Indian J. Applied & Pure Bio. Vol. 41(1), 420-431 (2026). **ISSN: 0970-2091**

From Brain to Behaviour: A Review of Neuropsychiatric Treatments for Mood and Anxiety Disorders

[1]*Muskan Tomar, [2]Harshit Shringi and [3]Manoj Goyal

[1,3]Department of Pharmacology Indore Mahavidyalaya, Indore - 453111 (India)
[2]Parijat College of Pharmacy, Indore - 452010 (India)
Corresponding Author – Muskan Tomar
Department of Pharmacology
***Email** – muskantomar808@gmail.com

Abstract

Among the most common neuropsychiatric conditions, depression and anxiety impact millions of people worldwide and present serious public health issues. These disorders cause significant emotional, cognitive, and functional impairments due to intricate interplay between neurobiological, psychosocial, and environmental variables. With an emphasis on their neuropsychological foundations, this study offers a thorough examination of both established and novel therapy approaches for treating anxiety and depression. For many individuals, traditional pharmaceutical treatments including benzodiazepines, serotonin-norepinephrine reuptake inhibitors (SNRIs), and selective serotonin reuptake inhibitors (SSRIs) continue to be the first choice. Nevertheless, these treatments frequently have drawbacks because of their partial response, delayed commencement of action, and adverse effects. Consequently, non-pharmacological and neuromodulator methods such as transcranial magnetic stimulation (TMS), electroconvulsive treatment (ECT), mindfulness-based interventions, and cognitive-behavioural therapy (CBT) are gaining popularity. Ketamine, esketamine, and psychedelic-assisted therapies are examples of new medicines that have been developed recently and give quick and potentially life-changing results in situations that are resistant to previous treatments. Research on neuroinflammation, the gut-brain axis, and personalized medicine has also created new avenues for tailored treatment. There is potential for better results when various therapy methods that are customized for each patient's neurobiological profile are integrated. The significance of a multimodal treatment strategy that incorporates pharmaceutical, psychological, and developing therapies is emphasized by this review. In order to maximize the effectiveness of treatment and get a deeper understanding of the mechanisms behind these diseases, it also emphasizes the necessity of further study.

Keywords - Depression, Anxiety, Neuropsychiatric disorders, Pharmacological treatments, Cognitive Behavioral Therapy (CBT), Mental health treatment.

The causes and symptoms of depression vary greatly because of the intricate interactions between psychological patterns, biological vulnerabilities, and social pressures. Treatment for depression is equally complicated and necessitates a highly customized strategy that may include a mix of therapy, medication, and lifestyle modifications.

Depression and anxiety are among the most prevalent mental health disorders globally, contributing significantly to the global burden of disease. These symptoms are linked to a markedly lower quality of life and are considered to be among the most concerning for those with Parkinson's disease.[49] If affective dysregulation is linked to inadequate dopaminergic supplementation, then managing depression and anxiety by appropriate dopaminergic medication may be successful. Given its shown efficacy, the usage of selective serotonin reuptake inhibitors may be taken into consideration.[50] Following symptom identification, a thorough symptom history is necessary to establish a diagnosis of major depressive disorder (MDD); excluding medical conditions including thyroid disease that may exacerbate depressed symptoms; and assessing for additional mental illnesses that exhibit depressed symptoms, including substance-related disorders, anxiety disorders, and bipolar I or II disorder.[22,33]

Neurobiological Basis of Depression and Anxiety :

Neuroanatomical Pathways: These disorders are linked to changes in parts of the brain, Including the hippocampus, amygdala, and prefrontal cortex. Neurotransmitter Systems Involved: Dopamine, serotonin, and norepinephrine imbalances are important. Genetic and Epigenetic Contributions: These illnesses are a result of a combination of environmental influences and genetic predispositions.[18] Neuroinflammation and Oxidative Stress: The pathophysiology of anxiety and depression has been connected to both chronic inflammation and oxidative stress. Neuroimaging and Biomarkers: Potential biomarkers for diagnosis and therapy response have been found thanks to developments in neuroimaging.[44]

Conventional Pharmacological Treatments:

The main mechanism of serotonin reuptake inhibition is shared by all drugs in the two larger classes of antidepressants (serotonin norepinephrine reuptake inhibitors [SNRIs] and selective serotonin reuptake inhibitors [SSRIs]). Individual agent differences are comparatively small. Nonetheless, it is typical to see that different patients within the same class tolerate or react differently to different antidepressants. Secondary pharmacologic features that differ among the different antidepressants may have an impact on individual differences in medication response; genetic variations affecting the pharmacokinetics and pharmacodynamics of antidepressants may also have an impact on the variability observed in patient responses.

Non-Pharmacological Therapies :

Cognitive Behavioral Therapy (CBT): A structured, time-limited therapy focusing on modifying dysfunctional thoughts and behaviours. In addition to pharmaceutical therapies, cognitive behavioral therapy (CBT) is a significant treatment option for depressive disorders.

Figure 1. Conventional Pharmacological Treatment

Although newly created digital cognitive behavioral therapy techniques have significant benefits since they are more accessible, it is yet unknown how successful they are in comparison to traditional CBT.[7] To find every study that used a cognitive behavioral therapy (CBT)-based intervention—whether in-person or online—for patients suffering from serious depression, we carried out a thorough literature search.[8] The acceptability and efficacy of guided iCBT for the treatment of anxiety and depression in routine care are supported by this study.[27] Health care providers should use interventions that have been validated in randomized controlled clinical trials because of the large degree of heterogeneity between interventions and contexts. If iCBT is used successfully, it could be a useful tool for expanding healthcare in a variety of settings.[26] Mindfulness-Based Interventions: Incorporate mindfulness practices to help individuals manage symptoms and reduce relapse rates.

(a) Effectiveness of Mindfulness-Based Stress Reduction (MBSR): MBSR considerably lowers symptoms of anxiety and depression in both clinical and non-clinical populations.[29]
(b) Mindfulness-Based Cognitive Therapy (MBCT) • Effectiveness: MBCT helps patients with recurrent depression avoid relapsing and lessens the symptoms of anxiety.[32]
(c) MBIs vs. Pharmacotherapy: Research revealed that MBSR was equally effective as escitalopram (SSRI) in treating anxiety disorders.[23]
(d) Meta-Analysis Summary Results: Meta-analyses indicate that MBIs improve well-being and offer moderate-to-large reductions in stress, anxiety, and depression.[19]
(e) Proof from Neurobiology Results: MBIs improve activation in brain regions linked to self-referential processing, emotion management, and attention, according to neuroimaging research.[46]

Psychodynamic and Interpersonal Therapies: Focus on unconscious processes and interpersonal relationships to alleviate symptoms.

PDT (Psychodynamic Therapy)

a. Idea emphasizes how early experiences, unconscious processes, and unresolved issues impact mental health today. seeks to increase emotional intelligence and self-awareness.

b. Effectiveness in Treating Depression and Anxiety: Research indicates that short-term psychodynamic therapy is highly beneficial in the treatment of anxiety and depression. Results: A meta-analysis of 23 RCTs revealed that improvements were maintained after therapy ended and that effect sizes were moderate (g = 0.71 for depression).[10]

IPT (Interpersonal Therapy)

IPT was created to treat depression and focuses on social isolation, role changes, conflict, grieving, and interpersonal functioning. aids patients in enhancing their social connections and adjusting to life changes. Evidence of Efficacy in Depression: IPT is a proven treatment for major depressive disorder (MDD), even in older adults and adolescents.

Results: IPT is superior to a placebo or standard treatment, and it is as effective as CBT (effect size d = 0.63), according to a meta-analysis.[6]

Evidence of Efficacy in Anxiety Disorders: Modified IPT has demonstrated potential in the treatment of generalized anxiety disorder and social anxiety disorder. Findings: IPT for social anxiety was not inferior than CBT, according to RCT data.[45]

Long-Term Advantages

Following treatment, both PDT and IPT show a consistent reduction in anxiety and depression symptoms; some research even suggests that these improvements may remain for up to two years.[1]

Lifestyle Modifications (Exercise, Diet, Sleep): Regular physical activity, balanced nutrition, and adequate sleep have been shown to improve symptoms.

Exercise a. Contribution to Anxiety and Depression

Frequent resistance and aerobic exercise is linked to notable decreases in feelings of anxiety and sadness. Exercise elevates mood by lowering inflammation, raising endorphins, and increasing brain-derived neurotrophic factor (BDNF).

b. Proof Exercise had moderate to large impacts (SMD = 0.66), according to a meta-analysis of 49 trials.

Resistance and aerobic training are both beneficial; supervised programs produce superior results.[42]

Nutrition & Diet

a. Contribution to anxiety and depression

A Mediterranean diet rich in fruits, vegetables, nuts, seafood, and whole grains is linked to a decreased risk of depression. Vitamins, zinc, magnesium, and omega-3 fatty acids are among the nutrients that are involved in the control of neurotransmitters.

b. Proof

The SMILES experiment showed that, in comparison to a control group, dietary improvements decreased depression symptoms in patients with major depressive disorder.[25]

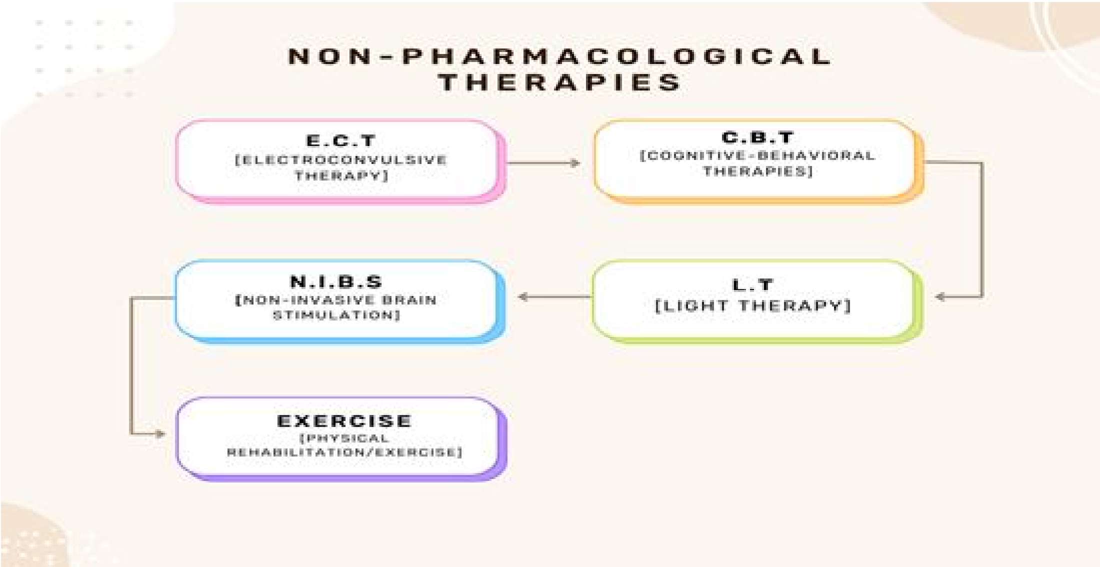

Figure 2. Representing Non – Pharmacological Therapies to neuropsychiatric Treatments.

Neuromodulation Techniques :

Electroconvulsive Therapy (ECT): Effective for severe or treatment-resistant depression, involving electrical stimulation of the brain under anaesthesia. One well-known somatic treatment is electroconvulsive therapy (ECT), which involves passing regulated electrical currents through the brain to cause brief seizures.

mainly used for bipolar depression, severe depression, treatment-resistant depression (TRD), and some severe anxiety disorders. Action Mechanism

ECT is thought to result in: regulation of neurotransmitters (*e.g.*, noradrenaline, serotonin, and dopamine) elevated expression of BDNF (Brain-Derived Neurotrophic Factor) and neuroplasticity, Hypoactivity in the limbic and prefrontal cortex, which are important in mood regulation, returns to normal.[40] Transcranial Magnetic Stimulation (TMS): A non-invasive method using magnetic fields to stimulate nerve cells in the brain. A non-invasive neuromodulation method called transcranial magnetic stimulation (TMS) stimulates brain nerve cells using magnetic fields. usually focuses on the dorsolateral prefrontal cortex (DLPFC), a part of the brain that controls mood. Action Mechanism TMS increases or decreases neural activity in specific brain regions by modifying cortical excitability and synaptic plasticity. Whereas low-frequency TMS ($\leq$1 Hz) is inhibitory, high-frequency TMS ($\geq$5 Hz) is excitatory.[34] Vagus Nerve Stimulation (VNS): Involves electrical stimulation of the vagus nerve and is considered for treatment-resistant cases. The FDA-approved neuromodulation treatment known as Vagus Nerve Stimulation (VNS) involves electrically stimulating the vagus nerve, also known as cranial nerve X. VNS was first authorized to treat epilepsy, but it has also demonstrated effectiveness in treating treatment-resistant depression (TRD).

Action Mechanism
Monoaminergic neurotransmission (serotonin, norepinephrine) is modulated by the VNS. reduces the stress response by influencing the hypothalamic-pituitary-adrenal (HPA) axis, increases cerebral blood flow and neuroplasticity in brain regions linked to mood, including the prefrontal cortex and amygdala.[20]

Deep Brain Stimulation (DBS): An invasive procedure targeting specific brain regions, currently under investigation. An invasive neurosurgery technique called deep brain stimulation (DBS) modifies aberrant neural activity by implanting electrodes in particular brain areas.

DBS was first used to treat movement disorders, such Parkinson's disease, but it has also showed promise in treating psychiatric diseases that are resistant to treatment, like major depression and obsessive-compulsive disorder (OCD). Action Mechanism Targeted activation of brain regions such as the nucleus accumbens or the subgenual cingulate gyrus (Brodmann area 25) modulates: circuits that are dysfunctional in regulating mood, The loop of limbic, cortical, striatal, pallidal, and thalamic Neurotransmission (dopamine, serotonin) and neuroplasticity.[35]

Emerging Brain Stimulation Methods: Includes techniques like transcranial direct current stimulation (tDCS), showing promise in early studies.

Direct Current Stimulation in the Brain (tDCS) a non-invasive method of regulating cortical excitability by applying mild electrical currents to the scalp.

Cathodal stimulation reduces excitability, while anodal stimulation increases it. shown to target the dorsolateral prefrontal cortex (DLPFC) in order to alleviate depression symptoms.[4]

TBS, or theta burst stimulation
a brand-new repeating TMS procedure that delivers brief high-frequency stimulation bursts. The FDA has approved intermittent TBS (iTBS) for major depressive disorder (MDD). Similar effectiveness to traditional TMS, but with shorter session lengths.[3]

Without the need for surgery, Low-Intensity Focused Ultrasound (LIFU) modifies neuronal activity in deep brain regions using ultrasonic waves.

Although there are currently few human trials, preclinical research points to potential for mood and anxiety control.[15]

Using oscillating electrical currents, transcranial alternating current stimulation (tACS) synchronizes brain oscillations at particular frequencies (gamma, theta, and alpha). emerging as a possible treatment for anxiety and depression that can improve mood and cognitive function.[21]

Preclinical Optogenetics and Magneto genetics
Using light to manipulate genetically altered neurons, optogenetics holds promise for breaking down mood circuitry. Utilizing magnetic fields to regulate cell activity, magneto

genetics is being investigated for deep brain modulation in mood disorders.[47]

Novel and Emerging Therapeutic Approaches:

Ketamine and Esketamine:

Rapid-acting antidepressants used in treatment-resistant depression. An NMDA receptor antagonist called ketamine has demonstrated quick antidepressant and anxiolytic effects, particularly in major depressive disorder (MDD) and treatment-resistant depression (TRD).

The FDA authorized esketamine, the S-enantiomer of ketamine, as a nasal spray for TRD in 2019.[11]

Action Mechanism
inhibits the glutamate receptor known as the NMDA receptor, which results in increased transmission of glutamate, increased neuroplasticity and synaptogenesis in the hippocampus and prefrontal cortex,
mTOR signalling pathway modification, Elevation of mood in a matter of hours.

Ketamine's Clinical Effectiveness Within 1 to 24 hours, intravenous ketamine (0.5 mg/kg) rapidly reduces depression symptoms. beneficial for sudden suicidal thoughts. Limitations: Possible dissociation or psychotomimetic effects; brief duration (7–10 days).[9]

Esketamine's FDA-approved intranasal formulation for TRD exhibits clinical efficacy. Has long-lasting antidepressant benefits when taken frequently.
adverse effects that are less psychotomimetic than those of racemic ketamine.[12]

4.5. Application to Anxiety Conditions
In early trials, ketamine showed encouraging benefits in treating GAD, PTSD, and social anxiety disorder (SAD). More controlled experiments are required.[43]

Psychedelic-Assisted Therapies :

Substances like psilocybin are being studied for their potential in treating depression and anxiety. Under the supervision of psychotherapy, psychedelic compounds including psilocybin, LSD, MDMA, and ayahuasca are used in supervised therapeutic settings as part of psychedelic-assisted therapy (PAT). There is mounting evidence that these drugs help relieve PTSD, anxiety, and depression quickly and effectively, particularly when conventional therapies are ineffective.

Therapy Assisted by Psilocybin The primary ingredient in magic mushrooms, psilocybin, is a serotonin 5-HT2A receptor agonist. Clinical trials show that depressed symptoms, such as treatment-resistant depression (TRD) and end-of-life worry, are significantly, quickly, and sustainably reduced in cancer patients.[5] Psychotherapy Assisted by MDMA MDMA (3,4-methylenedioxymethamphetamine) decreases fear reactions and improves empathy and emotional processing. demonstrated in controlled trials to be beneficial for anxiety-related disorders and post-traumatic stress disorder (PTSD).[37]

Anti-inflammatory and Neuroprotective Agents:

Targeting neuroinflammation and oxidative stress pathways. NSAIDs, or non-steroidal anti-inflammatory drugs Celecoxib,

a COX-2 inhibitor, is one NSAID that has been investigated as an adjuvant treatment for serious depression. demonstrated to lower inflammatory cytokines that are increased in depression, such as TNF-α and IL-6.[30]
PUFAs, or omega-3 polyunsaturated fatty acids.

By regulating cytokine production and neuronal membrane fluidity, omega-3 polyunsaturated fatty acids (EPA and DHA) have anti-inflammatory and neuroprotective properties. Improvements in major depressive disorder (MDD) and anxiety symptoms have been reported in clinical trials.[38]

Curcumin

Turmeric (*Curcuma longa*) contains curcumin, which has potent anti-inflammatory, antioxidant, and neuroprotective qualities. lowers oxidative stress and alters monoaminergic pathways and BDNF expression.
shown to have effects similar to those of antidepressants in people.

Gut-Brain Axis and Microbiota-Targeted Interventions:

Exploring the role of gut microbiota in mental health. The system of two-way communication between the central nervous system (CNS) and the gastrointestinal (GI) tract is known as the gut-brain axis. The generation of neurotransmitters, immunological responses, and inflammatory responses are all influenced by gut microbiota and are linked to mental health conditions like anxiety and depression.

Gut Microbiota's Function in Mental Health

Neuroactive substances that impact mood and cognition, such as GABA and serotonin precursors, are produced by the gut bacteria. Microbial imbalance, or dysbiosis, has been connected to altered neurotransmission in anxiety and depression, elevated inflammation, and a stress response.[16]

"Psychobiotics" or probiotics

In both people and animals, some probiotic strains (Bifidobacterium, Lactobacillus) have demonstrated anxiolytic and antidepressant-like properties. They affect neurotransmitter metabolism, lower systemic inflammation, and modify the HPA axis.[48]

Prebiotics

Prebiotics, such as fructooligo saccharides and galactooligo saccharides, promote the development of good gut flora.[41] Certain prebiotics have been shown to enhance emotional processing and lower cortisol levels.

Digital and Mobile Health Interventions:

Utilizing technology for therapy delivery and symptom monitoring. Applications for mobile devices (Apps) Numerous applications provide emergency help, mood monitoring, guided meditation, and CBT-based therapies. Their efficacy for mild to severe anxiety and depression is supported by some data.[13]

Cognitive behavioral therapy conducted online (iCBT) Through websites or apps, iCBT offers structured therapy modules. Effective for moderate to severe anxiety and depression, with outcomes on par with in-person therapy.[2]
Interventions Using Virtual Reality (VR) Technology VR treatment provides social skill

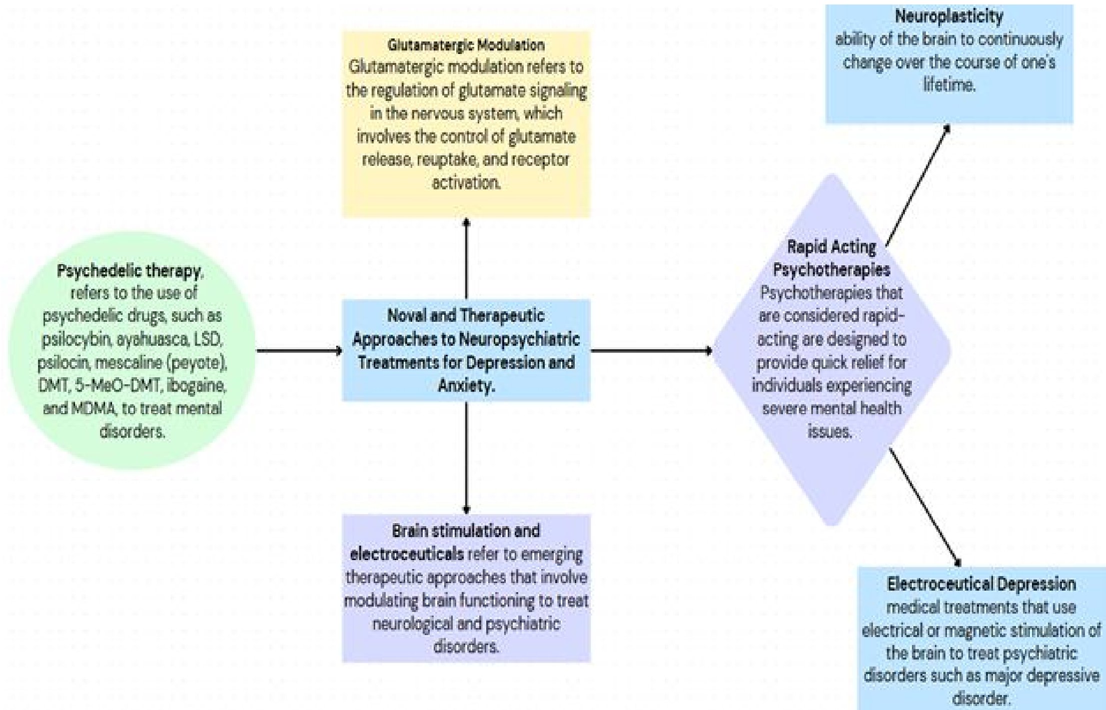

Figure – 3. Noval and Therapeutic Approaches to Neuropsychiatric Treatments for Depression and Anxiety.

development, relaxation training, and exposure therapy in immersive settings. VR therapies have demonstrated potential in the treatment of social anxiety disorders, PTSD, and phobias.[17] Telepsychology as well as Telepsychiatry Psychiatric consultations and therapy are provided through video calls in telepsychiatry. found to be just as successful as face-to-face therapy for sadness and anxiety, especially in isolated and underprivileged locations.[24]

Devices Wearable and Biofeedback

Heart rate monitors, fitness trackers, and smartwatches are examples of devices that give real-time biofeedback and measure physiological stress signs, such as heart rate variability and sleep patterns.
proven to enhance mood management and lessen stress and anxiety.[36]
Through video conversations, telepsychiatry and video counselling provide remote access to qualified specialists.
shown to be just as successful for a variety of mental health issues as in-person sessions.

Anxiety and depression are complex neuropsychiatric conditions that have a major influence on mental health worldwide. This study emphasizes the wide range of therapeutic strategies, from cutting-edge neuromodulation methods and new digital health solutions to traditional pharmaceutical therapies. The mainstays of treatment continue to be traditional antidepressants, anxiolytics, and psychotherapy techniques like cognitive behavioral therapy and mindfulness-based therapies. The investigation of new approaches, such as brain stimulation therapies (such as TMS, ECT, VNS, and DBS), ketamine and psychedelic-assisted therapies, and microbiota-targeted interventions through the gut-brain axis, is necessary due to treatment resistance and side effects.[28]

Furthermore, because they provide scalable, easily available, and customized interventions, digital and mobile health technologies are quickly changing the face of mental health care. Holistic management also heavily relies on lifestyle changes like exercise, food adjustments, and sleep optimization.[14] Notwithstanding these developments, issues with long-term effectiveness, side effect reduction, and therapy customization still exist. Incorporating multimodal therapy, finding biomarkers for personalized medicine, and thoroughly assessing novel interventions like digital medicines and gut microbiome manipulation should be the main goals of future research. In conclusion, the most promising route to all-encompassing, patient-centred treatment for anxiety and depression is a mix of pharmaceutical, psychotherapy, neuromodulator, and lifestyle approaches.[16,31]

References :

1. Abbass A., J. Town, and E. Driessen (2014). *World Psychiatry. 13*(3): 312–323.
2. Andrews G., A. Basu, P. Cuijpers, M. G. Craske, P. McEvoy, and C.L. English, *et al.* (2018). *PLoS One. 13*(1): e0189659.
3. Blumberger D.M., F. Vila-Rodriguez, K.E. Thorpe, K. Feffer, Y. Noda, and P. Giacobbe, *et al*. (2018). *Lancet. 391*(10131): 1683–1692.
4. Brunoni A.R., L. Valiengo, A. Baccaro, T.A. Zanao, J.F. de Oliveira, and A. Goulart, *et al*. (2013). *JAMA Psychiatry. 70*(4): 383–391.
5. Carhart-Harris R.L., B. Giribaldi, R. Watts, M. Baker-Jones, A. Murphy-Beiner, and R. Murphy, *et al*. (2021). *N Engl J Med. 384*(15): 1402–1411.
6. Cuijpers P., A.S. Geraedts, P. van Oppen, G. Andersson, J.C. Markowitz, and A. van Straten (2011). *Am J Psychiatry. 168*(6): 581–592.
7. Cuijpers P., S. Quero, H. Noma, M. Ciharova, C. Miguel, and E. Karyotaki, *et al*. (2021). *World Psychiatry. 20*(2): 283–293.
8. Cuijpers P., M. Sijbrandij, S.L. Koole, G. Andersson, A.T. Beekman, and C.F. Reynolds (2013). *World Psychiatry. 12*(2): 137–148.
9. Daly E.J., M.H. Trivedi, A. Janik, H. Li, Zhang Y., and X. Li, *et al*. (2019). *JAMA Psychiatry. 76*(7): 676–685.
10. Driessen E., H.L. Van, F.J. Don, J. Peen, R.A. Schoevers, and P. Cuijpers, *et al*. (2015). *Clin Psychol Rev. 42:* 1–15.
11. Duman R.S., G.K. Aghajanian, G. Sanacora, and J.H. Krystal (2019). *Neuron. 102*(1): 75–93.
12. Feder A., M.K. Parides, J.W. Murrough, A.M. Perez, J.E. Morgan, and S. Saxena, *et al*. (2014). *JAMA Psychiatry. 71*(6): 681–688.
13. Firth J., J. Torous, J. Nicholas, R. Carney, S. Rosenbaum, and J. Sarris (2017). *World Psychiatry. 16*(3): 287–298.
14. Firth J., J. Torous, B. Stubbs, J. A. Firth, R. Marx, and R. Carney, *et al.* (2019).

World Psychiatry. 18(2): 119–129.
15. Folloni D., L. Verhagen, R. B. Mars, E. Fouragnan, C. Constans, and J.F. Aubry, *et al*. (2019). *Neuron. 101*(6): 1109–1115.
16. Foster J.A., and K.A. Neufeld (2013). *Trends Neurosci. 36*(5): 305–313.
17. Freeman D., S. Reeve, A. Robinson, A. Ehlers, D. Clark, and B. Spanlang, *et al*. (2017). *Psychol Med. 47*(14): 2393–2400.
18. Ghielen I., S. Rutten, R. E. Boeschoten, M. Houniet-de Gier, E.E. van Wegen, and Heuvel O.A. van den, *et al*. (2019). *J Psychosom Res. 122:* 43–51.
19. Goldberg S.B., R.P. Tucker, P.A. Greene, R.J. Davidson, B. E. Wampold, and D.J. Kearney, *et al*. (2018). *Clin Psychol Rev. 59:* 52–60.
20. Groves D.A., and V.J. Brown (2005). *Neurosci Biobehav Rev. 29*(3): 493–500.
21. Herrmann C.S., S. Rach, T. Neuling, and D. Strüber (2013). *Front Hum Neurosci. 7:* 279.
22. Hirschfeld R.M., J.B. Williams, R.L. Spitzer, J.R. Calabrese, L. Flynn, and P.E. Keck Jr., *et al*. (2000). *Am J Psychiatry. 157*(11): 1873–1875.
23. Hoge E.A., E. Bui, L. Marques, C.A. Metcalf, L.K. Morris, and D.J. Robinaugh, *et al*. (2022). *JAMA Psychiatry. 79*(7): 576–584.
24. Hubley S., S.B. Lynch, C. Schneck, M. Thomas, and J. Shore (2016). *World J Psychiatry. 6*(2): 269–282.
25. Jacka F.N., A. O'Neil, R. Opie, C. Itsiopoulos, S. Cotton, and M. Mohebbi, *et al*. (2017). *BMC Med. 15*(1): 23.
26. Kessler R.C., P. Berglund, O. Demler, R. Jin, D. Koretz, and K.R. Merikangas, *et al*. (2003). *JAMA. 289*(23): 3095–3105.
27. Kessler R.C., and E.J. Bromet (2013). *Annu Rev Public Health. 34:* 119–138.
28. Khan A., and W.A. Brown (2015). *World Psychiatry. 14*(3): 294–300.
29. Khoury B., T. Lecomte, G. Fortin, M. Masse, P. Therien, and V. Bouchard, *et al*. (2015). *J Psychosom Res. 78*(6): 519–528.
30. Köhler O., M.E. Benros, M. Nordentoft, M.E. Farkouh, R.L. Iyengar, and O. Mors, *et al*. (2014). *JAMA Psychiatry. 71*(12): 1381–1391.
31. Krystal J.H., C.G. Abdallah, G. Sanacora, D.S. Charney, and R. S. Duman (2019). *Neuron. 101*(5): 774–788.
32. Kuyken W., R. Hayes, B. Barrett, R. Byng, T. Dalgleish, and D. Kessler, *et al*. (2016). *JAMA Psychiatry. 73*(6): 565–574.
33. Lam R.W., D. McIntosh, J. Wang, M.W. Enns, T. Kolivakis, and E. E. Michalak, *et al*. (2016). *Can J Psychiatry. 61*(9): 510–523.
34. Lefaucheur J.P., A. Aleman, C. Baeken, D.H. Benninger, J. Brunelin, and V. Di Lazzaro, *et al*. (2020). *Clin Neurophysiol. 131*(2): 474–528.
35. Lozano A.M., H.S. Mayberg, P. Giacobbe, C. Hamani, R.C. Craddock, and S.H. Kennedy (2008). *Biol Psychiatry. 64*(6): 461–467.
36. Mehler D.M.A., M.O. Sokunbi, I. Habes, K. Barawi, L. Subramanian, and M. Range, *et al*. (2020). *Psychol Med. 50*(10): 1530–1537.
37. Mitchell J.M., M.P. Bogenschutz, A. Lilienstein, C. Harrison, S. Kleiman, and K. Parker-Guilbert, *et al*. (2021). *Nat Med. 27*(6): 1025–1033.
38. Mocking R.J.T., I. Harmsen, J. Assies, M.W.J. Koeter, H.G. Ruhé, and A.H. Schene (2016). *Transl Psychiatry. 6*(3):

e756.
39. Ng Q.X., S.S. Koh, H.W. Chan, and C.Y. Ho (2017). *J Am Med Dir Assoc. 18*(6): 503–508.
40. Sackeim H.A. (2017). *JAMA Psychiatry. 74*(8): 779–780.
41. Schmidt K., P.J. Cowen, C.J. Harmer, G. Tzortzis, S. Errington, P.W. Burnet (2015). *Psychopharmacology (Berl). 232*(10): 1793–1801.
42. Schuch F.B., D. Vancampfort, J. Richards, S. Rosenbaum, P.B. Ward, and B. Stubbs (2016). *Am J Psychiatry. 175*(7): 631–648.
43. Short B., J. Fong, V. Galvez, W. Shelker, C.K. Loo (2018). *Lancet Psychiatry. 5*(1): 65–78.
44. Siniscalchi K.A., M.E. Broome, J. Fish, J. Ventimiglia, J. Thompson, and P. Roy, *et al.* (2020). *J Prim Care Community Health. 11:* 2150132720931261.
45. Stangier U., E. Schramm, T. Heidenreich, M. Berger, and D.M. Clark (2011). *Am J Psychiatry. 168*(7): 692–700.
46. Tang Y.Y., B.K. Hölzel, and M.I. Posner (2015). *Nat Rev Neurosci. 16*(4): 213–225.
47. Tye K.M., and K. Deisseroth (2013). *Nat Rev Neurosci. 14*(8): 488–502.
48. Wallace C.J.K., R. Milev (2017). *Ann Gen Psychiatry. 16*(1): 14.
49. Weintraub D. (2020). *Neurotherapeutics. 17*(4): 1511–1524.
50. Zhuo C., R. Xue, L. Luo, F. Ji, H. Tian, and H. Qu, *et al*. (2017). *Medicine (Baltimore)*. 96(22):e6698.