Fırat Tıp Dergisi / Firat Med J 2026; 31(1): 59-65

**RESEARCH ARTICLE**

# Distinct Verbal Memory Patterns Distinguish Bipolar Depression From Unipolar Depression in Terms of Neurocognitive Profile

## Farklı Sözel Bellek Örüntüleri, Nörobilişsel Profili Açısından Bipolar Depresyonu Unipolar Depresyondan Ayırır

Elvan ÇİFTÇİ[1]

[1]Uskudar University NP Brain Hospital, Psychiatry, Istanbul, Turkey

E.C. 0002-7452-2616.

Corresponding Author: Elvan ÇİFTÇİ (elvanlciftci@gmail.com

**ABSTRACT**

**Objective:** Neurocognitive impairments are common in both unipolar depression (UDp) and bipolar depressive disorder (BDp), yet the extent and nature of these deficits remain poorly understood. This study aimed to compare cognitive profiles in BD and UD during depressive episodes using comprehensive neuropsychological testing.

**Material and Method:** We retrospectively enrolled seventy-eight patients from NP Brain Hospital who had been diagnosed with DSM-5 UDp (n =39) and BDp (n =39) according to a validated assessment. Participants underwent a battery of neuropsychological tests assessing five cognitive domains: working memory, executive functioning, visual memory, verbal memory, and face recognition. Tests included the Wechsler Memory Scale (WMS), the Verbal Memory Processes Test (VMPT), and the Stroop Color and Word Test (SCWT).

**Results:** Despite overlapping neurocognitive impairments, BDp and UDp exhibit distinct profiles, BDp patients demonstrated greater impairment in VMPT learning, criteria, maximum recall, and delayed recall ($p < 0.01$), highlighting more severe verbal memory dysfunction than UDp using the Mann-Whitney U test. No significant differences were found in working memory, executive functioning, face-image recognition or non-verbal memory between groups.

**Conclusion:** Specific cognitive profiles deficits such as verbal memory impairments provide valuable targets for early interventions between UDp and BDp. This study reinforces the critical role of neuropsychological testing in the early diagnosis and management of mood disorders. Future research should focus on addressing methodological limitations, integrating multimodal approaches, and tailoring treatments to specific neurocognitive profiles to improve patient outcomes.

**Keywords:** Bipolar disorder, unipolar depression, cognitive impairment, executive function, verbal memory, neuropsychological testing.

**ÖZET**

**Amaç:** Nörokognitif bozukluklar hem unipolar depresyonda (UDp) hem de bipolar depresif bozuklukta (BDp) yaygındır, ancak bu eksikliklerin kapsamı ve doğası tam olarak anlaşılamamıştır. Bu çalışma, kapsamlı nöropsikolojik testler kullanarak depresif dönemler sırasında BDp ve UDp'deki bilişsel profilleri karşılaştırmayı amaçladı.

**Gereç ve Yöntem:** DSM-5'e göre doğrulanmış bir değerlendirmeye göre, UDp (n =39) ve BDp (n =39) tanısı almış NP Beyin Hastanesi'nden yetmiş sekiz hastayı retrospektif olarak çalışmaya dahil ettik. Katılımcılara beş bilişsel alanı değerlendiren bir dizi nöropsikolojik test uygulandı: çalışma belleği, yürütücü işlevler, görsel bellek, sözel bellek ve yüz tanıma. Testler arasında Wechsler Bellek Ölçeği (WMS), Sözel Bellek İşlemleri Testi (VMPT) ve Stroop Renk ve Kelime Testi (SCWT) yer aldı. İstatistiksel karşılaştırmalar yapıldı.

**Bulgular:** Mann-Whitney U testi kullanılarak yapılan analize, BDp ve UDp örtüşen nörokognitif bozukluklara rağmen, BDp hastaları UDp hastalarına göre VMPT öğrenmesinde, kriterlerde, maksimum hatırlamada ve gecikmiş hatırlamada daha fazla bozulma gösterdi ($p < 0.01$), bu da UDp'den daha ciddi sözel bellek bozukluğunu vurguladı. Gruplar arasında çalışma belleği, yürütücü işlevler, yüz görüntüsü tanıma veya sözel olmayan bellek açısından anlamlı bir fark bulunmadı.

**Sonuç:** Sözel bellek bozuklukları gibi belirli bilişsel profil eksiklikleri, UDp ve BDp arasındaki erken müdahaleler için değerli hedefler sağlar. Bu çalışma, duygudurum bozukluklarının erken tanı ve yönetiminde nöropsikolojik testlerin kritik rolünü pekiştirmektedir. Gelecekteki araştırmalar, metodolojik sınırlamaları ele almaya, multimodal yaklaşımları entegre etmeye ve hasta sonuçlarını iyileştirmek için tedavileri belirli nörobilişsel profillere göre uyarlamaya odaklanmalıdır.

Received: 03.11.2024
Accepted:19.06.2025

**Anahtar Sözcükler:** Bipolar bozukluk, unipolar depresyon, bilissel bozukluk, yürütücü işlev, sözel bellek, nöropsikolojik testler.

Patients with major depressive disorder (MDD) and bipolar disorder (BD), both symptomatic and symptom-remitted, frequently have cognitive abnormalities. The degree of neurocognitive impairment that distinguishes these two illnesses is still unknown, though.

Frontal lobe and intelligence tests have been used to compare the neuropsychological performance of patients with unipolar and bipolar mood disorders during acute episodes of depression. Compared to unipolar depressed patients, bipolar patients exhibited a greater level of cognitive dysfunction associated with frontal lobe activity during an acute depressive episode. These findings may support other research suggesting some similarities between bipolar disorder and schizophrenia as well as pathogenic differences between bipolar and unipolar affective disorder (1).

One important neurocognitive component of impulsivity in mood disorders is response inhibition. Xia et al. (2) investigated whether oscillatory patterns can serve as early indicators for BD and examined the similarities and differences between the brain circuit changes linked to response inhibition in BD and unipolar depression (UDp). While rIFG aberrant activity is more exclusive to BD, prefrontal control network inefficiency is a common mechanism in mood disorders. In the early stages of BD, neural responses during response inhibition may be used as a diagnostic biomarker.

In the study of Xu et al. (3), a battery of neuropsychological tests was administered to three groups of patients with UDp (n =293), bipolar I (n =92), and bipolar II (n =131) both at baseline (during a depressive episode) and after six weeks of treatment. Cognitive performance was compared with 202 healthy controls. Processing speed, attention, memory, verbal fluency, and executive function were among the cognitive domains. The three patient groups (bipolar I, bipolar II, and UDp) exhibited cognitive dysfunction in verbal fluency, processing speed, memory, and executive function during the acute depressive state, but not in attention when compared to controls. Bipolar I depressed patients outperformed bipolar II and UDp depressed patients in verbal fluency and executive function, according to post comparisons. There was no discernible difference between UDp and bipolar II people, except in visual memory. Bipolar I and II patients who were clinically remitted only showed cognitive impairment in visual memory and processing speed after six weeks of treatment. In addition to processing speed and visual memory, remitted UDp patients also displayed cognitive impairment in executive function. Processing speed and visual memory scores were comparable across the three remitted patient groups. During an acute depressive episode, patients with bipolar I, bipolar II, and UDp all exhibit a similar pattern of cognitive impairment; however, the impairment is more severe for bipolar I patients than for bipolar II and UDp patients. Both bipolar and UDp patients exhibit cognitive deficits in visual memory and processing speed during clinical remission, and executive dysfunction may be a trait-marker for UDp but a status-maker for bipolar disorder (3).

It is well recognized that MDD and BD are frequently associated with cognitive deficits. However, there is disagreement over which specific cognitive processes in each domain of functioning can be impaired to cause disability. Working memory, inhibition, cognitive flexibility, and attention over an extended period of time in individuals with BD and MDD were assessed look for disability and quality of life, and assess how factors associated with cognitive reserve-such as education and daily cognitive stimulation-affect cognitive function. While patients with MDD improved on working memory and attention tests, the BD group demonstrated improvements in attention. Working memory function in BD was linked to the areas of cognition and movement of functioning, as well as with physical and environmental aspects of daily life. Social ties and the quality of life in the environment were linked to cognitive flexibility in MDD. Ponsani et al. (4) showed that working memory and cognitive flexibility could be intriguing targets for therapies meant to enhance daily functioning and quality of life in people with BD and MDD.

The prefrontal cortex (PFC) and hippocampus, areas essential for verbal memory encoding and retrieval, are linked to both structural and functional abnormalities in BD (Bourne et al., 2013). Due to oxidative stress and neuroinflammatory processes that impair synaptic plasticity and memory consolidation, hippocampal atrophy is more noticeable in BD (Fernandes et al., 2017). Verbal memory performance may deteriorate due to fronto-temporal connectivity impairment caused by dysregulated dopamine and glutamate neurotransmission in BD, especially during depressive episodes (Bortolato et al., 2016). According to the "Kindling Hypothesis" (Post, 1992), recurrent mood episodes in BD lead to cumulative damage to the temporal lobes, which progressively exacerbates memory impairments. The purpose of this study is to compare the cognitive profiles of unipolar and bipolar depressive episodes. Both unipolar and bipolar depression are thought to result in neurocognitive impairments, but we hypothesize that verbal memory impairments will be more severe in bipolar depressive disorder.

## MATERIAL AND METHOD

### Participants

Retrospective data were collected from the NP Brain Hospital database of patients with DSM-5-classified Bipolar I disorder, depressive (BDp) (n =39, age= 47±4.5 (38-52)) and unipolar depressive (UDp) (n =39, age = 34±17 (22-66)) patients. According to

The Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5), which requires the presence of at least one manic episode for a Bipolar I diagnosis, verified by the patient's psychiatrists. Using psychiatric diagnostic interviews, bipolar and unipolar depressive phases were assessed using the DSM-5 criteria (SCID-5). Exclusion criteria included: neurological conditions that significantly impair cognitive function, such as cerebrovascular events, epilepsy, and intellectual disabilities whose symptoms began shortly after birth; conditions that are known to impair cognition, begin at birth, and manifest before the age of 18; neurodevelopmental problems, such as attention deficit hyperactivity disorder and autism spectrum disorders; neurological disorders, such as infantile cerebral palsy; medical conditions, such as meningitis; head injuries; substance or alcohol use disorders; and intoxication at the time of data collection. The Uskudar University Ethics Committee approved the study.

**Evaluation**: A battery of neuropsychological tests was conducted by skilled clinical psychologists. The five neurocognitive domains were given the following measures: 1. Working memory and complex attention; 2. Executive functioning; 3. Face-image recognition; 4. Non-verbal memory; 5. Verbal memory; and according to the specified domain, the following tests were used to assess the neurocognitive condition of the patients:

**Complex attention and working memory:** In the well-known Digit Span test, participants must repeat a series of progressively longer digits. The majority of healthy people perform within the seven plus/minus two span of apprehension range, which is a solid indicator of simple attention. The digit span test is best viewed as an attentional measure, and while some believe it to be reflective of a memory task, it actually places very little strain on memory in general. In order to show this idea, amnestic individuals with Korsakoff's syndrome and Alzheimer's disease frequently exhibit a normal digit span forward in spite of profound anterograde amnesia. In contrast to digits ahead, digit span reverse is a qualitatively distinct task that more heavily depends on working memory abilities (5).

**Executive Functions:** These tools measure cognitive flexibility, problem-solving skills, and the ability to shift strategies based on new information.

The Verbal Fluency Test assesses patients' phonemic fluency by asking them to pronounce the letters K, A, and S (Controlled Oral Word Association-COWAT test) and fruit and animal name pairs respectively.

Stroop Color-Word Interference Test (SCWT): One neuropsychological test that is commonly used to assess a person's ability to avoid cognitive interference, known as the Stroop Effect, which occurs when processing one sensory attribute interferes with processing another (6). Strong interference between language and color information is known as the Stroop effect, a well-known and classic human behavioral phenomenon. By deducting the time needed for color naming from the time needed for word reading, the interference score is determined.

**Face-Image Recognition:** Benton Face Recognition Test: The Benton Face Recognition Test evaluates people between the ages of 16 and 74 on their ability to recognize pictures of unknown faces (7). The following tasks are completed in the facial recognition test, which consists of six options and a target photograph shot from the front:

1. Among the options, locate the identical target photograph.
2. Compare three images of the same individual taken from various perspectives with the target photograph.
3. Comparing the target photo to one that was taken in a different lighting setting (one that was 1/4, 2/4, or 3/4 less than normal lighting).

**Visual (nonverbal) memory, both short- and long-term:** Wechsler Memory Scale-Fourth Edition: Auditory memory, visual memory, and visual working memory are among the memory functions that are evaluated by the WMS-IV. Tasks including both immediate and delayed recall and recognition are used to gather comprehensive data on a person's memory functions. A quick assessment of cognitive status is also included in the WMS-IV.

**Verbal memory, short- and long-term:** The Verbal Memory Processes Test (VMPT), developed by Öktem, evaluates the ability to learn verbal material and to recall and recognize learned material from memory. It is frequently used to assess patients in neurology and psychiatry. Based on the Rey Auditory Verbal Learning Test (8), this word-list learning test was created by Öktem (9). The test consists of two stages. In the first stage, the examiner reads a list of 15 words and asks the participant for immediate recall. Recall is reassessed after 45 minutes to evaluate verbal long-term memory. Immediate memory and long-term recall scores are utilized in this study to assess verbal short-term/ working memory and long-term learning.

These neuropsychological tools provided a detailed overview of the cognitive profiles of participants, enabling a systematic comparison between unipolar and bipolar depressive patients during acute depressive episodes.

**Statistical Analysis**

The statistical analysis was conducted using IBM Statistics for Windows 22.00 (Chicago, IL, USA). Kolmogorov-Smirnov test was used to assess normality. To assess demographic features for non-normally distributed variables, the frequency, median, interquartile range, and range [minimum - maximum] were used. We used non-parametric testing such as Mann-Whitney U Test, because the results of neuropsychological tests did not fit the normal distribution based on Kolmogorov-Smirnov test results. Chi-Square test ($\chi^2$) was used to compare categorical variables between the two groups. The Mann-Whitney U (MWU) test was used to compare non-normally distributed neuropsychological tests among bipolar and unipolar depression patients. The cutoff for statistical significance was set at a two-tailed p-value of 0.05.

## RESULTS

The BDp group consisted of 16 males and 23 females (median age ± interquartile range (IQR)**:** 34 ± 17 years, range: 22-66), while the UDp group included 19 males and 20 females (median age ± IQR**:** 47 ± 4.5 years**,** range: 38–52). Patients with BDp and UDp were evaluated based on their neurocognitive performance during acute depressive states.

A statistical comparison revealed no significant differences in age (z score**=** -0.425, p = 0.671) or gender ($\chi2$=0; p =1) distribution between the two groups, ensuring comparability in demographic variables (Table 1).

**Table 1.** Demographic characteristics of participants and distribution of BDp and UDp groups.

| Groups | Bipolar Depression Phase (BDp) (n =39) | Unipolar Depression Phase (UDp) (n =39) | p value |
|---|---|---|---|
| Gender | 16 M, 23 F | 19 M, 20 F | (p =1) * |
| Age | 34±17 (22-66) | 47±4.5 (38-52) | (p =0.671) ** |

*M males, and F females, no significant gender (Chi Square test, $\chi2$=0; p =1) differences between BDp and UDp.

**Age was nonnormally distributed, no significant age differences between BDp and UDp (MWU test, z score**=**-0.425; p = 0.671).

Table 2 provides an overview of the median and interquartile range (range= minimum-maximum) for each group (UDp and BDp), as well as the significance level (p-value) to determine whether the differences between groups are statistically significant.

The MWU test for UDp versus BDp subjects' neurocognitive test comparison are given for all tests (Table 2).

**Table 2.** Comparison of cognitive function scores in various areas of bipolar depression and unipolar depression groups.

| Cognitive Domain | Test | UDp (Median ± IQR) | BDp (Median ± IQR) | Z score | p-value |
|---|---|---|---|---|---|
| **Working Memory and Complex Attention** | Digit Span (Forward) | 5.5± 1 (3-8) | 6± 2 (5-8) | -1.299 | .194 |
| | Digit Span (Backward) | 4± 2 (3-7) | 4± 1 (2-6) | -.677 | .499 |
| **Executive Functioning** | Verbal Fluency | 30.5±23.75 (9-72) | 35±22 (9-61) | -.159 | .874 |
| | Stroop False | 1± 3.75 (0-12) | 0± 0 (0-10) | -1.955 | .051 |
| | Stroop Spontaneous | 2± 4.75 (0-12) | 1± 1.5 (0-11) | -1.369 | .171 |
| | Stroop Time | 45.5± 91.25 (20-160) | 49.9± 40 (23-96) | -.063 | .950 |
| **Face- Image Recognition** | Benton Facial Recognition Test | 42.2± 6 (36-52) | 47± 6.5 (36-54) | -.634 | .526 |
| | Image Recognition | 1± 0.75 (0-1) | 1± 0 (0-1) | -.473 | .636 |
| **Non-Verbal Memory** | WMS VI Immediate | 9.5± 5.75 (2-16) | 10± 5 (5-14) | -1.588 | .112 |
| | WMS VI Delayed | 8± 8.25 (1-14) | 8± 6 (2-14) | -1.746 | .081 |
| | WMS VI Recognition | 1± 1.75 (0-2) | 0± 1 (0-3) | -.128 | .898 |
| **Verbal Memory** | VMPT Immediate | 5± 2 (2-9) | 5± 3 (2-10) | -1.333 | .183 |
| | VMPT Learning | 113.5± 46.75 (47-116) | 101± 32.5 (66-136) | -2.675 | .007 |
| | VMPT Criteria | 5± 7 (0-10) | 0± 5 (0-8) | -2.571 | .010 |
| | VMPT Maximum | 15± 4.75 (7-15) | 13± 4 (8-15) | -2.605 | .009 |
| | VMPT Delayed | 10± 4 (2-15) | 10± 5 (3-14) | -2.823 | .005 |
| | VMPT Delayed Recognition | 5± 4.75 (0-14) | 5± 5 (1-12) | -1.096 | .273 |
| | VMPT False Recognition | 0± 0 (0-6) | 0± 0 (0-2) | -.159 | .874 |
| | VMPT Total Memory | 15± 0 (9-15) | 15± 0 (11-15) | -.426 | .670 |

*UDp: Unipolar Depression, BDp: Bipolar Depression Phase, WMS: Weschler Memory Scale, VMPT: Verbal Memory Processing Test. Results are given as Median ± Interquartile Range (min-max). MWU test is used for comparison.

In this table, the Vmpt Learning-Criteria-Max-Delayed test demonstrates a statistically significant difference between the UDp and BDp groups, with a p-value <0.05, indicating notable disparities in performance for this test.

## DISCUSSION

Comprehensive assessment across five cognitive domains, including verbal memory, executive functions, and attention, provides valuable insights into the cognitive profiles of BDp and UDp. While some measures, such as verbal learning (e.g., VMPT Learning, Criteria, Max, and Delayed), showed slight variations, most cognitive deficits were shared between the two groups. These findings align with prior research emphasizing overlapping neurocognitive impairments in mood disorders.

Barreiros et al. (10) investigated the P300 event-related potential as a biomarker for distinguishing cognitive dysfunction in BDp and UDp. Their study revealed that while UDp patients exhibited P300 amplitudes similar to healthy controls (HC), BDp patients demonstrated significantly reduced P300 amplitudes compared to both groups. This suggests that certain brain processes related to attention and context updating remain impai-

red in BDp even during symptom remission. Although their classification model achieved a modest accuracy of 53.5%, these findings highlight the potential of electrophysiological signatures like P300 in differentiating BDp from UDp.

These findings are consistent with Xu et al. (3) and Cai et al. (11), who identified memory and executive dysfunction as core features of mood disorders. For example, Xu et al. (3) observed that young adults with depression, whether unipolar or bipolar, exhibit notable deficits in verbal memory, despite intact processing speed, attention, and executive functions. They suggested that early neuropsychological testing could identify at-risk individuals, emphasizing the importance of early intervention. Targeted therapies addressing verbal memory deficits during the early stages of mood disorders may help prevent long-term neurobiological deterioration. Similarly, Cai et al. (11) reported that individuals with BDp show greater impairments in executive function and memory than those with UDp, supporting the notion that these cognitive domains are particularly vulnerable in BDp.

Galimberti et al. (12) further expanded on these findings using network analysis to explore cognitive differences between MDD and BD. They reported that while both groups exhibited modest cognitive impairments, network metrics revealed distinct patterns. Memory impairments were more central in MDD, whereas executive dysfunction played a dominant role in BD. These findings suggest that while overall cognitive deficits may appear similar across mood disorders, the underlying neural mechanisms and their clinical implications differ, reinforcing the need for tailored therapeutic strategies.

Samamé et al. (13) also showed that verbal memory as measured by list learning tests showed a substantial overall effect size favoring major depressive illness during euthymia, while the other variables under analysis showed no significant between-group differences. The groups' cognitive outcomes during depression episodes were comparable. Based on the evidence now available, it is not viable to assume distinct cognitive characteristics for bipolar disorder and major depression. It is still unclear if the variations observed in verbal memory are a reflection of varied underlying processes or if sample features or varying exposure to factors that impair cognition are the most likely explanations.

Certain mood fluctuations are a hallmark of MDD and BD. Changes in cognitive domains like impulsivity, risk-taking, and decision-making have been documented in both diseases. Ramírez-Martín et al. (14) showed that Impulsivity levels were higher in BD and MDD individuals than in HC participants; the difference was particularly noticeable in BD participants. Inhibitory control was also worse in BD participants than in MDD participants. Overall, both mood disorders (BD and MDD) were linked to poor decision-making. Neither group showed any discernible deterioration in risk-taking behavior.

Godard et al. (15) provided additional longitudinal evidence, demonstrating that psychosocial and cognitive impairments in both UDp and BDp persist even during subsyndromal and euthymic states. Their study highlighted widespread occupational and relational dysfunction alongside heterogeneous neurocognitive deficits, with attentional processes frequently compromised. Despite treatment, these impairments remained after one year, significantly impacting daily functioning. These findings underscore the chronic nature of cognitive and psychosocial deficits in mood disorders.

Interestingly, while many studies report more pronounced deficits in BDp, this study observed minimal differences between UDp and BDp, which may reflect methodological or demographic factors. This calls for further research into the impact of age, illness duration, and symptom severity on cognitive profiles.

Nevertheless, the emphasis on early intervention remains crucial. Hermens et al. (16) also showed that when compared to controls, both unipolar and bipolar individuals displayed notable deficits in verbal memory and attentional switching. Psychomotor speed, executive control, and attention to normal mental processes were unaffected in either mood disorder group. The unipolar and bipolar groups did not differ from one another on a variety of neuropsychological measures, according to effects size calculations. Compared to older patients, young adults with contemporary depression disorders seem to exhibit different neurocognitive abnormalities. Even when processing speed, attention, and executive abilities are intact, poor verbal memory can be a distinguishing feature of both unipolar and bipolar depression in early adulthood. According to this study, young individuals in the early stages of severe mood disorders may benefit from neuropsychological testing.

Few treatments have been shown to be linked to cognitive benefits, even though most mood disorder treatments have generally benign cognitive profiles. The detrimental cognitive profiles of some, like lithium, have been the subject of comparatively thorough research. Data from 2876 BD participants from 31 primary studies (5) were included in a recent mega-analysis, which found that while there was some evidence that not taking medication improved performance on two of 11 measures, there was little evidence that medication had an impact on performance. According to one meta-analysis, medication only affects BD patients' processing speed (21). The effect of polypharmacy on mood disorder patients' cognitive abilities has not been thoroughly investigated in many studies. Antidepressant treatment appears to improve one aspect of executive functioning (Stroop test performance) in MDD patients without psychosis, according to a small meta-analysis (three studies; n =122) (22). Antidepressants with pro-cognitive effects, like vortioxetine, may be more beneficial for UDp, whereas mood stabilizers, like lithium, may help to preserve hippocampal volume in BDp (5).

Pharmacological and non-pharmacological strategies addressing verbal memory and executive function deficits may mitigate long-term neurobiological changes and enhance functional outcomes. Training on mental tasks and processes is a common component of cognitive remediation. Following 10 weeks of cognitive training in 38 patients with mood disorders, increa-

ses in brain activity as determined by functional neuroimaging are observed (23). The prefrontal, temporal, and parietal regions showed increased activations during the working memory task, and the hippocampus showed increased activations during the recollection memory task following training. These increases were linked to improvements in verbal working memory and delayed recall measures (23).

In conclusion, these findings reinforce the existence of both shared and distinct cognitive deficits in UDp and BDp, particularly highlighting verbal memory impairments in UDp. For early differentiation of BDp from UDp, particularly when mood episodes are the only presenting feature, include verbal memory tests (e.g., VMPT, Rey AVLT) in routine evaluations for Enhanced Differential Diagnosis Screening tool for depressive episodes. Future studies should explore the integration of neuropsychological assessments and biomarkers, such as the P300 component, to refine diagnostic differentiation and personalize treatment approaches for mood disorders. Cognitive Remedial Therapy focuses on encoding techniques like chunking and semantic clustering to address hippocampal dysfunction and attentional lapses, prioritizing retrieval practice and attentional training for BDp.

**Limitations**

The relatively small number of participants in each group (39 UDp, 39 BDp) may limit the generalizability of findings and the ability to detect subtle differences. Data collection from a hospital database might have biases related to incomplete information or diagnostic inconsistencies. The wide age range (22-66 years) in the BDp group introduces potential confounding, as cognitive performance is age-dependent. The lack of a younger UDp sample also complicates direct comparisons. The study provides a snapshot of cognitive deficits during depressive phases but does not assess changes over time or the impact of treatment on cognitive recovery. Comorbidities or medication effect were not analyzed in the study.

**Future Directions**

Longitudinal Studies can assess cognitive changes over time, particularly during remission phases, to determine the trajectory of deficits and their trait versus state markers. Future research should include power calculations to ensure adequate sample sizes and statistical robustness. Incorporate larger, more homogenous samples to clarify distinctions between UDp and BDp, particularly in younger populations. To be able to integrate biomarkers, combine neurocognitive testing with neuroimaging or electrophysiological markers (e.g., P300 amplitude) to enhance diagnostic accuracy and explore underlying mechanisms. Test the impact of interventions targeting verbal memory, such as cognitive training or pharmacological therapies, on cognitive and functional outcomes in early-stage patients.

**Conclusion**

This study highlights verbal memory deficits as a prominent feature of mood disorders, with overlapping cognitive impairments observed between UDp and BDp. The findings align with previous research suggesting memory and executive dysfunction as hallmark features of these conditions. However, specific measures such as Vmpt Learning, Criteria, Max, and Delayed tests revealed differences between UDp and BDp, reinforcing the value of neuropsychological assessments in distinguishing these disorders. Verbal memory profiling offers a low-cost, scalable marker to refine diagnosis, guide treatment, and mitigate disability in mood disorders. Integrating these findings into standard neuropsychological batteries could reduce diagnostic delays and optimize outcomes.

## REFERENCES


1. Borkowska A, Rybakowski JK. Neuropsychological frontal lobe tests indicate that bipolar depressed patients are more impaired than unipolar. Bipolar Disord 2001; 3: 88-94.
2. Xia Y, Wang X, You W et al. Impulsivity and neural correlates of response inhibition in bipolar disorder and their unaffected relatives: A MEG study. J Affect Disord 2024; 351: 430-41.
3. Xu G, Lin K, Rao D et al. Neuropsychological performance in bipolar I, bipolar II and unipolar depression patients: a longitudinal, naturalistic study. J Affect Disord 2012; 136: 328-39.
4. Ponsoni A, Branco LD, Cotrena C, Shansis FM, Fonseca RP. A longitudinal study of cognition, functional outcome and quality of life in bipolar disorder and major depression. Appl Neuropsychol Adult 2023; 30: 757-63.
5. Bourne C, Aydemir Ö, Balanzá-Martínez V et al. Neuropsychological testing of cognitive impairment in euthymic bipolar disorder: an individual patient data meta-analysis. Acta Psychiatr Scand 2013;128: 149-62.
6. Fernandes BS, Berk M. Staging in bipolar disorder: one step closer to precision psychiatry. Braz J Psychiatry 2017; 39: 88-9
7. Bortolato B, Miskowiak KW, Köhler CA, Vieta E, Carvalho AF. Cognitive dysfunction in bipolar disorder and schizophrenia: a systematic review of meta-analyses. Neuropsychiatr Dis Treat 2015; 11: 3111-25.
8. Post RM. Transduction of psychosocial stress into the neurobiology of recurrent affective disorder. Am J Psychiatry 1992; 149: 999-1010.
9. Reynolds CR. Forward and backward memory span should not be combined for clinical analysis. Arch Clin Neuropsychol 1997; 12: 29-40.
10. Stroop JR. Studies of interference in serial verbal reactions. J Experiml Psychol Gen 1992; 121: 15-23.
11. Benton AL, Hamsher K, Varney NR, Spreen O. Contributions to neuropsychological assessment, Oxford University Press, New York, 1983.
12. Rey A. L'examin clinique en psychologie. Paris, France: Presses Universitaires de France, 1958.
13. Öktem Ö. Sözel bellek süreçleri testi (bir ön çalışma). Nöropsikiyatri Arşivi 1992; 29:196-206.
14. Barreiros AR, Breukelaar IA, Chen W et al. Neurophysiological markers of attention distinguish bipolar disorder and unipolar depression. J Affect Disord 2020; 274: 411-9.
15. Cai Y, Kuang W, Guo T, Yan L, Zhu J, & Chen H. Zhong nan da xue xue bao. Yi xue ban-J Cent South University. Med Sci 2012; 37: 1152-5.
16. Galimberti C, Bosi MF, Caricasole V, Zanello R, Dell'Osso B, Viganò CA. Using network analysis to explore cognitive domains in patients with unipolar versus bipolar depression: a prospective naturalistic study. CNS Spectr 2020; 25: 380-91.
17. Samamé C, Szmulewicz AG, Valerio MP, Martino DJ, Strejilevich SA. Are major depression and bipolar disorder neuropsychologically distinct? A meta-analysis of comparative studies. Eur Psychiatry 2017; 39: 17-26.
18. Ramírez-Martín A, Sirignano L, Streit F et al. Impulsivity, decision-making, and risk behavior in bipolar disorder and major depression from bipolar multiplex families. Brain Behav 2023; 14: e3337.
19. Godard J, Baruch P, Grondin S, Lafleur MF. Psychosocial and neurocognitive functioning in unipolar and bipolar depression: a 12-month prospective study. Psychiatry Res 2012; 196: 145-53.
20. Hermens DF, Naismith SL, Redoblado Hodge MA, Scott EM, Hickie IB. Impaired verbal memory in young adults with unipolar and bipolar depression. Early Interv Psychiatry 2010; 4: 227-33.
21. Bora E, Yucel M, Pantelis C. Cognitive endophenotypes of bipolar disorder: A meta-analysis of neuropsychological deficits in euthymic patients and their first-degree relatives. J Affect Disord 2009; 113: 1-20.
22. Wagner S, Doering B, Helmreich I, Lieb K, Tadic A. A meta-analysis of executive dysfunctions in unipolar major depressive disorder without psychotic symptoms and their changes during antidepressant treatment. Acta Psychiatr Scand 2012; 125: 281-92.
23. Meusel LA, Hall GB, Fougere P, McKinnon MC, MacQueen GM. Neural correlates of cognitive remediation in patients with mood disorders. Psychiatry Res 2013; 214: 142-52.