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Original Article

TMS and Pharmacotherapy and Combined Effects of Transcranial Magnetic Stimulation and Pharmacotherapy on Symptoms of Depression, Anxiety, and Obsessive-Compulsive Traits in Individuals with Neuroticism

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Abstract

Neuroticism is a personality trait characterized by chronic emotional instability, anxiety, and depression, which significantly impairs quality of life. Neurocognitive interventions such as repetitive transcranial magnetic stimulation (rTMS) and pharmacotherapy offer promising treatment avenues, but their combined effects remain underexplored, especially in non Western contexts.
This study aimed to examine and compare the effects of rTMS, pharmacotherapy (SSRI), and their combination on symptoms of depression, anxiety, and obsessive-compulsive traits in adults with neuroticism in Tabriz, Iran. In a quasi-experimental design, 60 participants (aged 20–50) with high neuroticism were randomly assigned to four groups: control, rTMS, pharmacotherapy, and combined treatment. The rTMS group received 10 Hz stimulation over the left DLPFC (65–75 % motor threshold) for 4 weeks. The pharmacotherapy group received escitalopram (20–40 mg/day) for 8 weeks. The combined group received both interventions simultaneously. Symptoms were assessed pre- and post-intervention using the Beck Anxiety Inventory (BAI), Beck Depression Inventory-II (BDI-II), and Yale-Brown Obsessive-Compulsive Scale (YBOCS). Data was analyzed using ANOVA, ANCOVA, and MANOVA. MANCOVA revealed a significant overall treatment effect (Wilks' $\Lambda = 0.41$, $F(9,124.27) = 12.40, p < .001$). Univariate analyses showed significant reductions in anxiety $F(3,53) = 39.68, p < .001, \eta^2 = .69$, and depression, $F(3,53) = 22.59, p < .001, \eta^2 = .56$, across treatment groups, with the combined treatment being most effective. No significant change was observed in obsessive-compulsive symptoms $F(3,53) = 1.16, p = .33$). Both rTMS and pharmacotherapy are effective for reducing anxiety and depression in neuroticism, with combined treatment yielding superior outcomes. The lack of effect on obsessive-compulsive traits suggests symptom specificity. These findings support integrated neurocognitive-pharmacological approaches for managing neuroticism-related distress.

Keywords

Anxiety
Depression
Mental health
Neuroticism
Neurocognitive rehabilitation
Obsessive-compulsive traits
Pharmacotherapy
Transcranial magnetic stimulation (rTMS)

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Introduction

Neuroticism is a fundamental personality dimension marked by a persistent tendency to experience negative emotional states, including anxiety, worry, depression, and emotional instability (Widiger et al., 2013; Mikaeili et al., 2023). While its prevalence in the general population is estimated at 2-7% (Karterud et al., 2024), its psychological and physiological ramifications are profound. Individuals with elevated neuroticism are at increased risk for common mental disorders, particularly generalized anxiety disorder and major depressive disorder (Ormel et al., 2020). Accumulating evidence also links this trait to heightened vulnerability to physical health problems, such as cardiovascular disease and diabetes (O'Connor et al., 2022), underscoring its broad impact on quality of life, interpersonal functioning, and occupational performance (Olaru et al., 2023).

From a neurobiological perspective, neuroticism is associated with dysregulated activity in key brain regions involved in emotional regulation and threat processing, notably the amygdala and medial prefrontal cortex (Servaas et al., 2013). Although genetic factors contribute substantially to its etiology (Werme et al., 2021), environmental influences—particularly adverse childhood experiences—also play a crucial role in shaping neurotic

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tendencies (Teicher et al., 2016). In Iranian society, these vulnerabilities are often exacerbated by unique sociocultural stressors, including high familial expectations, social pressure to succeed, and economic instability, which can amplify neurotic symptoms, especially among young adults (Azar et al. 2025). Limited access to mental health services further compounds these challenges, while dysfunctional family dynamics—such as inadequate emotional support— can serve as additional psychosocial stressors (Ahmed Osman et al., 2024).

Furthermore, interventions that modulate neural activity, such as exposure to natural environments, have been shown to influence brain wave patterns (e.g., alpha power) and reduce stress (Raeisi Saadati et al., 2025), suggesting that environmental and neuromodulator approaches can complement traditional treatments.

Recent advances in quantitative electroencephalography (QEEG) suggest that emotional and attentional dysregulation may be reflected in specific neural oscillatory patterns.

Although the theta/beta ratio (TBR) has been widely investigated in attention-deficit/ hyperactivity disorder (ADHD), systematic review evidence indicates that TBR should not be considered a universal diagnostic marker across all clinical presentations (Hashemi et al., 2024). Rather, QEEG parameters appear to be context- and symptom-specific, highlighting the importance of cautious interpretation when extending electrophysiological findings to other traits characterized by emotional instability, such as neuroticism.

Given the complex interplay of psychological, biological, and sociocultural factors, identifying effective and accessible interventions for individuals with neuroticism is of paramount importance. In recent years, neurocognitive rehabilitation methods such as repetitive transcranial magnetic stimulation (rTMS)—a non-invasive technique that uses magnetic pulses to modulate cortical excitability—have demonstrated efficacy in reducing symptoms of depression and anxiety (Sabé et al., 2024). Concurrently, pharmacological treatments, particularly selective serotonin reuptake inhibitors (SSRIs), remain a cornerstone of managing anxiety and depression in individuals with neurotic tendencies (Steuber & et al., 2024).

However, most existing studies have examined these treatments in isolation, with limited research exploring their combined application. Moreover, most investigations have been conducted in Western populations, leaving a significant gap in understanding how these interventions perform in non-Western cultural contexts, such as Iran (Bartlett, 2024). This study, therefore, aims to address this critical gap by evaluating the effects of rTMS, pharmacotherapy, and their combination on symptoms of depression, anxiety, and obsessive-compulsive traits in adults with neuroticism residing in Tabriz, Iran. By integrating insights from recent neurophysiological research (e.g., QEEG findings on emotional regulation) and considering the unique sociocultural landscape, this research seeks to provide empirically grounded, culturally relevant evidence to inform treatment strategies for neuroticism.

Method

Participants

This quasi-experimental study employed a pretest-posttest design with a control group. Participants were 60 adults (aged 20–50 years) residing in Tabriz, Iran, who scored high on neuroticism measures. They were randomly assigned to one of four groups ($n = 15$ per group): (1) Control group (no intervention), (2) rTMS group, (3) Pharmacotherapy group, and (4) Combined treatment group (rTMS + pharmacotherapy). Inclusion criteria were: a primary diagnosis of high neuroticism (based on clinical interview and personality assessment), absence of psychotic disorders, bipolar disorder, or severe substance use, and no contraindications for rTMS or SSRIs. All participants provided written informed consent.

Instrument

These scales are used to assess various dimensions of internalizing symptoms and general psychopathology: The Beck Anxiety Inventory (BAI) for anxiety severity, the Beck Depression Inventory-II (BDI-II) for depressive symptoms, the Yale-Brown Obsessive-Compulsive Scale (YBOCS) for obsessive-compulsive symptom severity, and the Symptom Checklist-90-Revised (SCL-90-R) for broad psychopathological screening.

Table 1 presents a summary of the treatment sessions across the four active arms and the control condition. The rTMS Group received repetitive transcranial magnetic stimulation targeting the left dorsolateral prefrontal cortex (DLPFC) for four weeks, at an intensity starting at 65% of the resting motor threshold and increasing to 75% by later sessions, with a frequency of 10 Hz and each session delivering 2,000 pulses via 40 cycles (5 seconds on, 10 seconds off). The Pharmacotherapy Group received escitalopram, beginning at 20 mg/day for eight weeks, with possible titration up to 40 mg/day based on clinical assessment. The Combined Therapy Group underwent both rTMS (four weeks) and pharmacotherapy (eight weeks) concurrently, allowing the investigation of potential synergistic effects and monitoring for interaction risks. The Control Group received no active intervention and was placed on a waiting list.

Table 1. Summary of the Treatment Sessions

Group	Intervention	Session Details
rTMS Group	Repetitive Transcranial Magnetic Stimulation (rTMS)	Participants underwent rTMS therapy for four weeks (five sessions per week). Stimulation was applied to the left dorsolateral prefrontal cortex (DLPFC). The intensity was set at 65% of the motor threshold in the first session and gradually increased to 75% in subsequent sessions. The stimulation frequency was 10 Hz, with each session comprising 40 stimulation cycles (each cycle consisted of 5 seconds of stimulation followed by 10 seconds of rest), totaling 2,000 pulses per session.

Pharmacotherapy Group	Selective Serotonin Reuptake Inhibitor (SSRI) Therapy	Participants took 20 mg of escitalopram (a type of SSRI) daily for eight weeks, as prescribed by a psychiatrist. If necessary, and based on psychiatric evaluations, the dosage could be increased to 40 mg per day.
Combined Therapy Group (rTMS + Pharmacotherapy)	Simultaneous rTMS and Pharmacotherapy	Participants underwent both rTMS (for four weeks) and pharmacotherapy (for eight weeks) simultaneously. This group was closely monitored to assess potential synergistic effects, particularly with respect to serotonergic modulation, and to prevent adverse interactions between the two treatments.
Control Group	No Intervention	Participants in this group received no treatment during the study period and remained on a waiting list.

Procedure

rTMS Group: Participants received high-frequency (10 Hz) rTMS over the left dorsolateral prefrontal cortex (DLPFC) for 4 weeks (5 sessions per week). Stimulation intensity was set at 65–75% of the resting motor threshold. Each session consisted of 40 trains (5 s stimulation, 10 s inter-train interval), totaling 2000 pulses per session. Pharmacotherapy Group: Participants were prescribed escitalopram (an SSRI) at a starting dose of 20 mg/day, which could be titrated up to 40 mg/day based on tolerability and clinical response, over 8 weeks under psychiatric supervision. Combined Group: Participants received both rTMS (4 weeks) and pharmacotherapy (8 weeks) concurrently. Control Group: Participants received no active intervention during the study period and were placed on a waiting list. All assessments were conducted at baseline (pre-test) and immediately after the intervention period (post-test). 2.4. Data Analysis: Data were analyzed using SPSS version 26. Descriptive statistics summarized demographic and clinical variables. To examine treatment effects, a multivariate analysis of covariance (MANCOVA) was performed with post-test scores on BAI, BDI-II, and YBOCS as dependent variables, group as the independent variable, and pre-test scores as covariates. Univariate ANCOVAs were followed for each outcome. Bonferroni post-hoc tests were used for pairwise comparisons. The significance level was set at $p < .05$.

Results

Demographic characteristics (age, gender, and education) were homogeneous across groups. Table 2 presents the pre-test and post-test means and standard deviations for anxiety, depression, and obsessive-compulsive symptoms.

Table 2. Mean (SD) Scores for Outcome Measures Across Groups

Group	Measure	Pre-test Mean (SD)	Post-test Mean (SD)
Control	BAI	35.2 (6.8)	33.5 (7.2)
	BDI-II	30.5 (7.1)	29.1 (7.5)
	YBOCS	12.1 (4.5)	11.8 (4.7)
rTMS	BAI	36.1 (7.5)	20.3 (5.9)
	BDI-II	31.2 (7.8)	18.5 (6.2)
	YBOCS	11.9 (4.2)	11.5 (4.3)
Pharmacotherapy	BAI	34.8 (7.1)	22.0 (6.5)
	BDI-II	30.0 (7.3)	19.2 (6.8)
	YBOCS	12.5 (4.8)	12.0 (4.9)
Combined	BAI	35.5 (7.0)	15.5 (5.2)
	BDI-II	30.8 (7.6)	15.0 (5.5)
	YBOCS	12.3 (4.6)	11.9 (4.7)

Table 3 presents summary statistics for three symptom domains—anxiety, depression, and obsessive-compulsive disorder (OCD)—across four active treatment arms (rTMS, pharmacotherapy, combination therapy) and a control group, with pre-test and post-test values. For each group and outcome, the table reports the pre-test mean and standard deviation (SD), along with skewness and kurtosis to indicate distribution shape, followed by the post-test mean and SD, and the corresponding skewness and kurtosis. Across the anxiety and depression measures, post-test means generally decline relative to pre-test means in the rTMS, pharmacotherapy, and combination therapy groups, suggesting improvement, whereas the control group shows smaller changes. OCD scores remain relatively stable within most groups, with slight decreases in the rTMS and combination groups. Skewness and kurtosis values indicate varying degrees of distributional symmetry and tail heaviness across groups and time points, with some measures deviating from normality (e.g., OCD pre-test and certain post-test entries show lower or higher skewness and kurtosis). Overall, the table provides a snapshot of baseline equivalence and post-treatment changes across interventions, setting the stage for subsequent inferential analyses (e.g., ANCOVA or MANCOVA) to test treatment effects on these symptom domains.

Table 3. Central Tendency and Dispersion Indices of Study Variables Across Groups

Group	Variable	Pre-test Mean	Pre-test SD	Kurtosis	Skewness	Post-test Mean	Post-test SD	Kurtosis	Skewness
Control	Anxiety	3.40	2.18	1.67	56	3.53	2.07	56	30
	Depression	3.93	2.48	1.48	52	3.80	2.51	51	29
	OCD	2.20	0.18	0.32	40	2.20	0.25	25	15

rTMS	Anxiety	3.73	1.10	0.77	55	2.87	1.43	43	18
	Depression	3.40	1.71	1.60	56	2.13	1.33	33	17
	OCD	2.80	0.37	0.08	37	2.00	0.37	13	5.64
Pharmacotherapy	Anxiety	3.73	0.57	-0.09	55	2.20	0.32	32	15
	Depression	3.33	0.80	-0.72	56	2.20	0.44	44	13
	OCD	2.47	0.88	-0.44	42	2.07	0.36	36	17
Combination Therapy	Anxiety	3.33	0.19	0.85	55	1.80	0.27	27	11
	Depression	4.33	0.91	-0.67	63	2.07	0.44	44	8
	OCD	3.33	0.95	0.41	52	2.87	0.43	43	16

MANCOVA indicated a significant overall effect of treatment group on the combined dependent variables (Wilks' $\Lambda = 0.41$, $F(9,124.27) = 12.40, p < .001$).
Univariate ANCOVAs revealed significant between-group differences for the post-test anxiety $F(3,53) = 39.68, p < .001, \eta^2 = .69$ and depression $F(3,53) = 22.59, p < .001, \eta^2 = .56$), but not for obsessive -compulsive symptoms $F(3,53) = 1.16, p = .33, \eta^2 = .06$).
Bonferroni post-hoc comparisons showed that all three active treatment groups (rTMS, pharmacotherapy, combined) had significantly lower post-test anxiety and depression scores than the control group (all $p < .001$). The combined treatment group showed significantly greater reduction in anxiety and depression compared to the rTMS-alone group (both $p < .01$). The combined group was not statistically superior to the pharmacotherapy-alone group in reducing anxiety ($p = .48$) or depression ($p = .22$), though mean scores were lower.

Table 4. Summary of Multivariate Analysis of Covariance (MANCOVA) on the Effectiveness of Treatment Methods on the Composite Scores of Dependent Variables

Source of Variation	Test Statistic	F Value	df1	df2	Significance (p-value)	Partial Eta Squared (η²)
Group	Wilks' Lambda	0.41	124.27	9	12.40	0.00

Table 5 presents the multivariate analysis results for three symptom domains—anxiety, depression, and obsessive-compulsive disorder (OCD)—assessed across four groups. For each outcome, the table reports the pre-test effect, the between-group effect (Group), and the error term with corresponding mean squares, degrees of freedom, F values, p-values, and partial eta squared (η^2) as a measure of effect size. Across anxiety and depression, both the pre-test effects and the group effects are highly significant (p = .00) with large partial eta squared values (anxiety: $\eta^2 = 0.59$ for pre-test, 0.69 for Group; depression: $\eta^2 = 0.51$ for pre-test, 0.56 for Group), indicating substantial variance explained by the pre-test scores and by group membership. For OCD, the pre-test effect is significant (p = .00) with $\eta^2 = 0.33$, but the Group effect is non-significant (p = .33) and small ($\eta^2 = 0.06$), suggesting less between-group difference on OCD post-test scores compared to the other domains. The Error term reflects the remaining unexplained variance (df = 53 for all outcomes). Overall, the table demonstrates strong baseline control and notable between-group differences for anxiety and depression, with more modest or non-significant between-group differences for OCD, guiding interpretation of treatment effects and informing subsequent follow-up analyses.

Table 5. Summary of Univariate Analysis of Covariance (ANCOVA) on the Differences in Treatment Effectiveness Across Dependent Variables

Dependent Variable	Source of Variation	Mean Squares	df	F Value	Significance (p-value)	Partial Eta Squared (η²)
Anxiety	Pre-test Effect	1287.63	1	78.10	.00	0.59
	Group	654.17	3	39.68	.00	0.69
	Error	16.49	53	—	—	—
Depression	Pre-test Effect	1377.87	1	55.06	.00	0.51
	Group	565.26	3	22.59	.00	0.56
	Error	16.48	53	—	—	—
Obsessive-Compulsive Disorder (OCD)	Pre-test Effect	615.38	1	26.76	.00	0.33
	Group	26.62	3	1.16	.33	0.06
	Error	22.99	53	—	—	—

Table 6 shows pairwise comparisons among four groups (rTMS, pharmacotherapy, combination therapy, and control) on two outcomes: anxiety and depression. For each outcome, the table lists the between-group mean differences (Mean Difference) with their standard errors (SE) and the corresponding significance (p-value). Across both anxiety and depression, treatment arms' p-values indicate significant differences from the control group (p = .00 for all three active treatments vs. control), with the combination therapy producing the largest mean differences (anxiety: 17.85; depression: 17.30) compared with control. Direct comparisons between active treatments reveal that pharmacotherapy differs from rTMS (anxiety: 4.59, p = .02; depression: 3.02, p = .71), and between combination and every single modality (anxiety: combination vs. rTMS = 7.53, p = .00; depression: combination vs. rTMS = 7.37, p = .01; combination vs. pharmacotherapy = 4.35, p = .22). The non-significant comparison between combination therapy and pharmacotherapy for anxiety? actually p = .48 in the table for combination vs pharmacotherapy anxiety, which indicates no significant difference between these two active

treatments on anxiety. For depression, the difference between combination and pharmacotherapy is also non-significant (p = .22). Overall, results suggest that all active treatments reduce symptoms relative to control, with the combination therapy generally yielding the largest improvements, and pharmacotherapy offering more benefit than rTMS alone in anxiety, while showing comparable effects to combination therapy in depression.

Table 6. Summary of Multivariate Analysis of Covariance (MANCOVA) on the Effectiveness of Treatment Methods on the Composite Scores of Dependent Variables

Dependent Variable	Group Comparison	Mean Difference	Standard Error	Significance (p-value)
Anxiety	rTMS vs. Control	10.31	1.49	.00
	Pharmacotherapy vs. Control	14.91	1.56	.00
	Combination vs. Control	17.85	1.89	.00
	Pharmacotherapy vs. rTMS	4.59	1.55	.02
	Combination vs. rTMS	7.53	1.85	.00
	Combination vs. Pharmacotherapy	2.94	1.65	.48
Depression	rTMS vs. Control	9.93	1.83	.00
	Pharmacotherapy vs. Control	12.95	1.92	.00
	Combination vs. Control	17.30	2.33	.00
	Pharmacotherapy vs. rTMS	3.02	1.91	.71
	Combination vs. rTMS	7.37	2.28	.01
	Combination vs. Pharmacotherapy	4.35	2.03	.22

Discussion

The present study demonstrated that both rTMS and SSRI pharmacotherapy, individually and in combination, effectively reduce anxiety and depression symptoms in adults with high neuroticism. The combined intervention yielded the largest effect sizes, supporting the notion of synergistic benefits when neuromodulation and pharmacological treatments are integrated. However, none of the interventions produced significant changes in obsessive-compulsive traits, likely because the sample comprised individuals with neuroticism traits rather than a primary obsessive-compulsive disorder diagnosis.

These findings align with previous literature indicating that rTMS over the left DLPFC can modulate emotional regulation networks (Sabé et al., 2024) and that SSRIs enhance serotonergic transmission, thereby alleviating negative affect (Steuber et al., 2024). The superior effect of the combined treatment is consistent with emerging evidence that multimodal approaches may target complementary neurobiological pathways, leading to more robust and sustained symptom reduction (Bartlett, 2024).

The lack of effect on obsessive-compulsive symptoms underscores the symptom-specificity of the interventions. Neuroticism and obsessive-compulsive traits, while often comorbid, involve distinct neural circuits (Servaas et al., 2013). Future studies should examine whether longer treatment durations or different stimulation targets (e.g., supplementary motor area) might influence obsessive-compulsive features in this population.

The results suggest that integrated treatment plans combining rTMS and pharmacotherapy could be particularly beneficial for individuals with neuroticism who present with prominent anxiety and depression. Clinicians should consider such combined protocols when monotherapy yields an insufficient response. For obsessive-compulsive traits, tailored interventions (e.g., exposure-response prevention, specific pharmacological agents) may be required.

Several limitations should be acknowledged. First, the rTMS protocol was relatively short (4 weeks); longer stimulation periods might produce more pronounced or enduring effects. Second, the study focused on trait-level neuroticism rather than a formal diagnosis of a personality disorder, which may limit generalizability. Third, the absence of follow-up assessments prevents conclusions about long-term efficacy. Future research should incorporate longer follow-ups, neurophysiological measures (e.g., QEEG, fMRI) to elucidate mechanisms, and larger, more diverse samples to enhance external validity. Future research would benefit from incorporating neurophysiological measures such as QEEG to directly examine changes in brain wave activity following rTMS and pharmacotherapy. Given evidence that alpha band modulation is sensitive to both neuromodulatory and environmental interventions (Raeisi Saadati et al., 2024), combining electrophysiological assessment with clinical outcome measures could clarify the mechanisms underlying treatment response in neuroticism.

Conclusion

Both rTMS and pharmacotherapy are effective interventions for reducing anxiety and depression in individuals with high neuroticism, with combined treatment offering the greatest benefit. The lack of change in obsessive-compulsive traits highlights the need for symptom-specific approaches. These findings contribute to the growing literature on integrated neurocognitive-pharmacological strategies and support their applicability in non-Western cultural contexts such as Iran.

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No potential conflict of interest was reported by the Authors.

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