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HERBAL ANTIDEPRESSANT: AN EVIDENCE-BASED REVIEW OF PLANTS USED IN THE TREATMENT OF DEPRESSION AND ANXIETY

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ABSTRACT

Depression and anxiety damage brain cells, altering clinical outcomes and quality of life. Memory loss, anxiety, sleep disorders, and dementia affect millions globally due to mental health issues. Since ancient times, herbal therapies for neurological diseases have been used, and 88% of people worldwide use traditional medicine or medicinal herbs for general health. Herbal remedies enhance conventional treatments with plant substances. Central nervous system serotonin (5-HT), BDNF, glutamate, dopamine, and norepinephrine activities contribute. Molecular antidepressants alter serotonin, norepinephrine, and dopamine levels in depressed brains. Traditional plant-based medicine is essential in underdeveloped nations. Herbal and plant-based depression and anxiety treatments are growing in industrialized nations. About 25% of pharmaceuticals are plant-based, and 75% of people worldwide use traditional medicine. The 520 drugs contain 30% natural compounds or their derivatives, with 75% treating cancer and 60% infectious diseases. Fatty acids, flavonoids, polyphenols, alkaloids, triterpenoids, essential oils, and saponins are anxiolytics and antidepressants.

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INTRODUCTION

Mental health is an important morbidity indicator. In 2008, 29.2% of adults had a history of mental illness, and 1 in 5 had experienced it in the previous year. 2017;20 Noorbala AA FS, Kamali K, et al. WHO created a 2013–2020 evidence-based mental health program. World Health Organization, 2013–2020 mental health action plan Representative cohort studies that compare pre-pandemic data to the first few weeks of global lockdowns demonstrate that average psychological distress scores and clinically severe mental disease rates have increased. Pierce M, Hope H, Ford T, et al. 2020 Steinhoff, Bechtiger, et al. 2020. According to research on prior epidemics like SARS, quarantine exacerbated post-traumatic stress disorder and depressive symptoms. Hawryluck L, Reynolds DL, Garay JR, Mihashi M, Liu X, et al. 2004–2012 Psychological changes during the COVID-19 epidemic are unknown.

1. Depression types: 1. Major Depressive Disorder (MDD) produces chronically low mood, lack of interest in previously enjoyed activities, food and sleep disorders, attention issues, and feelings of worthlessness or guilt. S.J. Rupke, D. Blecke, et al. (2006) Symptoms include: Different varieties of depression have different symptoms, duration, and daily effects. These include being depressed almost every day, losing or gaining a lot of weight, sleeping too much or being restless, being tired or exhausted, and having suicidal

thoughts or attempts. The symptoms must interrupt daily living for two weeks.

2. Dysthymia = chronic depression: PDD produces poor emotions for two years (one year for toddlers and teens). Lower than MDD symptoms. PDD is milder and lasts longer, causing depression, low self-esteem, and apathy. At least two years without break. Cui, L., Li, S., Wang, S., Wu, X., Liu, Y., Yu, W., & Li, B. (2024).

3. Bipolar bipolar disorder produces depression and mania or hypomania. Depression is like major depression, except manic episodes can be impetuous, overconfident, or enthusiastic. Bipolar I (full manic episodes) and II (hypomanic episodes) are the main forms.

4. Psychotic depression: This severe sadness includes psychosis like delusions or hallucinations.

5. Unusual Depression: A person with this form of depression may feel better after good things happen.

Etiology: Major depression has a complicated genetic and environmental cause. Even though anyone can get depressed, first-degree relatives of depressives are three times more probable. Some evidence suggests hereditary variables affect late-onset depression less than early-onset. Senior depression has biological risk factors. Depression is connected to Parkinson's, Alzheimer's, stroke, MS,

seizures, cancer, macular degeneration, and chronic pain. Problems in life induce depression. Depressed people may have experienced the death of a loved one, a lack of social support, the burden of caring for others, financial concerns, interpersonal issues, and disagreements. [(2015) McCloud, Caddy, et al.] Unknown cause of severe depression. Researchers claim neurotransmitter availability, receptor alteration, and sensitivity cause emotions. Dale, Bang-Andersen, Sánchez (2015). Clinical and preclinical research shows 5-HT action in the CNS. Glutamate, BDNF, DA, and NE are involved. IHME Global Health Data Exchange 2023 SSRIs show CNS 5-HT activation causes severe depression. Brain receptor change, gene expression, neurotransmitter availability, and time-dependent intercellular signaling are involved. Fall and winter bring SAD, which disappears in spring and summer. CNS 5-HT, circadian rhythms, and light are linked to seasonal affective disorder. Depressive symptoms are worsened by vascular injuries that disrupt front striatal connections between the dorsolateral prefrontal cortex, orbitofrontal cortex, anterior cingulate, and Amygdala and hippocampus induce depression. Prozac targets molecules According to the depression hypothesis, antidepressants alter serotonin (5 HT), norepinephrine, and dopamine levels in depressed brains [(2006); Al-Harbi, K. S. (2012)].

Pathophysiology Depression medications are licensed. Isocarboxazid, selegiline, and tricyclic antidepressants (amitriptyline, imipramine) are effective but cardiotoxic. [Wang, Dwivedi, et al. (2021)] Second-generation antidepressants targeting serotonin or norepinephrine transporters are safe, efficacious, convenient, and pharmacodynamically sound. Duloxetine, bupropion, mirtazapine, vortioxetine. MDD patients reported low remission rates despite many medications. 50% of depressed people cannot take one antidepressant, while 20%–30% do not respond to others. Many monotherapy-like drugs treat severe depression. Herbal antipsychotics are better and safer. Depression treatment with ketamine is popular. Ketamine alleviated depression more than placebo in a clinical experiment [T. L. McCloud, C. Caddy, J. Jochim, J. Rendell, J. M. Diamond, P. R. Shuttleworth + A. Cipriani (2015)].

Aids suicidal depressives. Ketamine restores forehead global brain connections and stress-induced nuclear accumulation hypertrophy on FMRI. Methods show ketamine reductions.

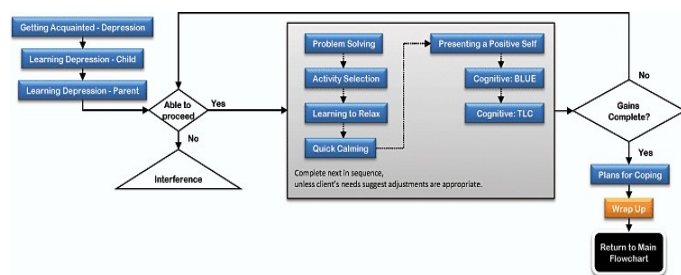


Fig. 1. Depicting the flow of depression

Types of Depression

MDD, or major depression: MDD is characterized by persistently low mood, lack of interest in previously enjoyed activities, changes in diet and sleep patterns, difficulties concentrating, and feelings of worthlessness or guilt [S.J. Rupke, D. Blecke, M. Renfrow, et al. (2006)] Some symptoms are Different types of depression have different symptoms, duration, and effects on daily life. These include being depressed most of the day, practically every day; losing or gaining a lot of weight; sleeping too much or being restless; being fatigued or lacking energy; and having suicide thoughts or attempts. Duration: Symptoms must disrupt daily life for two weeks.

Dysthymia-persistent depressive disorder: Chronic depression (PDD) causes low moods for at least two years (one year for kids and teens). Less severe than MDD symptoms. Though milder and longer-lasting, PDD often causes sadness, low self-esteem, and a lack of energy or interest. Duration: About two years without respite [Cui, L., Li, S., Wang, S., Wu, X., Liu, et al.]

Bipolar: bipolar disorder causes extreme mood swings (mania or hypomania) and depression. Depression is similar to major depression, although manic periods can make people impulsive, overconfident, or exuberant. Major types of bipolar disorder are Bipolar I (full manic episodes) and Bipolar II (hypomanic episodes).

Psychotic depression: Psychosis, such as delusions or hallucinations, is also present in this severe form of depression.

Unusual Depression: In this subtype of depression, the person still has depressive symptoms but may feel better after wonderful things happen.

Targets of Molecular Antidepressants -Depression hypothesis says that antidepressants alter serotonin (5 HT), norepinephrine, and dopamine levels in depressed patients' monoamine systems. Licensed depression antidepressants exist. Despite cardiotoxicity, monoamine oxidase inhibitors like isocarboxazid and selegiline and tricyclic antidepressants like amitriptyline and imipramine target the serotonin and norepinephrine transporter and are effective [P. Templeton, J. F. Mendes, O. Remes (2021)].

Second-generation antidepressants targeting serotonin or norepinephrine transporters are safe, effective, convenient, and pharmacodynamically sound. Citalopram, fluoxetine, venlafaxine, duloxetine, bupropion, mirtazapine, and multimodal antidepressants. Despite many treatments, MDD patients rarely recover. 50% of depressed persons cannot take one antidepressant, while 20%–30% do not respond to others. Many monotherapy-like medicines address severe depression. Herbal antipsychotics are superior and safer. NMDA receptor antagonist ketamine is a popular fast-acting antidepressant. Ketamine decreased depression more than placebo in clinical trials. [S.J. Rupke, D. Blecke, M. Renfrow (2006)] say it aids depressed suicide contemplators. FMRI shows ketamine recovers forehead global brain connections and stress-induced nuclear accumulation hypertrophy. Ketamine activates AMPA receptors and decreases NMDA receptors, affecting BDNF signaling. FDA-approved rapid-acting ketamine (SPRAVATO) treats non-responding depression. [WHO Traditional Medicine Strategy 54 (2013)], [2014–2023Mental and behavioral disorders (F00-F99). Chapter V (2016) (accessed February 6, 2018)].



Fig. 2. Difference between Depression and Anxiety Disorder

Current treatment and prevention Depression is treated and prevented with medications and nondrugs.[JW Williams Jr., JM Gierisch, J. McDuffie, J.L. Strauss (2014)]. Nonpharmacological treatments include ECT, VNS, cranial electrotherapy, MBT, wake therapy, yoga, exercise, music, and psychotherapy.

Traditional Treatment of Depression

No.	Plant	Family and Common name	Parts of plants showing Antidepressant property	Type of extracts used	Models/Tests performed	Mechanism of antidepressant activity	Nutraceutical compounds responsible
1.	Agapanthus campanulatus	Family: Agapanthaceae Common name: Bell agapanthus	Leaves, Flowers, Roots	Aqueous, Ethanolic	[3H]-citalopram- binding assay, TST, FST, Serotonin transporter (SERT), Norepinephrine transporter (NET) and Dopamine transporter (DAT) uptake inhibition assay	High affinity for SERT, Inhibition of SERT, NET and DAT	Flavonoids
2	Akebiae fructus	Chinese herb found in: Jiangsu, Anhui, Zhejiang, and Hunan provinces of China	Family: Lardizabalaceae Common name: Akebia Fruit powder	Ethanolic	TST, FST, Chronic unpredictable mild stress test (CUMS), Locomotor activity, Sucrose preference test (SPT), Monoamine uptake assay	High affinity for SERT, NET and DAT; affinity being highest for NET > DAT > SERT; seen for both rat and human transporters. Also, inhibits uptake activity of all three transporters in a dose dependant manner; inhibition of NET being the highest	Hederagenin
3	Albizzia julibrissin	Family: Fabaceae Common names: Mimosa, pink siris or Persian silk tree	Stem bark	Ethanolic	TST	Via activation of the serotonergic system through the 5- HT1A receptor system	Saponins
4	Albizzia lebeck	Family: Fabaceae Common names: Lebbek Tree, Flea Tree etc	Bark	Ethanolic	TST, FST, Locomotor activity		
5	Allium cepa	Family: Amaryllidaceae Common names: Bulb onion, common onion	Bulb powder	Aqueous	FST, Locomotor activity	Prevented an increase in the metabolite/neurotransmitter ratio; suggesting its mechanism as an inhibitor of Monoamine oxidase (MAO)	Quercetin glycosides (Flavonoids)
6	Aloysia polystachya	Family: Verbenaceae Common names: Beebrushes, Té de burro	Aerial parts	Hydro-alcoholic	FST		
7	Anemarrhena asphodeloides	Family: Asparagaceae Common name: Zhi Mu	Leaves		FST, MAO Inhibition assay	Via interaction with norepinephrine (NE) and Serotonin (5-HT) systems, Inhibition of MAO-A and MAO-B activities	Sarsasapogenin
8	Aniba riparia	Family: Lauraceae Common names: Louro-rosa, Canelacheirosa	Unripe fruit	Ethanolic	TST, FST	Via interaction with serotonergic, noradrenergic and dopaminergic receptor systems	Riparin II, Riparin III
9	Apocynum venetum Linn.	Family: Apocynaceae Common names: Luobuma, European dogbane or Dogbane leaf	Leaves	Ethanolic	FST, Locomotor activity, TST	Increase in NE and Dopamine (DA) levels in the hippocampus. Interaction with dopaminergic D1 and D2 receptor systems	Hyperoside, Isoquercitrin (Flavonoids)
10	Areca catechu	Family: Arecaceae Common names: Betel nut, Chalia, Pinang palm, Indian nut	Areca nut	Ethanolic	FST, TST, Locomotor activity, MAO Inhibition assay	Increase in 5-HT and NE levels in the hippocampus, MAO-A inhibitor	Possibly Saponins Compound(s) responsible for MAO inhibition still need to be identified
11	Asparagus racemosus	Family: Asparagaceae Common names: Wild asparagus, Shatavari	Roots	Methanolic	FST, Learned helplessness (LH), TST, Locomotor activity, MAO Inhibition assay	Inhibition of MAO-A and MAO-B activities, Via interaction with serotonergic, noradrenergic, dopaminergic, and GABAergic receptor systems	
12	Bacopa monnieri	Family: Plantaginaceae Common names: Brahmi, Water hyssop, Thyme-leafed gratiola	Whole plant	Methanolic	FST, LH, Foot shock stress, Shuttle box test	Via interaction with serotonergic, noradrenergic receptor systems and Inhibition of MAO-A and MAO-B activities	
13	Benincasa hispida (Thunb.) Cogn.	Family: Cucurbitaceae Common names: Ash gourd, Kushmanda, Petha, White gourd, Winter gourd, Winter melon, White pumpkin, Ash pumpkin	Fruits, Seeds	Methanolic, Aqueous	TST, FST, Locomotor activity, MAO-A assay, Object recognition task, Morris water maze	Via interaction with serotonergic, noradrenergic, dopaminergic and GABAergic receptor systems, Inhibition of MAO-A activity	
14	Boophone distica	Family: Amaryllidaceae Common names: Gifbol, Bushman poison bulb, Tumble weed	Bulb	Ethanolic	[3H]-citalopram- binding assay, TST, FST, SERT, NET and DAT uptake inhibition assay	Inhibition of SERT, NET and DAT	Buphanidrine, Buphanamine (Alkaloids)
15	Bupleurum falcatum	Family: Apiaceae Common names: Chinese Thorough wax		Methanolic			

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16	Camellia sinensis	Family: Theaceae Common names: Green tea, White tea	Leaves, Buds	Ethanollic, Methanolic, Aqueous	TST, FST, Hole cross test, Open field test (OFT), Thiopental sodium induced sleeping time test		Theanine
17	Canavalia brasiliensis	Family: Fabaceae Common name: Brazilian jackbean	Seeds		FST	Via interaction with serotonergic, noradrenergic and dopaminergic receptor systems	Lectins
18	Carthamus tinctorius L.	Family: Asteraceae Common name: Sa flower		Aqueous, Ethanollic	TST, FST		
19	Casimiroa edulis	Family: Rutaceae Common names: Zapote blanco, Matasano, Sleepy zapote	Leaves	Hydroalcoholic	FST		
20	Cayratia japonica	Family: Vitaceae Common names: Bushkiller, Yabu Garashi, Japanese Cayratia Herb	Whole plant, Fruit	Methanolic	MAO Inhibition assay	MAO inhibition	Flavonoids
21	Centella asiatica	Family: Apiaceae Common names: Asiatic pennywort, Indian pennywort	Leaves	Ethanollic	FST, Hole board test (HBT)	Involved in ameliorating the function of HPA axis and increasing levels of monoamine neurotransmitters	Total triterpenes
22	Cimicifuga racemosa	Family: Ranunculaceae Common names: Actaea racemosa, Black cohosh, Black snakeroot		Ethanollic, Isopropanoliceous extracts	TST	Partial agonist of serotonin receptor subtypes	
23	Cissampelos sympodialis	Family: Menispermaceae Common names: Milona, Bindweed	Leaves	Ethanollic, Hydroalcoholic	FST		Total tertiary alkaloids
24	Citrus paradisi var. duncan.	Family: Rutaceae Common name: Grapefruit	Leaves	Methanolic	FST		
25	Clitoria ternatea Linn.	Family: Fabaceae Common names: Butterfly pea, Blue-pea, Cordofan-pea	Roots, Aerial parts	Ethanollic, Methanolic	TST, FST, Locomotor activity		
26	Coleus forskohlii	Family: Lamiaceae Common names: Plectranth barbatus, Indian Coleus			FST	Increasing brain cAMP availability	Forskolin
27	Convolvulus pluricaulis	Family: Convolvulaceae Common name: Shankpushpi	Whole plant	Ethanollic	TST, FST, Locomotor activity	Possibly through restoration of brain monoamines	
28	Crocus sativus L	Family: Iridaceae Common names: Saffron, Autumn crocus	Petal, Stigma	Aqueous, Ethanollic	FST	Induces dopamine and glutamate release in the brain	Kaempferol, Safranal, Crocin
29	Curcuma longa	Family: Zingiberaceae Common name: Turmeric	Rhizome	Aqueous	TST, FST, MAO Inhibition assay	Inhibition of MAO-A activity in mouse whole brain	Turmerone
30	Echium amoenum	Family: Boraginaceae Common names: Boraginaceous, Ox-tongue	Flowers	Aqueous	Clinical trial setting		
31	Eleutherococcus senticosus	Family: Araliaceae Common names: Siberian Ginseng, Eleuthero, Ciwujia, Ezoukogi	Root bark	Aqueous	FST		
32	Emblica officinalis	Family: Phyllanthaceae Common names: Phyllanthus emblica, Indian gooseberry, Dhatrik, Amla	Fruit	Aqueous	TST, FST, MAO Inhibition assay, Locomotor activity	Inhibition of MAO-A activity	
33	Galphimia glauca	Family: Malpighiaceae Common names: Thryallis, Noche buena	Aerial parts	Methanolic	FST		
34	Gastrodia elata	Family: Orchidaceae	Rhizome	Hydroalcoholic, Ethanollic	TST, FST	Possibly by regulating both serotonergic and dopaminergic systems	
35	Gentiana kochiana	Family: Gentianaceae Common names: Gentiana acaulis, Ciminalis acaulis, Gentiana excisa	Aerial parts	Diethylether	FST, MAO Inhibition assay	Inhibition of MAO-A activity	
36	Glycyrrhiza glabra	Family: Fabaceae Common name: Liquorice	Roots	Aqueous, Hydroalcoholic, Ethanollic	TST, FST, Locomotor activity, MAO Inhibition assay	Mediated by increase in brain NE and DA, Inhibition of MAO-A activity	
37	Glycyrrhiza uralensi	Glycyrrhiza uralensi	Roots	Aqueous	SPT, FST, TST, CUMS, Locomotor activity	Increased 5-HT and NE in the mouse hippocampus, hypothalamus and cortex	
38	Gossypim herbaceum	Family: Malvaceae Common name: Levant cotton	Leaves	Aqueous		Activation of adenylyl cyclase-cAMP pathway in signal transduction system	
39	Hippeastrum vittatum	Family: Amaryllidaceae Common name: Knight's-star-lily	Bulb	Ethanollic	FST		Montanine (Alkaloid)
40	Humulus lupulus	Family: Cannabaceae Common name: Hops		CO2 extract	FST		Alpha-acid

Depression causes dehydration, infection, ulceration, DVT, and other problems, but osteoporosis and CVD persist. Duke, Janick, Simon (Eds.), 1993 Depression treatment emphasizes food, exercise, and hydration. Teen depression treatment with CBT and IPT works and is advised. Self-perceptions that affect emotions and behavior are corrected with CBT. IPT reduces loneliness, grief, and role change [J.O. Fajemiroye, D.M. DaSilva, D.R. DeOliveira, I.A. Costa]. (2016) Depressed youth benefit from CBT, IPT, and attachment-based family therapy, research reveals. Yoga, music, and exercise improve mood and sleep. Sleep restriction and wake therapy significant since outcomes are measured the day after treatment. Chronotherapy changes biological cycles to treat diseases fast [H. Kim, S.Y. Kim, S.Y. Lee, C.G. Jang, et al. (2007)]. It relieves endogenous depression 70% more than neurotic depression 48%. Some medications are marginally better than psychotherapy for severe depression. [O. Remes, J. F. Mendes, P. Templeton (2021)]. A recent study investigates depression's biological, psychological, and social causes. [Brain sciences, 11(12), 1633]. The first treatment for clinical depression was TCAs and MAOIs. They were superseded by SNRIs and SSRIs due to their safety and side effects. High-risk suicide patients are often killed by EECT if treatment fails. The FDA sometimes allows vagus nerve stimulation cranial electrotherapy. Psychotherapy and medication are recommended for complex depression. [T. L. McCloud, C. Caddy, J. Jochim, J. Rendell, J. Diamond et al., al.].

Medicinal herbs for depression therapy - Many modern drugs come from plants [M. Lähteenvuo and J. Tiihonen (2021)]. Traditional medicine is essential for basic health in developing countries and has become a norm for good health [Wu, Bunney, et al. (1990)] The use of herbal medicines and plant products to treat anxiety and depression has expanded in industrialized nations. About 75% of people worldwide use traditional medicines for basic health [T.G. Dinan et al. (1999)]. Plants are used to make 25% of prescription medications, proving their therapeutic value. Natural chemicals or derivatives made 30% of the 520 drugs, according to Cragg et al. [D.J. Newman, G.M. Cragg, K.M. Snader (2003)]. Newman et al.'s expanded study found that over 60% of these drugs treat infectious diseases and 75% treat cancer [Brigitta B. et al. 2002]. Studies have revealed that fatty acids, flavonoids, polyphenols, alkaloids, triterpenoids, essential oils, and saponins are anxiolytic and antidepressant-like [American psychiatry Association, Diagnostic and Statistical Manual of Mental Disorders, fifth edition (dsm-v), American psychiatry publication, 2013]. Herbal drugs are increasingly employed in therapy due to their improved quality and efficacy [UNESCO, Culture and Health, Orientation Texts-World Decade for Cultural Development 1988-1997].

CONCLUSION

In conclusion, depression is a complex illness that necessitates an all-encompassing therapeutic strategy that considers social, psychological, and biological aspects. Even while treatment options and our understanding of its genesis have advanced, significant work remains in terms of early detection, stigma reduction, and care access. To more effectively address the worldwide burden of depression, research into novel therapies and preventative measures must continue. Establishing settings where people with depression feel empowered and supported to seek help requires cooperation between communities, legislators, and healthcare professionals.

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