD fell between PSZ and HC with a significant
difference between both patient groups.

Our results indicate a gradient of reduced working memory capacity across the
schizo-bipolar spectrum, with PSZ showing a stronger impairment with a small to
medium effect size. By comparison, working memory capacity reduction in PBD
appeared to be less pronounced with a small effect size. These findings match
previous studies on working memory dysfunction in both disorders, which indicate a
similar intermediate level of working memory impairment in PBD (Hamilton, Altshuler
et al. 2009; Barch and Sheffield 2014). Our findings are also well in line with previous
studies demonstrating a comparable gradient of impairment across a wide range of
other cognitive domains (Goldberg 1999; Schretlen, Cascella et al. 2007; Ivleva,
Morris et al. 2010; Lewandowski, Cohen et al. 2011; Hill, Reilly et al. 2013; Reilly and
Sweeney 2014).

Our three participant groups differed in their levels of premorbid IQ. We report
significantly higher IQ in PBD and HC as compared to in PSZ, and no significant
difference between PBD and HC. However, there is evidence, that premorbid
intelligence scores tend to be lower in PSZ (Crawford, Besson et al. 1992;
Khandaker, Barnett et al. 2011), and supranormal in multiple measurements in PBD.
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(Bora 2015). Nevertheless, we did not observe a significant influence of IQ on $K$, which is unsurprising considering we measured crystallized intelligence. In comparison, fluid intelligence seems to be correlated with working memory capacity (Fukuda, Vogel et al. 2010). Therefore, the divergent levels of premorbid IQ in our cohorts should not have affected our results.

Recently, a large multi-center study using both a change localization and a multiple change detection task reported an overall reduction of visual working memory capacity across psychotic disorders, i.e. schizophrenia, schizoaffective disorder and BP-I with a history of psychosis (Gold, Barch et al. 2018). The authors did not observe a significant difference between any of the three patient groups in either task. For the change localization task, visual working memory capacity was significantly lower in all three patient groups. Interestingly, for the multiple change detection task this reduction was most pronounced in patients with bipolar I disorder with a lifetime history of psychosis, with only a trend towards a significant reduction in patients with schizophrenia. These results indicate that visual WM capacity reduction in BP-I with a lifetime history of psychosis is similar in magnitude to schizophrenia. However, it remains unclear, whether this similarity is attributable to the shared presence of psychosis, because the study did not include either bipolar I disorder without a lifetime history of psychosis or bipolar II disorder. In contrast to Gold et al., we observed an intermediate level of visual working memory capacity reduction in PBD. Several factors could account for this discrepancy. Firstly, we studied the full bipolar spectrum including BP-I without a history of psychosis and BP-II, rather than focusing only on the effect of psychotic illness across diagnostic categories. Interestingly, our exploratory subgroup analyses did not indicate a difference in visual working memory capacity between BP-I and BP-II. We also did not observe an effect of lifetime history of psychosis within the BPD group. However, these post-hoc
findings need to be interpreted with caution due to the relatively small size of our subgroups.

There were also important differences in the paradigms employed. The number of possible changes might have influenced results. Gold et al. observed a significant difference between healthy controls and all patient groups for their change localization task, which was a single change paradigm. By contrast, group differences were less pronounced in their multiple change detection paradigm with either zero, one, two or five changes. Furthermore, we consistently maintained a set size of four objects, while Gold et al used a set size of five objects. This difference might have affected results, because performance continues to decline as set size increases (Luck and Vogel 1997). This interpretation would also be in line with the notion, that deficits in PBD become more pronounced in tasks greatly exceeding their working memory capacity. It has been suggested, that a larger set size might have influenced subjects' strategies and minimized between-group differences (Gold, Barch et al. 2018). Conversely, there was some indication of a ceiling effect in the HC group in our data with 14 HC having a $K$ of 4 compared to 3 PBD and 3 PSZ (Figure 2). This implies, that our set size might have possibly underestimated working memory capacity differences between HC and both PBD and PSZ. Using both set sizes within the same study might help to clarify this issue.

The observation of a transdiagnostic reduction of working memory capacity raises the question, which cognitive mechanisms might be responsible. It has been proposed that these limits of working memory capacity are determined primarily by the amount of information which can be held in the focus of attention (Cowan 2001). Specifically, top-down attention appears to be crucial for the selection of information to be stored in working memory. The efficiency of this “gatekeeper” function has a substantial impact on working memory capacity (Vogel, McCollough et al. 2005; Cowan and
Morey 2006; McNab and Klingberg 2008). Thus, impaired attentional processes in patients might have contributed to our results. There is evidence for a selective impairment of top-down attentional control in schizophrenia, which may impair working memory encoding (Mayer, Fukuda et al. 2012). It has also been suggested, that attentional filtering could be mainly impaired in auditory information processing, while PSZ tend to show hyper focusing of attention when processing visual information (Luck, Leonard et al. 2019). Specifically, this hyper focusing might limit the amount of items which patients can focus on, thereby restricting the amount of information they can successfully encode into memory (Leonard, Kaiser et al. 2013; Luck, Hahn et al. 2019). To our knowledge, the potential influence of attentional dysfunction on working memory impairment has not yet been studied in bipolar disorder.

A number of behavioral and neuroimaging studies have provided evidence for a primary impairment of working memory during the encoding stage (Tek, Gold et al. 2002; Hartman, Steketee et al. 2003; Lee and Park 2005; Kim, Park et al. 2006; Haenschel, Bittner et al. 2007; Javitt, Rabinowicz et al. 2007; Fuller, Luck et al. 2009; Haenschel, Bittner et al. 2009; Gold, Hahn et al. 2010; Hahn, Robinson et al. 2010; Anticevic, Repovs et al. 2011; Mayer, Fukuda et al. 2012; Bittner, Linden et al. 2015). So far, it remains unclear, to which degree disturbances during the initial encoding of information contribute to reduced working memory capacity in bipolar disorder and schizophrenia. Interestingly, there is some evidence for differential mechanisms underlying working memory impairment in schizophrenia and bipolar disorder. While both groups were impaired in a spatial delayed response task, only patients with schizophrenia recorded more false memory responses by confidently responding that the information was correctly encoded although it was not (Mayer and Park 2012). Furthermore, there is evidence for an additional impairment of working memory...
maintenance in schizophrenia (Reilly, Harris et al. 2006; Stephane and Pellizzer 2007; Badcock, Badcock et al. 2008), which could also contribute to reduced working memory capacity. Conversely, the presence of working memory maintenance deficits has not yet been investigated in patients with bipolar disorder. Future studies should try to elucidate the contribution of specific component processes to reduced working memory capacity across both disorders.

Importantly, two different models for working memory storage have been discussed. Originally, a discrete-slots model was proposed, where a specific number of items are stored up to capacity, and nothing is stored from the remaining items (Miller 1956; Luck and Vogel 1997; Cowan 2001). More recently, working memory capacity has been studied using a limited-resource model in which a dynamic precision resource spreads out across objects such that a smaller amount of objects are encoded with higher precision (Bays and Husain 2008; Peters, Rahm et al. 2018).

Although our paradigm was able to measure working memory capacity in a slot model, it would be interesting to conduct an investigation of both the discrete-slot model and limited-resource models with the same patient groups.

CONCLUSION

To summarize, our data provide evidence for reduced visual working memory capacity in both bipolar disorder and schizophrenia. The observed gradient of cognitive dysfunction is compatible with the existence of a neurodevelopmental continuum (Owen and O'Donovan 2017), which implies that risk factors disturbing brain development and cognition play a larger role in schizophrenia than in bipolar disorder. This would place bipolar disorder between schizophrenia and healthy controls on a neurodevelopmental gradient, matching the degree and persistence of overall functional impairment in both disorders. In order to illuminate this issue
further, the neurophysiological underpinnings of the gradient of working memory capacity reduction need to be investigated. This would help to determine, whether the gradient of cognitive impairment can be explained by a gradual manifestation of the same neurophysiological disturbances across diagnostic categories, or by different mechanisms. Similarly, it remains an open question how shared and distinct genetic and environmental risk factors for either disorder might influence working memory capacity on the cognitive and neurophysiological level. Given their relevance for patients’ functional capacity, future studies should also examine whether pro-cognitive interventions such as cognitive remediation could improve these deficits across diagnostic categories. Finally, our results underscore the utility of established constructs based on cognitive neuroscience for the investigation of impaired information processing in bipolar disorder similar to such endeavors in schizophrenia research (Barch and Smith 2008).
SUPPLEMENTARY INFORMATION

Abbreviations

PBD: patients with bipolar disorder
PSZ: patients with schizophrenia
HC: healthy controls
YMRS: Young Mania Rating Scale
MADRS: Montgomery-Åsberg Depression Rating Scale
PANSS: The Positive and Negative Syndrome Scale
BP-I: patients with bipolar I
BP-II: patients with bipolar II
SZ: patients with schizophrenia
SZAFF: patients with schizoaffective disorder
BP HPS+: bipolar with a history of psychotic symptoms
BP HPS-: bipolar without a history of psychotic symptoms
K: Pashler’s K
SCID-I: Structural Clinical Interview
SD: Standard deviation
YOE: years of education
PYOE: parental years of education
DOI: duration of illness
MWT-B: German Mehrfachwahl-Wortschatz-Intelligenz Test

Acknowledgements

The authors are grateful to Benjamin Peters for helpful discussions, to Peter Hustedt, Michael Grube, Christoph Fehr, Martin Hambrecht for their support in recruiting
patients, as well as to Lisa Goldschmidt, Tobias Lehmann, and Deliah Macht for help with data acquisition.

Authors’ contributions

All authors made substantial contributions to the conception or design of the work, or the acquisition, analysis or interpretation of data. Authors RAB and LR designed the experiment. Authors CVB-S, CM and LR acquired the data. Authors CVB-S, JSM, SM and RAB analyzed the data. Authors CVB-S and RAB wrote the first draft of the manuscript. All authors contributed to and revised the manuscript. All authors read and approved the final manuscript.

Funding

C.V Barnes-Scheufler was supported by a “main doctus” scholarship from The Polytechnic Foundation of Frankfurt am Main.

Availability of data and materials

The datasets used during the current study are available from the corresponding author on reasonable request.
Ethics approval and consent to participate

The ethics committee of the University Hospital Frankfurt approved all study procedures. The study was carried out following the rules of the Declaration of Helsinki.

Consent for publication

Not applicable.

Competing interests

The authors declare that they have no competing interests.
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