NÖRO PSİKİYATRİ ARŞİVİ
ARCHIVES OF NEUROPSYCHIATRY

RESEARCH ARTICLE

Article No: 87

Arch Neuropsychiatry 2026;63:555–566
https://doi.org/10.29399/npa.29364

Neurocognitive Performance and Peripheral microRNA Expression in Schizophrenia and Obsessive-Compulsive Disorder: A Comparative Study

Emel UYSAL[1], Demet SAĞLAM AYKUT[1], Evrim ÖZKORUMAK KARAGÜZEL[1], Alper Han ÇEBİ[2], Mücahit GÖKTAŞ[3]

[1]Department of Psychiatry, Faculty of Medicine, Karadeniz Technical University, Trabzon, Türkiye
[2]Department of Medical Genetics, Faculty of Medicine, Karadeniz Technical University, Trabzon, Türkiye
[3]Private Practice, Kocaeli, Türkiye

ABSTRACT

Introduction: Schizophrenia and obsessive-compulsive disorder (OCD) are two chronic psychiatric disorders associated with partially overlapping cognitive impairments and neurobiological processes. The aim of this study was to comparatively examine neurocognitive performance and the expression levels of selected peripheral microRNAs (miR-134, miR-137, miR-150, miR-502, and miR-1306) in patients with schizophrenia and OCD relative to healthy controls, and to exploratorily assess potential heterogeneity within OCD by considering autogenous and reactive subtypes.

Methods: This cross-sectional study included 20 patients with schizophrenia, 20 patients with OCD, and 20 healthy controls matched for age, sex, and years of education. All participants underwent a standardized neurocognitive test battery assessing executive functions, attention, processing speed, and verbal memory. Plasma miRNA levels were measured from peripheral blood samples using quantitative real-time polymerase chain reaction (qRT-PCR). Within the OCD group, autogenous and reactive obsession subtypes were additionally analyzed.

Results: Both schizophrenia and OCD groups showed poorer performance than healthy controls in measures of attention, processing speed, and executive functioning. Verbal memory impairment was more widespread in the schizophrenia group, whereas it was more limited in the OCD group. After controlling for depression and anxiety levels as covariates, many neurocognitive differences remained significant. miR-134 levels were lower in the OCD group, while miR-502 levels were lower in both the OCD and schizophrenia groups compared with healthy controls. Exploratory analyses yielded limited findings suggesting a possible association between miR-502 and executive function measures.

Conclusion: The findings suggest a more widespread pattern of neurocognitive impairment in schizophrenia, whereas OCD appears to involve a more selective cognitive impairment characterized predominantly by executive dysfunction, with verbal memory difficulties potentially related to executive processes. Alterations in selected miRNA levels may provide exploratory clues regarding shared and distinct peripheral molecular processes, while the potential relationship between miR-502 and executive functioning warrants confirmation in larger and longitudinal studies.

Keywords: microRNAs, neuropsychological tests, obsessive-compulsive disorder, schizophrenia

Cite this article as: Uysal E, Sağlam Aykut D, Özkorumak Karagüzel E, Çebi AH, Göktaş M. Neurocognitive Performance and Peripheral microRNA Expression in Schizophrenia and Obsessive-Compulsive Disorder: A Comparative Study. Arch Neuropsychiatry 2026;63:555–566. doi: 10.29399/npa.29364

INTRODUCTION

Schizophrenia and obsessive–compulsive disorder (OCD) are two important neuropsychiatric disorders associated with a chronic course and marked functional impairment. Clinical observations and epidemiological findings suggest that there may be a partial overlap between these two disorders not only at the level of comorbidity but also in terms of the underlying neurobiological processes (1). The observation of obsessive–compulsive symptoms in patients with schizophrenia and psychotic features in patients with OCD, as well as the reported comorbidity rates exceeding the expected lifetime prevalence, support this view (1).

In schizophrenia, widespread and persistent cognitive impairments in attention, working memory, executive functions, and processing speed are considered among the core features of the disorder (2). In OCD, although cognitive impairment is generally milder, selective difficulties, particularly in executive functions, have been reported. It has been suggested that impairments in cognitive flexibility, response inhibition, set shifting, and error monitoring processes may be associated with dysfunction in fronto-striatal circuits (3). In addition, more limited but consistent impairments in verbal memory, visuospatial memory, and reasoning have been reported (3). These findings suggest that the patterns of cognitive impairment observed in schizophrenia and OCD may be associated with partially overlapping frontal and cortico-striatal network dysfunctions (4). Therefore, the comparative examination of neurocognitive performance in patients with schizophrenia and OCD may contribute to a better understanding of both shared cognitive mechanisms and possible selective cognitive weaknesses specific to OCD.

Highlights

- **Neurocognitive impairment was more widespread in schizophrenia than OCD.**
- **OCD showed an intermediate cognitive profile between controls and schizophrenia.**
- **miR-134 and miR-502 levels differed across the study groups.**
- **miR-502 levels may relate to executive function measures.**

Correspondence Address: Emel Uysal, Department of Psychiatry, Faculty of Medicine, Karadeniz Technical University, Trabzon, Türkiye • **E-mail:** dremelkorkmaz@gmail.com
Received Date: 19.02.2026, **Accepted Date:** 29.05.2026, **Publication Date:** 11.09.2026

The heterogeneous clinical structure of OCD suggests that cognitive and biological processes do not exhibit a uniform pattern across all patients. In the model proposed by Lee and Kwon, obsessions are divided into two subtypes: autogenous and reactive (5). Autogenous obsessions are characterized by thoughts emerging without an external trigger and involving intense ego-dystonicity, whereas reactive obsessions are characterized by thoughts triggered by external stimuli and involving realistic threat perception (5). Previous studies have shown that these subtypes may partially differ in terms of clinical and cognitive characteristics (6,7). However, the biological correlates of this distinction have not yet been sufficiently elucidated.

In recent years, molecular studies have shown that complex neuropsychiatric disorders such as schizophrenia and OCD may also be associated with differences in gene regulatory processes. In this context, microRNAs (miRNAs) have become a focus of interest due to their regulatory roles in neuronal development, synaptic plasticity, and neuroinflammatory processes (8). In schizophrenia, miRNA research has particularly shown that molecules such as miR-137 may be associated with genetic risk for the disorder (9). In contrast, data regarding miRNA profiles in OCD are relatively limited and are mostly based on exploratory studies conducted with small samples.

In the present study, five miRNAs previously reported to be associated with synaptic plasticity, neurodevelopment, and neuroinflammation were examined using an exploratory approach. miR-134 is associated with synaptic development and plasticity (10); miR-137 is linked to neuronal maturation processes and has been identified as a genetic risk locus for schizophrenia (9); miR-150 has been associated with immune response and neuroinflammatory processes (11); miR-502 has been identified as one of the candidate miRNAs found to be increased in the plasma of patients with schizophrenia (12) and has been shown to target the *GABRA1* gene, which may have an effect on inhibitory neurotransmission (13); miR-1306 has been associated with neuroprotective and neuroplastic processes (14).

The aim of this study was to comparatively evaluate selected miRNA levels and neurocognitive performance in patients with schizophrenia and OCD in comparison with healthy controls. In addition, whether there are differences between autogenous and reactive obsession subtypes in the OCD group in terms of these biological and cognitive measures will be explored. Through this approach, it is aimed to provide preliminary findings regarding the shared and distinct patterns between schizophrenia and OCD by jointly addressing miRNA profiles and neurocognitive functions.

METHODS

Study Design and Participants

This study is a cross-sectional and observational study conducted at Karadeniz Technical University Faculty of Medicine Psychiatry Outpatient Clinic. Participants were selected using a consecutive sampling method from patients followed in our outpatient clinic between January 2017 and December 2018. All participants were evaluated using the Structured Clinical Interview for DSM-5 Clinical Version (SCID-5-CV), a structured clinical interview based on DSM-5 diagnostic criteria.

The study included 20 patients with schizophrenia, 20 patients with OCD, and 20 healthy controls similar in terms of age, sex, and educational level. Healthy controls were selected from university staff and volunteers from the surrounding community, and the absence of any psychiatric disorder was confirmed using the SCID-5-CV.

Individuals aged between 18 and 65 years who provided written informed consent and whose diagnosis of schizophrenia or OCD was confirmed using the SCID-5-CV were included in the study. Individuals with an additional psychiatric diagnosis other than schizophrenia or OCD, those presenting both autogenous and reactive obsession types in the OCD group, those with a history of dementia, central nervous system disease, or head trauma associated with loss of consciousness, those with intellectual disability according to clinical evaluation and developmental history, and those with a lifetime history of alcohol or substance use disorder were excluded from the study.

All patient groups had been clinically stable under the same medication regimen for at least 8 weeks prior to assessment. Clinical stability was defined as the absence of a significant increase in symptoms during follow-up assessments using the Positive and Negative Syndrome Scale (PANSS) in patients with schizophrenia and the Yale-Brown Obsessive-Compulsive Scale (Y-BOCS) in patients with OCD. In the control group, psychotropic medication use and the presence of a first-degree relative with a history of psychotic disorder or OCD were accepted as exclusion criteria.

Clinical Assessment Instruments

Depression and anxiety levels were assessed using the Beck Depression Inventory (BDI) (15,16) and the Beck Anxiety Inventory (BAI) (17,18), respectively.

The type and severity of OCD symptoms were evaluated using the Yale-Brown Obsessive-Compulsive Scale (Y-BOCS) (19,20).

In patients with OCD, autogenous and reactive obsession subtypes were classified based on the model proposed by Lee and Kwon (5). Based on the Y-BOCS symptom checklist, aggressive, religious, and sexual obsessions were included in the autogenous subtype, whereas contamination, doubt, symmetry, somatic, and hoarding obsessions were included in the reactive subtype. According to this classification, 9 patients were included in the autogenous OCD group and 11 patients in the reactive OCD group.

Neurocognitive Assessment

All neurocognitive tests were administered by the same researcher in a quiet environment using a standard test order.

Executive functions and conceptual flexibility were assessed using the Wisconsin Card Sorting Test (WCST) (21,22).

Selective attention and response inhibition were evaluated using the interference condition of the Stroop Test (Stroop V); only the reaction times of correct responses were taken into account in the analyses (23,24).

Attention, psychomotor speed, and cognitive flexibility were measured using the Trail Making Test (TMT-A and TMT-B) (25,26).

Verbal learning and memory processes were assessed using the Rey Auditory Verbal Learning Test (RAVLT) (27,28).

miRNA Expression Analysis

Peripheral venous blood samples (3 mL) were collected into EDTA-containing tubes and processed on the same day. Plasma samples were stored at -80°C until analysis. All samples were processed under the same laboratory conditions and evaluated in comparable analysis sessions. Total miRNA isolation was performed using the Qiagen miRNeasy Serum/Plasma Kit in accordance with the manufacturer's instructions.

miRNA expression levels were measured using quantitative real-time polymerase chain reaction (qRT-PCR) on the Rotor-Gene Q real-time PCR system using Qiagen miScript Primer Assays and miScript® SYBR® Green PCR Kit Master Mix. In order to control RNA isolation and amplification efficiency, synthetic Caenorhabditis elegans miRNA (cel-miR-39) was added as an exogenous spike-in control and used in the normalization of miRNA expression levels.

Raw Ct values were obtained through the Qiagen GeneGlobe Data Analysis Center portal. Relative miRNA expression levels were calculated

based on normalized Ct values and exported for subsequent statistical analyses. Normalized miRNA expression values were used in subsequent statistical analyses.

Ethical Approval

The study was approved by the Karadeniz Technical University Faculty of Medicine Scientific Research Ethics Committee (Decision No: 24237859/178). All participants were informed verbally and in writing about the study, and written informed consent was obtained in accordance with the principles of the Declaration of Helsinki.

Statistical Analysis

The distribution of continuous variables was evaluated using skewness and kurtosis coefficients; values within the range of ±2.5 were accepted as indicators of normal distribution. Missing data in BDI and BAI scores were handled using the Expectation-Maximization (EM) algorithm.

For between-group comparisons, one-way analysis of variance (ANOVA) was applied for normally distributed variables. Welch ANOVA was used when the assumption of homogeneity of variances was violated. Correction for multiple comparisons was applied in post hoc pairwise comparisons. Duncan's test was used in parametric analyses in which homogeneity of variance was met, whereas Tamhane's T2 test was used when homogeneity was not met. Variables not showing normal distribution were compared using the Kruskal-Wallis H test; when significance was detected, Bonferroni-corrected Dunn post hoc analyses were applied. In order to evaluate the potential confounding effect of depressive and anxiety symptoms, BDI and BAI scores were included in the model as covariates, and group differences in neurocognitive test scores were examined using Quade's ANCOVA, a non-parametric covariance analysis approach, since the assumption of normal distribution was not met. Bonferroni-corrected post hoc analyses were applied to control for the effect of multiple testing in pairwise group comparisons.

Categorical variables were compared using Fisher's exact test due to low expected cell frequencies. Pairwise comparisons between two independent groups were performed using the independent samples *t* test for normally distributed variables and the Mann-Whitney *U* test for non-normally distributed variables.

Relationships between miRNA expression levels and neuropsychological test scores were examined using Spearman's rank correlation coefficient depending on distribution characteristics. For variables in which significant correlations were detected, exploratory linear regression analyses were performed to evaluate the contribution of miRNA levels to cognitive performance measures. These regression analyses were not intended for confirmatory modeling and were considered hypothesis-generating due to the small sample size.

All statistical tests were conducted as two-tailed, and the statistical significance level was set at $p < 0.05$. Statistical analyses were performed using IBM Statistical Package for Social Sciences (SPSS) program version 26 (IBM Corp., Armonk, NY, USA).

RESULTS

Sociodemographic and Clinical Characteristics

The groups did not show significant differences in terms of age, sex, marital status, duration of education, educational level, or income status (all $p > 0.05$) (Table 1). When clinical variables were examined, the duration of illness was found to be longer in the OCD group compared with the schizophrenia group ($p=0.006$), whereas the age at illness onset was found to be lower ($p=0.001$) (Table 1). Beck Depression Inventory (BDI) scores were found to be significantly higher in the OCD group compared with the control group ($p=0.002$); however, the scores of the schizophrenia group did not differ statistically from those of the OCD and control groups. No significant difference was found between the groups in terms of BAI scores ($p=0.073$).

Neurocognitive Test Results

TMT-A and TMT-B durations were found to be significantly longer in the OCD and schizophrenia groups compared with the control group ($p=0.004$ and $p=0.015$, respectively) (Table 2). A significant difference was found

Table 1. Sociodemographic and clinical characteristics of the study groups

	Control (n=20)	OCD (n=20)	SCH (n=20)	p	Effect size
Age	22 (18–41)	22 (18–40)	23.5 (19–42)	0.335[h]	0.003[e]
Sex					
Female	11 (55)	11 (55)	11 (55)	1.000[x]	0.000[v]
Male	9 (45)	9 (45)	9 (45)		
Marital status					
Single	16 (80)	18 (90)	18 (90)	0.713[x]	0.139[v]
Married	4 (20)	2 (10)	2 (10)		
Years of education	12 (5–17)	12 (5–18)	12 (5–16)	0.999[h]	0.000[e]
Education level					
Primary education	5 (25)	5 (25)	5 (25)	1.000[x]	0.042[v]
High school	11 (55)	11 (55)	10 (50)		
Bachelor's degree	4 (20)	4 (20)	5 (25)		
Monthly income					
Below minimum wage	14 (70)	18 (90)	17 (85)	0.344[x]	0.220[v]
Above minimum wage	6 (30)	2 (10)	3 (15)		
Duration of illness (years)	-	8.5 (2–27)	3 (1–20)	**0.006[m]**	**0.761[d]**
Age at illness onset	-	15.25±5.66	20.3±2.68	0.001[t]	1.140[d]
BDI	5 (2–11.22)[a]	18 (2–30)[b]	10.61 (2–21)[ab]	**0.002[h]**	
BAI	4.37 (2–9.13)	12 (0–36)	9 (1–22)	0.073[h]	

h: Kruskal-Wallis H test; x: Fisher's exact test; t: independent samples t test; m: Mann-Whitney U test; e: eta squared; d: Cohen's d value; v: Cramer's V coefficient; data are presented as median (min–max) or mean ± SD; OCD: obsessive-compulsive disorder; SCH: schizophrenia; BDI: Beck Depression Inventory; BAI: Beck Anxiety Inventory.

between the groups in terms of Stroop Test Part V durations (p=0.008); the longest durations were observed in the schizophrenia group, whereas the shortest durations were observed in the control group.

Rey Auditory Verbal Learning Test (RAVLT) learning (A1–5), short-term recall (A6), and delayed recall (A7) scores were found to be lowest in the schizophrenia group and highest in the control group (all p <0.05) (Table 2).

Significant differences were observed between the groups in WCST parameters. The total number of correct responses was highest in the control group and lowest in the schizophrenia group, whereas the total number of errors was lowest in the control group and highest in the schizophrenia group (p <0.001). Perseverative responses and error counts showed an increasing pattern across groups (all p <0.05). Conceptual level response count and the number of completed categories were found to

Table 2. Comparison of miRNA levels and neuropsychological test scores between groups

	Control (n=20)	OCD (n=20)	SCH (n=20)	p	η^2
miR-134	30.71 (21.83–33.64)[a]	27.68 (17.25–32.21)[b]	27.44 (25.27–34.89)[ab]	**0.004[h]**	0.163
miR-137	36.15 (20.09–38.93)	36.01 (23.34–39.89)	35.01 (27.7–39.75)	0.281[h]	0.009
miR-150	32.19 (22.45–38.75)	27.53 (20.24–31.7)	27.97 (19.06–33.02)	0.073[h]	0.057
miR-502	24.74±1.67[a]	22.59±2.44[b]	21.85±1.98[b]	**<0.001[f]**	0.273
miR-1306	28.14±0.88	28.04±2.08	29.18±1.85	0.083[w]	0.090
TMT-A (sec)	20 (13–80)[a]	31 (14–71)[b]	41.5 (24–112)[b]	**0.004[h]**	0.159
TMT-B (sec)	48 (30–150)[a]	74.5 (38–245)[b]	91.5 (56–205)[b]	**0.015[h]**	0.112
TMT B-A (sec)	26 (16–70)	43 (18–174)	57 (24–105)	0.062[h]	0.062
Stroop V (sec)	17 (11–33)[a]	23 (15–43)[b]	37 (19–67)[c]	**0.008[h]**	0.136
RAVLT A1	10 (5–11)	8 (4–11)	5 (3–10)	0.067[h]	0.060
RAVLT A1–5	64.5 (44–68)[a]	57.5 (39–69)[b]	31.5 (14–56)[c]	**0.007[h]**	0.806
RAVLT B1	7 (4–13)	7 (3–13)	4 (2–5)	0.388[h]	0.140
RAVLT A6	14 (8–15)[a]	12 (7–15)[b]	6 (2–11)[c]	**0.026[h]**	0.093
RAVLT A7	14 (9–15)[a]	11 (8–14)[b]	5.5 (2–10)[c]	**0.005[h]**	0.153
RAVLT recognition list	15 (11–15)	14.5 (10–15)	11.5 (6–15)	0.087[h]	0.051
WCST total correct responses	54.25±2.15[a]	47±5.27[b]	43±5.52[c]	**<0.001[f]**	0.521
WCST total errors	9.75±2.15[a]	17±5.27[b]	21±5.52[c]	**<0.001[f]**	0.521
WCST perseverative responses	5 (3–9)[a]	8.5 (3–14)[b]	11 (3–18)[c]	**0.005[h]**	0.150
WCST perseverative errors	5.05±1.43[a]	8.05±2.68[b]	9.55±3.53[c]	**<0.001[w]**	0.338
WCST non-perseverative errors	4.5 (3–9)[a]	9 (3–16)[b]	11.5 (4–22)[c]	**0.001[h]**	0.231
WCST conceptual level responses	52.3±3.63[a]	43.8±7.49[b]	37.45±8.48[c]	**<0.001[w]**	0.453
WCST completed categories	4 (2–5)[a]	3 (2–5)[b]	2 (0–3)[c]	**0.018[h]**	0.105
WCST trials to complete first category	11 (11–21)[a]	12.5 (11–32)[b]	25 (10–65)[c]	**0.004[h]**	0.158
WCST failure to maintain set	0 (0–3)	0 (0–3)	1 (0–4)	0.095[h]	0.047
WCST learning-to-learn score	0 (-4.37–3.32)[a]	-0.47 (-16.73–13.64)[b]	-6.19 (-24.64 - -3.18)[c]	**0.006[h]**	0.147

f: One-way ANOVA; w: Welch ANOVA; h: Kruskal-Wallis H test; η^2: effect size; a-b: there is no statistically significant difference between groups sharing the same letter; data are presented as median (min–max) or mean ± SD; miRNA: microRNA; TMT: Trail Making Test; RAVLT: Rey Auditory Verbal Learning Test; WCST: Wisconsin Card Sorting Test.

Table 2.1. Comparison of neuropsychological test scores between groups after controlling for BDI and BAI scores as covariates

	Control (n=20)	OCD (n=20)	SCH (n=20)	F	p	η^2
TMT-A (sec)	20 (13–80) [a]	31 (14–71) [b]	41.5 (24–112) [b]	20.134	**<0.001**	0.423
TMT-B (sec)	48 (30–150) [a]	74.5 (38–245) [b]	91.5 (56–205) [b]	19.247	**<0.001**	0.412
TMT B-A (sec)	26 (16–70) [a]	43 (18–174) [b]	57 (24–105) [b]	14.144	**<0.001**	0.340
Stroop V (sec)	17 (11–33) [a]	23 (15–43) [b]	37 (19–67) [c]	25.463	**<0.001**	0.481
RAVLT A1	10 (5–11) [a]	8 (4–11) [a]	5 (3–10) [b]	16.489	**<0.001**	0.375
RAVLT A1–5	64.5 (44–68) [a]	57.5 (39–69) [b]	31.5 (14–56) [c]	45.259	**<0.001**	0.622
RAVLT B1	7 (4–13) [a]	7 (3–13) [a]	4 (2–5) [b]	26.106	**<0.001**	0.514
RAVLT A6	14 (8–15) [a]	12 (7–15) [b]	6 (2–11) [c]	48.091	**<0.001**	0.636
RAVLT A7	14 (9–15) [a]	11 (8–14) [b]	5.5 (2–10) [c]	59.845	**<0.001**	0.685
RAVLT Recognition List	15 (11–15) [a]	14.5 (10–15) [b]	11.5 (6–15) [c]	20.750	**<0.001**	0.430
WCST Total Correct Responses	54.5 (50–58) [a]	47 (36–58) [b]	43.5 (28–53) [b]	32.443	**<0.001**	0.541
WCST Total Errors	9.5 (6–14) [a]	17 (6–28) [b]	20.5 (11–36) [b]	32.443	**<0.001**	0.541
WCST Perseverative Responses	5 (3–9) [a]	8.5 (3–14) [ab]	11 (3–18) [b]	9.470	**<0.001**	0.256
WCST Perseverative Errors	5 (3–8) [a]	8.5 (3–12) [ab]	11 (3–14) [b]	12.437	**<0.001**	0.311
WCST Non-Perseverative Errors	4.5 (3–9) [a]	9 (3–16) [b]	11.5 (4–22) [b]	27.775	**<0.001**	0.502
WCST Conceptual Level Responses	52.5 (44–58) [a]	44 (24–58) [b]	38 (16–53) [b]	28.726	**<0.001**	0.511
WCST Completed Categories	4 (2–5) [a]	3 (2–5) [b]	2 (0–3) [c]	30.913	**<0.001**	0.529
WCST Trials to Complete First Category	11 (11–21) [a]	12.5 (11–32) [b]	25 (10–65) [b]	18.763	**<0.001**	0.406
WCST Failure to Maintain Set	0 (0–3) [a]	0 (0–3) [a]	1 (0–4) [b]	7.190	**0.002**	0.207
WCST Learning-to-Learn Score	0 (-4.37–3.32) [a]	-0.47 (-16.73–13.64) [a]	-6.19 (-24.64 - -3.18) [b]	13.591	**<0.001**	0.393

F: Covariance analysis (ANCOVA) test statistic; η^2: Effect size; a–b: There is no statistically significant difference between groups sharing the same letter. Data are presented as median (min–max) or mean ± SD; TMT: Trail Making Test; RAVLT: Rey Auditory Verbal Learning Test; WCST: Wisconsin Card Sorting Test.

be lowest in the schizophrenia group and highest in the control group, whereas the OCD group exhibited an intermediate level of performance in most parameters (p <0.05). The learning-to-learn score was markedly lower in the schizophrenia group (p=0.006) (Table 2).

When BDI and BAI scores were controlled as covariates in order to exclude the potential confounding effects of depression and anxiety scores, changes were observed in the neuropsychological test profiles of the groups (Table 2.1).

Following covariance analysis, between-group differences emerged in TMT B–A difference duration and RAVLT A1, B1, and Recognition scores, for which no differences had initially been detected. In TMT B–A, the OCD and schizophrenia groups showed lower performance than controls, whereas the schizophrenia group exhibited a more unfavorable pattern particularly in RAVLT A1, B1, and Recognition performance (all p <0.001).

In WCST parameters, in many core variables such as WCST Total Correct Responses, Total Errors, Non-Perseverative Errors, and Conceptual Level Responses, in which all three groups initially differed from one another in a graded manner, the performance difference between the OCD and schizophrenia groups disappeared after adjustment for depression and anxiety (all p <0.001). Both clinical groups became statistically similar to each other and exhibited significantly lower performance only compared with healthy controls (Table 2.1).

miRNA Levels

Significant differences were found between the groups in terms of miR-134 and miR-502 levels (Table 2). miR-134 levels were found to be lower in the OCD group compared with the control group (p=0.004). miR-502 levels, on the other hand, were higher in the control group compared with both the OCD and schizophrenia groups (p <0.001).

In contrast, no significant differences were found between the groups in terms of miR-137, miR-150, and miR-1306 levels (all p >0.05) (Table 2).

Exploratory Correlation Analyses

In the OCD group, miR-502 levels showed a positive correlation with Stroop Test Part V duration (r=0.490, p=0.028) and a negative correlation with the number of completed WCST categories (r=–0.473, p=0.035). In addition, miR-1306 levels showed a positive correlation with the WCST failure to maintain set score (r=0.491, p=0.028). No significant relationship was found between the other miRNAs and neurocognitive tests (Table 3).

Table 3. Relationships between miRNA levels and neuropsychological test scores in patients with OCD

		miR-134	miR-137	miR-150	miR-502	miR-1306
TMT-A (sec)	r	0.242	-0.150	0.255	0.311	0.230
	p	0.304	0.529	0.277	0.182	0.329
TMT-B (sec)	r	0.192	-0.090	0.068	0.139	-0.045
	p	0.417	0.707	0.777	0.558	0.850
TMT B-A (sec)	r	0.079	-0.052	0.025	0.046	-0.172
	p	0.740	0.828	0.917	0.848	0.469
Stroop V (sec)	r	-0.011	-0.208	-0.001	0.490	-0.029
	p	0.965	0.380	0.997	**0.028**	0.903
RAVLT A1	r	-0.435	-0.148	-0.212	-0.418	-0.273
	p	0.055	0.534	0.370	0.067	0.244
RAVLT A1–5	r	-0.252	-0.322	-0.065	-0.242	-0.217
	p	0.283	0.166	0.784	0.303	0.358
RAVLT B1	r	-0.315	-0.361	-0.420	-0.272	-0.335
	p	0.176	0.118	0.065	0.245	0.148
RAVLT A6	r	-0.282	-0.143	-0.033	-0.358	-0.130
	p	0.228	0.547	0.889	0.122	0.584
RAVLT A7	r	-0.159	-0.074	0.038	-0.297	-0.110
	p	0.502	0.756	0.873	0.203	0.646
RAVLT recognition list	r	-0.170	0.002	0.022	0.066	-0.134
	p	0.473	0.992	0.927	0.781	0.574
WCST total correct responses	r	-0.217	0.003	0.076	-0.281	0.149
	p	0.357	0.990	0.749	0.231	0.529
WCST total errors	r	0.217	-0.003	-0.076	0.281	-0.149
	p	0.357	0.990	0.749	0.231	0.529
WCST perseverative responses	r	0.426	-0.159	0.108	0.180	-0.009
	p	0.061	0.503	0.652	0.449	0.968
WCST perseverative errors	r	0.393	-0.151	0.120	0.163	-0.007
	p	0.086	0.526	0.616	0.491	0.976
WCST non-perseverative errors	r	0.057	-0.035	-0.149	0.418	-0.136
	p	0.812	0.884	0.530	0.067	0.567
WCST conceptual level responses	r	-0.144	0.017	-0.005	-0.305	0.159
	p	0.544	0.942	0.982	0.192	0.504
WCST completed categories	r	-0.293	0.047	-0.248	-0.473	-0.019
	p	0.209	0.845	0.292	**0.035**	0.938
WCST trials to complete first category	r	0.188	0.001	0.074	0.138	-0.080
	p	0.428	0.997	0.756	0.562	0.737
WCST failure to maintain set	r	0.345	0.009	0.073	0.333	0.491
	p	0.136	0.971	0.759	0.151	**0.028**
WCST learning-to-learn score	r	-0.006	-0.164	0.084	-0.270	-0.051
	p	0.981	0.515	0.741	0.279	0.842

r: Spearman's rho correlation coefficient.

In order to visualize the findings, correlation patterns were presented in the form of heat maps (Figure 1).

In the schizophrenia group, miR-502 levels showed positive correlations with TMT-A (r=0.484, p=0.031), TMT-B (r=0.524, p=0.018), and Stroop

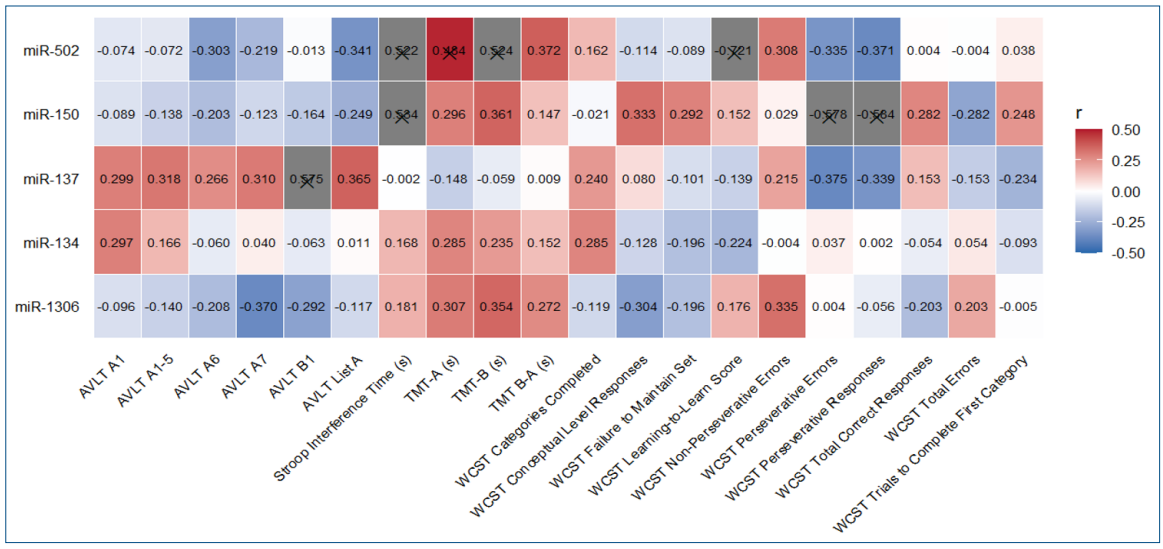

Figure 1. Heat map showing the relationships between miRNA levels and TMT, Stroop, RAVLT, and WCST Scores in patients with OCD (r: Spearman's rho correlation coefficient; x: p <0.05).

Table 4. Relationships between miRNA levels and neuropsychological test scores in patients with schizophrenia

		miR-134	miR-137	miR-150	miR-502	miR-1306
TMT-A (sec)	r	0.285	-0.148	0.296	0.484	0.307
	p	0.224	0.533	0.205	**0.031**	0.188
TMT-B (sec)	r	0.235	-0.059	0.361	0.524	0.354
	p	0.319	0.806	0.118	**0.018**	0.126
TMT B-A (sec)	r	0.152	0.009	0.147	0.372	0.272
	p	0.523	0.970	0.535	0.106	0.247
Stroop V (sec)	r	0.168	-0.002	0.534	0.522	0.181
	p	0.479	0.992	**0.015**	**0.018**	0.446
RAVLT A1	r	0.297	0.299	-0.089	-0.074	-0.096
	p	0.204	0.200	0.708	0.755	0.688
RAVLT A1–5	r	0.166	0.318	-0.138	-0.072	-0.140
	p	0.483	0.171	0.562	0.764	0.556
RAVLT B1	r	-0.063	0.575	-0.164	-0.013	-0.292
	p	0.792	**0.008**	0.489	0.957	0.211
RAVLT A6	r	-0.060	0.266	-0.203	-0.303	-0.208
	p	0.802	0.257	0.391	0.193	0.378
RAVLT A7	r	0.040	0.310	-0.123	-0.219	-0.370
	p	0.866	0.183	0.605	0.352	0.108
RAVLT recognition list	r	0.011	0.365	-0.249	-0.341	-0.117
	p	0.965	0.113	0.289	0.141	0.624
WCST total correct responses	r	-0.054	0.153	0.282	0.004	-0.203
	p	0.820	0.519	0.229	0.986	0.391
WCST total errors	r	0.054	-0.153	-0.282	-0.004	0.203
	p	0.820	0.519	0.229	0.986	0.391
WCST perseverative responses	r	0.002	-0.339	-0.584	-0.371	-0.056
	p	0.992	0.143	**0.007**	0.107	0.815
WCST perseverative errors	r	0.037	-0.375	-0.578	-0.335	0.004
	p	0.876	0.103	**0.008**	0.149	0.987
WCST non-perseverative errors	r	-0.004	0.215	0.029	0.308	0.335
	p	0.987	0.362	0.904	0.187	0.149
WCST conceptual level responses	r	-0.128	0.080	0.333	-0.114	-0.304
	p	0.590	0.738	0.152	0.632	0.193
WCST completed categories	r	0.285	0.240	-0.021	0.162	-0.119
	p	0.223	0.308	0.931	0.496	0.619
WCST trials to complete first category	r	-0.093	-0.234	0.248	0.038	-0.005
	p	0.697	0.320	0.292	0.872	0.982
WCST failure to maintain set	r	-0.196	-0.101	0.292	-0.089	-0.196
	p	0.407	0.672	0.212	0.707	0.408
WCST learning-to-learn score	r	-0.224	-0.139	0.152	-0.721	0.176
	p	0.533	0.701	0.676	**0.019**	0.627

r: Spearman's rho correlation coefficient.

Test Part V duration, and a negative correlation with the WCST learning-to-learn score (r=–0.721, p=0.019). miR-150 levels showed a positive correlation with Stroop Test Part V duration and negative correlations with WCST perseverative responses and perseverative error counts. miR-137 levels were positively associated with the RAVLT B1 score. Other relationships were not found to be significant (Table 4). Findings are presented in Figure 2.

Exploratory Linear Regression Analyses

In the OCD group, while the predictive regression model (Model 1) established for Stroop Test Part V duration, for which a significant relationship was detected in correlation analysis, was not statistically significant (p=0.112); only the model in which miR-502 predicted the number of completed WCST categories was found to be significant (F (1,18)=5.547, p=0.030; adjusted R^2=0.193) (Table 5).

In the schizophrenia group, in the exploratory model including miR-150 and miR-502, both of which showed significant relationships in correlation analyses, a significant model associated with Stroop Test Part V duration was obtained (F (2,17)=6.275, p=0.009; adjusted R^2=0.357). Similarly, the model including miR-137 for the RAVLT B1 score and the model including miR-502 for the WCST learning-to-learn score were also found to be significant at the exploratory level (Table 6).

Exploratory Subgroup Analyses

No significant differences were found between the autogenous OCD, reactive OCD, schizophrenia, and control groups in terms of sociodemographic variables (all p>0.05). In neurocognitive tests, significant differences were found between the groups, particularly in terms of TMT and Stroop Test Part V durations (all p<0.001); the most prominent executive function impairment was observed in the schizophrenia group (Table 7).

When BDI and BAI scores were controlled as covariates, significant differences in most neurocognitive test performances were observed to persist between the autogenous OCD, reactive OCD, schizophrenia, and control groups (all p ≤0.001) (Table 7.1). However, in terms of the autogenous and reactive OCD groups, the cognitive dissociations observed particularly in executive function and verbal memory domains were found to be substantially reduced; the groups exhibited similar performance patterns in many neuropsychological measures (Table 7.1).

In terms of miRNA levels, significant differences were found between the groups for miR-134, miR-502, and miR-1306 (Table 7). miR-134

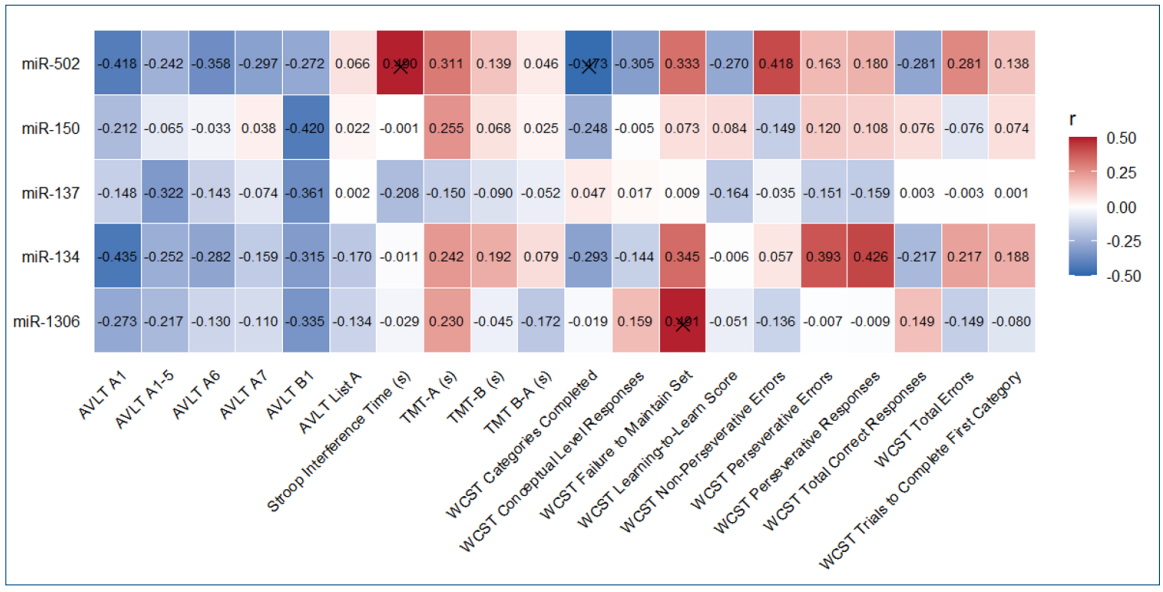

Figure 2. Heat map showing the relationships between miRNA levels and TMT, Stroop, RAVLT, and WCST scores in patients with schizophrenia (r: Spearman's rho correlation coefficient; x: p <0.05).

Table 5. Linear regression analyses examining the effects of miRNA levels on neuropsychological performance in OCD

	β_0 (%95 CI)	SE	β_1	Test Statistic	p	r_1	r_2	VIF
Model 1: Stroop V								
Constant	-0.161 (-31.674–31.352)	14.999		-0.011	0.992			
miR-502	1.103 (-0.285–2.491)	0.660	0.366	1.670	0.112	0.366	0.366	1.000
Model 2: WCST completed categories								
Constant	7.551 (3.783–11.32)	1.794		4.210	**0.001**			
miR-502	-0.186 (-0.352 - -0.02)	0.079	-0.485	-2.355	**0.030**	-0.485	-0.485	1.000
Model 3: WCST failure to maintain set score								
Constant	-2.704 (-7.993–2.585)	2.517		-1.074	0.297			
miR-1306	0.116 (-0.072–0.304)	0.090	0.292	1.296	0.211	0.292	0.292	1.000

Model 1: F=2.789; p=0.112; Adjusted R^2=0.086; DW=2.569
Model 2: F=5.547; p=0.030; Adjusted R^2=0.193; DW=2.076
Model 3: F=1.679; p=0.211; Adjusted R^2=0.035; DW=2.136
SE: standard error; β_0: unstandardized coefficient; β_1: standardized coefficient; r_1: zero-order correlation coefficient; r_2: partial correlation coefficient; CI: confidence interval; VIF: variance inflation factor; DW: Durbin-Watson statistic.

Table 6. Linear regression analyses examining the effects of miRNA levels on neuropsychological performance in patients with schizophrenia

	β_0 (%95 CI)	SE	β_1	Test statistic	p	r_1	r_2	VIF
Model 1: TMT-A								
Constant	-83.294 (-229.718–63.13)	69.695		-1.195	0.248			
miR-502	6.236 (-0.44–12.912)	3.178	0.420	1.962	0.065	0.420	0.420	1.000
Model 2: TMT-B								
Constant	-90.537 (-301.025–119.951)	100.189		-0.904	0.378			
miR-502	9.232 (-0.366–18.829)	4.568	0.430	2.021	0.058	0.430	0.430	1.000
Model 3: Stroop V								
Constant	-82.935 (-155.571 - -10.299)	34.428		-2.409	**0.028**			
miR-150	1.961 (0.146–3.777)	0.861	0.424	2.279	**0.036**	0.489	0.484	1.022
miR-502	3.041 (0.305–5.777)	1.297	0.436	2.345	**0.031**	0.499	0.494	1.022
Model 4: RAVLT B1								
Constant	-2.305 (-7.125–2.515)	2.294		-1.005	0.328			
miR-137	0.177 (0.039–0.314)	0.066	0.536	2.692	**0.015**	0.536	0.536	1.000
Model 5: WCST Perseverative Responses								
Constant	27.741 (8.543–46.94)	9.138		3.036	0.007			
miR-150	-0.621 (-1.304–0.063)	0.325	-0.410	-1.908	0.072	-0.410	-0.410	1.000
Model 6: WCST Perseverative Errors								
Constant	23.067 (8.028–38.105)	7.158		3.222	0.005			
miR-150	-0.484 (-1.019–0.052)	0.255	-0.408	-1.898	0.074	-0.408	-0.408	1.000
Model 7: WCST Learning-to-Learn Score								
Constant	36.296 (-4.621–77.213)	17.744		2.046	0.075			
miR-502	-2.053 (-3.905 - -0.2)	0.803	-0.670	-2.555	**0.034**	-0.670	-0.670	1.000

Model 1: F=3.851; p=0.072; Adjusted R^2=0.065; DW=1.103
Model 2: F=4.084; p=0.058; Adjusted R^2=0.140; DW=1.221
Model 3: F=6.275; p=0.009; Adjusted R^2=0.357; DW=1.701
Model 4: F=7.247; p=0.015; Adjusted R^2=0.247; DW=2.385
Model 5: F=3.640; p=0.072; Adjusted R^2=0.122; DW=1.285
Model 6: F=3.604; p=0.074; Adjusted R^2=0.121; DW=1.242
Model 7: F=6.528; p=0.034; Adjusted R^2=0.380; DW=1.474
SE: standard error; β_0: unstandardized coefficient; β_1: standardized coefficient; r_1: zero-order correlation coefficient; r_2: partial correlation coefficient; CI: confidence interval; VIF: variance inflation factor; DW: Durbin-Watson statistic.

Table 7. Comparison of miRNA levels and neuropsychological test scores among control, autogenous OCD, reactive OCD, and schizophrenia groups

	Control (n=20)	Autogenous OCD (n=9)	Reactive OCD (n=11)	SCH (n=20)	p	η^2
miR-134	30.71 (21.83–33.64)^{a}	28.01 (17.25–32.21)^{b}	27.66 (23.49–32.12)^{ab}	27.44 (25.27–34.89)^{ab}	**0.010^{h}**	0.149
miR-137	36.15 (20.09–38.93)	37.01 (32.41–39.89)	35.47 (23.34–39.28)	35.01 (27.7–39.75)	0.178^{h}	0.034
miR-150	32.19 (22.45–38.75)	26.48 (20.24–31.7)	28.42 (26.07–31.28)	27.97 (19.06–33.02)	0.069^{h}	0.073
miR-502	24.74±1.67^{a}	23.05±2.69^{b}	22.21±2.27^{c}	21.85±1.98^{c}	**<0.001^{f}**	0.283
miR-1306	28.14±0.88^{ab}	29.08±2.0^{ab}	27.18±1.78^{a}	29.18±1.85^{b}	**0.037^{w}**	0.191
TMT-A (sec)	20 (13–80)^{a}	32 (19–46)^{ab}	30 (14–71)^{ab}	41.5 (24–112)^{b}	**<0.001^{h}**	0.394
TMT-B (sec)	48 (30–150)^{a}	83 (44–149)^{ab}	59 (38–245)^{ab}	91.5 (56–205)^{b}	**<0.001^{h}**	0.357
TMT B-A (sec)	26 (16–70)^{a}	46 (25–103)^{ab}	34 (18–174)^{ab}	57 (24–105)^{b}	**<0.001^{h}**	0.272
Stroop V (sec)	17 (11–33)^{a}	24 (15–31)^{ab}	22 (18–43)^{ab}	37 (19–67)^{b}	**<0.001^{h}**	0.443
RAVLT A1	10 (5–11)^{a}	9 (5–10)^{ab}	7 (4–11)^{ab}	5 (3–10)^{b}	**<0.001^{h}**	0.347
RAVLT A1–5	64.5 (44–68)^{a}	56 (44–63)^{a}	58 (39–69)^{a}	31.5 (14–56)^{b}	**<0.001^{h}**	0.608
RAVLT B1	7 (4–13)^{a}	7 (4–9)^{a}	7 (3–13)^{a}	4 (2–5)^{b}	**<0.001^{h}**	0.469
RAVLT A6	14 (8–15)^{a}	12 (9–15)^{a}	12 (7–15)^{a}	6 (2–11)^{b}	**<0.001^{h}**	0.619
RAVLT A7	14 (9–15)^{a}	12 (8–14)^{a}	11 (8–14)^{a}	5.5 (2–10)^{b}	**<0.001^{h}**	0.664
RAVLT recognition list	15 (11–15)^{a}	15 (11–15)^{a}	14 (10–15)^{ab}	11.5 (6–15)^{b}	**<0.001^{h}**	0.394
WCST total correct responses	54.25±2.15^{a}	48.11±5.3^{b}	46.09±5.32^{c}	43±5.52^{c}	**<0.001^{f}**	0.529
WCST total errors	9.75±2.15^{a}	15.89±5.3^{b}	17.91±5.32^{bc}	21±5.52^{c}	**<0.001^{f}**	0.529
WCST perseverative responses	5 (3–9)^{a}	8 (3–13)^{b}	9 (4–14)^{ab}	11 (3–18)^{b}	**<0.001^{h}**	0.237
WCST perseverative errors	5.05±1.43^{a}	7.67±2.35^{b}	8.36±3.01^{b}	9.55±3.53^{b}	**<0.001^{w}**	0.341
WCST non-perseverative errors	4.5 (3–9)^{a}	7 (3–14)^{ab}	9 (4–16)^{b}	11.5 (4–22)^{b}	**<0.001^{h}**	0.451
WCST conceptual level responses	52.3±3.63^{a}	45.11±6.09^{b}	42.73±8.62^{bc}	37.45±8.48^{c}	**<0.001^{f}**	0.458
WCST completed categories	4 (2–5)^{a}	3 (2–5)^{ab}	4 (2–5)^{a}	2 (0–3)^{b}	**<0.001^{h}**	0.506
WCST trials to complete first category	11 (11–21)^{a}	11 (11–19)^{a}	15 (11–32)^{ab}	25 (10–65)^{b}	**<0.001^{h}**	0.343
WCST failure to maintain set	0 (0–3)^{a}	1 (0–2)^{ab}	0 (0–3)^{a}	1 (0–4)^{b}	**0.002^{h}**	0.170
WCST learning-to-learn score	0 (-4.37–3.32)^{ab}	-3.79 (-16.73–4.04)^{ac}	3.33 (-2.38–13.64)^{b}	-6.19 (-24.64 - -3.18)^{c}	**<0.001^{h}**	0.233

f: One-way ANOVA; w: Welch ANOVA; h: Kruskal-Wallis H test; η^2: effect size; a-b-c: there is no statistically significant difference between groups sharing the same letter; data are presented as median (min-max) or mean ± SD; miRNA: microRNA; TMT: Trail Making Test; RAVLT: Rey Auditory Verbal Learning Test; WCST: Wisconsin Card Sorting Test.

Table 7.1. Comparison of neuropsychological test scores between groups after controlling for BDI and BAI scores as covariates

	Control (n=20)	Autogenous OCD (n=9)	Reactive OCD (n=11)	SCH (n=20)	F	p	η^2
TMT-A (sec)	20 (13–80)[a]	32 (19–46)[b]	30 (14–71)[b]	41.5 (24–112)[b]	13.291	**<0.001**	0.425
TMT-B (sec)	48 (30–150)[a]	83 (44–149)[b]	59 (38–245)[b]	91.5 (56–205)[b]	12.806	**<0.001**	0.416
TMT B-A (sec)	26 (16–70)[a]	46 (25–103)[b]	34 (18–174)[ab]	57 (24–105)[b]	9.840	**<0.001**	0.353
Stroop V (sec)	17 (11–33)[a]	24 (15–31)[ab]	22 (18–43)[b]	37 (19–67)[b]	16.676	**<0.001**	0.481
RAVLT A1	10 (5–11)[a]	9 (5–10)[ab]	7 (4–11)[ab]	5 (3–10)[b]	10.799	**<0.001**	0.375
RAVLT A1–5	64.5 (44–68)[a]	56 (44–63)[b]	58 (39–69)[b]	31.5 (14–56)[c]	29.865	**<0.001**	0.624
RAVLT B1	7 (4–13)[a]	7 (4–9)[a]	7 (3–13)[a]	4 (2–5)[b]	19.056	**<0.001**	0.514
RAVLT A6	14 (8–15)[a]	12 (9–15)[a]	12 (7–15)[a]	6 (2–11)[b]	31.512	**<0.001**	0.636
RAVLT A7	14 (9–15)[a]	12 (8–14)[b]	11 (8–14)[b]	5.5 (2–10)[c]	39.194	**<0.001**	0.685
RAVLT recognition list	15 (11–15)[a]	15 (11–15)[a]	14 (10–15)[a]	11.5 (6–15)[b]	15.484	**<0.001**	0.446
WCST total correct responses	54.5 (50–58)[a]	47 (41–58)[b]	46 (36–56)[b]	43.5 (28–53)[b]	22.075	**<0.001**	0.551
WCST total errors	9.5 (6–14)[a]	17 (6–23)[b]	18 (8–28)[b]	20.5 (11–36)[b]	22.075	**<0.001**	0.551
WCST perseverative responses	5 (3–9)[a]	8 (3–13)[ab]	9 (4–14)[ab]	11 (3–18)[b]	6.606	**0.001**	0.268
WCST perseverative errors	5 (3–8)[a]	8 (3–10)[ab]	9 (4–12)[ab]	11 (3–14)[b]	9.024	**<0.001**	0.334
WCST non-perseverative errors	4.5 (3–9)[a]	7 (3–14)[b]	9 (4–16)[b]	11.5 (4–22)[b]	18.224	**<0.001**	0.503
WCST conceptual level responses	52.5 (44–58)[a]	45 (36–58)[b]	43 (24–56)[b]	38 (16–53)[b]	19.012	**<0.001**	0.514
WCST completed categories	4 (2–5)[a]	3 (2–5)[a]	4 (2–5)[a]	2 (0–3)[b]	20.516	**<0.001**	0.533
WCST trials to complete first category	11 (11–21)[a]	11 (11–19)[ab]	15 (11–32)[bc]	25 (10–65)[c]	13.757	**<0.001**	0.433
WCST failure to maintain set	0 (0–3)[a]	1 (0–2)[ab]	0 (0–3)[a]	1 (0–4)[b]	6.180	**0.001**	0.256
WCST learning-to-learn score	0 (-4.37 – 3.32)[ab]	-3.79 (-16.73 – 4.04)[ac]	3.33 (-2.38 – 13.64)[b]	-6.19 (-24.64 - -3.18)[c]	18.095	**<0.001**	0.570

F: covariance analysis (ANCOVA) test statistic; η^2: effect size; a-b: There is no statistically significant difference between groups sharing the same letter. Data are presented as median (min-max) or mean ± SD; TMT: Trail Making Test; RAVLT: Rey Auditory Verbal Learning Test; WCST: Wisconsin Card Sorting Test.

levels were found to be higher in the control group compared with the autogenous OCD group. miR-502 levels were highest in the control group and lower in the reactive OCD and schizophrenia groups, showing a gradual decrease. miR-1306 levels were found to be highest in the schizophrenia group. No significant differences were found in terms of miR-137 and miR-150 levels.

DISCUSSION

The present study aimed to investigate how cognitive performance and the expression levels of selected microRNAs (miR-134, miR-137, miR-150, miR-502, and miR-1306) differ between groups in OCD and schizophrenia; additionally, it aimed to exploratorily evaluate whether there is heterogeneity in cognitive and miRNA levels in the autogenous and reactive subtypes of OCD.

In our sample, the finding of an earlier age at onset and longer duration of illness in OCD is consistent with the tendency of OCD to frequently begin in adolescence–early adulthood and follow a chronic course, whereas schizophrenia may begin in later adolescence/early adulthood and show a relative plateau tendency over time (1).

Neuropsychological Test Performance

The covariance analysis conducted to evaluate the potential confounding effect of depressive and anxiety symptoms suggests that the neurocognitive differences observed in schizophrenia and OCD may not be explained solely by mood symptoms. In the literature, it has been suggested that executive function and memory impairments in patients with OCD may be secondary to accompanying depressive symptoms and that cognitive differences may decrease when these variables are controlled (29,30). However, in our study, when BDI and BAI scores were controlled as covariates, it was observed that group differences persisted in some cognitive domains, whereas they became more apparent in others. In particular, the emergence of significant group differences following covariance analysis in TMT B–A difference duration and RAVLT immediate registration (A1, B1) and recognition performance, which had not shown significant differences in the initial analyses, is noteworthy. In contrast, the reduction in differentiation between the OCD and schizophrenia groups in certain executive function parameters suggests that depressive and anxiety symptoms may contribute dimensionally to specific cognitive domains. When these findings are evaluated together, it is considered that the observed neurocognitive patterns may not be explained solely by accompanying mood symptoms; however, they should also not be interpreted as processes entirely independent of these symptoms. Contemporary approaches also emphasize that cognitive impairments observed in psychopathologies can be addressed as a transdiagnostic dimension or "C factor" (cognitive dysfunction), which may show commonality across diagnoses (31).

The changes observed in WCST findings evaluating executive functions suggest that there may be both overlapping and differentiating cognitive patterns between schizophrenia and OCD. Following adjustment for depression and anxiety, the difference between the OCD and schizophrenia groups disappeared in some core parameters such as WCST Total Correct Responses, Total Errors, Non-Perseverative Errors, and Conceptual Level Responses, which initially showed a graded pattern of control, OCD, and schizophrenia in the initial analyses; both clinical groups demonstrated similar levels of performance and differed from healthy controls. This situation suggests that there may be greater commonality than expected between the two clinical groups, particularly in concept formation, cognitive flexibility, and executive control processes (32,33). On the other hand, the finding that the TMT B–A difference, which did not show a significant difference in the initial analyses, became significant following covariance analysis and that the marked group differentiation in Stroop interference duration persisted suggests that difficulties in attentional control, inhibition, and cognitive flexibility processes continue even when depressive and anxiety symptoms are controlled (34,35).

In contrast, it is noteworthy that the schizophrenia group exhibited a more unfavorable pattern than the other groups in the WCST Learning-to-Learn and Failure to Maintain Set parameters. In particular, the finding

that the "Failure to Maintain Set" parameter, which was initially not significant, became significant following covariance analysis suggests that impairment in more complex executive processes such as developing strategies using feedback, sustaining attention, and learning from errors may be more pronounced in schizophrenia. The widespread pattern of neurocognitive impairment in schizophrenia, encompassing executive functions, attention, processing speed, and memory domains, has been supported by accumulating meta-analytic and observational literature over many years (36,37). Therefore, our findings suggest that cognitive impairment in schizophrenia may differ from OCD not only in terms of severity but also in terms of scope and complexity.

The pattern observed in verbal memory (RAVLT) findings also supports the differentiation between the cognitive architectures of schizophrenia and OCD. Following covariance analysis, the lower performance of the schizophrenia group compared with the other groups in immediate registration (A1, B1), learning (A1–5), free recall (A6, A7), and recognition processes indicates a more widespread verbal memory impairment in schizophrenia encompassing encoding, learning, and retrieval processes (36,37). In contrast, it is noteworthy that the OCD group, differing from schizophrenia in A1, B1, and Recognition List performance, exhibited a profile closer to healthy controls. This situation suggests that basic registration and storage processes may be relatively preserved in OCD (38). Nevertheless, the relatively lower performance of the OCD group in learning (A1–5) and free recall (A6, A7) may be related not to a primary memory impairment but rather to difficulties in executive function processes involved in organizing information, strategic encoding, and retrieval. This interpretation is consistent with the literature suggesting that executive dysfunction in OCD may secondarily affect memory performance (32,38).

In conclusion, our findings support a more widespread and global pattern of neurocognitive impairment in schizophrenia, whereas in OCD, despite relatively preserved basic memory processes, more selective difficulties related to executive functions are supported. However, due to the sample size, the findings need to be confirmed in larger samples.

miRNA Analyses

The low plasma miR-134 levels detected in the OCD group contradict previous findings reporting elevated levels in medication-free patients (39); however, this situation may be explained by sample characteristics and particularly differences in pharmacotherapy. Although the role of miR-134 in limiting synaptic plasticity (10) may theoretically be associated with cognitive rigidity in OCD, data showing that this molecule decreases in stress and depressive processes (40) suggest that the present finding may be related to higher severity of depressive symptoms. However, evidence indicating that psychopharmacological agents may regulate miRNA expression (41) suggests that treatment effect may be a determining factor in this decrease. The absence of changes in miR-134 levels in the schizophrenia group differs from large-sample studies reporting reductions in various miRNAs, including miR-134 (42). Considering the sensitivity of miRNA profiles to clinical stage and treatment status (43), this situation may be related to sample size and the masking effect of antipsychotic use.

The finding of lower plasma miR-502 levels in both the OCD and schizophrenia groups compared with controls may indicate a shared peripheral miRNA signature in the etiology of these two disorders. Recent bioinformatic analyses demonstrating common risk genes and simultaneous involvement of serotonergic/dopaminergic/glutamatergic pathways in both disorders provide a basis supporting this molecular overlap (44). Experimental studies have shown that miR-502 negatively regulates GABAergic transmission by targeting the *GABRA1* gene (13). Postmortem reductions in GAD67 and *GABRA1* expression in schizophrenia (45) and GABAergic alterations detected in OCD (46) suggest a common "GABAergic dysfunction" in both disorders. In this context, the decrease in miR-502 observed in our study may be interpreted as a potential compensatory mechanism aimed at increasing receptor (*GABRA1*) expression in order to compensate for the existing GABAergic deficiency. It is known that antipsychotic treatments may affect miRNA profiles (47). The fact that miR-502 showed a change in the same direction (decrease) in both disease groups may indicate a common neurobiological process; however, it may also be a consequence of different classes of pharmacological agents used in patients with schizophrenia and OCD similarly affecting the same biological pathways (down-regulation). Since the pharmacological heterogeneity and sample size in our study were not suitable for statistically controlling medication effects, it cannot be definitively distinguished whether this decrease reflects disease-specific biology or a shared medication effect. This constitutes an important limitation that should be considered in the interpretation of our results.

The absence of a significant difference in miR-137 levels between the patient groups and controls in our study should be evaluated in light of the complex picture in the literature regarding the peripheral expression of this molecule. Findings regarding miR-137 in schizophrenia show marked heterogeneity depending on sample material and clinical stage. While a study conducted with plasma samples reported increased miR-137 particularly in early-stage patients (48), a recent study using whole blood reported a marked decrease in patients (49). The authors attributed this inconsistency not only to differences in sample type but also to the masking role of hormonal interactions such as sex and estrogen/prolactin (49). It has also been emphasized that dysregulation of miR-137 may not be consistently demonstrated unless risk variants in the miR-137 gene are taken into consideration and that the effect of this molecule may be particularly specific to neurodevelopmental processes during adolescence/early adulthood (50). The absence of altered levels in the OCD group also suggests that although there is strong evidence for central pathologies such as cortico-striato-thalamo-cortical circuit dysfunctions and glutamatergic/dopaminergic dysregulations in the pathophysiology of OCD (51), the sensitivity of miR-137 in reflecting this central pathology may be limited.

The absence of differences in miR-1306 and miR-150 levels between groups is consistent with the limited and heterogeneous data in the literature (52). miR-1306 has been defined mainly in the context of 22q11.2 deletion syndrome and neuronal models rather than peripheral biomarker studies (53). While miR-150 shows heterogeneous results across different tissues and methods in schizophrenia (52), to our knowledge, no specific data have yet been reported for OCD.

Exploratory Correlation and Regression Results

Exploratory correlation and regression analyses provide preliminary findings indicating that miR-502 may be associated with executive functions in both clinical groups.

In the OCD group, miR-502 levels were found to be associated with Stroop interference duration at the correlation level and directly with WCST performance in regression analysis; in the schizophrenia group, miR-502 levels were found to be associated with TMT, Stroop interference, and WCST learning-to-learn scores, suggesting that this molecule may accompany cognitive control and flexibility processes. Considering that executive function impairments are associated with fronto-striatal circuit dysfunctions in schizophrenia and OCD (32), these findings are conceptually meaningful. Experimental studies showing that miR-502 may target the *GABRA1* gene regulating GABAergic transmission (13), together with GABAergic alterations reported in both disorders (45,46), provide a possible biological basis for the observed relationships. However, considering the limited and selective effects detected in

regression analyses, as well as sample size and multiple comparisons, our findings should be evaluated as exploratory in nature; these findings should be interpreted not as confirmatory evidence but as hypothesis-generating preliminary findings.

Exploratory Subgroup Analysis Results

Due to the clinical heterogeneity of OCD, exploratory analyses were conducted by dividing OCD into autogenous and reactive subtypes. Due to the limited sample size, subgroup analyses should be considered descriptive/hypothesis-generating. Although the similarity of sociodemographic variables between groups suggests that the confounding effects of variables such as age, sex, and education may be limited, this does not provide causal inference. Although studies reporting an earlier age at onset and higher male ratio in autogenous OCD exist in the literature (54), there are also studies reporting no sociodemographic differences between subtypes (55).

In our study, impairments in executive functions, verbal memory, and processing speed were observed in both OCD subtypes in terms of neurocognitive performance; no marked cognitive differentiation was detected between autogenous and reactive obsession subtypes. Particularly when depression and anxiety levels were controlled as covariates, cognitive differences between the two subtypes were largely reduced, and similar patterns emerged in TMT, RAVLT, and WCST performance. This finding appears to be consistent with the general neuropsychological literature reporting executive function and memory impairments in OCD independently of subtypes (32). Nevertheless, the limited dissociations observed in some measures, such as Stroop interference performance, TMT B–A difference, and WCST learning-to-learn score, suggest that subtle cognitive differences may exist between autogenous and reactive obsession subtypes and that this pattern should be re-evaluated in larger samples.

The limited differences observed in miR-134 and miR-502 levels provide indirect and exploratory clues that the autogenous-reactive distinction may not be entirely unrelated to biological processes. However, considering sample size, treatment effects, and the exploratory nature of the analyses, it appears more appropriate to evaluate these findings within a dimensional and hypothesis-generating framework rather than interpreting them as discriminative biological markers for OCD subtypes.

The limited sample size reduces the power of subgroup analyses in particular and limits generalizability; therefore, autogenous/reactive findings should be considered exploratory/hypothesis-generating. Since heterogeneity in psychotropic medication use may create pharmacoepigenetic effects on miRNA expression, part of the molecular patterns may reflect treatment effects. Variables such as illness duration, symptom severity, clinical stability, and motivation may also affect neuropsychological performance; therefore, interpretations should be made cautiously. The selected miRNA approach and the limited number of biological parameters may increase sensitivity to statistical error in small samples; therefore, the findings need to be confirmed in larger samples, preferably with predefined primary hypotheses and strengthened quality control reporting. In the future, larger/homogeneous samples, medication-naïve groups, covariance control of mood indicators, and longitudinal designs, as well as multi-level (genetic-neuroimaging-neuropsychology) approaches, are recommended.

As a result, this study by comparatively evaluating neurocognitive performance and selected peripheral miRNA levels in schizophrenia and obsessive-compulsive disorder within the same sample framework, indicates that the two disorders may exhibit both overlapping and differentiating patterns in terms of cognitive and peripheral biological characteristics. The findings suggest a more widespread and global pattern of cognitive impairment in schizophrenia encompassing attention, executive functions, and verbal memory domains; whereas in OCD, a relatively more selective pattern of neurocognitive impairment is suggested, in which executive functions are particularly prominent and difficulties in verbal memory performance may be more related to executive processes. The differences observed in selected miRNA levels provide exploratory findings regarding peripheral molecular variables that should be evaluated together with cognitive patterns. Although exploratory analyses revealed preliminary findings particularly indicating that miR-502 may be associated with executive function indicators, confirmation of these relationships requires studies conducted in larger, preferably medication-naïve and longitudinal samples. Overall, our study suggests that a multidimensional approach that addresses cognition as a fundamental axis and positions molecular variables within this framework may provide a useful conceptual framework for understanding the comparative neurobiology of schizophrenia and OCD and may constitute a preliminary basis for future confirmatory studies.

Ethics Committee Approval: The study was approved by the Karadeniz Technical University Faculty of Medicine Scientific Research Ethics Committee (Decision No: 24237859/178).

Informed Consent: Individuals aged between 18 and 65 years who provided written informed consent and whose diagnosis of schizophrenia or OCD was confirmed using the SCID-5-CV were included in the study.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept- EU, DSA; Design- EU, DSA; Supervision- EU, DSA; Resource- EU, DSA, MG; Materials- EU, DSA; Data Collection and/or Processing- EU, DSA, EÖK, AHÇ, MG; Analysis and/or Interpretation- EU, DSA, EÖK; Literature Search- EU, DSA, AHÇ; Writing- EU, DSA; Critical Reviews- EÖK.

Conflict of Interest: The authors declared that there is no conflict of interest.

Financial Disclosure: This study was supported by the Karadeniz Technical University Faculty of Medicine Scientific Research Projects Unit (Project No: 3579295). The funding organization had no role in the study design, data collection, analysis and interpretation of data, writing of the manuscript, or the decision to submit the manuscript for publication.

Artificial Intelligence Use: During the preparation of this manuscript, the authors utilized artificial intelligence to support language editing, improve the clarity and readability of the text, and assist in the organization of the manuscript. Following the use of this tool, the authors carefully reviewed, revised, and made the necessary adaptations to all content and assume full responsibility for the accuracy, originality, and integrity of the published work.

REFERENCES

1. Pardossi S, Cuomo A, Fagiolini A. Unraveling the boundaries, overlaps, and connections between schizophrenia and obsessive-compulsive disorder (OCD). J Clin Med. 2024;13:4739. [Crossref]
2. McCutcheon RA, Keefe RSE, McGuire PK. Cognitive impairment in schizophrenia: aetiology, pathophysiology, and treatment. Mol Psychiatry. 2023;28:1902–1918. [Crossref]
3. Jalal B, Chamberlain SR, Sahakian BJ. Obsessive-compulsive disorder: etiology, neuropathology, and cognitive dysfunction. Brain Behav. 2023;13:e3000. [Crossref]
4. Venkatasubramanian G, Rao NP, Behere RV. Neuroanatomical, neurochemical, and neurodevelopmental basis of obsessive-compulsive symptoms in schizophrenia. Indian J Psychol Med. 2009;31:3–10. [Crossref]
5. Lee HJ, Kwon SM. Two different types of obsession: autogenous obsessions and reactive obsessions. Behav Res Ther. 2003;41:11–29. [Crossref]
6. Besiroglu L, Agargun MY, Ozbebit O, Aydin A. A discrimination based on autogenous versus reactive obsessions in obsessive-compulsive disorder and related clinical manifestations. CNS Spectr. 2006;11:179–186. [Crossref]
7. Moulding R, Kyrios M, Doron G, Nedeljkovic M. Autogenous and reactive obsessions: further evidence for a two-factor model of obsessions. J Anxiety Disord. 2007;21:677–690. [Crossref]
8. Hu Z, Li Z. miRNAs in synapse development and synaptic plasticity. Curr Opin Neurobiol. 2017;45:24–31. [Crossref]
9. Ripke S, Sanders AR, Kendler KS, Levinson DF, Sklar P, Holmans PA, et al. Genome-wide association study identifies five new schizophrenia loci. Nat Genet. 2011;43:969–976. [Crossref]

10. Schratt GM, Tuebing F, Nigh EA, Kane CG, Sabatini ME, Kiebler M, et al. A brain-specific microRNA regulates dendritic spine development. Nature. 2006;439:283–289. [Crossref]
11. Hu YZ, Li Q, Wang PF, Li XP, Hu ZL. Multiple functions and regulatory network of miR-150 in B lymphocyte-related diseases. Front Oncol. 2023;13:1140813. [Crossref]
12. Wei H, Yuan Y, Liu S, Wang C, Yang F, Lu Z, et al. Detection of circulating miRNA levels in schizophrenia. Am J Psychiatry. 2015;172:1141–1147. [Crossref]
13. Sharma B, Torres MM, Rodriguez S, Gangwani L, Kumar S. MicroRNA-502-3p regulates GABAergic synapse function in hippocampal neurons. Neural Regen Res. 2024;19:2698–2707. [Crossref]
14. Meng S, Wang B, Li W. CircAXL knockdown alleviates Aβ1-42-induced neurotoxicity in Alzheimer's disease via repressing PDE4A by releasing miR-1306-5p. Neurochem Res. 2022;47:1707–1720. [Crossref]
15. Beck AT, Ward CH, Mendelson M, Mock J, Erbaugh J. An inventory for measuring depression. Arch Gen Psychiatry. 1961;4:561–571. [Crossref]
16. Hisli N. Beck Depresyon Envanterinin üniversite öğrencileri için geçerliği ve güvenirliği. Psikoloji Derg. 1989;7:3–13.
17. Beck AT, Epstein N, Brown G, Steer RA. An inventory for measuring clinical anxiety: psychometric properties. J Consult Clin Psychol. 1988;56:893–897. [Crossref]
18. Ulusoy M, Sahin NH, Erkmen H. Turkish version of the Beck Anxiety Inventory: psychometric properties. J Cogn Psychother. 1998;12:163–172.
19. Goodman WK, Price LH, Rasmussen SA, Mazure C, Fleischmann RL, Hill CL, et al. The Yale-Brown Obsessive Compulsive Scale. I. Development, use, and reliability. Arch Gen Psychiatry. 1989;46:1006–1011. [Crossref]
20. Tek C, Ulug B, Rezaki BG, Tanriverdi N, Mercan S, Demir B, et al. Yale-Brown Obsessive Compulsive Scale and US National Institute of Mental Health Global Obsessive Compulsive Scale in Turkish: reliability and validity. Acta Psychiatr Scand. 1995;91:410–413. [Crossref]
21. Berg EA. A simple objective technique for measuring flexibility in thinking. J Gen Psychol. 1948;39:15–22. [Crossref]
22. Karakaş S. Bilnot Bataryası El Kitabı: Nöropsikolojik Testler için Araştırma ve Geliştirme Çalışmaları. Ankara: Dizayn Ofset; 2004.
23. Stroop JR. Studies of interference in serial verbal reactions. J Exp Psychol. 1935;18:643–662. [Crossref]
24. Karakaş S, Erdoğan E, Soysal Ş, Ulusoy T, Ulusoy İY, Alkan S. Stroop Test TBAG Form: standardisation for Turkish culture, reliability and validity. Turkish J Clin Psy. 1999;2:75–88.
25. Reitan RM. Validity of the Trail Making Test as an indicator of organic brain damage. Percept Mot Skills. 1958;8:271–276. [Crossref]
26. Türkeş N, Can H, Kurt M, Elmastaş-Dikeç B. İz Sürme Testinin 20-49 yaş aralığında Türkiye için norm belirleme çalışması. Türk Psikiyatri Derg. 2015;26:189–196. [Crossref]
27. Rey A. L'examen clinique en psychologie (The Clinical Psychological Examination). Paris: Presses Universitaires de France; 1964.
28. Can H, Doğutepe E, Torun Yazıhan N, Korkman H, Erdoğan Bakar E. İşitsel sözel öğrenme testi yapı geçerliği. Türk Psikiyatri Derg. 2016;27:195–203.
29. Basso MR, Bornstein RA, Carona F, Morton R. Depression accounts for executive function deficits in obsessive-compulsive disorder. Neuropsychiatry Neuropsychol Behav Neurol. 2001;14:241–245.
30. Moritz S, Birkner C, Kloss M, Jacobsen D, Fricke S, Böthern A, et al. Impact of comorbid depressive symptoms on neuropsychological performance in obsessive-compulsive disorder. J Abnorm Psychol. 2001;110:653–657. [Crossref]
31. Abramovitch A, Short T, Schweiger A. The C factor: cognitive dysfunction as a transdiagnostic dimension in psychopathology. Clin Psychol Rev. 2021;86:102007. [Crossref]
32. Abramovitch A, Abramowitz JS, Mittelman A. The neuropsychology of adult obsessive-compulsive disorder: a meta-analysis. Clin Psychol Rev. 2013;33:1163–1171. [Crossref]
33. Aumer P, Brandt GA, Hirjak D, Bähner F. Impaired cognitive flexibility in schizophrenia: a systematic review of behavioral and neurobiological findings. Biomark Neuropsychiatry. 2024;11:100111. [Crossref]
34. Önder Uzgan B, Tetik Oktay M, Aykaç C, Ermiş Ç, Alkın T. Neurocognitive flexibility, perfectionism, obsessive beliefs in patients with obsessive compulsive disorder. Turkish J Clin Psy. 2021;24:439–449. [Crossref]
35. Westerhausen R, Kompus K, Hugdahl K. Impaired cognitive inhibition in schizophrenia: a meta-analysis of the Stroop interference effect. Schizophr Res. 2011;133:172–181. [Crossref]
36. Heinrichs RW, Zakzanis KK. Neurocognitive deficit in schizophrenia: a quantitative review of the evidence. Neuropsychology. 1998;12:426–445. [Crossref]
37. Schaefer J, Giangrande E, Weinberger DR, Dickinson D. The global cognitive impairment in schizophrenia: consistent over decades and around the world. Schizophr Res. 2013;150:42–50. [Crossref]
38. Roth RM, Baribeau J, Milovan D, O'Connor K, Todorov C. Procedural and declarative memory in obsessive-compulsive disorder. J Int Neuropsychol Soc. 2004;10:647–654. [Crossref]
39. Yue J, Zhang B, Wang H, Hou X, Chen X, Cheng M, et al. Dysregulated plasma levels of miRNA-132 and miRNA-134 in patients with obsessive-compulsive disorder. Ann Transl Med. 2020;8:996. [Crossref]
40. Zhang HP, Liu XL, Chen JJ, Cheng K, Bai SJ, Zheng P, et al. Circulating microRNA-134 sheds light on the diagnosis of major depressive disorder. Transl Psychiatry. 2020;10:95. [Crossref]
41. Funatsuki T, Ogata H, Tahara H, Shimamoto A, Takekita Y, Koshikawa Y, et al. Changes in multiple microRNA levels with antidepressant treatment are associated with remission and interact with key pathways: a comprehensive microRNA analysis. Int J Mol Sci. 2023;24:12199. [Crossref]
42. Yu HC, Wu J, Zhang HX, Zhang GL, Sui J, Tong WW, et al. Alterations of miR-132 are novel diagnostic biomarkers in peripheral blood of schizophrenia patients. Prog Neuropsychopharmacol Biol Psychiatry. 2015;63:23–29. [Crossref]
43. Lai CY, Yu SL, Hsieh MH, Chen CH, Chen HY, Wen CC, et al. MicroRNA expression aberration as potential peripheral blood biomarkers for schizophrenia. PLoS One. 2011;6:e21635. [Crossref]
44. Ghanbarzehi A, Sepehrinezhad A, Hashemi N, Karimi M, Shahbazi A. Disclosing common biological signatures and predicting new therapeutic targets in schizophrenia and obsessive-compulsive disorder by integrated bioinformatics analysis. BMC Psychiatry. 2023;23:40. [Crossref]
45. Hashimoto T, Bazmi HH, Mirnics K, Wu Q, Sampson AR, Lewis DA. Conserved regional patterns of GABA-related transcript expression in the neocortex of subjects with schizophrenia. Am J Psychiatry. 2008;165:479–489. [Crossref]
46. Biria M, Banca P, Heal MP, Keser E, Sawiak SJ, Rodgers CT, et al. Cortical glutamate and GABA are related to compulsive behaviour in individuals with obsessive-compulsive disorder and healthy controls. Nat Commun. 2023;14:3324. [Crossref]
47. Martinez B, Peplow PV. MicroRNAs as potential biomarkers for diagnosis of schizophrenia and influence of antipsychotic treatment. Neural Regen Res. 2024;19:1523–1531. [Crossref]
48. Chen BY, Lin JJ, Lu MK, Tan HP, Jang FL, Lin SH, et al. Neurodevelopment regulators miR-137 and miR-34 family as biomarkers for early and adult onset schizophrenia. NPJ Schizophr. 2021;7:35. [Crossref]
49. Peng Q, Dai Z, Yin J, Lv D, Luo X, Xiong S, et al. Schizophrenia plausible protective effect of microRNA-137 is potentially related to estrogen and prolactin in female patients. Front Psychiatry. 2023;14:1187111. [Crossref]
50. Thomas KT, Zakharenko SS. MicroRNAs in the onset of schizophrenia. Cells. 2021;10:2679. [Crossref]
51. Gargano SP, Santos MG, Taylor SM, Pastis I. A closer look to neural pathways and psychopharmacology of obsessive compulsive disorder. Front Behav Neurosci. 2023;17:1282246. [Crossref]
52. Han S, Li Y, Gao J. Peripheral blood microRNAs as biomarkers of schizophrenia: expectations from a meta-analysis that combines deep learning methods. World J Biol Psychiatry. 2024;25:65–81. [Crossref]
53. Zhao D, Lin M, Chen J, Pedrosa E, Hrabovsky A, Fourcade HM, et al. MicroRNA profiling of neurons generated using induced pluripotent stem cells derived from patients with schizophrenia and schizoaffective disorder, and 22q11.2 deletion. PLoS One. 2015;10:e0132387. [Crossref]
54. Yucens B, Erdogan NO, Gündüz M, Tumkaya S. Comparison of autogenous and reactive type obsessive-compulsive disorders in terms of clinical characteristics: a meta-analysis study. J Psychiatr Res. 2024;176:338–347. [Crossref]
55. Besiroglu L, Sozen M, Ozbebit O, Avcu S, Selvi Y, Bora A, et al. The involvement of distinct neural systems in patients with obsessive-compulsive disorder with autogenous and reactive obsessions. Acta Psychiatr Scand 2011;124:141–151. [Crossref]