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# Effect of Repetitive Transcranial Magnetic Stimulation on Neurocognitive Functions in Moderate and Severe Depression: A Longitudinal Study

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**ABSTRACT**

**Objective:** We aimed to investigate the effects of transcranial magnetic stimulation (TMS) on depressive symptoms and neurocognitive functions during treatment and follow-up.

**Methods:** A total of 65 patients diagnosed with major depressive disorder, with a >16 scores in Hamilton Depression Rating Scale (Ham-D) and a >18 scores in Beck Depression Inventory (BDI), participated in the study. The Ham-D, BDI, Beck Anxiety Inventory (BAI), Trail Making Tests A and B, Stroop Colour and Word Test, Number Sequence Test, Öktem Verbal Memory Processes Test, and Verbal Fluency Test were administered at baseline, 1st-, and 3rd-month for both treatment and control groups.

**Results:** A total of 65 patients were enrolled in the study, of whom 25 in the TMS group and 26 in the non-TMS group completed the follow-up. At the first month, 73.5% of patients in the TMS group showed a full treatment response, compared to 29.03% in the non-TMS group (p=0.001). At the third month, the treatment response rate decreased to 40% in the TMS group, whereas it was 42.3% in the non-TMS group, and the significant difference between the groups disappeared (p=0.918). In terms of cognitive functions, no significant changes were observed in either group at the first- and third-month follow-ups compared to baseline.

**Conclusion:** TMS had a strong acute antidepressant effect; however, this effect diminished over time during the follow-up period. Although partial improvement was observed in cognitive functions, this improvement did not reach statistical significance.

**Keywords:** Cognitive functions, major depressive disorder, transcranial magnetic stimulation

## INTRODUCTION

Major depressive disorder (MDD) is one of the most prevalent psychiatric conditions, characterized by high recurrence rates and significant functional impairment (Gilmour and Patten 2007). Even when remission is achieved through antidepressant treatment, residual symptoms such as fatigue and cognitive dysfunction often persist (Stahl et al. 2003). Studies have shown that, despite heterogeneity in individual studies, the most common cognitive deficits in MDD are related to attention, memory, psychomotor speed, and executive functions (Bortolato et al. 2016). Therefore, improving cognitive functions is a primary objective of newly developed treatments (Dam et al. 2022).

In recent years, transcranial magnetic stimulation (TMS) has emerged as a preferred biological treatment approach due to its non-invasive nature and ease of application (Rachid 2018). The Canadian Network for Mood and Anxiety Treatments (CANMAT) guidelines rate the evidence for both high- and low-frequency TMS as Level 1 for the treatment of mood disorders (Lefaucheur et al. 2020). Functional neuroimaging studies in depressed individuals report decreased activity in the left prefrontal cortex (specifically Broadmann areas BA 9 and BA 46) and altered activation in a cortico-subcortical network that includes the subgenual and anterior cingulate cortices. High-frequency (HF) TMS applied to the left dorsolateral prefrontal cortex (DLPFC) has shown antidepressant efficacy by activating this region in both acute and long-term

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phases (Bagherzadeh et al. 2016). Repetitive TMS (rTMS) induces slow and sustained neuroplastic changes in the stimulated region, influencing dopaminergic and adrenergic neurotransmitter regulation and gene expression (Bagherzadeh et al. 2016). Furthermore, rTMS has been reported to increase plasma levels of brain-derived neurotrophic factor (BDNF) (Yukimasa et al. 2006), regulate the hypothalamic-pituitary-adrenal (HPA) axis (Keck, 2003), and exhibit anti-inflammatory effects by increasing interferon-α levels (Bikson et al. 2020). It also enhances endogenous opioid release in regions such as the periaqueductal gray matter and anterior cingulate, contributing to its antidepressant effects (de Andrade et al. 2011).

While the optimal timing for TMS in the depression treatment algorithm remains unclear, clinical practice suggests that applying TMS at the onset of a depressive episode in patients under 65 years of age, particularly those who have not responded to two pharmacological treatments, may result in higher success rates (George and Aston-Jones, 2010). Negative predictors of TMS treatment response include high treatment resistance scores, prolonged episode duration, advanced age, and psychotic symptoms (Unsalver and Tarhan, 2017).

The effect of TMS on cognitive functions has been well-documented, with studies suggesting that it influences long-term potentiation, a mechanism critical for learning and memory (Gentner et al. 2008). Recent advances in imaging studies have shown that rTMS enhances synaptic connectivity, modulates receptor and neuromodulator expression, and regulates the functions of dispersed brain circuits (Sharbafshaaer et al. 2023). Increased regional cerebral blood flow observed during high-frequency stimulation (Loo et al. 2003) and post-stimulation increases in prefrontal gamma oscillatory activity have been linked to improvements in cognitive functions (Barr et al. 2009). HF-rTMS applied to the DLPFC has been shown to immediately impact cognition by inducing an anti-inflammatory response (Cha et al. 2022) and increasing superoxide dismutase activity, a factor associated with cognitive performance (Zhu et al. 2019). Additionally, enhanced glutamate neurotransmission observed in patients with vascular cognitive impairment may contribute to cognitive function preservation (Pennisi et al. 2016).

Reports of TMS's efficacy in enhancing cognition in patients with dementia and mild cognitive impairment have raised questions about its potential impact on depression-related cognitive deficits (Di Lazzaro et al. 2021). This study hypothesized that repetitive TMS applied to the prefrontal cortex would result in overall improvement in executive cognitive functions supported by the DLPFC. It aimed to evaluate both the acute and long-term efficacy of TMS in MDD and its potential to improve cognitive functions.

### Study Hypotheses

TMS treatment will reduce depressive symptoms in patients with moderate and severe MDD.

TMS treatment will be more effective in treating depression during follow-up compared to pharmacotherapy alone.

TMS treatment will demonstrate a greater tendency for cognitive function improvement compared to pharmacotherapy alone in patients with moderate and severe MDD.

Improvements in cognitive functions during follow-up will be greater in the TMS-treated group compared to the pharmacotherapy-only group.

## METHOD

### Participants and Protocol

This study commenced following approvals obtained from the Interventional Clinical Research Ethics Committee of Selçuk University (decision dated 29.07.2021, no: E-40209705-050.01.04-113332) and the Turkish Medicines and Medical Devices Agency (approval dated 09.09.2021, no: E-68869993-511.06-532642). Prior to participation, all individuals were informed about the study and provided written informed consent. All procedures performed in this study adhered to institutional/national research committee ethical standards and the 1964 Helsinki Declaration and its later amendments or comparable ethical standards.

The study sample comprised 34 volunteers aged 18-65 years who were diagnosed with major depressive disorder based on the Structured Clinical Interview for DSM-5 Disorders, Clinician Version (SCID-5-CV), meeting moderate to severe depression criteria according to depression scale scores (Ham-D >16 and BDI >18) (Eskin et al. 2013; Akdemir et al. 1996). Of these, 32 participants received transcranial magnetic stimulation (TMS) therapy alongside ongoing pharmacological treatment, while 2 received TMS therapy without pharmacological treatment. Additionally, 31 individuals who met the same criteria but did not receive TMS therapy were included as the control group; 28 were on pharmacological treatment only, and 3 had previously received but discontinued pharmacological therapy. None of the participants exhibited additional psychiatric disorders at the diagnostic level. TMS therapy participants continued their existing pharmacological treatments (except for the 2 individuals receiving only TMS).

For the control group, depressive symptoms and cognitive functions were monitored at baseline, after 20 TMS sessions (1 month), and at the 3-month follow-up. Data collection included the Beck Depression Inventory (BDI), Hamilton Depression Rating Scale (Ham-D), Beck Anxiety Inventory

(BAI), and neuropsychological tests administered by a psychologist. Individuals outside the age range of 18-65 years, those diagnosed with other psychiatric disorders comorbid with depression via SCID-5-CV, and individuals with epilepsy, cerebrovascular diseases, active suicidal ideation, psychotic major depressive disorder, or contraindications for magnetic stimulation (e.g., intracranial implants or pacemakers) were excluded.

### TMS Protocol

The TMS protocol was administered at the TMS unit of the Psychiatry Clinic at Selçuk University under the supervision of a psychiatrist and a psychiatric nurse, using the Magventure TMS therapy device, an advanced high-performance magnetic stimulator. The MC-B70 coil, a butterfly-shaped, non-cooled coil suitable for focused stimulation, was employed. The coil's slightly curved design facilitated close contouring.

During the first session, the resting motor threshold (RMT) was determined by gradually increasing the stimulus at a point 5 cm lateral to the vertex on the mid-interaural line until involuntary muscle contractions were observed in the contralateral hand. The stimulation intensity was set at 120% of the RMT. The treatment site was identified using the "5 cm rule," designating a point 5 cm anterior to the motor cortex point along the parasagittal plane, corresponding to the left dorsolateral prefrontal cortex (DLPFC). The coil was positioned at a 45° angle on the scalp. Each TMS session consisted of 10 Hz stimulation with 40 pulses in each sequence lasting 3,900 milliseconds, with a 26 second gap between the sequences, administered over 75 trains. Each session lasted approximately 37 minutes, with a total of 20 sessions performed.

### Data Collection Tools

**Hamilton Depression Scale (Ham-D):** Developed by Hamilton in 1967, this scale is used not for diagnosis but to measure the severity of depressive symptoms. It tends to prioritize somatic complaints in its evaluation. The scale's validity and reliability were tested by Akdemir et al. in 1996. The maximum score is 53. Scores of 0–7 indicate no depression, 8–15 indicate mild depression, 16–28 moderate depression, and 29 and above severe depression (Akdemir et al. 1996). In this study, the scale was administered by a clinician.

**Beck Depression Inventory (BDI):** The Beck Depression Inventory is a 21-item self-report scale designed to measure characteristic attitudes and symptoms of depression (Beck et al. 1961). It is filled out by the participant and does not serve to diagnose depression but to objectively determine the severity of depressive symptoms. Participants are instructed to select the statement that best describes how they felt over the past week, including the day of administration (Hisli, 1988). Scores of 8 and below indicates no depression, 8–17 indicate mild depression, 18–29 moderate depression, and 30 and above severe depression. The validity and reliability of the scale in Turkish were established by Hisli in 1988 (Eskin et al. 2013). In this study, the inventory was completed by the patient.

**Beck Anxiety Inventory (BAI):** This self-report inventory measures anxiety symptoms experienced in the past week (Beck et al. 1988). Its Turkish validity and reliability study was conducted by Ulusoy et al. in 1998. The total score categorizes anxiety as follows: 8–15 indicates low anxiety, 16–25 moderate anxiety, and 26–63 high anxiety (Ulusoy et al. 1998). In this study, the inventory was completed by the patient.

**Trail Making Test (TMT):** Initially developed by Partington in 1938, the test was first published as part of the Army Individual Test Battery (Partington and Leiter, 1949). It consists of two sections, A and B, and assesses skills such as working memory, visual search, attention, processing speed, mental flexibility, set-shifting, and response inhibition. The Turkish validity and reliability study was conducted by Cangöz et al. in 2007 (Cangöz et al. 2007). In the first section (TMT-A), participants were instructed to connect numbered circles (1–25) sequentially. In the second section (TMT-B), participants alternated between numbers and letters in sequence (e.g., 1-A, 2-B, 3-C). Reaction times and error counts were recorded for each section. This test was administered by a clinical psychologist.

**Stroop Test TBAG Form:** The phenomenon that naming objects or colors takes longer than reading their associated words was first discovered by McKeen Cattell in 1886, with Stroop demonstrating the "color-word interference effect" in 1935 (Stroop, 1935). The Turkish form, developed as part of TÜBİTAK's BİLNOT Battery project, combines the original Stroop Test and the Victoria Form (Karakaş and Başar, 1993). It assesses perceptual set shifting, response inhibition, and focused attention, with particular sensitivity to orbitofrontal cortex impairments (Karakaş et al. 1999). The test consists of five cards: ST-1 and ST-2 measure reading speed; ST-3 and ST-4 measure color naming speed; and ST-5 evaluates task performance under interference. This test was administered by a clinical psychologist.

**Number Sequence Learning Test (NSLT):** Originally developed by Zangwill in 1943, the test's Turkish validity and reliability study was conducted by Karakaş et al. It assesses memory and learning ability and is sensitive to medial temporal lobe and hippocampal impairments (Karakaş and Karakaş, 2001). The test involves recalling randomized sequences of numbers ranging from 1 to 9, presented twice consecutively. Based on the participant's age and education, one of two 8- or

9-item sequences was selected, and participants were asked to recall them in the correct order. Testing continued for up to 12 repetitions or until the participant successfully repeated the sequence twice in succession. This test was administered by a clinical psychologist.

**Verbal Öktem Memory Processes Test:** It is designed to examine verbal learning and memory across multiple factors. It is used to assess the left hippocampus, medial temporal lobe, and frontal lobe. The test evaluates various memory-related parameters, such as the transfer from short-term to long-term memory, the ability to maintain long-term memory storage, and the processes of retrieval and recognition. It also looks at hippocampal-type memory loss and secondary types of memory loss. The test examines short-term memory, learning processes, and the ability to retain and recall information. The Turkish validation and reliability study of this test was conducted in 1992 by Öktem and colleagues. The test is performed by having participants recall a list of words over several trials. In the initial trial, the number of words remembered is used to assess immediate memory and the ability to maintain attention. After reading the same list nine times, the total number of recalled words forms the learning score. About 40 minutes later, the number of words remembered is used to evaluate long-term verbal memory performance. The spontaneous recall score is then calculated. Words that were not listed are evaluated as long-term memory false recall. Additionally, the recognition section examines how many words are recognized with cues. The test is administered by a clinical psychologist.

**Verbal Fluency Test:** Developed by Benton in 1967, verbal fluency tests evaluate various cognitive functions, including language ability and executive functions (Borkowski et al. 1967). The Turkish standardization uses the phonemes K, A, and S instead of the original F, A, and S (Umaç, 1997). Phonemic fluency was assessed with these phonemes, and semantic fluency was evaluated using the category of "items in a market." Participants were given one minute per category to list relevant words, and their responses were recorded. Total correct words and errors were noted. This test was administered by a clinical psychologist.

### Statistical Analysis

All statistical analyses were conducted using IBM SPSS 21.0 and R version 3.6.0. Normality of data distribution was evaluated using the Shapiro-Wilk test and Q-Q plots, while homogeneity of variances was assessed with Levene's test. Normally distributed data were expressed as mean ± standard deviation or adjusted mean (95% CI), while non-normally distributed data were expressed as median (min-max). Categorical variables were presented as frequency (n) and percentage (%). Group comparisons utilized independent samples t-tests or chi-square tests. Changes over time in depression, anxiety, and neuropsychological test scores were analyzed using repeated measures ANOVA or Friedman tests, with post-hoc analyses conducted using Bonferroni or Bonferroni-adjusted Nemenyi tests. For parameters without significant changes, Bonferroni-adjusted Wilcoxon tests were used. Group comparisons at each follow-up point employed independent t-tests or Mann-Whitney U tests. Covariance analyses were applied where baseline differences existed, using ANCOVA for normally distributed data or the non-parametric Quade method otherwise. Remission rates at 1 and 3 months were compared using Yates-corrected chi-square tests. A p-value of $<0.05$ was considered statistically significant.

## RESULTS

### Sociodemographic and Clinical Characteristics

The study included 34 volunteers diagnosed with major depressive disorder (MDD) who underwent TMS therapy (18 females, 16 males, aged 18-65) and 31 volunteers using only pharmacological treatments (18 females, 13 males). The study was completed by 25 TMS-treated patients and 26 patients not receiving TMS. The mean age of the TMS group was calculated as 35.58±13.67, while the non-TMS group had a mean age of 33.06±12.31, with no significant difference between the groups. No statistically significant differences were observed between the TMS and non-TMS groups regarding gender, marital status, years of education, employment status, or residential area. The sociodemographic characteristics of the groups are presented in Table 1.

When comparing the clinical features of MDD between TMS and non-TMS groups, no statistically significant differences were found concerning age of onset, total duration of illness, history of pharmacological treatment, inpatient treatment history, family history of psychiatric disorders, history of physical illness, history of suicide attempts, or smoking, alcohol, and substance use. However, the TMS group experienced more depressive episodes than the non-TMS group. A significant difference was noted in the history of previous treatments and current pharmacological treatments between the groups. Additionally, 14.7% (n=5) of patients in the TMS group had previously undergone neuromodulation techniques, all of which occurred more than one year prior. Clinical characteristic of MDD in both groups are presented in Table 2.

### Evaluation of TMS Treatment Efficacy

Both groups showed significant improvements in BDI scores at the 1-month and 3-month follow-ups compared to baseline. However, no significant difference was observed between the 1-month and 3-month BDI scores within the groups. Due to the significant difference in BDI scores between the groups, statistical analyses of follow-up data were adjusted accordingly. The mean depression scale scores and

**Table 1.** Sociodemographic Characteristics of the Groups

| | | TMS | Control | p value |
|---|---|---|---|---|
| Age (mean ± SD) | | 35.58 ±13.67 | 33.06 ±12.31 | 0.439[a] |
| Gender (n) | Female | 18 | 18 | 0.869[b] |
| | Male | 16 | 13 | |
| Marital Status (n) | Married | 19 | 9 | 0.111[b] |
| | Single | 13 | 19 | |
| | Divorced/Widowed | 2 | 3 | |
| Education (mean ± SD) | | 10.85 ±3.9 | 12.32 ±4.8 | 0.143[a] |
| Employment status (n) | Unemployed | 25 | 20 | 0.772[b] |
| | Employed | 9 | 11 | |

TMS: Individuals receiving TMS treatment; Control: Individuals not receiving TMS treatment
[a]Independent samples t-test, [b]Chi-square test,
Mean: Mean, SD: Standard deviation, n: Number

**Table 2.** Clinical Characteristics of Major Depressive Disorder in Groups

| | | TMS | Control | p value |
|---|---|---|---|---|
| Age at Onset of Illness (mean ± SD) | | 23±9.01 | 22±9.17 | 0.754[a] |
| Duration of Illness (mean ± SD) | | 13.08±9.6 | 10.75±8.39 | 0.162[a] |
| Number of Episodes (mean ± SD) | | 5.91±3.64 | 3.38±1.76 | **<0.001**[a] |
| History of Previous Treatment (n, %) | Pharmacotherapy | 15(%44.1) | 27 (%87) | |
| Current Pharmacological Treatment (n,%) | Psychotherapy | 2(%5.9) | 0 (%0) | **<0.001**[b] |
| | ECT or TMS | 5 (%14.7) | 0 (%0) | |
| | Combined Treatments | 12(%35.3) | 4 (%13) | |
| | No pharmacological treatment | 2(%5.8) | 3(%9.6) | |
| | SSRI Monotherapy | 10(%29.4) | 16 (%51.6) | 0.127[b] |
| | SNRI Monotherapy | 10(%29.4) | 9 (%29) | |
| | Combined Antidepressants | 12(%35.2) | 3 (%9.6) | |
| History of Inpatient Treatment (n, %) | None | 26 (%76.5) | 28(%90.3) | 0.271[b] |
| | Yes | 8 (%23.5) | 3(%9.6) | |
| History of Physical Illness (n, %) | None | 17 (%50) | 14 (%45.2) | 0.805[b] |
| | Yes | 17(%50) | 17 (%54.8) | |
| History of Suicide Attempt (n, %) | None | 24 (%70.6) | 24(%77.6) | 0.583[b] |
| | Yes | 10 (%29.4) | 7 ( %22.6) | |
| Family History (n, %) | None | 16 (%47.1) | 17 (%54.8) | 0.802[b] |
| | Yes | 18 (%52.9) | 14 (%45.16) | |
| Smoking (n, %) | Yes | 13 (%38.2) | 9 (%29) | 0.880[b] |
| | Quit | 2 (%5.9) | 8 (% 25.8) | |
| | None | 19 (%55.9) | 14 (45.2) | |
| Alcohol Use (n, %) | Yes | 2 (%5.9) | 7(%22.58) | 0.229[b] |
| | Quit | 3(%8.8) | 3 (%9.6) | |
| | None | 29 (%85.3) | 21(%67.7) | |
| History of Psychoactive Substance Use (n, %) | Yes | 0 (%0) | 0 (%0) | 0.183[b] |
| | Quit | 1 (%2.9) | 2 (%6.5) | |
| | None | 33 (97.1) | 29 (%93.5) | |

TMS: Individuals receiving TMS treatment; Control: Individuals not receiving TMS treatment.
Bold values indicate statistical significance.
[a]Independent t-test for, [b]Chi-Square test, SD: Standard Deviation, n: Number, SSRI: Selective Serotonin Reuptake Inhibitors, SNRI: Serotonin-Norepinephrine Reuptake Inhibitors
Note: Alcohol and psychoactive substance use do not meet the criteria for addiction.

changes are shown in Table 3. While there was no significant difference in BAI scores between the groups at baseline or during follow-up, significant improvements were observed in both groups at the 1-month follow-up, particularly in the TMS group. However, this improvement diminished by the 3-month follow-up. HAM-D scores at baseline, 1-month, and 3-month follow-ups showed significant differences compared to baseline within both groups, but no significant differences were found between 1-month and 3-month scores. The mean depression scale scores and changes are detailed in Table 3.

When comparing HAM-D response rates, the TMS group demonstrated significantly higher response rates at the 1-month follow-up compared to the non-TMS group. At 3 months, the difference in response rates between the groups disappeared. At the 1-month follow-up, 73.5% (n=24) of the TMS group responded to treatment, compared to 33.3% (n=9) in the non-TMS group. By the 3-month follow-up, the response rate in the TMS group declined to 40%, while the non-TMS group showed an increase in response rates. HAM-D response comparisons are presented in Table 4.

**Table 3.** Mean Depression Scale Scores and Changes in the Groups

| | | Baseline | 1st Month | 3rd Month | Change over time ( p) | | | |
|---|---|---|---|---|---|---|---|---|
| **Parameters** | | **Mean ± SD** | **Median (min - max)** | **Median (min - max)** | | **Posthoc** | | |
| | | | | | | **0-1 m** | **0-3 m** | **1-3 m** |
| BDI | TMS | 34.40 ± 10.29 | 15.88 (12.47-19.29) | 17.93 (13.5-22.37) | **p<0.001** | **p<0.001** | **p<0.001** | 0.662 |
| | Control | 23.79 ± 8.75 | 20.75 (16.95-24.54) | 20.06 (15.53-24.59) | **0.02** | **0.021** | **0.005** | P<0.999 |
| | Group difference (p) | p<0.001 | 0.076 | 0.53 | | | | |
| BAI | TMS | 28.08 ± 12.86 | 17.47 ± 12.86 | 21.07 ± 12.24 | **p<0.001** | **p<0.001** | 0.1 | p<0.999 |
| | Control | 24.80± 11.16 | 19.57± 12.63 | 17.46 ± 12.30 | **0.02** | **0.007** | 0.31 | P<0.999 |
| | Group difference (p) | 0.278 | 0.51 | 0.3 | | | | |
| HAMD-D | TMS | 20.85 ± 5.30 | 8.0 (6.18-9.83) | 12.65 (9.93-15.37) | **p<0.001** | **p<0.001** | **p<0.001** | 0.62 |
| | Control | 18.67 ± 3.28 | 12.09(10.08-14.11) | 10.51 (7.73-13.28) | **p<0.001** | **0.003** | **P<0.001** | 0.716 |
| | Group difference (p) | **0.049** | **0.005** | 0.287 | | | | |

BDI: Beck Depression Inventory, BAI: Beck Anxiety Inventory, HAM-D: Hamilton Depression Rating Scale,

TMS: Individuals receiving TMS treatment; Control: Individuals not receiving TMS treatment, m: Month SD: Standard Deviation, Min: Minimum, Max: Maximum.

Note: The study was completed by 25 patients who received TMS treatment and 26 patients who did not.

Change over time (p): Refers to the comparison of scale scores within the same group across different control time points."Repeated measures ANOVA and Friedman test were used. Bonferroni confidence intervals and Bonferroni-corrected Nemenyi post-hoc tests were used to identify significant changes. Non-significant parameters in the Friedman test were evaluated with Bonferroni-corrected Wilcoxon tests for post-hoc comparisons.

Group difference (p): Independent sample t-test and Mann Whitney U test were used.

For parameters that were found to be significantly different between groups at baseline, to avoid bias, covariance analysis for normally distributed parameters and the Quade method were used for 1st and 3rd month follow-ups. Non-parametric covariance analysis methods were applied for parameters that did not show normal distribution, testing the equality of two regression lines. Bold values indicate statistical significance.

**Table 4.** HAM-D Response Comparisons Between Groups

| HAM-D | Response to Treatment* | TMS (n,%) | Control (n,%) | P value |
|---|---|---|---|---|
| 1st Month Follow-Up | None | 3 (%8.8) | 13(%41.93) | **0.001** |
| | Partial | 6 (%17.16) | 9 (%29.03) | |
| | Full | 25(%73.5) | 9(%29.03) | |
| 3rd Month Follow-Up | None | 10 (%40) | 9(%34.61) | 0.918 |
| | Partial | 5 (%20) | 6(%23.07) | |
| | Full | 10 (%40) | 11(%42.30) | |

TMS: Individuals receiving TMS treatment; Control: Individuals not receiving TMS treatment. The study was completed by 25 patients receiving TMS treatment and 26 patients not receiving TMS treatment. Ham-D: Hamilton Depression Rating Scale, n: Number of patients.
*No response: <25% decrease in HAM-D score; Partial response: 25%-50% decrease in HAM-D score; Response: ≥50% decrease in HAM-D score.

**Table 5.** Comparison of Neuropsychological Test Performances

| | | Baseline | 1 st month | 3rd month | Change over time (p) | | | |
|---|---|---|---|---|---|---|---|---|
| | | Mean ± SD | Mean ± SD | Mean ± SD | | post hoc | | |
| | | | | | | 0-1 | 0-3 | 1-3 |
| Trail Making A test | Group A | 46.82 ± 17.71 | 46.54 ± 17 | 44.09 ± 28.84 | 0.384 | 0.204 | 0.081 | 0.394 |
| | Group B | 97.87 ± 14.47 | 34.88 ± 14.63 | 34.16 ± 14.98 | 0.781 | 0.234 | >0.999 | 0.062 |
| | Group difference (p) | 0.06 | 0.111 | 0.286 | | | | |
| Trail Making B test | Group A | 75.78 ± 43.54 | 80.62 ± 54.75 | 68.76 ± 12.67 | 0.352 | 0.822 | 0.498 | 0.296 |
| | Group B | 88.13 ± 8.31 | 73.01 ± 4.38 | 77.24 ± 8.16 | 0.125 | 0.151 | 0.379 | >0.999 |
| | Group difference (p) | 0.373 | 0.308 | 0.641 | | | | |
| Stroop 1st card | Group A | 11.44 ± 3.08 | 11.50 ± 3.88 | 15.17 ± 20.70 | 0.846 | 0.649 | 0.932 | 0.637 |
| | Group B | 9.97 ± 2.78 | 9.54 ± 1.9 | 9.03 ± 2.9 | 0.764 | 0.334 | 0.159 | 0.485 |
| | Group difference (p) | 0.056 | 0.028 | 0.031 | | | | |
| Stroop 2nd card | Group A | 13.04 ± 4.66 | 15.57 ± 8.10 | 12.97 ±2.87 | 0.115 | 0.158 | 0.179 | 0.265 |
| | Group B | 11.27 ± 4.92 | 11.14 ± 4.19 | 13.39 ± 7.01 | 0.112 | 0.909 | **0.05** | **0.002** |
| | Group difference (p) | 0.013 | 0.588 | 0.506 | | | | |
| Stroop 3rd card | Group A | 15.81 ± 5.64 | 15.50 ± 6.26 | 14.90 ± 4.77 | 0.747 | 0.514 | **0.008** | **0.003** |
| | Group B | 13.87 ± 3.75 | 12.81 ± 3.2 | 12.47 ± 3.96 | 0.024 | P>0.999 | **0.025** | 0.157 |
| | Group difference (p) | 0.145 | 0.098 | 0.151 | | | | |
| Stroop 4th card | Group A | 22.16 ± 10.56 | 20.05 ± 9.86 | 17.25 ± 5.35 | 0.009 | 0.337 | 0.004 | 0.337 |
| | Group B | 17.21 ± 6.34 | 15.86 ± 5.84 | 14.51 ± 6.27 | **p<0.001** | 0.137 | **p<0.001** | 0.55 |
| | Group difference (p) | 0.047 | 0.811 | 0.041 | | | | |
| Stroop 5th card | Group A | 33.24 ± 13.13 | 30.08 ± 14.49 | 29.62 ± 10.80 | 0.311 | 0.14 | 0.103 | 0.689 |
| | Group B | 29.01 ± 10.74 | 25.71 ± 12.59 | 26.67 ± 14.11 | 0.019 | 0.043 | **0.004** | 0.534 |
| | Group difference (p) | 0.252 | 0.099 | 0.081 | | | | |
| Number Sequencing Test | Group A | 8.48 ± 8.21 | 7.66 ± 9.35 | 9.29 ± 7.97 | 0.941 | 0.673 | 0.756 | 0.431 |
| | Group B | 9.92 ± 9.87 | 10.15 ± 9.43 | 9.92 ± 9.60 | 0.546 | 0.754 | 0.917 | 0.955 |
| | Group difference (p) | 0.679 | 0.231 | 0.721 | | | | |
| Verbal Memory Processes Test Instant Learning | Group A | 5.84 ± 1.97 | 5.30 ± 1.77 | 5.29 ± 1.90 | 0.179 | 0.117 | 0.037 | 0.95 |
| | Group B | 6.15 ± 1.84 | 5.05 ± 1.69 | 6.23 ± 2.5 | 0.192 | 0.215 | 0.697 | 0.151 |
| | Group difference (p) | 0.767 | 0.503 | 0.131 | | | | |
| Verbal Memory Processes Test Total Learning | Group A | 100.18 ± 24.74 | 94.69 ± 24.12 | 94 ± 23.35 | 0.16 | **0.013** | **0.018** | 0.297 |
| | Group B | 107 ± 23.90 | 101 ± 25.65 | 102 ± 30.88 | 0.112 | 0.055 | 0.509 | 0.431 |
| | Group difference (p) | 0.317 | 0.262 | 0.19 | | | | |
| Verbal Memory Processes Test Reaching Criteria | Group A | 2.33 ± 3.73 | 2.24 ± 3.57 | 1.87 ± 3.74 | 0.908 | 0.875 | 0.611 | p>0.999 |
| | Group B | 2.19 ± 3.23 | 2.30 ± 3.48 | 2.11 ± 3.08 | 0.436 | 0.859 | 0.779 | 0.717 |
| | Group difference (p) | 0.963 | 0.347 | 0.401 | | | | |
| Verbal Memory Processes Test Highest Learning | Group A | 11.93 ± 3.35 | 11.60 ± 3.75 | 11.99 ± 2.96 | 0.658 | 0.152 | 0.424 | 0.982 |
| | Group B | 12.34 ± 3.36 | 12.57 ± 2.56 | 17.11 ± 21.47 | 0.892 | 0.426 | 0.421 | 0.273 |
| | Group difference (p) | 0.152 | 0.39 | 0.554 | | | | |

**Table 5.** Continued...

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| Verbal Memory Processes Test Spontaneous Recall | Group A | 10.33 ±3.26 | 9.06 ± 4.22 | 9.20 ± 4.25 | 0.308 | 0.055 | 0.984 | **0.042** |
| | Group B | 11.19 ± 2.56 | 10.30 ± 3.12 | 10.96 ± 3.82 | 0.205 | 0.06 | 0.697 | 0.271 |
| | Group difference (p) | 0.344 | 0.256 | 0.153 | | | | |
| Verbal Memory Processes Test Recognition | Group A | 4.27 ± 2.92 | 5.51 ± 3.39 | 5.20 ± 3.4 | 0.511 | **0.034** | 0.113 | p>0.999 |
| | Group B | 3.7 ± 2.49 | 4.5 ± 2.94 | 3.57 ± 2.87 | 0.179 | 0.087 | 0.761 | 0.106 |
| | Group difference (p) | 0.574 | 0.318 | 0.083 | | | | |
| Verbal Memory Processes Test Total Recall | Group A | 13.60 ± 3.87 | 13.54 ± 3.92 | 14.75 ± 0.67 | 0.186 | 0.796 | 0.705 | 0.079 |
| | Group B | 14.92 ± 0.27 | 14.34 ± 0.460 | 15 | 0.146 | 0.317 | 0.102 | 0.157 |
| | Group difference (p) | 0.079 | 0.118 | 0.065 | | | | |
| Verbal Memory Processes Test Perseveration | Group A | 3 ± 3.29 | 3.33 ± 3.59 | 2.95 ± 2.33 | 0.917 | 0.657 | 0.748 | 0.974 |
| | Group B | 2.6 ± 2.07 | 3.80 ± 3.76 | 3.84 ± 3.85 | 0.667 | 0.35 | 0.337 | 0.841 |
| | Group difference (p) | 0.888 | 0.654 | 0.631 | | | | |
| Verbal Fluency Test Semantic Fruit | Group A | 8.33 ± 2.10 | 8.87 ± 2.47 | 9.41 ± 3.80 | 0.909 | **0.001** | 0.041 | 0.826 |
| | Group B | 9.26 ± 2.49 | 8.57 ± 2.30 | 8.50 ± 2.12 | 0.767 | 0.0821 | 0.866 | 0.701 |
| | Group difference (p) | 0.547 | 0.951 | 0.317 | | | | |
| Verbal Fluency Test Semantic Animal | Group A | 16.87 ± 4.82 | 17.57 ± 4.28 | 20.16 ± 7.42 | 0.012 | 531 | **0.003** | 0.154 |
| | Group B | 17.92 ± 4.23 | 18.69 ± 4.24 | 20.65 ± 5.73 | 0.077 | 0.392 | **0.032** | **0.035** |
| | Group difference (p) | 0.52 | 0.37 | 0.669 | | | | |
| Verbal Fluency Test Lexikal | Group A | 30.66 ± 11.66 | 32.94 ± 12.10 | 32.75± 15.51 | 0.068 | 0.011 | **0.021** | 0.337 |
| | Group B | 31.57 ± 11.06 | 36.42 ± 12.27 | 39.07 ± 12.81 | **0.011** | **0.002** | **0.011** | **0.03** |
| | Group difference (p) | 0.837 | 0.285 | 0.115 | | | | |

TMS: Individuals receiving TMS treatment; Control: Individuals not receiving TMS treatment. Mean: Mean, SD: Standard Deviation. The study was completed by 25 patients who received TMS treatment and 26 patients who did not.

Change over time (p): (Changes over time) Repeated measures ANOVA and Friedman test were used. Bonferroni confidence intervals and Bonferroni-corrected Nemenyi post-hoc tests were used to determine significant measurements. For non-significant parameters in the Friedman test, p-values were calculated using Bonferroni-corrected Wilcoxon tests for post-hoc comparisons.

Group difference (p): Independent sample t-test and Mann Whitney U test were used.

No significant differences were observed in the Trail Making Test (Parts A and B), Digit Span Test, Verbal Memory Processes Test, or Stroop Test scores at baseline, 1-month, or 3-month follow-ups within or between the groups. However, in the Verbal Fluency subtest, the non-TMS group showed significant improvement in the semantic test between the 1-month and 3-month follow-ups, while no significant differences were found between baseline and other follow-up points. In the TMS group, significant improvement was observed in the verbal fluency subtest between baseline and the 1-month follow-up, but no significant differences were found between other intervals. The neuropsychological test performance comparisons within and between the groups are summarized in Table 5.

## DISCUSSION

The findings of this study suggest that TMS exhibits substantial acute efficacy in treating depression in MDD patients; however, this effect appears to diminish over time during follow-ups. A study conducted in Turkey on treatment-resistant depression (TRD) reported that 63% of patients responded to TMS therapy, with 42.1% achieving remission (Akpınar et al. 2022). Similarly, the literature supports the efficacy of TMS in alleviating depressive symptoms, emphasizing the necessity of maintenance TMS for sustained benefits (Chang, 2020). Cohen et al. reported an average remission duration of 119 days following rTMS applied to the left or right DLPFC in 204 MDD patients (Cohen et al. 2009).

In this study, BAI scores, based on self-reports, showed no significant differences between the TMS and non-TMS groups at baseline or during follow-ups. However, both groups experienced significant improvement at the 1-month follow-up, particularly the TMS group, with this effect diminishing by the 3-month follow-up. This outcome aligns with the expected correlation between reductions in depression scores and anxiety scores. The observed decrease in acute treatment efficacy during follow-up was also reflected in anxiety symptoms. A study involving 697 MDD patients examining the efficacy of rTMS on anxiety symptoms found a positive correlation between reductions in anxiety disorders and improvements in depressive symptoms across three TMS protocols (Chen et al. 2019).

Cognitive functions were assessed using the Trail Making Test (Parts A and B), Stroop Test, Digit Span Test, Verbal Memory Processes Test, and Verbal Fluency Test. No statistically significant differences were observed between the groups in cognitive test performance at baseline or during follow-ups. In a meta-analysis by Martin et al. (2016), no significant differences were found between TMS and sham TMS in executive functions, attention, processing speed, visual memory, verbal memory, visuospatial memory, or working memory. The lack of improvement in cognitive functions despite improvements in depressive symptoms was attributed to rTMS not causing a general upregulation of DLPFC function. Similarly, in another study, 20 sessions of high-frequency rTMS were applied to the left DLPFC in TRD patients. Although depression scores improved, no significant improvement in cognitive functions was observed. It was suggested that rTMS might independently modulate cognitive abilities and depressive symptoms by activating different neural pathways and regions (Kedzior et al. 2012).

The dorsolateral prefrontal cortex (DLPFC), which is the primary target for transcranial magnetic stimulation (TMS), plays a critical role in several cognitive functions, including working memory, attention, and the processing of episodic information (Koechlin et al. 1999). Research suggests that repetitive transcranial magnetic stimulation (rTMS) applied to the DLPFC may stimulate cortical neurons in this region, potentially modulating neural pathways to optimize neural network efficiency in information processing. One hypothesis is that rTMS may enhance cognitive functions by promoting increased interactions between the prefrontal cortex, hippocampus, and the frontoparietal network. Additionally, rTMS could activate the precuneus, a crucial node in the default mode network (DMN), within the frontoparietal network. These changes in neural pathways are believed to contribute to improvements in cognitive abilities, such as memory, processing speed, and attention (Kim et al. 2019).

Despite these promising hypotheses, only a limited number of studies have reported improvements in working memory, cognitive flexibility, or verbal fluency, and no significant effects of rTMS on problem-solving, planning, or reasoning have been observed. A meta-analysis focusing on TMS's effects on attention found that while TMS did not enhance alertness, it did show potential for improving selective and sustained attention. In the context of learning and memory, memory complaints that commonly follow electroconvulsive therapy (ECT) have not been reported with rTMS (Guse et al. 2010).

The duration of rTMS's cognitive effects has been explored in only a few studies. While it is suggested that cognitive improvement may persist for some time due to the alleviation of depressive symptoms (Guse et al. 2010), many studies have examined cognitive improvement independently of antidepressant response (Martis et al. 2003). The biological mechanisms through which rTMS exerts cognitive-enhancing effects remain unclear, and until a better understanding of the underlying pathophysiology is achieved, these mechanisms remain speculative (Martin et al. 2016). Furthermore, methodological differences—such as the number of rTMS sessions, variations in stimulation protocols, and differences in sample characteristics—pose challenges in evaluating the true effects of rTMS on cognitive functions.

## CONCLUSION

In this study, no correlation was found between the reduction in depressive symptoms and improvements in cognitive functions. The cognitive-enhancing effects of TMS are thought to be linked to specific biological mechanisms, but these remain unclear and will require further investigation to better understand the underlying pathophysiology. Although TMS has been suggested to support neurogenesis, neuronal plasticity, and even provide neuroprotection in MDD patients, these findings need to be replicated in larger samples, with extended follow-up periods and the inclusion of neuroimaging techniques to confirm these effects.

## LIMITATIONS

This study has several limitations. These include a small sample size, participant dropout due to the COVID-19 pandemic, a wide age range in the sample, concurrent antidepressant utilization in the TMS group, absence of a sham control, baseline differences in depression scores between groups, limited cognitive function tests, and lack of consideration

for confounding factors like age, gender, education level, and treatment history. Consequently, the findings cannot be generalized. Nevertheless, the study's follow-up design adds value due to the scarcity of such studies in this field.

**Ethics Commite Approval:** This study commenced following approvals obtained from the Interventional Clinical Research Ethics Committee of Selçuk University (decision dated 29.07.2021, no: E-40209705-050.01.04-113332) and the Turkish Medicines and Medical Devices Agency (approval dated 09.09.2021, no: E-68869993-511.06-532642). Prior to participation, all individuals were informed about the study and provided written informed consent.

**Conflict of Interest:** The authors have no conflicts of interest to declare.

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**Author Contributions:** G.K.S.: Contributed to the design of the study and drafted the acquisition, analysis, manuscript and performed the statistical analyses. Z.Ç.: Contributed to the acquisition. Ö.G.: Offered critical revisions of the draft, and all authors read and approved the final manuscript.

**Data Availability Statement:** The data that support the findings of this study are not publicly available due to ethics restrictions but available from the corresponding author upon reasonable request with individual permission from local institutions ethics board.

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