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From Early Recognition to Precision Psychiatry: Advancing Mental Health Research Across the Lifespan

Mental disorders remain among the leading causes of disability worldwide, with depression, schizophrenia, substance use disorders, and obsessive-compulsive spectrum disorders contributing substantially to the global burden of disease. Despite remarkable advances in neuroscience and psychopharmacology, many psychiatric disorders continue to be diagnosed late, follow heterogeneous clinical courses, and demonstrate variable responses to treatment. Consequently, modern psychiatry increasingly emphasizes early detection, identification of biological and clinical markers, personalized interventions, and multidisciplinary care.

The current issue of "Biological Psychiatry and Psychopharmacology" reflects these priorities by bringing together original research, narrative reviews, assessment tools, and doctoral research addressing several key areas of contemporary psychiatric science.

The issue opens with an original population-based study by Tamaliūnaitė and Laurinaitienė, examining the prevalence and severity of depressive symptoms among individuals without a previously diagnosed mental disorder. Their findings remind us that clinically relevant depressive symptoms frequently remain unrecognized within the general population. Identifying vulnerable demographic groups is essential for developing effective prevention strategies and improving access to early mental health interventions.

Schizophrenia remains one of the most challenging disorders in psychiatry, and two comprehensive reviews in this issue address complementary aspects of its management. Zilionytė, Dargytė and Steiblienė summarize the current evidence regarding neuromodulation techniques – including transcranial magnetic stimulation, transcranial direct current stimulation, and electroconvulsive therapy – as promising adjunctive treatment options for patients with persistent symptoms. In parallel, Zubrickas and Ješkevičius review growing evidence linking sleep and circadian rhythm disturbances with the pathophysiology of schizophrenia, highlighting their potential role as biomarkers for disease progression, prognosis, and therapeutic response. Together, these articles illustrate the movement of psychiatry toward biologically informed and individualized treatment approaches.

Personality pathology increasingly receives attention as an important determinant of psychiatric outcomes. In their narrative review, Neverauskaitė and Jakienė explore the complex relationships between obsessive-compulsive personality disorder, anxiety disorders, mood disorders, and suicidal behavior. Their review emphasizes that personality traits influence not only symptom expression but also treatment response and long-term prognosis, reinforcing the importance of comprehensive personality assessment in routine psychiatric practice.

Rare psychiatric syndromes continue to challenge clinicians because of their diagnostic complexity. Remeikaitė and Bulotienė present a review of Capgras syndrome accompanied by illustrative clinical cases, providing valuable guidance for differential diagnosis and improving recognition of this fascinating but uncommon delusional misidentification syndrome.

Reliable assessment instruments remain indispensable in both clinical work and research. This issue introduces the Lithuanian version of the Short Alcohol and Alcohol Problem Perception Questionnaire (SAAPPQ), presented by Justina Kievišienė. The availability of validated instruments facilitates both high-quality research and the evaluation of healthcare professionals' attitudes toward patients with alcohol use disorders, ultimately supporting better addiction care.

Finally, two doctoral thesis summaries demonstrate the interdisciplinary nature of modern health sciences. Giedrė Vaičienė investigates the relationships between objective and subjective movement system indicators and chronic nonspecific low back pain among young office workers, illustrating the growing integration of rehabilitation medicine and behavioral health. Justina Kievišienė explores the concept of the "use of self" in social work with individuals experiencing alcohol-related problems, highlighting the importance of therapeutic relationships and person-centered care within addiction services.

Taken together, the contributions in this issue underscore several important trends shaping contemporary psychiatry. First, mental disorders should be identified earlier, often before formal diagnosis becomes established. Second, advances in neuroscience continue to expand opportunities for precision diagnostics through biomarkers, neurophysiology, and digital assessment. Third, successful psychiatric care increasingly requires integration of biological, psychological, and social perspectives rather than reliance on any single explanatory model.

As psychiatry moves toward precision medicine, collaboration between clinicians, neuroscientists, psychologists, rehabilitation specialists, and public health researchers becomes ever more essential. We hope that the articles presented in this issue will stimulate further research, encourage interdisciplinary collaboration, and ultimately contribute to improving patient care and mental health outcomes.

We sincerely thank all authors, reviewers, and members of the editorial board for their valuable contributions to this issue. Their dedication continues to strengthen the scientific quality and clinical relevance of Biological Psychiatry and Psychopharmacology.

Vesta Steiblienė, MD, PhD
Editor
Biological Psychiatry and Psychopharmacology

Prevalence and severity of depressive symptoms and their association with sociodemographic factors in the Lithuanian population without a previously diagnosed mental disorder

Lietuvos gyventojų, neturinčių diagnozuoto psichikos sutrikimo, depresijos simptomų išreikštumas, paplitimas bei sąsajos su sociodemografiniais rodikliais

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SUMMARY

Introduction and aim. Depression is one of the most common mental disorders worldwide and represents a significant public health concern. However, depressive symptoms may also occur in individuals who have not received a formal psychiatric diagnosis. The aim of this study was to assess the prevalence and severity of depressive symptoms and their association with sociodemographic factors in adults without a previously diagnosed mental disorder.

Methods. A cross-sectional online survey was conducted, with a final analytical sample of 410 participants. Depressive symptoms were assessed using the Patient Health Questionnaire-9 (PHQ-9). Additionally, data were collected on sociodemographic characteristics (gender, age, education, marital status, place of residence, and parental status) and lifestyle factors (physical activity, perceived stress levels, smoking, alcohol consumption, and illicit drug use). Non-parametric statistical tests were used for data analysis.

Results. Clinically significant depressive symptoms (PHQ-9 ≥10) were identified in 22.4% of participants. Women reported significantly higher PHQ-9 scores than men. Higher depressive symptom severity was also observed among younger participants and individuals without children. Increased perceived stress and lower levels of physical activity were significantly associated with higher PHQ-9 scores ($p < 0.001$).

Conclusions. A notable proportion of adults without a prior psychiatric diagnosis reported clinically significant depressive symptoms. This may indicate that depressive symptoms in the community can remain unrecognized, represent subclinical or transient emotional distress, or reflect psychological difficulties that individuals manage without seeking professional help.

Keywords: depression; depressive symptoms; PHQ-9; prevalence; epidemiology; sociodemographic factors

SANTRAUKA

Įvadas ir tikslas. Depresija yra vienas dažniausių psichikos sutrikimų pasaulyje ir svarbi visuomenės sveikatos problema. Tačiau depresijos simptomai gali pasireikšti ir asmenims, kuriems nėra diagnozuotas psichikos sutrikimas. Šio tyrimo tikslas – įvertinti depresijos simptomų paplitimą, išreikštumą bei jų sąsajas su sociodemografiniais rodikliais tarp suaugusiųjų, kuriems anksčiau nebuvo diagnozuotas psichikos sutrikimas.

Metodai. Buvo atlikta skerspjūvio (angl. cross-sectional) internetinė apklausa, kurios galutinę analizuojamą imtį sudarė 410 respondentų. Depresijos simptomai vertinti naudojant Paciento sveikatos klausimyną (PHQ-9). Papildomai rinkti duomenys apie respondentų sociodemografines charakteristikas (lytį, amžių, išsilavinimą, šeiminę padėtį, gyvenamąją vietą, vaikų turėjimą) bei gyvensenos veiksnius (fizinį aktyvumą, patiriamo streso lygį, rūkymą, alkoholio bei narkotinių medžiagų vartojimą). Duomenų analizei taikyti neparametriniai statistiniai testai.

Rezultatai. Kliniškai reikšmingi depresijos simptomai (PHQ-9 ≥ 10) buvo nustatyti 22,4 proc. dalyvių. Moterys pasižymėjo statistiškai reikšmingai aukštesniais PHQ-9 balais nei vyrai. Didesnis depresijos simptomų išreikštumas taip pat buvo stebimas tarp jaunesnio amžiaus respondentų ir asmenų, neturinčių vaikų. Didesnis patiriamo streso lygis ir mažesnis fizinis aktyvumas buvo statistiškai reikšmingai susiję su aukštesniais PHQ-9 balais ($p < 0,001$).

Išvados. Reikšminga dalis suaugusiųjų, kuriems anksčiau nebuvo diagnozuotas psichikos sutrikimas, nurodė patiriantys kliniškai reikšmingus depresijos simptomus. Tai gali rodyti, kad depresijos simptomai bendruomenėje gali likti neatpažinti, atspindėti subklinikinį ar laikiną emocinį distresą arba atspindėti psichologinius sunkumus, su kuriais asmenys susidoroja nesikreipdami profesionalios pagalbos.

Raktažodžiai: depresija; depresijos simptomai; PHQ-9; paplitimas; epidemiologija; demografiniai rodikliai

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INTRODUCTION

Depression is one of the most common mental disorders worldwide and is associated with impaired functioning, reduced quality of life, increased healthcare use, and a higher risk of suicide [1–4]. In Lithuania, approximately one in eight individuals have been diagnosed with a mental or behavioral disorder, with prevalence increasing over the past decade [5]. However, many individuals experiencing depressive symptoms do not seek professional help and remain untreated [6,7].

Research suggests that depressive symptoms exist along a continuum within the general population and are not limited to clinically diagnosed disorders [8–10]. Many individuals experience clinically relevant depressive symptoms without meeting full diagnostic criteria for major depressive disorder or without seeking professional help. These subthreshold or undiagnosed depressive symptoms may nevertheless be associated with impaired functioning, reduced well-being, and an increased risk of developing major depressive episodes in the future [10–12]. Therefore, assessing depressive symptom prevalence in the general population is essential for understanding the broader burden of mental health problems and identifying individuals who may benefit from early intervention.

The Patient Health Questionnaire-9 (PHQ-9) is a widely used screening instrument for depressive symptoms [13,14]. It is a brief self-report measure based on DSM criteria for major depressive disorder [14,15] and has demonstrated good reliability and validity across different populations [14,16,17]. Due to its brevity and strong psychometric properties, it is commonly used in epidemiological studies and population-based surveys [14,16–18].

The PHQ-9 allows both the identification of probable depressive disorder and the assessment of symptom severity. A score of ≥10 is commonly used to indicate clinically significant depressive symptoms and has good sensitivity and specificity for detecting major depressive disorder [16,18]. This cutoff is widely used in clinical and epidemiological research [14,16,17].

Recent studies show that many individuals report moderate to severe depressive symptoms despite not having a formal diagnosis of depression [19–21]. This may be due to stigma, limited access to mental health services, lack of awareness, or attributing symptoms to situational stressors rather than mental illness [7,22].

Untreated depressive symptoms are associated with reduced productivity, interpersonal difficulties, poorer physical health, and an increased risk of more severe psychiatric disorders [5,23]. Therefore, population-based studies using validated screening instruments are important for identifying unmet mental health needs and informing public health strategies.

While many studies examine depression in clinical populations, fewer focus on individuals without a diagnosed mental disorder. Assessing depressive symptoms in this group may reveal a hidden burden of psychological distress not captured by clinical statistics [19–21].

Therefore, the aim of the present study was to assess the prevalence of depressive symptoms using the PHQ-9 questionnaire in a sample of adults and to determine the proportion of individuals experiencing clinically significant depressive symptoms (PHQ-9 ≥10) despite not having a previously diagnosed mental disorder.

MATERIALS AND METHODS

Study Design and Participants

A cross-sectional survey was conducted using an anonymous online questionnaire between 15 December 2025 and 22 February 2026. The questionnaire was administered using an online survey platform (Google Forms) and distributed through social media platforms, specifically Facebook and Instagram. The invitation to participate was shared in various public and private community groups to ensure a diverse range of respondents. The recruitment invitation explicitly stated that the study targeted individuals who had never consulted a psychiatrist or received a formal mental health diagnosis. To ensure data integrity and adherence to this criterion, a mandatory control question regarding prior psychiatric diagnoses was included in the questionnaire. Participation in the survey was voluntary, and adults over the age of 18 were invited to take part in the study. Ethical approval for the study was obtained from the Lithuanian University of Health Sciences Bioethics Center (2025-BEC2-1312).

A total of 438 adults completed the survey. Participants were asked whether they had ever received a diagnosis of a mental disorder from a healthcare professional. Individuals who reported such a diagnosis (n = 28) were excluded from further analyses in order to focus on depressive symptoms among individuals without a previously diagnosed mental disorder. The final analytical sample consisted of 410 participants.

MEASURES

Depressive symptoms

Depressive symptoms were assessed using the Patient Health Questionnaire-9 (PHQ-9) [13,24]. The Lithuanian version of the instrument has demonstrated good psychometric properties, including reliability and construct validity, in previous research conducted in a student population [25]. The PHQ-9 consists of nine items corresponding to the diagnostic criteria for a major depressive episode according to the Diagnostic and Statistical Manual of Mental Disorders (DSM) [15]. Participants were asked to indicate how often they experienced each symptom during the past two weeks. Each item is rated on a four-point Likert scale ranging from 0 (“not at all”) to 3 (“nearly every day”), resulting in a total score ranging from 0 to 27.

A cut-off score of ≥10 was used to indicate clinically relevant depressive symptoms, consistent with validation studies demonstrating optimal sensitivity and specificity for detecting probable major depressive disorder at this threshold [16,17].

Symptom severity categories were defined as follows: minimal (0–4), mild (5–9), moderate (10–14), moderately severe (15–19), and severe depression (20–27) [13].

Sociodemographic and lifestyle variables

The questionnaire included several sections designed to collect data on sociodemographic characteristics and lifestyle

factors. Sociodemographic variables: Participants provided information on gender, age, education level, place of residence, marital status, parental status, employment, and work capacity.

Age was assessed as a categorical variable using predefined age groups (e.g., 18–25, 26–35 years) as specified in the questionnaire design. Due to the small number of respondents in the oldest age categories, these groups were not included in comparative statistical analyses in order to ensure the validity and interpretability of the results.

Lifestyle and health-related factors: Stress was assessed with a single binary question ("Do you often experience stress?", Yes/No). The term "often" was not further defined and reflects the participant's subjective perception of stress frequency. Physical activity was measured by weekly duration ("None", "3 hours/week", "6 hours/week", or "10 hours or more/week"). Health-related behaviors, including smoking, alcohol consumption, and drug use, were assessed using binary (Yes/No) questions. Illicit drug use was not further specified and reflects participants' self-reported use of substances considered illegal.

Exclusion criterion: A specific question regarding prior mental disorder diagnoses based on ICD-10 AM classification was used to ensure participants met the inclusion criteria.

Statistical Analysis

Data were initially organized and processed using Microsoft Excel [26] and subsequently analyzed using IBM SPSS Statistics version 21.0 (IBM Corp., Armonk, NY, USA) [27]. Results were considered statistically significant when $p < 0.05$.

Descriptive statistics were used to summarize the characteristics of the study sample. Continuous variables were presented as means and standard deviations, while categorical variables were reported as frequencies (n) and percentages.

The distribution of PHQ-9 scores was assessed for normality. As the distribution deviated from normality, non-parametric statistical tests were applied. The Mann-Whitney U test was used for comparisons between two independent groups, while the Kruskal-Wallis test was used for comparisons across more than two groups.

In addition, the proportion of participants with clinically significant depressive symptoms (PHQ-9 ≥10) was calculated for descriptive purposes only, and no inferential statistical tests were applied to these comparisons.

RESULTS

The final analytical sample consisted of 410 participants. The sociodemographic characteristics of the study sample are presented in Table 1. The majority of participants were female (73.2%) and most respondents were between 26 and 35 years of age (42.4%). Over half of the participants were married (54.6%), and 59.3% reported having children. The majority of participants reported experiencing frequent stress (67.8%), 22.0% reported no regular physical activity. Most respondents did not report illicit drug use (96.6%).

The mean PHQ-9 score in the total sample was 6.16 (SD = 5.30), with a median score of 4 (IQR 2–9). According to PHQ-9 severity categories, 206 participants (50.2%) had minimal depressive symptoms, 112 (27.3%) had mild symptoms, 50 (12.2%) had moderate symptoms, 34 (8.3%) had moderately severe symptoms, and 8 (2.0%) had severe depressive symptoms. Overall, clinically significant depressive symptoms (PHQ-9 ≥10) were identified in 92 participants (22.4%). The

Table 1. Sociodemographic characteristics of the study sample (N = 410)

Variable	Category	n	%
Gender	Female	300	73.2
	Male	108	26.3
	Other	2	0.5
Age group	18–25 years	30	7.3
	26–35 years	174	42.4
	36–45 years	66	16.1
	46–55 years	72	17.6
	56–65 years	55	13.4
	66–75 years	9	2.2
	76–85 years	2	0.5
	≥86 years	2	0.5
Education	University	254	62.0
	College / non-university	75	18.3
	Vocational	44	10.7
	Secondary	25	6.1
	Primary	10	2.4
	Elementary	2	0.5
Place of residence	City	209	51.0
	Large city	131	32.0
	Rural area	37	9.0
	Small town	33	8.0
Marital status	Married	224	54.6
	In a relationship	92	22.4
	Single	68	16.6
	Divorced	26	6.3
Children	None	167	40.7
	Has children	243	59.3
Frequent stress	Yes	278	67.8
	No	132	32.2
Physical activity	None	90	22.0
	3 hours/week	183	44.6
	6 hours/week	78	19.0
	10+ hours/week	59	14.4
Smoking	Yes	106	25.9
	No	304	74.1
Alcohol consumption	Yes	219	53.4
	No	191	46.6
Drug use	Yes	14	3.4
	No	396	96.6

distribution of depressive symptom severity is presented in Table 2.

Comparisons of PHQ-9 scores across sociodemographic variables revealed several statistically significant differences (Table 3). Women reported significantly higher PHQ-9 scores than men (p = 0.004). In addition, the proportion of participants with clinically significant depressive symptoms (PHQ-9 ≥10) was higher among women (24.3%) compared with men (18.3%).

Statistically significant differences were also observed across age groups (p < 0.001). The highest PHQ-9 scores were observed among participants aged 18–25 years, whereas lower scores were generally observed in older age groups. Due to the very small number of participants in older age categories, these groups were not included in comparative statistical analyses.

Marital status was also significantly associated with depressive symptom severity (p = 0.015). The highest prevalence of PHQ-9 ≥10 was observed among single participants, while the lowest prevalence was found among married participants.

Participants without children reported significantly higher PHQ-9 scores compared with those who had children (p < 0.001).

Among lifestyle variables, depressive symptom severity was strongly associated with frequent stress (p < 0.001). Participants who reported frequently experiencing stress had substantially higher PHQ-9 scores compared with those who did not.

Significant differences in PHQ-9 scores were also observed according to physical activity levels (p < 0.001). Participants who reported no regular physical activity had the highest PHQ-9 scores.

Finally, a statistically significant association was found between depressive symptoms and illicit drug use (p = 0.003), with higher PHQ-9 scores observed among respondents who reported drug use. The relatively small number of participants reporting illicit drug use should be considered when interpreting these findings.

No statistically significant differences in PHQ-9 scores were observed according to education level, place of residence, source of income, work capacity, smoking status, or alcohol consumption (p > 0.05).

Detailed statistical calculations are presented in Table 3.

Table 3. Comparison of PHQ-9 scores between participant groups

Variable	Category	Mean	SD	p
Gender	Female	6.63	5.33	0.004
	Male	4.77	4.90	
Age group	18–25	9.90	6.04	<0.001
	26–35	6.94	5.39	
	36–45	5.12	5.08	
	46–55	5.11	4.70	
	56–65	4.58	4.44	
Marital status	Single	6.95	5.40	0.015
	In a relationship	5.82	4.95	
	Married	5.21	4.72	
	Divorced	6.54	5.15	
Children	None	7.53	5.85	<0.001
	Has children	5.21	4.68	
Frequent stress	Yes	7.63	5.42	<0.001
	No	3.06	3.38	
Physical activity	None	8.29	6.12	<0.001
	3 h/week	5.44	4.78	
	6 h/week	5.51	4.84	
	≥10 h/week	5.85	5.12	
Drug use	No	6.01	5.19	0.003
	Yes	10.29	6.94	
Education	University	6.08	4.90	0.139
	Other*	6.25	5.62	
Place of residence	Large city	6.89	5.17	0.139
	Other**	5.79	5.23	
Smoking	Yes	7.33	6.42	0.108
	No	5.75	4.80	
Alcohol use	Yes	6.65	5.62	0.094
	No	5.59	4.86	

TM – mean; SD – standard deviation; *Other education includes: college, vocational, secondary, primary, and elementary education.; **Other place of residence includes: city, small town, and rural areas.

DISCUSSION

This study found that 22.4% of participants had clinically significant depressive symptoms (PHQ-9 ≥10) despite not having a prior psychiatric diagnosis. This supports previous research indicating that depressive symptoms are widely distributed in the general population and often remain undetected [6,19,28].

Table 2. Distribution of depressive symptom severity according to PHQ-9

PHQ-9 category	Score range	n	%
Minimal	0–4	206	50.2
Mild	5–9	112	27.3
Moderate	10–14	50	12.2
Moderately severe	15–19	34	8.3
Severe	20–27	8	2.0
Clinically significant depressive symptoms	PHQ-9 ≥10	92	22.4

The prevalence observed in this study (22.4%) is higher than estimates reported in large-scale epidemiological studies (10–11%) [19,20]. This difference may be explained by characteristics of the study sample, including potential selection bias due to voluntary participation and recruitment via social media, which may have led to self-selection bias, as individuals experiencing psychological distress may be more likely to engage in mental health-related surveys [7,22]. In addition, the higher proportion of younger participants may have contributed to these findings, as younger individuals may be particularly vulnerable to depressive symptoms due to increased exposure to academic, occupational, and social

pressures, as well as uncertainty regarding future prospects. A high prevalence of reported stress (67.8%), which was associated with higher PHQ-9 scores, may have further influenced the results. Furthermore, the PHQ-9 assesses symptoms over the past two weeks and may capture transient distress rather than clinically diagnosed depression [13,16,19–21].

Only 6.4% of respondents (n = 28) were excluded due to a prior mental disorder diagnosis, which is lower than the prevalence in the Lithuanian population (approximately one in eight) [5]. This discrepancy is likely related to the recruitment strategy, as the study targeted individuals without prior psychiatric diagnoses, making such individuals less likely to participate. Those with a diagnosis who did participate may have overlooked this criterion but were identified through a control question.

Previous studies show that many adults report depressive symptoms without meeting diagnostic criteria or receiving a formal diagnosis [10–12]. These findings support the concept that depressive symptoms exist along a continuum [10,29].

The present study also identified gender differences in depressive symptom severity, with women reporting significantly higher PHQ-9 scores than men. This finding is consistent with epidemiological research showing that depressive disorders occur approximately twice as frequently among women as among men [4,21]. Age-related differences were also observed, with younger participants reporting higher depressive symptom scores, consistent with previous findings [20,21,30].

Lifestyle factors were associated with depressive symptoms, with higher perceived stress linked to higher PHQ-9 scores. Chronic stress is a well-established factor associated with depressive symptoms [4,23]. Lower physical activity was also associated with higher symptom scores, consistent with evidence that regular activity may have protective effects [31].

Illicit drug use was associated with higher depressive symptom scores, whereas no significant associations were observed for alcohol consumption or smoking. This may reflect differences in substance use patterns, as well as methodological factors, since all variables were assessed using binary (yes/no) measures that do not capture frequency or intensity of use. Previous research suggests that stronger associations are more likely to be observed in cases of heavier or problematic use, while illicit drug use may be more closely linked to psychological distress [21].

This study has several limitations. The cross-sectional design does not allow causal inferences. Data were based on self-report and may be subject to reporting bias. Additionally, the use of an online survey may have introduced selection bias.

Age was measured using predefined categories rather than as a continuous variable, limiting the assessment of linear relationships and the inclusion of older age groups in comparative analyses. Stress and illicit drug use were assessed using single self-report items (yes/no), which may have limited measurement precision and introduced variability in interpretation. The study sample was not representative of the Lithuanian population due to the use of convenience sampling and recruitment via social media, resulting in an overrepresentation of women and younger individuals; therefore, the findings should be interpreted with caution.

Despite these limitations, the study has several strengths. The use of a validated screening instrument (PHQ-9) allowed standardized assessment of depressive symptoms [13,16,32]. Furthermore, the study specifically focused on individuals without a previously diagnosed mental disorder, thereby providing insight into the potential burden of undetected depressive symptoms within the general population.

Overall, the results suggest that depressive symptoms may remain unrecognized in a considerable proportion of individuals without a formal psychiatric diagnosis, highlighting the importance of early identification and targeted prevention strategies.

CONCLUSION

A notable proportion of adults without a previously diagnosed mental disorder reported clinically significant depressive symptoms. These findings suggest that depressive symptoms may be relatively common in the general population even in the absence of a formal psychiatric diagnosis. These findings highlight the potential importance of early identification of depressive symptoms in the general population and the need for increased awareness and accessible screening strategies. Further research is needed to better understand whether such symptoms represent unrecognized depression, subthreshold depressive conditions, or temporary psychological distress related to everyday life stressors.

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Effectiveness of neuromodulation methods in the treatment of schizophrenia: a narrative literature review

Neuromoduliacijos metodų veiksmingumas gydant šizofreniją: naratyvinė literatūros apžvalga

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SUMMARY

Introduction. Schizophrenia is a chronic, severe and complex mental disorder. Although antipsychotic medications are effective in reducing positive symptoms, their impact on negative and cognitive symptoms is limited. Treatment resistance is common, affecting about 10% of patients at disease onset and up to 40% in chronic stages. In response to these challenges, neuromodulation techniques have been developed, allowing targeted modulation of brain networks and potentially benefiting the treatment of therapy-resistant symptoms. This review examines recent studies on neuromodulation approaches for schizophrenia. It focuses on methods such as repetitive transcranial magnetic stimulation (rTMS), deep transcranial magnetic stimulation (dTMS), transcranial alternating current stimulation (tACS). The analysis will focus on the efficacy of these methods in improving positive and negative symptoms, cognitive impairaments.

Aim. To review the most recent scientific literature on the effectiveness of neuromodulation techniques in the treatment of schizophrenia, and to evaluate their impact on clinical symptoms, including effects on positive and negative symptoms of schizophrenia, cognitive functions, and treatment outcomes.

Methods. A narrative literature search and analysis were conducted using the PubMed database in December 2025, applying keywords related to schizophrenia and non-invasive neuromodulation techniques (rTMS, dTMS, tACS, and tDCS), identifying 855 records. Publications were selected in multiple stages using filters for the last „10 years", „English" language, „human" studies, and predefined inclusion criteria. Titles and abstracts were screened, duplicates and low-priority studies were excluded, and 39 publications were ultimately included in the final analysis.

Results. The reviewed studies show that non-invasive neuromodulation techniques have variable efficacy in schizophrenia. rTMS demonstrates the strongest evidence, particularly high-frequency stimulation of the left DLPFC, which effectively reduces negative symptoms and shows some cognitive benefits, while effects on auditory hallucinations remain inconsistent. dTMS has not shown reliable efficacy for positive symptoms or cognition. tACS and tDCS appear safe and promising, with preliminary evidence suggesting

SANTRAUKA

Įvadas. Šizofrenija yra lėtinis, sunkus ir kompleksinis psichikos sutrikimas. Nors antipsichoziniai vaistai vertinami kaip veiksmingi mažinant teigiamus sutrikimo simptomus, jų poveikis neigiamiems ir kognityviniams simptomams yra gana ribotas. Gydymo atsparumas yra dažnas - apie 10 proc. pacientų nuo sutrikimo pradžios ir iki 40 proc. lėtiniuose sutrikimo etapuose. Dėl šių iššūkių buvo pradėti taikyti neuromoduliacijos metodai, kurie leidžia tikslingai veikti smegenų tinklus ir gali būti naudingi gydant vaistams atsparius sutrikimo simptomus. Šioje apžvalgoje nagrinėjami naujausi tyrimai apie neuromoduliacijos metodus, taikomus šizofrenijos gydymui. Dėmesys skiriamas tokiems metodams kaip pasikartojanti transkranijinė magnetinė stimuliacija (rTMS), gilioji transkranijinė magnetinė stimuliacija (dTMS), transkranijinė kintamosios srovės stimuliacija (tACS) ir transkranijinė tiesioginės srovės stimuliacija (tDCS).

Tikslas. Apžvelgti naujausią mokslinę literatūrą apie neuromoduliacijos metodų veiksmingumą gydant šizofreniją, įvertinti jų poveikį klinikiniams simptomams, apimant poveikį teigiamiems, neigiamiems šizofrenijos simptomams ir kognityvinėms funkcijoms bei gydymo rezultatams.

Metodologija. Literatūros paieška ir analizė buvo atlikta naudojant PubMed duomenų bazę 2025 m. gruodį, taikant raktažodžius, susijusius su šizofrenija ir neinvazinės neuromoduliacijos metodais (rTMS, dTMS, tACS ir tDCS), identifikuojant 855 įrašus. Publikacijos buvo atrenkamos keliais etapais, taikant filtrus paskutinių „10 metų" laikotarpiui, „anglų" kalba, „žmogaus" tyrimai ir iš anksto nustatytus įtraukimo kriterijus. Buvo peržiūrėti straipsnių pavadinimai ir santraukos, pašalinti pasikartojantys ir mažesnio prioriteto tyrimai, o galutinei analizei įtrauktos 39 publikacijos.

Rezultatai. Apžvelgti tyrimai rodo, kad neinvazinės neuromoduliacijos technikos pasižymi nevienodu veiksmingumu gydant šizofreniją. rTMS turi stipriausią įrodymų bazę, ypač taikant didelio dažnio stimuliaciją kairiajame DLPFC, kuri veiksmingai mažina neigiamus simptomus ir rodo tam tikrą teigiamą poveikį kognityvinėms funkcijoms, tačiau poveikis klausos haliucinacijoms išlieka nevienareikšmis. dTMS neparodė patikimo veiksmingumo

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potential benefits for positive, negative symptoms, and selected cognitive domains; however, findings are heterogeneous and require confirmation in larger, well-controlled trials.

Conclusions. Neuromodulation should be seen as additional to antipsychotic medication treatment for schizophrenia-especially in treatment-resistant cases. Many methods still lack strong evidence for the treatment efficacy, although rTMS, tDCS show the most promising outcomes in current studies.

Keywords: schizophrenia, repetitive transcranial magnetic stimulation (rTMS), deep transcranial magnetic stimulation (dTMS), transcranial alternating current stimulation (tACS), transcranial direct current stimulation (tDCS).

teigiamiems simptomams ar kognityvinėms funkcijoms. tACS ir tDCS laikomi saugiais ir perspektyviais metodais, o preliminarūs duomenys rodo galimą naudą teigiamiems ir neigiamiems sutrikimo simptomams bei atskiroms kognityvinėms funkcijoms, tačiau rezultatai yra heterogeniški ir reikalauja patvirtinimo didesniuose, gerai kontroliuojamuose tyrimuose.

Išvados. Neuromoduliacija turėtų būti naudojama kaip papildomas, šizofrenijos gydymo metodas, ypač gydymui atspariems atvejams. Daugelio neuromoduliacinių metodų veiksmingumas vis dar nėra pakankamai įrodytas, nors apžvelgtoje literatūroje duomenys apie rTMS, tDCS pateikia daugiausiai žadančius gydymo veiksmingumo rezultatus.

Raktažodžiai: šizofrenija, pasikartojanti transkranialinė magnetinė stimuliacija (rTMS), gilioji transkranijinė magnetinė stimuliacija (dTMS), transkranijinės kintamosios srovės stimuliacija (tACT), transkranijinė tiesioginės srovės stimuliacija (tDCS).

INTRODUCTION

Schizophrenia is a severe and complex psychiatric disorder that affects approximately 1% of the global population and is recognized as one of the leading causes of disability worldwide [1]. First described by Emil Kraepelin in the 1890s as "dementia praecox," schizophrenia has long been understood as a disorder affecting multiple brain functions, including memory, attention, motor activity, and perception [2]. The globally prevalence of schizophreni has increased from 13.1 million cases in 1990 to 20.9 million in 2016, contributing substantioally to years lived with disability [3].

Schizophrenia most commonly emerges in late adolescence or early adulthood, with the greatest disease burden occuring between 25 and 54 years of age-coinciding with peak productive years [3]. Functional outcomes vary widely, with some individuals maintaining relatively preserved functioning, while others experience severe disability and a reduced life expectancy [1].

The pathophysiology of schizophrenia involves complex interactions among multiple neurotransmitter systems and neural circuits, particularly within frontal, temporal, and mesostriatal regions [4]. Dysconnectivity within frontotemporal and frontoparietal networks has been associated with both positive and negative symptoms [5,6], whereas hypoactivity of the prefrontal cortex and disrupted connectivity with temporoparietal and contralateral regions are linked to cognitive impairments [7].

Clinically, schizophrenia is characterized by positive symptoms such as delusions, hallucinations and disorganized thinking; negative symptoms including affective flattening amotivation and alogia; as well as cognitive dysfunction that significantly impairs social and occupational functioning [8-10]. Notably, cognitive and social functioning often decline prior to the onset of psychosis [11].

Although antipsychotic medications are effective in reducing positive symptoms, their efficacy in treating negative and cognitive symptoms remains limited. These symptom domains are major predictors of long-term functional outcomes [10,12]. Approximately 10% of patients are resistant to standard treatments at disease onset, with this proportion increasing to around nearly 40% in chronic stages of illness [9].

In response to these therapeutic challenges and limitations, various neuromodulation techniques have been developed to target specific dysfunctional neural circuits and enhance neuroplasticity. These include transcranial magnetic stimulation (TMS), repetitive TMS (rTMS), deep TMS (dTMS), transcranial alternating current stimulation (tACS). Compared to pharmacotherapy, these approaches offer the potential for more precise modulation of specific brain regions and may provide benefits for negative, cognitive and treatment-resistant symptoms [12].

Despite advances in pharmacological treatment, schizophrenia remains a heterogeneous disorder associated with a substantial socioeconomic burden, highlighting the need for alternative and adjunctive therapeutic strategies [13].

Therefore, the aim of this narrative literature review is to evaluate the effectiveness of neuromodulation methods in the treatment of schizophrenia, with particular focus on positive, negative symptoms domains and cognitive functioning as well as on clinical outcomes.

METHODS

A narrative literature search, review and analysis were conducted using the computerised databases – PubMed in December 2025. The search strategy included the following keywords: schizophrenia and "repetitive transcranial magnetic stimulation," or "deep transcranial magnetic stimulation," or "transcranial alternating current stimulation," or "transcranial direct current stimulation," resulting in a total of 855 identified bibliographic records. The selection of publications was carried out in several stages. In the first stage, after applying filters for the last "10 years", "English" language, and "human" studies, 451 publications were excluded. In the second stage, the "free full text" filter was applied, leading to the exclusion of an additional 215 publications. In the third stage, inclusion criteria were applied: clinical trials, controlled clinical trials, randomized controlled trials, meta-analyses, systematic reviews, and narrative reviews; 66 publications that did not meet these criteria were excluded, and the remaining publications were selected for further analysis.

During the subsequent evaluation, article titles and abstracts were reviewed to determine whether the studies directly assessed the effects of neuromodulation on schizophrenia;

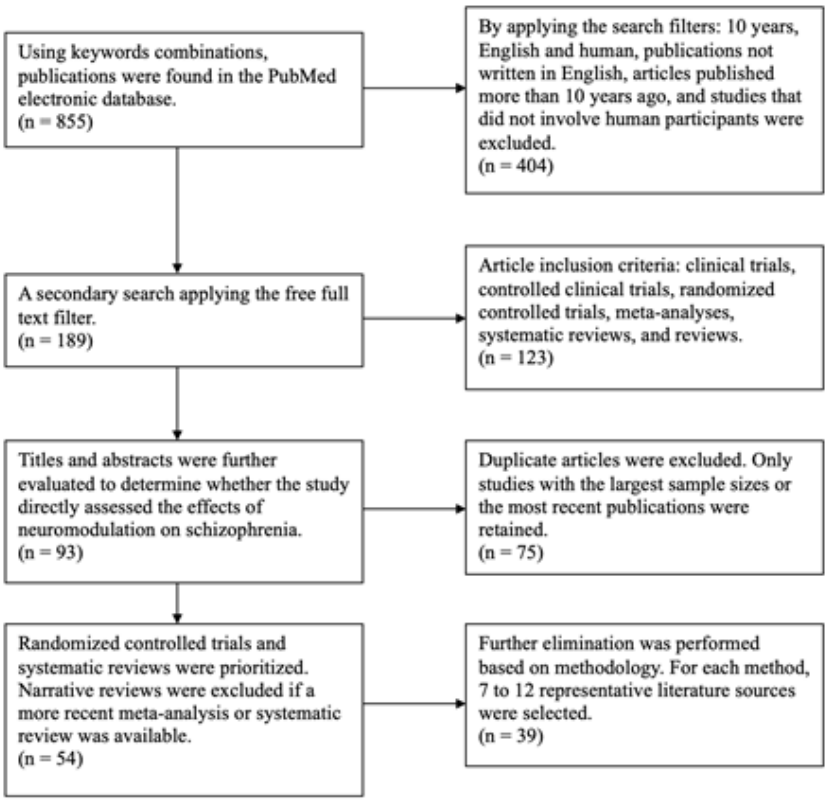

Figure 1. Study selection flow diagram.

theoretical reviews, editorials and opinion articles without empirical data were also excluded, resulting in the exclusion of 30 publications. After removing duplicate articles and retaining only studies with the largest sample sizes or the most recent data, a further 18 publications were excluded. In the prioritization process, preference was given to randomized controlled trials and systematic reviews, while narrative reviews were excluded when a more recent meta-analysis or systematic review was available, leading to the exclusion of 21 publications. During the representativeness balancing stage, 7 to 12 representative literature sources were selected for each research method, resulting in the exclusion of an additional 15 publications. After all selection stages, 36 sources remained; however, due to the limited number of studies-three older studies were additionally included, resulting in a final literature list of 39 publications.

RESULTS

The reviewed 39 studies indicate that neuromodulation techniques show varying effectiveness in the treatment of schizophrenia.

1. Transcranial magnetic stimulation (TMS)

TMS is a noninvasive neuromodulation technique that modulates cortical activity through rapidly changing magnetic field, inducing low-intensity electrical currents in targeted brain regions. The technique was first developed by Barker and colleagues and is fundamentally derived from Faraday's nineteenth-century discovery of electromagnetic induction. Over the past three decades, TMS has been extensively investigated and has shown clinical promise for the management of psychiatric disorders in adults [14]. Clinical evidence supports its efficacy across a multiple psychiatric conditions, including schizophrenia, major depressive disorder, obsessive-compulsive disorder, and substance use disorders. In schizophrenia, high-frequency stimulation of the left dorsolateral prefrontal cortex (DLPFC) has been associated with improvements in negative symptoms. Overall, treatment response appears strongly dependent on stimulation parameters, highlighting the importance of disorder- and site-specific protocol optimization to maximize therapeutic outcomes [15].

Transcranial magnetic stimulation is a promising noninvasive technique for psychiatric disorders, particularly schizophrenia. Its clinical effectiveness depends on optimized, disorder- and target-specific stimulation parameters.

2. Repetitive transcranial magnetic stimulation (rTMS)

rTMS is a non-invasive brain stimulation technique that can modulate the activity of discrete cortical regions, allowing direct manipulation of local brain activation and indirect manipulation of the target's associated neural networks [16]. rTMS involves the application of a pulsatile magnetic field over the scalp to generate an electric field within a targeted brain region. This induced electric field modulates ion flow across neuronal membranes, resulting in changes in neuronal polarization and activity. Consequently, rTMS alters the functional state of neurons in the stimulated area. Evidence suggests that these effects may be mediated by mechanisms similar to long-term potentiation (LTP) and long-term depression (LTD), potentially leading to lasting modifications in neural circuitry. rTMS is a flexible neuromodulatory

Table 1. Efficacy of high-frequency rTMS in reducing negative symptoms of schizophrenia: summary of studies

Author (year)	Study aim	Sample characteristics	Main findings
Quan WX et al. (2015) [22]	Evaluate high-frequency rTMS over the left DLPFC for negative symptoms.	117 patients with prominent negative symptoms. 20 sessions of active rTMS (n = 78) or sham rTMS (n = 39).	Negative symptoms improved significantly in active group; improvements maintained up to 24 weeks; no significant change in overall clinical severity.
Li Z et al. (2016) [21]	To investigate the effect of rTMS on negative symptoms in schizophrenia, and the timing of its effects.	47 patients with schizophrenia, randomized into: active rTMS over left DLPFC (n = 25) and sham stimulation (n = 22).	No significant difference at 4 weeks; significant reduction in negative symptoms in the active rTMS group at 8 weeks; indicates a delayed effect of rTMS on negative symptoms.
Zhuo K et al. (2019) [23]	Examine adjunctive 20-Hz rTMS on negative symptoms.	60 patients, 4 - week, randomized, double-blind, sham-controlled trial. Participants were assigned to either active rTMS (n = 33) or sham rTMS (n = 27).	Significant reduction in negative symptoms.
Kumar N et al. (2020) [20]	Evaluate efficacy of high-frequency (20 Hz) rTMS over the left DLPFC of negative symptoms.	100 patients with schizophrenia and predominantly negative symptoms randomized, sham-controlled, double-blind 20 sessions over 4 weeks.	Significant improvement in negative symptoms (anhedonia, alogia, avolition, attention impairment) vs. sham; effects maintained through follow-up.
Brandt SJ et al. [2021] [16]	Explore rTMS for schizophrenia symptoms and advances integrating neuroimaging.	Literature review of treatment-resistant schizophrenia.	rTMS modulates cortical activity and neural networks; mixed results due to heterogeneity; neuroimaging integration may improve precision and outcomes.
Patel S et al. (2023) [24]	Analyze safety and efficacy of rTMS in schizophrenia, depressionm, and obsessive-compulsive disorder.	Umbrella meta-analysis: 28 meta-analyses (9 on schizophrenia).	rTMS significantly reduced negative symptoms in schizophrenia; effectiveness depends on target site, frequency, and neuropathology.
Wang WY et al. (2024) [25]	Assess neuronavigation - enhanced rTMS for schizophrenia and depression.	Review of clinical studies; sample sizes not specified.	Neuronavigation improves precision of targeting, leading to enhanced efficacy; allows individualized targets based on structural/functional brain variations.
Yi S et al. (2024) [18]	Integrate evidence on rTMS for negative symptoms.	20 reviews, 33 analyses; patients with prominent negative symptoms; high-frequency rTMS >100% motor threshold, ≥3 weeks.	rTMS significantly decreased negative symptoms in 26 analyses; high-frequency DLPFC stimulation for ≥3 weeks is most effective; moderate-quality evidence for anhedonia, avolition, social withdrawal.

technique, and its physiological effects depend on stimulation parameters. High-frequency stimulation (10–25 Hz) generally increases cortical excitability, whereas low-frequency stimulation (≈1 Hz) tends to produce cortical inhibition [16].

These neurophysiological effects of rTMS are particularly relevant for schizophrenia, as they may help normalize dysregulated cortical circuits associated with negative symptoms and cognitive deficits [17].

2.1. The effects of repetitive transcranial magnetic stimulation on negative symptoms

Negative symptoms constitute core characteristics of schizophrenia and profoundly impact daily functioning and overall quality of life [18].These symptoms are frequently unresponsive to antipsychotic medications and may be resistant to conventional treatment [19,20].

Repetitive transcranial magnetic stimulation (rTMS) represents an alternative therapeutic approach that may be effective in alleviating negative symptoms [19]. Hypofrontality is thought to play a key role in the pathogenesis of negative symptoms, and rTMS may contribute to its restoration [6]. It is important to note that the therapeutic effects of rTMS may not manifest immediately but may emerge following the completion of a treatment course; for instance, benefits have been observed as late as eight weeks after treatment initiation [21].

In summary, the studies presented in the table below (table 1) rTMS, particularly high-frequency stimulation targeting the dorsolateral prefrontal cortex (DLPFC), is a promising intervention for alleviating negative symptoms in patients with schizophrenia. Evidence from randomized controlled trials, meta-analyses, and reviews indicates that rTMS can significantly reduce symptoms such as anhedonia, avolition, alogia, and social withdrawal, with effects maintained for weeks to months. Overall, rTMS represents a valuable adjunctive therapy, especially for patients who are resistant to conventional pharmacological treatments [8–17].

2.2. The effects of repetitive transcranial magnetic stimulation on positive symptoms (for auditory hallucinations)

Auditory hallucinations (AVH) are among the most common symptoms of schizophrenia, significantly increasing the risk of suicide and violence in patients. rTMS is considered a potential treatment for AVH. In recent years, image-guided rTMS has received increasing attention, as it allows more precise targeting of brain regions associated with hallucinations. In their publication, Hua Q et al. investigated the efficacy of image-guided rTMS for treating auditory hallucinations in patients with schizophrenia. The study enrolled 62 patients experiencing AVH. Findings demonstrated that active rTMS was significantly more effective than sham (placebo) treatment in alleviating AVH symptoms, with the beneficial effects maintained throughout the entire follow-up period [26]. These findings indicate that image-guided rTMS could serve as an effective adjunctive intervention for alleviating auditory hallucinations in patients with schizophrenia.

Nonetheless, the literature also reports conflicting findings. In a study conducted by Patel S et al., rTMS was found to have no statistically significant effect on auditory hallucinations. In this study, rTMS did not demonstrate consistent efficacy in alleviating auditory hallucinations in patients with schizophrenia [24].

In summary, image-guided rTMS appears to be a promising adjunctive intervention for alleviating auditory hallucinations in patients with schizophrenia. Nevertheless, existing findings are inconsistent, highlighting the need for further large-scale, methodologically rigorous studies to reliably evaluate the efficacy of rTMS in the treatment of these symptoms.

2.3. The effects of repetitive transcranial magnetic stimulation on cognitive functions

Cognitive deficits represent a core feature of schizophrenia, profoundly affecting patients daily functioning and overall quality of life [18]. One emerging research avenue is the use of rTMS to modulate brain activity. Evidence suggests that high-frequency rTMS can induce significant and lasting improvements in specific cognitive domains, including executive functions, working memory, and language abilities [18]. A study assessing the impact of rTMS on cognitive functions enrolled 60 patients with schizophrenia. The findings indicated that rTMS did not result in significant improvements in working memory or other cognitive domains [23]. Jiang Y et al. conducted a meta-analysis involving 351 patients, which indicated that high-frequency rTMS significantly enhanced working memory compared with placebo stimulation. The maximal effect was observed when rTMS targeted the DLPFC, with the total number of pulses not exceeding 30,000. No significant improvements were observed in other cognitive domains, including executive functions, attention, information processing speed, or language abilities [27].

Nonetheless, high-frequency rTMS exhibited long-lasting benefits for working memory and, in some cases, for language function in patients with schizophrenia [18,27].

Despite the limited quality of available evidence, rTMS demonstrates potential efficacy in improving specific cognitive functions in patients with schizophrenia [18].

In summary, high-frequency rTMS shows potential benefits for improving working memory in patients with schizophrenia, particularly when targeting the DLPFC, although evidence for broader cognitive improvements remains limited and inconsistent.

3. Deep transcranial magnetic stimulation (dTMS)

Deep transcranial magnetic stimulation (dTMS) is an advanced variation of conventional TMS, designed to stimulate deeper and broader brain areas using a specialized H-coil [28]. Similar to traditional TMS, it induces neuronal depolarization through focused magnetic pulses [28,29]. Moreover, a distinct H-coil structure can generate an electromagnetic field that reaches approximately 4 cm beneath the skull surface [28,30]. The magnetic field produced by the H-coil enables modulation of deeper layers of the prefrontal cortex [28,29]. Clinically, this deeper reach may contribute to faster therapeutic outcomes, potentially requiring shorter treatment durations compared to standard TMS [31].

dTMS has been studied as an adjunctive treatment for individuals with medication-resistant schizophrenia. Open-label trials have suggested potential benefits in reducing auditory hallucinations, improving negative symptoms, and enhancing cognitive performance [28,29].

3.1. The effects of deep transcranial magnetic stimulation on positive symptoms (for auditory hallucinations)

Hyperactivity in the left temporoparietal cortex, a brain region associated with speech perception and often targeted using TMS. dTMS is designed to reach subcortical structures more effectively by summing electromagnetic fields from multiple peripheral coil windings [32].

A sham-controlled study included 18 patients, who received deep stimulation via the Brainsway H1 coil, directed at the left temporoparietal cortex over a 10- day period. Each session lasted 10 minutes and used low-frequency stimulation (1 Hz) at 110% of the motor threshold. The sham treatment replicated the sensations and sound of stimulation but produced only a minimal electric field within the brain. This study did not show any significant differences between real and sham stimulation [32].

Supporting this, a recent systematic review found little reliable evidence for the antipsychotic effects of dTMS in schizophrenia, particularly in patients with persistent auditory hallucinations. The review suggests that limiting stimulation to the left temporoparietal cortex may not be sufficient, especially given that multiple brain regions could be involved [31].

Current evidence does not strongly support dTMS targeting the left temporoparietal cortex as an effective intervention for treatment-resistant auditory hallucinations in schizophrenia (Table 2). The likely involvement of broader neural circuits, along with possible limitations in stimulation parameters, may explain the lack of consistent clinical benefit [31,32].

Further research is needed to refine both target selection and stimulation protocols before dTMS can be recommended for auditory hallucinations [31,32].

Table 2. Efficacy of dTMS in reducing auditory hallucinations symptoms of schizophrenia: summary of studies

Author (year)	Study aim	Sample characteristics	Main findings
Xue J, Zhai Z, Li X et. al. (2025) [30]	Deep transcranial magnetic stimulation over the anterior cingulate cortex in treatment-resistant schizophrenia.	17 patients with schizophrenia undergoing 20 sessions of 1 Hz dTMS over 4 weeks, acitve (n = 8) and sham stimulation (n = 9).	No statistical difference between the active and the sham treated groups.
Rosenberg et. al. (2012) [32]	Low-frequency deep TMS to the left temporoparietal cortex.	18 schizophrenic patients with refractory auditory hallucinations were recruited. Participants were assigned to either active dTMS (n = 9) or sham dTMS (n = 9).	Non-significant differences.

3.2. The effects of deep transcranial magnetic stimulation on cognitive functions

Deep transcranial magnetic stimulation (dTMS) has been studied as a possible intervention for cognitive symptoms in schizophrenia, but so far the findings have been inconclusive.

Open-label studies suggested that dTMS might lead to cognitive improvements. However, sham-controlled trials did not find a significant effect. Both the real and sham groups showed similar improvements, making it difficult to attribute the changes to dTMS [33,34].

dTMS appears less reliable in improving cognitive or clinical symptoms in schizophrenia. Reviews suggest that observed benefits may not be directly tied to active brain stimulation [33,34].

However, none of the available studies reported cognitive worsening or memory impairment after dTMS. This supports the idea that the procedure is at least cognitively safe for individuals with schizophrenia [33,34].

Overall, dTMS may not be an effective treatment for treating cognitive symptoms of schizophrenia. More controlled trials are needed, especially those that fine-tune stimulation parameters and better define treatment targets [33,34].

4. Transcranial alternating current stimulation (tACS)

Transcranial alternating current stimulation (tACS) is a noninvasive technique that delivers sinusoidal electric currents to the scalp, modulating cortical oscillations and promoting synaptic plasticity. In schizophrenia, tACS has been investigated as a tool to normalize abnormal brain rhythms linked to cognitive and behavioral deficits [35]. tACS is also feasible for home use and allows individualization based on patient anatomy and functional characteristics [36].

4.1. The effects of transcranial alternating current stimulation on positive symptoms

Preliminary trials suggest that tACS is safe, well-tolerated, and may reduce positive symptoms such as auditory hallucinations [36,37]. However, its effectiveness appears to depend on stimulation parameters such as frequency, intensity, and electrode placement. Most studies are small and methodologically heterogeneous, indicating the need for larger, well-controlled trials to confirm efficacy and optimize protocols [38,39].

In particular, 10 Hz tACS has demonstrated potential for reducing persistent delusions. In patients experiencing ongoing delusional symptoms, add-on treatment administered twice daily for 20 minutes was associated with a significant reduction in delusion severity as early as the fifth day of stimulation. Together, these findings indicate that 10 Hz tACS may represent a promising therapeutic approach for positive symptoms, although further systematic investigation is required [40].

4.2. The effects of transcranial alternating current stimulation on negative symptoms and cognitive functions

Preliminary trials suggest that tACS is safe and well tolerated and may improve negative symptoms and cognition, with theta- and gamma-frequency protocols showing particular potential for enhancing these outcomes [36,37].

Haller N et al. demonstrated that patients who received 40 Hz prefrontal tACS stimulation twice daily for 10 minutes exhibited improvements in negative symptoms and cognitive impairment [41]. Relatedly, tDCS has also ademonstrated potential for improving cognition and reducing symptoms in schizophrenia, particularly when targeting frontotemporal networks [42,43].

Overall, tACS is a promising, safe, and well-tolerated adjunctive intervention for schizophrenia, with potential to improve positive and negative symptoms as well as cognition. However, larger, well-controlled studies are needed to establish optimal stimulation protocols and confirm its clinical efficacy.

5. Transcranial direct current stimulation (tDCS)

Transcranial direct current stimulation (tDCS) is a non-invasive neuromodulation approach that delivers low-intensity, continuous electrical current to the scalp [44,45]. The technique involves the application of a weak current, typically 1-2 mA, through at least two electrodes, resulting in measurable effects on cortical tissue and excitability [45,46].

Compared with other neuromodulation methods such as rTMS, tDCS provides several clinical advantages, including lower cost, high portability, and good tolerability [44,46]. Due to these practical and clinical benefits, tDCS represents a promising and accessible therapeutic tool for neuromodulation in neuropsychiatric disorders [46].

5.1. The effects of transcranial direct current stimulation on cognitive functions

Cognitive deficits in schizophrenia often affect working memory, executive function, attention, speech, and sometimes appear as broader impairments. In Stuchlikova Z and Klirova M literature review, 12 randomized controlled trials have investigated the use of tDCS to improve cognition, but the findings remain mixed. Anodal stimulation over the left prefrontal cortex was the most frequently used method that

showed the most potential. In most studies, the cathode was placed over the opposite orbitofrontal area, prefrontal cortex, or at the vertex. While activating the prefrontal cortex with tDCS may enhance the effects of cognitive training, the evidence is still inconclusive, and further research is needed [45].

In the study by Jeon et al., 56 patients were enrolled- 26 in the active and 28 in the sham group. After ten treatment sessions, no significant differences in cognitive outcomes were found between the groups. However, the study had limitations, including a small sample size [47].

tDCS may be a promising intervention for cognitive deficits in schizophrenia. However, further research is needed, that includes individualized stimulation protocols and tDCS longevity [48].

5.2. The effects of transcranial direct current stimulation on negative symptoms

People with schizophrenia often show reduced activity in the brain's prefrontal cortex. This has been linked to their negative symptoms [49]. These findings show that tDCS, particularly anodal stimulation over the left dorsolateral prefrontal cortex, may counteract the frontal hypoactivity associated with negative symptoms in schizophrenia [50]. As a result, tDCS has emerged as a promising alternative neuromodulation technique [49].

A study by Valiengo et. al. tested tDCS on 100 patients with mostly negative symptoms. In this study, active tDCS was more effective than sham stimulation in reducing negative symptoms. Patients receiving active tDCS showed higher response rates, defined as a 20% improvement in negative symptoms [49].

A meta-analysis by Yu et al., which included the Valiengo et al. trial, reviewed 12 studies on tDCS for negative symptoms. Only 4 of these showed significant clinical improvement, while the remaining studies failed to demonstrate consistent therapeutic effects in the treatment of schizophrenia [51].

Furthermore, inconsistency in the studies underlines the need for further high- quality trials with larger sample sizes, extended follow- up periods [51].

5.3. The effects of transcranial direct current stimulation on positive symptoms (for auditory hallucinations)

Several case studies have reported that tDCS can reduce auditory hallucinations in patients with schizophrenia. In these studies, the cathode was placed over the left temporo-parietal junction, and the anode over the left dorsolateral prefrontal cortex. Most protocols used two tDCS sessions per day for five consecutive days. Active tDCS led to a significantly greater reduction in AVH than sham stimulation [49,52,53].

Chang et. al. study with a larger patient group showed that tDCS had no significant effect on treating AVH symptoms in schizophrenia [54]. This shows that, to confirm the effectiveness of tDCS in treating AVH, further studies with larger patient samples are needed.

Although some studies show significant improvement in AVH symptoms, they were conducted a long time ago with small study groups, which suggest that the results may be insufficient.

DISCUSSION

This narrative literature review evaluated the effectiveness of various neuromodulation methods in the treatment of schizophrenia, with a focus on clinical symptoms, cognitive functioning, and treatment response. Overal, the findings suggest that neuromodulation approaches may serve as promising adjunctive intervention alongside standard pharmacological treatment, particulary for symptoms that remain insufficiently controlled by antipsychotic medication. This growing interest in non-invasive neuromoduliation reflects the need for additional therapeutic strategies targeting the complex neurobiological mechanism underlying schizophrenia.

Among the reviewed techniques, rTMS demonstrated the most consistent evidence of clinical benefits particularly for negative symptoms. Several randomized controlled trials, meta-analyses, and systematic reviews have reported reduction

Table 3. Efficacy of tDCS in reducing auditory hallucinations symptoms of schizophrenia: summary of studies

Author (year)	Study aim	Sample characteristics	Main findings
Bose A et. al. (2018) [52]	Effects of fronto-temporal tDCS stimulation on AVH.	25 patients with schizophrenia undergoing twice a day tDCS 20-min sessions. The active stimulation (n = 12) and sham stimulation group (n = 13).	Significant reduction of AVH in the active group.
Mondino M et. al. (2015) [53]	Effects of fronto-temporal tDCS stimulation on AVH.	23 schizophrenic patients received 10 active sessions (2 mA, 20min) or sham tDCS (2 mA for the first 30 seconds). Participants were assigned to either active tDCS (n = 11) or sham tDCS (n = 12).	There is a significant difference between active and sham groups. In the active group AVH is reduced by 28% compared to 10% for the sham group.
Brunelin J et. al. (2012) [49]	tDCS treatment for hallucinations.	30 schizophrenic patients received 10 active sessions (2 mA, 20min) or sham tDCS (2 mA for the first 40 seconds). Active tDCS (n = 15) or sham (n = 15).	Significant reduction after active tDCS compared to sham stimulation. 31% reduction in hallucination severity, compared with an 8% reduction after 10 sham sessions.
Chang et. al. (2018) [54]	Fronto-temporal tDCS in the treatment of AVH.	60 schizophrenic patients received 10 active sessions (2 mA, 20min. Active tDCS (n = 30) or sham (n = 30).	No significant changes in the severity of AVH.

symptoms such as anhedonia, avolition, alogia, and social withdrawal, with effects maintained for weeks to months [8-17]. However the effects of rTMS on auditory hallucinations and cognitive functions, more less clear, suggesting that further well-designed clinical trials are necessary, as current evidence is inadequate. Compared with other neuromodulation methods reviewed in this study, rTMS currently has the strongest evidence base, as it has been investigated in a larger number of randomized controlled trials and meta-analyses.

dTMS was theoretically expected to offer advantages due to its ability to reach deeper brain structures [30]. However, available clinical evidence does not consistently support its efficacy for auditory hallucinations or cognitive impairments in schizophrenia. The limited effects suggest that simply increasing the depth of stimulation is insufficient. Therefore, selecting the right targets and individualized stimulation protocols is important for achieving better clinical outcomes.

Meanwhile, tACS has emerged as a potentially safe and well- tolerated neuromodulation technique with the potential to benefit both positive and negative symptoms associated with schizophrenia [35]. However, available clinical studies are inconsistent due to small sample sizes and methodological variability. Consequently, the clinical effectiveness of tACS remains uncertain and requires further inverstigation in larger trials.

tDCS may be beneficial for cognitive impairments, negative symptoms and auditory hallucinations (AVH) in individuals with schizophrenia. Anodal stimulation of the left prefrontal cortex has shown potential for improving cognitive function [45]. However, findings across studies have been inconsistent. Furthermore, stimulation targeting the left dorsolateral prefrontal cortex has also been explored for negative symptoms. While some studies have reported clinically meaningful improvements, meta- analyses suggest that such effects are not consistently replicated [50,51]. Moreover, tDCS has shown promising results in the reduction of AVH, although additional research is needed to confirm the effectiveness as a treatment for schizophrenia. Differences in study results may be related to variations in stimulation parameters, treatment duration, sample size, and the heterogeneity of study populations.

Finally, the included studies differed in their design, stimulation protocols, and outcomes measures, which makes direct comparison of the results more challenging.

CONCLUSION

This review suggests that neuromodulation techniques should not be considered as a primary treatment for schizophrenia, but rather as adjunctive interventions. They are particularly valuable for patients with treatment- resistant schizophrenia. This review highlights that many neuromodulation techniques still lack well- conducted studies to confirm their effectiveness in threatening schizophrenia.The available evidence suggests the non-invasive neuromodulation techniques differ in their therapeutic impact on schizophrenia. Of these methods, rTMS is supported by the strongest body of evidence, particularly when high-frequency stimulation is applied to the left DLPFC. This approach has been consistently associated with reductions in negative symptoms, such as anhedonia, avolition, and social withdrawal. In addition, rTMS appears to confer modest and domain-specific cognitive benefits, most notably in working memory. By contrast, its effects on positive symptoms, especially auditory hallucinations, have been less consistent across studies. dTMS, on the other hand, has not demonstrated clear or reliable efficacy in improving positive symptoms or cognitive deficits in sham-controlled trials. tACS and tDCS are generally considered safe and well tolerated. Early findings indicate that these techniques may offer benefits for both positive and negative symptoms, as well as selected cognitive domains. Nevertheless, the evidence remains heterogeneous, and further large-scale, methodologically robust studies are necessary to establish their clinical effectiveness and to define optimal stimulation protocols.

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The role of sleep and circadian rhythm disturbances in the pathophysiology of schizophrenia and their potential as biomarkers: a literature review

Miego ir cirkadinio ritmo sutrikimų vaidmuo šizofrenijos patofiziologijoje ir jų, kaip biomarkerių potencialas: literatūros apžvalga

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SUMMARY

Introduction. Sleep disturbances are highly prevalent in patients with schizophrenia and are increasingly recognized as an integral component of the disorder rather than merely secondary symptoms. Growing evidence suggests that alterations in sleep architecture and circadian rhythms are closely linked to neurobiological mechanisms and clinical manifestations of schizophrenia.

Aim. To review the biological mechanisms underlying sleep disturbances in schizophrenia and to evaluate their potential role as biomarkers of disease progression.

Methods. A literature search was conducted using the PubMed database, including studies published up to April 5, 2026. Relevant publications were identified using combinations of the following Medical Subject Headings (MeSH) terms: "schizophrenia", "sleep wake disorders", "circadian rhythm" and "biomarkers". A total of 23 peer-reviewed full-text articles published in English within the past 10 years were included in the analysis. Descriptive analysis was performed to summarize the findings.

Results. Sleep disturbances are common in schizophrenia and affect approximately 30–80% of patients. They are associated with poorer clinical outcomes, including more severe symptoms, cognitive impairment, and reduced quality of life. Polysomnographic studies have demonstrated shorter total sleep time, reduced slow-wave sleep, and alterations in rapid eye movement (REM) sleep parameters. Neurophysiological alterations, such as decreased sleep spindle density, reflect underlying thalamocortical dysfunction. Circadian rhythm disturbances are also frequent and include irregular sleep–wake patterns and altered melatonin secretion, and these changes can be assessed using objective measures. Growing evidence suggests that sleep-related parameters such as spindle activity, circadian variability, and inflammatory markers may serve as potential biomarkers, although their clinical applicability remains limited.

Conclusions. Sleep disturbances in schizophrenia are closely linked to the neurobiological mechanisms and clinical course of the disorder. Objective sleep measures show promise as potential biomarkers of disease progression and treatment response; however further research is needed to establish their reliability, validity and clinical utility.

Keywords: schizophrenia, sleep wake disorders, circadian rhythm, biomarkers.

SANTRAUKA

Įvadas. Miego sutrikimai yra labai dažni sergant šizofrenija ir vis dažniau laikomi ne tik antriniais simptomais, bet ir svarbia ligos dalimi. Vis daugiau tyrimų rodo, kad miego architektūros ir cirkadinių ritmų pokyčiai yra glaudžiai susiję su šizofrenijos neurobiologiniais mechanizmais ir klinikinėmis išraiškomis.

Tikslas. Apžvelgti biologinius mechanizmus, susijusius su miego sutrikimais sergant šizofrenija, ir įvertinti jų potencialą kaip ligos progresavimo biomarkerių.

Metodai. Mokslinių straipsnių paieška atlikta elektroninėje duomenų bazėje „PubMed", įtraukiant publikacijas iki 2026 m. balandžio 5 d. Mokslinėms publikacijoms rasti naudoti šie raktažodžiai ir jų deriniai: šizofrenija, miego-budrumo sutrikimai, cirkadinis ritmas ir biomarkeriai. Iš viso į analizę įtraukti 23 pilno teksto recenzuoti moksliniai straipsniai anglų kalba, publikuoti per pastaruosius 10 metų. Išvadoms apibendrinti buvo atlikta aprašomoji analizė.

Rezultatai. Miego sutrikimai yra dažni sergant šizofrenija ir nustatomi maždaug 30–80 proc. pacientų. Jie siejami su sunkesne ligos eiga, labiau išreikštais simptomais, kognityviniais sutrikimais ir prastesne gyvenimo kokybe. Polisomnografinių tyrimų duomenys rodo trumpesnį bendrą miego laiką, sumažėjusį lėtųjų bangų miegą ir pakitusius REM miego parametrus. Neurofiziologiniai pokyčiai, tokie kaip sumažėjęs miego verpsčių tankis, atspindi talamokortikinių tinklų disfunkciją. Taip pat dažni cirkadinių ritmų sutrikimai, įskaitant nereguliarų miego-budrumo ritmą ir pakitusią melatonino sekreciją. Vis daugiau duomenų rodo, kad su miegu susiję rodikliai, tokie kaip miego verpsčių aktyvumas, cirkadinio ritmo kintamumas ir uždegiminiai žymenys, gali turėti potencialą kaip biomarkeriai ligos progresavime, tačiau jų klinikinis pritaikomumas išlieka ribotas.

Išvados. Miego sutrikimai sergant šizofrenija yra glaudžiai susiję su ligos neurobiologiniais mechanizmais ir klinikine eiga. Objektyvūs miego rodikliai gali būti perspektyvūs biomarkeriai, leidžiantys vertinti ligos progresavimą ir gydymo efektyvumą, tačiau būtini tolesni tyrimai jų patikimumui ir klinikiniam pritaikymui nustatyti.

Raktažodžiai: šizofrenija, miego sutrikimai, cirkadiniai ritmai, biomarkeriai, miego architektūra

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INTRODUCTION

Sleep disturbances are common and clinically significant symptoms in patients with schizophrenia. Poor sleep quality affects a substantial proportion of patients, with a reported prevalence of approximately 63.4% [1]. Historically considered secondary symptoms, sleep disturbances are now recognized as potentially bidirectionally related to psychotic symptoms, contributing to their persistence and forming a vicious cycle [2].

Sleep disturbances are common and clinically significant symptoms in patients with schizophrenia. Poor sleep quality affects a substantial proportion of patients, with a reported prevalence of approximately 63.4% [1]. Historically considered secondary symptoms, sleep disturbances are now recognized as potentially bidirectionally related to psychotic symptoms, contributing to their persistence and forming a vicious cycle [2].

Schizophrenia is associated with disturbances in circadian regulation and alterations in sleep regulatory processes, leading to disrupted sleep architecture, including reduced total sleep time and impaired sleep continuity [3]. Polysomnographic studies have demonstrated abnormalities in REM sleep, such as shortened REM latency, reduced REM duration, and increased REM density [3]. In addition, reduced sleep spindle density, amplitude, and duration have been consistently associated with cognitive impairment, reflecting thalamocortical network dysfunction [4]. These findings suggest that sleep parameters may serve as objective indicators of disease progression and treatment response.

Circadian rhythm disturbances are also a key feature of schizophrenia. Patients frequently exhibit irregular sleep-wake patterns, altered melatonin secretion, and misalignment of biological rhythms, which may reflect underlying pathophysiological processes [2]. At the molecular level, alterations in clock gene expression and neuroendocrine regulation further support the association between sleep disturbances and the biological mechanisms of the disorder [5].

Accumulating evidence regarding the role of sleep in schizophrenia has led to the exploration of novel diagnostic and therapeutic approaches. Modulation of sleep rhythms, including non-invasive brain stimulation, has been suggested as a potential strategy for improving both sleep quality and clinical outcomes [6]. In this context, objective sleep parameters, such as sleep spindle activity, REM sleep characteristics, and circadian rhythm alterations, may serve as potential biomarkers for assessing disease progression and treatment response. These biomarkers could contribute to improved clinical outcomes by enabling earlier detection of deterioration and more targeted treatment strategies; however, their clinical application remains limited. The aim of this literature review is to examine the biological mechanisms underlying sleep disturbances in schizophrenia and evaluate their potential as biomarkers .

METHODS

A literature search was conducted using the electronic PubMed database. The search included studies published up to April 5, 2026. Relevant publications were identified using combinations of the following Medical Subject Headings (MeSH) terms: “schizophrenia,” “sleep-wake disorders,” “circadian rhythm,” and “biomarkers.”Only full-text peer-reviewed scientific articles published in English within the last 10 years were considered eligible for inclusion. The review included various types of scientific publications, such as clinical trials, randomized controlled trials, meta-analyses, systematic reviews and narrative reviews; consequently, duplication of reported findings and referenced primary studies was unavoidable.

In total, 23 publications were included in the analysis, with additional relevant sources identified through the reference lists of selected articles. The included studies examined various aspects of sleep disturbances in schizophrenia, including sleep architecture, circadian rhythm alterations and neurobiological mechanisms. Studies that did not meet the inclusion criteria were excluded. Descriptive analysis was performed for the literature review.

RESULTS

1. Prevalence and Clinical Characteristics of Sleep Disturbances

Sleep disturbances are very common in schizophrenia and are considered a major part of the disease’s clinical course [7]. Systematic reviews show that about 30–80% of patients with schizophrenia deal with sleep issues, which are linked to worse clinical outcomes [8]. Meta-analyses of polysomnography studies comparing patients with healthy individuals reveal that these disturbances often involve a shorter total sleep time, taking longer to fall asleep, and a decrease in slow-wave sleep [9]. Additionally, poor sleep is associated with a lower quality of life, cognitive decline, and more severe symptoms [8,10]. Sleep disturbances are closely associated with psychotic symptoms, such as hallucinations and delusions, and may play a contributory role in their onset and maintenance [7].

Review studies indicate that the most common problems include trouble falling asleep, difficulties staying asleep, poor sleep quality, fragmented sleep, nightmares, hypersomnia, disrupted sleep-wake rhythms, and irregular sleep schedules [2,10,11]. In addition, sleep disturbances may emerge during the prodromal phase of the disease, often before psychosis fully develops, making them one of the most common early symptoms [12] These disturbances may also serve as early warning indicators of emerging or worsening psychosis [13].

Sleep disturbances in schizophrenia are heterogeneous, with their presentation varying according to the stage of the disorder and individual patient characteristics [2]. Nevertheless, accumulating evidence indicates that, despite this variability, sleep disturbances remain closely associated with the clinical course of schizophrenia, with specific patterns linked to greater disease severity and poorer functional outcomes [13]. In addition, antipsychotic medications can also influence sleep in schizophrenia by affecting sleep quality and architecture, and may both improve and disrupt sleep while contributing to sleep disorders; therefore, they should be considered an important confounding factor when evaluating sleep disturbances [2,11].

2. Sleep Architecture and Neurophysiological Disruptions

Schizophrenia is marked by objectively measurable changes in sleep architecture [14]. Polysomnography meta-

Table 1. Studies on prevalence, clinical features, and outcomes of sleep disturbances in schizophrenia

Authors of the study/article	Type	Sample	Conclusions
Ferrarelli (2021) [7]	Narrative review	N/A	Sleep disturbances in schizophrenia are common and include abnormalities in sleep architecture and sleep oscillations. They are closely associated with psychotic symptoms, and may contribute to their onset and persistence.
Azargoonjahromi et Bahrampour (2025) [8]	Umbrella review	N/A	Sleep disturbances are common in schizophrenia and are associated with cognitive impairment, more severe psychotic symptoms, poorer quality of life, circadian rhythm disruption, and genetic variations related to sleep regulation.
Chan et al. (2017) [9]	Systematic review and meta-analysis	31 studies (35 included in review): 574 patients with schizophrenia and 515 HC	Schizophrenia is associated with significant disturbances in both sleep continuity and sleep architecture. Patients show reduced total sleep time, decreased sleep efficiency, prolonged sleep onset latency, and increased wake time.
Kaskie et Ferrarelli (2020) [10]	Narrative review	N/A	Sleep disturbances in schizophrenia are associated with abnormalities in sleep architecture and EEG oscillations, reflecting thalamocortical dysfunction. These alterations are linked to greater symptom severity, cognitive impairment, and poorer quality of life.
Freeman et Waite (2025) [2]	Narrative review	N/A	Sleep and circadian disturbances are common in schizophrenia, associated with worse symptoms, cognition, and functioning, and may represent a causal and modifiable factor in psychosis.
Kaskie et al. (2017) [11]	Systematic review	54 studies on schizophrenia and sleep disorders included after screening	Sleep disturbances are highly prevalent in schizophrenia, often precede illness onset, and are associated with worse symptoms, cognitive impairment, and poorer quality of life.
Ashton et Jagannath (2020) [12]	Narrative review	N/A	Sleep and circadian disruption are core features of schizophrenia, linked to symptom severity and quality of life, and may appear in the prodromal phase before psychosis onset. A bidirectional relationship with dopamine signaling may contribute to the disorder's pathophysiology.
Waite et al. (2020) [13]	Systematic review	58 studies (37 clinical, 21 non-clinical) investigating sleep disturbances and psychotic experiences	Sleep disturbances are highly prevalent in psychosis, associated with the onset and persistence of psychotic experiences, and may act as an early warning sign of developing or worsening psychosis. They represent a causal and modifiable factor, and their treatment can improve both sleep and psychotic symptoms.

N/A – not applicable; HC – healthy controls

analyses show that patients have significantly reduced total sleep time, prolonged sleep latency, increased wake after sleep onset, lower sleep efficiency, and reduced stage 4 and slow-wave sleep [9]. Meta-analytic data also indicate significant alterations in REM sleep in patients with schizophrenia, including a shorter time to the first REM episode and reduced REM duration [3]. There is a growing body of evidence suggesting that sleep disturbances in schizophrenia are linked to specific neurobiological mechanisms and one of the most consistently reported findings is a reduction in sleep spindles, which has been associated with thalamocortical network dysfunction [4,14,15] Sleep spindle abnormalities have been associated with disruptions in gamma-aminobutyric acid (GABA) and glutamatergic neurotransmission, as well as alterations in T-type calcium channel function [14].

Besides structural sleep changes, circadian rhythm disorders are also frequently found in schizophrenia, including irregular sleep-wake patterns, delayed or advanced sleep phases, and, in some cases, non-24-hour rhythms [2]. These disturbances are characterized by irregular sleep timing and a shift toward increased nighttime wakefulness and are influenced by a combination of biological and behavioral factors, including anxiety, psychotic symptoms, reduced daytime activity, and environmental conditions [2]. Fragmented sleep is also a common issue, characterized by waking up frequently and a lack of continuous rest [8]. Long-term objective observations show that circadian disruptions in schizophrenia are linked to getting less daylight exposure and having highly variable sleep-wake rhythms [16].

3. Genetic and Molecular Mechanisms

The biological foundation of these circadian disruptions is backed by evidence showing altered circadian locomotor output cycles kaput (CLOCK) gene expression and impaired melatonin regulation in schizophrenia [17]. Studies have shown that individuals going through their first psychotic episode have reduced CLOCK, period circadian regulator 2 (PER2), and cryptochrome circadian regulator 1 (CRY1)

Table 2. Studies on sleep and circadian disturbances in schizophrenia

Authors of the study/article	Type	Sample	Conclusions
Chan et al. (2017) [9]	Systematic review and meta-analysis	31 studies (35 included in review): 574 patients with schizophrenia and 515 HC	Schizophrenia is associated with disturbances in sleep continuity and architecture, including reduced total sleep time and efficiency, prolonged sleep latency, increased wakefulness, and alterations in slow-wave and REM sleep.
Morra et Barbato (2025) [3]	Systematic review and meta-analysis	1,845 patients with schizophrenia, 1,269 HC	Schizophrenia is associated with significant REM sleep alterations, including reduced REM time and shortened REM latency, while REM density is increased.
Fahey et Lopez (2024) [5]	Genome-wide association study	GWAS datasets: SCZ (n=160,779); UK Biobank: insomnia (n=239,918)	Significant shared genetic variation was identified between schizophrenia and sleep-related phenotypes. Schizophrenia polygenic risk scores were associated with insomnia and evening chronotype.
Ferrarelli (2024) [4]	Narrative review	N/A	Sleep spindle abnormalities are present across schizophrenia stages, including early and high-risk phases, and are linked to thalamocortical dysfunction, cognitive impairment, symptom severity, and GABAergic and glutamatergic dysfunction.
Manoach et al. (2019) [15]	Narrative review	N/A	Sleep spindle deficits are consistently observed in schizophrenia and are associated with impaired memory consolidation, cognitive deficits, and thalamocortical dysfunction.
Castelnovo et al., (2025) [14]	Narrative review	N/A	Sleep spindle abnormalities in schizophrenia are linked to thalamocortical dysfunction and cognitive impairment, and are associated with GABAergic and glutamatergic imbalance and altered T-type calcium channel function.
Ferrarelli (2024) [4]	Narrative review	N/A	Reduced sleep spindle density, amplitude, and duration are consistently observed in schizophrenia and are associated with cognitive impairment, symptom severity, and thalamocortical dysfunction; alterations are linked to GABAergic and glutamatergic neurotransmission and calcium channel abnormalities.
Freeman et Waite (2025) [2]	Narrative review	N/A	Sleep and circadian disturbances include insomnia, fragmented sleep, hypersomnia, nightmares, and circadian disruption; they are associated with worse symptoms, cognition, and functioning, and may represent a causal, modifiable factor in psychosis.
Azargoonjahromi et Bahrampour (2025) [8]	Umbrella review	N/A	Sleep disturbances are common in schizophrenia and are associated with cognitive impairment, more severe psychotic symptoms, poorer quality of life, circadian rhythm disruption, and genetic variations related to sleep regulation.
Skeldon et al. (2022)	Case-control study	20 patients with schizophrenia and 21 HC	Patients with schizophrenia exhibited prolonged and irregular sleep patterns, reduced bright light exposure, and circadian rhythm disruption, suggesting that personalized light interventions may improve sleep-wake regulation.

N/A – not applicable; HC – healthy controls; REM – Rapid Eye Movement; GABA – gamma-aminobutyric acid.

messenger ribonucleic acid (mRNA) expression in their blood mononuclear cells, while patients with chronic schizophrenia show disrupted CRY1 and PER2 rhythms in their fibroblasts [17]. Furthermore, irregular melatonin release is observed in schizophrenia, and melatonin receptor 1 (MTRN1) polymorphisms are linked to more frequent insomnia, which adds support to the idea that circadian imbalance plays a role in the disorder's pathogenesis [18]. A large-scale genome-wide association study analysis discovered a significant positive genetic correlation between longer sleep duration and the risk of developing schizophrenia [19]. It was also found that a higher genetic risk for schizophrenia is associated with an evening chronotype and insomnia, suggesting that these conditions partially share a genetic foundation [5]. This data shows that circadian system issues in schizophrenia are not merely accompanying symptoms; they actually may reflect underlying molecular mechanisms of the disease.

A metabolomics study showed that 16 metabolites, mostly lipids, are linked to sleep disturbances in schizophrenia, pointing to major changes in metabolic pathways involving glycerophospholipids, butanoate, and sphingolipids [20]. In addition, melatonin has been implicated in these disturbances, with evidence suggesting altered secretion and regulation linked to sleep and circadian abnormalities

Table 3. Studies on molecular and neurobiological mechanisms of sleep disturbances in schizophrenia

Authors of the study/article	Type	Sample	Conclusions
Johansson et al. (2016) [17]	Case-control study	11 patients with chronic schizophrenia and 11 HC; additional sample: 14 first-episode psychosis patients and 14 HC	Altered expression of clock genes (CRY1, PER2, CLOCK) and loss of normal circadian rhythmicity were observed in schizophrenia; patients also showed poor sleep quality and impaired circadian regulation
Boiko et al. (2024) [18]	Narrative review	N/A	Circadian and sleep–wake disturbances are linked to symptom severity and quality of life, involve genetic, neurobiological, and metabolic mechanisms, and altered melatonin signaling supports their role in pathogenesis.
Lane et al. (2017) [19]	Genome-wide association study	112,586 UK Biobank participants	Sleep disturbances have a genetic basis and share pathways with neuropsychiatric and metabolic disorders; longer sleep duration is genetically linked to schizophrenia risk.
Fahey et al. (2024) [5]	Genome-wide and pathway-based polygenic score analysis	239,918 UK Biobank participants	The study shows shared genetic variation between sleep traits and schizophrenia; polygenic risk is linked to chronotype and insomnia, with specific pathways potentially mediating circadian disruption.
Yan et al. (2024) [20]	case-control study	59 drug-naïve patients with schizophrenia, 36 HC	The study identified 16 characteristic metabolites (mainly lipid-related) significantly associated with sleep disturbances in drug-naïve schizophrenia.
Moon et al. (2022) [21]	Narrative review	N/A	Melatonin dysregulation is common in psychiatric disorders, including schizophrenia, where reduced melatonin levels and pineal gland abnormalities are linked to sleep and circadian disturbances.
Künzel et al. (2018) [22]	Case-control study	9 drug-free patients with schizophrenia and 9 HC	Patients with schizophrenia show altered sleep architecture and altered endocrine regulation, with significantly increased GH response to GHRH.
Lee et al. (2019) [23]	Case-control study	144 outpatients with schizophrenia and 134 HC	Sleep disturbances in schizophrenia are associated with increased inflammatory markers (hs-CRP, IL-6); poorer sleep correlates with higher inflammation and worse cognition, especially in women.

N/A – not applicable; HC – healthy controls; GH – growth hormone; GHRH – growth hormone-releasing hormone; hs-CRP – high-sensitivity C-reactive protein; IL-6 – interleukin-6; CRY1 – cryptochrome circadian regulator 1; PER2 – period circadian regulator 2; CLOCK – circadian locomotor output cycles kaput.

[21]. Neuroendocrine mechanisms may contribute to sleep disturbances in schizophrenia, as growth hormone-releasing hormone (GHRH) plays a key role in sleep regulation by promoting slow-wave sleep and growth hormone release, and its dysregulation has been reported in the disorder [22]. Furthermore, sleep disturbances in schizophrenia are associated with systemic inflammation, with elevated levels of pro-inflammatory cytokines correlating with poorer sleep quality [23]. Overall, these findings suggest that metabolic, neuroendocrine, and inflammatory alterations may represent potential biomarkers linked to sleep disturbances and the underlying pathophysiology of schizophrenia; however, their clinical applicability remains limited and requires further validation.

DISCUSSION

This review summarizes current evidence on sleep disturbances in schizophrenia and their underlying biological mechanisms. The findings are consistent with previous literature showing that sleep disturbances are highly prevalent and closely associated with symptom severity, cognitive impairment, and poorer functional outcomes.

However, the interpretation of these findings is limited by substantial methodological heterogeneity across studies. Differences in sample characteristics (e.g., first-episode vs. chronic patients), assessment methods and study design make direct comparisons challenging. Importantly, relatively few studies differentiate sleep disturbances across illness stages. Available evidence suggests that sleep abnormalities may already be present in prodromal and early stages of schizophrenia and may fluctuate with disease course, potentially worsening during acute exacerbations and partially improving during remission, although longitudinal data remain limited.

Medication effects represent another important confounding factor. Antipsychotic treatment can significantly influence sleep architecture, for example by increasing total sleep time or altering REM sleep, which complicates the distinction between illness-related and treatment-related changes.

From a biological perspective, the strongest evidence for sleep disturbances as potential biomarkers currently comes from neurophysiological and genetic domains. Sleep spindle deficits, reflecting thalamocortical dysfunction, are among the most replicated findings and show associations with cognitive impairment and illness severity. In parallel, genetic studies indicate shared pathways between sleep traits and schizophrenia, while circadian rhythm disruptions and melatonin signaling abnormalities further support a biological basis. Inflammatory markers have also been implicated in sleep disturbances in schizophrenia, however, the current body of evidence remains less extensive compared to neurophysiological and genetic biomarkers. In addition, circadian rhythm disturbances can be objectively assessed using methods such as actigraphy, polysomnography, and light exposure monitoring. Parameters including sleep timing variability and sleep–wake rhythm regularity may represent promising markers of circadian dysfunction in schizophrenia; however, most current evidence remains associative, and their clinical applicability has not yet been fully established.

Although sleep disturbances show promise as potential biomarkers, their clinical applicability should be interpreted with caution. Despite consistent associations, current evidence is insufficient to support their routine use in clinical practice due to a lack of standardized measurement protocols, limited longitudinal validation, and uncertainty regarding specific symptoms, disease stage and effects of medications. At present, sleep disturbances are better considered as clinically relevant features and potential targets for intervention rather than biomarkers for early intervention.

Overall, sleep disturbances appear to be an important component of schizophrenia, with potential implications for both research and clinical practice. Further studies are needed to better establish their role as reliable biomarkers and to evaluate targeted interventions aimed at improving sleep and clinical outcomes.

CONCLUSIONS

Sleep disturbances are highly prevalent in schizophrenia and involve changes in both sleep architecture and circadian rhythms. These issues are tightly connected to the disease's clinical course, symptom severity, and how well the patient functions daily.

Data from polysomnography and actigraphy show that schizophrenia features specific changes in sleep structure, including less slow-wave sleep, altered REM parameters, and fewer sleep spindles. All of these reflect the core neurobiological mechanisms of the disorder. Circadian problems, such as sleep-wake desynchronization, high sleep variability, and low daytime light exposure, can be objectively measured and may serve as potential biomarkers.

Genetic, molecular, and neuroendocrine findings suggest that sleep disturbances in schizophrenia are linked to altered CLOCK gene expression, abnormal melatonin regulation, metabolic shifts, and inflammation, confirming their deep connection to the disease's pathophysiology.

Even though various sleep parameters and biological markers show real promise as biomarkers, their clinical application is still quite limited right now. Since most of the current data relies on associations, we need further prospective and interventional studies to properly validate them.

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Associations of obsessive-compulsive personality disorder with suicidal behavior, anxiety and mood disorders: a narrative literature review

Obsesinio-kompulsinio asmenybės sutrikimo sąsajos su suicidiniu elgesiu, nerimo ir nuotaikos sutrikimais: naratyvinė literatūros apžvalga

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SUMMARY

Obsessive-Compulsive Personality Disorder (OCPD) may represent a clinically significant yet understudied risk factor for suicidal behavior, particularly when comorbid with anxiety and mood disorders. OCPD is characterized by perfectionism, rigidity, and a need for control. Although frequently comorbid with anxiety and mood disorders, its relationship with suicide-related outcomes remains insufficiently examined.

The objective of this review is to examine the associations of OCPD with suicidal behavior, anxiety and mood disorders, with particular attention to the risk of suicidal ideation and suicide attempts.

Materials and methods. A literature search was conducted in PubMed, PsycINFO, Web of Science, Scopus, and Google Scholar from April and to May 2026. The main search terms included "obsessive-compulsive personality disorder", "OCPD", "anankastic personality", "suicide", "suicidal ideation", "suicide attempt", "anxiety disorders", "depression", and "bipolar disorder." After removing duplicate records, 88 articles were identified. Based on the eligibility criteria, 11 articles were included in the final analysis.

Results. OCPD appears to be clinically relevant in mood and anxiety disorder populations. Studies suggest associations between OCPD or OCPD-related traits and depressive symptoms, anxiety, stress-related impairment, perfectionism, hopelessness, reduced reasons for living, suicidal ideation, non-suicidal self-injury, and suicide attempts. Direct evidence remains limited, but available findings indicate that OCPD may increase suicide-related vulnerability, particularly in the context of depression. However, many studies used cross-sectional designs, assessed traits rather than formal OCPD diagnosis.

Conclusion. OCPD and related traits may be important factors in suicide risk assessment, especially among individuals with mood disorders, anxiety disorders, hopelessness, and marked perfectionism. Further longitudinal research is needed to clarify causal pathways and identify which OCPD features most strongly predict suicidal behavior.

Keywords. Obsessive-compulsive personality disorder; suicidal behavior; mood disorders; anxiety disorders; perfectionism

SANTRAUKA

Obsesinis-kompulsinis asmenybės sutrikimas (OKAS) gali būti kliniškai reikšmingas, tačiau nepakankamai ištirtas savižudiško elgesio rizikos veiksnys, ypač kai jis pasireiškia kartu su nerimo ir nuotaikos sutrikimais. OKAS būdingas perfekcionizmas, rigidiškumas ir kontrolės poreikis.

Tikslas. Šios naratyvinės apžvalgos tikslas – išnagrinėti OKAS sąsajas su suicidiniu elgesiu, nerimo ir nuotaikos sutrikimais, ypatingą dėmesį skiriant suicidinėms mintims ir savižudybės bandymams.

Metodai. Literatūros šaltinių paieška atlikta PubMed, PsycINFO, Web of Science, Scopus ir Google Scholar duomenų bazėse 2026 m. balandžio - gegužės mėn. Pagrindiniai paieškos terminai apėmė: „obsessive-compulsive personality disorder", „OKAS", „anankastic personality", „suicide", „suicidal ideation", „suicide attempt", „anxiety disorders", „depression" ir „bipolar disorder". Pašalinus šaltinių dublikatus, identifikuoti 88 straipsniai. Į galutinę analizę, remiantis atrankos kriterijais, įtraukta 11 straipsnių.

Rezultatai. OKAS yra kliniškai reikšmingas nuotaikos ir nerimo sutrikimų turinčiose populiacijose. Tyrimai rodo sąsajas tarp obsesinio-kompulsinio asmenybės sutrikimo arba su obsesiniu-kompulsiniu asmenybės sutrikimu susijusių bruožų ir depresijos simptomų, nerimo, su stresu susijusio funkcionavimo sutrikimo, perfekcionizmo, beviltiškumo, sumažėjusių gyvenimo motyvų, suicidinių minčių, nesuicidinio savęs žalojimo ir suicidinių bandymų. Tiesioginių įrodymų vis dar nedaug, tačiau turimi duomenys rodo, kad OKAS gali didinti riziką suicidiniam elgesiui, ypač depresijos kontekste. Vis dėlto daugelyje tyrimų taikytas skerspjūvio tyrimo planas, vertinti bruožai, bet ne formali OKAS diagnozė.

Išvados. OKAS ir su juo susiję bruožai gali būti svarbūs veiksniai vertinant savižudybės riziką, ypač asmenims, sergantiems nuotaikos ar nerimo sutrikimais, patiriantiems beviltiškumą ir pasižymintiems ryškiu perfekcionizmu. Reikalingi tolesni ilgalaikiai tyrimai, siekiant išaiškinti priežastinius mechanizmus ir nustatyti, kurie OKAS bruožai geriausiai prognozuoja suicidinio elgeso riziką.

Raktažodžiai: obsesinis-kompulsinis asmenybės sutrikimas; suicidinis elgesys; nuotaikos sutrikimai; nerimo sutrikimai; perfekcionizmas.

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INTRODUCTION

Suicide is a major global public health concern, accounting for an estimated 700,000 deaths annually worldwide [1]. Although mental disorders are strongly associated with an increased risk of suicides, the strength and nature of this relationship remain subject to methodological and diagnostic variability. In contrast, the contribution of personality disorders-particularly Obsessive-Compulsive Personality Disorder (OCPD) has received comparatively limited attention [2] compared with extensively studied conditions such as Borderline Personality Disorder, which has been strongly associated with elevated suicide risk [3]. OCPD is defined by enduring patterns of perfectionism, rigidity, and a need for control, and is commonly observed among individuals with anxiety and mood disorders. Despite this high comorbidity, its potential role in shaping suicide risk remains insufficiently characterized, with existing findings limited and sometimes inconsistent [2]. The co-occurrence of OCPD traits with mood disorders has been associated with greater clinical complexity and may adversely affect treatment response, with some evidence suggesting a potential increase in suicide risk [4]. A clearer understanding of these relationships may inform more precise risk assessment and the development of targeted clinical interventions.

In the DSM-5, OCPD is classified as a Cluster C personality disorder marked by perfectionism, rigidity, and a need for control. Diagnosis requires the presence of multiple maladaptive traits, particularly those reflecting excessive control, perfectionism, and rigidity. As with other personality disorders, these traits are enduring, inflexible, and pervasive across contexts, typically emerging by early adulthood [5]. These features-particularly maladaptive perfectionism and cognitive rigidity – have been hypothesized to contribute to vulnerability under conditions of stress, failure, or loss, which are key contexts in the emergence of suicidal ideation.

OCPD should be distinguished from Obsessive-Compulsive Disorder (OCD), as the two conditions are frequently conflated despite important diagnostic differences. OCD is characterized by intrusive, unwanted obsessions and repetitive compulsions that are usually experienced as distressing and ego-dystonic [5]. In contrast, OCPD reflects enduring personality traits that are often ego-syntonic and consistent with the individual's self-concept [2,5]. This distinction has clinical relevance because individuals with OCPD may be less likely to recognize their traits as problematic, which may delay help-seeking and complicate treatment engagement. Although OCPD traits may occur in individuals with OCD, available evidence does not support a simple continuum between OCD and OCPD [6].

Several theoretical frameworks have been proposed to explain how OCPD personality traits may be linked to suicidal behavior. The interpersonal-psychological theory of suicide proposes that suicidal desire arises from the combination of thwarted belongingness and perceived burdensomeness, accompanied by hopelessness, while the capability for suicide is acquired through repeated exposure to painful or fear-inducing experiences [7]. OCPD traits – particularly interpersonal rigidity, social withdrawal, and excessive self-criticism – may contribute to thwarted belongingness and perceived burdensomeness and may also reinforce hopelessness in the context of perceived interpersonal failure [8]. The diathesis – stress model conceptualizes personality traits as enduring vulnerabilities that interact with environmental stressors to precipitate suicidal crises. Core features of OCPD, such as cognitive rigidity and maladaptive perfectionism, may impair adaptive coping in the face of failure, loss, or uncontrollable stressors, thereby increasing vulnerability to hopelessness and suicidal ideation [9]. The perfectionism social disconnection model further suggests that perfectionistic concerns and socially prescribed perfectionism – both central to OCPD – promote social disconnection, which may in turn increase suicide risk [10]. Although these models differ in emphasis, they converge on the role of interpersonal dysfunction, maladaptive coping, and cognitive rigidity as central mechanisms linking personality pathology to suicidal behavior, although empirical support for these pathways in OCPD specifically remains limited.

This narrative review examines the association between OCPD and suicidal behavior, with a particular focus on individuals with comorbid anxiety and mood disorders. Specifically, it seeks to explore the extent to which OCPD traits are associated with suicidal ideation and behavior, to examine potential underlying mechanisms, and to identify inconsistencies and gaps in the existing evidence. In this context, suicidal behavior is considered to encompass suicidal ideation, suicide attempts, and death by suicide. By clarifying these relationships, the review aims to inform risk assessment strategies and highlight priorities for future research.

METHODS

Review design

This study was conducted as a narrative review examining the association between Obsessive-Compulsive Personality Disorder (OCPD) and suicide-related outcomes in the context of anxiety and mood disorders. A narrative design review was selected because the available literature was limited and heterogenous in terms of study designs, populations, diagnostic approaches, and outcome measures.

Data sources and search strategy

A literature search was conducted across five major databases: PubMed, PsycINFO, Web of Science, Scopus, and Google Scholar. Primary search terms included: "obsessive-compulsive personality disorder", "OCPD", "anankastic personality", "suicide", "suicidal behavior", "suicidal ideation". "suicide attempt", "anxiety disorders", "panic disorder", "generalized anxiety disorder", "social anxiety", "mood disorders", "depression", and "bipolar disorder". The search was conducted from April to May 2026.

Eligibility criteria

Studies were included if they: (1) reported original empirical data on OCPD or OCPD-related traits; (2) examined suicidal behavior, suicide-related outcomes, or clinically relevant risk factors such as suicidal ideation, suicide attempts, non-suicidal self-injury, hopelessness, depression, anxiety, perfectionism, reasons for living, or functional impairment; (3) investigated OCPD or OCPD-related traits in relation to anxiety or mood disorders; (4) were published in peer-reviewed journals in English.

Studies were excluded if they: (1) focused exclusively

on OCD without assessing OCPD or OCPD-related traits, defined in this review as maladaptive perfectionism, rigidity/inflexibility, excessive conscientiousness, overcontrol, need for order, restricted emotional expression, or excessive self-criticism; (2) consisted only of single case reports or very small case descriptions without broader clinical relevance; (3) examined perfectionism or obsessive-compulsive traits without any clear connection to OCPD; (4) were review articles, editorials, or commentaries without original data; or (5) did not provide clinically relevant information on mood disorders, anxiety disorders, suicidal behavior, or suicide-related risk factors.

Selection of studies

Following title, abstract, and full-text screening 79 records were excluded for not meeting OCPD-specific criteria. 9 studies met the eligibility criteria. Two additional studies were identified through hand-searching, resulting in 11 studies included in the review (see Table 1).

Data extraction and narrative synthesis

For each included study, the following information was extracted: study design, sample characteristics (size, demographics, clinical population), assessment methods for OCPD and suicidal behavior, prevalence estimates, comorbidity patterns, statistical associations, and key findings. Given the heterogeneity of study designs, populations, and outcome measures, findings were synthesized narratively and organized according to the review's primary themes: prevalence and clinical significance of OCPD, comorbidity with mood disorders, comorbidity with anxiety disorders, direct evidence linking OCPD to suicidal ideation or suicidal behavior, indirect mechanisms linking OCPD traits to suicide-related vulnerability, and treatment implications.

RESULTS

Characteristics of Included Studies

A total of 11 studies were included in the review: 9 studies identified through database searching and 2 additional studies identified through hand-searching. The included studies varied in design, population, assessment methods, and outcome measures. Most studies were observational, cross-sectional, or clinical cohort studies, while one study was a small case series. Study populations included community samples, psychiatric outpatient and inpatient samples, patients with anxiety and mood disorders, medical students, and individuals with elevated perfectionism or stress-related exhaustion. Detailed characteristics and findings of the included studies are presented in Table 1.

Table 1. Characteristics of the studies

Author(s) & Year	Study Design	Sample (N)	Population / Setting	Key OCPD Findings	Suicide / Behavioral Outcomes	Notes / Limitations
Ansell et al., 2010	Cross-sectional outpatient study; factor analysis	130	Monolingual Spanish-speaking Hispanic psychiatric outpatients	OCPD prevalence 26%. A two-factor structure was identified: perfectionism and interpersonal rigidity.	Perfectionism was associated with depression and suicidal ideation; interpersonal rigidity was associated with anger and aggression.	Clinical and ethnically specific sample; cross-sectional design limits causal interpretation.
Aydın et al., 2025	Cross-sectional survey; mediation analysis	164	Medical students in Turkey	Assessed OCPD-related beliefs rather than formal OCPD diagnosis. OCPD belief scores were higher in the hopelessness group.	No direct suicidal behavior outcome was assessed. Depression mediated the association between OCPD-related beliefs and hopelessness, a clinically relevant suicide-risk factor.	Self-report measures; cross-sectional design; findings reflect OCPD-related beliefs rather than diagnosed OCPD.
Bowen et al., 2019	Population-based secondary analysis; regression/ network analysis	7,839	British Adult Psychiatric Morbidity Survey	Assessed OCPD traits rather than formal OCPD diagnosis. Traits were associated with suicidality-related outcomes independent of depression and mood instability.	OCPD traits were associated with suicidal ideation, non-suicidal self-injury, and suicide attempts.	Cross-sectional; trait measure rather than clinical OCPD diagnosis.
Diaconu et al., 2009	Three-group clinical comparison; structured interviews	311	Psychiatric consultation sample; depressed + OCPD vs depressed without Axis II vs controls	OCPD examined as independent risk factor for suicidal behavior among depressed patients.	OCPD associated with nonfatal suicidal behavior beyond depression risk.	Cross-sectional/ retrospective;
Dyck et al., 2001	Cross-sectional anxiety-disorder comorbidity study	622	Anxiety-disorder outpatients: GAD, panic disorder ± agoraphobia, social phobia	Examined personality pathology patterns; OCPD/Cluster C relevance in anxiety disorders.	No suicide-specific outcome. Major depression associated with OCPD	Limited by diagnostic era (DSM-III-R), specialized sample, correlational design
Grant et al., 2012	National epidemiological survey	43,093	U.S. adults; NESARC	Lifetime DSM-IV OCPD prevalence 7.8%, similar in men and women.	No suicide-specific outcome in abstract. OCPD was associated with mood, anxiety, and other PD comorbidity.	Strong population sample, but not suicide-focused.

Continuation of the table 1. Characteristics of the studies

Author(s) & Year	Study Design	Sample (N)	Population / Setting	Key OCPD Findings	Suicide / Behavioral Outcomes	Notes / Limitations
Ozkan & Altindag, 2005	Clinical panic-disorder study	122 adults (71 women, 41 men)	Subjects with panic disorder	Personality disorders increased panic-disorder clinical severity; Mood disorders had a high comorbidity with personality disorders (11.6%).	Comorbid OCPD-panic disorder more prevalent in patients with suicidal ideation.	Cross-sectional; OCPD was associated with suicidal ideation but not suicide attempts. Findings may reflect broader personality-disorder comorbidity rather than OCPD-specific risk.
Raja & Azzoni, 2007	Clinical case series	7	Patients with mood disorder + OCPD admitted after near-lethal suicide attempts	OCPD was described as complicating mood disorder presentation and increasing stress response.	All cases involved near-lethal suicide attempts; authors suggest OCPD may increase severity of suicidal behavior in mood disorders.	Very small N; no control group; cannot infer prevalence or causality.
Redden, Mueller, & Cougle, 2022/2023	Clinical study of elevated perfectionism; treatment-response component	74	Students with elevated perfectionism	OCPD was associated with higher perfectionism, personal standards, social anxiety, and current anxiety disorder.	No suicide-specific outcome Behavioral outcome: response to exposure-based perfectionism treatment.	Small university-student sample selected for elevated perfectionism; findings may not generalize to broader clinical OCPD populations
Reinhardt et al., 2024	Clinical longitudinal follow-up	84	Female outpatients with stress-related exhaustion; 7-year follow-up	OCPD was the most common PD; reported as ~40% among those assessed for OCPD.	No direct suicide outcome. OCPD was linked to higher burnout/depression and longer symptom duration/recovery concerns.	Small sample, only females included, self-reported status of mental health
Smith et al., 2017	Open comparison inpatient treatment study	161 total; OCPD n=52	Psychiatric inpatients; OCPD vs other PD vs no PD	OCPD group entered treatment with more depression, anxiety, emotion dysregulation, and emotional non-acceptance.	No suicide-specific outcome. Treatment outcomes: OCPD patients improved but had lower anxiety remission at discharge.	Predominantly Caucasian inpatient sample; self-report; non-manualized treatment.

Prevalence of OCPD

OCPD is among the more common personality disorders in both community and clinical samples, although estimates vary according to diagnostic criteria, assessment method, and population studies. Epidemiological studies estimate the prevalence of OCPD in the general population at approximately 2–8% [2,11,12]. Research [11], using data from the National Epidemiologic Survey on Alcohol and Related Conditions, reported a lifetime DSM-IV OCPD prevalence of 7.8%, with OCPD associated with elevated rates of psychiatric comorbidity, including mood and anxiety disorders.

Rates of OCPD appear higher in psychiatric and stress-related clinical samples. In a Hispanic outpatient psychiatric sample, OCPD prevalence was reported at 26%, substantially higher than general population estimates [13]. In a longitudinal clinical sample of female patients with stress-related exhaustion, OCPD was the most common personality disorder and was associated with greater depressive and burnout symptoms over follow-up [14]. These findings show that OCPD is relatively common in some clinical and stress-related samples. However, these comorbidity findings should be interpreted as indirect evidence with respect to suicide risk, as they do not necessarily establish a direct association with suicidal behavior.

OCPD and Mood Disorders

OCPD appears to be clinically important in the context of mood disorders, particularly depression. Several studies suggest that OCPD or OCPD-related traits are associated with greater depressive symptom burden, chronicity, and functional impairment [14,15]. In psychiatric inpatients, individuals with OCPD showed higher baseline levels of depressive symptoms, generalized anxiety symptoms, emotion regulation difficulties, and emotional non-acceptance compared with controls, although they improved during intensive multimodal psychiatric treatment [15].

Evidence from non-clinical or mixed samples also supports an association between OCPD-related features and depressive vulnerability. In a cross-sectional study of medical students, OCPD-related beliefs were associated with hopelessness, and depression statistically mediated the relationship between OCPD beliefs and hopelessness [16]. This finding is relevant because hopelessness is a well-established suicide-related risk factor. However, the study measured OCPD-related beliefs rather than formal OCPD diagnosis and did not directly assess suicidal behavior. Therefore, it should be interpreted as indirect evidence regarding suicide-related vulnerability rather than direct evidence of suicidal behavior.

OCPD has also been reported in association with bipolar disorder and other mood disorder presentations [11]. A case report [4] described seven patients with mood disorders and comorbid OCPD who had made near-lethal suicide attempts. The authors suggested that OCPD traits such as rigidity,

overcontrol, and heightened sensitivity to stressful life events may complicate mood disorder presentation and may be relevant to the severity of suicidal behavior. However, because this evidence comes from a small uncontrolled case series, it cannot establish that OCPD independently predicts suicide attempts.

OCPD and Anxiety Disorders

OCPD also shows clinically relevant associations with anxiety disorders. In the Harvard/Brown Anxiety Research Project, personality pathology was common among patients with generalized anxiety disorder, panic disorder with or without agoraphobia, and social phobia, with Cluster C personality pathology being particularly relevant in these populations [17]. Similarly, in individuals with panic disorder, comorbid personality disorders were associated with greater clinical severity, although OCPD-specific findings require careful interpretation because not all outcomes were specific to OCPD [18].

Research on perfectionism further supports the relationship between OCPD traits and anxiety symptoms. In a study of individuals with elevated perfectionism, those meeting criteria for OCPD demonstrated higher general and domain-specific perfectionism, greater personal standards, higher social anxiety, and increased rates of current anxiety disorders compared with those without OCPD [19]. These findings indicate an association between OCPD, perfectionism, and anxiety symptoms. However, because this study did not primarily examine suicidal behavior, its relevance to suicide risk is indirect.

OCPD and Suicidal Behavior

Direct evidence linking OCPD to suicidal ideation and suicidal behavior remains limited but clinically important. A study [20] examined depressed patients with and without comorbid OCPD and found that comorbid OCPD was associated with increased nonfatal suicidal behavior. The study also reported that depressed patients with OCPD had fewer reasons for living and lower scores on protective dimensions such as moral objections to suicide compared with depressed patients without OCPD [20]. These findings suggest that OCPD may be associated not only with increased suicide-related risk but also with diminished protective factors in the context of depression.

Trait-based evidence also suggests a relationship between obsessive-compulsive personality features and suicide-related outcomes. In a large community sample from the British Adult Psychiatric Morbidity Survey, obsessive-compulsive personality traits were independently associated with suicidal ideation, non-suicidal self-injury, and suicide attempts after controlling for depression and mood instability [8]. The study demonstrated that the association between obsessive-compulsive personality traits and suicide-related outcomes remained significant after controlling for depression and mood instability. However, the study assessed personality traits rather than formal OCPD diagnosis, and its cross-sectional design limits conclusions about causality.

Evidence regarding death by suicide in individuals with OCPD is extremely limited. No robust prospective studies identified in this review specifically examined suicide deaths among individuals diagnosed with OCPD. Therefore, while available findings support an association between OCPD-related pathology and suicidal ideation or nonfatal suicidal behavior, conclusions regarding fatal suicidal behavior remain premature.

OCPD-Related Traits and Suicide-Related Vulnerability

Several studies examined OCPD-related traits that may be clinically relevant to suicide-related vulnerability. Maladaptive perfectionism may increase vulnerability to self-criticism, shame, perceived failure, and hopelessness when personal standards cannot be met. In a Hispanic outpatient sample study [13], the perfectionism of OCPD was associated with depression and suicidal ideation, whereas interpersonal rigidity was more closely associated with anger and aggression. These findings indicate that different OCPD dimensions were associated with different clinical correlates in this sample.

Other studies reported associations between OCPD-related beliefs, hopelessness, depression, anxiety symptoms, emotional non-acceptance, and functional impairment [14-16, 19]. These variables may be clinically relevant to suicide related vulnerability; however, most studies did not directly examine whether they mediated the relationship between OCPD and suicidal behavior.

DISCUSSION

This review suggests that obsessive-compulsive personality disorder (OCPD) and OCPD-related traits may be clinically relevant to suicide-related vulnerability, especially in individuals with mood and anxiety disorders. According to the studies findings, OCPD was associated with depressive and anxiety symptoms, stress-related impairment, perfectionism, hopelessness, and functional difficulties [11,13–16]. Direct evidence linking OCPD to suicidal behavior remains very limited; however, available studies suggest associations with suicidal ideation, non-suicidal self-injury, suicide attempts, fewer reasons for living, and reduced protective factors, particularly in depressed or community samples [8,20]. These findings indicate that OCPD may be relevant to suicide risk assessment, especially when it occurs alongside depression, anxiety, hopelessness, or marked perfectionism. Perfectionism appears to be one of the most clinically relevant traits, linking OCPD with suicide-related vulnerability. A study [13] found that the perfectionism dimension of OCPD was associated with depression and suicidal ideation, suggesting that perfectionistic traits may be especially relevant when internalizing distress.

The relationship between OCPD and mood disorders appears important. Depression is one of the strongest clinical correlates of suicidal ideation and behavior, and several studies suggest that OCPD is associated with greater depressive burden or more persistent symptoms [14,15]. Another study found that depression mediated the association between OCPD-related beliefs and hopelessness in medical students, supporting the possibility that depressive symptoms could correlate with OCPD traits and suicide-related vulnerability [16]. However, given than much of the available evidence is cross-sectional, it remains unclear whether OCPD traits increase vulnerability to depression, and whether depression, in turn, reinforces OCPD-related beliefs. Clinically, these findings suggest that assessment of OCPD traits may be useful in patients with mood or anxiety disorders, particularly when suicidal ideation,

chronic depression, severe perfectionism, hopelessness is present. Clinicians should consider evaluating perfectionistic standards, fear of mistakes, rigidity, intolerance of uncertainty, emotional restriction, and reactions to perceived failure. These traits may not be reported because OCPD features are often perceived as appropriate or desirable [1,2].

The relationship between OCPD and anxiety disorders is also clinically relevant, although its connection to suicidal behavior is less direct. OCPD traits such as perfectionism, intolerance of uncertainty, rigidity, and excessive control may intensify anxiety symptoms, particularly in panic disorder, generalized anxiety disorder, and social anxiety disorder [17–19]. However, most studies examining OCPD and anxiety disorders did not directly assess suicidal behavior, so conclusions regarding suicide risk in this subgroup remain preliminary.

Several theoretical frameworks may help explain the possible relationship between OCPD-related traits and suicide-related vulnerability. Cognitive rigidity and overcontrol may be relevant because individuals with OCPD may struggle to adapt flexibly to failure, loss, interpersonal conflict, or uncertainty. Within a diathesis–stress framework, these enduring traits may interact with environmental stressors to intensify distress and reduce adaptive coping [9]. Similarly, the interpersonal theory of suicide suggests that perceived burdensomeness, thwarted belongingness, and hopelessness are central to suicidal desire [7]. OCPD-related interpersonal rigidity, emotional restriction, and excessive self-criticism could plausibly contribute to these mental states, although direct empirical testing of these pathways in OCPD populations remains limited.

The perfectionism social disconnection model provides another relevant framework. This model proposes that perfectionistic concerns and socially prescribed perfectionism may promote social disconnection, which may in turn increase suicide-related vulnerability [10]. Because perfectionism is a core feature of OCPD. This model may help explain the contribution of OCPD traits to interpersonal isolation, shame, and hopelessness. However, perfectionism should not be treated as equivalent to OCPD, since OCPD also includes rigidity, overcontrol, excessive conscientiousness, and inflexibility.

This review has some noteworthy limitations. Very few studies directly examine OCPD and suicidal behavior as primary outcomes. Several assess OCPD traits, beliefs, or perfectionism rather than formal OCPD diagnosis. Many are cross-sectional or retrospective, limiting causal relationships. The studies also vary substantially in sample type, diagnostic method, and outcome measurement. Evidence regarding death by suicide in individuals with OCPD is especially limited. Future research should use longitudinal designs, distinguish diagnosed OCPD from dimensional traits, and examine whether specific features such as perfectionism, rigidity, overcontrol, or interpersonal inflexibility independently predict suicidal ideation, suicide attempts, or death by suicide.

CONCLUSIONS

This narrative review suggests that OCPD and OCPD-related traits may be clinically relevant to suicidal behavior, particularly among individuals with mood and anxiety disorders. Available studies link OCPD features with depression, anxiety, hopelessness, perfectionism and suicidal ideation.

However, the evidence remains limited. Few studies directly examine OCPD and suicidal behavior and many rely on cross-sectional designs, small samples or trait-based measurement rather than OCPD diagnoses. Evidence regarding suicide death is especially lacking. Therefore, current findings should be interpreted with caution.

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Diagnosis of Capgras syndrome and illustrative clinical cases: a literature review

Capgraso sindromo diagnostika ir klinikiniai atvejai: literatūros apžvalga

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SUMMARY

Introduction. Capgras syndrome is a psychopathological phenomenon. It belongings to the group of delusional misidentification syndromes and is characterized by a delusional belief that a close person or object has been replaced by an identical double. Although the syndrome is rare, it has significant clinical importance due to diagnostic challenges and its associations with various psychiatric and neurological disorders.

Methods. A literature search was conducted in international scientific databases and platforms, including PubMed, ScienceDirect, and ResearchGate. The following keywords were used: "Capgras syndrome", "delusional misidentification syndrome", "delusion of doubles", and "misidentification delusion".
The search covered publications published between 1923 and 2025. Articles published in English describing cases of Capgras syndrome, its etiopathogenesis, clinical features, diagnostic aspects, and treatment options were included in the analysis.
Based on titles and abstracts, some publications were excluded due to not meeting the inclusion criteria. 49 were included in the literature review.

Results. The literature analysis showed that Capgras syndrome most commonly occurs as a secondary phenomenon in the context of psychotic disorders, substance-induced psychosis, traumatic brain injury, or neurodegenerative diseases. Clinical presentation is highly variable, ranging from isolated delusional beliefs to complex psychopathological conditions with hallucinations or behavioral disturbances. Diagnosis is mainly based on clinical assessment, as there is no unified diagnostic algorithm. Treatment is complex and individualized, most commonly involving antipsychotic medications, with better outcomes observed in cases of functional etiology.

Conclusions. Capgras syndrome is a heterogeneous condition, with clinical presentation and prognosis depending on its etiology. Early recognition of the syndrome, comprehensive psychiatric and neurological evaluation, and individualized treatment are essential to reduce the risk of complications and improve patients' quality of life.

Keywords: Capgras syndrome, delusional misidentification syndromes, diagnosis, psychosis

SANTRAUKA

Įvadas. Capgraso sindromas yra sutrikusios identifikacijos sindromų grupei priklausantis psichopatologinis reiškinys, pasireiškiantis kliedesiniu įsitikinimu, kad artimas asmuo ar objektas buvo pakeistas identišku antrininku. Nors sindromas yra retas, jis turi reikšmingą klinikinę svarbą dėl diagnostikos sudėtingumo ir sąsajų su įvairiais psichikos bei neurologiniais sutrikimais.

Tikslas. Pateikti Capgraso sindromo diagnostikos ypatumus ir sindromą iliustruojamus klinikinius atvejus.

Metodai. Literatūros paieška buvo atlikta tarptautinėse mokslinėse duomenų bazėse ir platformose, įskaitant PubMed, ScienceDirect, ResearchGate. Paieškos metu buvo naudojami raktiniai žodžiai: "Capgras syndrome", "delusional misidentification syndrome", "delusion of doubles", "misidentification delusion".

Paieška aprėpė publikacijas, paskelbtas 1923–2025 m. Į analizę įtraukti anglų kalba publikuoti straipsniai, kuriuose aprašomi Capgraso sindromo atvejai, jo etiopatogenezė, klinikiniai požymiai, diagnostikos ypatumai ir gydymo galimybės.

Remiantis straipsnių pavadinimais ir santraukomis, dalis publikacijų buvo atmesta dėl neatitikimo įtraukimo kriterijams. Iš viso į literatūros apžvalgą buvo įtraukti 49 darbai.

Rezultatai. Literatūros analizė parodė, kad Capgraso sindromas dažniausiai pasireiškia kaip antrinis reiškinys psichozinių sutrikimų, psichoaktyvių medžiagų sukeltos psichozės, galvos smegenų traumų ar neurodegeneracinių ligų kontekste. Klinikinė raiška yra įvairi – nuo izoliuotų kliedesinių įsitikinimų iki kompleksinių psichopatologinių būklių su haliucinacijomis ar elgesio sutrikimais. Diagnostika daugiausia grindžiama klinikiniu vertinimu, nes nėra vieningo diagnostinio algoritmo. Gydymas yra kompleksinis ir individualizuotas, dažniausiai taikant antipsichozinius vaistus, o geresni rezultatai stebimi funkcinės kilmės atvejais.

Išvados. Capgraso sindromas yra heterogeniška būklė, kurios klinikinė raiška ir prognozė priklauso nuo etiologijos. Ankstyvas sindromo atpažinimas, išsamus psichiatrinis ir neurologinis įvertinimas bei individualizuotas gydymas yra būtini siekiant sumažinti komplikacijų riziką ir pagerinti pacientų gyvenimo kokybę.

Raktažodžiai: Capgraso sindromas, sutrikusios identifikacijos sindromai, diagnostika, psichozė

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INTRODUCTION

"You look exactly like my husband, but I know you are not him – you are his double." These words illustrate one of the most intriguing phenomena in psychiatry – delusional misidentification syndromes (DMS). This rare but clinically significant group of psychiatric disorders is characterized by distorted recognition of people, places, or objects [1].

One of the most well-known conditions within this group is Capgras syndrome. It is defined by a delusional belief that a close person has been replaced by an identical impostor [2] Cases have also been described in which patients believe that not only people but also their homes or pets have been replaced by an imposter [3,4]. The course of these delusions may vary. They can be transient, recurrent, or persistent, depending on the underlying disorder and comorbid conditions [5].

Other disorders classified as delusional misidentification syndromes include Fregoli delusion, intermetamorphosis, and subjective doubles [5]. Capgras syndrome was first described in the early 20th century by the French psychiatrist Jean Marie Joseph Capgras (1873–1950) together with Reboul-Lachaux in their work "L'illusion des sosies" ("The Illusion of Doubles"). The publication recounts the tragic case of Madame de Rio Branco, who, after the death of four children, became convinced that her only surviving daughter had been abducted and replaced by an identical double. Over time, the delusion expanded to include her husband, physicians, police officers, and eventually, according to the patient, herself [6].

A key feature of Capgras syndrome is the patient's firm belief that a familiar person or even the patient themselves has been replaced by an identical impostor. This occurs despite preserved consciousness and often intact cognitive functioning [7]. Patients frequently exhibit mistrust toward others. In some cases, this may progress to hostility or aggressive behavior [8]. Research suggests that these disorders may be associated with impairments in short-term memory and cognitive information processing [9].

Due to its rarity, Capgras syndrome is often unrecognized or is diagnosed late. Its symptoms may be misattributed to schizophrenia spectrum disorders, various forms of dementia, or organic brain pathology [3,10]. The aim of this paper is to discuss diagnostic features of Capgras syndrome and present illustrative clinical cases.

MATERIALS AND METHODS

A literature search was conducted using international scientific databases and platforms, including PubMed, ScienceDirect, and ResearchGate. The search terms used were "Capgras syndrome" and "delusional misidentification syndromes".

Articles were included if they were published in English, focused on Capgras syndrome or delusional misidentification syndromes, and provided relevant clinical, diagnostic, or therapeutic information. Case reports, case series, review articles, and observational studies were included.

Articles were excluded if they were not directly related to the topic, lacked full-text availability, or did not provide sufficient clinical or diagnostic data.

The selection process was performed in several stages. First, titles and abstracts were screened for relevance. Subsequently, full-text articles were assessed for eligibility based on predefined inclusion and exclusion criteria. A total of 46 articles published between 1923 and 2025 were included in this literature review.

Etiology and Epidemiology

Capgras syndrome is a complex condition that may occur in the context of various psychiatric and neurological disorders. For this reason, its epidemiological prevalence remains difficult to determine [11–13]. In most cases, it is described as a secondary phenomenon accompanying other psychiatric or organic disorders. The lack of a unified diagnostic framework further complicates comparisons across studies [14].

Available data suggest that Capgras syndrome occurs more frequently in the context of functional psychiatric disorders (60–75% of cases), although a significant proportion of cases is also associated with structural or neurodegenerative brain pathology (25–40%) [15]. Due to its etiological diversity, the prevalence of the syndrome in the general population is considered very low, approximately 0.12%. In psychiatric inpatient settings, however, reported prevalence rates are higher, ranging from 1.3% to 4.1% [16,17].

Recent studies suggest that Capgras syndrome may be more common in the early stages of psychosis and may present during the first psychotic episode [18,19]. The syndrome may affect individuals of any age. In younger patients, it is more commonly associated with psychotic disorders, whereas in older individuals it is more frequently linked to neurodegenerative or cerebrovascular diseases [3,10].

In the context of organic pathology, Capgras syndrome is most commonly reported in patients with Lewy body dementia, frontotemporal dementia, and Alzheimer's disease [12,20]. Overall, the syndrome is considered a clinical phenomenon that may involve both functional and organic mechanisms. Approximately 56% of cases are of functional origin, 43% organic, and the remainder are classified as mixed or unspecified [21].

Current models suggest that Capgras syndrome arises from impaired communication between facial recognition systems and emotion-processing neural networks, coupled with deficits in cognitive functions responsible for assessing reality [22–26]. The etiological and clinical heterogeneity of the syndrome necessitates thorough psychiatric and neurological assessment in clinical practice.

DIAGNOSIS

Capgras syndrome presents with a wide range of clinical manifestations, and its onset and course are largely dependent on the underlying pathology. Reported cases demonstrate that the disorder may occur in both young patients with psychotic disorders and elderly individuals with organic brain lesions or neurodegenerative diseases [16,27].

Despite attempts to systematize diagnostic approaches, there is currently no universally accepted diagnostic algorithm for Capgras syndrome. One proposed framework suggests combining clinical history with neuroimaging, toxicological testing, and longitudinal observation [28]. In everyday practice, however, diagnosis is primarily based on the core symptom: a fixed, argument-resistant belief that a familiar person has been replaced by an identical impostor.

This delusion most often targets close family members

particularly spouses, children, or parents and persists despite contradictory evidence [29, 30]. According to DSM-5 classification, such symptoms fall within the spectrum of psychotic disorders and may occur in individuals without prior psychiatric history [31].

The course of the syndrome varies depending on its etiology. In some cases, particularly when the underlying condition is well controlled, symptoms may resolve. In contrast, in progressive neurodegenerative diseases or chronic psychotic disorders, CS often becomes persistent and treatment-resistant, with possible relapses [32].

The absence of clear diagnostic criteria, combined with diverse symptomatology, contributes to delayed recognition. Clinical presentation may range from isolated delusional beliefs to complex psychopathological states involving dissociation, hallucinations or behavioral changes. Although aggressive behavior is relatively uncommon, it remains clinically significant due to potential safety risks [16].

Capgras syndrome is particularly challenging in cases associated with organic pathology. In such cases, symptoms may develop gradually and appear long after a traumatic brain injury or neurological event [3,33]. Delusions may be overshadowed by other symptoms, such as cognitive impairment or apathy, and may therefore be misinterpretation as delirium [15,16]. Studies indicate that in these cases delusions are frequently transient, with diagnosis occurring several months after the precipitating event [3].

Symptom onset often coincides with significant clinical events, such as acute psychotic episodes or progression of neurological disease. The course may vary from brief episodes to chronic, recurrent delusional thinking [10]. The relationship with the perceived impostor is usually negative and may intensify over time, occasionally leading to aggression [34]. However, in some cases, patients maintain a positive relationship, believing the impostor to be a better version of the original person [35].

Functional forms of Capgras syndrome are more often associated with complex psychopathological features, including auditory hallucinations, aggressive behavior, or other misidentification syndromes (Fregoli, intermetamorphosis, subjective doubles),whereas organic forms more commonly involve visual hallucinations. Although these associations should not be considered as a diagnostic criteria, as mixed presentations are common [16].

Some studies have explored potential biological markers of Capgras syndrome, including platelet monoamine oxidase (MAO) activity. Reduced MAO activity has been reported in certain psychiatric disorders. It was hypothesized that this parameter might have diagnostic value, particularly during a first psychotic episode. However, subsequent studies found no significant differences between patients with Capgras syndrome, patients with schizophrenia, and healthy individuals. Therefore, the clinical utility of this marker remains limited [11].

In clinical practice, the psychopathology of Capgras syndrome is often described using the concepts of negative and positive double symptoms. The negative double symptom is characterized by the belief that close relatives are impostors attempting to deceive the patient. These beliefs are typically justified by perceived minor changes in behavior or appearance [36].

Differential diagnosis is also important. Capgras syndrome must be distinguished from conditions such as delusion of subjective doubles and prosopagnosia. In subjective doubles, the patient is convinced in the existence of their own double with a separate identity [37, 38]. In contrast, prosopagnosia involves impaired facial recognition due to brain damage, although emotional responses to familiar faces may remain intact [39, 40]. Capgras syndrome has therefore been described as a “mirror image” of prosopagnosia, where recognition is preserved but emotional response is disrupted [41].

Given these diagnostic challenges, comprehensive clinical assessment remains essential. This should include psychiatric evaluation, neurological examination, and, when necessary, additional investigations. The following section presents and discusses clinical cases of Capgras syndrome, illustrating both functional and organic etiologies.

One case described in the literature involves a 23-year-old woman with no prior psychiatric history. She presented with acute anxiety, paranoia, and delusions following intensive use of multiple psychoactive substances (methamphetamine, marijuana, cocaine, and MDMA). The patient believed that her family members were impostors and experienced auditory hallucinations. A diagnosis of substance – induced Capgras syndrome was established based on clinical evaluation, psychiatric consultation, and a detailed medical history [28].

Treatment with aripiprazole and escitalopram resulted in a reduction in delusional intensity and overall clinical improvement. This case highlights that Capgras syndrome may occur not only in primary psychotic disorders, but also in the context of substance – induced psychosis [28].

Another case describes a 75-year-old man previously diagnosed with Lewy body dementia. He developed a delusional belief that his wife had been replaced by one or more identical impostors. The diagnosis was based on psychiatric evaluation and neurological history, considering

Table 1. **Main clinical and diagnostic characteristics of Capgras syndrome**

Category	Key findings
Etiology	Functional (psychotic disorders) organic (neurogenerative diseases, TBI), mixed
Epidemiology	Rare; higher prevalence in psychiatric settings
Clinical presentation	Delusional misidentification, hallucinations, variety of symptoms
Diagnosis	Based on clinical assessment; no standardized algorithm
Differential diagnosis	Prosopagnosia, subjective doubles, other DMS
Treatment	Antipsychotics, multidisciplinary, individualized
Prognosis	Depends on etiology; better in functional cases

progressive cognitive decline.

Unlike typical cases, this patient did not exhibit fear or hostility toward the wife. Instead, he maintained a positive and affectionate attitude. Delusions resolved spontaneously as dementia progressed, and no antipsychotic treatment was initiated. This case illustrates that Capgras syndrome may manifest in neurodegenerative diseases without cold or aggressive behavior [20].

The final case involves a 52-year-old man who presented with one week of aggressive behavior and a firm belief that his wife had been replaced by a double. His medical history revealed a severe traumatic brain injury two months earlier, resulting in loss of vision in the right eye. The clinical picture included visual and auditory hallucinations, disorientation, memory and attention deficits, and anosognosia. Brain MRI revealed bilateral frontal lobe damage and right globe post-traumatic enucleation [2].

The patient was diagnosed with trauma-induced psychotic disorder with Capgras syndrome. Treatment with haloperidol and valproate led to a reduction in delusional intensity and aggressive behavior. Although the delusion resolved, mild anxiety persisted. This case demonstrates the role of structural brain damage in the development of Capgras syndrome [2].

TREATMENT

The treatment of Capgras syndrome remains challenging. This is due to the rarity of the condition, its clinical variety, and the frequent presence of psychiatric or neurological disorders. Currently, there are no standardized treatment guidelines [14].

In clinical practice, a comprehensive approach is typically used. It includes pharmacological treatment, psychosocial interventions, and management of the underlying disorder. Antipsychotic medications are considered the main treatment option. Depending on the clinical presentation antidepressants, benzodiazepines, or antiepileptic drugs may also be prescribed. Treatment response largely depends on the underlying etiology. Better outcomes are generally observed in functionally based cases, whereas organic forms are often more treatment-resistant [42–44].

An essential component of management is the establishment of a safe therapeutic environment, avoiding direct confrontation of delusional beliefs, which may exacerbate anxiety or provoke aggressive behavior [45]. Psychotherapeutic interventions, particularly when combined with pharmacotherapy, may help reduce illness-related stress and improve overall functioning [11,35].

In cases involving severe psychosis, aggressive behavior, or increased suicide risk, hospitalization may be necessary. More intensive treatment strategies, including electroconvulsive therapy, should be considered when indicated [34,46].

DISCUSSION

Capgras syndrome remains a diagnostically challenging condition due to its heterogeneous clinical presentation and diverse etiological background. Based on the reviewed literature, the syndrome is better understood as a psychopathological phenomenon that may arise in different psychiatric and neurological contexts, rather than as a distinct diagnostic entity [3,10,21].

One of the main limitations of the available literature is the predominance of case reports and small case series. Although these studies provide valuable insights into the clinical presentation of Capgras syndrome, they limit the ability to generalize findings, particularly in terms of prevalence, prognosis, and treatment effectiveness. In addition, differences in diagnostic approaches and terminology across studies complicate comparison of results and contribute to variability in reported findings [14,18].

The literature suggests certain differences between functional and organic forms of Capgras syndrome. Functional cases, most often associated with psychotic disorders, tend to present with more complex delusional systems and are frequently accompanied by auditory hallucinations or behavioral disturbances. In contrast, organic forms are more commonly linked to neurodegenerative diseases, traumatic brain injury, or structural brain lesions, and are often associated with cognitive impairment and visual hallucinations [3,12,16]. However, this distinction is not absolute, and mixed clinical presentations are common [16].

The clinical cases presented in this paper further illustrate the variability of Capgras syndrome across different clinical contexts. Despite differences in age, underlying pathology, and symptom expression, all cases shared a core feature – a fixed delusional belief involving misidentification of a familiar person. At the same time, emotional responses toward the perceived impostor varied considerably, ranging from hostility to preserved or even positive emotional attachment [20]. These findings highlight the importance of individualized clinical assessment.

Another important challenge is the lack of standardized diagnostic criteria. In clinical practice, diagnosis is primarily based on clinical evaluation and phenomenological assessment

Table 2. **Summary of representative clinical cases of Capgras syndrome**

Case	Age/Sex	Etiology	Main symptoms	Treatment	Outcome
Case 1	23F	Substance induced psychosis	Delusions, hallucinations	Aripiprazole and escitalopram	Improvement
Case 2	75M	Lewy body dementia	Misidentification, no hostility	No treatment	Spontaneous resolution
Case 3	52M	Traumatic brain injury	Delusions, aggression, hallucinations	haloperidol and valproate	Partial improvement

Taken together, these clinical cases demonstrate several shared features of Capgras syndrome, including a fixed delusional belief involving a familiar person and the presence of psychotic symptoms such as hallucinations or behavioral disturbances. At the same time, they highlight considerable variability in etiology, age of onset, emotional response toward the perceived impostor, and treatment outcomes. These findings support the need for individualized diagnostic evaluation and management, depending on the underlying condition.

rather than objective markers [28]. Although neuroimaging and other investigations may support diagnostic clarification, their role remains complementary. This contributes to delayed recognition and possible misdiagnosis, especially in cases associated with organic pathology [3,33].

From a therapeutic perspective, treatment outcomes appear to depend largely on the underlying etiology. Functional cases may respond more favorably to antipsychotic treatment, whereas organic forms are often more resistant and require a broader multidisciplinary approach [42–44]. The absence of standardized treatment guidelines reflects both the rarity of the syndrome and its clinical heterogeneity [14].

Overall, the findings of this review emphasize the need for more consistent diagnostic approaches and further research into the mechanisms underlying Capgras syndrome. A better understanding of its neurobiological and psychological aspects may improve both diagnostic accuracy and treatment strategies.

CONCLUSIONS

Capgras syndrome is a rare and heterogeneous condition belonging to the group of delusional misidentification syndromes. It may occur in both psychiatric and neurological contexts, with manifestations depending on the underlying etiology.

Diagnosis remains challenging due to a variety of symptoms and frequent comorbidities, requiring comprehensive psychiatric and neurological assessment.

Treatment should be individualized and multidisciplinary, focusing on the underlying disorder and symptom management. Early recognition and appropriate intervention may improve patient outcomes and reduce the risk of complications.

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Trumpasis alkoholio ir su alkoholio vartojimu susijusių problemų suvokimo klausimynas (angl. *Short Alcohol and Alcohol Problem Perception Questionnaire, SAAPPQ*)

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Profesinį požiūrį lemia tiek asmeninė patirtis, tiek profesinė praktika bei specifinis kontekstas, kuriame dirba specialistai. Šie veiksniai lemia, kaip požiūris formuojasi ir kaip pasireiškia praktikoje. Todėl specialistų požiūris tampa ypač svarbus dirbant su jautriomis klientų grupėmis, tokiomis kaip asmenys, patiriantys alkoholio vartojimo problemų, kurias dažnai yra lydi stigmatizacija, socialinė atskirtis ar įvairūs psichosocialiniai sunkumai (Finn et al., 2023; Seabra et al., 2025; Tan et al., 2022; Vargas et al., 2020). Metodika, sudaranti galimybę kompleksiškai įvertinti profesinio požiūrio aspektus šiame kontekste, prisideda prie efektyvesnių intervencijų bei geresnių profesinių rezultatų.

Trumpasis alkoholio ir su alkoholio vartojimu susijusių problemų suvokimo klausimynas (angl. *Short Alcohol and Alcohol Problems Perception Questionnaire, SAAPPQ*) (Anderson & Clement, 1987) yra skirtas profesiniam požiūriui į darbą su asmenimis turinčiais alkoholio vartojimo problemų įvertinti ir yra tinkamas taikyti įvairių sveikatos priežiūros sričių specialistams (Anderson et al., 2004; Clancy & Oyefeso, 2019; Geirsson et al., 2005; Iqbal et al., 2015; Johnson et al., 2022; Kennedy et al., 2019; Munoz et al., 2019; Williamson, 2019). Klausimyną sudaro dešimt teiginių, vertinamų 5 balų Likerto skalėje (1 – „visiškai nesutinku“, 5 – „visiškai sutinku“), o aukštesni balai atspindi pozityvesnį požiūrį į darbą su asmenimis, patiriančiais alkoholio vartojimo problemų (3, 4 ir 6 teiginiams taikomas atvirkštinis vertinimas).

SAAPPQ apima penkias subskales: profesinio vaidmens adekvatumą (angl. *role adequacy*; 1 & 2 teiginiai), vaidmens teisėtumą (angl. r*ole legitimacy*; 3 & 4 teiginiai), motyvaciją (angl. *motivation*; 5 & 6 teiginiai), pasitenkinimą darbu (angl. *work satisfaction*; 7 & 8 teiginiai) ir savivertę atliekant užduotis (angl. *task-specific self-esteem*; 9 & 10 teiginiai). Šios subskalės leidžia identifikuoti du latentinius konstruktus – terapeutinį įsipareigojimą (angl. *therapeutic commitment*) ir vaidmens saugumą (angl. *role security*), kurie atspindi specialistų pasirengimą ir pasitikėjimą dirbant su šia klientų grupe (Anderson & Clement, 1987). Tokia struktūra leidžia ne tik įvertinti bendrą specialistų požiūrį, bet ir detaliai analizuoti atskiras profesinio požiūrio dimensijas, svarbias efektyviam darbui su asmenimis, patiriančiais alkoholio vartojimo problemų.

Lietuviška SAAPPQ versija buvo parengta taikant dvigubo vertimo (angl. *double-translation*) metodą, remiantis OECD (2012) rekomendacijomis (Kievišienė, 2024). Klausimyną į lietuvių kalbą nepriklausomai vertė kvalifikuotas vertėjas ir tyrėja, turintys atitinkamos srities kompetencijų. Vertimo versijos buvo lyginamos ir suderintos, įtraukiant du socialinio darbo srities specialistus. Kilę neatitikimai buvo sprendžiami diskusijų ir konsultacijų metu, siekiant užtikrinti semantinį tikslumą ir konceptualų atitikimą originaliai metodikai. Atsižvelgiant į tai, kad SAAPPQ buvo sukurtas 1987 m., dalis formuluočių šiuolaikiniame kontekste gali būti laikomos stigmatizuojančiomis, todėl šiame tyrime naudota terminologija buvo adaptuota, siekiant užtikrinti neutralios ir etiškos kalbos vartojimą (pagal Mahmoud et al., 2020).

Kaip pažymi Price ir kt. (2015), skalės validumas yra sprendimas, paremtas įvairiais įrodymais, todėl SAAPPQ validumas grindžiamas keliais argumentais. Visų pirma, šiame tyrime nustatytas pakankamas vidinis suderinamumas (Cronbach $\alpha = 0{,}764$), rodantis gero lygio patikimumą. Be to, metodika plačiai taikyta įvairiuose tyrimuose ir skirtingose šalyse (Anderson et al., 2003; Crothers & Dorrian, 2011; Geirsson et al., 2005; Kennedy et al., 2019; Munoz et al., 2019; Berstein et al., 2022), taip pat naudota tarptautiniuose tyrimuose, tokiuose kaip WHO devynių šalių analizė (Anderson et al., 2003) ar AMPHORA (Drummond et al., 2013). Šiuose tyrimuose nustatytas stabilus skalių pasiskirstymas skirtinguose kontekstuose (Keurhorst et al., 2016). Taigi, SAAPPQ pasižymi pakankamu patikimumu ir validumu bei yra tinkamas instrumentas specialistų profesiniam požiūriui vertinti.

Apibendrinant galima teigti, kad ši metodika yra plačiai naudojama, kultūriškai adaptyvi ir suteikia galimybę išsamiai suprasti specialistų darbo su asmenimis, patiriančiais alkoholio vartojimo problemų, ypatumus.

Trumpasis alkoholio ir su alkoholio vartojimu susijusių problemų suvokimo klausimynas (angl. *SAAPPQ*)

Nr.	Teiginys	Visiškai nesutinku	Nesutinku	Nei sutinku, nei nesutinku	Sutinku	Visiškai sutinku
1.	Manau, kad apie alkoholio vartojimo priežastis žinau pakankamai, kad galėčiau atlikti savo pareigas darbe su asmenimis, turinčiais alkoholio vartojimo problemų.	1	2	3	4	5
2.	Aš jaučiu, kad galiu tinkamai konsultuoti savo klientus apie alkoholio vartojimą bei jo padarinius.	1	2	3	4	5
3.	Aš jaučiu, kad nedaug kuo galiu didžiuotis savo darbe su asmenimis, turinčiais alkoholio vartojimo problemų.	1	2	3	4	5
4.	Apskritai, esu linkęs (linkusi) manyti, kad man prastai sekasi dirbti su asmenimis, turinčiais alkoholio vartojimo problemų .	1	2	3	4	5
5.	Aš noriu dirbti su asmenimis, kurie turi alkoholio vartojimo problemų.	1	2	3	4	5
6.	Pesimizmas yra realiausias požiūris į asmenis, kurie turi alkoholio vartojimo problemų.	1	2	3	4	5
7.	Manau, kad turiu teisę paklausti klientų apie jų alkoholio vartojimą, kai to reikia.	1	2	3	4	5
8.	Manau, kad mano klientai žino, kad aš turiu teisę paklausti jų apie jų alkoholio vartojimą, kai to reikia.	1	2	3	4	5
9.	Apskritai, darbas su asmenimis, kurie turi alkoholio vartojimo problemų man teikia pasitenkinimą.	1	2	3	4	5
10.	Apskritai, man patinka dirbti su asmenimis, kurie turi alkoholio vartojimo problemų.	1	2	3	4	5

Pastaba: SAAPPQ (požiūrio į darbą su asmenimis, turinčiais alkoholio vartojimo problemų) klausimynas gali būti naudojamas moksliniams ir klinikiniams tyrimams, tačiau publikuojant rekomenduojama pateikti nuorodą į originalų šaltinį: Anderson, P., & Clement, S. (1987). The AAPPQ Revisited: Measurement of general practitioners' attitudes to alcohol problems. British Journal of Addiction, 82, 753–759.

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Giedre Vaiciene – Relationships between objective and subjective movement system indicators and chronic nonspecific low back pain among young office workers

Dr. Giedre Vaiciene is a physical therapist and researcher at the Lithuanian University of Health Sciences (LSMU). She earned her MSc from LSMU, where she also completed her PhD studies, defending her doctoral thesis, "Relationships between objective and subjective movement system indicators and chronic nonspecific low back pain among young office workers," on May 27, 2025. Currently, Dr. Vaiciene works as a researcher at the Laboratory of Behavioural Medicine (within the Neuroscience Institute) and as a physical therapist at the Stress Diseases Department of LSMU's Palanga Clinic.

INTRODUCTION

Chronic nonspecific low back pain (LBP) is a multifaceted issue characterized by the complex interplay of biological, psychological, and social factors. It is a leading cause of disability among young and middle-aged individuals, and its management remains challenging due to the myriad factors contributing to pain chronification. Scientific studies suggest that a purely biomedical approach is insufficient, highlighting the need to evaluate a broader biopsychosocial context that incorporates physical, psychological, and social aspects.

Office work and low physical activity levels are recognized as major health challenges in modern society. Prolonged sitting not only leads to muscle imbalances but also alters biomechanical body patterns, resulting in increased stress on the spine and musculoskeletal structures. Additionally, static postures at work, stress, and poor sleep can amplify pain perception and increase the likelihood of chronicity. To systematically capture this complexity, these factors can be structured using the International Classification of Functioning, Disability, and Health (ICF) model, which integrates biological, functional, social, and environmental dimensions.

By integrating both objective clinical measures of the movement system and subjective patient-reported experiences, the ICF framework provides a comprehensive baseline to evaluate the pain-related functional status of young office workers. Such analysis provides a foundation for more targeted research, contributing directly to the development of precise physiotherapy diagnoses and personalized recommendations aimed at preventing long-term functional limitations and improving patients' quality of life.

AIM

To identify and evaluate the relationships between objective and subjective movement system indicators and chronic nonspecific low back pain in young office workers.

OBJECTIVES

1. To assess the spinal functional status of young office workers using the culturally adapted and validated Lithuanian version of the Spine Functional Index questionnaire.
2. To determine and evaluate the associations between movement system indicators, subjective functional status, and chronic nonspecific low back pain intensity in young office workers.
3. To analyze relationships between office work experience and both subjective and objective functional status in young office workers.
4. To evaluate the factors influencing the subjective functional status of young office workers based on the biopsychosocial model.

CONCLUSIONS

1. Using the culturally adapted and validated Lithuanian version of the Spine Functional Index, it was determined that the majority of young office workers assessed their subjective functional status as better than average. The questionnaire results did not reveal significant differences between genders but reliably identified associations between functional limitations and pain experience, confirming its suitability for evaluating spine functional status in this population.
2. Subjective functional limitations in young office workers are associated with higher pain intensity, older age, longer duration of low back pain, poorer body awareness, and increased lumbar erector spinae muscles activation. A weak relationship was identified between vertebral rotation and greater subjective functional limitations, but no significant associations were found between other postural parameters and either chronic nonspecific LBP intensity or functional status. Muscle strength indicators varied: in the male group, functional limitations were linked to reduced trunk muscle strength, whereas no such associations were observed in females. In both sexes, greater imbalances in left and right hip extensor muscle strength were significantly associated with increased functional limitations, although these imbalances were not linked to chronic nonspecific LBP intensity.
3. Chronic nonspecific LBP intensity reveals a relationship between longer office work duration and greater subjective functional limitations, even in the absence of a direct influence of work duration. Increased bilateral lumbar erector spinae muscles electrical activity serves as a critical mediating factor through which prolonged office work worsens subjective functional status. Isolated relationships were identified between longer office work duration, reduced maximal strength of trunk extensors and flexors, and poorer subjective functional status. However, trunk muscle strength was not a significant mediating factor in the relationship between work duration and subjective functional status.
4. Increased vertebral rotation, greater imbalance in lumbar erector spinae muscles electrical activity during dynamic movement, larger asymmetry in hip extensor muscle strength between the left and right sides, higher pain intensity, and increased emotional awareness of bodily sensations – have a significant direct effect on poorer subjective functional status. Conversely, factors such as age, low back pain duration, body awareness domains such as not-distracting, attention regulation, and trusting, as well as imbalances in trunk flexor and extensor strength, had no statistically significant impact. Other factors, such as postural parameters, office work duration, and subjective physical activity levels, showed no direct associations with subjective functional status and were therefore excluded from the analysis models.

Justina Kievisiene – Use of Self in Social Work with Individuals Having Alcohol Usage Problems in Lithuania

Dr. Justina Kievišienė is a health psychologist and researcher who completed her PhD studies at the Faculty of Social Sciences, University of Lapland, Finland. She defended her doctoral dissertation in 2026. Her research focused on social work practice, particularly the role of professional attitudes and competencies in working with individuals experiencing alcohol use problems. The work emphasizes strengthening evidence-based social work practice and developing professional Use of Self.

Justina Kievišienė is now working as a researcher at the Laboratory of Behavioral Medicine at the LSMU Neuroscience Institute and contributes to clinical and research activities at the institute.

INTRODUCTION

Alcohol use problems remain a significant social and public health issue in Lithuania, contributing to a wide range of challenges including physical and psychological health problems, social issues, and family dysfunction. Social workers are often at the frontline of addressing these issues, working with individuals whose alcohol use is either the primary concern or an underlying factor in broader social problems. Despite the prevalence of these challenges, social work with individuals experiencing alcohol use problems is still relatively under-researched in Lithuania.

Effective intervention in such cases requires not only technical knowledge and skills but also the ability of the social worker to integrate personal and professional dimensions in practice. Therefore, the Use of Self framework is employed to highlight the importance of self-awareness, reflective practice, and the integration of personal and professional qualities in developing effective client relationships. In this context, professional attitudes and competencies play a crucial role, as they directly influence the quality of interaction, client engagement, and intervention outcomes.

Since alcohol use problems are still highly stigmatized, which affects both clients and professionals, negative attitudes, lack of motivation, and insufficient competencies can reduce the effectiveness of social work interventions. Therefore, it is important to gain a deeper understanding of how social work is currently practiced in Lithuania and to identify the key professional factors influencing this practice.

AIM

To explore social work with individuals experiencing alcohol usage problems in Lithuania and to analyze professional attitudes and competences as components of the "Use of Self" framework.

OBJECTIVES

1. To analyze the context of alcohol consumption in Lithuania and its implications for social work practice.
2. To examine social work methods, services, practices, and challenges when working with individuals with alcohol use problems.
3. To investigate social workers' professional attitudes toward working with individuals experiencing alcohol use problems.
4. To assess social workers' professional competences and identify key competence dimensions in the context of alcohol use problems.

CONCLUSIONS

1. The study revealed that social work with individuals experiencing alcohol use problems in Lithuania is still in a developmental stage and is primarily oriented toward individual-level interventions. The individual casework method is the most commonly used approach, while group work and community-based interventions remain underused. The most frequent services provided by social workers include consultation, information provision, and mediation, while care services are less commonly applied. One of the main challenges identified in practice is the lack of client motivation, which significantly affects intervention outcomes.

2. The findings also showed that social workers generally hold positive professional attitudes toward working with individuals experiencing alcohol use problems. Role legitimacy and a sense of professional responsibility were particularly strong. However, lower levels of work motivation and job satisfaction indicate the need for additional support for professionals in this field. Education and work experience were found to influence attitudes, with higher education levels and moderate work experience associated with more positive attitudes.

3. In terms of professional competences, the study identified several key competence areas, including consultation, emotional support, case management, teamwork, and relationship-building. Social workers demonstrated strong abilities in interpersonal work and client support, but competences related to professional resilience and community work were less developed. This highlights the need for further development of holistic competence, integrating both procedural skills and meta-competences such as self-awareness and emotional regulation.

4. Overall, the study emphasizes the importance of the "Use of Self" as a central component of effective social work practice. By integrating professional knowledge, skills, attitudes, and personal awareness, social workers can build stronger relationships with clients and achieve more effective outcomes. The findings also suggest a need to strengthen social work education and practice by promoting self-reflection, reducing stigma, and enhancing competences, particularly in community-based and preventive interventions.