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Undervalued Botanical Bioactives: A Comprehensive Review on the Multi-Target Therapeutic Potential of *Curcuma longa* L. Leaves in Mitigating Acute Respiratory Distress Syndrome (ARDS)

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ABSTRACT

Acute Respiratory Distress Syndrome (ARDS) continues to be a leading cause of critical care deaths and is defined by a hyper-inflammatory "dark triad" of matrix degradation, cytokine storms and uncontrolled oxidative stress. Although the therapeutic properties of the rhizome of Curcuma longa are extensively studied, the therapeutic potential of its leaves has been relatively underutilized. In vitro profiling of crude extracts of Pakistani Curcuma longa leaves shows potent anti-inflammatory and tissue-protective activity. The leaf extract significantly inhibits proteinase activity by 43 ± 1.3% and effectively downregulates key pro-inflammatory cascades by inhibiting Matrix Metalloproteinase (MMP-9) by 61 ± 6%, Interleukin-1 beta (IL-1β) by 67 ± 7%, and Tumor Necrosis Factor-alpha (TNF-α) by 52 ± 5% at the benchmark optimization threshold of 1.00 mg/mL. Moreover, the extract also has competitive binding capacity for copper ions, with a 63.9% inhibition of tyrosinase activity directly opposing the production of downstream reactive oxygen species (ROS). Compared to the use of conventional antioxidants such as kojic acid, inhibition reaches 77.0% in their combination, thus extending the possibilities for synergistic formulations. This review compiles these biochemical data, highlighting the molecular mechanisms of kappa, MAPK, Nrf2 pathways, the application of delivery systems including nanoencapsulation to enhance the low oral bioavailability, and a platform for future in vivo clinical translation.

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INTRODUCTION

Introduction & Clinical Pathophysiology of ARDS

Acute Respiratory Distress Syndrome (ARDS) is a life-threatening, high mortality disease of the respiratory system that accounts for approximately 10% of worldwide admissions to intensive care units (ICUs) (Vellingiri *et al.*, 2020; Bardají-Carrillo *et al.*, 2024). The disease is characterized pathologically by noncardiogenic pulmonary edema, severe hypoxemia and decreased lung compliance, and is caused by direct or indirect injury to the alveolar-capillary membrane (Matthay *et al.*, 2019; Ma *et al.*, 2025). On first insult, neutrophils and hyperactivated alveolar macrophages enter and trigger a locally generated series of events that begins a destructive cycle of tissue breakdown (Lupu *et al.*, 2020; Qiao *et al.*, 2024). If the process proceeds with post-viral or severe viral ARDS, it quickly turns into a "cytokine storm", an uncontrolled systemic response (Dubey *et al.*, 2026). This hyper-inflammatory event leads to high plasma levels of toxic pro-inflammatory mediators such as Interleukin-6 (IL-6), Interleukin-1 beta (IL-1β), and Tumor Necrosis Factor-alpha (TNF-α) (Bahmani *et al.*, 2022; Sagris *et al.*, 2022). The signaling molecules cause the endothelial cells to become more permeable, and protein-rich fluid and cell debris accumulate in the alveolar air spaces, forming hyaline membranes that severely diminish gas exchange.

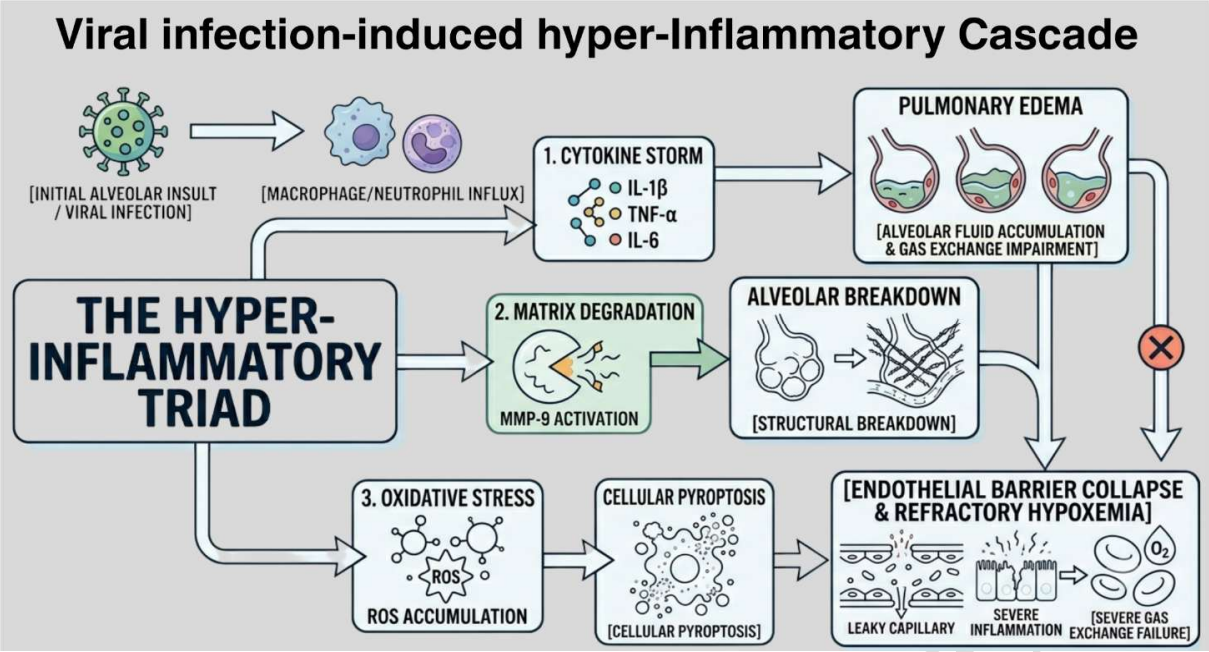

Fig. 1: provides a detailed depiction of the complex biological process that ultimately results in severe damage to the lungs. It elaborates on the process by which the 'Hyper-Inflammatory Triad,' consisting of cytokine storm, matrix degradation, and oxidative stress, causes pulmonary edema and alveolar breakdown as a consequence. Ultimately, the image illustrates that these synergistic impacts lead to a breakdown of the endothelial barrier and the development of refractory hypoxemia, thereby emphasizing the complete cessation of effective gas exchange.

Conventional treatment for the cascade is usually a high dose of a systemic corticosteroid (such as dexamethasone) or a monoclonal antibody (such as tocilizumab) (Chen and Ostermann, 2022; Liu *et al.*, 2025). These therapies are, however, challenging because of their high cost, their inconsistent response rates, and the potential side effects of other therapies, such as long-term immunosuppression and delayed tissue healing. There is a critical need for multi-target, low-cost nutraceutical interventions.

Phytochemical Landscape

Turmeric Leaves vs. Roots

Historically, classical ethnopharmacology has focused primarily on the bright yellow rhizome of Curcuma longa L., but recent analytical studies of the plant's discarded leaves reveal that these leaves possess their own unique and rich phytochemical profile. The rhizome usually contains typical curcuminoids, but the leaves also contain a complex of volatile essential oils (1,8-cineole, alpha$-terpinene, limonene, zingiberene) and polyphenols, flavonoids (quercetin, isorhamnetin), and phenolic acids (Destryana *et al.*; Ibáñez and Blázquez, 2020) . However, the main advantage of raw leaf extracts over isolated curcumin is that:

Polyphenolic Synergy

The secondary metabolites in the leaf complex act synergistically to maintain cellular membranes in a stable state and improve the antioxidant status (El-Saadony *et al.*, 2025; Espinoza *et al.*, 2026).

Broad Therapeutic Index

This natural combination does not cause the gastrointestinal or renal toxicity that is sometimes associated with the long-term use of synthetic, nonsteroidal anti-inflammatory drugs (NSAIDs) or with the use of one isolated molecule in higher doses (Memarzia *et al.*, 2021).

Sustainable Harvest

Using foliage means harvesting without damaging the tree. It is a very valuable and cost-effective resource for countries facing severe public health challenges like Pakistan (Fuloria *et al.*, 2022; Khan *et al.*, 2023).

Mechanistic Action of Leaf Bioactives on Inflammatory Cascades

Protease Suppression and Extracellular Matrix Protection

In severe pulmonary inflammation, neutrophils are activated to produce destructive proteases, like neutrophil elastase (NE), which break down structural collagen and elastin in the lung (Cheetham *et al.*, 2024). At the same time, zinc-dependent endopeptidases, specifically Matrix Metalloproteinase-9 (MMP-9), remodel the extracellular matrix (ECM) which contributes to the chronic pulmonary fibrosis seen in ARDS survivors (Vianello *et al.*, 2022; Wolosowicz *et al.*, 2025). The evaluation in vitro shows that the extract of the leaves of Curcuma longa is effective in inhibiting this enzyme tissue destruction (Narwaria *et al.*, 2025). The extract inhibits proteinase at 1.00 mg/mL by 43 ± 1.3% at the benchmark concentration. It also decreases the expression of active-matrix metalloproteinase, with a 61 ± 6% inhibition of MMP pathways. This effect is inhibitory to breakdown of structural proteins and aids in maintaining the integrity of the alveolar-capillary barrier.

Downregulation of Pro-Inflammatory Cytokines (IL-1β and TNF-α)

Once the pathogens enter the alveolar space, the NLRP3 inflammasome is activated in immune cells (Zhang *et al.*, 2021). This multi-protein complex is responsible for its activation of caspase-1, which cleaves pro-IL-1β into its mature and bioactive form, and triggers the cytokine feedback loop (Singh *et al.*, 2025). At the same time, there is an increase in the production of another inflammatory molecule called tumour necrosis factor (TNF)-α, which further contributes to inflammation in the local tissue of the lungs (Mazzon and Cuzzocrea, 2007; Gong *et al.*, 2021). The extract of the leaves breaks this cycle by inhibiting the transcription through NF-kappa and MAPK pathways. The experimental data shows very high anti-inflammatory potency at an optimisation level of 1.00 mg/mL:

- IL-1β Pathway: Provides 67 ± 7% inhibition of the IL-1β cascade.
- TNF-α Expression: Inhibits the activity of TNF-α by 52 ± 5%.

The leaf extract interferes with these important upstream signals, thereby decreasing the overactivation of macrophages and preventing them from entering the autocrine feedback loops of cytokines which result in severe ARDS.

Mitigation of Oxidative Stress via Tyrosinase and Free Radical Scavenging

Oxidative stress is a state of imbalance; when the lungs' natural defenses are outstripped by the cumulative effects of excess reactive oxygen and nitrogen species, which directly damage cellular lipids, proteins and DNA (Albano *et al.*, 2022; Bezerra *et al.*, 2023). Tyrosinase is well known for its role in melanin production that requires copper, but it is also a source of oxidative stress through the formation of intermediate free radicals that exacerbate lung damage (Song *et al.*, 2024). Various bioactives from curcuma longa leaves directly inhibit the pathway by competitively binding copper-ions (Bhosale, 2021). The extract inhibits tyrosinase activity at 1.00 mg/mL is 63.9%. This inhibition rises to 77.0% when combined with the standard antioxidant kojic acid. This synergistic effect is beneficial to free radical scavenging, the protection against the lipid peroxidation of delicate alveolar epithelial cells and promotion of tissue healing.

Methodological Framework for Screening Botanical Extracts

To establish reliable data for publication, screening must follow reproducible laboratory methodologies:

Comparative Efficacy of *Curcuma longa* Leaves vs. Commercial Standards

The following collated data compares the experimental activity of the optimized Curcuma longa leaf extract at threshold 1.00 mg/mL with the known positive pharmacological controls: Proteinase Efficacy Profile

Proteinase Efficacy Profile

- Diclofenac Sodium (g/mL): 84 ± 1.1% inhibition
- C. longa Leaf Extract (g/mL): 39 ± 12.5% inhibition

Matrix Metalloproteinase (MMP) Profile

- Doxycycline (mg/mL): 83 ± 8% inhibition (Score: 3)
- C. longa Leaf Extract (mg/mL): 61 ± 6% inhibition (Score: 2)

Interleukin-1 Beta (IL-1β) Profile

- Dexamethasone (1.00 mg/mL): 80 ± 8% inhibition (Score: 3)
- C. longa Leaf Extract (1.00 mg/mL): 67 ± 7% inhibition (Score: 2)

Tumor Necrosis Factor Alpha (TNF-α) Profile

- Dexamethasone (1.00 mg/mL): 80 ± 8% inhibition (Score: 3)
- C. longa Leaf Extract (1.00 mg/mL): 52 ± 5% inhibition (Score: 2)

Tyrosinase Monotherapy vs. Synergistic Complexation

- Raw Leaf Extract (mg/mL): 63.9% enzyme inhibition (Mean OD: 0.69 ± 0.05)
- Leaf Extract + Kojic Acid (1.00 mg/mL): 77.0% enzyme inhibition (Mean OD: 0.44 ± 0.05)

Translational Hurdles: Bioavailability & Formulations

Although promising in vitro findings, there are two major challenges in converting raw *Curcuma longa* leaf extracts into clinical therapies:

The Solubility and Saturation Barrier

Raw leaf extracts may have non-linear dose response at high concentrations (Sethi *et al.*, 2025). For example, the higher concentrations of crude (2 mg/mL or 5 mg/mL) may result in lower measurable benefits because of poor aqueous solubility or cytotoxic saturation from phytochemicals at those higher concentrations.

Rapid metabolism

The main compounds, in particular natural polyphenols and curcuminoids, are quickly metabolized and eliminated by the gastrointestinal tract and therefore have a low systemic bioavailability (Bertoncini-Silva *et al.*, 2024).

To address these challenges, modern drug research is developing new drug delivery systems: Enclosing the leaf extract in polymeric nanoparticles (NP) or solid lipid nanocarriers (SLN), can greatly enhance its water solubility and helps protect the labile bioactives from early degradation (Borges *et al.*, 2020). Lipid bilayer vesicles can be used to target delivery to inflamed pulmonary tissues. The use of targeted dry powder inhalers or nebulized micro-formulations circumvents gastrointestinal breakdown altogether, which is the case with inhalable micro-aerosols (Bertoni *et al.*, 2019). This delivery method eliminates the need to pass through the alveolar-capillary barrier into the deep lung tissue where the therapeutic compound is delivered at a high concentration in direct contact with cell structures.

Synergy with Conventional Protocols & Future Directions

Curcuma longa leaf extract is a promising alternative to conventional medicines, complementing existing therapies rather than replacing them. The extract when used with

Table 1: Overview of assays for enzyme inhibition against standard controls for detection of anti-inflammatory activity.

Assay Type	Target Enzyme / Substrate	Experimental Condition	Key Quantifiable Outcome	Reference Control	Reference
Proteinase Inhibition	Trypsin / Azocasein	Incubation at 37°C, termination via 10% TCA	Spectrophotometric optical density at 280 nm	Diclofenac Sodium	(Ovia *et al.*, 2021)
MMP Assay	Matrix Metalloproteinase-9	Streptavidin-HRP conjugation, incubation at 37°C	Colorimetric absorbance measured at 450 nm	Doxycycline	(Paul *et al.*, 2006)
IL-1β ELISA	Human Interleukin-1 beta	96-well pre-coated microplate capture system	Absorbance at 450 nm against calibration curve	Dexamethasone	(Kosoko, 2022)
TNF-α ELISA	Tumor Necrosis Factor Alpha	Antibody coating with target herbal extracts	Colorimetric response evaluation at 450 nm	Dexamethasone	(De Rubis, 2023)
Tyrosinase Assay	Mushroom Tyrosinase / L-DOPA	Phosphate buffer (pH 6.8), dopachrome detection	Optical measurement at 490 nm wavelength	Kojic Acid	(Wang *et al.*, 2021)

other steroids such as dexamethasone may be able to use less amounts of steroids, which means less risk of secondary systemic infections or delayed wound healing (Stati *et al.*, 2020). Furthermore, its unique anti-fibrotic effects could potentially be beneficial in preventing long-term lung scarring and remodeling in those who have recovered from an acute case of respiratory illness, a condition frequently observed after severe respiratory disease (Yu *et al.*, 2025). Future studies should go beyond the lab bench to living models:

In Vivo Validation
Evaluation of extract in animal model of acute lung injury (ALI) induced by lipopolysaccharide (LPS) for safety assessment and for protection of tissues.

Phytochemical Standardization
Developing uniform quality and strength through the use of liquid chromatography-mass spectrometry (HPLC-MS) standards for regional cultivars of plants.

Targeted Biomarker Tracking
Systematic clinical trial studies to comprehensively characterize its therapeutic effects in humans, which would include monitoring of specific inflammatory markers (e.g. CRP, active-matrix metalloproteinases, plasma cytokine levels).
Rather than replacing existing medical treatments, *Curcuma longa* leaf extract shows great

Conclusions
The extract of the leaves of Curcuma longa is therefore an excellent natural therapeutic option for the treatment of acute respiratory distress syndrome due to its multi-target effects. This plentiful natural material is able to exert multiple effects that limit the cytokine storm, inhibit the matrix metalloproteinases and reduce oxidative stress in the lungs, thus impacting several important pathways of tissue injury in the lungs. Advanced nanomedicine delivery systems can maximize its therapeutic effects, although issues exist in terms of oral bioavailable and standardized formulations. The potential benefit of this resource is a cost-effective, sustainable, and effective approach to managing respiratory health.

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Ethics Statement: This research was conducted in accordance with standard laboratory ethical guidelines for in vitro studies. The study did not involve human or animal clinical trials; therefore, institutional review board (IRB) approval was not required for the experimental protocols described.

Author's Contribution: MSQ and SS performed the research, MK and HN performed statistical analysis, MZM and NA drafted the manuscript, and MF supervised the project and prepared the final draft.

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