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Review Article

Reviews on New Pharmacological Interventions for Redefining COPD Management

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ABSTRACT

Chronic obstructive pulmonary disease is characterized by obstructed airflow, persistent inflammation of the airways, and structural alterations in the lungs. It is a heterogeneous, progressive pulmonary disorder. However, despite the wide use of long-acting bronchodilators and inhaled steroids, many patients continue to suffer from persistent symptoms, exacerbations, and an accelerated decline in lung function. This disconnect represents an unmet need for more powerful and effective treatments. New dual-function bronchodilators such as ensifentrine and immunomodulatory or anti-inflammatory medications represent a trend toward treatments that do more than simply dilate the airways. It would signal a shift toward targeting the core processes driving this disease, rather than merely symptom relief. Large COPD cohorts do not respond uniformly to biologic therapies. There is an increasing body of evidence that certain phenotypes, in particular those with eosinophilic or type-2 inflammatory features, derive true benefit from specific genetic variants. Still in the trial stage of development, cellular and regenerative strategies, such as mesenchymal stem cells and those employing extracellular vesicles, hold real promise to rebuild injured lung tissue. Ultimately, COPD care is shifting toward precision medicine: utilizing digital health tools, biomarkers, imaging, and multi-omics profiles to create personalized treatments for each individual.Standing back and considering all these developments together, there is cause for guarded optimism that in the not-too-distant future, we might move away from the mainly symptom-based strategy toward approaches more steeped in underlying mechanisms that result in improved long-term outcomes and slower disease progression.

INTRODUCTION

Chronic obstructive pulmonary disease remains one of the most important causes of morbidity and mortality worldwide, but the burden is increasing

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in low- and middle-income countries. Classically defined by the presence of persistent airflow limitation, COPD is now considered a heterogeneous, multicomponent disease characterized by complex interplay between genetic susceptibility, environmental exposures (such as tobacco smoke, biomass fuel, and air pollution), and abnormal inflammatory response.[1] Over the past twenty years, GOLD has evolved from a spirometry-centered classification to a multidimensional approach that integrates symptoms, exacerbation history, and biomarkers such as blood eosinophil count to guide therapy.[2] Despite significant advances in both pharmacological and non-pharmacological interventions, COPD still causes a high health and economic burden worldwide, which underlines the need for therapeutic reevaluation.[3]The optimization of conventional inhaled therapy, including long-acting β_2-agonists, long-acting muscarinic antagonists, and inhaled corticosteroids, many patients continue to experience exacerbations and progressive lung function decline. This has led to the exploration of new pharmacological treatments, especially biologic therapies targeting specific inflammatory pathways, including type-2 inflammation. New evidence shows that monoclonal antibodies, such as dupilumab, mepolizumab, benralizumab, and tezepelumab, may reduce exacerbation rates significantly, improve lung function, and enhance the quality of life in eosinophilic COPD subpopulations. These new advances represent a major step toward precision immunomodulation in COPD management.[4]

The current management of COPD is aimed at alleviating symptoms, preventing exacerbations, and slowing the progression of the disease. The pharmacological therapy is based on long-acting bronchodilators, namely, LABA and LAMA, usually combined with ICS in selected patients. While these drugs improve lung function, exercise tolerance, and quality of life, a large number of patients continue to suffer from persistent exacerbations and inflammation despite maximal inhaled therapy. This "residual risk" underlines the unmet need for innovative treatment strategies targeting underlying molecular pathways beyond bronchodilation and corticosteroid effects. In recent years, there has been the development of biologic and precision-targeted therapies aimed at the modulation of specific inflammatory mediators implicated in the pathogenesis of COPD. These have shown benefits in COPD subsets characterized by type 2 (T2) inflammation, especially among patients with elevated blood eosinophil counts. These monoclonal antibodies reduce the frequency of exacerbation, improve lung function, and enhance health status; hence, a paradigm shift toward immunomodulatory precision therapy in COPD[2]. The success of these interventions underlines the importance of identifying biomarker-defined responder populations that optimize treatment outcomes and minimize unnecessary exposure to broad anti-inflammatory drugs. Meanwhile, studies in genetic epidemiology and multi-omics have produced new insights into the heterogeneity of COPD. Now, investigations are focused on disease endotypes based on shared biological mechanisms rather than clinical features alone. Precision medicine approaches that incorporate genomics, transcriptomics, proteomics, and metabolomics seek to personalize treatment by linking an individual's molecular signature to specific pharmacologic targets; this approach offers hope for early detection of at-risk individuals damage[4].

Novel Bronchodilator Therapies:

Novel bronchodilator therapies are transforming the management of COPD. There is emerging evidence that FEV_1 as a sole measure of airflow

limitation does not capture symptom burden or dyspnea severity and exacerbation risk; it therefore has limited utility to guide bronchodilator choice. Small-airway inflammation, hyperinflation, and emphysema all are important contributors to clinical impairment and often persist despite traditional bronchodilator regimens, further supporting advanced therapeutic approaches[5]. Dual LABA/LAMA combinations represent a major advance, improving dyspnea, lung function, and quality of life across all severities of airflow obstruction, extending treatment decisions from spirometric thresholds to symptom and exacerbation profiles[5]. Similarly, triple therapy ICS/LABA/LAMA has also demonstrated superior bronchodilation and exacerbation reduction even in less severe obstruction, further reinforcing the move toward personalized therapy based on treatable traits. Precision medicine has further supported the idea that some airway-predominant phenotypes and particular molecular endotypes may be more susceptible to bronchodilators. Omics and imaging are increasingly important for tailored therapies. Lastly, via improved deposition and adherence, new inhaler platforms and novel smart-delivery systems are further enhancing practical efficacy of bronchodilators. In combination, these advances bring forth a new era in precision bronchodilation for COPD, matching therapy to clinical heterogeneity and extending beyond conventional spirometry-guided approaches[6].

Anti-Inflammatory and Immunomodulatory Agents :

Immunomodulatory and anti-inflammatory drugs are becoming important parts of COPD treatment, especially for patients who, even with the best bronchodilator therapy, continue to present exacerbation and chronic symptoms. Ensifentrine is the first-in-class inhaled dual PDE3/4 inhibitor, targeting simultaneously inflammatory pathways and airway smooth-muscle tone, thus possessing both bronchodilator and anti-inflammatory properties, representing a major step forward in COPD management. Ensifentrine decreases the number of inflammatory cells in sputum, including neutrophils, eosinophils, macrophages, and lymphocytes, following the lipopolysaccharide challenge in clinical trials, mediated through inhibition of PDE4, which blocks the release of several pro-inflammatory mediators from neutrophils, eosinophils, monocytes, and macrophages[7]. Its potent immunomodulatory profile is also demonstrated in preclinical models, showing marked inhibition of eosinophil recruitment, along with reduced cytokine production and suppression of TNF-α release.In this context, clinical research showing that ensifentrine has the ability to reduce moderate and severe exacerbations by 36–43% over a period of 24 weeks provides further evidence that ensifentrine's anti-inflammatory properties yield clinically significant results. While tailored immunomodulators are still in the research phases, conventional anti-inflammatory therapies, such as inhaled corticosteroids, do benefit eosinophilic phenotypes except for ensifentrine[8].

Biologic Therapies in COPD :

Although biologic medications have been studied in depth as targeted treatments for COPD, with the aim of modifying certain inflammatory pathways, data indicate that their clinical efficacy remains limited in most patients with COPD. Several biologics targeting eosinophilic or type-2-driven inflammation, such as mepolizumab, benralizumab, and dupilumab, have been tested in large clinical trials. However, most studies have failed to demonstrate consistent, clinically meaningful improvements in lung function or exacerbation reduction in unselected COPD

cohorts 1[9]. The primarily neutrophilic, steroid-resistant, and heterogeneous inflammatory profile of COPD, very different from asthma, the disease for which these biologics were initially developed, is for the most part responsible for the poor effect 1. New evidence, however, suggests that biologics may still play a role in some COPD phenotypes, particularly in patients with high circulating eosinophils or those with asthma-COPD overlap, in whom suppression of the IL-5 or IL-4/13 pathway may improve outcomes 1[9]. Novel immune targets under current investigation include the epithelial cytokines and pathways related to macrophages, although these are still in the early phases of exploration 1. It is hoped that, although biologics have not reached the same therapeutic efficacy in COPD as they have in asthma, a precision medicine approach driven by biomarkers, imaging features, and endotype definition will help in further defining biologics' future role in COPD management[10].

Mucokinetic and Mucoregulatory Drugs :

Mucokinetic drugs increase ciliary activity, decrease mucus adhesiveness, or improve airflow so as to promote the expectoration of secretions from the airways. Examples are ambroxol, which increases surfactant production to enhance a reduction in viscosity and adhesiveness of mucus, and β_2-agonists, which raise ciliary beat frequency and improve mucociliary transport. Mucoregulatory medications, on the other hand, inhibit excessive production or alter the composition of mucus at the glandular level[11][12].Examples include corticosteroids, lowering mucus hypersecretion by modifying the inflammatory process; macrolides that suppress mucin gene expression by exerting an immunomodulatory effect; and anticholinergics such as ipratropium and tiotropium, which reduce glandular secretion by blocking muscarinic receptors[13]. Carbocysteine is a well-studied mucoregulatory drug that has antioxidant properties and restores normal viscoelasticity to mucus by regulating the appropriate ratio between sialomucin and fucomucin. The combined use of mucokinetic and mucoregulatory medications can offer symptomatic relief such as the reduction of cough, sputum production, and airway blockage. However, clinical evidence may vary depending on the phenotype of the patient and the severity of the disease. Trials in conditions such as COPD and ventilated critical-care patients have outlined only a limited, context-specific benefit in this regard[14].

Antioxidant and Redox-Modulating Therapies:

Antioxidant and redox-modulating therapies represent new promising pharmacological approaches in COPD, as oxidative stress is recognized as a central driver of airway inflammation, structural lung damage, and corticosteroid resistance. COPD is defined as a persistent imbalance between oxidants, originating from cigarette smoke and environmental pollutants on one side, and the endogenous antioxidant defenses, mainly reduced glutathione (GSH)[15]. This oxidative burden contributes to epithelial apoptosis, mucus hypersecretion, protease-antiprotease imbalance, and accelerated disease progression. Thus, therapeutic approaches aimed at restoring redox balance have gained significant attention in the redefinition of COPD management[16]. NAC increases intracellular GSH and is a direct ROS scavenger. The HIACE and PANTHEON clinical trials reported that high-dose, prolonged administration of NAC resulted in a significant decrease in the frequency of exacerbation and symptomatic improvement in stable COPD patients, particularly in those with the chronic bronchitis phenotype[16][17]. A thiol-based mucoregulatory antioxidant, carbocisteine, has been shown to decrease oxidative biomarkers

as well as the rate of exacerbation in the PEACE study and seems to be an effective adjuvant therapy[18]. Recent developments target treatments directed against the Nrf2 pathway. While Nrf2 controls over 200 genes for antioxidants and cytoprotective enzymes, its activity is severely suppressed in COPD patients because of chronic oxidative load and/or reduction in histone deacetylase-2 expression [19]. Sulforaphane and synthetic Nrf2 activators have so far demonstrated restoring antioxidant defenses, increasing synthesis of GSH, and reinstating corticosteroid sensitivity through upregulation of HDAC2 levels[20][21]. Though still in the trial stage, the results so far highlight the potential for Nrf2 activators to be disease-modifying agents. Another area of growth is mitochondrially targeting antioxidants, including MitoQ and SkQ1. These antioxidants selectively accumulate in mitochondria, the primary site of intracellular ROS production. It has been hypothesized that mitochondrial dysfunction might play an active role in the pathophysiology of COPD through a variety of mechanisms, including structural damage, hampered ATP generation, and cellular senescence[22]. MitoQ has been described, in preclinical models, to decrease inflammation, improve endothelial function, and reduce mitochondrial oxidative stress[11]. Clinical evidence of these drugs is scant in COPD; however, they are among very promising agents acting at the source of oxidative damage.Clinical data in COPD are lacking, but these agents represent promising candidates for the precision targeting of oxidative injury. Moreover, there has been interest in inhibiting certain key ROS-producing enzymes. NOX inhibitors, especially those of NOX2 and NOX4, have shown the potential to decrease airway remodeling, mucus production, and the release of pro-inflammatory cytokines in experimental models of COPD[12]. Inhibitors of xanthine oxidase, including allopurinol, have also been tested for their ROS-reducing action. Although studies indicate systemic oxidative stress is reduced, direct clinical benefits in COPD remain to be determined so far[13]. Although encouraging, variability in clinical response also suggests heterogeneity in COPD and phenotype specificity in antioxidant strategies. Challenges persist for optimal dosing, treatment duration, and delivery, especially in inhaled antioxidant formulations. Not with standing such limitations, antioxidant and redox-modulating therapies therefore represent an area that is rapidly evolving within COPD pharmacotherapy, in which new agents and strategies are being developed with the goal of slowing disease progression, reducing exacerbations, and improving corticosteroid and other anti-inflammatory agent effectiveness[20].

Regenerative And Cellular Therapies :

Cellular and regenerative treatments represent a new frontier in the treatment of COPD, with an emphasis on lung tissue regeneration and functional capacity restoration rather than only palliative care. MSCs are intriguing options for the therapy of COPD because they have been shown to possess strong immunomodulatory, anti-inflammatory, and tissue-repair properties. They can be produced from bone marrow, adipose tissue, or umbilical cord sources. Preclinical studies have demonstrated that MSCs reduce airway inflammation, inhibit neutrophil recruitment, and stimulate alveolar epithelial regeneration by paracrine signaling mechanisms[23]. Systemically given MSCs have been shown in early-phase clinical trials to be safe and well-tolerated in patients with moderate to severe COPD; nevertheless, improvements in quality of life, indicators of systemic inflammation, and frequency of exacerbations have been reported by some, but changes in lung

function have been consistently restricted[23]. Furthermore, endothelial progenitor cells and induced pluripotent stem cell-derived epithelial progenitors are investigated as a way to regenerate microvascular systems and repair emphysematous lung tissue, thereby addressing structural degradation at the center of COPD pathophysiology. The use of EV treatment, including MSC-derived exosomes, is one of the more modern approaches. This cell-free approach has strong regeneration signals and few safety issues[24]. While promising, larger controlled trials will be needed to establish long-term efficacy and optimize methods of cell delivery and characterize patient subpopulations most likely to benefit from regenerative interventions[24].

Personalized And Precision Medicine Approaches :

In contrast, regenerative and cellular therapies are emerging investigational approaches for COPD targeted at the restoration or replacement of damaged lung tissue, rather than merely symptomatic control. Among these, MSCs have gained much attention because of their anti-inflammatory, immune-modulating, and tissue-repair properties. This is based on the rationale that MSCs secrete paracrine factors capable of suppressing chronic inflammation, promoting epithelial repair, and potentially modifying disease progression. However, the pharmacological update in 2024 again highlights that despite promising safety profiles, there is still inadequate evidence to suggest routine use of MSC or other regenerative therapies in COPD management, because large-scale trials to date have not convincingly demonstrated robust clinical efficacy. Other regenerative approaches including induced pluripotent stem cells, ex-tracellular vesicle-based therapies, and gene-modulating therapies, which are at an early preclinical stage, underline further the challenges involved in the reversibility of structural lung damage. Regenerative and cellular therapies thus hold theoretical promise, but require additional mechanistic studies and high-quality trials before they can be established as part of routine COPD treatment pathways[25].

Non-Pharmacological Adjuncts with Pharmacotherapy Relevance

Among these emerging strategies, regenerative and cellular therapies have gained considerable interest to address the irreversible structural damage characteristic of chronic obstructive pulmonary disease. In these, MSCs have been the most studied, given their immunomodulatory, anti-inflammatory, and tissue-repair capability. Preclinical models indicate that MSCs exert alveolar inflammation reduction, neutrophilic infiltration inhibition, and epithelial repair promotion by paracrine signaling and growth factor release. Clinical trials such as the Phase II START study examined intravenous allogeneic MSCs in moderate-to-severe COPD, revealing a solid safety profile along with reductions in systemic inflammation markers such as C-reactive protein, although significant lung function improvements were not seen. More recent studies explore adipose-derived MSCs, bone marrow–derived MSCs, and iPSC-derived lung progenitors; all show potential to enhance alveolar regeneration and improve extracellular matrix remodeling in experimental COPD models. Another newer strategy involves the use of MSC-derived EVs that can transport pro-repair microRNAs, with a presumably lower risk compared to live-cell transplantation. Although these newer regenerative therapies are in early clinical development, ongoing advances in stem-cell engineering, exosome biology, and lung tissue biofabrication provide hope for future disease-

modifying treatment options in COPD. However, larger, well-designed trials are required to confirm long-term safety and functional benefits and to define optimal delivery strategies[26].

FUTURE DIRECTIONS :

Precision medicine, biomarker-driven therapy selection, and the development of personalized therapies that target the processes driving disease progression, rather than just symptom-based treatments, are the more promising future directions in COPD management. Advances in molecular phenotyping and imaging are foreseen to further advance treatment strategies by enabling clinicians to match therapies such as biologics, anti-inflammatory agents, or regenerative approaches to specific inflammatory endotypes[26]. Furthermore, digital health tools, AI-assisted monitoring, and smart inhaler technologies will likely lead to better adherence and real time symptom assessment, hence reducing the risk of reoccurrence of exacerbations.4 In summary, new emerging drug classes include epithelial cytokine inhibitors, new PDE inhibitors, and therapies that restore mucociliary function, which may expand the options for patients who continue to exacerbate despite optimal inhaled therapy. Further, combining pharmacotherapy with cellular and gene-based interventions may eventually provide disease-modifying potential. Thus, the future of COPD treatment is moving toward integrated, mechanism-based, and personalized care models, given the complexity and heterogeneity of this disease .

CONCLUSION :

Advances in pharmacology, immunology, and precision medicine are changing the management of COPD, as emerging therapies push beyond the traditional bronchodilators and inhaled corticosteroids. Dual-acting drugs, such as ensifentrine, targeted anti-inflammatory drugs, and specific biologics that address unmet needs in patients who continue to have persistent symptoms and exacerbations despite adjustment of conventional therapy, are examples of new classes of therapeutics. Despite there having been no overall benefit of biologic therapy to date in unselected COPD patients, early data suggests that they may prove useful in certain inflammatory phenotypes, demonstrating the need for biomarker-guided treatment strategies. Regenerative and cellular treatments, on the other hand, propose the exciting prospect of disease-modifying and tissue-healing benefits not possible with the existing pharmacological approaches, though these are still strictly experimental. Future COPD management will increasingly rely on integrated, phenotype-driven treatment models combining innovative pharmacological agents with digital health tools, advanced diagnostics, and personalized monitoring. Further elucidation of the causes of the disease and treatment targets from ongoing research has the potential to yield more precise, effective, and personalized COPD therapy, improving quality of life and long-term outcomes in patients.

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