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Review Article

A Review of Medicinal Plants Utilized for Treating Chronic Obstructive Pulmonary Disease (COPD)

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Abstract

Chronic Obstructive Pulmonary Disease (COPD) represents a progressive and irreversible inflammatory lung disorder characterized by chronic airflow limitation, persistent respiratory symptoms, and significant extrapulmonary effects. This article presents a comprehensive exploration of COPD, focusing on its pathophysiology, epidemiology, etiological factors, clinical manifestations, diagnostic protocols, staging, and current therapeutic strategies. The disease encompasses two primary phenotypes: chronic bronchitis and emphysema, both resulting in obstructed pulmonary airflow due to structural lung damage and inflammatory response to noxious particles or gases, predominantly tobacco smoke. The systemic impact of COPD extends beyond the lungs, often resulting in cardiovascular complications, skeletal muscle dysfunction, and metabolic syndromes. Emphasis is placed on the role of oxidative stress, protease-antiprotease imbalance, and chronic inflammatory mediators in the progression of the disease. The article further investigates advanced diagnostic tools, including spirometry, imaging modalities, and biomarker analysis. Current pharmacological and non-pharmacological interventions are critically reviewed, highlighting bronchodilators, corticosteroids, pulmonary rehabilitation, and emerging therapies targeting specific molecular pathways. The potential of gene-based therapies, biologics, and stem cell approaches are also discussed in the context of future innovation. This work is intended to serve as a foundational reference for the development of novel diagnostic and treatment modalities and to aid in the formulation of patentable innovations targeting COPD management and control.

Keywords: Chronic Obstructive Pulmonary Disease (COPD); Spirometry

Introduction

Chronic Obstructive Pulmonary Disease (COPD) is a major global health burden characterized by persistent respiratory symptoms and airflow limitation due to airway and/or alveolar abnormalities. These abnormalities are usually the result of significant exposure to noxious particles or gases, most commonly tobacco smoke, but also environmental pollutants, occupational hazards, and genetic factors. COPD is a progressive, non-reversible disease that ranks among the leading causes of morbidity and mortality worldwide, significantly impacting the quality of life and imposing a substantial socioeconomic burden on healthcare systems.

The pathology of COPD encompasses a spectrum of pulmonary alterations, primarily chronic bronchitis and emphysema. Chronic bronchitis is defined by chronic productive cough for at least three months over two consecutive years, associated with inflamma-

tion and hypersecretion of mucus in the bronchi. Emphysema involves the destruction of alveolar walls and permanent enlargement of the airspaces distal to the terminal bronchioles, leading to impaired gas exchange and reduced lung elasticity. Although traditionally categorized as separate entities, these pathologies frequently coexist in varying proportions in individual patients, necessitating a unified approach to diagnosis and management under the umbrella of COPD.

From a pathophysiological perspective, COPD is driven by a complex interplay of inflammatory processes, oxidative stress, and an imbalance between proteases and antiproteases within the lung parenchyma. These mechanisms contribute to structural remodeling of the airways, loss of alveolar attachments, and increased mucus production—hallmarks of airflow obstruction. Systemic inflammation also plays a critical role, contributing to comorbid conditions such as cardiovascular disease, metabolic syndrome, and osteoporosis, which further complicate disease management.

Despite being preventable and treatable, COPD is frequently underdiagnosed, especially in its early stages, when therapeutic interventions are most effective. Timely diagnosis and appropriate disease staging through tools such as spirometry, imaging studies, and biomarker profiling are essential for guiding management strategies. Conventional treatment options focus on symptom control, exacerbation prevention, and quality-of-life improvement through pharmacologic agents (such as bronchodilators and corticosteroids), pulmonary rehabilitation, and lifestyle modifications. However, the disease continues to progress in many patients, underscoring the need for innovative therapeutic approaches.

This article aims to present an in-depth examination of COPD from a biomedical and clinical perspective, exploring its epidemiological trends, etiological drivers, cellular and molecular mechanisms, diagnostic frameworks, and current as well as emerging treatment modalities. The goal is to provide a scientific basis for the development of novel technologies and interventions that may be considered for intellectual property protection and future implementation in clinical practice.

Epidemiology of COPD

Chronic Obstructive Pulmonary Disease (COPD) is a significant public health challenge, with a growing prevalence that is closely linked to environmental exposure, behavioral patterns, socio-economic status, and demographic transitions. As of the most recent global estimates, COPD affects over 390 million individuals worldwide and is currently ranked as the third leading cause of death, according to the World Health Organization (WHO). This increasing prevalence, despite improvements in healthcare access and technology, highlights the multifactorial and systemic nature of the disease and underscores the urgency for preventive strategies and therapeutic innovation.

Global Prevalence and Incidence

COPD prevalence exhibits considerable geographical variation, influenced by regional smoking rates, air pollution levels, occupational exposures, and access to medical services. The highest burden is observed in low- and middle-income countries (LMICs), where biomass fuel combustion and inadequate respiratory protection contribute to significant morbidity, particularly among women and children. In high-income countries, the primary etiological agent remains cigarette smoking, although improved air quality and anti-smoking campaigns have led to a plateau or slight decline in incidence.

According to the Global Burden of Disease (GBD) study, the age-standardized prevalence rate of COPD was approximately 5.9% globally in adults over 40 years of age, with marked underdiagnosis in early-stage cases. Notably, COPD prevalence increases steeply with age, peaking in individuals aged 65 years and older. The disease exhibits a male predominance historically, though recent trends show rising prevalence in females due to increased smoking rates and higher exposure to indoor air pollutants in domestic settings.

Mortality and morbidity trends

COPD accounts for over 3.2 million deaths annually, with over 90% of these deaths occurring in LMICs. The chronic nature of the

disease leads to progressive decline in pulmonary function and frequent exacerbations, which are associated with hospitalizations, significant health costs, and elevated mortality rates. Exacerbation-related hospital admissions are a major contributor to the economic and social burden of COPD, with a 1-year post-hospitalization mortality rate ranging from 20% to 40%, depending on disease severity and comorbid conditions.

Risk factors and at-risk populations

While cigarette smoking remains the predominant risk factor globally, non-smoking-related COPD constitutes a substantial and growing proportion of cases. Risk factors contributing to COPD epidemiology include:

- Indoor air pollution, particularly from burning biomass fuels for cooking and heating.
- Occupational exposure to dust, vapors, chemicals, and fumes.
- Ambient air pollution, especially in densely populated urban areas.
- Genetic predispositions, such as α1-antitrypsin deficiency.
- Early-life events, including low birth weight, childhood respiratory infections, and suboptimal lung development.

At-risk populations also include individuals with low socioeconomic status, inadequate nutrition, and limited access to health education and preventive care.

Epidemiological surveillance and reporting gaps

Despite its significant burden, COPD remains underdiagnosed and underreported, particularly in rural and underserved regions. Limitations in access to spirometry and standardized diagnostic tools, coupled with low awareness among primary care providers, contribute to delayed diagnosis and missed intervention opportunities. Furthermore, inconsistent case definitions, variable diagnostic criteria, and lack of integration of COPD into national surveillance systems impair accurate epidemiological tracking.

Future projections

Modeling studies predict a continued rise in COPD prevalence and mortality over the coming decades, particularly in regions undergoing rapid industrialization without adequate environmental regulation. The anticipated demographic shift toward an aging population further exacerbates this trend, positioning COPD as a key target for future healthcare policies, research funding, and therapeutic development.

Predisposing factors for COPD

Chronic Obstructive Pulmonary Disease (COPD) arises from a complex interaction between environmental exposures and individual susceptibility. While exposure to inhaled irritants—most notably tobacco smoke—is the primary external trigger, not all individuals exposed to such agents develop the disease. This disparity underscores the importance of various predisposing factors that enhance vulnerability to COPD, either by amplifying the inflammatory response to insults or by impairing the repair and defense mechanisms of the pulmonary system.

The following are the principal predisposing factors associated with COPD:

- **Tobacco Smoking:** Cigarette smoking is the most well-established and potent risk factor for COPD, accounting for up to 80–90% of cases in high-income countries. The inhalation of tobacco smoke triggers a chronic inflammatory response in the airway epithelium, leading to mucosal damage, impaired mucociliary clearance, neutrophilic infiltration, and eventual parenchymal destruction. The risk correlates directly with smoking intensity and duration, as measured by pack-years. Importantly, passive smoke exposure or second-hand smoke also increases the risk of developing COPD, particularly in children and non-smoking adults.
- **Indoor and Outdoor Air Pollution:** Prolonged exposure to biomass fuel combustion—wood, charcoal, animal dung, and crop residues—is a significant risk factor, especially in rural and low-resource settings where such materials are used for cooking and heating. Women and children are disproportionately affected due to domestic exposure. Urban air pollutants, including nitrogen dioxide (NO_2), ozone (O_3), sulfur dioxide (SO_2), and particulate matter (PM2.5), are known to contribute to airway inflammation and exacerbate existing COPD.
- **Occupational Exposures:** Individuals employed in industries involving dust, vapors, gases, and fumes (VGDF)—such

as mining, textile manufacturing, construction, welding, and agriculture—are at elevated risk of developing COPD, even in the absence of smoking. Chronic inhalation of these irritants causes persistent airway inflammation and fibrotic changes, especially when exposure is unregulated or protective equipment is not used consistently.

- **Genetic Susceptibility:** Although rare, alpha-1 antitrypsin (AAT) deficiency is a well-documented genetic disorder that predisposes individuals to early-onset emphysema, particularly in the lower lobes of the lungs. AAT is a protease inhibitor that neutralizes neutrophil elastase; its deficiency allows unchecked proteolytic activity, resulting in alveolar wall destruction. Beyond AAT deficiency, emerging genome-wide association studies (GWAS) have identified other genetic polymorphisms associated with increased COPD susceptibility, particularly in genes related to inflammation, oxidative stress, and lung development.
- **Age and Gender:** COPD prevalence increases significantly with age, typically manifesting in individuals over the age of 40. Age-related decline in lung function, combined with cumulative environmental exposure, contributes to disease onset in older adults. Historically, males exhibited higher COPD prevalence due to higher smoking rates and occupational exposures; however, this gender gap is narrowing as more women smoke and are exposed to indoor air pollutants. Additionally, women may be more biologically susceptible to the harmful effects of smoke due to smaller airway calibers and hormonal influences on inflammatory pathways.
- **Early-Life Events:** Lung health in adulthood is strongly influenced by early-life factors, including:

    - Low birth weight and prematurity, which impair pulmonary development.
    - Childhood respiratory infections, especially viral infections like RSV (respiratory syncytial virus), which cause long-term airway remodeling.
    - Environmental tobacco smoke during infancy and childhood, leading to impaired lung growth and increased airway hyperresponsiveness.

    - These factors can result in suboptimal peak lung function in early adulthood, which may predispose individuals to accelerated lung function decline and early-onset COPD when exposed to environmental irritants later in life.

- **Asthma and Airway Hyperresponsiveness:** Patients with a history of chronic asthma, particularly those with poor disease control, may be at higher risk of developing fixed airflow obstruction resembling COPD. Chronic airway inflammation, bronchial remodeling, and overlapping phenotypes (often referred to as Asthma-COPD Overlap Syndrome, or ACOS) contribute to the development of COPD-like pathology.
- **Nutritional Deficiency and Low Body Mass Index (BMI):** Malnutrition and low BMI have been associated with increased susceptibility to pulmonary infections and impaired immune responses. Deficiencies in antioxidants such as vitamin C, E, and beta-carotene may exacerbate oxidative damage in the lung tissue, especially under sustained exposure to irritants.
- **Socioeconomic Status (SES):** Low SES is correlated with increased risk for COPD due to multiple converging factors, including poor housing conditions, limited access to healthcare, poor nutrition, increased occupational exposures, and higher smoking rates. Education level and income status also influence disease awareness and access to preventive measures.

Pathogenesis of COPD

The pathogenesis of Chronic Obstructive Pulmonary Disease (COPD) is characterized by a complex interplay between environmental insults and host responses, resulting in chronic inflammation, tissue remodeling, and irreversible airflow limitation. The disease process involves both large and small airways, alveolar structures, and the pulmonary vasculature, leading to a progressive decline in lung function. Understanding the pathogenesis is essential for identifying novel therapeutic targets and designing disease-modifying interventions.

Inhalation of Noxious Stimuli: The initiating event

The pathogenic cascade of COPD begins with the chronic inhalation of noxious agents, most commonly cigarette smoke, biomass fuel emissions, industrial pollutants, and environmental particulate matter. These substances contain a wide range of oxidants and toxic chemicals that initiate a local immune response in the lungs. Airway epithelial cells and alveolar macrophages recognize these irritants and respond by releasing pro-inflammatory mediators such as interleukin-8 (IL-8), tumor necrosis factor-alpha (TNF-α), and granulocyte-macrophage colony-stimulating factor (GM-CSF).

Persistent inflammation and immune dysregulation

Unlike acute inflammation, which resolves upon removal of the irritant, COPD is driven by chronic, non-resolving inflammation. Key features of this persistent inflammatory state include:

- Neutrophilic infiltration in the airways, particularly in chronic bronchitis.
- CD8+ cytotoxic T lymphocyte predominance, which contributes to alveolar wall destruction.
- Macrophage activation, which sustains the inflammatory response by producing matrix metalloproteinases (MMPs) and reactive oxygen species (ROS).
- Dysregulated innate immunity, including altered responses by dendritic cells and natural killer (NK) cells.

This chronic inflammation leads to structural changes in the airways and alveolar units, resulting in permanent airflow obstruction.

Oxidative stress

Oxidative stress is a central mechanism in COPD pathogenesis. Inhaled oxidants and endogenously generated reactive oxygen species (ROS) overwhelm the lung's antioxidant defenses, leading to:

- Lipid peroxidation and damage to cell membranes.
- Oxidative modification of proteins and DNA.
- Inactivation of antiproteases such as α1-antitrypsin.
- Upregulation of pro-inflammatory gene expression via transcription factors like NF-κB and AP-1.

The imbalance between oxidants and antioxidants contributes to tissue injury, mucus hypersecretion, and chronic inflammation.

Protease–antiprotease imbalance

The balance between proteolytic enzymes (e.g., neutrophil elastase, MMPs) and their natural inhibitors (e.g., α1-antitrypsin, tissue inhibitors of metalloproteinases—TIMPs) is crucial for maintaining lung tissue integrity. In COPD:

- Protease activity is markedly increased due to sustained neutrophil and macrophage infiltration.
- Antiprotease activity is reduced either by genetic deficiency (as in α1-antitrypsin deficiency) or oxidative inactivation.

This imbalance leads to destruction of elastin fibers and degradation of extracellular matrix (ECM), resulting in emphysematous changes and loss of alveolar attachments that support the small airways.

Airway remodeling and mucus hypersecretion

Chronic inflammation triggers structural remodeling of the airways, involving:

- Goblet cell hyperplasia and mucous gland hypertrophy, leading to excessive mucus production.
- Fibrosis of the small airways due to fibroblast proliferation and collagen deposition.
- Smooth muscle hyperplasia and increased airway wall thickness.
- These changes contribute to fixed airflow obstruction, particularly in the small airways (<2 mm in diameter), where resistance to airflow is most pronounced.

Alveolar destruction and emphysema formation

In emphysema-dominant COPD, the destruction of alveolar septa and capillary networks leads to:

- Airspace enlargement beyond the terminal bronchioles.
- Loss of alveolar surface area for gas exchange.
- Reduced elastic recoil, impairing expiratory airflow.

This results in air trapping, hyperinflation, and increased work of breathing, particularly during exertion.

Pulmonary vascular changes

In addition to airway and parenchymal involvement, COPD is associated with pulmonary vascular remodeling, which includes:

- Endothelial dysfunction and intimal thickening.
- Smooth muscle hypertrophy in pulmonary arterioles.
- Hypoxic vasoconstriction.

These changes contribute to pulmonary hypertension, particularly in advanced stages, and increase the risk of right heart failure (cor pulmonale).

Systemic Inflammation and Extrapulmonary Effects

COPD is now recognized as a systemic disease with extrapulmonary manifestations. Chronic pulmonary inflammation can spill over into systemic circulation, leading to:

- Skeletal muscle wasting and cachexia.
- Cardiovascular disease, including coronary artery disease and stroke.
- Metabolic abnormalities, such as insulin resistance.
- Osteoporosis and anemia.

These comorbidities significantly affect the prognosis, morbidity, and mortality associated with COPD.

Medicinal plants used for COPD

The growing interest in plant-based medicine has brought renewed attention to the therapeutic potential of medicinal plants in the management of Chronic Obstructive Pulmonary Disease (COPD). Several botanicals exhibit anti-inflammatory, antioxidant, bronchodilatory, immunomodulatory, and mucolytic properties—mechanisms that align with key pathological targets in COPD. These properties make medicinal plants valuable not only in symptom management but also in slowing disease progression and enhancing lung function.

Below is a comprehensive overview of select medicinal plants with documented or emerging relevance in COPD therapy.

Adhatodavasica (Malabar Nut)

- **Active compounds**: Vasicine, vasicinone.

Pharmacological actions

- Bronchodilator and expectorant activity
- Reduces bronchial congestion and enhances mucociliary clearance
- Inhibits pro-inflammatory cytokines such as TNF-α and IL-6.
- **Relevance in COPD:** Used in Ayurvedic and Unani systems to relieve chronic cough, wheezing, and breathlessness. Vasicine has been shown to improve airflow and reduce airway resistance in animal models.

Glycyrrhiza glabra (Licorice Root)

- **Active compounds**: Glycyrrhizin, liquiritin, flavonoids.

Pharmacological actions

- Potent anti-inflammatory and antioxidant
- Inhibits phospholipase A2 and cyclooxygenase pathways
- Soothes inflamed airways and suppresses mucus hypersecretion.
- **Relevance in COPD:** Helps attenuate oxidative stress and airway remodeling. Widely used in traditional Chinese medicine for chronic respiratory disorders.

Tylophora indica

- **Active compounds**: Tylophorine alkaloids.

Pharmacological actions

- Immunosuppressive and anti-allergic
- Inhibits mast cell degranulation and histamine release
- Promotes bronchodilation and relieves dyspnea.
- **Relevance in COPD:** Traditionally used for bronchitis and asthma; recent studies suggest benefit in inflammatory airway diseases including COPD.

Curcuma longa (Turmeric)

- **Active compounds**: Curcumin, demethoxycurcumin.

Pharmacological actions

- Inhibits NF-κB signaling and downstream inflammatory cascades
- Scavenges reactive oxygen species (ROS)
- Modulates gene expression of inflammatory cytokines.
- **Relevance in COPD:** Curcumin has been shown to reduce airway inflammation, oxidative damage, and remodeling in preclinical COPD models. Also supports immune regulation.

Ocimum sanctum (Holy Basil)

- **Active compounds**: Eugenol, ursolic acid, rosmarinic acid.

Pharmacological actions

- Antioxidant and anti-inflammatory
- Enhances pulmonary function and reduces allergic responses
- Inhibits lipid peroxidation and neutrophil infiltration.
- **Relevance in COPD:** Protects against oxidative and inflammatory damage to lung tissues; improves respiratory endurance and comfort.

Solanum xanthocarpum

- **Active compounds**: Solasodine, solanine.

Pharmacological actions

- Anti-inflammatory and mucolytic
- Reduces bronchial inflammation and airway resistance.
- **Relevance in COPD**: Often used in combination with other herbs for treatment of chronic bronchitis and productive cough associated with COPD.

Zingiber officinale (Ginger)

- **Active compounds**: Gingerols, shogaols.

Pharmacological actions

- Inhibits inflammatory enzymes (COX-2, LOX)
- Antioxidant and smooth muscle relaxant
- Enhances mucociliary clearance.
- **Relevance in COPD**: Promotes bronchodilation and reduces airway inflammation, with potential synergistic effects when used alongside conventional bronchodilators.

Withaniasomnifera (Ashwagandha)

- **Active compounds**: Withanolides, sitoindosides.

Pharmacological actions

- Adaptogenic and anti-inflammatory
- Modulates stress response and improves pulmonary endurance
- Immunomodulatory action through cytokine balance.
- **Relevance in COPD**: Supports systemic resilience, mitigates fatigue, and enhances pulmonary rehabilitation outcomes in COPD patients.

Mechanisms of action across plant-based therapies

Most of the above medicinal plants exert therapeutic effects via one or more of the following mechanisms relevant to COPD:

- Inhibition of inflammatory mediators (e.g., TNF-α, IL-1β, IL-6)
- Antioxidant activity, neutralizing ROS and reducing oxidative tissue damage
- Bronchodilation, mediated via β2-agonist-like activity or smooth muscle relaxation
- Mucolysis and expectorant action, aiding in clearing mucus buildup
- Immunomodulation, restoring balance between pro- and anti-inflammatory responses
- Inhibition of NF-κB and MAPK pathways, central to inflammation and remodeling.

Formulations and delivery

Medicinal plants are commonly delivered through decoctions, capsules, syrups, inhalants, or incorporated into polyherbal formulations. Some modern approaches explore standardized extracts and nano formulations to enhance bioavailability and therapeutic efficacy. Research into herb-drug interactions and pharmacokinetic profiling is ongoing to ensure safe integration with standard COPD treatments [1-14].

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