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Review Article

An Overview of the Anxiolytic, Antidepressant, and Neurobehavioral Effects of Nardostachys Jatamansi: From Traditional Medicine to Modern Pharmacological Evidence

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ABSTRACT

Nardostachys jatamansi (D.Don) DC., commonly known as jatamansi, is a high-altitude medicinal plant traditionally used in Ayurveda and other Asian medical systems for conditions involving disturbed sleep, emotional agitation, and neurological dysfunction. Modern pharmacological investigations have examined its effects on anxiety-like and depression-like behaviour, cognition, stress responses, and neuronal injury. This narrative review critically integrates traditional knowledge with experimental and emerging clinical evidence, with particular attention to the relationship between phytochemical composition and neuropsychopharmacological activity. Reported constituents include sesquiterpenes and sesquiterpenoids such as jatamansone (valeranone), nardostachone, nardosinone and related compounds, together with volatile-oil components and other secondary metabolites. Rodent studies suggest anxiolytic-like effects associated with GABAergic signalling, including modulation of the GABA-benzodiazepine receptor complex, while antidepressant-like effects have been linked to monoamine oxidase inhibition, altered monoaminergic tone and possible GABA-related mechanisms. Separate neurobehavioral studies report effects on learning, memory, stress-associated cognitive impairment and motor behaviour. Antioxidant, anti-inflammatory and neuroprotective pathways may contribute to these outcomes, but the evidence is heterogeneous and often based on non-standardised extracts. Early controlled clinical studies, including adjunctive treatment in depression and investigation in post-stroke depression, are encouraging but insufficient to establish efficacy or safety as a standalone treatment. Major limitations include small clinical samples, inconsistent botanical authentication, variable extraction methods, incomplete pharmacokinetic data and limited long-term toxicological evaluation.

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Standardised preparations, rigorous clinical trials and sustainable sourcing are essential before therapeutic recommendations can be made.

INTRODUCTION

Anxiety and depressive disorders are common and frequently coexist with sleep disturbance, impaired concentration, reduced motivation and changes in stress responsiveness. Although established pharmacotherapies are effective for many patients, incomplete response, delayed onset of benefit and adverse effects continue to motivate the investigation of complementary pharmacological approaches. Medicinal plants are of interest because they contain chemically diverse constituents that may influence several biological pathways. However, traditional use alone does not establish clinical efficacy, and plant-derived products require the same attention to identity, quality, dose, safety and interaction potential as other pharmacologically active substances.

Nardostachys jatamansi (D.Don) DC. is a perennial aromatic herb of the family Caprifoliaceae (traditionally placed in Valerianaceae). Its rhizomes and roots have a long history of use in South Asian traditional medicine. Ayurvedic descriptions associate jatamansi with calming, sleep-promoting and cognition-related uses. Contemporary studies have investigated extracts and isolated constituents in animal models of anxiety, depression, memory impairment, stress and neurodegeneration. A comprehensive review of the plant's pharmacology was published recently; therefore, the present article adopts a narrower and more critical focus on the anxiolytic, antidepressant and broader neurobehavioral domains, emphasising how mechanisms, experimental models and translational evidence intersect [1].

The central question is not simply whether jatamansi produces a behavioural effect, but whether reproducible effects can be linked to defined chemical preparations, plausible molecular mechanisms and clinically meaningful outcomes. This review therefore distinguishes between traditional indications, preclinical signals and clinical evidence, and highlights the gaps that must be addressed before clinical use can be recommended.

REVIEW METHODOLOGY

This article was prepared as a narrative review. Literature was identified through targeted searches of PubMed, PubMed Central, journal websites and scholarly search platforms using combinations of the terms “Nardostachys jatamansi”, “jatamansi”, “anxiety”, “anxiolytic”, “depression”, “antidepressant”, “neurobehavioral”, “GABA”, “monoamine oxidase”, “memory”, “stress”, “neuroprotection” and “clinical trial”. Priority was given to primary experimental studies, controlled clinical investigations, pharmacological reviews and authoritative botanical or journal sources. The search was updated to include literature available up to November 2026.

Studies were considered relevant when they investigated authenticated N. jatamansi material, extracts or isolated constituents and reported outcomes related to anxiety-like behaviour, depression-like behaviour, cognition, locomotion, stress, neuronal protection or clinical symptoms. Because the included studies differ in species, extract type, dose, duration, model and outcome measures, a quantitative meta-analysis was not attempted. Findings are presented narratively, and animal behavioural outcomes are described as “anxiolytic-like” or “antidepressant-like” rather than as proof of treatment efficacy in humans.

A limitation of this approach is that a narrative search may not capture every relevant publication and does not provide the reproducibility of a registered systematic review. Before journal submission, the authors should repeat the search using documented database-specific search strings, record the number of records screened and retained, and verify every bibliographic detail against the original article.

BOTANICAL AND ETHNOMEDICINAL PROFILE

1 Botanical Identity And Distribution

N. jatamansi is a small, perennial, rhizomatous herb associated with high-altitude Himalayan habitats. The underground rhizome and roots are the principal medicinal parts described in traditional use. Correct botanical identification is essential because common names can be applied inconsistently and related aromatic species may enter commercial supply chains. A

pharmacological study should report the accepted scientific name, author citation, plant part, collection location, collection season, authentication authority and voucher specimen number.

2 Traditional Uses Relevant To Neuropsychopharmacology

Traditional sources describe jatamansi as a medicinal material used in preparations intended to calm the mind, support sleep and address selected neurological or emotional complaints. These descriptions provide a rationale for investigating central nervous system activity, but traditional diagnostic categories do not map directly onto modern psychiatric diagnoses. For example, a traditional indication described as restlessness or disturbed sleep cannot be equated automatically with generalised anxiety disorder or major depressive disorder. The appropriate interpretation is that traditional practice generated hypotheses that can be tested using validated pharmacological and clinical methods.

3 Plant Part And Preparation

Rhizome and root preparations, powdered material, aqueous extracts, alcoholic extracts, methanolic extracts and volatile-oil fractions are not chemically interchangeable. Solvent polarity, extraction temperature, duration, particle size, storage and geographical origin can alter the relative abundance of constituents. Consequently, a behavioural result obtained with one ethanolic extract cannot be assumed to apply to a commercial powder, essential oil or differently prepared extract.

PHYTOCHEMICAL CONSTITUENTS AND THEIR PHARMACOLOGICAL RELEVANCE

The phytochemical profile of N. jatamansi includes volatile and non-volatile constituents, with sesquiterpenes and sesquiterpenoids receiving substantial attention in pharmacological research. Jatamansone, also known as valeranone, and compounds such as nardostachone, nardosinone and jatamansinol have been reported in the literature. The exact composition varies with plant material and analytical method. Other reported classes include coumarin-related compounds, lignans, neolignans and additional phenolic or terpenoid constituents [1].

1 Sesquiterpenes And Sesquiterpenoids

Sesquiterpenoid constituents are frequently investigated as candidate contributors to the biological activity of jatamansi. Nardosinone has been studied in neuroinflammatory and neuroprotective experimental systems, while jatamansinol has been investigated in a Drosophila model of amyloid-beta-associated neurotoxicity. These studies support interest in individual constituents but do not establish that any single compound is responsible for the behavioural effects of a whole extract [2,3].

2 Whole Extracts Versus Isolated Constituents

Whole extracts may contain multiple constituents with additive, synergistic, antagonistic or pharmacokinetic interactions. An isolated compound may produce a clear effect in a cellular assay while contributing little to the activity of a crude extract at the concentration actually achieved in vivo. Conversely, a multi-constituent extract may show activity that is not reproduced by one marker compound. Comparative studies should therefore evaluate the whole extract, chemically standardised fractions and selected isolated constituents in parallel.

3 Need For Chemical Standardisation

A reproducible jatamansi preparation should be characterised by chromatographic fingerprinting, quantitative marker analysis and stability testing. High-performance liquid chromatography (HPLC), gas chromatography-mass spectrometry (GC-MS) and liquid chromatography-mass spectrometry (LC-MS) can be used according to the chemical fraction under investigation. A single marker is unlikely to represent the full pharmacological profile; a multi-marker specification and a validated fingerprint would provide a stronger quality-control strategy.

Table 1: Selected Phytochemical Groups and Their Research Relevance

Constituent / Chemical Group	Reported Research Interest	Interpretive Caution
Jatamansone (valeranone)	Frequently cited sesquiterpenoid associated with the pharmacology of jatamansi.	Presence does not prove it is the sole active principle.
Nardosinone	Investigated in neuroinflammation and experimental neuroprotection.	Cell and animal findings require clinical validation.
Jatamansinol	Studied in an amyloid-beta Drosophila neurotoxicity model.	A model-specific effect cannot be generalised to human dementia.
Volatile-oil constituents	May contribute to aroma and biological activity of essential-oil fractions.	Composition is sensitive to origin, processing and storage.
Other terpenoid / phenolic constituents	Potential antioxidant or other pharmacological contributions.	Concentration and relevance differ among preparations.

PROPOSED NEUROPHARMACOLOGICAL MECHANISMS

1 GABAergic Signalling And Anxiolytic-Like Activity

Gamma-aminobutyric acid (GABA) is the major inhibitory neurotransmitter in the adult mammalian central nervous system. GABA-A receptors are ligand-gated chloride channels, and benzodiazepines enhance GABA-A receptor function through an allosteric binding site. In a mouse study, an N. jatamansi extract was evaluated using the elevated plus maze, open-field test, light-dark box and Vogel conflict test. Seven days of treatment produced anxiolytic-like behavioural changes alongside alterations in brain GABA, monoamines and antioxidant parameters. The effects were attenuated by flumazenil and picrotoxin, supporting involvement of the GABA-benzodiazepine receptor complex [4].

These antagonist experiments provide mechanistic evidence, but they do not prove direct binding of a jatamansi constituent to the benzodiazepine site. Antagonists can modify behaviour through their own pharmacology, and whole-extract studies cannot identify the responsible molecule. Binding assays, receptor-subtype studies, electrophysiology and exposure measurements are needed to establish the precise molecular interaction.

2 Monoaminergic Modulation And Antidepressant-Like Activity

Serotonin, noradrenaline and dopamine contribute to mood, motivation, reward processing and stress adaptation. Monoamine oxidase (MAO) enzymes metabolise monoamine neurotransmitters. In a mouse study, repeated oral administration of an ethanolic extract of N. jatamansi reduced immobility in the forced swim and tail suspension tests and reduced brain MAO-A and MAO-B activities. The investigators also reported that the effect was comparable to reference antidepressants in the experimental setting [5].

The findings are consistent with a possible monoaminergic contribution, but enzyme inhibition measured in whole-brain homogenate does not establish selective MAO inhibition in humans. Clinically relevant MAO inhibition could also raise interaction concerns, particularly if a preparation is combined with serotonergic or sympathomimetic medicines. Human enzyme studies, dose-exposure relationships and formal interaction assessments are therefore important.

3 GABA And The Complexity Of Antidepressant Mechanisms

The same antidepressant-like study reported reversal of part of the behavioural effect after pretreatment with baclofen, a GABA-B receptor agonist, suggesting that GABA-related signalling may contribute [5]. This observation should not be simplified into a claim that jatamansi uniformly increases or decreases GABA. GABA-A and GABA-B receptors have different functions, and effects may vary by brain region, dose, duration and experimental condition. The apparent involvement of GABA in both anxiolytic and antidepressant studies illustrates the complexity of whole-extract pharmacology.

4 Oxidative Stress And Antioxidant Defence

Oxidative stress can influence neuronal membrane integrity, mitochondrial function, inflammatory signalling and synaptic activity. Several experimental investigations of jatamansi have examined antioxidant enzymes and lipid peroxidation alongside behavioural outcomes. Antioxidant effects could contribute indirectly to preserved neuronal function, especially in models involving chronic stress or toxin-induced injury. Nevertheless, antioxidant capacity in a chemical assay is not equivalent to brain target engagement, and behavioural benefits should not be attributed to antioxidant activity without mechanistic intervention studies.

5 Neuroinflammation And Cell-Survival Pathways

Emerging studies have examined N. jatamansi extracts and nardosinone in microglial and Parkinsonian models. Reported findings include suppression of selected pro-inflammatory markers and changes in cellular energy metabolism. A 2026 investigation also reported activation of the Keap1-Nrf2-antioxidant response element pathway in experimental systems, including dopaminergic neuronal protection [2,6]. These findings broaden the mechanistic framework beyond classical neurotransmitters, but their relevance to anxiety and depression remains indirect until tested in disease-relevant behavioural models.

6 Neurotrophic And Synaptic Plasticity Pathways

Brain-derived neurotrophic factor (BDNF) supports neuronal survival and synaptic plasticity and is frequently investigated in depression research. A recent randomised, double-blind, placebo-controlled adjunctive study reported changes in serum BDNF alongside clinical and neurophysiological measures in participants receiving jatamansi with escitalopram [7]. Serum BDNF is not a direct measure of BDNF activity in specific brain regions, and it should be interpreted as a peripheral biomarker rather than proof of a defined central mechanism.

ANXIOLYTIC EFFECTS: EXPERIMENTAL EVIDENCE

The most directly relevant mechanistic evidence for anxiolytic-like activity comes from the mouse study by Razack and colleagues. The investigators used several behavioural paradigms rather than relying on one test, which strengthens the internal consistency of the behavioural signal. The elevated plus maze measures the conflict between exploration and avoidance of exposed arms; the light-dark box evaluates approach-avoidance behaviour; the open-field test measures exploration and locomotor activity; and the Vogel conflict test assesses punished drinking behaviour [4].

The reported increase in open-arm exploration and time in the illuminated compartment, together with changes in punished responding, was interpreted as anxiolytic-like activity. The use of flumazenil and picrotoxin provided pharmacological evidence implicating GABA-A/benzodiazepine-related signalling. However, locomotor stimulation or sedation can confound several anxiety tests. An increase in movement may be mistaken for reduced anxiety, while sedation may suppress exploration and obscure an anxiolytic effect. Future work should combine behavioural testing with automated locomotor tracking, dose-response analysis and blinded scoring.

The same study reported relatively low brain accumulation after radiolabelling, raising a question about the extent to which the detected behavioural effects are mediated by direct central exposure versus active metabolites or indirect peripheral pathways [4]. This observation makes pharmacokinetic characterisation particularly important. Quantitative measurement of candidate constituents and metabolites in plasma, cerebrospinal fluid and relevant brain regions would help connect dose, exposure and behavioural response.

ANTIDEPRESSANT EFFECTS: EXPERIMENTAL EVIDENCE

Dhingra and Goyal investigated the antidepressant-like effects of an ethanolic extract in Swiss albino mice. Repeated administration over 14 days reduced immobility in both the forced swim test and tail suspension test. The study also reported reduced MAO-A and MAO-B activity and a possible contribution from GABA-B-related signalling [5]. The inclusion of locomotor assessment was important because general motor stimulation can reduce immobility independently of an antidepressant-like action.

A further report assessed methanolic extract in normal and sleep-deprived mice using forced swim, tail suspension and locomotor activity tests. The reported findings suggested antidepressant-like effects at the tested doses and improvement in locomotor activity in sleep-deprived animals [8]. These results are hypothesis-generating, but differences in extract solvent and experimental design make direct comparison difficult.

The forced swim and tail suspension tests are screening tools that detect changes in stress-coping behaviour; they do not reproduce the full clinical syndrome of major depressive disorder. Immobility should not be interpreted as depression itself. Stronger preclinical evidence would combine chronic stress models, reward-related behaviour, social interaction, sleep measures, cognitive endpoints and physiological markers, while including appropriate controls for motor effects.

The clinical evidence is beginning to develop. A randomised, double-blind, placebo-controlled study examined jatamansi as an adjunct to escitalopram in major depressive disorder. The published report describes 60 participants receiving ongoing escitalopram who were allocated to jatamansi or placebo for two months, with reported improvements in depressive symptoms and selected neurobiological measures in the adjunctive group [7]. The findings are promising but do not establish jatamansi as monotherapy, and replication in larger, independently conducted trials is required.

A separate small randomised, double-blind controlled study examined jatamansi powder in post-stroke depression, reporting improvement in depression rating and quality-of-life measures over six weeks [9]. The small sample and specific post-stroke population limit generalisation to broader depressive disorders. Clinical trial registration, product characterisation, allocation concealment, adverse-event reporting and longer follow-up should be assessed carefully when interpreting such studies.

NEUROBEHAVIORAL EFFECTS BEYOND MOOD

1 Learning And Memory

N. jatamansi has been studied in learning and memory paradigms, including elevated plus maze transfer latency and passive avoidance tasks. An ethanolic extract administered to young and aged mice was reported to improve selected learning and memory measures and to attenuate amnesia induced by diazepam or scopolamine [10]. The reversal of scopolamine-associated impairment led the authors to propose a possible contribution from cholinergic transmission, while antioxidant activity was also discussed.

2 Stress-Associated Cognitive Impairment

Karkada and colleagues investigated whether N. jatamansi extract could protect against chronic restraint stress-associated learning and memory deficits using a radial arm maze task [11]. This line of research is relevant because chronic stress can affect hippocampal function, attention and spatial memory. It also provides a bridge between the plant's traditional calming reputation and measurable cognitive outcomes. However, effects in stress-exposed rodents cannot be directly translated into claims of improved academic performance, concentration or memory in healthy people.

3 Neurodegeneration-Related Behaviour

Jatamansinol has been studied in an amyloid-beta42 Drosophila model, with reported improvements in locomotor activity, learning and memory, alongside changes in oxidative stress and cholinesterase activity [3]. Other investigations have explored jatamansi extracts in Parkinsonian models and dopaminergic neuronal systems [2,6]. These experiments are useful for identifying neuroprotective pathways but are not evidence that jatamansi prevents or treats Alzheimer's disease or Parkinson's disease in humans.

4 Locomotor Activity, Sedation And Behavioural Confounding

A central nervous system-active botanical may influence arousal, muscle tone, exploration and sleep. These effects can alter the outcome of anxiety and depression tests independently of the intended pharmacological action. Every behavioural study should therefore include baseline locomotor measurements, assessment of sedation and coordination, and observation at multiple time points. A reduction in movement must not automatically be labelled anxiolysis or antidepressant activity.

Table 2: Summary of Selected Anxiolytic, Antidepressant and Neurobehavioral Studies

Research Domain	Representative Model / Study	Main Reported Observation	Evidence Level
Anxiety-like behaviour	Elevated plus maze, open field, light-dark box and Vogel conflict test in mice	Anxiolytic-like behavioural changes; antagonism by flumazenil/picrotoxin supported GABA-related involvement [4].	Preclinical animal study
Depression-like behaviour	Forced swim and tail suspension tests in mice	Reduced immobility; reduced brain MAO-A/MAO-B activity reported [5].	Preclinical animal study
Sleep-deprivation-associated behaviour	Forced swim, tail suspension and locomotor testing	Antidepressant-like signal reported with methanolic extract [8].	Preclinical report
Learning and memory	Passive avoidance and elevated plus maze paradigms	Improvement in selected memory outcomes and reversal of induced amnesia reported [10].	Preclinical animal study
Stress-related cognition	Chronic restraint stress and radial arm maze	Protection against stress-associated learning/memory deficits investigated [11].	Preclinical animal study
Major depressive disorder	Adjunctive jatamansi plus escitalopram, randomised double-blind study	Improvement in selected clinical and neurobiological measures reported [7].	Early clinical evidence
Post-stroke depression	Small randomised controlled study	Improvement in depression and quality-of-life measures reported [9].	Limited clinical evidence

INTEGRATED INTERPRETATION OF THE EVIDENCE

The available literature suggests that N. jatamansi may influence several interconnected systems rather than acting through a single pharmacological target. GABAergic signalling is most directly implicated in the anxiolytic-like study, while monoamine metabolism is implicated in the antidepressant-like mouse study. Antioxidant, anti-inflammatory and neurotrophic pathways may contribute to neuronal resilience and stress-associated outcomes. The convergence of these pathways is biologically plausible because mood, anxiety, cognition and stress adaptation depend on interacting neural circuits.

Nevertheless, mechanistic convergence should not be confused with proof of a multi-target therapeutic effect in humans. The studies use different extracts, doses, species and endpoints. The active constituents responsible for the reported behavioural effects remain incompletely identified, and the relationship between systemic exposure and central nervous system exposure is uncertain. Clinical evidence is emerging but remains too limited to define an effective dose, treatment duration, patient subgroup or interaction profile.

A useful translational model is to treat jatamansi as a candidate botanical whose efficacy depends on the quality and composition of the preparation. The chain of evidence should proceed from authenticated plant material to chemical fingerprint, in vitro target assays, pharmacokinetics, reproducible animal studies and adequately powered clinical trials. Failure at any stage should prompt reassessment rather than automatic progression to clinical claims.

SAFETY, TOXICITY AND HERB–DRUG INTERACTION CONSIDERATIONS

The absence of reported adverse effects in a small or short-duration study does not establish long-term safety. A botanical with central nervous system activity may cause sedation, dizziness, altered alertness or additive effects when combined with alcohol, sedatives, hypnotics, anxiolytics or other central nervous system

depressants. These are plausible precautions that require formal testing rather than confirmed interaction claims for every jatamansi product.

The reported MAO-related findings warrant particular attention. If a preparation meaningfully inhibits MAO in humans, concomitant use with serotonergic antidepressants, monoamine oxidase inhibitors or sympathomimetic medicines could create clinically important interactions. At present, the magnitude of this risk for standardised jatamansi products is not adequately defined. Patients should not add jatamansi to prescribed psychiatric treatment without discussing it with a qualified healthcare professional.

Future safety programmes should include acute and repeated-dose toxicity, genotoxicity where appropriate, reproductive and developmental toxicity, liver and kidney safety, cardiovascular monitoring, neurobehavioural safety and interaction studies. Product contaminants, heavy metals, pesticide residues, microbial load, adulterants and residual solvents should also be controlled. Safety conclusions must be tied to a defined product and dose, not generalised to all preparations bearing the name jatamansi.

QUALITY CONTROL, SUSTAINABILITY AND REGULATORY CHALLENGES

The therapeutic development of N. jatamansi depends on reliable botanical identity and sustainable supply. High-altitude medicinal plants may be vulnerable to habitat disturbance and unsustainable harvesting, particularly when underground parts are collected. Cultivation, community-based conservation, traceable sourcing and post-harvest standards should be integrated into product development. A pharmacologically promising plant cannot be considered a sustainable therapeutic resource if its supply chain damages wild populations.

Quality specifications should define the plant part, geographical origin, macroscopic and microscopic identity, chromatographic fingerprint, marker-compound range, moisture content, ash values, extractive values and limits for contaminants. Batch-to-batch consistency is essential for interpreting both clinical outcomes and adverse events. Regulatory classification and permitted claims vary by jurisdiction; traditional-use registration should not be represented as proof of efficacy for major depressive disorder or anxiety disorders.

RESEARCH GAPS AND FUTURE PERSPECTIVES

1 Standardised Extract Development

A priority is to develop a reproducible extract with a documented extraction process, chromatographic fingerprint and quantitative marker profile. Studies should compare multiple batches and report whether the pharmacological activity is retained after standardisation. The dose should be expressed both as extract mass and, where possible, as the amount of selected marker constituents.

2 Identification Of Active Constituents

Bioassay-guided fractionation, metabolomics and activity profiling can help identify constituents associated with anxiolytic-like and antidepressant-like outcomes. Isolated compounds should be evaluated at concentrations that are achievable after oral administration. Studies should also investigate whether combinations of constituents produce additive, synergistic or antagonistic effects.

3 Stronger Mechanistic Experiments

Mechanistic studies should combine receptor binding, functional assays, electrophysiology, enzyme assays and in vivo target engagement. GABA-A receptor subtype selectivity, GABA-B signalling, MAO-A/MAO-B activity, serotonergic and noradrenergic pathways, neuroinflammatory signalling and Nrf2-related antioxidant responses are reasonable priorities. Mechanistic claims should be supported by more than a single antagonist experiment or a change in a biochemical marker.

4 Improved Preclinical Design

Preclinical studies should be randomised, blinded and adequately powered, with pre-specified primary outcomes and transparent reporting of exclusions. Both sexes should be included where appropriate. Behavioural batteries should distinguish anxiety, depression-related coping, locomotion, sedation, sleep and cognition. Independent replication is particularly

important for findings based on a single laboratory or a single extract batch.

5 Clinical Trial Priorities

Clinical trials should evaluate a chemically standardised preparation, define the target diagnosis using accepted criteria, and use validated outcome scales. Trials should be sufficiently powered, registered prospectively and designed to assess both efficacy and safety. Adjunctive studies should specify background medication and monitor potential interactions. Longer follow-up is required to evaluate relapse, sustained benefit and delayed adverse events.

6 Pharmacokinetics And Brain Exposure

The pharmacokinetic profile of key constituents and metabolites remains a major knowledge gap. Oral absorption, metabolism, plasma protein binding, tissue distribution, blood-brain barrier penetration and elimination should be characterised. Linking exposure to behavioural response would clarify whether observed effects arise from direct central action, peripheral signalling or active metabolites.

7 Precision And Personalised Approaches

Future research may explore whether baseline symptom profiles, sleep disturbance, inflammatory markers, metabolic status or concomitant medication influence response. Such analyses should be exploratory until replicated and should not be used to market unvalidated personalised claims.

LIMITATIONS OF THE CURRENT EVIDENCE

The current evidence base is limited by heterogeneity in plant identity, plant part, extraction solvent, chemical composition, dose and duration. Many behavioural findings arise from small animal studies and use endpoints that only approximate components of human anxiety or depression. The number of controlled clinical investigations is limited, and existing trials involve specific contexts such as adjunctive treatment or post-stroke depression. Long-term safety, drug interactions, pharmacokinetics and the reproducibility of commercial products remain insufficiently characterised.

This review is narrative rather than systematic and does not provide a formal risk-of-bias assessment or pooled effect estimates. It should therefore be viewed as a critical overview of selected evidence, not a definitive evaluation of clinical efficacy. The bibliography should be checked against original publications before submission, especially for articles with online-first publication dates or incomplete metadata.

CONCLUSION

Nardostachys jatamansi is a traditional medicinal plant with a growing body of experimental research relevant to anxiety-like behaviour, depression-like behaviour, cognition and neuronal protection. Preclinical findings implicate GABAergic signalling in anxiolytic-like effects, monoamine oxidase and monoaminergic pathways in antidepressant-like effects, and antioxidant, anti-inflammatory and neurotrophic mechanisms in broader neurobehavioral outcomes. Early controlled clinical studies provide an initial translational signal, particularly for adjunctive use in depression and in post-stroke depression, but they are not sufficient to establish jatamansi as an independent treatment for psychiatric disorders.

The most important next step is not simply to conduct more studies with crude extracts, but to connect authenticated botanical material, reproducible chemical composition, pharmacokinetic exposure, validated mechanisms and well-designed clinical outcomes. Until larger trials and comprehensive safety studies are available, jatamansi should be regarded as a promising research candidate rather than a proven substitute for established treatment. Sustainable sourcing, rigorous quality control and transparent reporting will be essential for responsible development.

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