COPD & asthma: Why symptom control isn't enough anymore

Written by The Life Science Feed Editorial Team · Published 6 September 2026 · Edited by Mara Voss

Chronic obstructive pulmonary disease (COPD) and asthma represent significant global health burdens, characterized by persistent respiratory symptoms and recurrent exacerbations. While current therapeutic approaches effectively manage symptoms and reduce exacerbation frequency, they often fall short of fundamentally altering the underlying disease trajectory. The challenge remains to move beyond mere symptom control to interventions that can modify the natural course of these progressive conditions.

KEY TAKEAWAYS

- The Pivot: The focus in COPD and asthma management is shifting from symptom control and exacerbation reduction to modifying the long-term disease trajectory.
- The Data: No specific numeric data from trials is available for this general topic, as no research papers were provided.
- The Action: Clinicians should remain aware of evolving research into disease-modifying therapies that target underlying inflammatory and structural changes, rather than solely addressing symptoms.

COPD, a progressive lung disease, is primarily driven by chronic inflammation and structural changes in the airways and lung parenchyma. This leads to irreversible airflow limitation. The disease typically manifests in middle to later life, often linked to exposure to noxious particles or gases, most commonly cigarette smoke. Patients experience dyspnea, chronic cough, and sputum production, with acute exacerbations frequently leading to hospitalisation and accelerated lung function decline. The current standard of care involves bronchodilators, inhaled corticosteroids, and phosphodiesterase-4 inhibitors, aimed at symptom relief and reducing exacerbation rates. These therapies improve quality of life and reduce acute events, but they do not halt the relentless progression of lung damage.

Asthma, by contrast, is characterised by chronic airway inflammation and hyperresponsiveness, leading to recurrent episodes of wheezing, breathlessness, chest tightness, and coughing. While often reversible, severe asthma can lead to fixed airflow obstruction and significant morbidity. The disease typically presents earlier in life and involves a complex relationship of genetic and environmental factors. Current management relies on inhaled corticosteroids, long-acting beta-agonists, and in severe cases, biologics targeting specific inflammatory pathways. These treatments effectively control symptoms and prevent exacerbations for many, but a subset of patients continues to experience poorly controlled disease, highlighting an unmet need for therapies that address the underlying pathology more profoundly.

The Unmet Need for Disease Modification

The distinction between symptom management and disease modification is important for patient outcomes. For both COPD and asthma, existing treatments largely address the consequences of the disease, such as bronchoconstriction and inflammation, rather than altering the fundamental processes that drive progression. This is particularly evident in COPD, where lung function decline continues despite optimal therapy. In asthma, while biologics have revolutionised care for severe phenotypes, they primarily target specific inflammatory mediators, reducing exacerbations but not necessarily reversing the structural changes that can develop over time. The goal of disease trajectory modification is to intervene earlier and more effectively to prevent or slow these irreversible changes.

Targeting the early inflammatory cascade and structural remodelling processes is a key area of investigation. In COPD, this involves exploring therapies that can reduce small airway fibrosis, alveolar destruction, and chronic inflammation more comprehensively than current agents. For asthma, the focus extends to preventing airway remodelling, subepithelial fibrosis, and goblet cell hyperplasia, which contribute to persistent symptoms and fixed airflow obstruction. Understanding the specific cellular and molecular pathways that drive these changes is paramount. Clinicians looking for a comprehensive overview of respiratory conditions might find the Oxford Handbook of Respiratory Medicine a useful resource.

Pathophysiological Targets Beyond Symptom Relief

Several pathophysiological mechanisms are under investigation for their potential to modify disease trajectories. In COPD, this includes targeting oxidative stress, protease-antiprotease imbalance, and senescence pathways. Oxidative stress, often induced by cigarette smoke, contributes to inflammation and tissue damage. Therapies aimed at enhancing antioxidant defences or directly neutralising reactive oxygen species could theoretically slow disease progression. Similarly, restoring the balance between proteases and antiproteases, which is disrupted in COPD, might prevent further destruction of lung tissue. Cellular senescence, the irreversible arrest of cell division, also plays a role in chronic inflammation and tissue remodelling in COPD, making senolytics a potential therapeutic avenue.

For asthma, research is exploring targets related to epithelial dysfunction, innate immune responses, and airway smooth muscle remodelling. The airway epithelium acts as a critical barrier and orchestrates immune responses. Dysfunctional epithelial cells can contribute to chronic inflammation and hyperresponsiveness. Modulating epithelial repair mechanisms or targeting specific epithelial-derived cytokines, such as IL-33, could offer new therapeutic strategies. IL-33's dual role in COPD, for instance, highlights the complexity of these pathways. Innate immune cells, including mast cells and eosinophils, also contribute to asthma pathology, and novel approaches to modulate their activity are being explored. Preventing or reversing airway smooth muscle hypertrophy and hyperplasia could reduce airway hyperresponsiveness and fixed airflow obstruction.

The Role of Early Intervention

Modifying disease trajectory likely requires earlier intervention, potentially even before significant symptoms or irreversible structural changes have occurred. This necessitates improved diagnostic tools and biomarkers that can identify individuals at high risk of progression. For COPD, this might involve screening individuals with a history of smoking or environmental exposure for subtle lung function abnormalities or inflammatory markers. In asthma, identifying phenotypes at risk of developing severe, refractory disease or airway remodelling could allow for targeted preventative strategies. The concept of severe asthma beyond airway focus underscores the need for a broader

understanding of disease drivers.

Preventative strategies could include interventions to reduce exposure to environmental triggers, such as smoking cessation programmes, which remain the most effective way to slow COPD progression. But pharmacological interventions that can mitigate the early inflammatory and remodelling processes are also needed. This might involve novel anti-inflammatory agents or therapies that promote tissue repair and regeneration. The challenge lies in identifying the optimal window for intervention and the specific patient populations most likely to benefit from these approaches. The field is moving towards a more personalised medicine approach, tailoring treatments based on individual patient characteristics and disease endotypes.

Challenges and Future Directions

Developing therapies that truly modify disease trajectory presents significant challenges. Clinical trials for such agents would need to be long-term, measuring endpoints like sustained lung function improvement, prevention of structural changes, or reduction in long-term morbidity and mortality, rather than just short-term symptom control or exacerbation rates. Identifying appropriate biomarkers that correlate with long-term outcomes is also important for accelerating drug development. The heterogeneity of both COPD and asthma, with various endotypes and phenotypes, further complicates the development of universally effective disease-modifying treatments.

The potential rewards are substantial. Shifting from managing chronic, progressive disease to altering its fundamental course could dramatically improve patient quality of life, reduce healthcare burdens, and extend healthy lifespans. This requires a deeper understanding of disease pathogenesis, innovative drug discovery, and a willingness to rethink traditional clinical trial designs. The ongoing exploration of novel targets and therapeutic modalities offers hope for a future where COPD and asthma are not just controlled, but fundamentally reshaped. The development of triple therapy for COPD has already shown the benefits of comprehensive approaches, and future innovations will likely build on this.

> **CLINICAL IMPLICATIONS**
>
> The shift towards disease trajectory modification in COPD and asthma represents a significant conceptual leap for clinicians. We have long been comfortable with managing symptoms and reducing exacerbations, but the idea of fundamentally altering the disease's natural course demands a new perspective on therapeutic goals. This means looking beyond immediate relief to long-term structural and functional preservation.
>
> For patients, this could mean a future where their conditions are not just managed, but genuinely slowed or even halted in progression. It implies a move away from a reactive treatment model to a more proactive, preventative one. The challenge will be identifying who benefits most from these potentially more intensive or novel therapies, and when to initiate them.
>
> The pharmaceutical industry faces the task of developing agents that can demonstrate true disease modification, not just symptomatic improvement. This requires investment in longer, more complex trials and a deeper understanding of underlying pathophysiology. Regulatory bodies will need to adapt their frameworks to evaluate these novel endpoints, moving beyond traditional measures of acute efficacy.
>
> The success of disease trajectory modification will depend on a collaborative effort across research, clinical practice, and industry. It demands a willingness to challenge established paradigms and embrace a more ambitious vision for respiratory health. The current focus on control and stability, while valuable, is merely a stepping stone.

EDITORIAL & AI STANDARDS

AI tools are used solely to structure and summarise evidence from peer-reviewed primary sources. No AI-generated conclusions appear in The Life Science Feed without editor verification against the primary source.

REFERENCES

1. 1. Born CDC, Bhadra R, D'Souza G, et al. Combined Lifestyle Interventions in the Prevention and Management of Asthma and COPD: A Systematic Review. Nutrients. 2024;16(10). doi:10.3390/nu16101515
2. 2. Venkata AN. Asthma-COPD overlap: review of diagnosis and management. Curr Opin Pulm Med. 2020;26(2):155-161. doi:10.1097/MCP.0000000000000649
3. 3. Polverino F, Sin DD. The Developmental Origins of Asthma and COPD. Annu Rev Physiol. 2026;88(1):513-535. doi:10.1146/annurev-physiol-042924-084007
4. 4. Tanabe N, Nakagawa H, Sakao S, et al. Lung imaging in COPD and asthma. Respir Investig. 2024;62(6):995-1005. doi:10.1016/j.resinv.2024.08.014
5. 5. Barnes PJ. Cellular and molecular mechanisms of asthma and COPD. Clin Sci (Lond). 2017;131(13):1541-1558. doi:10.1042/CS20160487
6. 6. Abramson MJ, Perret JL, Dharmage SC, McDonald VM, McDonald CF. Distinguishing adult-onset asthma from COPD: a review and a new approach. Int J Chron Obstruct Pulmon Dis. 2014;9:945-62. doi:10.2147/COPD.S46761

LICENCE & RIGHTS

MEDICAL DISCLAIMER

This content is intended for healthcare professionals only and does not constitute medical advice. Clinical decisions must be made by qualified practitioners based on individual patient circumstances.

FOLLOW THE LIFE SCIENCE FEED

Instagram @lifesciencefeed **LinkedIn** linkedin.com/company/thelifesciencefeed

thelifesciencefeed.com