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Rania HALHEIT¹ *

Laboratory of Psychological and Educational Applications, Constantine 2

University 20 August 1955, Skikda, (Algeria)

Email : r.halheit@univ-skikda.dz

Zoubir BOUSSAFSAF²

Larbi Ben M'hidi University – Oum El Bouaghi – (Algeria)

Email : boussafsafzoubir@gmail.com



Abstract :

Cognitive impairments are among the main clinical features of bipolar disorder, particularly affecting executive functioning, attention, and memory. This theoretical paper, grounded in an interpretative neuropsychological perspective, examines the nature of these cognitive dysfunctions and the principal dimensions underlying their manifestation. It also reviews the major neurobiological, clinical, and contextual factors influencing cognitive performance and their impact on daily functioning.

The analysis suggests that cognitive impairments in bipolar disorder are heterogeneous and multidimensional. A better understanding of these deficits and the mechanisms contributing to their expression may support the development of integrated and individualized therapeutic strategies aimed at improving functional outcomes and overall quality of life.

keywords: Cognitive Impairments; Bipolar Disorder; Executive functioning; Attention; Memory.

الملخص :

تعد الاضطرابات المعرفية من أبرز السمات السريرية المصاحبة لاضطراب ثنائي القطب، حيث تؤثر بصورة خاصة في الوظائف التنفيذية والانتباه والذاكرة. وتهدف هذه الورقة النظرية، المستندة إلى منظور عصبي نفسي تفسيري، إلى دراسة طبيعة هذه الاختلالات المعرفية والأبعاد الأساسية الكامنة وراء ظهورها. كما تستعرض أهم العوامل العصبية البيولوجية والسريرية والبيئية المؤثرة في الأداء المعرفي وانعكاساته على الحياة اليومية.

وتشير هذه الورقة إلى أن الضعف المعرفي في اضطراب ثنائي القطب يتسم بالتعقيد والتعدد في مظاهره وأبعاده. ومن شأن الفهم الأعمق لهذه الاضطرابات والآليات المرتبطة بها أن يسهم في تطوير استراتيجيات علاجية متكاملة وفردية، بما يساعد على تحسين الأداء الوظيفي وجودة الحياة لدى المرضى.

الكلمات المفتاحية: الاضطرابات المعرفية؛ اضطراب ثنائي القطب؛ الوظائف التنفيذية؛ الانتباه؛ الذاكرة.

1. Introduction

Bipolar disorder is a chronic and recurrent psychiatric condition characterized by alternating episodes of mania and depression, interspersed with periods of euthymia or relative mood stability. The disorder typically emerges during adolescence or early adulthood (Grande et al., 2016; Price & Marzani-Nissen, 2012; Calesella, 2024).

According to the DSM-5, bipolar disorder comprises several diagnostic subtypes, including Bipolar I Disorder, Bipolar II Disorder, and Cyclothymic Disorder. Bipolar I Disorder is defined by the presence of at least one manic episode, whereas Bipolar II Disorder is characterized by recurrent major depressive episodes and hypomanic episodes. Cyclothymic Disorder involves chronic fluctuations between hypomanic and depressive symptoms that do not meet the full criteria for major mood episodes and persist for at least two years.

Beyond affective symptomatology, increasing attention has been directed toward the cognitive dimensions of bipolar disorder. In this context, growing evidence suggests that cognitive impairments constitute a central feature of the disorder. Masson et al. (2023) emphasize that patients exhibit significant deficits in attention, memory, and executive functioning, with varying levels of severity across different stages of the illness. Despite the increasing recognition of these disturbances, their underlying mechanisms remain only partially understood. Current evidence indicates that cognitive performance is shaped by a complex interaction of factors, including neurobiological alterations (Lopes & Fernandes, 2012), psychiatric comorbidities (Fountoulakis, 2014), and the effects of psychotropic medications (Camelo et al., 2013). Nevertheless, the precise contribution of these factors to cognitive dysfunction requires further investigation.

Given that cognitive impairments exert a direct and substantial impact on functional outcomes and quality of life (Baune & Malhi, 2015), while also limiting patients' ability to adapt effectively to the demands of everyday life (Brissos et al., 2008), understanding their nature and underlying mechanisms represents a critical step toward improving therapeutic interventions and developing effective cognitive rehabilitation programs.

Although international research on cognitive impairment in bipolar disorder has expanded considerably, studies conducted within Arab contexts,

particularly in Algeria, remain relatively scarce. Nevertheless, several national contributions provide valuable insights into the cognitive and biological dimensions of the disorder. Bensaida et al. (2015), for example, examined executive functioning in patients with bipolar disorder with and without comorbid attention-deficit/hyperactivity disorder (ADHD). Their findings demonstrated that individuals with comorbid bipolar disorder and ADHD exhibited more severe executive dysfunction than those with bipolar disorder alone, particularly in cognitive inhibition, mental flexibility, planning, and attention. Additionally, Dahdouh et al. (2014) reported evidence of genetic overlap across psychiatric disorders, suggesting that common genetic variants may confer vulnerability to multiple conditions. Complementing these findings, Dahdouh et al. (2013) highlighted the role of familial relatedness in schizophrenia and bipolar disorder, confirming family history as a significant risk factor for psychotic disorders.

Building on this body of evidence, the present theoretical paper aims to enhance understanding of cognitive impairments associated with bipolar disorder through a critical and interpretative review of the scientific literature. Specifically, it seeks to clarify the nature of these impairments, examine the factors influencing cognitive performance, analyze their impact on functional performance and quality of life, and discuss available therapeutic approaches and factors affecting their effectiveness. By integrating cognitive, clinical, and functional dimensions within a unified interpretative framework, this paper underscores the dynamic and heterogeneous nature of cognitive functioning in bipolar disorder and identifies directions for future research.

2. The Nature of Cognitive Impairments in Bipolar Disorder

Accumulating evidence indicates that cognitive disturbances in bipolar disorder are neither secondary nor incidental phenomena but may instead constitute an integral component of the disorder's clinical structure. Importantly, these impairments persist even during periods of euthymia, suggesting that they are linked to underlying neurobiological mechanisms rather than being solely mood-dependent. In particular, disturbances within fronto-limbic circuits, which play a crucial role in emotional regulation, and dysfunction of the ventral prefrontal cortex have been implicated, reinforcing the view that cognitive impairment is not merely a consequence of affective symptoms (Besche-

Richard, 2018). These observations support the growing conceptualization of cognitive dysfunction as a core dimension of bipolar disorder, extending beyond the traditional focus on affective symptomatology.

Nevertheless, cognitive impairments in bipolar disorder are not entirely stable in nature. Rather, they exhibit a dynamic profile, with the degree and pattern of dysfunction varying across cognitive domains. Certain deficits may be present in some functions but not others, depending on their sensitivity to mood fluctuations and the specific neurobiological substrates involved (wang et al, 2026; xu et al,2020) . For example, memory functioning is influenced by mood state. During manic episodes, patients tend to preferentially encode or recall positively valenced information, whereas during depressive episodes, memory is biased toward negative content. A similar pattern has been observed in emotion recognition, whereby individuals in depressive states tend to interpret facial expressions negatively, while those experiencing mania are more inclined toward positive interpretations. These findings indicate that although a core level of cognitive dysfunction may persist, specific cognitive operations remain modulated by affective state(Bigot,2021; Besche-Richard,2018).

Furthermore, the literature points to the existence of distinct cognitive subtypes within the bipolar population. While some individuals demonstrate relatively preserved cognitive functioning, others exhibit marked and widespread deficits across multiple domains. This heterogeneity may account for the substantial variability observed across empirical findings and further supports the notion that cognitive impairment in bipolar disorder is non-uniform and dynamically modulated (Besche-Richard, 2018).

In this regard, a systematic review conducted in the United Kingdom by Cullen et al. (2016), encompassing 30 studies of euthymic adults aged 18 to 70 years, reported substantial variability in the prevalence of cognitive impairment in bipolar disorder. Domain-specific prevalence rates ranged from 5.3% to 57.7% for executive dysfunction, 9.6% to 51.9% for attention and working memory deficits, 23.3% to 44.2% for impairments in processing speed and reaction time, 8.2% to 42.1% for verbal memory deficits, and 11.5% to 32.9% for visual memory impairments.

These findings indicate that executive functions, attention, and working memory are among the most frequently affected domains, whereas visual memory appears relatively less impaired. The considerable variability in

prevalence rates is largely attributable to methodological differences across studies, particularly the sensitivity and diversity of cognitive assessment tools employed, a limitation explicitly acknowledged by the authors. This interpretation is consistent with the heterogeneity emphasized by Besche-Richard (2018), suggesting that variability in findings reflects both genuine clinical heterogeneity and methodological constraints.

Cullen et al. (2016) further demonstrated that tasks requiring rapid performance under strict time constraints tend to yield higher impairment rates than non-timed tasks. Similarly, cognitive deficits are more pronounced in complex measures that integrate multiple cognitive processes than in simple single-domain assessments. These results underscore the influence of assessment methodology on the detection and characterization of cognitive impairment. They also highlight the importance of using sensitive and ecologically valid neuropsychological measures when evaluating cognitive functioning in bipolar disorder.

Overall, the findings suggest that cognitive impairment affects a substantial proportion of individuals with bipolar disorder and that its severity is closely linked to illness history. Longer illness duration, a greater number of manic episodes, and increased frequency of hospitalizations were consistently associated with poorer cognitive outcomes. The use of antipsychotic medications was also associated with greater cognitive impairment, although this relationship may partly reflect underlying illness severity, given that such medications are more commonly prescribed in more severe clinical presentations. Collectively, these findings support a multifactorial model in which cognitive impairment emerges from the interaction of clinical course, neurobiological vulnerability, and treatment exposure.

The impact of mood episodes on cognitive performance further varies according to episode type. Manic episodes are typically associated with accelerated thought processes and impaired attention, whereas depressive episodes are characterized by psychomotor slowing and reduced verbal fluency. Importantly, deficits in attention and executive functioning often persist during euthymic periods, reflecting an underlying neurobiological vulnerability and possible genetic predisposition. Recurrent mood episodes may exert cumulative effects, commonly described as neurocognitive scarring, with residual subthreshold symptoms persisting in some patients. These enduring impairments negatively affect daily functioning and increase vulnerability to relapse

(Bourgeois et al., 2014). This reinforces the view that cognitive dysfunction represents a stable and clinically meaningful dimension of bipolar disorder rather than a transient epiphenomenon of mood episodes.

Based on the synthesis provided by Masson et al. (2023), the cognitive domains most affected according to mood state can be summarized as follows:

During depressive episodes: impairments in memory, working memory, attention, verbal fluency, and executive functions.

During manic, hypomanic, and mixed episodes: deficits in learning and verbal memory, divided attention, social cognition, language, and executive functions.

During euthymic phases: persistent impairments in verbal memory, attention, processing speed, executive functioning, and decision-making abilities.

Table 1. Cognitive Profiles

Cognitive Profile	Percentage	Result
Global Cognitive Impairment	36.7%	Presence of severe and generalized impairment across most cognitive processes, as indicated by nearly all neuropsychological measures used in the study.
Selective Cognitive Impairment	30%	Cognitive deficits restricted to specific domains rather than across all functions, particularly verbal memory.
Preserved Cognitive Functioning	33.3%	No significant differences compared to healthy controls across any cognitive measure.

Source: Adapted from Russo et al. (2017)

Table 1 summarizes the findings of the cross-sectional neurocognitive study conducted by Russo et al. (2017), which aimed to investigate cognitive variability among individuals with bipolar disorder and to examine the association between cognitive subtypes and genetic vulnerability. To this end,

patients' cognitive performance was compared with that of their unaffected first-degree relatives (siblings) and healthy control participants.

The total sample consisted of 180 participants divided into three groups: 60 euthymic patients diagnosed with bipolar disorder, 49 unaffected siblings, and 71 healthy controls. Hierarchical cluster analysis identified three distinct cognitive profiles among the patient group. The results indicated that only the unaffected siblings of patients belonging to the Global Cognitive Impairment cluster exhibited cognitive difficulties comparable to those observed in patients, particularly in verbal memory and executive functioning, when contrasted with healthy controls. These findings suggest that global cognitive impairment may constitute a potential endophenotype, reflecting an underlying genetic vulnerability to bipolar disorder. This interpretation is consistent with broader evidence proposing that certain cognitive deficits may represent trait-like markers of vulnerability rather than mere consequences of illness expression.

This classification further highlights the heterogeneity of cognitive functioning in bipolar disorder, as the severity of impairment varied markedly across the identified cognitive profiles. Patients in the global cognitive impairment cluster exhibited the most pronounced deficits, whereas those in the preserved cognitive functioning cluster demonstrated relatively intact cognitive abilities. Clinically, these findings are significant, as they suggest that a subset of patients maintains relatively preserved cognitive functioning. This observation indicates that cognitive performance in bipolar disorder may not be solely determined by diagnostic status but could also be influenced by individual, biological, and contextual factors. More broadly, these findings emphasize the importance of individualized cognitive assessment and further support the view that cognitive functioning in bipolar disorder is characterized by substantial variability across patients.

2.1 Interpreting Cognitive Variability: Risk and Protective Factors

Cognitive variability in bipolar disorder can be understood as the result of a complex interaction between risk and protective factors. Among the most salient risk factors is the recurrence of mood episodes. The literature consistently demonstrates that the number of episodes, particularly manic episodes, is closely associated with the severity of cognitive decline (Xu et al., 2020). Repeated mood episodes may exert neurotoxic effects on the brain,

leading to impairments in executive functioning and memory (Lance, 2011). These findings collectively suggest a cumulative burden model in which repeated affective instability progressively compromises cognitive efficiency over time.

Recurrent hospitalizations represent an additional significant risk factor and have been associated with an increased likelihood of developing dementia. It has been estimated that each severe episode requiring hospitalization increases the subsequent risk of dementia by approximately 6%. Moreover, repeated episodes heighten the brain's vulnerability to relapse and contribute to what is commonly described as cognitive scarring, characterized by the persistence of residual cognitive deficits between mood episodes. This pattern further reinforces the idea that bipolar disorder may involve progressive neurocognitive deterioration rather than purely episodic dysfunction.(Bigot, 2021.; Forzan, 2014; Grande et al., 2016; Gadenne, 2018 ;Tursini, 2024)

Illness duration also constitutes a critical determinant of cognitive outcome, as impairments tend to worsen with disease progression (Montel, 2014). This observation supports the notion that bipolar disorder follows a progressive course rather than a static one. Within the framework of the staging model, cognitive deficits are thought to become more stable and pronounced in later, chronic stages of the disorder compared to earlier phases, suggesting a trajectory-dependent pattern of cognitive decline (Grande et al., 2016).

Symptom severity further plays a pivotal role in shaping cognitive outcomes. Mood episodes accompanied by psychotic features are associated with more severe and enduring cognitive impairments than non-psychotic episodes. Likewise, mixed states, defined by the simultaneous presence of manic and depressive symptoms, are linked to greater cognitive and functional disturbances (Bourgeois et al., 2014). Collectively, these factors contribute to substantial executive dysfunction, ultimately reducing patients' capacity for planning, self-regulation, and effective engagement in therapeutic interventions. This highlights how clinical phenomenology directly translates into measurable cognitive and functional consequences.

In contrast, protective factors, most notably cognitive reserve, are central to explaining interindividual differences in cognitive performance. Cognitive reserve refers to the brain's ability to sustain relatively preserved functioning despite the presence of neurological or psychiatric pathology, including bipolar disorder. It is commonly estimated using proxy indicators such as educational attainment and occupational complexity (Matsumoto & Hamatani, 2024). Higher levels of cognitive reserve may therefore play a crucial role in maintaining cognitive functioning and enhancing responsiveness to cognitive rehabilitation interventions.

The dynamic interplay between risk and protective factors accounts for the marked heterogeneity observed in cognitive outcomes among individuals with bipolar disorder and underscores the importance of individualized therapeutic approaches that are tailored to each patient's cognitive profile and clinical trajectory.

2.2 Cognitive Impairments as Endophenotypes

Montel et al. (2014) proposed that certain cognitive deficits associated with bipolar disorder may have a genetic basis, a hypothesis supported by their presence among patients' unaffected relatives. Several studies have reinforced this perspective by documenting attentional impairments in first-degree relatives (Camelo et al., 2013), as well as deficits in verbal memory and response inhibition (Fountoulakis, 2014). Collectively, these findings suggest that cognitive dysfunction may extend beyond the clinical manifestation of bipolar disorder and reflect an underlying endophenotype associated with genetic vulnerability. Consequently, cognitive impairment may represent a trait-like marker of susceptibility to the disorder rather than merely a consequence of illness expression.

To characterize cognitive deficits as endophenotypes, Montel et al. (2014) outlined four key criteria:

1. Heritability: cognitive deficits should have a genetic basis and be observable among relatives.

2. Association with the disorder: deficits should represent core clinical features of bipolar disorder.
3. State independence: impairments should be present irrespective of mood state or episode.
4. Presence in genetically at-risk relatives: deficits should be detectable even in the absence of a formal clinical diagnosis.

Collectively, these criteria support the conceptualization of endophenotypes as psychoneurobiological markers in genetic research and reinforce the hypothesis that certain cognitive impairments may precede the emergence of overt clinical symptoms, representing an early stage in the course of bipolar disorder. From a broader neuropsychological perspective, this framework also highlights the importance of considering cognitive dysfunction as a potential bridge between genetic vulnerability and clinical manifestation.

2.3 Differentiation from Other Psychiatric Disorders and Functional Impact

Bourgeois et al. (2014) observe that certain cognitive impairments, particularly executive dysfunction, are also present in schizophrenia. However, schizophrenia is generally associated with more severe and pervasive cognitive deficits, alongside more extensive neurobiological abnormalities. In contrast, cognitive impairment in bipolar disorder demonstrates a degree of relative specificity, as it is more strongly modulated by mood state and characterized by a pronounced interaction between cognitive and emotional processing. These differences suggest that, although there is overlap across psychiatric conditions, the pattern and modulation of cognitive deficits may vary significantly depending on diagnostic context.

These observations indicate that cognitive deficits cannot be considered exclusive diagnostic markers of bipolar disorder and should not be used in isolation to differentiate it from other psychiatric conditions. Moreover, psychiatric comorbidities, such as anxiety disorders and substance use disorders, may further exacerbate impairments in executive functioning and memory, thereby accelerating cognitive decline. Taken together, these findings highlight

that cognitive dysfunction in bipolar disorder is multifactorial in origin and context-dependent rather than diagnosis-specific.

Empirical evidence suggests that functional outcomes and quality of life in individuals with bipolar disorder are not determined solely by illness severity or diagnostic subtype but are substantially influenced by associated cognitive impairments. Numerous studies have demonstrated robust associations between deficits in processing speed, verbal memory, and executive functioning and poorer occupational and social outcomes, even during periods of mood stability (Wingo et al., 2009; Bourgeois et al., 2014; Özbek & Bora, 2025). This pattern underscores the clinical relevance of cognitive dysfunction as a key determinant of real-world functioning beyond symptom remission.

These findings support the notion that clinical remission does not necessarily equate to functional recovery. While some patients maintain satisfactory levels of functioning despite persistent symptoms, others experience marked functional decline despite achieving clinical stability. This heterogeneity underscores that functional performance emerges from a dynamic interplay between clinical status, cognitive impairments, and protective factors such as cognitive reserve and neural plasticity.

2.4 Therapeutic Interventions Targeting Cognitive Impairments

Among the most prominent therapeutic approaches targeting cognitive impairments in bipolar disorder are cognitive rehabilitation and cognitive remediation therapy. These psychological interventions aim to improve compromised cognitive abilities and enhance functional performance in everyday life, particularly in domains such as attention, memory, and executive functioning.

Although several studies have reported promising outcomes in terms of cognitive improvement and psychosocial functioning, the empirical evidence supporting the effectiveness of these interventions in patients with bipolar disorder remains limited and inconclusive (Kluwe-Schiavon et al., 2015; Strawbridge et al., 2021; Bourgeois et al., 2014). This inconsistency across findings may be partly explained by methodological heterogeneity, variability in

patient clinical profiles, and differences in intervention protocols, which collectively limit the comparability of results across studies.

Nevertheless, the adoption of integrative therapeutic approaches that combine these core interventions with complementary techniques, such as neurofeedback, is increasingly recommended. From a clinical neuropsychological perspective, such multimodal strategies may offer added value by addressing both cognitive dysfunction and underlying neural inefficiencies simultaneously. Accordingly, they may contribute to more comprehensive and potentially more effective treatment models by accounting for the cognitive and neurobiological complexity of bipolar disorder.

3. Factors Influencing Cognitive Performance in Bipolar Disorder

Cognitive variability among individuals with bipolar disorder cannot be fully understood without considering the complex interplay between neurobiological, clinical, and pharmacological factors, alongside protective mechanisms that shape individual cognitive trajectories. This interaction is commonly conceptualized within the framework of the neuroprogression hypothesis, which posits that recurrent manic episodes exert cumulative neurotoxic effects, leading to progressive neural alterations that compromise brain efficiency and result in worsening cognitive performance over time (Wingo et al., 2009).

In support of this hypothesis, Keramatian et al. (2021) demonstrated that patients with bipolar I disorder, characterized by the occurrence of full manic episodes, exhibit more pronounced impairments in verbal memory than individuals with bipolar II disorder. Nevertheless, the predominance of cross-sectional designs in this body of research represents a major methodological limitation, as it prevents definitive conclusions regarding whether cognitive impairments reflect pre-existing neurodevelopmental vulnerabilities or progressively worsen as the clinical course unfolds (Martínez-Arán et al., 2004). This methodological constraint is repeatedly echoed across the literature, reinforcing the need for longitudinal evidence.

At the neurobiological level, the literature consistently reports structural and functional brain abnormalities in bipolar disorder, particularly involving the prefrontal cortex, a region critically implicated in attention, executive functioning, and emotional regulation. Numerous studies have identified frontal lobe dysfunction, especially within prefrontal regions, as a key neural substrate underlying impairments in planning, response inhibition, and affective control (Boland & Alloy, 2013; Martínez-Arán et al., 2004).

Functional neuroimaging studies using functional magnetic resonance imaging (fMRI) and positron emission tomography (PET) have further revealed widespread functional abnormalities, including hypoactivation of the dorsolateral prefrontal cortex alongside hyperactivation of limbic structures such as the amygdala. These alterations have been linked to deficits in attentional control and impaired emotion regulation, reflecting disrupted fronto-limbic connectivity (Camelo et al., 2013).

Such dysfunctions may arise from abnormalities in neuronal and glial cell density, as well as from altered neural activation patterns during cognitively demanding tasks. Reduced engagement of frontal regions during working memory and sustained attention paradigms has been repeatedly documented in patients with bipolar disorder (Kessing & Miskowiak, 2018). In contrast, memory impairments, particularly in verbal and episodic memory, are thought to reflect dysfunction in medial temporal structures, including the hippocampus and middle temporal gyrus, which play a central role in learning, encoding, and retrieval processes (Huang et al., 2023).

Importantly, these neurobiological alterations appear to be associated with illness progression. Several studies have reported a relationship between longer illness duration and greater degrees of frontal cortical atrophy (Camelo et al., 2013). In other words, prolonged exposure to recurrent mood episodes, especially manic episodes, may lead to increasingly pronounced structural brain changes, lending support to the progressive deterioration hypothesis and reinforcing the notion of bipolar disorder as a neuroprogressive condition (Wingo et al., 2009).

However, the widespread reliance on cross-sectional methodologies complicates efforts to establish causal relationships between neurobiological

changes and cognitive impairment. It remains difficult to determine whether observed alterations result from the natural progression of the disorder, premorbid vulnerabilities, or confounding factors such as medication exposure. Indeed, some studies have failed to identify clear differences in white matter integrity between patients with varying cognitive profiles (Burdick et al., 2010), thereby limiting the extent to which specific cognitive deficits can be attributed to discrete structural abnormalities. These inconsistencies underscore the need for well-designed longitudinal studies capable of tracking neurobiological and cognitive trajectories over time (Martínez-Arán et al., 2004).

Beyond neurobiological mechanisms, comorbid conditions play a substantial role in shaping cognitive outcomes in bipolar disorder. Among the most frequently implicated are substance use disorders, particularly alcohol misuse, which has been associated with more severe impairments in executive functioning and memory compared to patients without such comorbidities. These effects may reflect either the direct neurotoxic impact of alcohol or the additive burden imposed by multiple pathological processes. Nonetheless, small sample sizes and the inclusion of patients with active mood symptoms in some studies limit the generalizability of these findings, highlighting the need for larger and methodologically rigorous investigations (Levy et al., 2008).

Sleep disturbances also represent an important and often underappreciated contributor to cognitive dysfunction. Sleep problems occur across all phases of bipolar disorder, preceding, accompanying, and following mood episodes, making them a relatively stable feature of the illness trajectory. The relationship between sleep disturbances and cognitive impairment is likely bidirectional, forming a vicious cycle: disrupted sleep impairs attention, executive control, and self-regulation, while executive dysfunction in turn compromises sleep organization. This interaction is largely mediated by the prefrontal cortex, which plays a critical role in both cognitive control and the regulation of sleep-wake cycles (Boland & Alloy, 2013).

Pharmacological factors further complicate the cognitive profile of bipolar disorder. Psychotropic medications exert heterogeneous and sometimes opposing effects on cognition. Some evidence suggests that antipsychotic treatment may be associated with poorer verbal memory performance, while

valproate use has been linked to less favorable cognitive outcomes compared to lithium (Özbek & Bora, 2025). Findings regarding lithium remain inconclusive: although some studies report mild memory impairments, others indicate preserved or even relatively enhanced cognitive functioning (Keramatian et al., 2021). These discrepancies likely reflect differences in illness severity, treatment duration, and clinical course rather than direct pharmacological effects per se. Moreover, the frequent use of polypharmacy in most clinical samples limits causal inference, underscoring the need for longitudinal studies involving early-stage or newly diagnosed patients (Burdick et al., 2010; Martínez-Arán et al., 2004).

In contrast to these risk factors, protective mechanisms, most notably cognitive reserve, play a crucial role in explaining interindividual differences in cognitive performance. Cognitive reserve refers to the brain's capacity to compensate for structural and functional damage by recruiting alternative neural strategies, thereby preserving cognitive functioning despite pathology. Higher levels of cognitive reserve have been consistently associated with better cognitive outcomes in bipolar disorder (Matsumoto & Hamatani, 2024; Özbek & Bora, 2025). However, the absence of standardized measures and the reliance on proxy indicators, coupled with predominantly cross-sectional study designs, represent important methodological limitations. These challenges highlight the need to harmonize assessment tools and develop more precise theoretical models to better elucidate the role of cognitive reserve in bipolar disorder (Matsumoto & Hamatani, 2024).

Conclusion

Bipolar disorder is consistently associated with deficits in executive functions, attention, and memory across different phases of the illness. Importantly, patients do not constitute a cognitively homogeneous group; rather, distinct cognitive subgroups can be identified, differing in both the extent and severity of impairment. This variability reflects the influence of multiple interacting factors, including illness severity, recurrence of mood episodes, neurobiological alterations, and levels of cognitive reserve. Together, these factors account for the marked heterogeneity observed in functional outcomes and quality of life among individuals with bipolar disorder.

Despite the growing body of research in this area, the precise mechanisms underlying cognitive dysfunction remain incompletely understood. Likewise, definitive cognitive markers among first-degree relatives have yet to be clearly established, with the exception of relatively consistent findings related to impairments in verbal learning and working memory. The effects of psychotropic medications, the widespread use of polypharmacy, and the protective role of cognitive reserve continue to require further systematic investigation to clarify their contribution to cognitive trajectories over the course of the illness. Within this context, integrating cognitive assessment into routine clinical evaluation and therapeutic planning is essential, as it represents a crucial step toward promoting functional recovery and improving overall quality of life.

The present review is subject to several limitations, including its theoretical nature, the predominant reliance on non-Arabic literature, and the limited depth devoted to the concept of cognitive reserve, despite its recognized clinical relevance. In addition, emerging therapeutic approaches, such as neurofeedback, were addressed only briefly. Nevertheless, emphasizing the cognitive dimension constitutes a necessary shift from an exclusive focus on mood symptom control toward a more comprehensive model that prioritizes functional performance and long-term recovery.

Accordingly, there is a clear need for longitudinal and experimental studies to achieve a more precise understanding of the trajectories of cognitive decline in bipolar disorder. Expanding research within Arab contexts is

particularly warranted to address existing gaps in the literature. Future investigations should also place greater emphasis on elucidating the mechanisms of cognitive reserve as a source of interindividual variability, as well as on evaluating neurofeedback as a promising adjunctive intervention alongside standard treatments to enhance cognitive functioning and support sustained functional recovery.

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