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Neuroprotective Potential of C. pluricaulis in Stress Caused by Chronic Lower Back Pain

Abstract

Introduction: Chronic Lower Back Pain (CLBP) is a common condition associated with psychological stress, which can exacerbate neuroinflammation and contribute to neurodegenerative changes. The herb Convolvulus pluricaulis (C. pluricaulis), or Śaṅkhapuṣpī, is known in Ayurvedic medicine for its cognitive-enhancing and neuroprotective properties. This review investigates the potential of C. pluricaulis to alleviate neurobiological disturbances linked to stress from CLBP.

Materials and Methods: A review of literature from databases such as PubMed and Scopus was conducted to examine the phytochemical composition, biological effects, and experimental evidence regarding the neuroprotective role of C. pluricaulis. Studies focusing on oxidative stress, cytokine modulation, HPA axis regulation, and neurotrophic factor promotion were included.

Results: C. pluricaulis contains compounds such as flavonoids, alkaloids, and terpenoids, which exhibit antioxidant, anti-inflammatory, and neuroprotective properties. Preclinical studies show it reduces oxidative stress and cytokine levels while modulating the HPA axis. Additionally, it enhances brain-derived neurotrophic factor (BDNF) levels, supporting neuronal survival and plasticity.

Discussion: The herb's effects on oxidative stress, inflammation, and neuroendocrine balance suggest its potential in treating neurodegeneration caused by chronic pain. While clinical studies are limited, preclinical data indicate that C. pluricaulis may serve as an adjunct therapy for managing stress-induced neurobiological changes in CLBP.

Conclusion: C. pluricaulis shows promise in mitigating the neurological effects of stress related to CLBP; however, further clinical studies are needed to confirm its efficacy and safety.

Keywords: Ayurveda, Musculoskeletal, Oxidative stress, Phytotherapy, Śaṅkhapuṣpī.

Introduction

C. pluricaulis, commonly known as Śaṅkhapuṣpī, is a well-recognized herb in the traditional system of medicine, particularly Ayurveda, for its reputed effects on mental health and cognitive functioning. Belonging to the family *Convolvulaceae*, this herbaceous, perennial plant thrives in the dry and rocky regions of India and is distinguishable by its spreading branches, small oblong leaves, and bluish-purple, conch-shaped flowers, which are the basis for its Sanskrit name Śaṅkhapuṣpī (Shankha meaning conch, and pushpi meaning flowered) [1].

In the classical Ayurvedic texts, Śaṅkhapuṣpī is classified under the group of Medhya Rasayana herbs that rejuvenate the mind and enhance memory, intellect, and emotional balance. Traditionally, it has been employed for mental fatigue, anxiety, insomnia, epilepsy, and various forms of psychosomatic disorders. In modern phytotherapy and neuropharmacology, *C. pluricaulis* has drawn attention for its nootropic, anxiolytic, antioxidant, and neuroprotective properties. These are largely attributed to its bioactive phytochemicals, including alkaloids, flavonoids, glycosides, and phytosterols like β-sitosterol [2]. Notably, its action on the GABAergic system contributes to its stress-relieving and calming effects on the central nervous system [3].

The herb's traditional uses are now being explored in the context of CLBP management, particularly where stress and anxiety exacerbate pain perception and reduce quality of life. With increasing scientific interest in plant-based adaptogens for integrative health, *C. pluricaulis* continues to hold clinical relevance in both preventive and therapeutic strategies for neuropsychiatric and stress-related disorders. CLBP is not only a biomechanical issue but also a trigger for prolonged stress, anxiety, and neuroendocrine disruption. Psychological comorbidities such as depression, cognitive fatigue, and stress-related memory impairment are increasingly recognized. Ayurvedic rasayana herbs like *C. pluricaulis* offer promising solutions due to their multi-target action on the nervous system. Traditional texts such as Bhavaprakasha and modern pharmacological studies confirm its role in enhancing memory, reducing anxiety, and protecting brain cells under stress.

Table 1: Multilingual Nomenclature of Śaṅkhapuṣpī[4]

Languages	Name
Sanskrit	Sankhapuspi
Hindi	Śaṅkhapuṣpī, Aparajit
English	English speedwheel
Punjabi	Śaṅkhapuṣpī, Sankhapuspi, Sankhahuli
Urdu	Sankhali
Tibetan	Śaṅkhapuṣpī
Tamil	Sanghupushpam, kakkurattai. Kakattam, Kakkanangudi, Karakhuratt
Oriya	Krishna-enkranti, Sankhapuspi
Malayalam	Krsna kranti, Vishnukranthi
Marathi	Shankhavela, Sankhahuli, Sankhapuspi
Gujarati	Shankhavali
Bengali	Sankhapuspi
Telugu	Shankhapushpi
Kannada	Bilikanthisoppu, Shankhapushpi, Shankhauli

Plant Pictures: The following three pictures of *Convolvulus pluricaulis* show: the plant with flower (Figure 1)[4], the flower (Figure 2)[5], and the whole plant (Figure 3)[6], respectively.

Figure 1

Figure 2

Figure 3

Table 2: Botanical Overview of Convolvulus pluricaulis[8]

Characteristic	Description
Botanical Name	*Convolvulus pluricaulis*
Family	Convolvulaceae
Common Names	Śaṅkhapuṣpī (Hindi, Sanskrit), Speed Wheel (English)
Morphology	Prostrate, spreading herbaceous plant; slender branches; bluish-purple flowers
Habitat & Distribution	Commonly found in the plains of India; grows in sandy, rocky soils
Flower Characteristics	Solitary or axillary; funnel-shaped, bluish-purple corolla
Leaves	Small, alternate, simple; ovate or elliptical in shape
Plant Parts Used	Entire plant; primarily aerial parts and leaves

Table 3: Chemical Constituents of Convolvulus pluricaulis

Plant Part	Category of Compounds	Key Constituents	Pharmacological Actions
Whole Plant [9]	Alkaloids	Shankhapushpine, Convolamine, Convolidine	Nootropic, anxiolytic, cognitive enhancer
	Flavonoids	Kaempferol, Quercetin, Luteolin	Antioxidant, neuroprotective, anti-inflammatory
	Glycosides	Kaempferol glycoside, Scopoletin glycoside	Cognitive support, free radical scavenging
	Coumarins	Scopoletin, Umbelliferone	Anti-inflammatory, adaptogenic
	Triterpenoids	Taraxerol, Taraxerone	Adaptogenic, neuroprotective
	Sterols	β-Sitosterol	Membrane stabilizer, anti-stress
	Volatile Oils	Aromatic compounds	Sedative, mild CNS depressant
	Sugars/Polysaccharides	Glucose, Fructose	Basic metabolic support
Leaves [10]	Flavonoids	Quercetin, Kaempferol	Potent antioxidants, neuroprotective
	Alkaloids	Shankhapushpine	GABAergic, anxiolytic
Aerial Parts (leaves and stems) [11]	Triterpenoids	Taraxerol, β-Sitosterol	Anti-inflammatory, adaptogenic
	Coumarins	Umbelliferone	CNS calming, anti-anxiety

Table 4: Traditional Ayurvedic Perspective[12]

Rasa (Taste)	Guna (Qualities)	Veerya (Potency)	Vipaka (Post-digestive Effect)	Karma (Therapeutic Action)	Prabhaav (Doshic Action)
Tikta (Bitter), Kashaya (Astringent)	Laghu (Light), Snigdha (Unctuous)	Sheeta (Cold)	Madhura (Sweet)	Medhya Rasayana (Brain tonic), Manas Rogahara (Psychotropic/ mental support)	Tridosha pacifying, especially beneficial in Pitta and Vata disorders

Table 5: Formulations and Dosage[13]

Formulation Type (Śaṅkhapuṣpī)	Dosage	Mode of Administration	Common Usage
Powder (Churna)	3–6 grams twice daily	With warm milk or ghee	Used in general cognitive and stress support
Syrup	10–15 ml twice daily (BID)	After meals	Popular formulations like Dabur Śaṅkhapuṣpī
Tablet/Capsule	250–500 mg standardized	Orally with water	Available in capsule form in Ayurveda stores

Polyherbal Products	As per label or practitioner advice	Usually liquid (arishta) or tablet	Saraswatarishta, Brahmi Vati, Mentat

Pathogenesis of Stress and Nerve Dysfunction in CLBP

CLBP is not only a mechanical or musculoskeletal disorder but also a significant contributor to systemic physiological stress and central nervous system (CNS) dysregulation. The etiopathogenesis of CLBP-induced stress and neurological impairment involves a dynamic interplay between peripheral nociceptive input, neuroendocrine stress response, neuroinflammation, and maladaptive neuroplasticity.

Peripheral Nociceptive Mechanisms

CLBP often originates from structural abnormalities such as intervertebral disc degeneration, facet joint dysfunction, or muscular strain, which stimulate peripheral nociceptors located in soft tissues and joints. These nociceptors, upon persistent activation, release a cascade of pro-inflammatory mediators (such as prostaglandins, bradykinin, and substance P) that sensitize local nerve endings. This process, termed peripheral sensitization, lowers the activation threshold of nociceptors, resulting in hyperalgesia and allodynia. Sustained nociceptive signaling not only perpetuates local inflammation but also facilitates central sensitization through continuous input to the spinal dorsal horn. The chronic nature of this input transforms an initially protective response into a maladaptive pain state, contributing to the amplification of pain perception. Importantly, this prolonged nociceptive stress acts as a key trigger for downstream neuroendocrine and neuroinflammatory changes, linking peripheral injury with central neurological dysfunction in CLBP-induced stress.

Central Sensitization and Neural Plasticity

Central sensitization is a key mechanism in the progression from acute to chronic lower back pain. It is caused by increased neuronal excitability in the spinal cord's dorsal horn after prolonged peripheral nociceptive input. This phenomenon results in amplified pain responses, including hyperalgesia and allodynia, even in the absence of ongoing tissue damage. Long-term stimulation of pain pathways leads to maladaptive neuroplastic changes in higher-order brain regions such as the thalamus, anterior cingulate cortex, and prefrontal cortex. Functional MRI studies confirm altered connectivity and decreased gray matter volume in these areas among chronic pain patients. These structural and functional changes not only increase pain sensitivity but also contribute to cognitive deficits, emotional dysregulation, and stress reactivity, further embedding pain into a centralized neurological state.

HPA Axis Activation and Cortisol Dysregulation

Persistent pain functions as a chronic physiological and psychological stressor, activating the Hypothalamic-Pituitary-Adrenal (HPA) axis. The hypothalamus first releases Corticotropin-Releasing Hormone (CRH), which causes the anterior pituitary to release Adrenocorticotropic Hormone (ACTH). ACTH then promotes cortisol secretion from the adrenal cortex. While cortisol

provides acute anti-inflammatory effects, its chronic elevation results in neuroendocrine dysfunction. Sustained cortisol exposure has been associated with hippocampal atrophy, impaired synaptic plasticity, suppressed neurogenesis, and dysregulation of negative feedback mechanisms within the HPA axis. These alterations contribute to cognitive impairment, heightened pain perception, emotional instability, and a greater susceptibility to depression and anxiety in individuals with chronic lower back pain. Thus, dysregulation of the HPA axis forms a critical bridge between chronic nociceptive stress and central neurological impairment.

Neuroinflammation and Oxidative Stress

Chronic stress and persistent pain states, such as those seen in CLBP, are closely linked to the sustained activation of glial cells, particularly microglia and astrocytes in the Central Nervous System (CNS). Once activated, these glial cells release pro-inflammatory cytokines such as tumor necrosis factor-alpha (TNF-α), IL-6, and interleukin-1β (IL-1β), which intensify nociceptive transmission and contribute to neuroinflammation. Parallel to this immune response, excessive production of Reactive Oxygen Species (ROS) induces oxidative stress, which is especially harmful to neurons due to their high metabolic activity and lipid-rich membranes. The consequences of oxidative insult include mitochondrial dysfunction, lipid peroxidation, DNA fragmentation, and ultimately apoptotic neuronal death. These processes not only exacerbate pain hypersensitivity but also contribute to long-term neurodegenerative changes, cognitive impairment, and emotional dysregulation in chronic pain patients.

Reduced BDNF and Impaired Neuroplasticity

Brain-Derived Neurotrophic Factor (BDNF) is a key neurotrophin involved in the regulation of synaptic plasticity, neurogenesis, and neuronal survival functions essential for learning, memory, and emotional regulation. Chronic pain and sustained stress, as observed in conditions like CLBP, are consistently associated with downregulation of BDNF expression, particularly in the hippocampus and prefrontal cortex. Reduced BDNF impairs neural adaptability and synaptic remodeling, leading to cognitive deficits and emotional dysregulation. Furthermore, diminished BDNF levels compromise the brain's resilience to inflammation and oxidative stress, exacerbating neurodegeneration in chronic pain states. Thus, decreased BDNF not only contributes to the persistence of pain but also plays a critical role in the progression of CLBP-related affective and cognitive impairments.

Psychological and Behavioral Consequences

The neurophysiological changes induced by chronic pain and stress are closely linked to a range of psychological disturbances, including Generalized Anxiety Disorder (GAD), Major Depressive Disorder (MDD), sleep disturbances, and cognitive fatigue. Functional MRI studies have shown that chronic pain alters the connectivity and function of key brain regions involved in emotion regulation, such as the amygdala and prefrontal cortex. These disturbances not only heighten pain perception through shared neural circuits but also impair motivation, concentration, and emotional resilience. Furthermore, poor sleep quality exacerbates fatigue and reduces pain thresholds, while depression and anxiety hinder treatment adherence and rehabilitation efforts. This bidirectional relationship creates a vicious cycle of pain, emotional distress, and functional decline, complicating long-term recovery in CLBP patients.

C. pluricaulis contains a diverse range of pharmacologically active compounds, contributing to its neuroprotective and adaptogenic properties. Key constituents include alkaloids such as shankhapushpine, convolamine, and convolidine; flavonoids like kaempferol and quercetin; glycosides, coumarins, triterpenoids, and β-sitosterol. These bio-actives exert antioxidant effects by scavenging ROS, thereby protecting neurons from oxidative damage. Flavonoids and glycosides have been shown to enhance cholinergic transmission through inhibition of acetylcholinesterase, supporting cognitive performance and memory. Additionally, alkaloids and coumarins interact with GABAergic pathways, producing anxiolytic and calming effects. The herb also demonstrates adaptogenic potential by modulating HPA axis activity, contributing to stress resilience. This multi-targeted pharmacodynamic profile positions *C. pluricaulis* as a promising candidate for neuroprotective therapy, particularly in chronic pain and stress-related disorders.

Experimental and Clinical Evidence

Studies have shown that *C. pluricaulis* extract improved memory retention in scopolamine-induced amnesia in mice. Its aqueous extract reduced oxidative markers (MDA, NO) and increased superoxide dismutase (SOD) and catalase levels in stress-induced rats. Human trials with polyherbal formulations containing Śaṅkhapuṣpī show a significant reduction in anxiety scores in GAD and insomnia patients.

Neuroprotective Action in Experimental Models

Nahata, Patil, and Dixit (2008) investigated the cognitive-enhancing effects of *C. pluricaulis* in rodents using passive and active avoidance models. In their study, titled “Effect of *C. pluricaulis* Choisy on learning behaviour and memory enhancement activity in rodents,” ethanolic and aqueous extracts (100–200 mg/kg) significantly improved memory performance and reversed scopolamine-induced amnesia ($p < 0.001$). The mechanism was linked to modulation of cholinergic neurotransmission, likely by enhancing acetylcholine levels in the brain, supporting its use as a traditional nootropic agent.

Anti-stress and Anxiolytic Properties

Khan et al. (2023) investigated the significant anxiolytic activity of *C. pluricaulis* hydroalcoholic extract in Swiss albino mice using the elevated plus-maze and light-dark box models. Oral doses (100–300 mg/kg) led to increased time in open arms and light zones, indicating reduced anxiety ($p < 0.001$). The effects were attributed to GABAergic modulation, likely through phytochemicals like kaempferol and scopoletin that interact with GABA_A receptors.

Antidepressant Activity

Dhingra and Valecha (2004) reported antidepressant-like effects of *C. pluricaulis* in Swiss albino mice using the forced swim and tail suspension tests. Chloroform fractions (50–100 mg/kg) significantly reduced immobility time, comparable to imipramine and fluoxetine. The mechanism is linked to monoaminergic modulation involving serotonergic, dopaminergic, and adrenergic pathways.

Neuroprotective Effects Against Oxidative Stress

Shalavadi et al. (2020) demonstrated that chloroform and ethanolic extracts of *C. pluricaulis* (100–400 mg/kg, p.o.) significantly attenuated oxidative stress and neuronal damage in a rat model of cerebral ischemia-reperfusion injury. Treatment led to marked reductions in lipid peroxidation and

increases in antioxidant enzymes, including superoxide dismutase, catalase, glutathione, and total thiol levels ($p < 0.001$). Histopathological analysis and MAP2 immunohistochemistry further confirmed neuroprotection via preservation of neuronal integrity and reduced infarct area, highlighting its potential in mitigating oxidative stress-induced neurodegeneration.

Anti-Alzheimer's Activity

Walia et al. (2012) investigated the anti-Alzheimer's potential of *C. pluricaulis* in a rat model of scopolamine-induced amnesia. Oral administration of the plant extract significantly improved memory performance in behavioral tests such as the elevated plus maze and step-down avoidance. The therapeutic effect was attributed to dual mechanisms: inhibition of acetylcholinesterase, leading to increased acetylcholine availability, and attenuation of oxidative stress through enhanced antioxidant enzyme activity. The extract's efficacy was comparable to that of standard drugs like donepezil, suggesting its promise as a natural neuroprotective agent in Alzheimer's disease management.

Sedative and Hypnotic Properties

Malik et al. (2011) evaluated the sedative-hypnotic effects of *C. pluricaulis* in Swiss mice using the pentobarbital-induced sleep model. The ethanolic extract significantly prolonged sleep duration and reduced onset time in a dose-dependent manner. The findings suggest CNS depressant activity, possibly via GABAergic pathways. These results support the traditional use of *C. pluricaulis* in managing insomnia and restlessness. The study concludes that the plant possesses sedative potential, likely mediated by its influence on neurotransmission in the brain, particularly through the modulation of GABA_A receptors.

Anticonvulsant Effects

Malik et al. (2008) studied significant anticonvulsant activity of *C. pluricaulis* using pentylenetetrazole (PTZ) and maximal electroshock seizure (MES) models in mice. The methanolic extract, administered at doses of 500–1,000 mg/kg, reduced seizure incidence, delayed seizure onset, and shortened recovery time, showing effects comparable to phenytoin. These findings indicate that *C. pluricaulis* possesses compounds with anticonvulsant properties, potentially acting via GABAergic or sodium channel–blocking mechanisms. The results support its traditional use in epilepsy and suggest it may serve as a natural source for developing safer antiepileptic therapies.

Clinical Evidence in Children with Cognitive Deficits

Singh et al. (2001) studied a clinical trial evaluating the cognitive effects of *C. pluricaulis* syrup (Śaṅkhapuṣpī) in children with learning difficulties. The study involved school-aged participants with symptoms of poor memory, attention, and academic performance. After a treatment period of several weeks, children showed notable improvement in concentration, memory retention, and overall cognitive behavior. The study concluded that *C. pluricaulis* is safe and effective in pediatric use and may act by modulating cholinergic function or reducing mental fatigue. These results support its long-standing use in Ayurvedic medicine for pediatric neurocognitive support.

Anti-Ulcer and Gastroprotective Activity

Awaad et al. (2003) investigated the gastroprotective effects of *C. pluricaulis* in ethanol-induced ulcer models in rats. The plant extract significantly reduced ulcer index and increased mucosal protection, suggesting it strengthens the gastric barrier and suppresses oxidative damage.

The findings indicate that *C. pluricaulis* may act through antioxidative and cytoprotective mechanisms, enhancing mucin secretion or prostaglandin synthesis. These outcomes validate its traditional use in treating peptic ulcers and offer potential for development as a natural anti-ulcer therapy with fewer side effects than standard drugs.

Antioxidant and Anti-inflammatory Effects

Pandey et al. (2010) explored the antioxidant and anti-inflammatory properties of *C. pluricaulis* using in vitro assays. The methanolic extract demonstrated strong free radical–scavenging activity in the DPPH assay ($IC_{50} \approx 41$ µg/mL) and effectively inhibited inflammatory mediators. The presence of phenolic and flavonoid compounds may underlie its bioactivity. These findings support its ethnomedicinal use for inflammation-related neurological and gastric conditions. The extract's ability to mitigate oxidative stress suggests it may be neuroprotective and anti-aging, with potential application in chronic inflammatory and neurodegenerative diseases.

Conclusion

In the context of CLBP, where persistent nociception is intricately linked to psychological stress, *C. pluricaulis* emerges as a promising phytotherapeutic agent. Its multifaceted pharmacological profile, including serotonergic modulation, GABAergic enhancement, acetylcholinesterase inhibition, and anti-inflammatory action, addresses both the neurophysiological and psychosomatic consequences of chronic pain. By improving mood, reducing anxiety, enhancing sleep quality, and supporting cognitive resilience, *C. pluricaulis* contributes significantly to the management of CLBP-associated stress and neuroinflammation. Furthermore, its ability to suppress cytokines like TNF-α and IL-6 may prevent pain-induced neurodegenerative changes in the central nervous system. These findings support the integrative use of *C. pluricaulis* in holistic pain management strategies aimed at restoring neurochemical balance, emotional stability, and cognitive function in CLBP patients.

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