Global Journal of Intellectual & Developmental Disabilities
ISSN: 2575-8586

Review Article
Volume 12 Issue 5 - November 2023
DOI: 10.19080/GJIDD.2023.12.555848

Glob J Intellect Dev Disabil

Neurobiologically based Treatments for Depression and its subtypes: A Review

Ghazal Farjami*

University of New England, Australia

Submission: October 26, 2023; **Published:** November 10, 2023

***Corresponding author:** Ghazal Farjami, University of New England, Australia, Email: gfarjami@myune.edu.au

Abstract

Major Depressive Disorder (MDD) places a heavy and growing burden on both the person and the community. The origin, extent, and recurrence of MDD may be influenced by a mix of hereditary factors, chronic unpleasant circumstances, and neurotransmitter imbalances; however, its specific aetiology is still unknown [1]. Therefore, it is essential to take into account all of these aspects and their effects when designing a therapy strategy for MDD. This review will.. examine the efficacy of four main treatment options for MDD: medication, talk therapy, neurological stimulation, and physical activity. Further, it explores the effectiveness of these interventions on different subtypes of MDD, particularly somatic depression, anhedonia, and melancholic depression.

Keywords: Depression; Depressive disorder; Neurotransmitters; Mental disorders; Antidepressant therapy; Psychotherapy

Introduction

Major Depressive disorder (MDD) patients are frequently treated with both psychotherapy and medication [2]. There are theories that provide insight into these treatments' probable workings, despite the fact that the precise mechanisms are not entirely understood. For instance, the monoamine theory, which is the foundation for many antidepressant drugs, postulates that MDD may result, if not entirely in some way, from imbalances in specific monoamine neurotransmitters [1]. These drugs are thought to lessen depressive symptoms by altering the brain's levels of particular neurotransmitters [3]. Psychotherapy has various forms, each of which is employed by a trained clinician to assist individuals in gaining a better understanding of their thoughts, emotions, and behaviours, as well as to develop skills and coping strategies to manage their condition [4]. Neurological stimulations directly target specific regions in the brain to modulate neural circuits and stimulate the release of neurotransmitters associated with depression [1]. Furthermore, Physical activity is increasingly recognized as a therapeutic strategy for depression due to its impact on increasing neural plasticity, promoting neural growth, and increasing the release of various neurotransmitters while also reducing inflammation [5]. All these treatment plans had a nonresponse rate of between 30% and 50% and onset delay of a few weeks [6].

According to the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition [DSM-5](DSM-5; diagnosing major depressive disorder (MDD)) [7], anhedonia is characterised as a significant reduction in interest or pleasure in nearly all activities throughout the day, reflecting an impairment in the brain's ability to process and experience rewards. Melancholic depression is marked by severe symptoms and treatment difficulties, among other subtypes of depression [1]. In addition to the common symptoms of MDD, individuals with melancholic depression have a higher incidence of experiencing persistent fatigue, anhedonia, slowed cognitive processes, disrupted sleep, appetite changes, feeling guilt, hopelessness, and suicidal thoughts. People who suffer from somatic depression are identified by excessive thoughts and feelings about physical health, either with or without pain.

Since the emergence of the first antidepressant in the 1950s, drug therapy has become one of the primary treatments for MDD [1]. According to Stafford et al.'s [8], study of antidepressant

prescribing patterns, there was an increase in the percentage of people receiving medication treatment during this time, going from 70% in 1987 to 89% in2001. In a more recent study conducted by Luo et al. [9], which analysed prescription patterns of antidepressants for adults in the United States over a 20-year period from 1996 to 2015, an overall decrease in the proportion of MDD patients receiving antidepressant therapy was observed.

Initially, tricyclic antidepressants (TCAs) such as Amitriptyline and Imipramine were the first class of widely prescribed antidepressants, but, over time, the use of selective serotonin reuptake inhibitors (SSRIs) like fluoxetine and citalopram became more prevalent due to their favourable tolerability and low incidence of adverse effects [10]. Throughout the years, novel drugs have been developed and widely used in the treatment of MDD. Some of them, as mentioned by Harmer et al. [10], include monoamine oxidase inhibitors (MAOIs), norepinephrine-dopamine reuptake inhibitors (NDRIs), serotonin- norepinephrine reuptake inhibitors (SNRIs), and noradrenergic and specific serotonergic antidepressants (NaSSAs).

The therapeutic effects of antidepressants are not immediate, and they often take several weeks to manifest observable clinical improvements. According to Cipriani et al. [11], these delays result from the time required for the neurotransmitter levels to become balanced and create adaptive changes in the brain regions impacted by depression. This explanation lends support to the monoamine hypothesis, which contends that low levels of serotonin, norepinephrine, and/or dopamine in the brain are the root cause of depression [10]. While the monoamine theory for antidepressants is widely accepted, some studies have shown data to the contrary. For instance, in a study conducted in 2000, Delgado [12], utilised the monoamine depletion paradigm to examine how lowering monoamine levels would affect depressive behaviours. The results from this study demonstrated that antidepressant benefits can be temporarily reversed by declining the level of monoamine, with the degree of reversal changing according to the class of antidepressant used. Notably, this study demonstrated that monoamine depletion neither worsened the depressive behaviour in individuals with MDD nor caused depressive behaviour in non-depressed individuals. These findings challenged the widespread notion that the primary mechanism by which antidepressants alleviate depression symptoms is through raising the levels of monoamine neurotransmitters.

The effectiveness of antidepressant medicines in the treatment of various subtypes of MDD has been examined in several trials. Kirsch and colleagues evaluated data from various clinical studies in a systematic review and meta-analysis in 2008 [13]. According to the findings of this study, for individuals with mild to moderate depression, the therapeutic response difference between antidepressants and a placebo was not clinically significant. This shows that people with moderate MDD may have limited success using antidepressants to treat their depressive symptoms and that others with severe MDD may benefit more from these medications. Later in 2009, Bauer and his team conducted a comparative meta-analysis to explore the efficacy of several antidepressant classes, including placebo, in patients with treatment-resistant depression [14]. The results of their study suggest that, compared to SSRIs, tricyclic antidepressants, and placebos, therapy with an SNRI antidepressant has a better effect, is easier to take, and has a much lower rate of relapse.

Moreover, recent studies [15,16], compared the effectiveness and safety of newer antidepressants like ketamine and esketamine to thoseof traditional monoamines. These studies showed that the immediate therapeutic rate of newer antidepressants is higher. However, it is yet unclear if these medications will be effective and safe over the long term, particularly for severe and treatment-resistant depression [2]. Hasselmann et al. [16], highlighted the higher incidence of suicidal behaviours among people who stopped using ketamine and emphasised the significance of post-discontinuation monitoring for those who use these new medications.

Psychotherapy

Talk therapy, commonly referred to as psychotherapy, encompasses various techniques, including interpersonal therapy, cognitive-behavioural therapy (CBT), and psychoanalysis [1]. Among these methods, CBT has been reported to have the highest therapeutic success rate in treating different types of affective disorders, including MDD [1]. However, it is important to note that there is no one-size-fits-all approach when it comes to psychotherapy. In 2012, Hofmann and colleagues [17], investigated the effectiveness of different psychotherapy approaches in the treatment of depressive disorders and suggested that the choice of psychotherapy should be based on each individual's specific case and situation. Similarly, Ahn and Wampold's meta-analysis from 2001 found no significant advantage to a particular psychological therapy. According to Ahn and Wampold et al. [18], treatment selection should be tailored to each patient's specific condition rather than just the type and severity of depressive symptoms.

Moreover, several studies have highlighted the role of the therapist in the psychotropic approach. For instance, Ahn and Wampold [18], demonstrated that using the same treatment technique by different therapists could yield vastly different treatment outcomes. In another study, Allen et al. [4], emphasised the importance of therapeutic alliance (TA) and highlighted that the outcome of psychotherapy is largely influenced by the therapist's ability to establish a strong bond with the patient rather than the specific therapy approach used. The therapeutic alliance is characterised by elements such as trust, emotional closeness,

How to cite this article: Alison Ghazal F. Efficacy Review of Neurobiologically-based Treatments for Depression and its Sub-types. Glob J Intellect Dev Disabil. 2023; 12(5): 555848. DOI: 10.19080/GJIDD.2023.12.555848

shared understanding of therapeutic goals and processes, and verbal and nonverbal behaviours, including eye contact, facial expressions, and even pauses by the therapist that signal care and support [4].

As stated in Sharpley [1], different research has compared the effectiveness of psychotherapy with medication and indicates that both treatments have shown positive results. Some studies, such as Kocsis et al. [19], suggest that when these two treatments are combined, the response is improved, especially for preventing relapse. In terms of deciding on the type of psychotherapy treatment, Sharpley and Bitsika [20], propose four criteria: patient demographics, the presence of melancholia, comorbidity with other psychiatric diagnoses, and genetic predisposition to depression. Studies on demographic factors show that patients with lower levels of social dysfunction respond better to psychotherapy, while those with impaired cognitive processing and more severe depression respond less to psychotherapy and may respond more to antidepressants [1]. Fournier et al. [21] have shown that married, unemployed individuals who have gone through adverse life events respond better to CBT compared to medication. Those with lower cognitive skills receive superior outcomes from medication therapy. As cited in Sharpley [1], the second factor contributing to the efficacy of psychotherapy is the presence of melancholic or non-melancholic depression. Melancholic depression may benefit from antidepressants [22], while non-melancholic depression works well with talk therapy. Comorbidity with other disorders, particularly personality disorders, is another factor affecting the results of psychotherapy. As cited in Sharpley [1], some studies reported a poorer response among depressed patients with personality disorders to talk therapy compared to those without personality problems. However, some studies challenge this argument and do not see an association between personality disorders and the rate of responsiveness to psychotherapy [1].

Neurological Stimulation

The poor adherence and limited efficacy of medication have sparked interest in alternative modalities for treating depression. The neuromodulation approach encompasses both invasive methods like deep brain stimulation (DBS) and vagal nerve stimulation (VNS) as well as non-invasive methods like electroconvulsive therapy (ECT), transcranial magnetic stimulation (TMS), trans-cranial direct current stimulation (tDCS), and real-time functional magnetic resonance imaging neurofeedback (rtfMRI-nf) [23].

The oldest and most established non-invasive neuromodulation modality is ECT [1]. During ECT, while the patient is under general anaesthesia, a controlled electric current is administered to the brain through the scalp to induce a seizure [23]. The exact mechanism of action for ECT is not clear yet, but some theories suggest that it works by regulating brain activity in the prefrontal cortex (PFC) and promoting neurogenesis in the hippocampus [24]. While ECT has been proven to be both effective and safe, it has some limitations, such as the need for professional equipment and the possible risks of major neurological complications in a smallpercentage of patients [24]. Demographic characteristics like theduration and severity of depression play a major role in the level of effectiveness of ECT, As emphasised by Holtzheimer & Mayberg [24].

Transcranial magnetic stimulation (TMS) is another safe and non-invasive brain stimulation method that has been shown to have a good outcome in treating MDD and TRD.In this technique, an electromagnetic coil will be placed on the patient's scalp, particularly over a part of the brain that needs to be targeted. This magnetic coil generates repetitive pulses that stimulate the brain cells and modulate the electrical activity of the brain [23]. According to research by Rotheneichner et al. [25], TMS improves hippocampus neurogenesis while lowering corticosteroid levels in the brain. In a 2012 study, Puigdemont et al. examined the effects of TMS on the cingulate gyrus of eight patients with TRD and found that depressive symptoms were eliminated without any noticeable adverse reactions. According to Brunoni et al. [23], conditions like previous treatment resistance, the length of the present depressive incident, and the lack of co-occuring anxiety symptoms could limit the beneficial effects of TMS therapy.

Like TMS, tDCS is a non-invasive and safe neuromodulation procedure that targets the brain's electrical activity. However, unlike TMS, which uses magnetic fields and electromagnetic pulses to stimulate neural activity, DCS works by sending small repetitive impulses that modulate the excitability of the cerebral cortex [26]. The mechanisms underpinning tDCS's antidepressant impact remain unclear; however, some research has indicated that tDCS therapy is linked to long-term brain plasticity [26]. Overall, contradictory findings from studies on the effectiveness of tDCS in treating MDD have been found [26].

An additional technique to alleviate the symptoms of depression is known as real- time functional magnetic resonance imaging neurofeedback (rtfMRI-NF). In fMRI-NF, patients in the magnetic scanner receive immediate feedback on blood oxygenation level-dependent activity (BOLD), which is connected to the vascular reaction to cerebral activity. Patients learn to voluntarily regulate this activity. Such training focuses on certain areas of the brain that are involved in cognitive and emotional functions [27]. Although research on the effectiveness of the fMRINF method as a therapeutic approach is still in its early stages, it shows promise in reducing the symptoms of depression by regulating the amygdala's activity and impacting limbic-thalamic-cortical pathways that are linked to depressive symptoms [28].

Deep Brain Stimulation (DBS) is a surgical procedure to implant a pulse generator device in the brain that activates or

How to cite this article: Alison Ghazal F. Efficacy Review of Neurobiologically-based Treatments for Depression and its Sub-types. Glob J Intellect Dev Disabil. 2023; 12(5): 555848. DOI: 10.19080/GJIDD.2023.12.555848

inhibits dysregulated neural activities. Brunoni et al. [23]. DBS has been demonstrated to improve depressive symptoms in patients diagnosed with treatment-resistant depression (TRD) by targeting the subgenual cingulate cortex (SCC),ventral capsule and striatum, and nucleus accumbens (NAc) [23]. The effectiveness of Deep Brain Stimulation (DBS) in addressing the subgenual cingulate cortex (SCC) was examined in a study carried out by Brown and associates in 2020. At the two-year milestone after the DBS treatment, the results showed an excellent 92% response rate among people with TRD. This suggests that after receiving DBS that targeted the SCC area ofthe brain, a sizable majority of patients experienced improvements in their feelings.

Vagus Nerve Stimulation (VNS) is a more recent FDA-approved therapeutic neuromodulation procedure that shows promise for individuals with severe affective disorders, including chronic and recurrent depression. In VNS, a pulse generator device is implanted beneath the skin of the chest. This device emits electrical impulses to an electrode threaded in the vagus nerve located in the patient's neck. The vagus nerve then transmits these electrical signals to brain areas involved in depressive symptoms, helping regulate impaired neural networks [23]. Compared to Electroconvulsive Therapy (ECT), which acts rapidly but may have significant cognitive side effects, VNS is generally well tolerated and lacks significant adverse effects. However, it is important to note that the therapeutic trajectory of VNS is longer and slower than that of ECT. Seeing that, it may not bethe optimal choice when rapid improvement is necessary.

Exercise

Extensive post-mortem and neuroimaging investigations have shown that people withMDD have significantly less hippocampus volume [29]. Research [29,30], shows that this volume change is caused by higher levels of glucocorticoids and lower levels of brain-derived neurotrophic factor (BDNF) in depressed individuals. As the result the rate of neurogenesis to slow down, which makes depressive symptoms appear. Based on this notion, Numerous studies have been conducted to investigate the potential impact of physical activity on the onset, treatment, and relapsepresentation of MDD.

Sleiman et al. [31], investigated the effects of physical activity on BDNF gene expression and hippocampal neurogenesis in mice. The researchers exposed mice to a running wheel for a duration of 4 weeks, which resulted in a significant increase in the concentration of BDNF in the hippocampus compared to the control group, suggesting that running promotes the induction of BDNF. Sleiman and the team (2016) highlighted the multifaceted role of BDNF in neuroplasticity, synaptic connections, and neural growth and suggested that individuals with higher levels of BDNF have a lower risk of developing anxietyand depression and may exhibit greater resistance to aversive environmental stimuli.

Xie and colleagues [32], conducted a systematic review of research on the antidepressant effects of exercise published between 2010 and 2021. The findings from thisreview indicated that moderate-to-high-intensity exercise three to five times a week significantly alleviates depressive symptoms and enhances the overall functioning of individuals with MDD. The same study showed that group aerobic exercise that involves focusing on mind-body coordination is the most effective type of physical activity for adhering to the treatment of depression.

Wang et al. [33], conducted a systematic review and meta-analysis to study exerciseinterventions among adolescents aged 12–18 with MDD. The study demonstrated that physical activity, particularly a combination of aerobic and resistance exercise, can have a positive impact on reducing depression among adolescents, including individuals with diagnosed depression and those with subsyndromal depression.

In addition to its rehabilitative effects, exercise has been shown to have a preventative impact on depressive symptoms. With the intention to investigate the preventative effect of exercise, Harvey et al. [34], conducted a longitudinal study following33,908 initially healthy subjects for 11 years, recording their physical and mental health conditions as well as their exercise patterns. The results of this study demonstrated that engaging in regular physical activity for at least one hour per week, regardless of intensity, was associated with a reduced risk of developing depressive disorders.

In elderly and physically vulnerable individuals, improving physical health has been shown to have a beneficial impact on their overall mental health. According to Heissel et al. [35] spending time in moderate-intensity physical activity, especially participation in a group exercise, stimulates the secretion of endorphins in the body, which act as natural painkillers and mood boosters. Fox et al. [5] argue that engaging in group exercise can foster a sense of belonging, promote self-efficacy and self-esteem, and alleviate feelings of loneliness, which are crucial factors in combating depressive symptoms and uplifting the mood. Engaging in group exercise activities can also elevate mood by providing social connection and support.

The impact of physical activity on mood and anhedonia, one of the core symptoms of MDD, was investigated in a study conducted by Sun et al. [36]. The findings fromthis study revealed the beneficial effect of physical activity on reducing anhedonia and elevating individuals' emotional well-being, concluding that more intense exercise leads togreater improvements in mood and motivation.

Therapeutic effects of medication, psychotherapy, brain stimulation, and exercise on anhedonia, melancholic depression, and somatic depression. Anhedonia is a common characteristic of depressive disorders. A combination of

How to cite this article: Alison Ghazal F. Efficacy Review of Neurobiologically-based Treatments for Depression and its Sub-types. Glob J Intellect Dev Disabil. 2023; 12(5): 555848. DOI: 10.19080/GJIDD.2023.12.555848

psychotherapy, medication, and physical activity may be effective in the treatment of anhedonia. Cao et al. [37], conducted a systematic review of 17 studies that used medication to treat anhedonia in MDD patients. The results of this review showed that mostof the examined antidepressants had favourable effects on measures of the anhedonia connected to MDD, indicating their potential effectiveness in treating this condition.

Furthermore, a study carried out by Sun and colleagues in 2022 showed that moderate- intensity group exercise has a significant positive impact on mood and promotes a sense of pleasure from the activities.

Multiple studies have explored the efficacy of antidepressants in treating melancholicdepression. For example, Thase et al. [38], conducted a randomised controlled trial comparing different antidepressants and found that both TCAs and SSRIs effectively reduced depressive symptoms in individuals with melancholia. These findings were further supported by a meta-analysis conducted by McIntyre et al. [39], which compared various classes of antidepressants, including SSRIs, TCAs, and SNRIs. The meta-analysis revealed that all types of antidepressant treatments were equally effective in addressing melancholic depression, with no significant differences observed between different medication classes.

These studies collectively provide compelling evidence supporting the usefulness of antidepressant therapy for melancholic depression. Brown W. A. [40,41], conducted a literature review comparing the effectiveness of antidepressants and psychotherapy in thetreatment of patients with melancholia and showed greater responsiveness to antidepressants compared to psychotherapy or placebo alone.

Miguel et al. [42], conducted a meta-analysis of 75 randomised trials involving 8209patients with somatic depression and showed that psychotherapy has a significant positive effect on reducing the severity of somatic depressive symptoms and increasing the quality oflife of individuals. The effectiveness of pharmacological interventions for somatoform disorders, characterised by medically unexplained physical symptoms (MUPS), was examined by Kleinstäuber et al. [43]. They assessed the efficacy of TCAs, NGAs, and combined treatments with SSRIs and antipsychotic medications [44-46]. The findings indicated low-quality evidence supporting the effectiveness of TCAs and NGAs in reducing the severity of MUPS. Additionally, the combination treatment of SSRIs and antipsychotics showed some positive effects.

Conclusion

In conclusion, medication, particularly antidepressants, has been a cornerstone in MDD treatment since the 1950s. Despite the initial dominance of tricyclic antidepressants, newer classes like SSRIs and other innovative drugs have emerged. However, the delayed onset of therapeutic effects and a nonresponse rate of 30-50% underscore the need for alternative or complementary interventions.

Talk therapy, encompassing various approaches such as CBT, interpersonal therapy, and psychoanalysis, offers a non-pharmacological avenue for MDD treatment. The effectiveness of psychotherapy, especially CBT, has been highlighted, with therapeutic alliance and individualized treatment plans playing crucial roles. Studies have indicated that combining medication and psychotherapy may yield improved outcomes, emphasizing the importance of tailoring treatment approaches to individual characteristics and conditions.

Neurological stimulation techniques, including electroconvulsive therapy (ECT), transcranial magnetic stimulation (TMS), trans-cranial direct current stimulation (tDCS), and deep brain stimulation (DBS), provide alternatives for treatment-resistant cases. These modalities target specific brain regions and neural circuits, showing promise in alleviating depressive symptoms, although their mechanisms of action are not fully understood. However, considerations regarding potential side effects, individual differences, and the need for professional equipment must be taken into account.

Physical activity has emerged as a recognized therapeutic strategy for MDD, influencing neural plasticity, neural growth, and neurotransmitter release while reducing inflammation. Exercise interventions, ranging from moderate-to-high-intensity workouts to group activities, have demonstrated positive effects in reducing depressive symptoms and preventing relapse. The impact of physical activity extends beyond symptom alleviation, addressing anhedonia, melancholic depression, and somatic depression, making it a holistic approach to MDD treatment.

Overall, the diverse nature of Major Depressive Disorder necessitates a comprehensive and individualized treatment approach. Integrating various therapeutic modalities and considering the specific characteristics of different MDD subtypes can enhance treatment efficacy and contribute to a more personalized and effective strategy for managing this pervasive mental health condition.

References

1. Sharpley C (2013) Understanding and treating depression: biological, psychological, and behavioural perspectives. Tilde University Press.
2. Karrouri R, Hammani Z, Benjelloun R, Otheman Y (2021) Major depressive disorder:Validated treatments and future challenges. World journal of clinical cases 9(31): 9350-9367.
3. Taylor C, Fricker AD, Devi LA, Gomes I (2005) Mechanisms of action of antidepressants: from neurotransmitter systems to signalling pathways. Cellular signalling 17(5): 549-557.
4. Allen ML, Cook BL, Carson N, Interian A, La Roche M, et al. (2017) Patient-Provider Therapeutic Alliance Contributes to Patient Activation in Community MentalHealth Clinics. Administration and policy in mental health 44(4): 431-440.

How to cite this article: Alison Ghazal F. Efficacy Review of Neurobiologically-based Treatments for Depression and its Sub-types. Glob J Intellect Dev Disabil. 2023; 12(5): 555848. DOI: 10.19080/GJIDD.2023.12.555848

5. Fox KR, Stathi A, McKenna J, Davis MG (2007) Physical activity and mental well- being in older people participating in the Better Ageing Project. European journal of applied physiology 100(5): 591-602.

6. Bschor T, Adli M (2008) Treatment of depressive disorders. Deutsches Arzteblattinternational 105(45): 782-792.

7. American Psychiatric Association D, American Psychiatric Association (2013) Diagnosticand statistical manual of mental disorders: DSM-5 (Vol. 5, No. 5) Washington, DC: American psychiatric association.

8. Stafford RS, MacDonald EA, Finkelstein SN (2001) National Patterns of MedicationTreatment for Depression, 1987 to 2001. Primary care companion to the Journal of clinical psychiatry 3(6): 232-235.

9. Luo Y, Kataoka Y, Ostinelli EG, Cipriani A, Furukawa TA (2020) National Prescription Patterns of Antidepressants in the Treatment of Adults with Major Depression in the US Between 1996 and 2015: A Population Representative SurveyBased Analysis. Frontiers in psychiatry 11: 35.

10. Harmer CJ, Duman RS, Cowen PJ (2017) How do antidepressants work? New perspectives for refining future treatment approaches. The lancet. Psychiatry 4(5): 409-418.

11. Cipriani A, Furukawa TA, Salanti G, Chaimani A, Atkinson LZ, et al. (2018) Comparative efficacy and acceptability of 21 antidepressant drugs for the acute treatment of adults with major depressive disorder: a systematic review and network meta- analysis. Lancet (London, England): 391(10128): 1357-1366.

12. Delgado PL (2000) Depression: the case for a monoamine deficiency. The Journal of clinicalpsychiatry 61 Suppl 6: 7-11.

13. Kirsch I, Deacon BJ, Huedo-Medina TB, Scoboria A, Moore TJ, et al. (2008) Initial severity and antidepressant benefits: a meta-analysis of data submitted to the Food and Drug Administration. PLoS medicine, 5(2): e45.

14. Bauer M, Tharmanathan P, Volz HP, Moeller HJ, Freemantle N (2009) The effect of venlafaxine compared with other antidepressants and placebo in the treatment of major depression: a meta-analysis. European archives of psychiatry and clinical neuroscience 259(3): 172-185.

15. Bahji A, Vazquez GH, Zarate CA Jr (2021) Comparative efficacy of racemic ketamineand esketamine for depression: A systematic review and meta-analysis. Journal of affective disorders 278: 542-555.

16. Hasselmann HW (2014) Ketamine as antidepressant? Current state and futureperspectives. Current neuropharmacology 12(1): 57-70.

17. Hofmann SG, Asnaani A, Vonk IJ, Sawyer AT, Fang A (2012) The Efficacy of Cognitive Behavioral Therapy: A Review of Meta-analyses. Cognitive therapy and research 36(5): 427-440.

18. Ahn H, Wampold BE (2001) Where oh where are the specific ingredients? A meta- analysis of component studies in counseling and psychotherapy. Journal of CounselingPsychology 48(3): 251-257.

19. Kocsis JH, Gelenberg AJ, Rothbaum BO, Klein DN, Trivedi MH, et al. (2009) Cognitive behavioral analysis system of psychotherapy and brief supportive psychotherapy for augmentation of antidepressant nonresponse in chronic depression: the REVAMP Trial. Archives of general psychiatry 66(11): 1178-1188.

20. Sharpley CF, Bitsika V, Christie DR (2011) Understanding the functionality of depression among Australian breast cancer patients: implications for cognitive and behavioural interventions. International journal of behavioural medicine 18(4): 319-324.

21. Fournier JC, DeRubeis RJ, Shelton RC, Hollon SD, Amsterdam JD, et al. (2009) Prediction of response to medication and cognitive therapy in the treatment of moderate to severe depression. Journal of consulting and clinical psychology 77(4):775-787.

22. Leventhal AM, Rehm LP (2005) The empirical status of melancholia: Implications forpsychology. Clinical Psychology Review 25(1): 25-44.

23. Brunoni AR, Teng CT, Correa C, Imamura M, Brasil-Neto JP, et al. (2010) Neuromodulation approaches for the treatment of major depression: challenges and recommendations from a working group meeting. Arquivos de neuro- psiquiatria 68(3): 433-451.

24. Holtzheimer PE, Mayberg HS (2012) Neuromodulation for treatment-resistantdepression. F1000 medicine reports 4: 22.

25. Rotheneichner P, Lange S, O'Sullivan A, Marschallinger J, Zaunmair P, et al. (2014) Hippocampal neurogenesis and antidepressive therapy: shocking relations. Neural plasticity 2014: 723915.

26. Ulrichsen KM, Kolskår KK, Richard G, Pedersen ML, Alnaes D, et al. (2022) No add-on effect of tDCS on fatigue and depression in chronic strokepatients: A randomized sham-controlled trial combining tDCS with computerizedcognitive training. Brain and behavior, 12(7): e2643.

27. Pilmeyer J, Huijbers W, Lamerichs R, Jansen JFA, Breeuwer M, et al. (2022) Functional MRI in major depressive disorder: A review of findings, limitations, andprospects. Journal of neuroimaging: official journal of the American Society of Neuroimaging, 32(4): 582-595.

28. Mennen AC, Turk-Browne NB, Wallace G, Seok D, Jaganjac A, et al. (2021) Cloud-BasedFunctional Magnetic Resonance Imaging Neurofeedback to Reduce the Negative Attentional Bias in Depression: A Proof-of-Concept Study. Biological psychiatry. Cognitive neuroscience and neuroimaging 6(4): 490-497.

29. Czéh B, Lucassen PJ (2007) What causes the hippocampal volume decrease in depression? Are neurogenesis, glial changes and apoptosis implicated? European archives of psychiatry and clinical neuroscience 257(5): 250-260.

30. Anacker C, Zunszain PA, Carvalho LA, Pariante CM (2011) The glucocorticoid receptor: pivot of depression and of antidepressant treatment? Psychoneuroendocrinology 36(3): 415-425.

31. Sleiman SF, Henry J, Al-Haddad R, El Hayek L, Abou Haidar E, et al. (2016) Exercise promotes the expression of brain derived neurotrophic factor (BDNF) through the action of the ketone body β-hydroxybutyrate. eLife, 5, e15092.

32. Xie Y, Wu Z, Sun L, Zhou L, Wang, et al. (2021) The Effects and Mechanisms of Exercise on the Treatment of Depression. Frontiers in psychiatry 12: 705559.

33. Wang X, Cai ZD, Jiang WT, Fang YY, Sun WX, et al. (2022) Systematic reviewand meta-analysis of the effects of exercise on depression in adolescents. Child and adolescent psychiatry and mental health 16(1): 16.

34. Harvey SB, Øverland S, Hatch SL, Wessely S, Mykletun A, et al. (2018) Exercise and the Prevention of Depression: Results of the HUNT Cohort Study. The American journal of psychiatry, 175(1): 28-36.

35. Heissel A, Heinen D, Brokmeier LL, Skarabis N, Kangas M, et al. (2023) Exercise as medicine for depressive symptoms? A systematic review and meta-analysis with meta-regression. British journal of sports medicine, bjsports-2022-106282. 57(16):1049-1057.

36. Sun CW, Wang YJ, Fang YQ, He YQ, Wang X, et al. (2022) The effect of physical activity on anhedonia in individuals with depressive symptoms. PsyCh journal 11(2): 214-226.

37. Cao B, Zhu J, Zuckerman H, Rosenblat JD, Brietzke E, et al. (2019) Pharmacological interventions targeting anhedonia in patients with major depressive disorder: A systematic review. Progress in neuro-psychopharmacology biological psychiatry 92: 109-117.

38. Thase ME, Haight BR, Richard N, Rockett CB, Mitton M, et al. (2005) Remission rates following antidepressant therapy with bupropion or

How to cite this article: Alison Ghazal F. Efficacy Review of Neurobiologically-based Treatments for Depression and its Sub-types. Glob J Intellect Dev Disabil. 2023; 12(5): 555848. DOI: 10.19080/GJIDD.2023.12.555848

selective serotonin reuptake inhibitors: a meta-analysis of originaldata from 7 randomized controlled trials. The Journal of clinical psychiatry 66(8): 974-981.

39. McIntyre RS, Filteau MJ, Martin L, Patry S, Carvalho A, et al. (2014) Treatment-resistant depression: definitions, review of the evidence, and algorithmic approach. Journal of affective disorders 156: 1-7.

40. Brown WA (2007) Treatment response in melancholia. Acta psychiatrica Scandinavica. Supplementum (433): 125-129.

41. Brown EC, Clark DL, Forkert ND, Molnar CP, Kiss ZHT, et al. (2020) Metabolic activity in subcallosal cingulate predicts response to deep brain stimulationfor depression. Neuropsychopharmacology: official publication of the American College of Neuropsychopharmacology 45(10): 1681-1688.

42. Miguel C, Karyotaki E, Ciharova M, Cristea IA, Penninx BWJH, et al. (2021) Psychotherapy for comorbid depression and somatic disorders: a systematic reviewand meta-analysis. Psychological medicine 53(6): 2503-2513.

43. Kleinstäuber M, Witthöft M, Steffanowski A, Van Marwijk H, Hiller W, et al. (2014) Pharmacological interventions for somatoform disorders in adults. The Cochrane database of systematic reviews (11): CD010628.

44. Cuijpers P, van Straten A, Andersson G, van Oppen P (2008) Psychotherapy for depression in adults: a meta-analysis of comparative outcome studies. Journal of consulting and clinical psychology 76(6): 909-922.

45. Proudman D, Greenberg P, Nellesen D (2021) The Growing Burden of Major DepressiveDisorders (MDD): Implications for Researchers and Policy Makers. PharmacoEconomics, 39(6): 619-625.

46. Puigdemont D, Pérez-Egea R, Portella MJ, Molet J, de Diego-Adeliño J, et al. (2012) Deep brain stimulation of the subcallosal cingulate gyrus: further evidence in treatment-resistant major depression. The international journal of neuropsychopharmacology 15(1): 121-133.

DOI: 10.19080/GJIDD.2023.12.555848

How to cite this article: Alison Ghazal F. Efficacy Review of Neurobiologically-based Treatments for Depression and its Sub-types. Glob J Intellect Dev Disabil. 2023; 12(5): 555848. DOI: 10.19080/GJIDD.2023.12.555848