ISSN 2582-6441 [Online]
RESEARCH JOURNAL OF PHARMACY AND LIFE SCIENCES
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Review Article

Methodical Investigation into the Pain- Relieving Effectiveness of *Tecoma Stans* Plant

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ARTICLE INFO

Date of submission: 15-06-2025
Date of Revision: 17-07-2025
Date of acceptance: 20-07-2025

Key Words:
Self-Nanoemulsifying Drug Delivery Systems, Nanoemulsions, Bioavailability

ABSTRACT

The current research focuses on the Anti-nociceptive activities of *Tecoma stans* plant. Natural botanicals are presently used in therapy more often than synthetic products since synthetic medications can have a variety of negative effects. *Tecoma stans* is a plant found in most tropical countries. This plant, which is a member of the Bignoniaceae family, has long been utilized in traditional Indian and Pakistani medicine. This plant has a large number of active chemical ingredients and has pharmacological effects. The different plant parts were found to have a variety of pharmacological actions, including acetylcholinesterase inhibitory activities, anti-inflammatory, analgesic, anticancer cardioprotective effect, genotoxic, cytotoxic, wound healing, antihyperglycemic, protect central nervous system, gastric ulcer healing, antiproliferative, antioxidant, antimicrobial, haemolytic activity, and anti-lipoxygenase. *Tecoma stans*, a member of the trumpet vine family, has a number of pharmacological characteristics that have been reported.

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INTRODUCTION

Tecoma stans has traditionally been used as a source of medicine contributing to human health and well-being [1]. Traditional herbal remedies are used by almost 80% of people worldwide, particularly in underdeveloped nations. Due to increased exploitation, environmentally unfriendly harvesting methods, habitat degradation, and unmonitored handling of medicinal plants, the genetic biodiversity and variety of traditional medicinal herbs are frequently extinct [2].

Tecoma stans is a medicinal herb that can be used to treat microbiological infections, diabetes mellitus, and gastrointestinal tract issues. It can also be used as a powerful tonic, vermifuge, and diuretic [3]. Traditional folk remedies use the leaves, bark, and root extracts because they contain physiologically active phytoconstituents. Numerous active components, including alkaloids, flavonoids, tannins, quinones, and traces of saponins and amino acids, were found in the initial phytochemical examination [4].

TECOMA STANS PLANT PROFILE

Tecoma stans generic name is derived from the word tecomaxochit, which indigenous Mexican peoples used to refer to plants with tubular flowers. These plants are also called yellow bells, yellow-elder, yellow trumpet bush, trumpet bush, Ginger-thomas, Esperanza, and Tronadora [5].

Figure1: *Tecoma stans* Plant

Tecoma stans are the plants which belongs to the family of Bignoniaceae that is having many synonyms and common names such as Bignonia stans, Stenolobium stans, Gelseminum stans, Kuntze Seem [5].

The genus Tecoma includes fourteen species of small trees or shrubs that belong to the Bignoniaceae family, which includes trumpet vines. While two species are found in Africa, twelve species are found in the Americas [6]. Although its natural habitat stretches from southern Texas, New Mexico, and Arizona to Bolivia and northern Argentina, as well as from Florida and the Bahamas to Trinidad in the Caribbean, it is found across South America and India. Additionally, [7] it is grown in tropical and subtropical regions of Australia, Asia, Africa, and the Pacific Islands [8].

Found in a bit salted and soils that are acid *Tecoma stans* blooms from embryos and the color green cuttings. The plant requires around two years to bloom. Clustered elongated fruits, yellow flowers, green compound, imparipinnate, lanceolate leaves with a serrated margin of two to five pairs of leaflets with an extra-large single terminal leaflet, and a slow growth rate of about twenty to thirty meters per year are some of the physical traits of *Tecoma stans*. The soft-touch, mid-green, lanceolate leaflets have serrated edges and extend around 10 cm in length[9].

At the extremities of the branches, trumpet-shaped clusters of flowers with five rounded lobes that range in colour from light to bright yellow are displayed. Fruits are around 20 cm long, narrow, slightly flattened to pointy capsules, and many winged, young, green seeds that turn pale brown when they ripen are kept in untidy clusters on the tree for months. These blossoms are frequently pollinated by bees and hummingbirds[10].

There are several other informal names for *Tecoma stans*. It's called Piliya or Pilakaner in Hindi. The most commonly used term for yellow bells is in English. It is referred to as Koranekelar in Kannada and Sonnapatti in Tamil. The name is Chandaprabha in Bengali and Pachagotla in Telegu. *Tecoma stans* are referred to as Ghantiful in Marathi[11].

CHEMICAL COMPONENTS

The leaf, bark, fruit, root, and flowers are just a few of the plant parts that contain several different types of chemicals. Several phytochemical studies are used to determine these components. The active chemical components are extracted using various solvents[7].Phytochemical studies demonstrate the presence of alkaloids, phenols, terpenoids, glycosides, flavonoids, saponins, carbohydrates, amino acids, phytosterols, monoterpenes, triterpenes, fixed oils, fats, gums, mucilage, resins, volatile oils, quinines, and tannins[12].

The sugars include fructose, sucrose, xylose, and glucose. Likewise, ursolic and oleanolic acids, along with amyrine, are triterpenoids. Chlorogenic, caffeic, vanillic, o-cumaric, and sinapicn acids are among the phenolic compounds found in the plant. Anthrallic acid, a crucial chemical component, is also present in *Tecoma stans*[5]. The alkaloids identified are tecomine and tecostamine[13]. Another alkaloid is 4-noractinidine. The leaves frequently contain these alkaloids. Several chemical substances can be extracted from leaves, including flavonoids, chryseriol, luteolin, indole oxygenase, boschniakine, 5-hydroxyskitanthine, 4-noractinidine, and 4-norskytanthine.The flowers may contain carotene. Different flavonones can also be extracted from the flowers. Kaempeferol

and 7,8-dihydroxy-4,6-dimethoxy flavones are two examples[14]. Lipids and fatty acids are found in seeds. Ingredients include stearic acid, palmitic acid, octadecenoic acid, octadecadieonic acid, and octadecatetraonic acid. Ethanolic preparations of flowers exhibit the presence of tecomanine and monoterpenoids[15].

TAXONOMICAL CLASSIFICATION

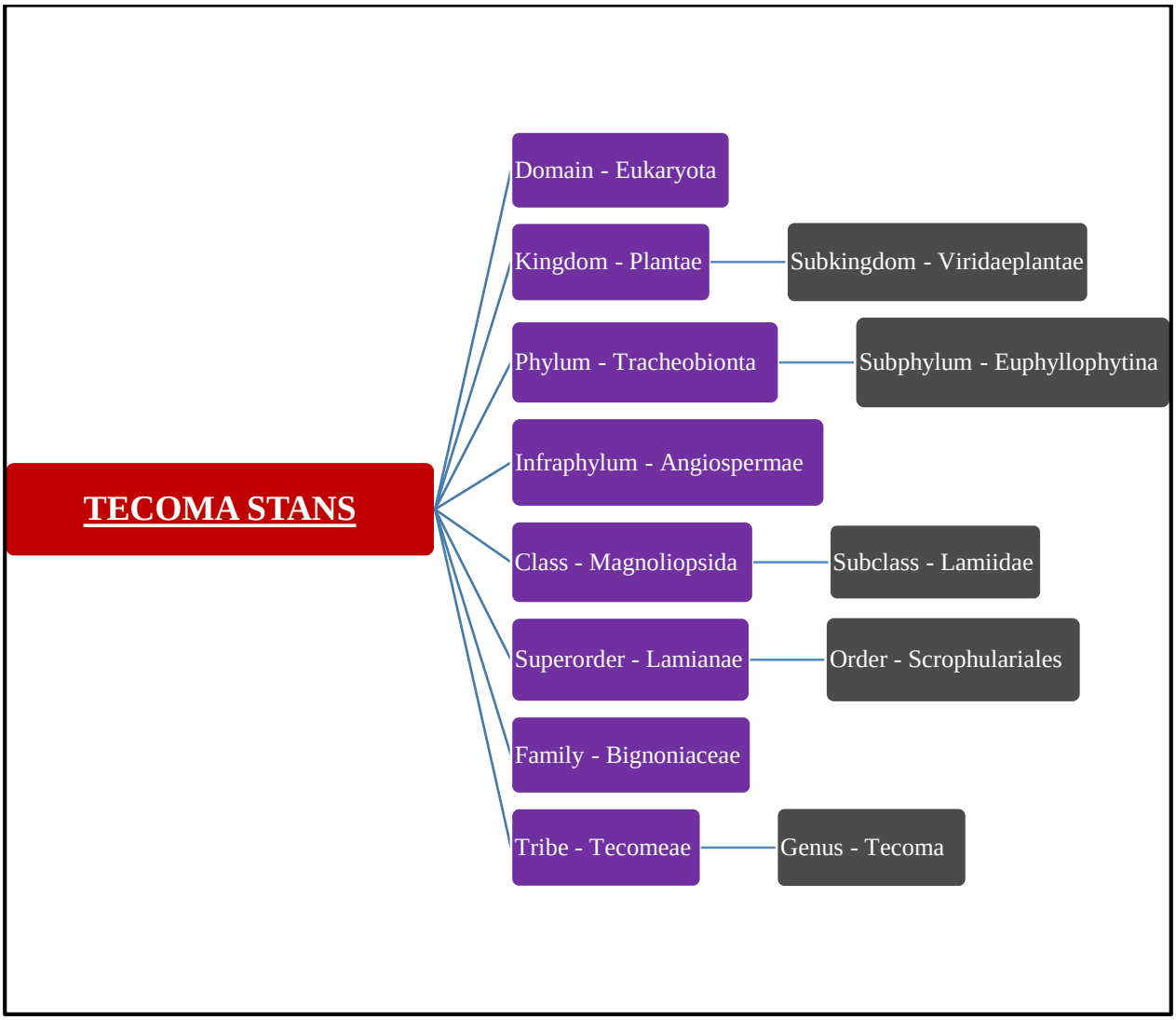

Figure 2: Taxonomical Classification of *Tecoma stans*

PHYSIOLOGY AND MECHANISM OF NOCICEPTION

Nociception is the process by which noxious (potentially tissue-damaging) stimuli are detected and transmitted to the brain. Specialized sensory neurons called nociceptors (free nerve endings) respond to mechanical, thermal, or chemical insults by generating action potentials. First-order (peripheral) nociceptors have pseudounipolar cell bodies in dorsal root (or trigeminal) ganglia and send one axon branch into the periphery (detecting injury) and one branch into the spinal cord[16].

When activated, nociceptors release excitatory neurotransmitters (primarily glutamate and substance P) onto second-order neurons in the dorsal horn.

These second-order neurons decussate and ascend via the spinothalamic tracts to the thalamus, and third-order thalamocortical neurons then project to somatosensory cortex for conscious pain perception[17].

Nociceptors and Peripheral Transduction

Nociceptors are unmyelinated or lightly myelinated free nerve endings located in skin, muscle, joints, and viscera (not in brain tissue). They transduce various harmful stimuli via specialized receptors and ion channels[18]. For example, TRP (transient receptor potential) channels (e.g. TRPV1) mediate heat and capsaicin responses, acid-sensing ion channels (ASICs) respond to pH changes, mechanosensitive channels detect stretch, and other chemoreceptors respond to histamine, bradykinin or prostaglandins released after injury. Different nociceptors are classified by modality[19]:

- **Mechanical nociceptors** – activated by intense pressure or stretch.
- **Thermal nociceptors** – activated by extreme heat or cold.
- **Chemical nociceptors** – activated by irritant chemicals (e.g. acid, histamine, bradykinin).
- **Polymodal nociceptors** – respond to combinations of mechanical, thermal, and chemical stimuli.

These receptors have peripheral receptive fields (e.g. smaller on fingertips for high acuity). In addition, injury releases inflammatory mediators (arachidonic acid metabolites, potassium, 5-HT, histamine, bradykinin, ATP, protons) that sensitize nociceptors (lower their activation threshold)[20]. For example, prostaglandins generated via COX enzymes do not directly cause pain but potentiate other stimuli on nociceptors[21]. Two main fibre types transmit the nociceptor signal: fast myelinated Aδ fibers and slow unmyelinated C fibers. **Aδ fibers** have medium diameter and relatively low thresholds. They conduct rapidly, producing the sharp, well-localized “first” pain and terminate in Rexed lamina I of the spinal dorsal horn, releasing glutamate. **C fibers** are small, unmyelinated, and have higher thresholds. They conduct slowly, producing a dull, burning “second” pain; they terminate mainly in lamina II (substantia gelatinosa) and release neuropeptides (substance P, CGRP) in addition to glutamate[22].

Spinal Cord Processing and Ascending Pathways

In the ventral posterolateral (VPL) nucleus of the thalamus, these signals synapse onto

third-order neurons, which project via the internal capsule to the primary somatosensory cortex for localization of pain. This three-neuron chain (peripheral → spinal → thalamocortical) ensures that noxious stimuli evoke rapid protective reflexes and conscious sensation[23].

Upon tissue damage, primary afferents release excitatory neurotransmitters in the dorsal horn: glutamate (via AMPA/NMDA receptors) is the principal fast excitatory transmitter, and substance P (via neurokinin-1 receptors) prolongs depolarization and promotes inflammation[24]. Glutamate rapidly depolarizes second-order neurons, while substance P enhances firing and can cause slow synaptic currents. These transmitters open ligand-gated ion channels on postsynaptic neurons, generating graded potentials that may trigger second-order action potentials if threshold is reached. Inhibitory interneurons in the dorsal horn (using GABA and glycine) can dampen this signalling, whereas descending facilitation (e.g. from brainstem serotonin or noradrenaline neurons) can enhance it. Overall, the synchronized synaptic transmission in the dorsal horn (and subsequent relay in brainstem/thalamus) constitutes the ascending pain pathway[25].

Cellular and Molecular Mechanisms

At the molecular level, nociceptor membranes contain many ion channels and receptors that transduce and modulate pain signals. Voltage-gated sodium channels (e.g. Nav1.7, Nav1.8) underlie action potential generation in nociceptors. Notably, gain-of-function mutations in Nav1.7 cause inherited erythromelalgia (excess pain), and loss-of-function causes congenital insensitivity to pain. TRP channels (TRPV1, TRPA1, TRPM8, etc.) respond to heat, chemicals, and cold by gating cation influx and depolarizing the nerve ending. ASIC channels respond to acid (protons), while P2X receptors respond to extracellular ATP from injured cells[26].

Once opened, these channels allow influx of sodium ions and calcium ions, generating the receptor potential. If this depolarization reaches threshold, it triggers a train of action potentials that travels along the fiber. The frequency of spikes encodes stimulus intensity, and the pattern recruits both Aδ (fast) and C (slow) fibers for the complex pain sensation. Central sensitization can occur when persistent C-fiber input leads to up regulation of NMDA glutamate receptors on second-order neurons: Magnesium ions block is relieved, and excessive calcium ions influx results in long-term potentiation of the pain synapse. This produces hyperalgesia (increased response to painful stimuli) and

allodynia (pain from normally non-painful stimuli) in chronic pain states[27].

Inflammatory mediators (e.g. bradykinin, histamine, prostaglandins, and cytokines) play dual roles: they sensitize peripheral nociceptors and induce local vasodilation. For example, cyclooxygenase (COX) enzymes convert arachidonic acid to prostaglandins which do not directly generate pain but lower nociceptor thresholds. Similarly, ATP and protons from ischemic tissue or cell lysis activate purinergic and acid-sensing channels. These mediators can also increase vascular permeability and recruit immune cells, creating a pro-nociceptive milieu[28].

Taken together, nociceptive signalling involves multiple molecular players: peripheral transducers (ion channels on nociceptors), synaptic neurotransmitters (glutamate, substance P, CGRP) and neuromodulators (GABA, glycine) in the spinal cord, and long-range tracts to the brain[1, 29].

In this diagram, nociceptor afferents (red) are shown releasing glutamate and substance P onto dorsal horn neurons, which ascend the spinothalamic tract. Neurotransmitter receptors (e.g. NMDA/AMPA, NK1) and second messenger cascades (e.g. phosphorylation of ion channels) amplify or dampen the signal at the cellular level. Clinically, aberrations in these molecular mechanisms (e.g. excess glutamate, loss of inhibition, channelopathies) underlie pathological pain conditions and guide target-specific therapies[30].

Descending Modulation and Endogenous Opioids

The central nervous system exerts powerful descending control over nociception, largely via endogenous opioid pathways. Higher brain centers (perigenual anterior cingulate, hypothalamus) and pain-related areas (periaqueductal gray – PAG, rostral ventromedial medulla – RVM) send projections to the dorsal horn that inhibit ascending transmission[31].

Activation of PAG neurons (e.g. by stress or analgesics) triggers the release of endorphins and enkephalins onto interneurons in the RVM and spinal cord. These endogenous opioids bind to μ, δ, and κ opioid receptors (allG protein-coupled receptor (GPCR) that are associated with G_i or G_o proteins) on pre- and post-synaptic membranes in lamina.Receptor activation causes presynaptic inhibition which results into decreased calcium ions influx and reduced neurotransmitter release from C/Aδ terminals) and postsynaptic hyperpolarization (allowing potassium ions to flow out of the cell, effectively reducing membrane excitability)[32].

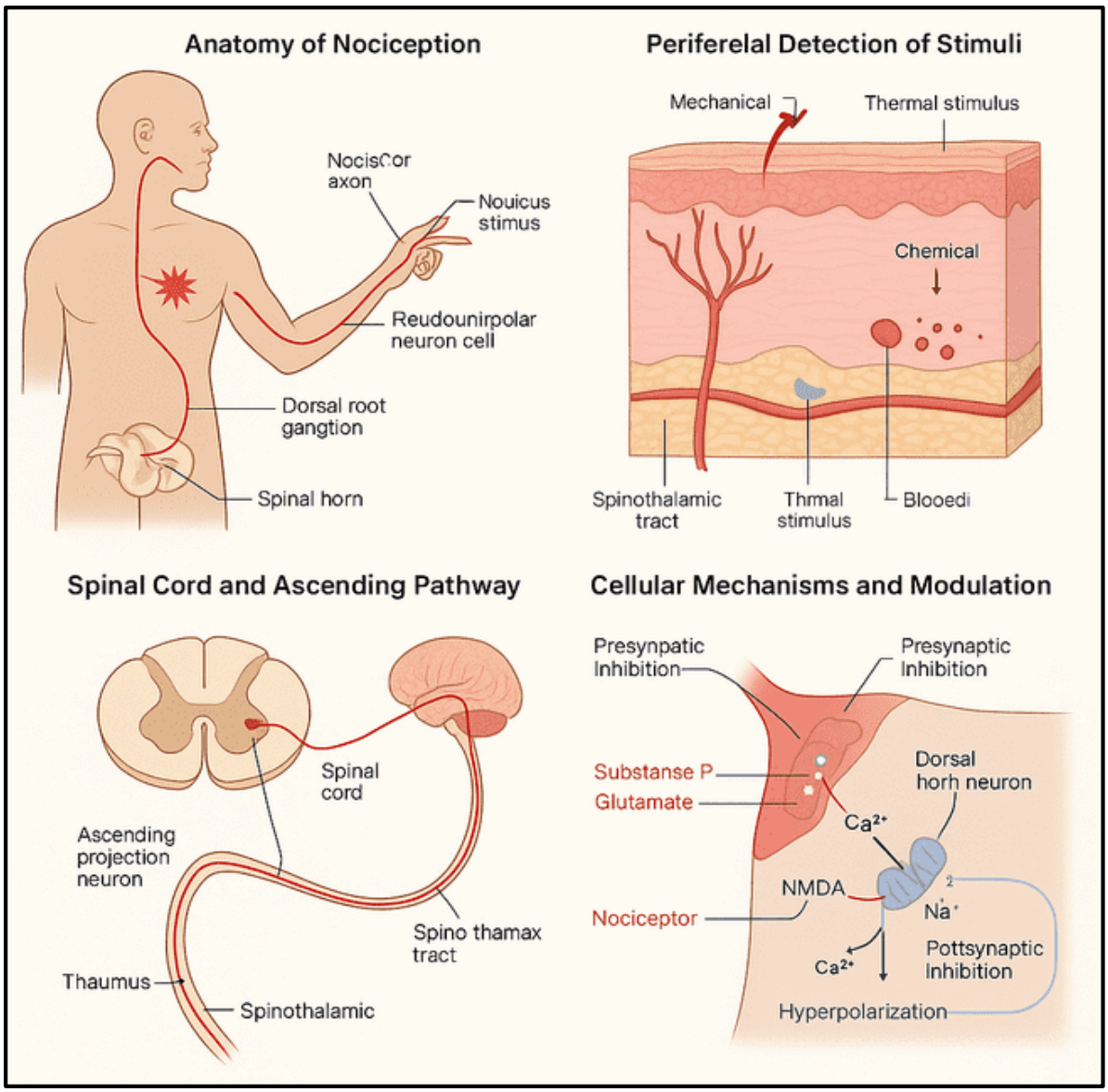

Figure 3: Mechanism of *Tecoma stans*

TECOMA STANS PLANT FOR ANTI-NOCICEPTIVE EFFECT

Tecoma stans, also referred to as yellow trumpet bush or yellow bells, is a flowering plant historically utilized in folk medicine for diverse applications. Its antinociceptive (pain-relieving) qualities have been the subject of recent pharmacological research, which attributes them to its abundant phytochemical composition. Here is a thorough synopsis[13]:

- **Traditional Uses:** *Tecoma stans* has been used in traditional medicine for:
  - Diabetes management (hypoglycaemic effects)
  - Stomach disorders
  - Anti-inflammatory purposes

- Fever and pain relief

Mechanisms of Antinociceptive Action

1. Inhibition of Inflammatory Mediators

- Down regulation of prostaglandin synthesis via cyclooxygenase (COX) inhibition.
- Reduced release of inflammatory cytokines such as TNF-α, IL-1β, and IL-6[33].

2. Modulation of Oxidative Stress

- Antioxidant constituents (e.g., flavonoids) reduce oxidative stress, which plays a key role in chronic pain and neuroinflammation[34].

3. Interaction with Opioid Receptors

- Some animal studies suggest partial mediation via opioid pathways, as pre-treatment with naloxone (opioid antagonist) attenuates the antinociceptive effects.
- This implies central modulation via μ-opioid receptors[35].

4. Inhibition of Nitric Oxide (NO) Pathways

Reduction in nitric oxide synthase (NOS) activity leads to decreased NO levels, which are known to contribute to pain signalling in the spinal cord[36].

5. Experimental Evidence

Animal Studies:

Several preclinical studies (mostly in mice and rats) have shown:

- **Significant reduction** in acetic acid-induced writhing (a model for visceral pain).
- **Increased latency** in the hot plate test (indicating central analgesic activity).
- **Reduced licking time** in the formalin test (both neurogenic and inflammatory phases)[37].

These effects are dose-dependent and comparable in efficacy to standard analgesics like diclofenac or morphine in some studies[38].

6. Clinical Relevance and Future Directions

- **Potential applications**: management of inflammatory pain, neuropathic pain, and adjunct use in chronic pain syndromes.
- **Advantages**: natural origin, antioxidant benefits, multi-targeted mechanisms.
- **Challenges**: lack of human trials, standardization of extract composition, and potential herb-drug interactions[39].

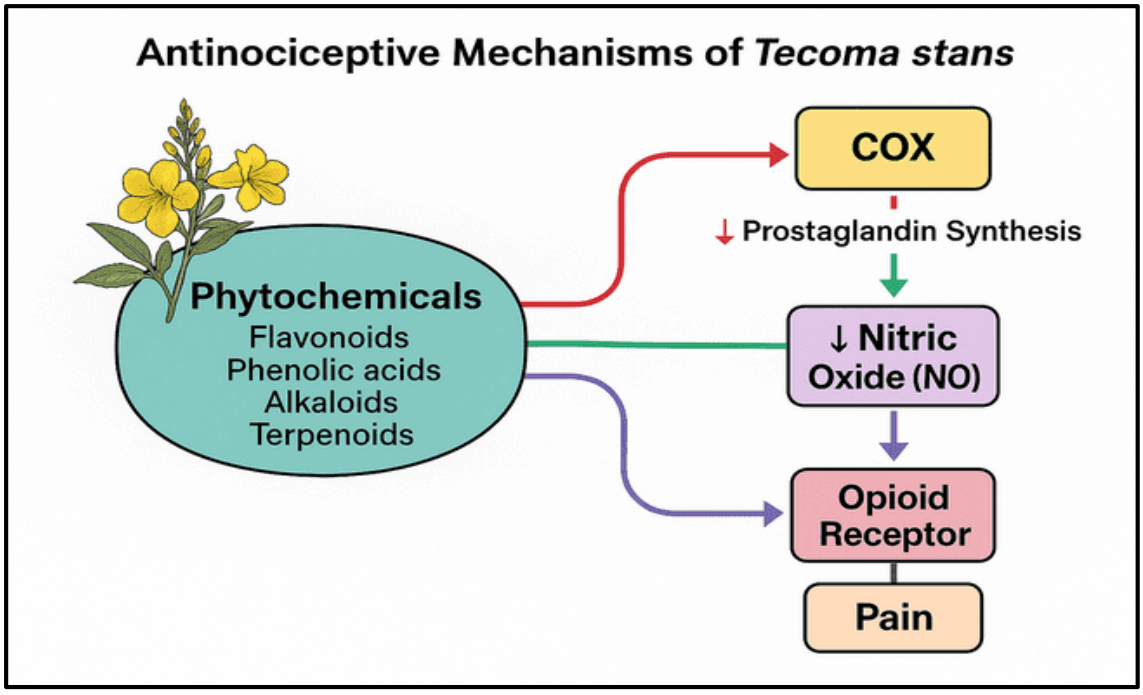

Figure 4: Antinociceptive Mechanisms of *Tecoma stans*

Additional Significant or Different Plants/Herbs or Agents with Anti-Nociceptive Properties

A wide range of medicinal plants and natural agents have demonstrated **antinociceptive** (pain-relieving) effects through various mechanisms, including inhibition of inflammatory mediators, modulation of ion channels, and interaction with neurotransmitter systems. Here's a detailed list categorized by mechanism and with potential clinical relevance[29]:

Table 1: Active Compounds and Mechanism of Action of different plants

Plant Name	**Active Compounds**	**Mechanism of Action**	**Evidence/Models**
Curcuma longa (Turmeric)	Curcumin	Inhibits COX-2, NF-κB; antioxidant activity	Formalin, writhing, and hot plate tests
Zingiber officinale (Ginger)	Gingerols, shogaols	COX inhibition, TRPV1 modulation	Clinical and animal studies
Salix alba (White willow bark)	Salicin	Inhibits prostaglandin synthesis (COX pathway)	Basis for aspirin; historical and clinical use
Cannabis sativa	THC, CBD	CB1/CB2 receptor modulation; anti-inflammatory	Human and preclinical models
Capsicum spp. (Chili pepper)	Capsaicin	TRPV1 agonist → desensitization	Topical analgesics;

			neuropathic pain treatment
Mitragyna speciosa (Kratom)	Mitragynine	Opioid receptor agonism	Preclinical studies and ethnobotanical use
Ocimum sanctum (Holy basil)	Eugenol, ursolic acid	Central and peripheral analgesic effects	Animal pain models
Piper nigrum (Black pepper)	Piperine	Modulation of TRP channels, antioxidant	Thermal and chemical nociception models

Natural Compounds or Nutraceutical Agents

- **Central Nervous System Modulation**:
    - Opioid receptor agonism (e.g., Kratom, Cannabis)
    - GABAergic enhancement (e.g., melatonin, valerian)
    - TRP channel modulation (e.g., capsaicin, piperine)[40]
- **Peripheral Mechanisms**:

Cyclooxygenase inhibition (e.g., turmeric, willow bark)

    - Inhibition of pro-inflammatory cytokines (e.g., IL-6, TNF-α)
    - Reduction in reactive oxygen species (e.g., flavonoids, thymoquinone)[41]
- **Ion Channel Effects**:
    - Sodium, potassium, calcium channel modulation
    - TRPV1, TRPA1, TRPM8 agonise or antagonism

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Mechanisms of Antinociceptive Action

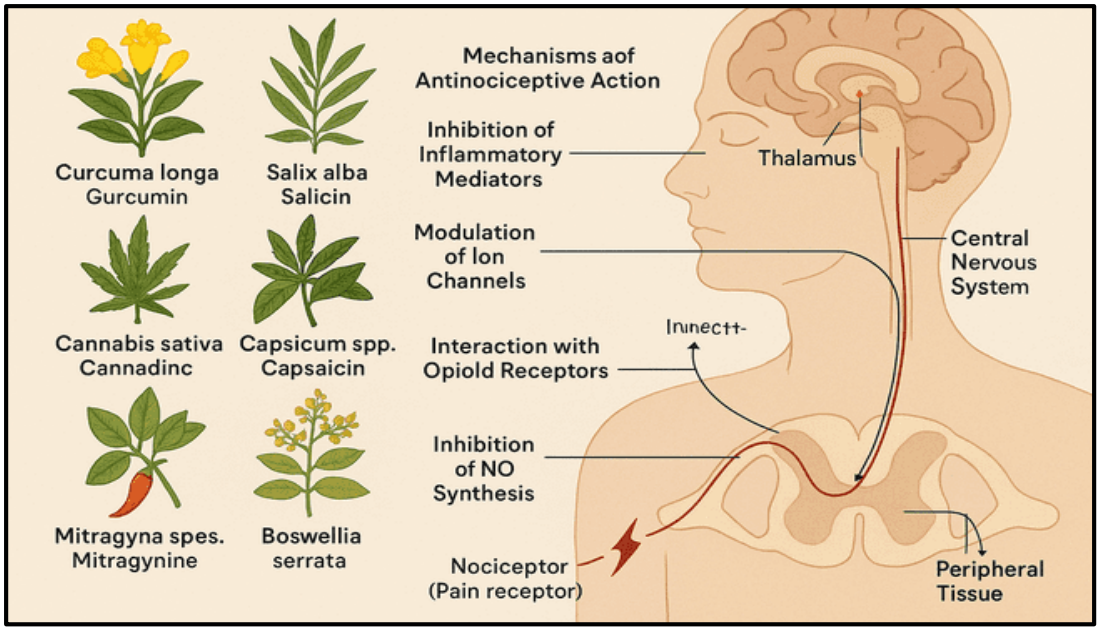

Figure 5: Plants with antinociceptive effects

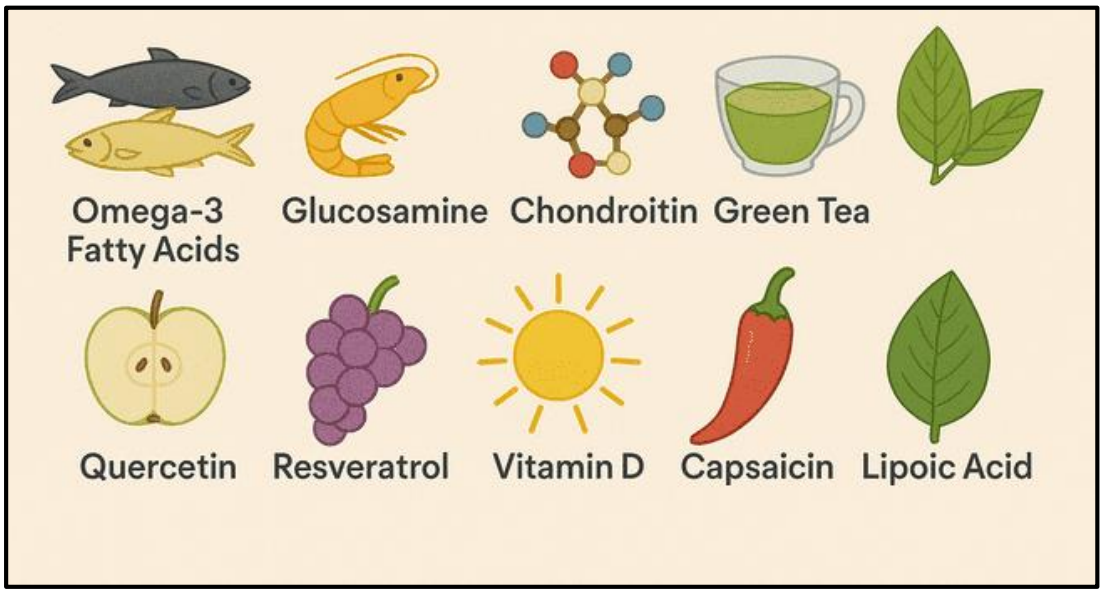

Figure 6: Neutraceutical agents with antinociceptive properties

Table 2: Neutraceutical compounds with their source, mechanism and uses

Compound	Source	Mechanism	Uses
Resveratrol	Grapes, berries	SIRT1activation, COX inhibition	Neuropathic pain, inflammation
Quercetin	Onions, apples	Mast cell stabilization, antioxidant	Antiallodynic, anti-hyperalgesic
Omega-3 fatty acids	Fish oil	Anti-inflammatory eicosanoids	Reduces chronic pain states
Palmitoylethanolamide (PEA)	Endogenous fatty acid amide	PPAR-α activation, mast cell modulation	Chronic pelvic pain, neuropathy

Berberine	Berberis spp.	Modulation of opioid and TRP channels	Useful in diabetic neuropathy
Melatonin	Pineal gland / dietary	Inhibits NO and cytokines, modulates GABAergic system	Effective in fibromyalgia, migraine

PHARMACOLOGICAL ACTIONS OF *TECOMA STANS*

Tecoma stans have been used as medicine since ancient times. Numerous medical applications exist for it. It was and still is acknowledged to be effective in treating diabetes.

Tecoma stans are made up of several chemical components that have different pharmacological effects. To illustrate *Tecoma stans*' pharmacological activities, numerous studies have been carried out [7,42].

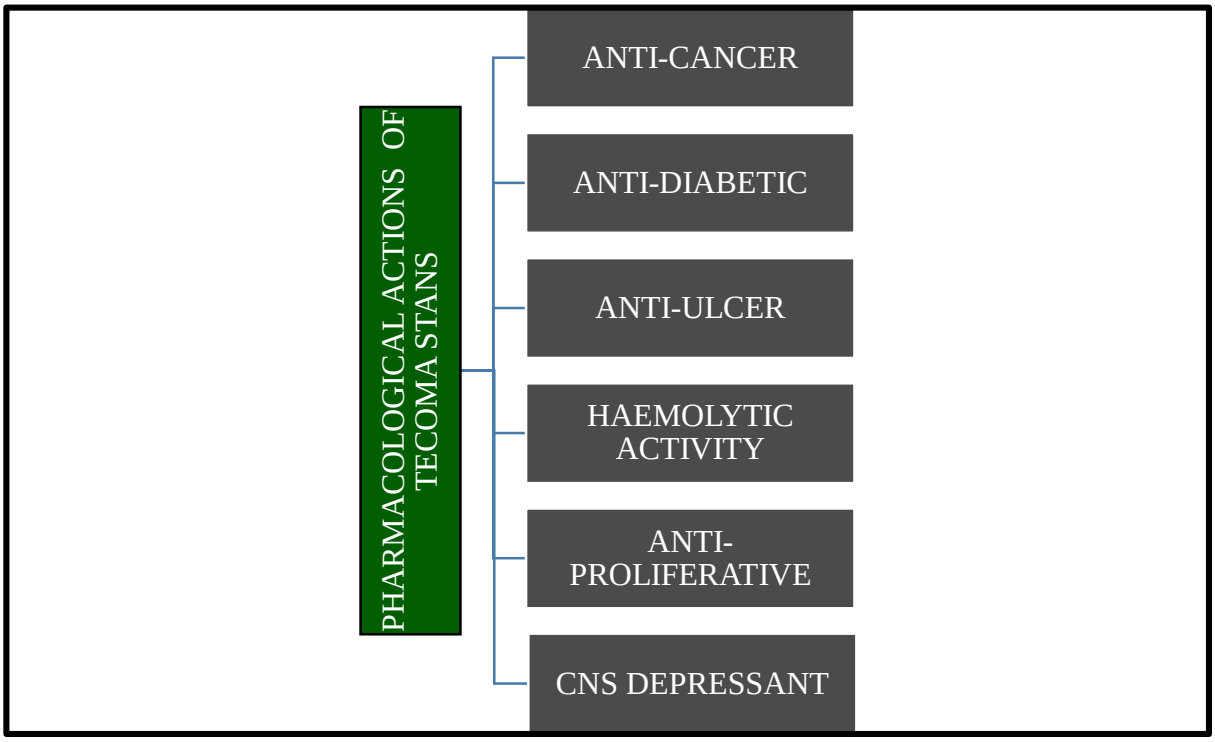

Figure 7: Pharmacological action of *Tecoma stans*

➢ ANTI-CANCER PROPERTIES

Uneven cell development is a hallmark of cancer, a horrible disease with over 100 different types that calls for a multipronged approach to management, prevention, and therapy[14]. It is also the second leading cause of death worldwide. Breast cancer is the most frequently diagnosed malignancy in women and is regarded as one of the chronic illnesses that they may experience (32.1 percent) during their lifetimes[43]. The

antiproliferative effect of various *Tecoma stans* components in breast cancer-MCF-7 cell lines is evaluated using the MTT assay. The *Tecoma stans* stem bark extract demonstrated the strongest anti-proliferative effect on the cell lines (MCF-7), but the bark, stem, root, and flower extracts all exhibited significant anti-proliferative activity[43, 44]. *Tecoma stans* contain flavonoids that have anticancer effects. Consuming a lot of flavonoids has been demonstrated to lower your risk of developing cancer. Flavonoids function by slowing the initiation, growth, and spread of tumours. Numerous investigations demonstrate the participation of multiple modes of action. Among the mechanisms of action are angiogenesis suppression, antiproliferation and differentiation, induction of apoptosis, cell cycle arrest, carcinogen inactivation, and reversal of multidrug resistance[45].

- **ANTI-MICROBIAL POTENCY**

Alcoholic and aqueous extracts of *Tecoma stans* exhibit antibacterial properties. Compared to leaf extracts, stem and bark extracts have a stronger antibacterial effect. Flavonoids are primarily responsible for *Tecoma stans*' antibacterial activity. Antimicrobial activity is mostly evaluated using the disc diffusion technique. Microbial diseases are responsible for a large number of global health problems[46]. Using plant sources as antimicrobial agents is essential for microorganism healing because manmade antimicrobial drugs and antibiotics may have unfavourable side effects. Agar medium, minimum inhibitory concentrations, and the disc diffusion method are some of the techniques used in antimicrobial testing on plant extracts. This approach uses both positive and negative organism strains as well as fungal strains to stop microbial growth, which is stopped by adding medications. The antibacterial activity was ascertained by computing the zone of inhibition and comparing it to the standard[47].

- **THE HAEMOLYTIC ACTIVITY**

The Haemolytic test, the Minimum Inhibitory Concentration method, and drop diffusion are used to quantify haemolytic activity. The result is determined by the zone of inhibition. The process that damages the cytoplasmic membrane and causes cell lysis and death is called haemolysis. Haemolytic activity is measured using the following method. Heparin, an anticoagulant, is placed in tubes containing human blood from healthy volunteers in the A, B, and O groups. Following three minutes of centrifugation at 3,000 rpm, the hRBCs were collected. Until the supernatant is colorless, the cells are repeatedly washed

with PBS solution[48]. The haemolytic test was performed in a microwell plate. PBS is put into each well. Additionally, ABCs were inserted into the wells. Serially diluted peptide solutions are added to the proper wells. While the hRBCs in Triton function as a positive control, the hRBCs in Triton function as a negative control. After an hour of incubation, the well's button formation is observed[49].

- **NEUROPHARMACOLOGY OF *TECOMA STANS***

Situations, although mice or rats are frequently used to test the central analgesic activity. Haffner's tail clip method in mice, tail flick or other radiant heat methods, hot plate methods in mice or rats, electrical stimulation (such as grid shock or stimulation of the tooth pulp or tail), monkey shock titration, and the formalin test in rats are a few techniques used to evaluate central analgesic activity[50].

- **WOUND HEALING ACTIVITY**

Tecoma stans has shown promising wound healing properties due to its rich content of flavonoids, tannins, and other antioxidants. These compounds help reduce inflammation, promote tissue regeneration, and speed up healing. Studies suggest its extracts support faster wound closure, making it a potential natural remedy for skin repair and care[3].

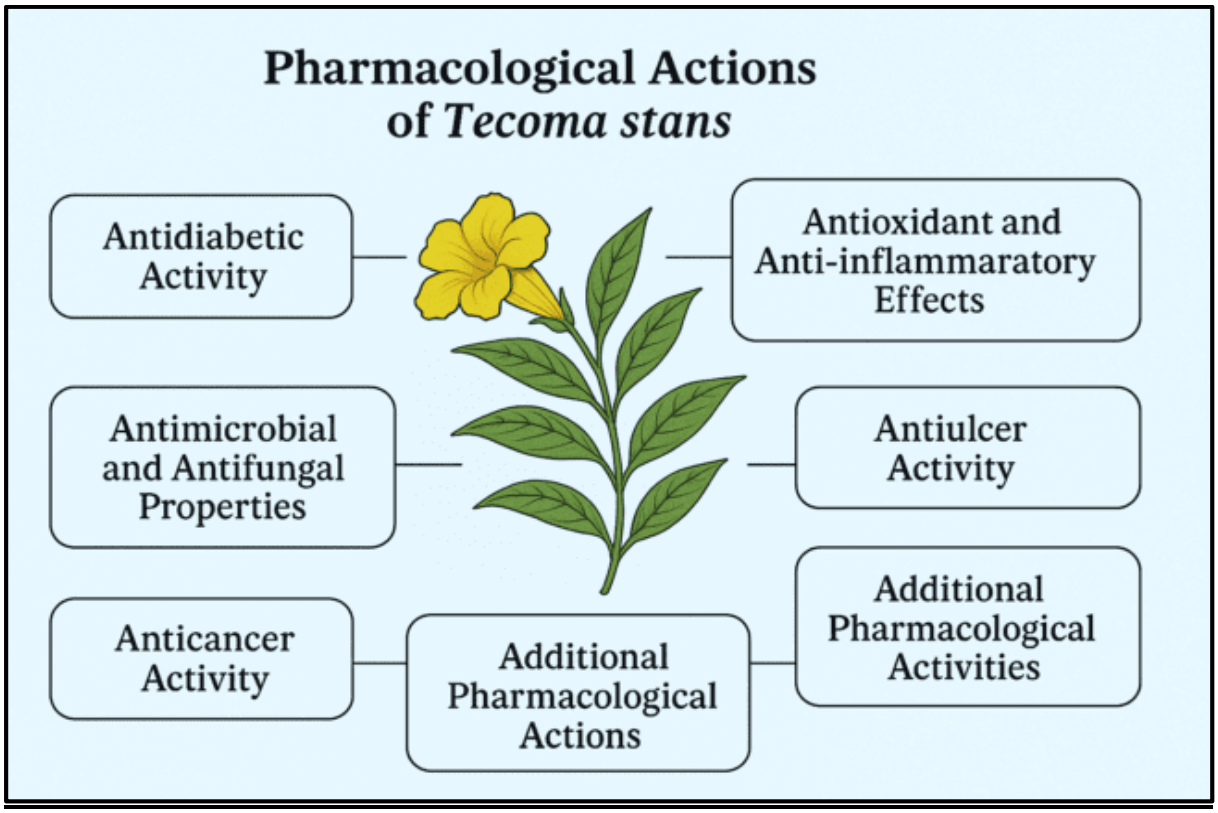

Figure 8: Pharmacological Action of *Tecoma stans*

CONCLUSION

The convergence of ethno pharmacology and modern neuroscience provides a fertile ground for developing safer, multi-targeted antinociceptive therapies from nature. With advancing research, phytochemicals and nutraceuticals could redefine pain management in both acute and chronic conditions. Based on the findings, it can be concluded that the methanolic extract of *Tecoma stans* floral buds exhibits significant antinociceptive activity, likely due to the presence of bioactive compounds such as flavonoids and triterpenoids. These results support its traditional use in pain management. Further pharmacological investigations and clinical studies are recommended to explore its full therapeutic potential and to isolate the specific compounds responsible for its analgesic effects.

CONFLICT OF INTEREST:

There is no such conflict of interest related to this investigation.

ACKNOWLEDGEMENTS:

This review article has got completed due to my parents who kept motivating me and finally we sincerely thank Prof. Rajaram Das Sir for his incredible guidance, constant support, and valuable insights throughout the preparation of this review article. Like to thank the almighty because of whose blessings this all had become possible.

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