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THERAPEUTIC POTENTIAL OF *ACHYRANTHES ASPERA* IN INFLAMMATORY LUNG DISEASES: PHYTOCHEMISTRY, MOLECULAR MECHANISMS, AND PHARMACOLOGICAL EVIDENCE

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ABSTRACT

Asthma, chronic obstructive pulmonary disease (COPD), acute lung injury (ALI), acute respiratory distress syndrome (ARDS), and pulmonary fibrosis are among the most common causes of morbidity and mortality worldwide. Persistent airway inflammation, oxidative stress, immunological dysregulation, and progressive lung tissue destruction that impairs respiratory function are the hallmarks of these illnesses. While traditional treatments like immunomodulators, bronchodilators, and corticosteroids are useful in treating symptoms, long-term use of these medications is frequently linked to side effects and reduced effectiveness in certain people. Plant-derived medicines with anti-inflammatory and antioxidant qualities are therefore gaining popularity. *Achyranthes aspera* L. (Amaranthaceae) is a medicinal herb commonly used in traditional medicine to treat respiratory diseases, inflammation, asthma, bronchitis, and cough. Triterpenoid saponins, flavonoids, alkaloids, phenolic compounds, terpenoids, ecdysteroids, and other bioactive phytochemicals with a variety of pharmacological actions are abundant in the plant. Through the regulation of important inflammatory mediators and signaling pathways, such as NF-κB, MAPK, COX-2, iNOS, and pro-inflammatory cytokines, *Achyranthes aspera* has been shown in experimental tests to have considerable anti-inflammatory, antioxidant, immunomodulatory, and antiallergic properties. The botanical traits, phytochemical components, molecular mechanisms, pharmacological data, and therapeutic potential of *Achyranthes aspera* in inflammatory lung disorders are summarized in this review. Additionally, it draws attention to research gaps and future directions for the development of phytotherapeutics based on. *Achyranthes aspera* for inflammatory respiratory illnesses.

KEYWORDS: *Achyranthes aspera*; Inflammatory lung diseases; Respiratory inflammation; Asthma; Chronic obstructive pulmonary disease (COPD); Acute lung injury; Pulmonary fibrosis; Phytochemistry; Anti-inflammatory activity; Oxidative stress.

INTRODUCTION

Asthma, chronic obstructive pulmonary disease (COPD), acute lung injury (ALI), acute respiratory distress syndrome (ARDS), and pulmonary fibrosis are all major causes of morbidity and mortality around the world. Persistent inflammation, oxidative stress, excessive production of pro-inflammatory cytokines, and increasing structural damage to lung tissues are the hallmarks of these illnesses, which impede respiratory function and lower quality of life. Corticosteroids, bronchodilators, and immunomodulatory drugs are examples of modern therapeutic approaches that successfully relieve symptoms, but their long-term usage is frequently linked to side effects, decreased therapeutic response, and expensive treatment costs. In order to prevent and treat respiratory inflammatory illnesses, there is a growing interest in finding natural compounds with strong anti-inflammatory and antioxidant properties.[1, 2]

The chemical variety and multi-target pharmacological activity of medicinal plants have long made them ideal sources of bioactive molecules for drug discovery. By controlling important molecular pathways involved in inflammation, plant-derived phytochemicals such flavonoids, saponins, alkaloids, phenolic compounds,

iuand terpenoids have shown notable anti-inflammatory, antioxidant, and immunomodulatory effects. Herbal remedies have the potential to reduce pulmonary inflammation and oxidative damage by modulating signaling pathways like nuclear factor-kappa B (NF-κB), mitogen-activated protein kinase (MAPK), cyclooxygenase-2 (COX-2), inducible nitric oxide synthase (iNOS), and pro-inflammatory cytokines, according to recent pharmacological studies.[2,3]

Achyranthes aspera L. (family Amaranthaceae), sometimes known as prickly chaff flower, is a medicinal herb found in tropical and subtropical climates. Asthma, bronchitis, cough, fever, inflammation, skin conditions, and gastrointestinal ailments have all been treated using the plant in traditional medical systems including Ayurveda, Siddha, and Unani. Numerous physiologically active components, including as triterpenoid saponins, flavonoids, alkaloids, steroids, glycosides, phenolic compounds, ecdysteroids, oleanolic acid, and ursolic acid, have been discovered by phytochemical studies. Numerous pharmacological actions, including as anti-inflammatory, antioxidant, antibacterial, antiallergic, immunomodulatory, and bronchodilatory effects, are caused by these phytoconstituents.[1, 4]

Extracts from *Achyranthes aspera L* have been shown in experiments to reduce the generation of inflammatory mediators, prevent oxidative stress, stabilize mast cells, and alter a number of inflammatory signaling pathways linked to respiratory disorders. According to these results, the plant may be useful in treating inflammatory lung conditions by lowering the generation of cytokines, decreasing the infiltration of inflammatory cells, and shielding lung tissues from oxidative damage. Nevertheless, there are still few clinical trials assessing its effectiveness and safety in respiratory conditions, despite promising preclinical data. Therefore, more research is needed to standardize plant extracts, pinpoint active ingredients, clarify molecular pathways, and demonstrate clinical efficacy through well planned human trials.[1, 3, 5]

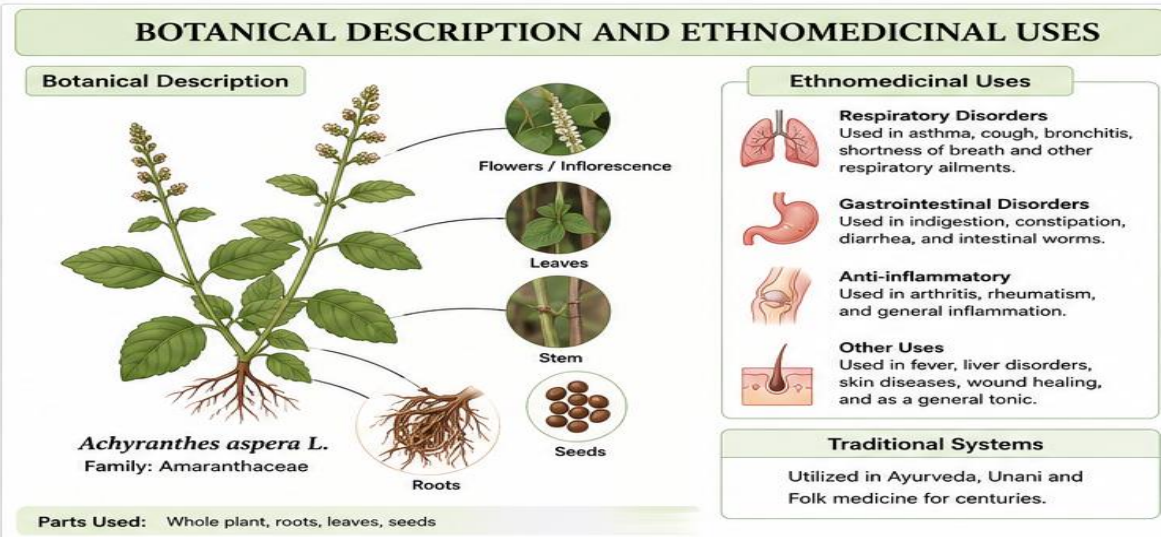
The current review highlights current understanding on the phytochemistry, molecular mechanisms, pharmacological data, and therapeutic potential of *Achyranthes aspera* in inflammatory lung disorders. Additionally, it addresses current research gaps and potential directions for the development of phytopharmaceuticals based on *Achyranthes aspera* as supplemental therapeutic agents for inflammatory respiratory illnesses.

BOTANICAL DESCRIPTION AND ETHNOMEDICINAL USES

Achyranthes aspera L. is an erect, annual or perennial herb from the Amaranthaceae family. It is frequently found in tropical and subtropical areas of Asia, Africa, Australia, and the Americas and is commonly referred to as prickly chaff flower in English. The plant frequently grows as a weed in meadows, open woods, roadsides, wastelands, and agricultural areas. It is renowned for its broad ecological distribution, drought tolerance, and extreme environmental adaptability.[6]

Botanical Description

The plant has an upright, branching, cylindrical stem covered in tiny hairs, and it typically grows to a height of 0.5 to 2.0 meters. The leaves are oblong to elliptic, simple, opposite, petiolate, and have a hairy surface and full edge. They are around 3 to 10 cm long. Long terminal or axillary spikes with many tiny greenish-white bisexual flowers make up the inflorescence. Persistent, spine-like bracts subtend each bloom and help spread seeds by adhering to human and animal fur or clothing. The taproot is cylindrical, yellowish-white, and has distinctive medicinal qualities; the fruits are utricles with a single smooth, reddish-brown seed.[7]



Taxonomically, *Achyranthes aspera* L belongs to the Kingdom Plantae, Division Magnoliophyta, Class Magnoliopsida, Order Caryophyllales, Family Amaranthaceae, Genus Achyranthes, and Species Achyranthes aspera L. The plant is commonly referred to by a variety of colloquial names, such as Apamarga (Sanskrit), Chirchita (Hindi), Nayuruvi (Tamil), Uttareni (Telugu), Apang (Bengali), and Kadaladi (Malayalam), which reflects its extensive use in traditional medicine over several geographical regions.[8]

Table 1: Taxonomical Classification of *Achyranthes aspera* L.[6]

Taxonomic Rank	Classification
Kingdom	Plantae
Subkingdom	Tracheobionta (Vascular plants)
Superdivision	Spermatophyta (Seed plants)
Division	Magnoliophyta (Angiosperms)
Class	Magnoliopsida (Dicotyledons)
Subclass	Caryophyllidae
Order	Caryophyllales
Family	Amaranthaceae
Genus	*Achyranthes*
Species	*Achyranthes aspera* L.

Ethnomedicinal Uses

Achyranthes aspera has been widely used in several indigenous healthcare practices as well as traditional medical systems like Ayurveda, Siddha, and Unani. The roots, leaves, stalks, seeds, flowers, and the entire plant are all utilized to treat a variety of illnesses. The plant has historically been used as an antibacterial, expectorant, diuretic, anti-inflammatory, antiasthmatic, analgesic, antipyretic, wound-healing, and immunomodulatory agent. Asthma, bronchitis, pneumonia, and other respiratory tract diseases have all been treated with decoctions and extracts of the roots and leaves in respiratory medicine. The plant's wide ethnopharmacological significance is further demonstrated by its use in the treatment of arthritis, skin conditions, gastrointestinal issues, urinary tract infections, diabetes, hemorrhoids, snakebite, insect bites, and fever.[9]

PHYTOCHEMICAL CONSTITUENTS OF *ACHYRANTHES ASPERA*

Achyranthes aspera L. contains a high concentration of physiologically active secondary metabolites, which contribute to its various pharmacological effects. Numerous phytoconstituents, including triterpenoid saponins, alkaloids, flavonoids, phenolic compounds, steroids, terpenoids, glycosides, tannins, ecdysteroids, polysaccharides, and essential minerals, have been found in the roots, stems, leaves, seeds, flowers, and entire plant, according to phytochemical investigations. The plant part, location, harvest season, and extraction technique all affect the qualitative and quantitative content of these chemicals.[10]

Triterpenoid saponins are thought to be the main bioactive components of *A. aspera* among the ingredients that have been found. These saponins have important hepatoprotective, immunomodulatory, antibacterial, anti-inflammatory, and antioxidant qualities. According to reports, they reduce nuclear factor-kappa B (NF-κB), inducible nitric oxide synthase (iNOS), and cyclooxygenase-2 (COX-2) signaling pathways to inhibit inflammatory mediators. By lowering oxidative stress and cytokine production, two significant pentacyclic triterpenoids extracted from the plant, oleanolic acid and ursolic acid, further enhance its anti-inflammatory and antioxidant properties.[11]

Flavonoids and phenolic compounds are another major family of phytochemicals found in *A. aspera.* By boosting natural antioxidant enzymes including glutathione peroxidase (GPx), catalase (CAT), and superoxide dismutase (SOD), these substances have potent free radical scavenging action and shield cells from oxidative damage. Additionally, flavonoids reduce tissue damage linked to chronic inflammatory diseases, such as respiratory ailments, by regulating inflammatory pathways and inhibiting lipid peroxidation.[12]

The plant also includes ecdysteroids, particularly 20-hydroxyecdysone (ecdysterone), which is of great pharmaceutical interest due to its antioxidant, anti-inflammatory, adaptogenic, and anabolic effects. Additionally, several solvent extracts of *A. aspera* have been found to contain alkaloids, glycosides, steroids, amino acids, carbohydrates, proteins, and tannins. Together, these phytochemicals support the plant's immunomodulatory, antibacterial, analgesic, antidiabetic, and wound-healing properties. Synergistic interactions between these phytoconstituents may improve the plant's overall therapeutic efficacy when compared to isolated substances, according to recent metabolomic studies.[13]

Table 2: Major Phytochemical Constituents of *Achyranthes aspera* L. and Their Pharmacological Activities.

Phytochemical Class	Major Phytoconstituents	Plant Part	Reported Pharmacological Activities	Reference
Triterpenoid saponins	Oleanolic acid saponins, Achyranthosides	Whole plant, Roots, Seeds	Anti-inflammatory, antioxidant, immunomodulatory, hepatoprotective	[10]
Triterpenoids	Oleanolic acid, Ursolic acid	Leaves, Roots	Anti-inflammatory, antioxidant, anti-fibrotic	[10], [11]
Ecdysteroids	20-Hydroxyecdysone (Ecdysterone), Ecdysone	Roots, Whole plant	Adaptogenic, antioxidant, anti-inflammatory, anabolic	[10], [13]

Flavonoids	Quercetin, Kaempferol derivatives	Leaves	Antioxidant, anti-inflammatory, free radical scavenging	[12]
Phenolic compounds	Gallic acid, Caffeic acid, Ferulic acid	Leaves, Whole plant	Antioxidant, antimicrobial, anti-inflammatory	[12], [14]
Alkaloids	Achyranthine	Roots, Whole plant	Cardioprotective, anti-inflammatory, antihypertensive	[10], [13]
Steroids	β-Sitosterol, Stigmasterol	Leaves, Seeds	Anti-inflammatory, hypolipidemic, antioxidant	[11], [14]
Terpenoids	Various mono- and triterpenoids	Whole plant	Anti-inflammatory, antimicrobial, analgesic	[10], [11]
Glycosides	Saponin glycosides	Whole plant	Immunomodulatory, anti-inflammatory	[13]
Tannins	Hydrolysable and condensed tannins	Leaves, Bark	Antioxidant, antimicrobial, wound healing	[12]
Amino acids	Arginine, Lysine, Glycine	Whole plant	Nutritional support, metabolic regulation	[13]
Carbohydrates	Reducing sugars, Polysaccharides	Whole plant	Energy source, immunomodulatory activity	[11]
Proteins	Structural proteins	Seeds	Nutritional and physiological functions	[13]
Fatty acids	Palmitic acid, Oleic acid, Linoleic acid	Seeds	Anti-inflammatory, membrane stabilization	[14]
Minerals	Potassium, Calcium, Magnesium, Iron, Zinc	Whole plant	Enzyme activation, antioxidant defense, metabolic regulation	[13]

Advances in chromatographic and spectroscopic techniques, such as high-performance liquid chromatography (HPLC), gas chromatography-mass spectrometry (GC-MS), liquid chromatography-mass spectrometry (LC-MS), nuclear magnetic resonance (NMR), and Fourier-transform infrared spectroscopy (FTIR), have enabled the identification and characterization of numerous bioactive constituents in *A. aspera*. The quality control, standardization, and authentication of herbal formulations meant for pharmaceutical purposes depend on these analytical techniques. The discovery of pharmacologically active compounds supports *A. aspera's* prospective development as a source of innovative phytopharmaceuticals for inflammatory illnesses and offers a scientific foundation for its traditional use.[14]

PATHOPHYSIOLOGY OF INFLAMMATORY LUNG DISEASES

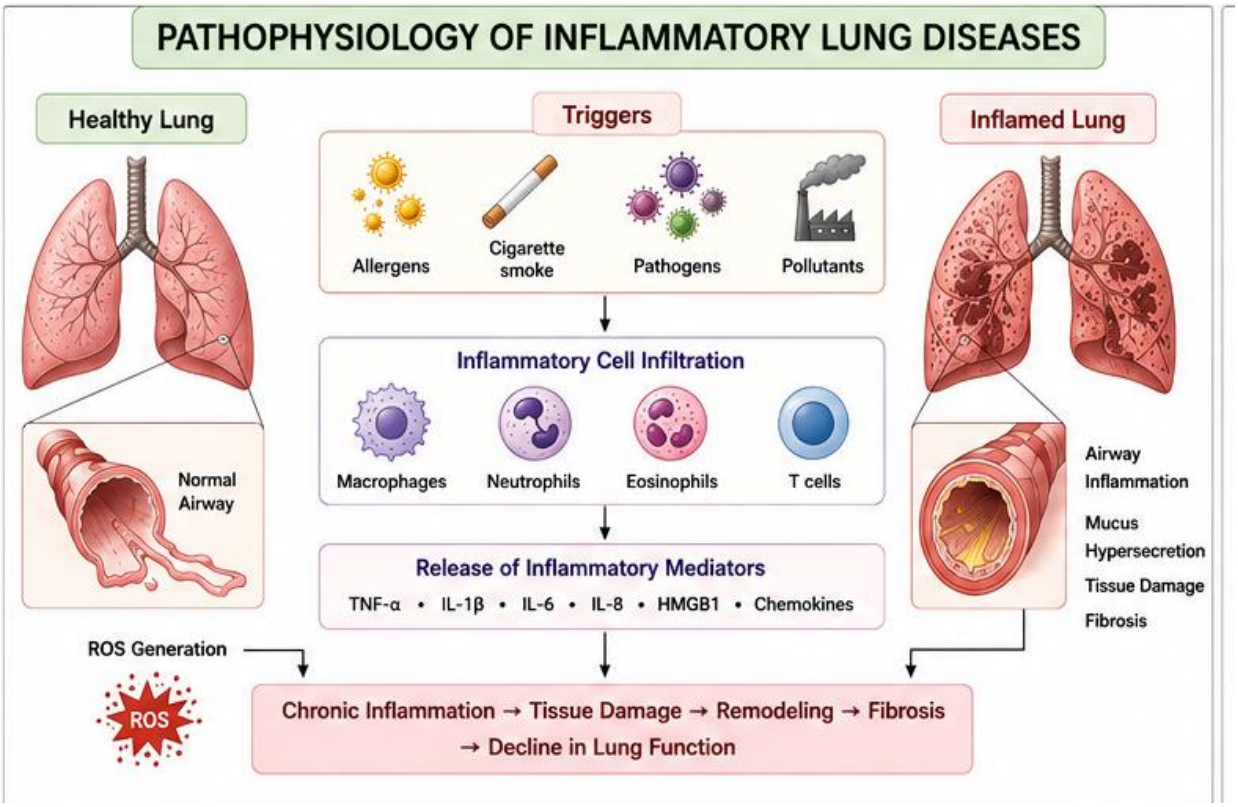
Acute lung injury (ALI), acute respiratory distress syndrome (ARDS), chronic obstructive pulmonary disease (COPD), asthma, and pulmonary fibrosis are examples of inflammatory lung diseases that are marked by immunological dysregulation, oxidative stress, and progressive lung architecture destruction. Environmental variables, infectious agents, genetic vulnerability, and host immune responses combine intricately to cause these disorders. Exposure to allergens, cigarette smoke, air pollutants, occupational chemicals, and respiratory infections stimulates airway epithelial cells and alveolar macrophages, beginning inflammatory cascades that ultimately impair normal pulmonary function.[15]

The initial line of defense against inhaled infections and environmental irritants is the airway epithelium. Following exposure to damaging stimuli, epithelial cells generate damage-associated molecular patterns (DAMPs), chemokines, and cytokines that recruit inflammatory cells such as neutrophils, eosinophils, macrophages, dendritic cells, and T lymphocytes into the lung tissue. Activated immune cells subsequently produce numerous pro-inflammatory mediators, including tumor necrosis factor-alpha (TNF-α), interleukin-1 beta (IL-1β), interleukin-6 (IL-6), interleukin-8 (IL-8), transforming growth factor-beta (TGF-β), and interferon-gamma (IFN-γ), which amplify inflammatory responses and contribute to airway remodeling and tissue injury.[16]

Another important element in the development of inflammatory lung disorders is oxidative stress. Lipid peroxidation, protein oxidation, DNA damage, and pulmonary epithelial cell death are the results of natural antioxidant defense mechanisms being overloaded by excessive production of reactive oxygen species (ROS) from activated inflammatory cells, damaged mitochondria, and environmental contaminants. Persistent oxidative stress further activates inflammatory signaling pathways and accelerates disease progression by enhancing cytokine production and extracellular matrix degradation.[17]

Numerous intracellular signaling pathways are crucial targets for treatment because they control pulmonary inflammation. The nuclear factor-kappa B (NF-κB) pathway is regarded as the primary regulator of inflammatory gene expression. Pro-inflammatory cytokines, chemokines, cyclooxygenase-2 (COX-2), inducible nitric oxide synthase (iNOS), and adhesion molecules are all transcriptionally induced when NF-κB

is activated. In a similar manner, the creation, apoptogen, cell cell proliferation, apoptosis, apoptosis, apoptosis, apoptosis, cell proliferation, cell proliferation, cell proliferation, cell proliferation, apoptosis, cell proliferation, apoptosis, apoptosis, apoptosis, cell proliferation, cell proliferation, cell proliferation, cell proliferation. Other signaling pathways, including Janus kinase/signal transducer and activator of transcription (JAK/STAT), phosphoinositide 3-kinase/protein kinase B (PI3K/Akt), and nuclear factor erythroid 2-related factor 2 (Nrf2), also play significant roles in balancing inflammatory and antioxidant responses within the lungs.[18]



Chronic airway remodeling, which is marked by goblet cell hyperplasia, mucus hypersecretion, smooth muscle hypertrophy, collagen deposition, fibrosis, and permanent airway constriction, is caused by persistent activation of these inflammatory pathways. Excessive fibroblast and myofibroblast activation in pulmonary fibrosis causes aberrant extracellular matrix buildup, which lowers lung elasticity and gas exchange capacity. Similar to this, pulmonary edema and respiratory failure are caused by widespread alveolar injury and increased vascular permeability, which are characteristics of ALI and ARDS.[19]

Developing successful treatment approaches requires an understanding of the molecular and cellular processes underlying inflammatory lung disorders. Novel pharmacological agents, such as plant-derived bioactive compounds like those in *Achyranthes aspera*, which have both anti-inflammatory and antioxidant properties, can be developed by focusing on inflammatory mediators, oxidative stress, and intracellular signaling pathways.[20]

MOLECULAR MECHANISMS OF INFLAMMATION

Inflammation is a complicated biological reaction that occurs when the innate and adaptive immune systems are exposed to infections, allergens, cigarette smoke, environmental pollution, or tissue damage. In inflammatory lung illnesses, activation of airway epithelial cells and alveolar macrophages causes the production of inflammatory mediators, which attract neutrophils, eosinophils, dendritic cells, and lymphocytes to the lungs. These immune cells create an overabundance of pro-inflammatory cytokines, chemokines, and reactive oxygen species (ROS), resulting in chronic inflammation, tissue damage, and decreased pulmonary function.[21]

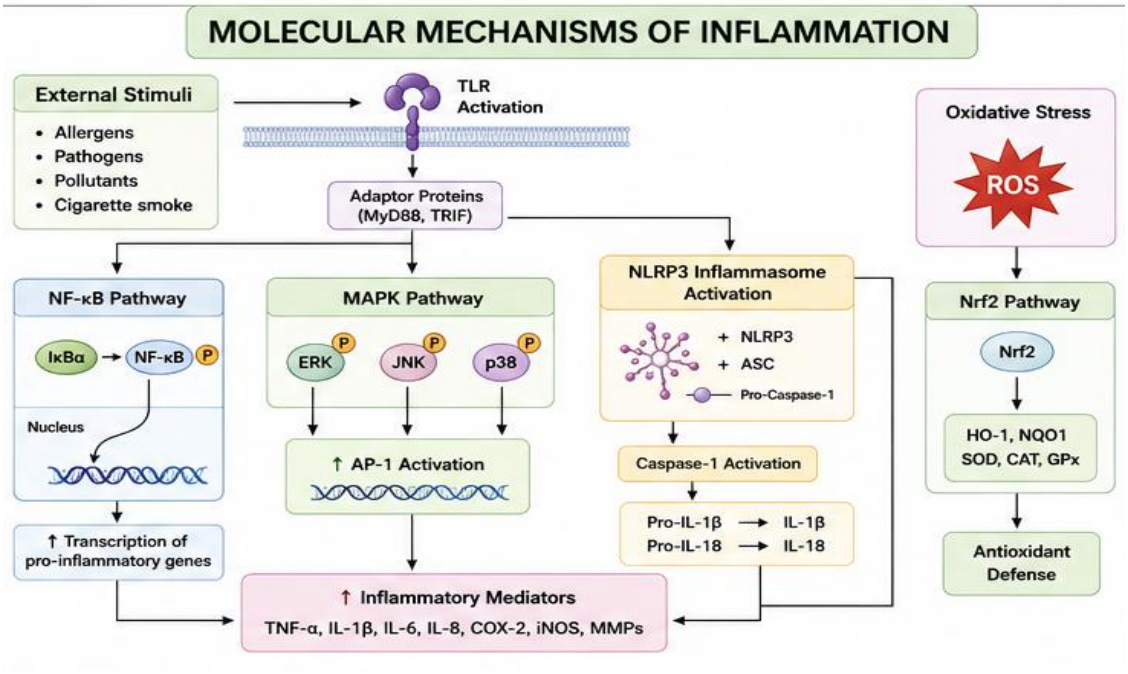
Inflammatory reactions are thought to be mostly regulated by the nuclear factor-kappa B (NF-κB) signaling pathway. Because of its connection with the inhibitor of κB (IκB), NF-κB is inactive in the cytoplasm under normal physiological circumstances. When inflammatory mediators like lipopolysaccharide (LPS), interleukin-1 beta (IL-1β), or tumor necrosis factor-alpha (TNF-α) stimulate IκB, it is phosphorylated and degraded, allowing NF-κB to go into the nucleus.

Numerous inflammatory genes, including as TNF-α, IL-6, IL-8, cyclooxygenase-2 (COX-2), inducible nitric oxide synthase (iNOS), and adhesion molecules, are transcriptionally induced by activated NF-κB, which amplifies inflammatory responses in lung tissues.[22]

The mitogen-activated protein kinase (MAPK) pathway, which includes ERK, JNK, and p38 MAPK, is also significant in pulmonary inflammation. The activation of these kinases controls inflammatory gene expression, cytokine production, apoptosis, cell proliferation, and tissue remodeling. Increased MAPK signaling activity has been found in asthma, COPD, and acute lung damage, which contributes to chronic airway inflammation and structural abnormalities in the respiratory tract.[23]

Oxidative stress contributes significantly to the pathophysiology of inflammatory lung disorders by increasing the formation of reactive oxygen species (ROS) and reactive nitrogen species (RNS). These reactive chemicals degrade cellular lipids, proteins, DNA, and mitochondria, resulting in death and decreased epithelial barrier function. ROS trigger NF-κB and MAPK signaling pathways, resulting in a positive feedback loop that perpetuates chronic inflammation. The Nrf2 signaling pathway, on the other hand, protects against oxidative harm by increasing the production of antioxidant enzymes such heme oxygenase-1 (HO-1), superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPx). Impaired Nrf2 signaling has been linked to an increased risk of chronic inflammatory lung disorders.[24, 25]

Inflammatory cytokines and chemokines are essential mediators of immune cell recruitment and activation. Cytokines such as TNF-α, IL-1β, IL-6, IL-17, and TGF-β increase inflammation, mucus production, airway remodeling, and fibrosis. Chemokines such as CXCL8 and CCL2 promote neutrophil and monocyte migration into lung tissues, maintaining inflammatory responses. Persistent overproduction of these mediators promotes disease progression and permanent pulmonary damage.[26]



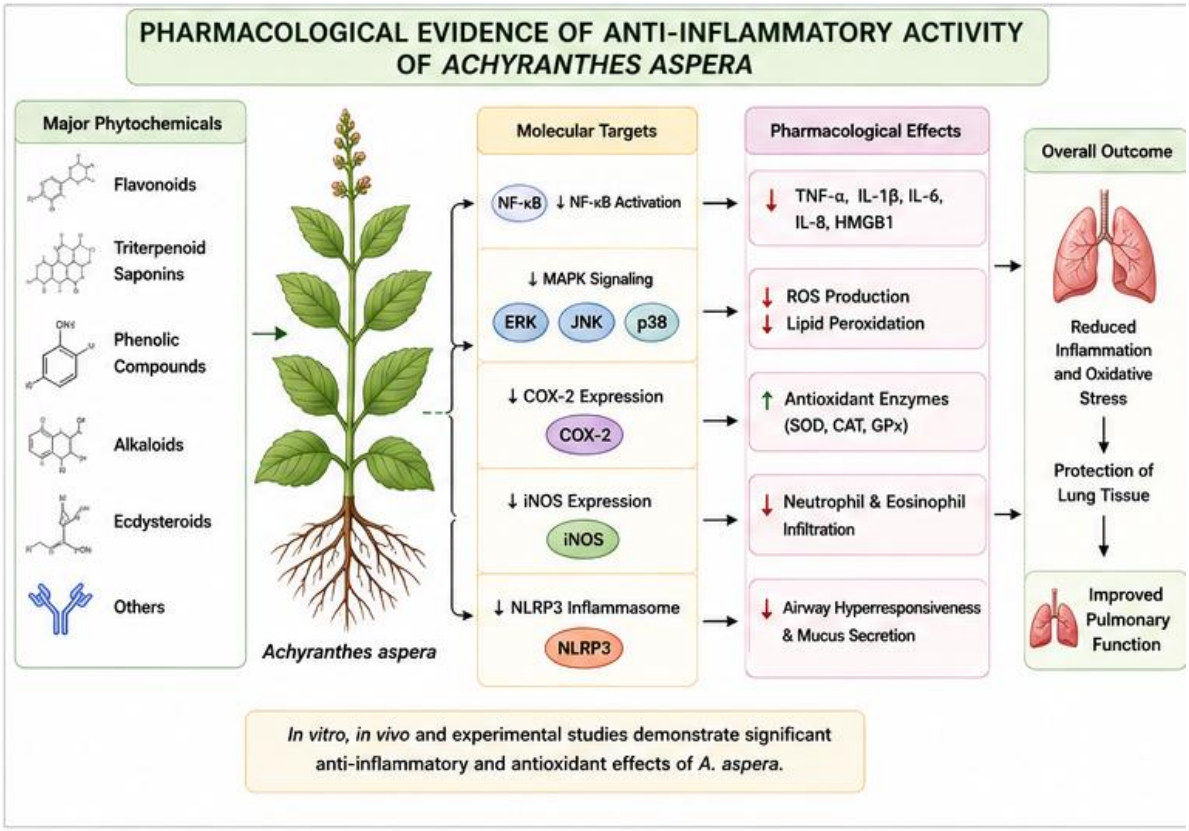
Understanding these molecular pathways has aided the discovery of new treatment targets for inflammatory lung disorders. Pharmacological drugs that suppress NF-κB, MAPK, COX-2, iNOS, and pro-inflammatory cytokines while increasing antioxidant defenses via Nrf2 activation have intriguing therapeutic promise. Medicinal herbs high in flavonoids, saponins, terpenoids, and phenolic compounds, such as *Achyranthes aspera*, may have a positive effect by regulating numerous inflammatory signaling pathways and lowering oxidative stress. Such multi-target activities point to their potential use as supplementary treatments for inflammatory respiratory diseases.

PHARMACOLOGICAL EVIDENCE OF ANTI-INFLAMMATORY ACTIVITY

Achyranthes aspera has received a lot of interest due to its wide range of pharmacological actions, notably its anti-inflammatory properties. Experimental studies have shown that extracts derived from the roots, leaves, seeds, and whole plants have strong anti-inflammatory action in both acute and chronic inflammatory models. These pharmacological effects are principally due to the synergistic action of triterpenoid saponins, flavonoids, alkaloids, phenolic compounds, ecdysteroids, and terpenoids, which modulate a variety of inflammatory mediators and signaling pathways.[27]

Several in vivo experiments have shown that methanolic and ethanolic extracts of *A. aspera* effectively prevent carrageenan-induced paw edema, cotton pellet-induced granuloma, formalin-induced inflammation, and xylene-induced ear edema in experimental mice. These data suggest that the plant inhibits both the exudative and proliferative stages of inflammation by decreasing vascular permeability, leukocyte infiltration, and inflammatory mediator release. In several experimental models, the observed pharmacological effects were shown to be equivalent to those of typical nonsteroidal anti-inflammatory medications (NSAIDs).[28]

Further in vitro studies have shown that *A. aspera* extracts prevent activated macrophages from producing nitric oxide (NO), prostaglandin E_2 (PGE_2), tumor necrosis factor-alpha (TNF-α), interleukin-1 beta (IL-1β), and interleukin-6 (IL-6). Additionally, the extracts reduce the manufacture of important inflammatory mediators by suppressing the expression of inducible nitric oxide synthase (iNOS) and cyclooxygenase-2 (COX-2). These findings imply that the plant's anti-inflammatory properties are mediated by modifying important cellular signaling pathways related to immune activation.[29]



A. aspera's anti-inflammatory properties are also greatly influenced by its antioxidant activity. The plant's bioactive phytochemicals efficiently scavenge reactive oxygen species (ROS), prevent lipid peroxidation, and boost endogenous antioxidant enzymes including glutathione peroxidase, catalase, and superoxide dismutase. The plant limits tissue damage during chronic inflammatory circumstances by lowering oxidative stress, preventing oxidative damage to pulmonary cells, and stopping the amplification of inflammatory signaling cascades.[30]

A. aspera's immunomodulatory qualities have been emphasized by recent pharmacological research, which shows that it can control leukocyte migration, cytokine release, mast cell degranulation, and macrophage activation. The plant may be used to treat respiratory inflammatory conditions including asthma and allergic bronchitis since experimental research indicates that it reduces eosinophilic infiltration and histamine production, which attenuates allergic inflammatory reactions. To determine its therapeutic potential and safety in human inflammatory lung disorders, further mechanistic research, standardized extract characterisation, pharmacokinetic analyses, and carefully planned clinical trials are needed, despite these optimistic results.[31]

POTENTIAL THERAPEUTIC ROLE IN ASTHMA

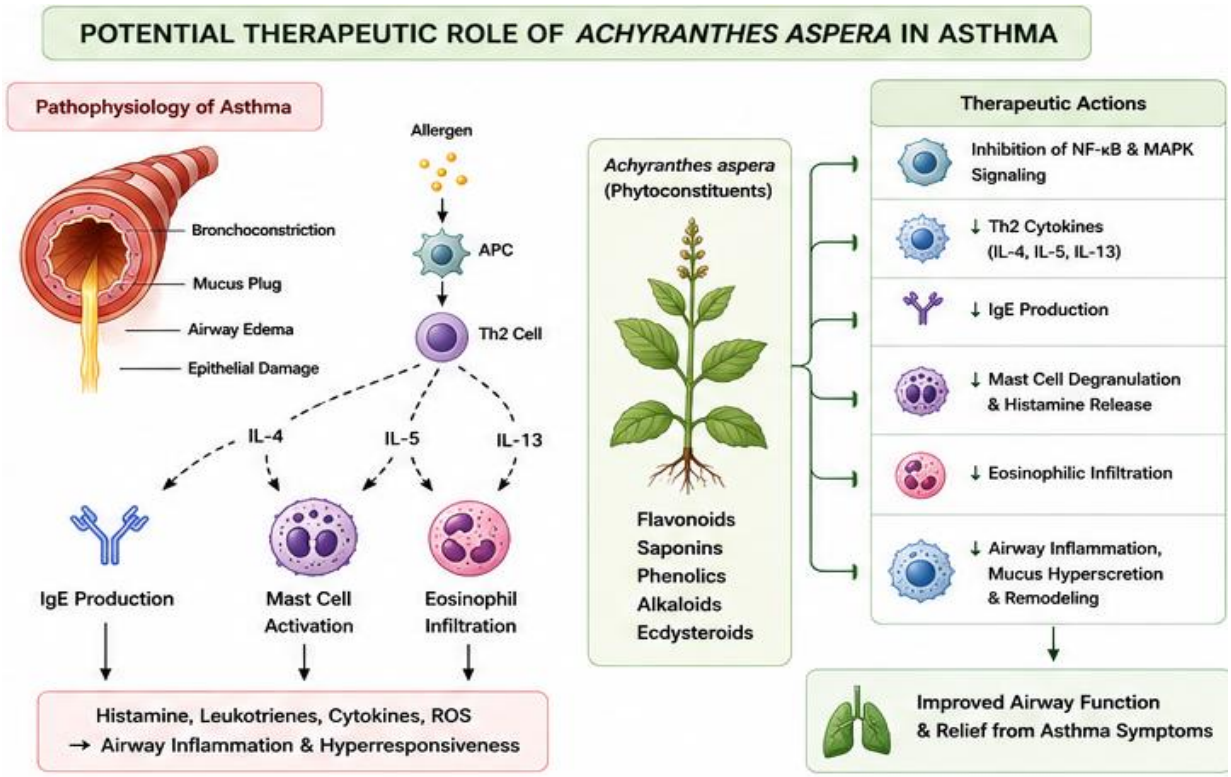
Asthma is a chronic inflammatory airway illness characterized by reversible airflow restriction, bronchial hyperresponsiveness, excessive mucus production, and airway remodeling. Type 2 immune responses including eosinophils, mast cells, T helper 2 (Th2) lymphocytes, and an excess of cytokines like interleukin (IL)-4, IL-5, and IL-13 are the main causes of the illness. Goblet cell hyperplasia, smooth muscle hypertrophy, and subepithelial fibrosis are among the structural changes

brought on by persistent airway inflammation that eventually impede respiratory performance. Long-term medication may be linked to side effects and inconsistent treatment responses, underscoring the need for alternate therapeutic agents even if inhaled corticosteroids and bronchodilators continue to be the mainstay of asthma care.[32]

Achyranthes aspera has long been used in Ayurveda and other indigenous medicine to treat asthma, persistent cough, bronchitis, and other respiratory conditions. Recent experimental research have offered scientific evidence to substantiate these conventional assertions. In mouse allergic asthma models, A. aspera extracts dramatically reduced eosinophilic infiltration, airway inflammation, serum immunoglobulin E (IgE) levels, and bronchial hyperresponsiveness. These findings indicate that the plant has considerable anti-asthmatic and immunomodulatory properties.[33]

A. aspera's antiasthmatic benefits are mostly due to its ability to control inflammatory mediators and oxidative stress. Bioactive substances such as triterpenoid saponins, flavonoids, phenolic compounds, and ecdysteroids block NF-κB and MAPK signaling pathways, resulting to decreased production of pro-inflammatory cytokines, including TNF-α, IL-1β, IL-6, IL-4, IL-5, and IL-13. In addition, the plant inhibits mast cell degranulation and histamine release, which reduces allergic airway inflammation and bronchoconstriction.[34]

Oxidative stress contributes significantly to asthma pathophysiology by inducing epithelium damage and increasing inflammatory responses. Experimental evidence suggests that *A. aspera* improves endogenous antioxidant defense mechanisms by increasing the activity of superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPx), while decreasing reactive oxygen species (ROS) generation and lipid peroxidation. These antioxidant actions may help preserve airway epithelial cells and reduce chronic airway inflammation.[35]



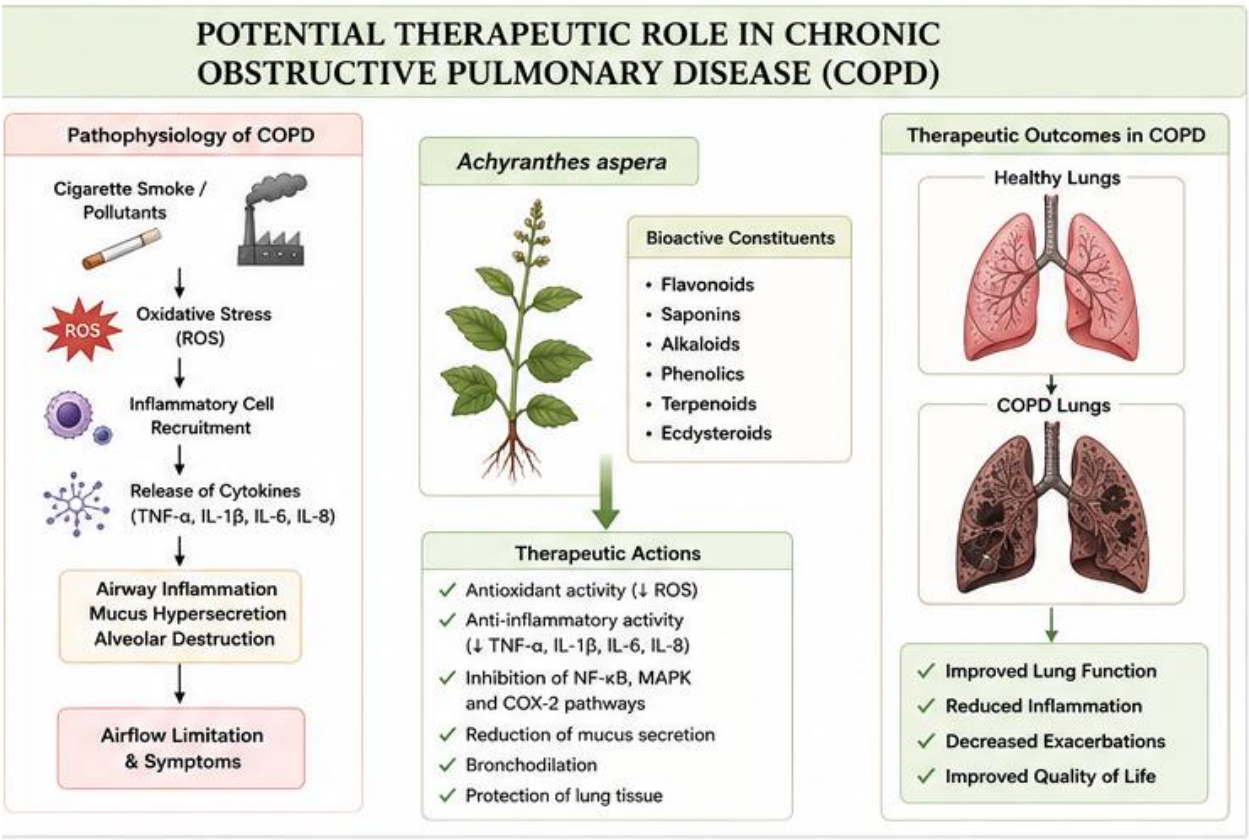
Although preclinical research clearly suggests that *Achyranthes aspera* has therapeutic promise in asthma, clinical data is sparse. Future study should concentrate on standardized extract formulation, the discovery of active phytoconstituents, pharmacokinetic evaluation, and randomized clinical trials to determine its effectiveness and safety as a supplemental treatment agent for asthma therapy.[36]

POTENTIAL THERAPEUTIC ROLE IN CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD)

Chronic obstructive pulmonary disease (COPD) is a progressive respiratory ailment characterized by persistent airflow restriction, chronic airway inflammation, emphysema, and lung structural remodeling. Cigarette smoke, environmental contaminants, occupational dust, and biomass fuel exposure are the primary risk factors that cause chronic inflammation in vulnerable persons. The disease causes increased infiltration of neutrophils, macrophages, and

$CD8^+$ T lymphocytes, as well as elevated levels of pro-inflammatory mediators such as TNF-α, IL-6, IL-8, MMPs, and ROS. This leads to progressive destruction of lung tissue and decline in pulmonary function.[37]

Achyranthes aspera's pharmacological qualities suggest that it might be used as a supplemental therapy for COPD. The herb contains powerful anti-inflammatory and antioxidant properties that may reduce chronic airway inflammation and oxidative stress, two important pathogenic processes implicated in COPD development. Phytochemicals in A. aspera, such as triterpenoid saponins, flavonoids, phenolic compounds, and oleanolic acid, suppress inflammatory cytokines and inhibit key signaling pathways like NF-κB and MAPK, reducing inflammatory responses in pulmonary tissues.[38]



Oxidative stress contributes significantly to COPD pathophysiology by causing epithelial cell damage, mucus hypersecretion, mitochondrial dysfunction, and the activation of inflammatory signaling pathways. Bioactive substances isolated from A. aspera have been shown to exhibit considerable free radical scavenging action and to increase endogenous antioxidant enzymes such as superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase. These antioxidant qualities might protect lung tissues from oxidative damage and slow the evolution of chronic airway inflammation.[39]

In addition to its antioxidant properties, *A. aspera* has been demonstrated to have immunomodulatory action by controlling macrophage activation, preventing excessive cytokine release, and decreasing inflammatory enzyme expression, such as cyclooxygenase-2 (COX-2) and inducible nitric oxide synthase. Such multi-target pharmaceutical activities may help to reduce airway remodeling, mucus hypersecretion, and the persistent inflammatory responses that are typical in COPD. Although direct experimental investigations on *A. aspera* in COPD models are rare, the shown anti-inflammatory and antioxidant pathways give a compelling scientific foundation for additional research.[40]

Future research should examine standardized extracts and extracted bioactive compounds from *Achyranthes aspera* in experimental COPD models and clinical trials. Detailed pharmacokinetic investigations, toxicity evaluations, and molecular studies are needed to evaluate its effectiveness and prove its potential as an additional treatment for COPD prevention and control.[41]

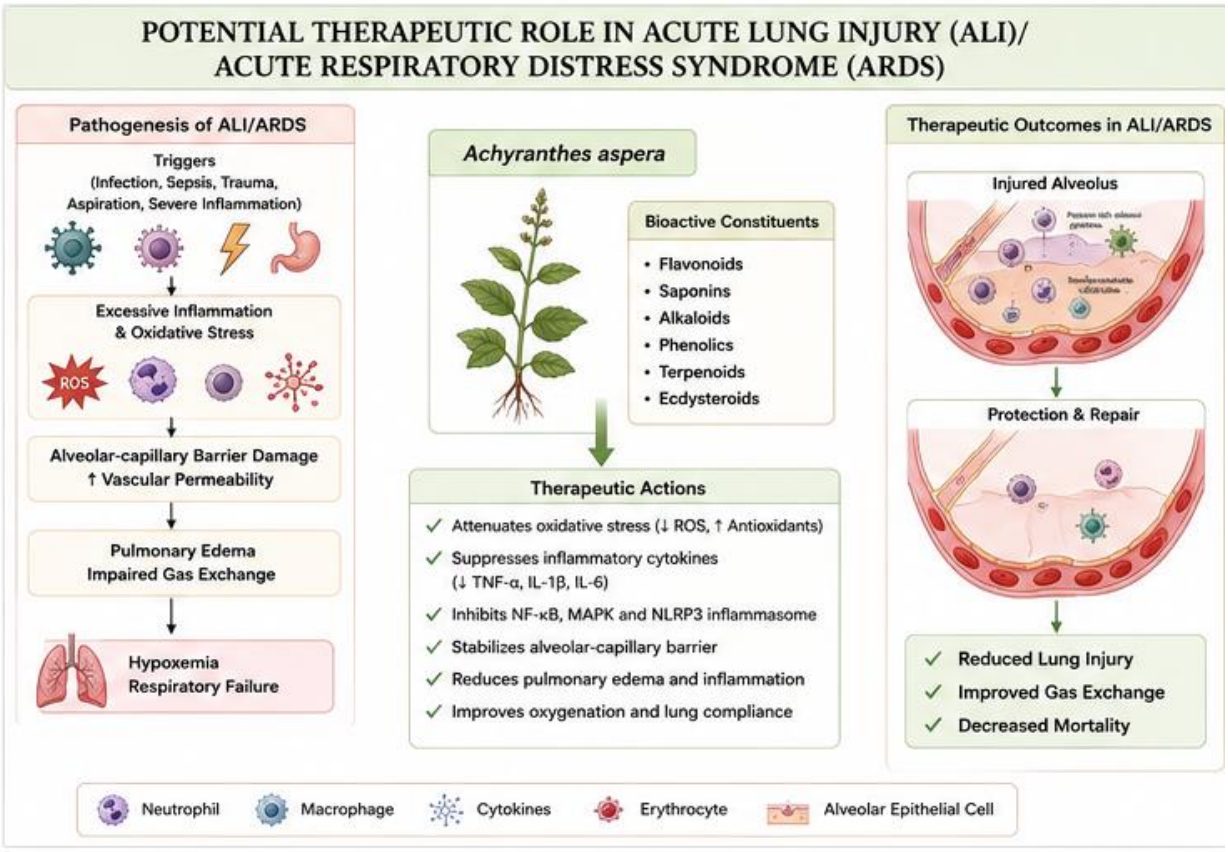
POTENTIAL THERAPEUTIC ROLE IN ACUTE LUNG INJURY (ALI)/ACUTE RESPIRATORY DISTRESS SYNDROME (ARDS)

Acute lung injury (ALI) and acute respiratory distress syndrome (ARDS) are severe inflammatory pulmonary illnesses marked by widespread alveolar destruction, breakdown of the alveolar-capillary barrier, pulmonary edema, excessive inflammatory cell infiltration, and severe hypoxia. These disorders can be caused by bacterial or viral infections, sepsis, pneumonia, aspiration, trauma, or the inhalation of hazardous chemicals. The etiology of ALI/ARDS involves

uncontrolled activation of neutrophils, macrophages, and pulmonary endothelial cells, which results in excessive production of inflammatory mediators, oxidative stress, and extensive lung tissue damage.[42]

In ALI/ARDS, pro-inflammatory cytokines such as TNF-α, IL-1β, IL-6, IL-8, and HMGB1 increase, causing an inflammatory response. These mediators activate intracellular signaling pathways, including NF-κB, MAPK, and NLRP3 inflammasome, leading to increased vascular permeability, epithelial cell apoptosis, and protein-rich fluid accumulation in alveolar spaces. Persistent oxidative stress exacerbates lung damage by inducing lipid peroxidation, mitochondrial failure, and inflammatory cell activation.[43]

Although direct investigations on *Achyranthes aspera* in ALI/ARDS models are lacking, the plant's pharmacological qualities indicate significant therapeutic promise. A. aspera extracts have strong anti-inflammatory and antioxidant properties, including the ability to limit inflammatory cytokine production, inhibit cyclooxygenase-2 (COX-2) and inducible nitric oxide synthase (iNOS), and reduce reactive oxygen species (ROS) synthesis. These pathways may aid to reduce pulmonary inflammation, protect alveolar epithelial cells, and maintain the integrity of the alveolar-capillary membrane following acute lung damage.[44]



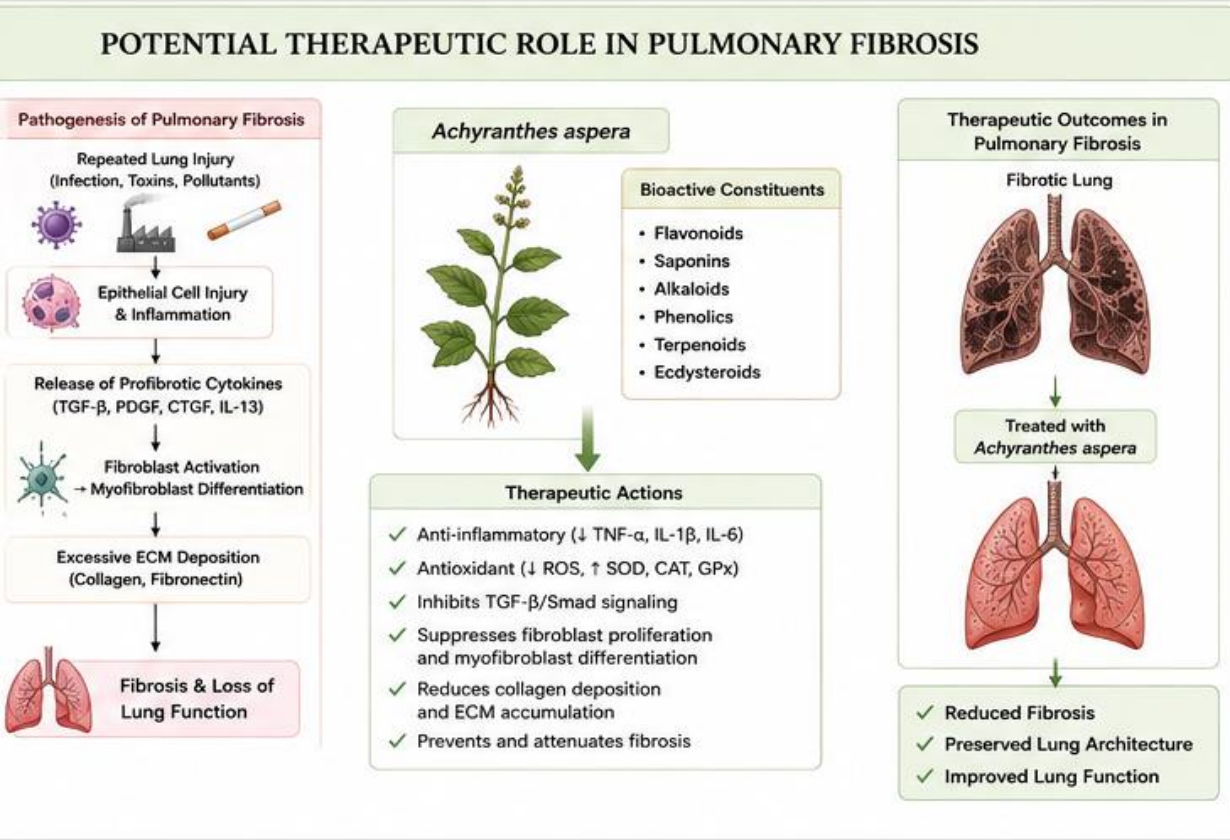
A. aspera phytochemicals, which include triterpenoid saponins, flavonoids, phenolic compounds, and ecdysteroids, have been shown to affect a variety of inflammatory signaling pathways linked to acute inflammatory diseases. Their antioxidant action boosts endogenous defense mechanisms by raising the activity of SOD, CAT, and GPx, limiting oxidative damage and decreasing inflammatory cell infiltration. Such multitarget pharmacological activities suggest that *A. aspera* might be a potential supplementary treatment for reducing the course of ALI and ARDS.[45]

Despite these promising results, clinical data supporting the use of *Achyranthes aspera* in ALI/ARDS is presently lacking. Future research should concentrate on standardized plant extracts and isolated bioactive compounds in experimental models of acute lung injury, followed by pharmacokinetic studies and well-designed clinical trials to determine therapeutic efficacy, optimal dosage, and long-term safety in patients with ALI and ARDS.[46]

POTENTIAL THERAPEUTIC ROLE IN PULMONARY FIBROSIS

Pulmonary fibrosis is a chronic interstitial lung disease marked by excessive extracellular matrix (ECM) deposition, irreversible damage of alveolar architecture, and gradual deterioration in pulmonary function. Repeated damage to alveolar epithelial cells triggers aberrant wound-healing responses that promote

fibroblast proliferation and differentiation into myofibroblasts, leading to excessive collagen deposition and fibrosis. Several molecular mediators, including as TGF-β, PDGF, CTGF, and pro-inflammatory cytokines, play key roles in the genesis and progression of pulmonary fibrosis.[47]



Chronic inflammation and oxidative stress are important drivers of pulmonary fibrogenesis. Prolonged activation of inflammatory cells, such as macrophages and neutrophils, triggers the production of TNF-α, IL-1β, IL-6, and ROS, which activate fibroblasts and increase extracellular matrix buildup. Activation of signaling pathways, including TGF-β/Smad, NF-κB, PI3K/Akt, and MAPK, contributes to fibroblast activation and fibrosis progression.[48]

Although direct experimental data for *Achyranthes aspera's* antifibrotic properties is scarce, its pharmacological profile suggests significant therapeutic promise. The plant has strong anti-inflammatory and antioxidant properties that may control inflammatory cytokine production, reduce oxidative stress, and inhibit molecular pathways implicated in fibrogenesis. Bioactive ingredients such as triterpenoid saponins, flavonoids, phenolic compounds, oleanolic acid, and ursolic acid have been shown in several animal models to modify inflammatory responses and reduce tissue fibrosis, implying potential advantages in pulmonary fibrosis.[49]

A. aspera's antioxidant properties may also help to protect against pulmonary fibrosis by reducing lipid peroxidation, scavenging reactive oxygen species, and increasing endogenous antioxidant enzymes like superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPx). Furthermore, inhibiting NF-κB-mediated inflammatory signaling and downregulating profibrotic cytokines may restrict fibroblast proliferation and excess extracellular matrix deposition. These multitarget pharmacological activities warrant further exploration of *A. aspera* as a possible supplementary treatment for pulmonary fibrosis.[50]

Despite these potential pharmacological qualities, no clinical trials have been conducted to determine the effectiveness of *Achyranthes aspera* in pulmonary fibrosis. Future research should look into its effects in established experimental models of pulmonary fibrosis, identify the active phytoconstituents responsible for antifibrotic activity, elucidate their molecular mechanisms, and conduct well-designed clinical trials to determine safety, pharmacokinetics, and therapeutic efficacy.[51]

SAFETY, TOXICITY AND PHARMACOKINETICS

The safety profile of *Achyranthes aspera* is a significant issue for its potential use as a treatment agent for inflammatory lung disease. Traditional medicine has utilized various components of the plant for millennia to treat respiratory, gastrointestinal, dermatological, and

inflammatory problems, indicating a generally acceptable safety profile when provided at therapeutic quantities. However, full toxicological and pharmacokinetic assessments are still scarce, with the majority of current material coming from preclinical trials. As a result, rigorous researches are required to determine its long-term safety, appropriate dose, and therapeutic usefulness.[52]

Acute and subacute toxicity tests in experimental animals have shown that *A. aspera* extracts are relatively safe at therapeutic levels. Oral administration of aqueous and ethanolic extracts did not result in substantial mortality, behavioral problems, or dramatic changes in body weight, food intake, hematological parameters, or biochemical indicators of liver and kidney function. Histopathological tests found no substantial tissue damage in key organs after short-term treatment. However, excessive dosages or extended exposure may result in dose-dependent hazardous consequences, underlining the significance of proper dose standardization.[53]

Reproductive toxicity investigations have shown that several *A. aspera* extracts exhibit antifertility and abortifacient characteristics in experimental animal models. These pharmacological effects are thought to be caused by unique bioactive ingredients that affect reproductive hormones and uterine function. As a result, the use of *A. aspera* during pregnancy should be treated with caution until enough clinical safety evidence is available. Further research is also needed to determine its safety during breastfeeding and in pediatric populations.[54]

There is limited information available about *A. aspera's* pharmacokinetics. According to limited research, key phytoconstituents such as triterpenoid saponins, flavonoids, and phenolic compounds are absorbed via the gut, then metabolized by the liver and excreted through the kidneys. Individual bioactive chemicals' absorption, bioavailability, tissue distribution, metabolism, excretion, and possible herb-drug interactions have yet to be fully described. Advanced analytical approaches, including liquid chromatography–mass spectrometry (LC–MS/MS) and pharmacokinetic modeling, are needed to discover the disposition characteristics of these drugs and enhance their therapeutic use.[55]

Before *Achyranthes aspera* may be used in clinical practice, more research should be conducted on chronic toxicity, genotoxicity, carcinogenicity, reproductive safety, pharmacokinetic behavior, and clinical safety using well-designed human trials. Standardization of plant extracts, identification of active phytoconstituents, and assessment of herb-drug interactions will all be required to assure uniform quality, effectiveness, and safety. Such research will give the scientific data needed to produce standardized A. aspera-based phytopharmaceuticals for inflammatory pulmonary illnesses.[56]

CONCLUSION

Achyranthes aspera L. is a medicinal plant with significant pharmacological value that has long been used in traditional systems of medicine to treat inflammatory and respiratory problems. Scientific evidence suggests that the plant has significant anti-inflammatory, antioxidant, immunomodulatory, antimicrobial, and antiallergic properties, which are primarily due to its diverse phytochemical constituents, which include triterpenoid saponins, flavonoids, phenolic compounds, alkaloids, terpenoids, steroids, and ecdysteroids. These bioactive chemicals control inflammation pathways, including NF-κB, MAPK, COX-2, iNOS, and pro-inflammatory cytokines, leading to reduced inflammation and oxidative stress.

According to experimental research, *A. aspera* shows remarkable therapeutic potential for the treatment of inflammatory lung illnesses such as asthma, chronic obstructive pulmonary disease (COPD), acute lung injury (ALI), acute respiratory distress syndrome (ARDS), and pulmonary fibrosis. Its capacity to inhibit inflammatory mediator production, minimize oxidative damage, normalize immunological responses, and preserve pulmonary tissues demonstrates its promise as a multi-target therapeutic agent. Furthermore, the plant's antioxidant capabilities may help to slow disease development by maintaining cellular integrity and increasing endogenous defense systems.

Despite these positive findings, the majority of the existing information comes from in vitro and animal studies, with minimal clinical research. The absence of standardized extracts, poor pharmacokinetic data, and limited long-term safety assessments continue to impede its use in clinical practice. Future research should focus on identifying active phytoconstituents, elucidating molecular processes, standardizing herbal formulations, improved drug delivery techniques, and well-designed clinical trials to establish its effectiveness and safety in human patients.

To summarize, *Achyranthes aspera* is a promising natural source of bioactive chemicals with substantial promise for the development of innovative phytopharmaceuticals that target inflammatory lung disorders. Continued interdisciplinary study combining pharmacognosy, phytochemistry, pharmacology, pharmaceutics, toxicology, and clinical medicine will be required to convert its traditional medicinal value into evidence-based therapeutic uses. With more scientific confirmation, A. aspera might emerge as a useful supplemental or adjuvant therapy for the prevention and treatment of chronic inflammatory respiratory diseases.

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