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Metacognitive functioning in bipolar disorder versus controls and its correlations with neurocognitive functioning in a cross-sectional design

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ABSTRACT

Introduction: Metacognition is an important factor in the development and persistence of bipolar disorder. One of the most striking examples of impairment in metacognitive functioning in bipolar disorder is the lack of insight these patients have in their disorder. Despite its importance, research regarding metacognition in bipolar disorder is scarce. Furthermore, the neurocognitive basis of metacognitive functioning is unknown.
Methods: The current study included 29 patients with bipolar disorder and 29 age, educational level and gender matched healthy controls. All the participants filled in a metacognition questionnaire that examined their metacognitive beliefs. In addition, it was tested how well they estimated their performance on a neurocognitive test-battery beforehand (metacognitive knowledge) and afterwards (metacognitive experience).
Results: Bipolar disorder patients showed maladaptive metacognitive beliefs in comparison with the healthy controls. They also showed impaired metacognitive knowledge and experience. That is, they overestimated their own cognitive performance. However, the latter result was also true for the healthy controls. In addition, metacognition had neurocognitive correlates. However, for the bipolar patients, depressive symptomatology had an important effect on this relationship and on metacognition in general.
Conclusion: Maladaptive metacognitive skills are related to depression in bipolar disorder. A more healthy metacognitive thinking should be promoted. An effective training for this could be a therapy that includes various elements, from basic cognitive- to higher order metacognitive training.

1. Introduction

Metacognitive functioning refers to the ability to think about our thinking [1]. The versatility of the term metacognition is been discussed in a recent paper of Moritz and Lysaker [2]. Metacognition finds its origin in experimental psychology. Here, it has been referred to as "the study of one's own cognitive processes or anything related to them" [3]. Metacognition as defined by Flavell [3], has two key components, which are "metacognitive knowledge" and "metacognitive experience". Metacognitive knowledge handles about the beliefs that we have about our general cognitive functioning. Metacognitive experience, on the other hand, involves the perceived performance on tasks that assess our cognition [3,4]. Overall, it is generally accepted that metacognition involves a wide range of abilities, including the formation and reasoning about representations of our own mental states and the states of individuals who are surrounding us, in order that we are able to make decisions, solve interpersonal conflicts or problems and to master subjective suffering [5–7]. As such, metacognition or the knowledge about one's own mental state is crucial to every day decision-making. Despite its importance, such metacognition has proven to be highly fallible with many examples of self-knowledge being at best imperfect [8,9]. One of the most striking examples of metacognitive failure is the lack of insight that patients with chronic mental disorders have regarding their illness [10]. This makes metacognitive functioning an important factor in the development and the persistence of psychiatric disorders [11]. For instance, metacognition could direct someone's focus on disorder congruent information or it could contribute in the development of unhelpful coping strategies such as rumination and excessively worrying [12] which in turn could lead to the maintenance of depressive symptoms [13,14]. Although this makes metacognitive functioning clinically a very relevant topic to study in mood disorders, research in bipolar disorder is scarce.

Investigations that studied metacognitive knowledge in bipolar disorder found that these patients poorly estimated their own cognitive performance prior the neurocognitive tests [4,15,16]. However, one study found that patients with bipolar disorder are accurate in estimating their performance on a specific task after completing it, which could suggest intact metacognitive experience [4]. Though, the latter investigation only asked the participants about their performance on one specific memory task, which makes its results impossible to generalize to

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overall cognitive functioning. Furthermore, Sarisoy et al. [17] assessed more general metacognitive functions in bipolar disorder, what we will call metacognitive beliefs. They found that this patient group has impaired metacognitive skills (measured by a metacognition questionnaire) in comparison with healthy controls. On the other hand, Popolo et al. [18] found no difference between bipolar disorder patients and healthy controls on one of the two questionnaires they presented.

Furthermore, although research in schizophrenia points to a neurocognitive basis of poor metacognitive functioning [19,20], only one study has examined this possibility in bipolar disorder [21]. The latter investigation found that deficiencies in executive functioning and verbal memory were correlated with impairments in metacognition. However, this study only included these two cognitive domains, leaving out the possibility that other cognitive functions are associated with poor metacognition in bipolar disorder.

Taken together, former studies regarding metacognition in bipolar disorder show mixed results. In addition, the neurocognitive basis of metacognitive functioning in bipolar disorder is unknown. Because of the clinical relevance of metacognition, the current investigation will study this topic in more detail by examine if patients with bipolar disorder show impairments in different components of metacognitive functioning in comparison with a healthy control group. In addition, the current research will assess if metacognition is associated with neurocognitive functioning in bipolar disorder. Because of the lack of previous research on metacognition in bipolar disorder this investigation tried to give as a complete picture as possible by including self-appraisal (metacognitive knowledge and metacognitive experience) and more general (metacognitive beliefs) assessments of metacognition.

Based on previous research [4,15–17] we hypothesize that patients with bipolar disorder will display impairment in metacognitive knowledge and metacognitive beliefs. Furthermore, we expect that metacognition, and more precise metacognitive beliefs, will have neurocognitive correlates in bipolar disorder [19,20]. Specifically, we expect positive associations between metacognitive functioning and memory and executive functioning.

2. Method

2.1. Participants

This study included 29 inpatients diagnosed with bipolar disorder as confirmed by the Mini International Neuropsychiatric Interview (MINI) [22] and 29 age, gender and educational level matched healthy controls ($p = .131$; $p = .296$; $p = .236$, respectably). In the patient group the mean age was 39.43 years old (SD = 10.69) and there were more women (19) included than men. Only two patients did not take any medication. All other participants were on combined treatment that included intake of benzodiazepines, antidepressants (tricyclics or paroxetine), mood stabilizers, and/or antipsychotic medication. The average duration of the illness at time of assessment was 21.25 years. In the control group the mean age was 34.28 years old (SD = 14.44) and more women participated (16) than men. The level of education that was most predominantly present in both groups was bachelor degree.

The exclusion criteria were: inability to speak and understand the Dutch language, present alcohol or drugs dependency, dementia or other neurological/medical conditions that are known to affect cognition or other medical problems that hindered the ability to complete the assessments. With regards to the healthy control group the following exclusion criteria were applied: current or past diagnosis of major depressive disorder, bipolar disorder, schizophrenia of schizoaffective disorder as defined by DSM-IV-TR criteria [23], bipolar disorder or schizophrenia of schizoaffective disorder in a first-degree family member, present alcohol or drugs dependency, current intake of psychopharmacologic medication, dementia or other neurological/medical conditions that are known to affect cognition or other medical problems that hindered the ability to complete the assessments. The local committee approved the study (EC 1718), and all participants provided written informed consent prior to the study entry.

2.2. Instruments

2.2.1. Mood questionnaires

The Young Mania Rating Scale (YMRS) [24] was used to assess the presence or absence of manic symptomatology. The YMRS is composed of 11 items and the total score can range from 0 to 60. A score of <12 indicates a remission of manic symptoms. Secondly, the Hamilton Depression Rating Scale (HDRS) [25] was used to measure the presence and severity of depressive symptomatology. It is a clinician-administered scale and contains 17 items. The total score can range from 0 to 52. A score of 0–7 is seen as normal, a score between 8 and 13 is accepted to represent a mild depression, a score between 14 and 18 is seen as a moderate depression, a score between 19 and 27 represents a severe depression, and scores over 20 indicate a very severe depression [25].

2.2.2. Neurocognitive assessment

By the use of the International Society for Bipolar Disorders Battery for assessment of Neurocognition (ISBD-BANC) [26] the neurocognitive functioning of the patients and the healthy controls was measured. The ISBD-BANC is a battery especially designed to measure neurocognitive functioning in bipolar disorder and takes between 1 and 2 h to complete. It holds the following cognitive domains: speed of processing (Trail Making Test-Part A, Brief Assessment of Cognition in Schizophrenia-Symbol Coding, and Category Fluency: Animal naming); attention (Continuous Performance Test-Identical Pairs); working memory (Wechsler Memory Scale-Spatial Span, Letter Number Span); learning (Hopkins Verbal Learning Test-Revised, Brief Visuospatial Memory Test-Revised); executive functioning (Neuropsychological Assessment Battery-Mazes, Trail Making Test-Part B, Color-Word Stroop); and social cognition (Managing Emotions). In order that higher scores indicated a better performance neurocognitive test scores that had inconsistent metrics were revised. The scores on each test were converted to z-scores based on the scores of the control group. Subsequent, the following neurocognitive domains were calculated: speed of processing, attention, working memory, learning, executive functioning, and social cognition. In addition, an overall cognition score was analyzed based on the average of all the z-scores.

2.2.3. Metacognitive measures

2.2.3.1. Metacognitive beliefs. The metacognitive beliefs of all the participants were assessed by the use of the Metacognitions Questionnaire short form (MCQ-30) [12]. The MCQ-30 is a self-rating scale and comprises 30-items. The participants reported on their metacognitive beliefs across five dimensions: cognitive confidence (e.g. “I do not trust my memory”), positive beliefs about worry (e.g. “worrying helps me to get things sorted in my mind”), cognitive self-consciousness (e.g. “I am constantly aware of my thinking”), uncontrollability and danger (“when I start worrying I cannot stop”), and the need to control thoughts (e.g. “if I could not control my thoughts, I would not be able to function”). Participants reported on a 4-point Likert scale (from 1 “I do not agree” to 4 “I agree very much”). Higher scores indicate more maladaptive metacognitive beliefs.

2.2.3.2. Metacognitive knowledge and experience. Both metacognitive knowledge and experience were calculated based upon the accuracy of the estimated neurocognitive performance following the method of Torres et al. [4]. Metacognitive knowledge was assessed by asking the subjects to rate their perceived general cognitive functioning before the start of the cognitive testing. More specifically, the following question was asked: “compared to healthy individuals of your age, your cognitive skills (concentration, memory, problem solving,…) are…”. The subjects were asked to answer this question on a Likert scale with the

following options (−3) profoundly below average, (−2) well below average, (−1) below average, (0) average, (+1) above average, (+2) well above average, or (+3) superior.

Metacognitive experience was assessed by asking the subjects how well they thought they performed on the cognitive tasks after completing the test battery. In the same manner as metacognitive knowledge was assessed the subjects filled in a Likert scale with the same options as described above.

The subdivision of the Likert scale was chosen in order that it would correspondent with the z-score value of the overall cognition score. For example, a score on the Likert rating scale of −2 means that the subjects rated their cognitive functioning well below average. Similarly, a z-score of −2 on overall cognition represents a score well below same-age healthy peers.

To compute the metacognitive knowledge and the metacognitive experience score, delta scores were calculated by subtracting the estimated score on the Likert scale from the overall cognition score. We calculated the scores in order that increasingly positive scores reflected an underestimation of their own cognitive ability. On the other hand, a negative score reflected an overestimation of their cognitive performance. This method was based on previous research [4,27–29].

2.3. Statistical analysis

All statistical analyses were conducted using SPSS version 24 (SPSS Inc., Chicago, IL, USA). In case the variables did not meet for the criteria for parametric tests, non-parametric test were applied. Patient-control difference in age, gender, and educational level, mood, cognitive functioning and metacognition, were examined by two-tailed independent *t*-tests, Pearson chi-square test, Wilcoxon rank test, and Mann Whitney *U* test, as appropriate. The associations between metacognitive functioning and cognition were assessed by the use of Pearson correlations.

3. Results

3.1. Clinical variables

In Table 1 it is shown that patients and controls differed significantly from one another for to the scores on the HDRS and the YMRS. Based on the mean HDRS and YMRS score, the patients displayed mild depressive symptoms and no manic symptomatology. The healthy control group scored well below the cut-off for depression and mania.

3.2. Cognitive functioning

Table 2 displays the differences between patients with bipolar disorder and healthy controls in regard to neurocognitive functioning. Patients showed impaired cognitive functioning for all of the included neurocognitive tests relative to healthy controls.

3.3. Metacognitive functioning

3.3.1. Metacognitive beliefs

The metacognitive beliefs of the patients and controls are shown in Table 3. In this table, it is shown that patients have a score that is significantly higher in comparison with the controls for the total score on the MCQ-30 and the metacognitive domains of uncontrollability and danger, cognitive confidence, and the need to control thoughts.

Table 1
Mean (SD) scores of mood questionnaires in patients and healthy controls.

	Patients	Controls	Sign.
HDRS	11.72 (5.71)	1.86 (2.21)	<0.001[a]
YMRS	4.79 (4.53)	0.69 (1.44)	<0.001[a]

Note. HDRS, Hamilton Depression Rating Scale; YMRS, Young Mania Rating Scale; *p*, level of significance.
[a] Significant difference.

Table 2
Cognitive functioning in patients and controls.

	Patients	Controls	Sign.
	Mean (SD)	Mean (SD)	*p*
Speed of processing	−0.971 (0.76)	0 (1)	<0.001[a]
Attention	−0.687 (0.12)	0 (1)	=0.025[a]
Working memory	−0.652 (0.69)	0 (1)	=0.002[a]
Learning	−0.810 (1.06)	0 (1)	=0.002[a]
Executive functioning	−1.552 (1.68)	0 (1)	<0.001[a]
Social cognition	−0.9275 (1.41)	0 (1)	=0.008[a]
Overall cognition	−0.894 (0.98)	0 (1)	<0.001[a]

Note. SD, Standard deviation; *p*, level of significance.
[a] Significant difference.

3.3.2. Metacognitive knowledge and experience

Table 4 displays that patients were less accurate in regards to their metacognitive knowledge and experience, however, there was no significant difference between patients and controls. However, both groups overestimated their performance on the neurocognitive tests beforehand and afterwards. This resulted in a score on metacognitive knowledge and metacognitive experience that was significantly different from the expected score (which should have been close to 0; [4]) for the patient group ($z = -2.83$, $p = .005$; $z = -2.19$, $p = .028$, respectably) and for the control group ($z = -2.03$, $p = .042$; $z = -2.13$, $p = .032$, respectably). Metacognitive knowledge and experience did not significantly differ between the patients and the control group. In addition, metacognitive knowledge and experience did not differ from each other in the patient group, nor in the control group.

3.4. Depressive symptomatology and metacognition

For the control group, no correlations between the score on the HDRS and the metacognitive assessments were found. The correlates for the patient group are displayed in Table 5.

3.5. Neurocognitive correlates with metacognition

The significant correlates between neurocognition, the MCQ-30 and metacognitive knowledge and experience for the patient group are shown in Table 6. Importantly, all of the correlations disappeared when controlling for depression rates. For the control group, the significant correlations between neurocognitive functioning and metacognition are displayed in Table 7.

4. Discussion

The purpose of the present study was to assess if individuals with bipolar disorder show impairments in metacognitive functioning and if metacognition in these subjects was linked to neurocognition. The

Table 3
Scores on the MCQ-30 for patients and controls.

	Patients	Controls	Sign.
	Mean (SD)	Mean (SD)	*p*
Cognitive confidence	13.00 (4.58)	9.13 (2.98)	<0.001[a]
Positive beliefs about worry	9.00 (3.54)	8.75 (2.35)	=0.776
Cognitive self-consciousness	14.75 (3.47)	15.07 (3.65)	=0.828
Uncontrollability and danger	16.33 (5.39)	10.72 (3.70)	<0.001[a]
Need to control thoughts	12.48 (4.18)	9.65 (2.75)	=0.006[a]
Total MCQ-30	63.78 (11.26)	50.93 (11.14)	<0.001[a]

Note. SD, Standard deviation; *p*, level of significance; MCQ-30, Metacognitions Questionnaire short form - 30.
[a] Significant difference.

Table 4
Metacognitive knowledge and experience for patients and controls.

	Patients	Controls	Sign.
	Mean (SD)	Mean (SD)	*p*
Metacognitive knowledge	−0.788 (1.18)	−0.357 (0.87)	0.228
Metacognitive experience	−0.702 (1.47)	−0.356 (0.74)	0.309

Note. SD, Standard deviation; *p*, level of significance.

main finding is that patients display maladaptive metacognitive beliefs in comparison with healthy controls. In addition, they show impaired metacognitive knowledge and experience. However, the latter result was also true for the healthy controls. Furthermore, metacognition has neurocognitive correlates in bipolar disorder. However, depressive symptomatology seems to have a profound effect on this relationship and on metacognition in general.

4.1. Impaired metacognition in bipolar disorder and healthy controls

The current paper assessed the general metacognitive functioning of the participants through questioning their metacognitive beliefs. Although Popolo et al. [18] were unable to find impaired metacognitive beliefs in bipolar disorder patients, our results are in line with the research of Sarisoy et al. [17] were patients had more maladaptive metacognitive beliefs in comparison with the healthy control group.

In particular, patients displayed more impaired cognitive confidence, negative beliefs about uncontrollability and danger, and the need to control their thoughts. Although our patient group did not differed significantly from the healthy control group on the estimation of their cognitive performance, it does seems that they have less confidence in their cognitive performance. This outcome was also found in previous research were bipolar disorder patients experienced greater cognitive complaints in comparison with a healthy control group [30]. Although this was not the main purpose of the current investigation, we showed that bipolar disorder patients indeed score significantly worse in comparison with our healthy control group on all the cognitive domains that were assessed. Our results thus further support the assumption that impaired neurocognitive functioning is indeed an important characteristic of bipolar disorder [31–34].

Secondly, similar to the research of Sarisoy et al. [17] we found that patients had more negative feelings about uncontrollability and danger of their worrying and that they had more the need to control their thoughts. The elevations of these two sub-scales could mirror the worrying and ruminative character of (depression in) bipolar disorder [35]. In regard to this, we found that depressive symptomatology was indeed linked to most scales of the MCQ-30. This is an important finding as negative beliefs about our inner responses and thoughts are likely to feed the cycle of depression. For example, they could contribute to fearing a depressive episode and lead to a tendency to catastrophize normal deviations in mood [36].

Furthermore, the objective metacognitive functioning of the patients was assessed by the estimation of their own performance on cognitive tests before and after they completed a cognitive test battery. In this manner, metacognitive knowledge was defined as the estimation of cognitive capacities beforehand. Metacognitive experience reflected the estimation of the performance on the tests that were executed by the participants.

Both the bipolar disorder patients and the control group overestimated their cognitive abilities before the start of the tests and afterwards. It means that they showed impairment in metacognitive knowledge and experience. These results are partially in accordance with the findings of Torres et al. [4], who found that bipolar disorder patients poorly estimated their own cognitive performance prior the neurocognitive tests (impaired metacognitive knowledge). However, in the current research, additionally to an impaired metacognitive knowledge, individuals with bipolar disorder made an inaccurate estimation of their performance after the completion of the tests (they tend to overestimated their performance), resulting in an impairment of metacognitive experience. The difference between these two studies could be explained by the different mood states of the patients at inclusion. That is, in the research of Torres et al. [4] patients where euthymic, whereas the patients in the current investigation were mildly depressed. However, this does not explain the finding of the current study, that patients and controls both had the tendency to overestimate their cognitive performance. This raised the question whether a lack of metacognitive knowledge is a general trait that could possibly serves as a protection for our self-esteem [37]. Nevertheless, there is evidence for an opposite relationship because this way of reasoning could have a negative effect on daily functioning. That is, persons who tend to overestimate their abilities are more likely to engage in behavior that leads to failure, which in turn could lead to more depressive symptoms and lower self-esteem [38]. The association between impaired metacognitive functioning and decreased self-esteem is demonstrated in previous research [17]. Furthermore, the finding that patients had an inaccurate metacognitive experience could suggest that they are

Table 5
Associations between HDRS and metacognition in the patient group.

Metacognitive domain	*r* with the HDRS	*p*
Cognitive confidence	0.602	0.001[a]
Positive beliefs about worry	−0.209	0.287
Cognitive self-consciousness	−0.482	0.009[a]
Uncontrollability and danger	0.577	0.002[a]
Need to control thoughts	0.274	0.166
Total MCQ-30	0.496	0.008[a]
Metacognitive knowledge	0.218	0.284
Metacognitive experience	0.366	0.066

Note. *r*, correlation coefficient; *p*, level of significance.
[a] Significant correlation.

Table 6
Significant correlations between neurocognition and metacognition for the patient group.

Metacognitive domain	Cognitive domain	*r*	*p*
Cognitive confidence	Speed of processing	−0.474	0.011[a]
	Working memory	−0.449	0.016[a]
	Learning	−0.376	0.049[a]
	Executive functioning	−0.448	0.019[a]
	Total cognition	−0.427	0.029[a]
Need to control thoughts	Speed of processing	−0.423	0.028[a]
	Executive functioning	−0.477	0.014[a]
	Social cognition	−0.389	0.049[a]
	Total cognition	−0.417	0.038[a]
Metacognitive knowledge	Attention	0.708	<0.001[a]
	Working memory	0.391	0.048[a]
	Learning	0.633	0.001[a]
	Executive functioning	0.456	0.019[a]
	Social cognition	0.400	0.043[a]
	Total cognition	0.564	0.003[a]
Metacognitive experience	Attention	0.449	0.022[a]
	Learning	0.401	0.042[a]
	Social cognition	0.428	0.029[a]
	Total cognition	0.390	0.049[a]

Note. *r*, correlation coefficient; *p*, level of significance.
[a] Significant correlation.

Table 7
Significant correlations between neurocognition and metacognition for the control group.

Metacognitive domain	Cognitive domain	*r*	*p*
Cognitive confidence	Speed of processing	−0.373	0.046[a]
Uncontrollability and danger	Speed of processing	0.395	0.037[a]
Cognitive self-consciousness	Executive functioning	−0.397	0.033[a]

Note. *r*, correlation coefficient; *p*, level of significance.
[a] Significant correlation.

not able to alter their beliefs on ground of new information. Although they just experienced the cognitive tests, they still are not able to correctly identify their own performance on it. This observation could also be of importance to insight in bipolar disorder. That is, in the prevention of new episodes it is essential that patients learn from past episodes to guide future behavior when a new episode is lurking [39]. Following this line of reasoning, it could be that the impaired metacognitive knowledge and experience that was found in the current research finds its origin in the lack of insight individuals with bipolar disorder have in their illness (e.g. clinical insight) and in their anomalous evaluation and interpretation of events (e.g. cognitive insight). However, this assumption needs further investigation [14].

The finding that patients show more cognitive complaints in comparison with our healthy control group on the MCQ-30, but did not differ from the control group in metacognitive knowledge nor experience might feel as somewhat contradictory. A possible explanation for this might be that the MCQ-30 specifically asked about impairments in memory, whereas for metacognitive knowledge and experience the patients were asked to rate their general cognitive functioning.

4.2. Neurocognitive correlates with metacognitive functioning

As expected, metacognitive functioning was linked to neurocognition in bipolar disorder. More specifically, it was found that better metacognitive functioning is associated with higher scores on the neurocognitive domains of speed of processing, working memory, learning, executive functioning and social cognition. The link between executive functioning and metacognition was already described in bipolar disorder [21] and is not surprising as executive functioning is thought of as processes that are involved in regulating and control of complex cognitive operations such as the adaptation of plans in view of new information and solving unexpected problems, all processes that are needed for an effective metacognitive functioning [40]. The only study that has assessed the association between cognition and metacognition in bipolar disorder also found that memory is linked to metacognitive functioning [21]. Furthermore, the current research describes an association with learning. Taken together, it could be that impairments in remembering and integrating life events might cause problems in metacognitive functioning, as this requires the ability to think about our thinking. In addition, we found that impaired processing speed is correlated with impairments in metacognitive beliefs, more specifically the need to control thoughts and worsted cognitive confidence. Although it is out of the scoop of the current paper, it could be that this relationship further complicates the construction of a complex idea about ourselves and others [41]. The formation of an impression about our own mental states and about the mental states of individuals who are surrounding us [42], requires adequate social cognition as this involves the interpretation of thoughts and emotions of ourselves and others [43]. As such, it may come as no surprise that the current research showed a link between metacognitive beliefs, -knowledge, -experience and social cognition. Although the latter assumption needs further investigation by the use of more specialized metacognitive assessments such as the Metacognition Assessment Scale (MAS-A) [18,21], our results provide further support for a cognitive base of metacognitive difficulties in severe psychiatric disorders, such as bipolar disorder.

4.3. Depressive symptomatology and metacognitive functioning

As mentioned in the introduction of the current paper, metacognition shares similarities with excessively worrying or rumination [12], which could promote depressive symptomatology [13,14]. As expected, we did find that more depressive feelings are associated with maladaptive metacognition. More specifically, depressive symptomatology was associated with a loss of confidence in own cognitive performance, the awareness of extensively ruminating and experiencing less ownership of worrying. Moreover, depression rates seem to have a large impact on the association between cognition and metacognition. Even to that extend that all the correlations disappeared when controlling for depressive symptomatology.

Our results thus further add to the evidence that more maladaptive metacognitive skills are associated with depression, which is often discussed in research regarding major depressive disorder [44]. In addition, the results of the current paper are in line with previous studies of our research group in bipolar disorder that identified a link between more depressive feelings and higher self-reflectiveness about own thinking, feelings and experiences [45]. Future studies in euthymic patients and longitudinal studies in patients in different mood states should be conducted to further clarify the relationship between metacognition and depression.

4.4. Clinical implementations

With this knowledge in mind, it seems that maladaptive metacognitive thinking should be prevented as this way of thinking can lead to negative consequences such as withdrawal from pleasant activities and moreover, it can lead to distorted interpretations of life events [46,47]. Hence, trainings are being developed that promote a healthy metacognitive thinking, by focusing on the reduction of unhelpful cognitive processes. For instance, they help the patient to interrupt rumination and in the reduction of unhelpful self-monitoring tendencies [39]. Although there are metacognitive trainings for depression [48], anxiety disorders [49], and schizophrenia [50], to date, there is no training that focuses on the promotion of a healthy metacognitive functioning in bipolar disorder.

One manner in which we can facilitate an improvement in metacognitive functioning is through directly tackle the difficulties by the implementation of a metacognitive training [50] such as described above. However, since cognitive problems are well known in bipolar disorder [31–34] and because they seem to be associated with poor metacognitive functioning, the implementation of cognitive remediation therapy could additionally have a positive effect on metacognition. That being said, there is increasing evidence that cognitive remediation therapy is more effective for functional outcome when there are elements included that cause a transfer to real life skills [51]. Recently, it was argued that such therapies would achieve even more of a transfer to everyday life [40].

Taken together, it seems that effective therapy in bipolar disorder should include various elements, from basic cognitive- to higher order metacognitive training. In this manner a more healthy form of metacognition could be generated, which could lead to less depressive symptomatology and better functional outcome in bipolar disorder.

This study has several strengths such as the inclusion of the ISBD-BANC for the assessment of neurocognition, the assessment of mood in the participants and various metacognitive assessments. However the latter strength can be seen as a limitation. That is, the concept of metacognitive knowledge that is taken here as a concept of metacognition, could also be seen as a form of clinical or cognitive insight which was also addressed in our discussion. However, it could also be seen of as a neurocognitive insight, which further complicates the interpretation of the current research. On the other hand investigations as the current show the complexity of metacognition and makes room for further important research regarding this topic. Another limitation is that we did not assess the effect of other comorbid symptoms such as anxiety, which too could have an effect on metacognitive functioning and neurocognitive functioning. In addition, we made use of categorical scales for the assessment of metacognitive beliefs, metacognitive experience and metacognitive knowledge. This could have caused a less precise measure of metacognition. Therefore, it could be helpful if future research includes continuous scales such as a visual analog scale. Lastly, because of the relative small sample size, the clinical implementations of the current paper should be seen as preliminary.

Declaration of Competing Interest

None.

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