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ANTIDEPRESSANT POTENTIAL OF NARDOSTACHYS JATAMANSI AND WITHANIA SOMNIFERA: A COMPARATIVE REVIEW

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Abstract
Major depressive disorder affects over 280 million people globally, yet conventional antidepressants are limited by delayed onset, adverse effects, and treatment resistance. This review, sourced from PubMed/MEDLINE, Embase, Cochrane Library, Scopus, and ClinicalTrials,gov (2000-May 2026), evaluates the antidepressant potential of two Ayurvedic Himalayan plants, *Nardostachys jatamansi* and *Withania somnifera. N. jatamansi* demonstrates rapid monoaminergic enhancement via MAO inhibition and SERT modulation, showing superior effects to fluoxetine in preclinical models. In contrast, *W.somnifera* exerts sustained effects through HPA axis normalization and BDNF upregulation, with strong clinical evidence. Both exhibit favorable safety profiles compared to SSRIs. Their complementary mechanisms suggest potential for combined use in managing treatment-resistant depression, highlighting the need for standardized formulations and well-designed clinical trials.

INTRODUCTION

Depression represents a common global health challenge, affecting over 280 million people and is one of the leading causes of disability in the world. It is a neuropsychiatric disorder that is characterized by persistent low mood, loss of interest, impaired cognition and emotional function, significantly affecting the quality of life(1–3). Conventional antidepressants such as selective serotonin reuptake inhibitors (SSRIs; e.g., fluoxetine, sertraline, escitalopram), serotonin norepinephrine reuptake inhibitors (SNRIs; e.g., venlafaxine, duloxetine), and tricyclic antidepressants (e.g., imipramine, amitriptyline) are being used widely and provides relief of symptoms but they show delayed onset of action, side effects like sexual dysfunction and weight gain and are also resistant in up to 30% of patients(2). These limitations have evoked the search for alternative approaches with better safety and therapeutic benefits. This systematic review synthesises evidence from PubMed/MEDLINE, Embase, Cochrane Library, Scopus and ClinicalTrials.gov (2000-May 2026).

Flavonoids, alkaloids, terpenoids, and glycosides are some of the phytochemicals that have been shown to have antidepressant properties through various mechanisms, including modulation of neurotransmitter systems, oxidative stress reduction, anti-inflammatory activity, and regulation of the hypothalamic pituitary adrenal (HPA) axis(4,5). Additionally, various substances derived from plants have had encouraging results in preclinical and clinical studies, indicating plant extracts as safer substitutes or adjuncts to traditional antidepressant treatments(6).

There are many medicinal plants in the Himalayas that have different pharmacological properties. Many of these plants have been used for a long time to treat central nervous system diseases. *Nardostachys jatamansi* (Jatamansi) and *Withania somnifera* (Ashwagandha) are two plants that stand out because they protect the brain and help with depression. These effects are based on Ayurvedic methods for easing insomnia and mental pain. Jatamansi primarily exhibits antidepressant effects by modulating monoamine neurotransmitters (such as serotonin and norepinephrine via monoamine oxidase (MAO) inhibition) and demonstrating significant antioxidant activity that alleviates oxidative stress in neuronal tissues(7). In contrast, Ashwagandha primarily functions by regulating stress responses, reducing cortisol levels, and

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improving neuronal function due to its adaptogenic properties(8).

Preclinical studies support the efficacy of jatamansi and ashwagandha. Jatamansi extracts (250-500mg/kg) exceed fluoxetine in forced swim and tail suspension tests, while Ashwagandha glycowithanolides (20-50 mg/kg) demonstrate comparable effects to imipramine in behavioural despair models. Emerging clinical data demonstrates that Jatamansi shows comparable results as imipramine for anxiety disorders that are also linked to depression, and that Ashwagandha can help with SSRIs in obsessive compulsive disorder (OCD) and mood stabilization(2,9–12).

Therefore, this review aims to comparatively evaluate the antidepressant potential of *Nardostachys jatamansi* and *Withania somnifera* with respect to their phytochemical constituents, pharmacological activities, and underlying mechanism of action.

PLANT PROFILE

Nardostachys jatamansi DC. (Family: Caprifoliaceae)

Nardostachys jatamansi, commonly known as Jatamansi or Indian spikenard, is a small, upright perennial herb that typically grows to a height of 30 to 60 cm, its base is rhizomatous and hairy. It can be found in China, Nepal, Bhutan, Tibet, Pakistan and India (especially Jammu & Kashmir, Himachal Pradesh, Uttarakhand and Sikkim). The high-altitude alpine Himalayas (3,000-5,000 m) are ideal for its growth. The International Union for Conservation of Nature (IUCN) has listed this plant as endangered because of overharvesting, despite its beautiful pinkish-purple flowers and highly fragrant rhizomes that are vital to traditional medicine(13–15).

Important phytochemicals include sesquiterpenes (valeranone, nardostachone, and jatamansone), valeranone derivatives, lignans, neolignans, coumarins (aristolochene), and alkaloids, which are primarily found in roots and rhizomes. These boost its other CNS-modulating effects and depressive efficacy(2,14,16).

Withania somnifera (L). **Dunal** (Family: Solanaceae)

Withania somnifera, is an upright, evergreen, tomentose shrub that can reach a height of 30 to 150 cm. It is also known as Indian ginseng or ashwagandha. It features small greenish-yellow blooms with reddish orange berries and ovate oblong leaves. It thrives in subtropical dry climates in India, Sri Lanka, Afghanistan, Pakistan, Nepal, the Middle East, Africa, and the Mediterranean, often on wastelands as high as 5,500 feet in the Himalayas(17–19).

The primary bioactive components are withanolides (steroidal lactones like withaferin A and withanolide D), alkaloids (withanine, somniferine, and tropine), sitoindosides, flavonoids, and saponins, which differ by chemotype and are concentrated in roots and leaves. These bolster its adaptogenic, depressive, and neuroprotective properties(20,21).

PHARMACOLOGICAL MECHANISMS

The antidepressant effect of medicinal plants is exerted through multiple neurobiological mechanisms including regulation of the HPA axis, modulation of neurotransmitter levels of monoamines like serotonin, dopamine and norepinephrine and also through antioxidant mechanism that scavenges reactive oxygen species (ROS) and also by anti-inflammatory process by diminishing the neuroinflammation. Jatamansi and ashwagandha exert its effects through different pathways and mechanisms that are harmoniously integrated to confer a therapeutic effect thereby proving to be potential herbal drugs in the management of depression(7,22).

Nardostachys jatamansi

The antidepressant effect of jatamansi was seen to be mediated by increasing the monoamine neurotransmitter systems, by increasing the levels of serotonin, dopamine, and norepinephrine in the brain. This is mediated by the MAO inhibition. Just like the other established MAO inhibitors, it also acts by preventing the breakdown of these neurotransmitters hence increasing their availability in the synaptic cleft. Jatamansi also has antioxidant activities which plays crucial role in depression as well as in neuroprotection. The antioxidant activities of jatamansi, reduced the oxidative stress associated with depression and the neuroprotective activities enhance the neuronal integrity, resulting in the reduction of the depressive like behaviours in several models of experimental depression(2,23).

Withania somnifera

Ashwagandha's antidepressant activity can be attributed to its action on the dysregulated HPA axis, which is the key neuropathology of depression, and its adaptogenic and anti-stress activity, which includes regulation of chronic stress evoked cortisol decrease, and thus leading to mood stability by showing a significant reduction in elevated cortisol levels; anti-inflammatory and antioxidant effects in neuronal cells; and that it works well against stress-induced models of depression by having an indirect effect on neurotransmitter system and promoting stress recovery mechanisms(24–27).

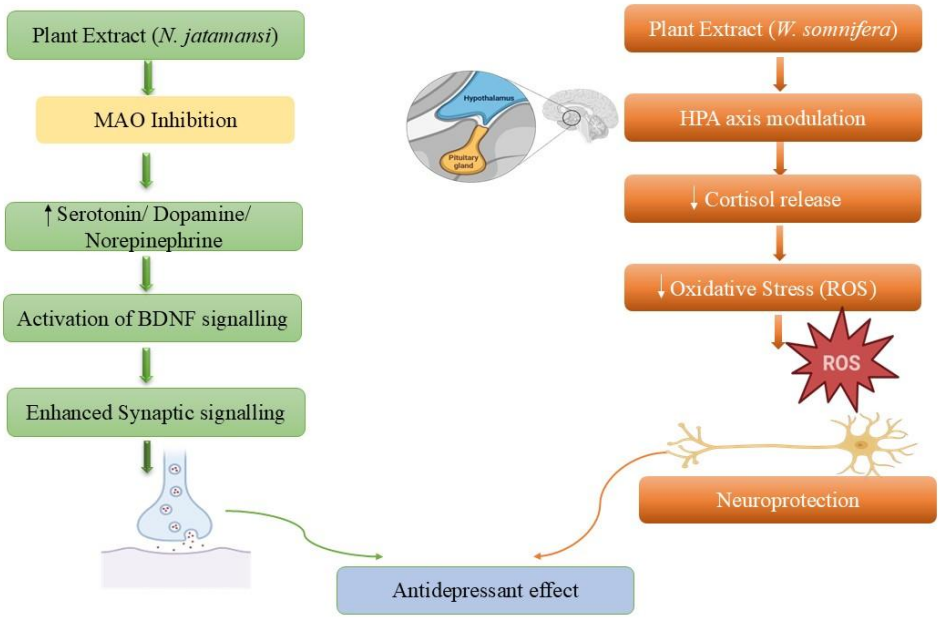

Figure 1: Integrated mechanistic pathways of plant extract mediated antidepressant effects

Comparative Analysis

Ashwagandha is primarily associated with stress-axis modulation through positive regulation of HPA axis and reduction of cortisol levels. On the other hand, jatamansi is primarily associated with direct monoaminergic stimulation through increased neurotransmitter levels and inhibition of MAO. Thus, the reciprocal antioxidant and neuroprotective properties of these two herbs that are complementary to the other's effects in alleviating the two major pathologies of depression namely monoamine depletion and chronic stress suggest possible synergistic benefits when used in conjunction and therefore possible uses in combination therapies(7,23–28).

PRECLINICAL EVIDENCE

The antidepressant efficacy of jatamansi and ashwagandha was validated through a number of preclinical studies primarily using the widely accepted standardized models of behavioural despair for assessing immobility as an index of depressive like behaviour.

Nardostachys jatamansi

Hydroalcoholic rhizome extract of jatamansi in different doses of 125, 250 and 500 mg/kg (p.o) showed dose dependent antidepressant activity. The maximum decrease in immobility time as compared to fluoxetine (20 mg/kg) was seen with 500 mg/kg in forced swim test (FST) and tail suspension test (TST). There was no effect on locomotor activity in open field test at 250-500 mg/kg dose which confirmed the specific behavioural effect rather than sedation. Further, the jatamansi hydroalcoholic rhizome extract in yohimbine potentiation test revealed the partial α2 adrenoreceptor antagonism which indicated the involvement and certain receptors in antidepressant behaviour. It also exhibited synergistic effects with the sub-therapeutic dose of fluoxetine (5 mg/kg)(2,9).

Withania somnifera

Both glycowithanolides from glycyrrhizan- extracted root extract showed antidepressive effects comparable to those of a reference drug imipramine in a battery of behavioural despair and learned helplessness tests in rats when administered orally at doses of 20, 50 mg/kg for 5 days. The glycowithanolides treated animals showed a decreased number of escape failures in the behavioural tests and an increased sucrose preference (anhedonia reversal). Chronic treatment with the glycowithanolides also reversed monoamine depletion in the brain (5-HT and NE) induced by unpredictable chronic mild stress (UCMS) without affecting decrease in body weight, which is a side effect of chronic administration of tricyclic antidepressants. The glycowithanolides treated animals also showed recovery of the HPA axis and increased expression of BDNF in the hippocampus(24–26).

Model/Test	*N. jatamansi*	*W. somnifera*	Standard drug (dose)	% immobility reduction	Reference
Forced Swim Test (FST)	Hydroalcoholic extract (500 mg/kg, p.o.)	Glycowithanolides (50 mg/kg, 5 days)	Fluoxetine (20 mg/kg)	42% vs 28%	(2,29)
Tail Suspension Test (TST)	250-500 mg/kg, p.o	Gycowithanolides (50 mg/kg)	Imipramine (10 mg/kg)	38% vs 35%	(2,30)
Learned helplessness	Limited data	20-50 mg/kg = imipramine)	Imipramine (10 mg/kg)	Equal	(25)
Chronic stress models	Hippocampal protection	Monoamine/BDNF restoration	-	Model-specific	(2,15)

Table 1: Comparative antidepressant Efficacy of *N. jatamansi* and *W. somnifera* in preclinical behaviour despair models

Summary of key preclinical findings across standardized depression models. N. jatamansi hydroalcoholic extracts demonstrate superior acute monoaminergic effects (FST: 42% vs fluoxetine 28% immobility reduction), while W. somnifera glycowithanolides match imipramine in chronic stress paradigms. All effective doses-maintained locomotor activity, confirming an4tidepressant specificity without sedation.

CLINICAL EVIDENCE AND SAFETY PROFILES

Although preclinical studies demonstrate robust antidepressant effects of jatamansi and ashwagandha these are met with divergent human clinical trial evidence, whose translation potential differs significantly between the two herbs with the evidence of ashwagandha being considerably higher than that for jatamansi and thus, more conductive to further clinical investigation and potential therapeutics.

N.jatamansi

Clinical evidence of jatamansi is limited and only derived from the Ayurvedic integrative clinical studies. A randomized control trial (RCT) involving 60 patients investigated the efficacy of standardized jatamansi extract (500 mg/kg) vs imipramine (75 mg/kg) in patients for 8 weeks with primary diagnosis of anxiety disorder with comorbidity of depression. Both regimens produced a similar degree of reduction in the Hamilton Anxiety Rating Scale (HAM-A) scores and were well-tolerated (68% vs 72% reduction from baseline). Open-label studies derived from the stress-related insomnia treatment indicated the beneficial effects of jatamansi when used in combination with other adaptogens and hence supporting its sedative properties(31,32).

Safety: Human doses up to 2 g/day exhibit only mild gastrointestinal symptoms (nausea 8%); in animals, the acute oral LD50 surpasses 5 g/kg. Throughout centuries of traditional use, there have been no reports of hepatotoxicity, sedation, or cardiotoxicity. Safety is confirmed by long-term ayurvedic formulations(31,32).

W. somnifera

There is significant clinical validation for ashwagandha. Root extract (300-600 mg/day standardized to 5% withanolides, 8 weeks) significantly decreases anxiety (HAM-A: 12.72 vs -4.47) and depression severity (HAM-D: -10.65 vs placebo -4.37; SMD = -1.85, $p<0.001$), according to a 2021 systematic review and meta-analysis (5RCTs, n=491). With a 56.5% HAM-A response rate compared to a 30.2% placebo, the effects are especially noticeable in stress-related depression subtypes. A 2025 RCT involving 91 subjects having the diagnosis depression and OCD showed that SSRIs were effective as an additional therapeutic intervention(26,33,34).

Safety: Based on data from more than 25 trials, it was observed that human doses up to 1,250 mg/day were tolerated well. Gastrointestinal (GI) distress (2.1%) and temporary sleepiness (3.4%) are the only mild side effects. There have been no reports of withdrawal, hepatotoxicity, or severe side effects and is safe for specific groups (elderly, equivalent to pregnancy Class B)(24,34).

Plant	Study design	Dose/duration	Sample size	Primary outcome	Effect size vs placebo/comparator	Reference
N.jatamansi	RCT	500 mg/day extract, 8 weeks	n=60	HAM-A ↓68%	Equivalent to imipramine (75 mg/day)	(32)
N.jatamansi	Open-label	Ayurvedic formulation	n=45	PSQI sleep improvement	Moderate effect	(31)
W.somnifera	Meta-analysis (5RCTs)	300-600 mg/day (5% withanolides), 8 weeks	n=491	HAM-D ↓23-44%	SMD=-1.85 (p<0.001)	(34)
W.somnifera	RCT (adjunctive)	300 mg BID + SSRI, 12 weeks	n=91	OCD-Depression composite ↓	Significant adjuvant benefit	(33)

Table 2: Summary of human clinical evidence *Clinical evidence highlights W. somnifera's robust RCT support (level Ib) versus N. jatamansi's preliminary findings (level IIb). HAM-A/D: Hamilton Anxiety/Depression scales; PSQI: Pittsburgh Sleep Quality Index.*

Safety parameter	***N. jatamansi***	***W. somnifera***	**SSRIs**	**Reference**
Acute LD50	>5 g/kg (rodents)	>5 g/kg (rodents)	N/A (therapeutic dosing)	(31,33)
Therapeutic AEs	Mild GI (8%)	Drowsiness (3.4%)	Sexual dysfunction (40%), nausea (25%)	(32,34)
Hepatotoxicity	None	None	Rare (0.1%)	(24,35)
Chronic use	Ayurvedic safe	25+ RCTs safe	Weight gain ↑	(31,34)
Special population	Traditional use	Safe for Elderly/pregnancy	Multiple cautions	(33,36)

Table 3: Comparative safety profiles *Both plants exhibit superior safety margins compared to SSRIs. N. jatamansi leverages centuries of Ayurvedic safety data; W. Somnifera benefits from extensive modern RCTs. LD50: lethal dose 50%; AEs: adverse events.*

CONCLUSION

Depression remains a crucial mental health issue worldwide due to its serious negative effect, delayed onset, and over one-third of cases are characterized by significant treatment failure. This comparative review highlights *Nardostachys jatamansi* and *Withania somnifera* as natural pharmacotherapeutics that, through different yet complementary mechanisms, effectively address these challenges.

In traditional models of behavioural despair, jatamansi surpasses fluoxetine by swiftly enhancing monoaminergic potentiation through the inhibition of MAO and the upregulation of serotonin transporter, leading to immediate antidepressant effects. Ashwagandha offers clinically proven adaptogenic neuroprotection by consistently reducing cortisol levels, stabilizing the HPA axis, and boosting BDNF. Meta-analysis has shown that it significantly reduces the severity of depression compared to a placebo.

Their unique therapeutic properties allow for strategic clinical application: ashwagandha can serve as a primary treatment or complement SSRIs for depression associated with chronic stress, whereas jatamansi is effective for acute episodes needing rapid monoamine level restoration. With high therapeutic indices (LD50 >5 g/kg), minimal side effects (less than 8% experience mild gastrointestinal issues or drowsiness), and a long history of traditional use, these plants offer significantly greater safety compared to synthetic antidepressants.

Developing standardized extracts, conducting adequately powered head-to head RCTs compared to SSRIs, exploring fixed-dose combination therapies for treatment resistant depression, employing biomarker-based patient selection, and implementing sustainable cultivation methods to address the endangered status of

jatamansi are all key areas of research important for clinical application.

Collectively, these Himalayan medicinal plants signify the next advancement in precise herbal medicine more effective, better tolerated, and offering a thorough mechanistic understanding. Focusing regulatory attention on pharmaceutical-grade formulations might significantly transform the approach to managing depression, effectively combining Ayurvedic healing knowledge with modern evidence-based medicine to help millions of people globally.

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