WORLD JOURNAL OF PHARMACEUTICAL RESEARCH
SJIF Impact Factor 8.453
Volume 14, Issue 11, 371-383. Review Article ISSN 2277–7105

A SYSTEMATIC REVIEW ON RISK FACTORS AND TREATMENT STRATEGIES FOR DEPRESSION

[1]*Aishwarya A. Nandyalkar, [2]Dr. Vishnu A. Kangralkar and [3]Sujata M. Halagekar

[1]Department of Pharmacology, Maratha Mandal College of Pharmacy, Belgaum - 590016, Karnataka, India.

Article Received on
08 April 2025,

Revised on 29 April 2025,
Accepted on 20 May 2025

DOI: 10.20959/wjpr202511-36878

***Corresponding Author**
Aishwarya A. Nandyalkar
Department of Pharmacology, Maratha Mandal College of Pharmacy, Belgaum - 590016, Karnataka, India.

ABSTRACT

Depression is a prevalent psychiatric condition characterized by persistent low mood and functional impairment, posing a significant public health challenge globally. Despite decades of research the precise neurobiological mechanisms underlying depression remain incompletely understood. Genetic predisposition plays a pivotal role in the onset and progression of depression as evidenced by family, twin and adoption studies. Although early genetic investigations such as linkage and association studies laid the groundwork, recent genome-wide association studies have highlighted the complexity of genetic contributions emphasizing that multiple variants with small effects interact with environmental factors to influence disease susceptibility. Concurrently, evolving hypotheses-ranging from neurotransmitter imbalances and hypothalamic-pituitary-adrenal (HPA) axis dysfunction to cytokine-mediated inflammation and impaired neuroplasticity—reflect the multifactorial nature of depression pathogenesis. Recent advances underscore the significance of systemic and multi-organ interactions in disease progression and open avenues for multitarget therapeutic strategies. Additionally, novel pharmacological agents are demonstrating promising outcomes in clinical settings. Depression is also frequently comorbid with various physical illnesses leading to worse prognoses, increased healthcare burden and complex treatment demands. This bidirectional relationship is potentially driven by shared biological and psychological risk factors and lifestyle stressors. Integrated treatment strategies, including lifestyle modifications, Pharmacological Treatments, Psychotherapy, Neuromodulation Techniques are emerging as effective approaches. This review provides a comprehensive synthesis of

recent developments in the etiology, diagnosis, comorbidity, and management of depression, highlighting ongoing research and future directions for optimized care.

KEYWORDS: Depression, Mood Disorder, Factors for Depression and Treatments.

INTRODUCTION

Depression is a common and serious mental health condition characterized by persistent feelings of sadness, loss of interest or pleasure in daily activities and a range of emotional and physical symptoms that interfere with everyday functioning.[1] The effects of depression are due to the changes in the monoamine neurotransmitters, specifically norepinephrine, serotonin and dopamine in the brain.[2] Depression is characterized by feelings of melancholy, hopelessness, despair and discouragement. Depression is also characterized by reduced thinking, pleasurable concentration and sleep disturbance.[3] It can affect anyone, regardless of age, gender or background and is influenced by a complex interplay of genetic, biological, psychological and environmental factors.[4]

The World Health Organization (WHO) states that depression is one of the main causes of disability and the most prevalent cause worldwide.[5] These mental illnesses develop into chronic or recurrent conditions that fundamentally impair an individual's ability to manage day-to-day tasks. Approximately 20% of people worldwide suffer from depression.[6] Depression is currently the second leading cause for both sexes combined in the 15–44 age group. Men's lifetime risk of depression ranges from 5% to 12%, whereas women's ranges from 10% to 25%. One of the main outcomes of the majority of depressive diseases is suicide. Depression and associated diseases account for about 60% of deaths.[7]

Every year, almost 1 million individuals die by suicide, which is equivalent to 2500 suicide deaths every day. Extreme discouragement and dejection are characteristic of depression a common mental illness. Even though depression only strikes a person once in their lifetime many people have multiple episodes and the symptoms appear throughout the day, often every day.[8]

Depression affects approximately 300 million individuals worldwide and is now one of the leading causes of disability. According to the WHO, Depression was ranked third in terms of disease burden in 2018, but by 2030, it is expected to rank first on the list for disease burden.[9]

Despite the high frequency and severity of depression not all people may benefit equally from current treatments like psychotherapy and medication. Even though antidepressants help a lot of people they can have negative side effects and takes a while to start working and cause problems with long-term use. Since a significant amount of the population is resistant to traditional antidepressants there is an urgent need for novel and more potent therapeutic options.[10]

RISK FACTORS FOR DEPRESSION

1. Genetic and Biological Factors.
2. Psychological Factors.
3. Social and Environmental Factors.
4. Demographic Factors.
5. Medical Conditions and Substance Use.

Genetic and Biological Factors

Genetic Predisposition[11,12]

Individuals with a family history of depression particularly first-degree relatives have a higher risk of developing the condition. Family and twin studies suggest that genetic factors account for approximately 40% to 50% of the risk for Depression. Genome-wide association studies have identified multiple genetic variants linked to depression highlighting the role of heredity.

Neurotransmitter Imbalances[13,14]

Neurotransmitter imbalances particularly involving serotonin, norepinephrine and dopamine play a significant role in the development and manifestation of depression. The monoamine hypothesis suggests that deficiencies or dysregulations in these neurotransmitters can lead to depressive symptoms. Serotonin is crucial for mood regulation, sleep and appetite; norepinephrine affects attention and response actions and dopamine is associated with motivation and reward mechanisms. Alterations in the levels or functioning of these neurotransmitters can disrupt neural communication, leading to the emotional and physical symptoms observed in depression. Understanding these imbalances has been fundamental in developing antidepressant treatments that aim to restore neurotransmitter levels and alleviate depressive symptoms.

Hormonal Changes[15,16]

Hormonal fluctuations significantly influence the onset and progression of depression, particularly in women. Key hormones such as estrogen, progesterone, cortisol and thyroid hormones play pivotal roles in mood regulation. For instance, during the postpartum period, abrupt declines in estrogen and progesterone levels have been linked to postpartum depression highlighting the impact of reproductive hormones on emotional well-being. Elevated cortisol levels often a response to chronic stress have been associated with depressive symptoms suggesting that dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis may contribute to mood disorders. Additionally, thyroid hormone imbalances such as hypothyroidism can manifest as depressive symptoms, underscoring the importance of endocrine health in mental well-being. Understanding these hormonal influences is crucial for developing targeted interventions and treatments for depression.

PSYCHOLOGICAL FACTORS

Personality Traits[17,18]

Personality traits play a significant role in the development and progression of depression. Research indicates that high levels of neuroticism a trait characterized by emotional instability and a propensity for negative emotions are consistently associated with an increased risk of depressive disorders. Individuals exhibiting high neuroticism are more susceptible to stress and may perceive everyday situations as threatening, leading to heightened emotional responses. Conversely, traits like low extraversion (indicative of social withdrawal) and low conscientiousness (reflecting disorganization and lack of goal-directed behavior) have also been linked to higher depression rates. These personality dimensions can influence how individuals respond to life stressors, potentially exacerbating or mitigating depressive symptoms. Understanding the interplay between personality traits and depression is crucial for developing targeted prevention and intervention strategies.

Cognitive Patterns[19]

Cognitive patterns significantly influence the development and maintenance of depression. Individuals with depression often exhibit negative cognitive biases, such as a tendency to interpret neutral or ambiguous information pessimistically and to recall negative experiences more readily than positive ones. These maladaptive thought patterns can perpetuate feelings of hopelessness and low self-worth. Neuroimaging studies have revealed that such cognitive distortions are associated with hyperactivity in brain regions like the amygdala, which

processes negative emotions and hypoactivity in areas like the dorsolateral prefrontal cortex, responsible for cognitive control and emotion regulation. This neural imbalance may underlie the persistent negative thinking and impaired emotional regulation observed in depression.

Social and Environmental Factors

Chronic Stress[20,21]

Chronic stress is a significant risk factor for depression, exerting profound effects on both brain structure and function. Prolonged exposure to stress activates the hypothalamic-pituitary-adrenal (HPA) axis, leading to sustained elevations in cortisol levels. This hormonal imbalance can result in hippocampal atrophy, reduced neurogenesis and impaired synaptic plasticity all of which are associated with depressive symptoms. Moreover, chronic stress induces neuroinflammation characterized by the release of proinflammatory cytokines, further exacerbating neuronal damage and mood disturbances. These neurobiological changes underscore the critical role of chronic stress in the onset and progression of depression.

Life Events[22]

Major life events such as divorce, job loss, sexual abuse or the death of a loved one are significant stressors that can trigger depressive episodes. These events can lead to a condition known as situational depression or adjustment disorder with depressed mood, characterized by symptoms like sadness, hopelessness, irritability and difficulty managing daily tasks. Symptoms typically emerge within 90 days of the triggering event and often resolve within six months. However, if symptoms persist or worsen it may indicate the development of major depressive disorder necessitating professional intervention.

Demographic Factors

Gender[23]

Gender plays a significant role in the prevalence and manifestation of depression. Women are nearly twice as likely as men to experience depression a disparity influenced by a combination of biological, hormonal and psychosocial factors. Hormonal fluctuations related to menstruation, pregnancy and menopause can affect mood regulation in women. Additionally, societal pressures, gender-based violence and socioeconomic disadvantages contribute to the increased risk. Women are also more likely to seek help and report depressive symptoms which may lead to higher diagnosis rates. Conversely, men may exhibit depression through irritability, anger or substance abuse and are less likely to seek treatment potentially leading to underdiagnosis.

Age[24,25]

Age is a key risk factor in depression with its prevalence and underlying causes varying across life stages. Young adults (18–29 years) show the highest rates of depressive symptoms about 21% driven by social pressures, financial stress and life transitions. In middle-aged adults (30–64 years), lifestyle factors such as smoking, alcohol use and physical inactivity can contribute to mental health decline often becoming noticeable by the mid-30s. Among older adults (65+) the prevalence of depression is debated; some studies report underdiagnosis while others indicate higher rates up to 31.74% primarily due to chronic illness, social isolation and reduced functional ability.

Medical Conditions and Substance Use

Chronic Illnesses[26]

Chronic illnesses significantly elevate the risk of developing depression due to various interrelated factors. The persistent stress and anxiety associated with managing a long-term health condition can contribute to depressive symptoms. Additionally, certain chronic diseases such as Parkinson's disease, cardiovascular diseases, cancer or stroke may cause brain changes that increase vulnerability to depression. Medications used to treat chronic illnesses can also have side effects that impact mood. Furthermore, individuals with multiple chronic conditions are at an even higher risk as the complexity of managing several health issues can exacerbate emotional distress.

Substance Abuse[27]

Substance abuse is a significant risk factor for depression, with a complex bidirectional relationship between the two conditions. Individuals may turn to substances like alcohol or drugs as a form of self-medication to alleviate depressive symptoms. However this often exacerbates the condition over time. Conversely, prolonged substance use can alter brain chemistry increasing the likelihood of developing depression. Studies indicate that approximately one-third of people with major depression also struggle with alcohol problems and nearly half of those with substance use disorders have co-occurring mental health conditions such as depression. This interplay underscores the importance of integrated treatment approaches that address both substance use and mental health disorders simultaneously.

TREATMENTS FOR DEPRESSION

1. Pharmacological Treatments.

2. Psychotherapy.
3. Neuromodulation Techniques.
4. Lifestyle Modifications.

Pharmacological Treatments[28,29]

Selective Serotonin Reuptake Inhibitors (SSRIs)

Selective Serotonin Reuptake Inhibitors (SSRIs), including fluoxetine, sertraline and escitalopram are commonly prescribed due to their efficacy and relatively favorable side effect profiles. They function by increasing serotonin levels in the brain, which can improve mood and emotional state. These medications are often considered first-line treatments for depression.

Serotonin-Norepinephrine Reuptake Inhibitors (SNRIs)

Serotonin-Norepinephrine Reuptake Inhibitors (SNRIs), such as venlafaxine and duloxetine target both serotonin and norepinephrine neurotransmitters. This dual action can be beneficial for patients who do not respond to SSRIs alone.

Atypical Antidepressants

This category includes medications like bupropion and mirtazapine, which have unique mechanisms of action. For instance, bupropion affects dopamine and norepinephrine levels and is often chosen for patients concerned about sexual side effects associated with other antidepressants.

Tricyclic Antidepressants (TCAs) and Monoamine Oxidase Inhibitors (MAOIs)

Tricyclic Antidepressants (TCAs) (e.g. amitriptyline) and Monoamine Oxidase Inhibitors (MAOIs) (e.g. phenelzine) are older classes of antidepressants. While effective, they are generally reserved for cases where newer medications are ineffective due to their potential for more severe side effects and dietary restrictions.

Psychotherapy[1,30,31]

Cognitive Behavioral Therapy (CBT)

Cognitive Behavioral Therapy (CBT) is a structured, time-limited therapy that focuses on identifying and modifying negative thought patterns and behaviors contributing to depression. Patients learn to recognize cognitive distortions and develop coping strategies to manage symptoms. Cognitive Behavioral Therapy (CBT) is typically structured over 12 to 16

sessions. Research indicates that Cognitive Behavioral Therapy (CBT) is effective for mild to moderate depression and can be combined with pharmacotherapy for enhanced outcomes.

Psychodynamic Therapy

This therapy explores unconscious processes and past experiences influencing current behavior and emotions. By gaining insight into unresolved conflicts patients can understand the root causes of their depression. Psychodynamic therapy has demonstrated efficacy especially in long-term treatment plans.

Interpersonal Therapy (IPT)

Interpersonal Therapy (IPT) addresses interpersonal issues and life events that may contribute to depressive episodes. The therapy typically spans 12–16 weeks and focuses on improving communication skills and social functioning. Studies have shown IPT to be effective particularly when depression is linked to relationship conflicts or significant life changes.

Neuromodulation Techniques[32,33,34]

Electroconvulsive Therapy (ECT)

Electroconvulsive Therapy (ECT) is a well-established medical treatment used primarily for individuals with severe or treatment-resistant depression. It involves the application of small electrical currents to the brain under general anesthesia to induce controlled seizures which are believed to cause changes in brain chemistry that rapidly relieve depressive symptoms. ECT is typically administered two to three times a week over several weeks and has shown high efficacy particularly for patients who do not respond to medications or psychotherapy. Despite its effectiveness, ECT may cause temporary side effects such as confusion and short-term memory loss but modern techniques have significantly reduced these risks. It is often considered when rapid improvement is necessary such as in cases of suicidal ideation or severe functional impairment.

Transcranial Direct Current Stimulation (tDCS)

Transcranial Direct Current Stimulation (tDCS) is a non-invasive neuromodulation technique explored for treating depression. It involves applying a low-intensity electrical current to specific brain regions typically the dorsolateral prefrontal cortex to modulate neuronal activity. Meta-analyses of randomized controlled trials have indicated that active tDCS can lead to modest improvements in depressive symptoms compared to sham treatments with effect sizes ranging from small to moderate. For instance, a comprehensive analysis reported

a Hedge's g of 0.46 suggesting a moderate effect size in favor of tDCS over placebo. tDCS is generally well-tolerated with minimal side effects making it a potential alternative or adjunctive therapy, especially for patients who do not respond to conventional treatments. However, the variability in study outcomes underscores the need for further large-scale, well-designed trials to establish standardized protocols and determine its long-term efficacy and safety.

Vagus Nerve Stimulation (VNS)

Vagus Nerve Stimulation (VNS) is an FDA-approved neuromodulation therapy for treatment-resistant depression (TRD) particularly in cases unresponsive to conventional treatments. The procedure involves surgically implanting a device that delivers mild electrical impulses to the left vagus nerve in the neck which then transmits signals to brain regions involved in mood regulation. While the exact mechanism is not fully understood, VNS is believed to influence neurotransmitter systems including serotonin and norepinephrine thereby alleviating depressive symptoms. Additionally, long-term observational studies have indicated sustained antidepressant effects with a 40% response rate observed over a 12-week period.

Deep Brain Stimulation (DBS)

Deep Brain Stimulation (DBS) is an investigational neuromodulation therapy for individuals with treatment-resistant depression (TRD) who have not responded to conventional treatments such as medications or psychotherapy. The procedure involves surgically implanting electrodes into specific brain regions most notably the subcallosal cingulate cortex that are associated with mood regulation. These electrodes deliver continuous electrical impulses to modulate neural activity and alleviate depressive symptoms. Clinical studies have reported response rates averaging around 60% though outcomes can vary among individuals and require careful optimization of stimulation parameters. While DBS is generally considered safe it is invasive and carries risks associated with surgery. Ongoing research aims to refine targeting techniques and improve patient selection to enhance efficacy.

Lifestyle Modifications[35,36]

Physical Activity

Regular physical activity is a well-established, evidence-based intervention for managing depression particularly in individuals with mild to moderate symptoms. Exercise has been shown to reduce depressive symptoms through various mechanisms including the release of

endorphins, improvement in sleep quality and reduction in stress hormones. Psychotherapy and medication may be effectively supplemented or replaced by exercise. Exercise enhances a variety of physical and cognitive traits in addition to mental wellness.

Sleep Hygiene

Sleep hygiene refers to a set of behavioral and environmental practices aimed at promoting consistent, uninterrupted and restful sleep which plays a crucial role in managing depression. Research indicates that poor sleep hygiene such as irregular sleep schedules, excessive screen time before bed and consumption of stimulants can exacerbate depressive symptoms. Conversely, adhering to good sleep hygiene practices like maintaining a consistent sleep schedule creating a comfortable sleep environment and avoiding caffeine or electronic devices before bedtime can improve sleep quality and in turn alleviate symptoms of depression.

CONCLUSION

Depression is a multifaceted illness influenced by biological, psychological and environmental risk factors across the lifespan. Off-time factors like low income, family history and chronic disease show stronger links with depression in unexpected age groups. Early psychiatric intervention and prevention of social deterioration are crucial. Current treatments offer partial relief with many patients not achieving full remission. Emerging pharmacological agents and brain stimulation techniques hold promise for treatment-resistant cases. Continuous care not just acute episode management is vital. Novel therapies must be paired with personalized strategies for better outcomes. Identifying modifiable risk factors can improve prevention. A multidisciplinary approach is key to addressing the complexity of depression.

REFERENCES

1. Otte C, Gold SM, Penninx BW, Pariante CM, Etkin A, Fava M, Mohr DC, Schatzberg AF. Major depressive disorder. Nature reviews Disease primers, Sep. 15, 2016; 2(1): 1-20.
2. Chuang CY, Shi YC, You HP, Lo YH, Pan TM. Antidepressant effect of GABA-rich monascus-fermented product on forced swimming rat model. Journal of agricultural and food chemistry, Apr. 13, 2011; 59(7): 3027-34.
3. Bee R, Maheshwari KK. Phytochemical screening of antidepressant activity of acyranthes aspera by using forced swimming test in rats.

4. Alkahtani J, Elshikh MS, Dwiningsih Y, Rathi MA, Sathya R, Vijayaraghavan P. In-vitro antidepressant property of methanol extract of Bacopa monnieri. Journal of King Saud University-Science, Nov. 1, 2022; 34(8): 102299.
5. Akshatha S, Shetty SK. Ayurvedic management to aid weaning of anti-depressants: A case report. Journal of Ayurveda Case Reports, Apr. 1, 2021; 4(2): 50-3.
6. Yousuf S, Haq SM, Rasool A, Zulfajri M, Hanafiah MM, Nafees H, Tasneem S, Mahboob M. Evaluation of antidepressant activity of methanolic and hydroalcoholic extracts of Acorus calamus L. rhizome through tail suspension test and forced swimming test of mice. Journal of Traditional Chinese Medical Sciences, Sep. 1, 2020; 7(3): 301-7.
7. Jawaid T, Imam SA. Antidepressant activity of methanolic extract of Verbena Officinalis Linn. plant in mice. Asian Journal of Pharmaceutical and Clinical Research, 2015; 8(4): 308 10.
8. Nikam P, Kangralkar VA, Godabole S, Goundar D, Kelkar P. Evaluation of anti-depressant activity of seed extract of eleusine coracana l. In experimental rats, 2022; 12(1): 1314-1320.
9. Cui L, Li S, Wang S, Wu X, Liu Y, Yu W, Wang Y, Tang Y, Xia M, Li B. Major depressive disorder: hypothesis, mechanism, prevention and treatment. Signal transduction and targeted therapy, Feb. 9, 2024; 9(1): 30.
10. Kushwaha Y, Tiwari P, Srivastava M, KA S, Rai P. Evaluation of Anti-Depressant Behaviour of Alcoholic Extract of Tectona grandis Linn. on Rats. Lat. Am. J. Pharm., 2023; 42(6): 1-8.
11. Lohoff FW. Overview of the genetics of major depressive disorder. Current psychiatry reports, Dec. 2010; 12: 539-46.
12. Tsuang MT, Taylor L, Faraone SV. An overview of the genetics of psychotic mood disorders. Journal of psychiatric research, Jan. 1, 2004; 38(1): 3-15.
13. Nutt DJ. Relationship of neurotransmitters to the symptoms of major depressive disorder. J Clin psychiatry, Jan. 1, 2008; 69(E1): 4-7.
14. Bhatt S, Devadoss T, Manjula SN, Rajangam J. 5-HT3 receptor antagonism: a potential therapeutic approach for the treatment of depression and other disorders. Current neuropharmacology, Sep. 1, 2021; 19(9): 1545-59.
15. Hendrick V, Altshuler LL, Suri R. Hormonal changes in the postpartum and implications for postpartum depression. Psychosomatics, Mar. 1, 1998; 39(2): 93-101.
16. Mokrani MC, Duval F, Erb A, Lopera FG, Danila V. Are the thyroid and adrenal system alterations linked in depression?. Psychoneuroendocrinology, Dec. 1, 2020; 122: 104831.

17. Struijs SY, Lamers F, Verdam MG, van Ballegooijen W, Spinhoven P, van der Does W, Penninx BW. Temporal stability of symptoms of affective disorders, cognitive vulnerability and personality over time. Journal of Affective Disorders, Jan. 1, 2020; 260: 77-83.
18. Struijs SY, de Jong PJ, Jeronimus BF, van der Does W, Riese H, Spinhoven P. Psychological risk factors and the course of depression and anxiety disorders: A review of 15 years NESDA research. Journal of Affective Disorders, Dec. 1, 2021; 295: 1347-59.
19. Solis E, Antypa N, Conijn JM, Kelderman H, Van der Does W. Psychometric properties of the Leiden Index of Depression Sensitivity (LEIDS). Psychological assessment, Feb. 2017; 29(2): 158.
20. Yang L, Zhao Y, Wang Y, Liu L, Zhang X, Li B, Cui R. The effects of psychological stress on depression. Current neuropharmacology, Jul. 1, 2015; 13(4): 494-504.
21. Hassamal S. Chronic stress, neuroinflammation, and depression: an overview of pathophysiological mechanisms and emerging anti-inflammatories. Frontiers in psychiatry, May 11, 2023; 14: 1130989.
22. Bandelow B, Gutermann J, Peter H, Wedekind D. Early traumatic life events, parental attitudes, family history, and birth risk factors in patients with depressive disorder and healthy controls. International journal of psychiatry in clinical practice, Feb. 1, 2013; 17(1): 56-63.
23. Parker G, Brotchie H. Gender differences in depression. International review of psychiatry, Oct. 1, 2010; 22(5): 429-36.
24. Schaakxs R, Comijs HC, van der Mast RC, Schoevers RA, Beekman AT, Penninx BW. Risk factors for depression: differential across age?. The American Journal of Geriatric Psychiatry, Sep. 1, 2017; 25(9): 966-77.
25. Sözeri-Varma G. Depression in the elderly: clinical features and risk factors. Aging and disease, Oct 21, 2012; 3(6): 465.
26. Gürhan N, Beşer NG, Polat Ü, Koç M. Suicide risk and depression in individuals with chronic illness. Community mental health journal, Jul. 1, 2019; 55: 840-8.
27. Siennick SE, Widdowson AO, Woessner MK, Feinberg ME, Spoth RL. Risk factors for substance misuse and adolescents' symptoms of depression. Journal of Adolescent Health, Jan. 1, 2017; 60(1): 50-6.
28. Elias E, Zhang AY, Manners MT. Novel pharmacological approaches to the treatment of depression. Life, Jan. 28, 2022; 12(2): 196.

29. Kovich H, Kim W, Quaste AM. Pharmacologic treatment of depression. American family physician, Feb. 2023; 107(2): 173-81.
30. Dekker JJ, Hendriksen M, Kool S, Bakker L, Driessen E, De Jonghe F, De Maat S, Peen J, Van HL. Growing evidence for psychodynamic therapy for depression. Contemporary Psychoanalysis, Apr. 3, 2014; 50(1-2): 131-55.
31. Srivastava K, Chatterjee K, Prakash J, Yadav A, Chaudhury S. Comparative efficacy of cognitive behavior therapy and interpersonal therapy in the treatment of depression: A randomized controlled study. Industrial Psychiatry Journal, Jan. 1, 2024; 33(1): 160-7.
32. Li M, Yao X, Sun L, Zhao L, Xu W, Zhao H, Zhao F, Zou X, Cheng Z, Li B, Yang W. Effects of electroconvulsive therapy on depression and its potential mechanism. Frontiers in psychology, Feb. 20, 2020; 11: 80.
33. Vlaicu A, Bustuchina Vlaicu M. New neuromodulation techniques for treatment resistant depression. International journal of psychiatry in clinical practice, Jun. 1, 2020; 24(2): 106-15.
34. Drobisz D, Damborská A. Deep brain stimulation targets for treating depression. Behavioural brain research, Feb. 1, 2019; 359: 266-73.
35. Noetel M, Sanders T, Gallardo-Gómez D, Taylor P, del Pozo Cruz B, Van Den Hoek D, Smith JJ, Mahoney J, Spathis J, Moresi M, Pagano R. Effect of exercise for depression: systematic review and network meta-analysis of randomised controlled trials. bmj., Feb. 14, 2024; 384.
36. Rahimi A, Ahmadpanah M, Shamsaei F, Cheraghi F, Sadeghi Bahmani D, Holsboer-Trachsler E, Brand S. Effect of adjuvant sleep hygiene psychoeducation and lorazepam on depression and sleep quality in patients with major depressive disorders: results from a randomized three-arm intervention. Neuropsychiatric disease and treatment, Jun. 22, 2016; 1507-15.