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Review Article

ROLE OF *HARIDRA* AS AN ANTI-DEPRESSANT

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ABSTRACT
Depression is characterized by decreased interest in daily activities, irritation and low concentration for extended periods. It is the leading causes of disability and 2nd leading cause of death (by suicide) in world with a prevalence rate of 5% of the world's adult population according to World Health Organization. However, due to stigma, a lack of effective treatments, and a dearth of mental health resources, it is frequently misdiagnosed and untreated. *Haridra* also identified as *Curcuma Longa* in Latin is one of the Ayurvedic drug which can be pivotal in treatment in depression as well as easily available, cost effective in long run with virtually no side effects. Curcumin present in *Haridra* is proven to be effective antidepressant. It is effective in regulating the levels of neurotransmitters, insulin resistance, oxidative and nitrosative stress, hypothalamic-pituitary-adrenal disruptions, and modulate inflammatory pathways, excitotoxicity, neuroplasticity and the endocannabinoid system. We will go over *Haridra's* effects through its phytoconstituent curcumin and how it affects the etiopathogenesis of depression in this review.

INTRODUCTION
Depression is the most prevalent mental illness that lowers quality of life and raises mortality risk. The GBD 2021 study reported that Depressive disorders resulted in 56.3 million DALYs (Disability-adjusted life years) globally in 2021, equivalent to 1.9% of total DALYs.

The 5th Edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) lists the following symptoms as indicative of major depressive disorder: low mood, energy loss, markedly diminished interest, psychomotor retardation, feelings of guilt or worthlessness, insomnia or hypersomnia, noticeable weight loss, impaired concentration, and frequent thoughts of death. Despite understanding the pathophysiology of MDD, approximately one half of patients with depression exhibit the treatment resistance, which leads to a failure of the first-line treatment. Even though the remission rates of around 70% after the fourth line of treatment has been seen.[1]

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Furthermore, many patients reduce or discontinue the antidepressant due to significant adverse effects including constipation, dry mouth, sleeping trouble, cardiotoxicity, neurotoxicity, orthostatic hypotension and sexual dysfunction.[2]

Haridra also known as turmeric, botanically named as *Curcuma Longa* is a part of *Zingeberacaece* family. *Haridra* has synonyms such as *Lakshami, Mangala, Mangalya, Pavitra,* and *Shiva* [3], which illustrate the meaning of the term. It has numerous beneficial qualities and can be used to treat a wide range of illnesses, including *Shotha, Gulma, Prameha, Kustha, Swasa, Kasa, Jwara*, and *Vrana*. But lately, studies on the phytoconstituents of *Haridra* have gained a lot of traction due to promising results in animal and clinical trials for the treatment of cancer, MDD, and other illnesses. The main bioactive substances in *C. longa* are curcumin, AR-turmerone, a-turmerone, b-turmerone desmethoxycurcumin and bisdemethoxycurcumin. The components of *Haridra*, most especially curcumin, have demonstrated encouraging pharmacological actions in contemporary pharmacological research because of their anti-neuroinflammatory, neuroprotective, chemo preventive, immune-modulatory, and potentially chemotherapeutic properties.[4] Curcumin may have

antidepressant benefits by boosting neurogenesis, decreasing brain inflammation, and altering neurotransmitters like dopamine and serotonin. Research has demonstrated encouraging outcomes in animal models and certain clinical trials, suggesting that curcumin may serve as a natural substitute or supplement to conventional antidepressant drugs. In traditional Chinese medicine, *Haridra* also known as *Jiang Huang* has been used effectively for alleviating stress, depression, pain, mania and other mental diseases.[5]

This review will provide an overview of the several possible mechanisms of curcumin for the treatment of depression, as evidenced by studies conducted on humans and animals. In addition, a brief overview of the ongoing difficulties with curcumin utilized in antidepressant treatments as well as recent pre- and clinical trials will be provided.

This review aims to offer a thorough assessment of research on *Haridra's* possible antidepressant mechanisms in depressive illnesses.

METHODS AND MATERIAL

- Articles on depression, pathophysiology of depression, mode of action of curcumin, action of curcumin in MDD was searched through online publishing sites like PubMed, Elsevier, Frontier, Wiley, etc.
- Articles about meta-analyses, animal and human research, and literary reviews were chosen and shortlisted.
- These papers were sorted in the separate category of review and researched article.
- Data from these articles were collected and carefully compiled in this article with proper references.

Curcumin and HPA axis

Anxiety and depression lead to the dysfunction of the hypothalamo-pituitary axis. The hypothalamus releases corticotropin-releasing hormone in response to stress which in turn, stimulates the pituitary gland to secrete adrenocorticotrophic hormone, which in turn triggers the adrenal cortex to release glucocorticoids. The HPA axis is strictly regulated because high levels of glucocorticoids are harmful to the body.[6] The control of negative feedback is primarily mediated by mineralocorticoid and glucocorticoid receptors.[7] Cortisol is the main glucocorticoid that regulates emotions, particularly fear and anxiety, metabolism, and cognitive functions in humans.[8]

Curcumin has been demonstrated to reduce the physiological changes and symptoms of depression due to cortisol in animal models. It is shown that curcumin might return corticosterone levels to baseline in stressed rats [9]. In a mouse model, researchers demonstrated that curcumin may offer immunity to corticosterone-induced neurotoxicity and a reduction of serotoninergic receptor mRNA levels.[10] Curcumin may also restore normal adrenal gland size in stressed mice.[11]

Curcumin and Neurotransmitters

Monoamines

The monoamine hypothesis states that deficit in neurotransmitters like serotonin, nor-epinephrine and dopamine in the certain areas of brain leads to pathophysiology of depressive disorders. The hormone norepinephrine is distributed throughout the brain and is involved in mood regulation as well as the body's reaction to stress and other stimuli. Depression is closely linked to α2- and β-adrenergic receptors in the frontal and prefrontal cortex, suggesting a hypofunction of the noradrenergic system. Research has indicated that depression is associated with α2-adrenergic receptor downregulation.[12]

Important fear regions in the brain, including as the hippocampus, cortex, and raphe nuclei, have abundant of serotonin receptors, and stimulation of these receptors causes both short-term and long-term alterations. The most researched serotonin receptor is 5-HT1A. It is a crucial component of the fear circuit, which controls the autonomic and motor reactions to stress. In the temporal lobe and hippocampal regions, there is frequently a downregulation of the 5-HT1A receptor in relation to anxiety and depression.[12] A research investigation revealed the role of 5-HT1A receptors in the hippocampus area in the reduced cognitive function that is frequently linked to mood disorders.[13] In contrast, the raphe nuclei's auto and heteroreceptors function by hyperpolarizing the membrane and lowering neuronal excitability.

The idea that curcumin can alter monoaminergic systems is bolstered by animal research. Researchers demonstrated that curcumin improved serotoninergic and dopaminergic transmission in addition to inhibiting MAO-A and MOA enzymes that reversed the depressive-like behavior induced by chronic stress in mice. [14,15] It has also been shown to raise dopamine, serotonin, and norepinephrine in the rat striatum, hippocampus, and frontal cortex.[16]

In an investigation on curcumin's effects on serotonin (5-HT) receptors in animals, it was reported that the antidepressant-like effect they saw was associated with the serotoninergic system, maybe as a result of an interaction with 5-HT1A/1B and 5-HT2C receptors.[17] The antidepressant effect of curcumin appeared to be associated with 5-HT1A receptor

expression, since curcumin administration raised 5-HT1A receptor mRNA levels in all hippocampus subfields.[18] In a different study, it was shown that giving them a 5-HT1B receptor antagonist averted curcumin's antidepressant-suchlike effects. [19]

Glutamate

According to the current theory, depression may result from excessive glutamate activity, particularly extra synaptic glutamate, which may be harmful to neuronal function. Activation of the kynurenine pathway by inflammatory mediators can lead to an increase in glutamate receptor agonism. Quinolinic acid, a byproduct of the kynurenine pathway, not only acts as an NMDA agonist but also directly releases glutamate. Inflammatory mediators can thereby create a situation where glutamate receptor agonism is excessive, leading to neurotoxicity. On the other hand, activation of NMDA receptors by glutamate or quinolinic acid may also activate microglia, which sets off a vicious cycle by increasing the production of inflammatory mediators. Furthermore, microglial-released inflammatory mediators have a deleterious effect on astrocyte production of EAAT, which may hinder glutamate elimination.[20]

It has been demonstrated that curcumin inhibits the release of glutamate in the rat pre-frontal cortex, counteracting this phenomenon in a manner that is comparable to, but more potent than, that of fluoxetine.[16] A synergistic interaction between NMDA and 5-HT receptors was proposed when it was seen that administering a sub-effective dose of curcumin in conjunction with a sub-effective dose of fluoxetine caused an antidepressant-like effect.[21] Additional in vitro research has provided clarification on these findings, demonstrating that curcumin can downregulate the expression of the GluN2B portion of NMDA receptors and counteract glutamate-induced neurotoxicity on hippocampus cells.[22] Studies showing curcumin's effects on the glutamatergic system were linked to higher BDNF levels, which may explain why curcumin is such an effective anti-depressant.[17]

Curcumin and Inflammation

Interaction with Immune System

The cross-sectional association between inflammation and major depression is confirmed by the compelling evidence of elevated circulating concentrations of C-reactive protein and interleukin-6 in patients with major depressive disorder who are not taking antidepressant medication. This implies that immunomodulatory research targeting IL-6 reduction in patient subgroups with elevated inflammatory profiles may hold promise for treating patients with elevated inflammation and depression.[23]

When given to LPS-treated rats, Curcumin reduced pro-inflammatory cytokine levels, restored glutathione depletion in the hippocampus, and alleviated depression symptoms. It also reversed inflammatory responses and neuronal abnormalities in mice with mild stress exposure, suggesting that inhibiting the IL-1b pathway could be its efficacy.[16]

Human patient investigations revealed that curcumin reduced the levels of the inflammatory cytokines TNF-a and IL-1b in depressed individuals as compared to the placebo group. Furthermore, curcumin has been demonstrated in further clinical trials to reduce the levels of TNF-a, IL-6, and CRP in patients' plasma.[24]

Depression and Inflammasome Activation

NLRP3 inflammasome activation is linked to microglial activation, which can enter an active state upon recognizing environmental stressors and are more prone to induce prolonged inflammatory responses. This inflammasome acts as a transducer of neuroinflammatory responses, causing persistent neuroinflammation due to chronic exposure to noxious threats like stressful psychological inputs.[25] It is activated by two-step process of priming and promoting. Priming involves NF-kb activation, which can be triggered by TNF or pathogen-associated molecular patterns. Additionally, corticosterone may activate glucocorticoid receptors, which in turn may increase NLRP3 transcription. Another way to activate the NLRP3 inflammasome is the reduction of intracellular potassium levels through P2X7 purinergic receptors which leads to potassium efflux in microglia. An activated NLRP3 inflammasome cleaves pro-IL-1b and pro-IL-18 into active IL-1b and IL-18. [16,26]

Research has demonstrated that long-term stress can activate the NLRP3 inflammasome in mouse brains, which can result in depression and impaired social behavior.[27] Patients with depressive disorders have active NLRP3 in their mononuclear cells, and certain antidepressants may reduce this activation.[28] It has been demonstrated that curcumin inhibits glutamate-induced cell apoptosis and decreases IL-1b release, thereby shielding stressed rats against IL-1b-induced apoptosis.[29] Additionally, it blocked the production of P2X7 receptors, which prevented NLRP3 activation, lowered NF-kb activation, and decreased the mRNA expression of proinflammatory cytokines (IL-1b, IL-6, and TNF-a).[30]

Depression is linked to an excessive activity of nitric oxide synthase, which is a key factor in neuroinflammation and neurotoxicity processes in stress and depression. Suicidal and depressed patients

often have elevated levels of plasma NO and its metabolites.[31] Curcumin has been shown to counteract the increase of NOS activity in human cultured neurons exposed to quinolinic acid. In animal models, curcumin can inhibit NOS hyperactivation and increase hippocampal NO. [16] It can also attenuate LPS-induced microglial activation and NF-kb activation in the hippocampus and pre-frontal cortex, and decrease NOS levels.[32]

Kynurenine Pathway

Kynurenine acid and quinolinic acid are the two metabolites produced by the kynurenine pathway. Kynurenine Acid is neuroprotective in nature, however, quinolinic acid is NMDA agonists which is excitotoxic leading to synaptic loss. It also brings upon oxidative stress. Researchers found kynurenine acid is lowered and quinolinic acid is found in higher levels in depression.

The enzyme known as IDO (indoleamine 2,3-dioxygenase) catalyzes the conversion of tryptophan into kynurenine in the kynurenine pathway. An overabundance of this enzyme reduces tryptophan's availability for serotonin, a key player in the pathogenesis of MDD. Cyclooxygenase-2 -2, a pro-inflammatory messenger that raises Prostagandin E2 synthesis, is a cofactor for the increase in IDO. Studies show elevated levels of PGE2 and COX-2 in depression.[33]

Curcumin shows reduction of Cyclooxygenase-2 expression and Prostaglandin E2 synthesis.[16]

Curcumin and Insulin

The hormone insulin inhibits the reuptake of serotonin and nor-epinephrine and suppresses the alpha-2 receptors in hypothalamic neurons. Decreased neuroplasticity and changed dopamine signaling are the results of insulin resistance, hence linking insulin with MDD. Additionally, insulin has anti-inflammatory qualities that are compromised by insulin resistance.[16]

Based on recent studies, curcumin can reduce insulin resistance in addition to MDD.

Curcumin and Oxidative Stress

Depression patients have lower antioxidative systems and increased oxidative stress markers compared to healthy individuals. Recent research has shown that curcumin administration can correct depressive behaviors, improve memory functions, and improve oxidative stress in chronically stressed mice. Curcumin administration also reduced oxidative stress in the pre-frontal cortex and impaired oxidative parameters.[16] Chronic stress can impair oxidative parameters and mitochondrial enzyme complex activities, but curcumin administration reverses these effects and improves depressive-like behavior.[34]

Curcumin and Neuroprotection

Curcumin has been shown to have a neuroprotective effect through its inhibition of inflammatory pathways and mitigation of glutamate excitotoxicity.[29] Studies have shown that curcumin administration can improve depressive behavior, reduce hippocampal neuronal cell death, attenuate long-term depression, increase Brain-derived neurotrophic factor, and inhibit COX-2, suggesting neuroprotection via anti-inflammatory effects.[35] Curcumin can also stimulate the production of neurotrophic factors, particularly Brain-derived neurotrophic factor. Curcumin has also been shown to increase Brain-derived neurotrophic factor levels in the amygdala, alleviating depressive behavior and upregulating Brain-derived neurotrophic factor mRNA in the limbic system. In human studies, curcumin has been shown to increase plasma Brain-derived neurotrophic factor levels compared to the placebo group.[24]

Haridra

Haridra is the most commonly used spice since ancient times and is advocated to be included in daily diet by acharyas. In India, *Haridra* is the part of almost every Indian cuisine and used in auspicious ceremonies as decoration and offering for prayers.

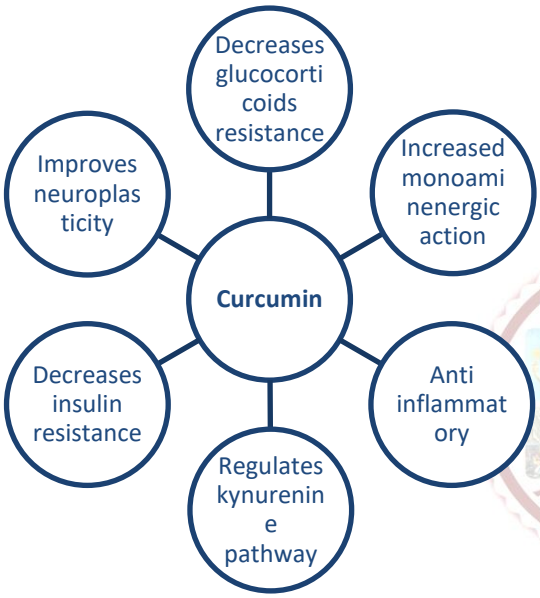
In Ayurveda, *Haridra* is sorted in *Kushtagna, Lekhniya, Kandugna, Vishagna, Tikta Skandha, Shiro-virechana, Haridradi, Mustadi, Shleshma samshanadi Gana.*

Rasa	*Tikta, Katu*
Guna	*Rukhsa, Laghu*
Virya	*Ushna*
Vipaka	*Katu*
Doshakarma	*Kaphavatashamaka, Pittarechaka*
Karma	*Vedanasthapana* - Alleviates pain *Ruchivardhaka* - Adds to the taste of food *Anulomana* - Helps in downward movement of *Vata* *Krimigna* - Removes disease causing microorganisms

	Raktaprasadana and *Vardhak* - Increases quality and quantity of blood. *Raktastambhaka* - Stops bleeding *Mutrasangrahniya* and *Virechaniya* - Helps in urine retention *Garbhashayashodhaka* - Cleanses uterus *Kushtagna* - Alleviates skin disorder *Jwaragna* - Alleviates fever [36]

Haridra although not mentioned specifically in classical books to be anti-depressant but in many drugs such as *Mahakalyanaka Ghrita, Manasamitra Vati, Siddhartaka Ghrita, Kalyanaka Ghrita, Shiva Ghrita,* etc. it is one of the main ingredients. These medicines are referred for either *Unmada* or *Manasa Vyadhi* in classics and prescribed for all kinds of *Manasik Vyadhi's* in practice.

RESULTS AND DISCUSSION



Numerous animal studies have demonstrated the effectiveness of curcumin in treating depression via a variety of pathophysiological routes. Turmeric, explained as *Haridra* in Ayurvedic texts can also be used for depressive disorders as it contains curcumin as its phytoconstituents and is been used as a content in various Ayurvedic formulations given for mental disorders. It has already been stated that *Haridra* works on *Vata Shaman* which is the cause of these *Vishada*, a disease corelated to depression from Ayurvedic perspective. Due to its *Vata Shamana* and *Shothahara* action it can counteract pathophysiology of MDD.

According to its mode of action, *Haridra*, one of the species that the Asian population eats, should result in fewer cases of MDD. But among Asians, the incidence of MDD is currently close to 37%. This might have resulted from curcumin's low bioavailability. Research on curcumin in both human and animal trials have produced encouraging results in terms of reducing the inflammatory alterations associated with MDD, which may be revolutionary. But there are doubts about its effectiveness because of its low bioavailability. It is natural to assume that *Haridra*, which contains only 3% curcumin, is less potent. But research has shown that turmeric has neuroprotective properties in elderly.[37]

A study on large population to detect efficacy of *Haridra* in comparison to its component standalone (curcumin) can put a light on whether bioavailability *Haridra* in human body has any relation to the form in which it is found. Furthermore, after determining its efficacy there is a need for addressing the dosage of *Haridra* or curcumin in long term patients of MDD.

CONCLUSION

Haridra, an Ayurvedic drug used in our daily food, has a neuroprotective effect and its phytoconstituent Curcumin is proven to be anti-depressant, anti-inflammatory and immune-modulatory. It modulates different pathophysiology of MDD and can be given to the patients in dosage for neurogenerative and anti-inflammatory action in MDD as an add-on drug with another anti-depressant. Single drug usage of *Haridra* as an anti-depressant should be render to more rigorous studies in a larger population because of curcumin's poor bioavailability.

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