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# Current and Emerging Biologic Therapies for the Treatment of Asthma: A Focus on Targeting Thymic Stromal Lymphopoietin (TSLP)

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## Abstract

Asthma is a chronic inflammatory airway disease that affects millions globally, contributing to substantial morbidity and mortality. A significant subset of patients remains uncontrolled despite conventional therapies. Biologic treatments that target type 2 inflammation, including monoclonal antibodies against IL-4, IL-5, IL-13, and IgE, have revolutionized asthma management; however, these therapies primarily address downstream inflammatory pathways. Thymic stromal lymphopoietin (TSLP), an epithelial-derived cytokine, has emerged as a pivotal upstream mediator in the pathophysiology of asthma, acting as a bridge between innate and adaptive immune responses. TSLP is critical in initiating and maintaining type 2 inflammation, positioning it as a promising therapeutic target. Tezepelumab (Tezspire), the first FDA-approved TSLP inhibitor, has demonstrated broad efficacy across various asthma phenotypes, effectively reducing exacerbation rates and improving lung function independent of eosinophil count or allergic status. Additionally, emerging TSLP-targeting biologics under development, such as the inhaled monoclonal antibody fragment ecleralimab (CSJ117), offer novel approaches to localized airway inflammation control. This review aims to explore the mechanisms, clinical efficacy, and economic implications of TSLP-targeting therapies, underscoring their potential to redefine the asthma biologics market and address the unmet therapeutic needs of patients with severe asthma.

***Keywords:*** asthma; biologic; cytokine; ecleralimab (CSJ117); Tezspire (tezepelumab); thymic stromal lymphopoietin (TSLP)

## Abbreviations

airway hyperresponsiveness (AHR), annual asthma exacerbation rate (AAER), Centers for Disease Control (CDC), compound annual growth rate (CAGR), fragment antigen-binding region (Fab), fragment crystallizable region (Fc), fractional exhaled nitric oxide (FeNO), forced expiratory volume in one second (FEV1), immunoglobulin (Ig), interleukin (IL), intravenous (IV), subcutaneous (SC), t-helper (Th), thymic stromal lymphopoietin (TSLP), thymic stromal lymphopoietin receptor (TSLPR), type 2 innate lymphoid cell (ILC-2), World Health Organization (WHO).

## Introduction

Asthma is one of the most common chronic inflammatory disorders of the airways; it is characterized by respiratory symptoms (ex., breathlessness and wheezing), eosinophilic inflammation, goblet cell hyperplasia, mucus hypersecretion, airway hyperresponsiveness (AHR) [Figure 1], and variable airflow limitation, as well as an array of clinical manifestations, immune mechanisms, and therapeutic responses [9, 32, 33]. According to the World Health Organization (WHO), in 2019, asthma was one of the most prevalent non-communicable diseases and the most common chronic childhood disease, and it has affected some 262 million people and contributed to 455,000 deaths worldwide [41]. According to the Centers for Disease Control (CDC), in the United States, asthma has affected over 26 million Americans, with nearly 1 million requiring treatment in an emergency department for symptoms and contributing to 3,500 deaths in one year [12]. The primary goal in asthma treatment is to minimize the risk of exacerbation and overall symptom burden, and anti-inflammatory (ex., inhaled or oral corticosteroids) and bronchodilators (ex., short- or long-acting adrenergic β-2 receptor agonists also referred to as β-2 agonists) have long been the mainstay treatments of asthma therapy [32]. In the majority of patients, asthma is adequately controlled through these conventional treatment methods; however, approximately fifteen percent of asthma patients, despite adherence to conventional therapies, will remain uncontrolled [23].

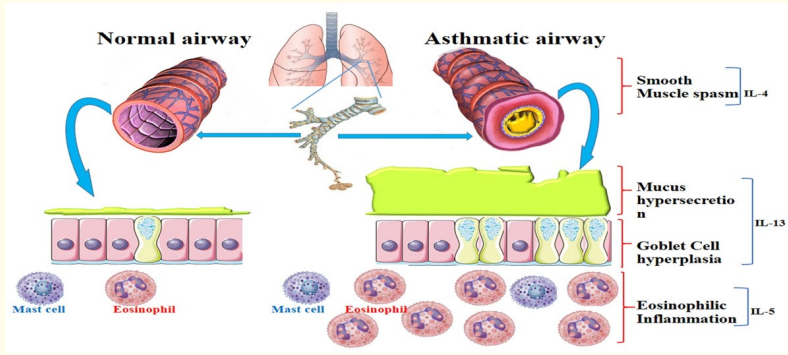


***Figure 1:*** Comparison of normal and asthmatic airway structure and inflammation [9].

Compared to the normal airway (left), asthmatic airways (right) exhibit thickened smooth muscle (mediated by IL-4), goblet cell hyperplasia and mucus hypersecretion (mediated by IL-13), and infiltration of eosinophils and mast cells into the epithelium (driven by IL-5). These changes contribute to airway narrowing, AHR, and clinical symptoms such as wheezing, breathlessness, and variable airflow limitation.

Biologic therapies have transformed the treatment landscape for patients with asthma who were previously poorly controlled with conventional therapies by providing targeted inhibition of key inflammatory pathways [37]. Monoclonal antibody therapies that target IL-4, IL-5, IL-13 (interleukins), and IgE (immunoglobulin E) have proven effective in improving lung function and reducing exacerbations in patients with type 2 high asthma; however, each of these therapies only addresses the downstream effectors of the asthma inflammatory cascade [10, 25, 28, 37].

Thymic stromal lymphopoietin (TSLP) is produced mainly by epithelial cells and plays a crucial role in connecting innate and adaptive immune responses; it has been identified as a key cytokine in the initiation and progression of type 2 inflammation, especially in the context of asthma [33]. Once released, TSLP activates various immune cells, including dendritic and mast cells, which helps create a pro-inflammatory environment [38]. This inflammatory setting significantly contributes to airway hyperresponsiveness (AHR) and the structural remodeling associated with asthma pathology [16]. Due to its essential role in asthma development, TSLP has become a

promising target for therapeutic interventions to mitigate type 2 inflammatory responses [33].

This review analyzes the mechanisms and clinical efficacy of existing and emerging biologics that inhibit TSLP signaling and examines the economics of the asthma biologics market.

### *Market Size of Asthma Biologics*

Driven by the chronic nature of the condition, the global asthma therapeutics market, including both biological and conventional therapies (such as bronchodilators and inhaled corticosteroids), in 2024 was valued at $27.81 billion and is expected to grow to a value of $46.66 billion by 2034 with a compound annual growth rate (CAGR) of 5.31% [Figure 2] [5]. In 2022, the asthma biologics market was valued at $6.5 billion and is projected to grow significantly over the next nine years, with a CAGR of 12.5%, reaching an estimated value of $19.2 billion by 2031 [Figure 3] [4].

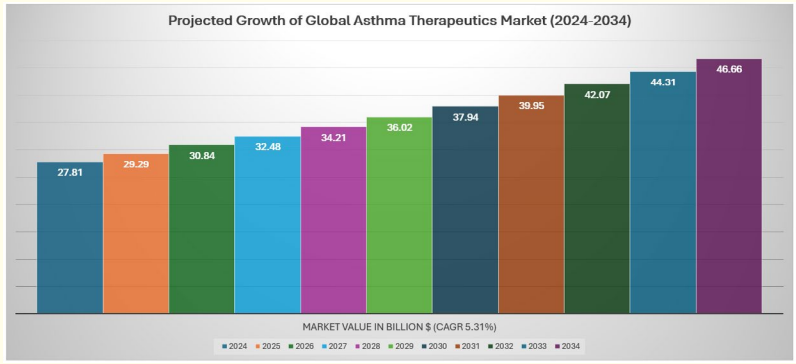


***Figure 2:*** The forecasted growth of the global asthma therapeutics market (2024-2034) [5].

The global asthma therapeutics market, including biologics and conventional treatments, was valued at $27.81 billion in 2024 and is projected to reach $46.66 billion by 2034, growing at a CAGR of 5.31%.

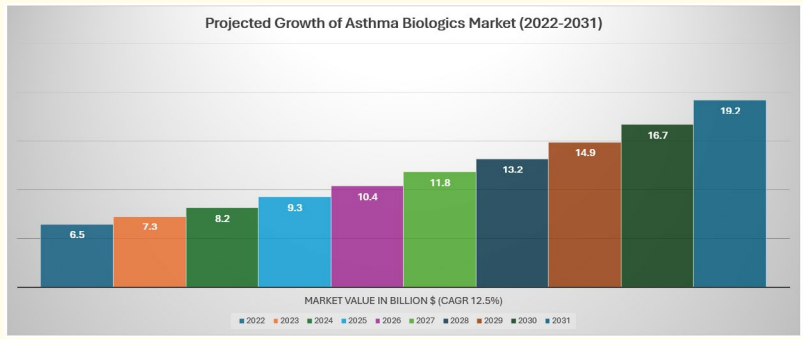


***Figure 3:*** The projected growth of the global asthma biologics market (2022–2031) [4].

In 2022, the asthma biologics market was valued at $6.5 billion. Fueled by the growing adoption of targeted therapies, this market is expected to expand at a CAGR of 12.5% and is projected to reach $19.2 billion by 2031.

### *Airway Inflammation in Asthma*

Immune responses play a central role in the pathology of asthma [11, 18, 25]. Type 2 high asthma features eosinophilic airway inflammation, which manifests through increased blood eosinophil levels or elevated fractional exhaled nitric oxide (FeNO) and typically shows a responsiveness to corticosteroids [10, 21]. In Type 2 high asthma, inhaled allergens, microbes, and pollutants interact with the airway epithelium and activate mediators such as TSLP, IL-25, and IL-33 cytokines [11]. Allergen exposures also activate the dendritic cells, which present the antigens to CD4+ T cells, promoting differentiation into Th2 cells, which subsequently induce the release of the cytokines IL-4, IL-5, and IL-13, driving the recruitment and activation of immune cells, IgE production by B cells and the activation of innate cells, leading to bronchoconstriction, airway hyperresponsiveness, mucus hypersecretion, and airway remodeling [18, 25]. In contrast, Type 2 low asthma is characterized by normal sputum and peripheral blood eosinophil counts, low FeNO levels, persistent symptoms, airflow limitation, and poor corticosteroid reaction [21]. Type 2 low asthma is associated with neutrophilic or paucigranulocytic inflammation, which triggers the activation of both T1 and T17 cells, and increased cytokine IL-17A mRNA levels have been observed in individuals with moderate to severe asthma [25].

### *Pharmaceutical Companies and Current FDA-Approved Asthma Biologics*

Six innovative asthma biologics have been developed and brought to market by various pharmaceutical companies during the last twenty years, summarized in Table 1.

| *Company* | *Biologic Product* | *Biologic Target* | *Drug Delivery Mechanism* | *FDA Approval Year* | *References* |
|---|---|---|---|---|---|
| Genentech/Novartis | Xolair (omalizumab) | IgE | Subcutaneous (SC) | 2003 | [17, 34] |
| GlaxoSmithKline | Nucala (mepolizumab) | IL-5 | Subcutaneous (SC) | 2015 | [19] |
| Teva Pharmaceuticals | Cinqair (reslizumab) | IL-5 | Intravenous (IV) | 2016 | [40] |
| AstraZeneca | Fasenra (benralizumab) | IL-5 | Subcutaneous (SC) | 2017 | [7] |
| Sanofi/Regeneron | Dupixent (dupilumab) | IL-4 & IL-13 | Subcutaneous (SC) | 2018 | [36] |
| AstraZeneca/Amgen | Tezspire (tezepelumab) | TSLP | Subcutaneous (SC) | 2021 | [6] |

***Table 1:*** Key pharmaceutical companies and their biologic products.

Genentech and Novartis worked together to co-develop Xolair (omalizumab), which targets and binds immunoglobulin E (IgE), and it was the first monoclonal antibody therapy for moderate to severe allergic asthma which was first FDA approved in 2003 [17, 34]. In 2024, the global revenue of Xolair was over $1.64 billion [29]. GlaxoSmithKline developed Nucala (mepolizumab), a drug that targets and inhibits the cytokine interleukin-5 (IL-5); it was the first anti-IL-5 targeting treatment and was FDA-approved in 2015 for the treatment of severe eosinophilic asthma [19]. In 2023, Nucala generated over £1.65 (over $2.2 billion USD) in revenue [20]. Teva Pharmaceuticals' Cinqair (reslizumab) is also an IL-5 antagonist that works by inhibiting IL-5 signaling and was first approved by the FDA in 2016 [40]. Teva Pharmaceuticals has not publicly disclosed specific revenue figures for its Cinqair (reslizumab) product. AstraZeneca's Fasenra (benralizumab) targets IL-5, but in this scenario, the drug functions as an IL-5 receptor antagonist instead of directly targeting the IL-5 cytokine itself as in the other direct IL-5 inhibitor Nucala, and Fasenra was first FDA approved the treatment of severe eosinophilic asthma in 2017 [7]. AstraZeneca reported that Fasenra generated over $1.55 billion in sales revenue during 2023 [8]. Sanofi and Regeneron collaborated to create Dupixent (dupilumab), which works to inhibit interleukin-4 (IL-4) and interleukin-13 (IL-13) signaling; it was the first-in-class drug to target these cytokines and was first approved by the FDA in 2018 to treat moderate to severe eosinophilic and oral corticosteroid-dependent asthma [36]. In 2024, Dupixent sales reached €13 billion [$13.5 billion USD]

[35]. However, it should be noted that Dupixent has five other FDA-approved uses for common indications, including atopic dermatitis (eczema), that could impact its overall revenue figures [35, 36]. AstraZeneca partnered with Amgen to develop Tezspire (tezepelumab), which was the first in its class to target an epithelial cytokine thymic stromal lymphopoietin (TSLP), and it was FDA-approved in 2021 to treat severe asthma without phenotype or biomarker limitations [6]. Amgen reported a sales revenue of $567 million from Tezspire (tezepelumab) in the year 2023 [3].

### *TSLP Inhibitors: Mechanism of Action*

First cloned in 1994, thymic stromal lymphopoietin (TSLP) is an epithelial cytokine (alarmin) with a four-helical bundle structure that is structurally similar to interleukin-7 (IL-7) and belongs to the hematopoietic cytokine family [13, 16]. TSLP is mainly derived from epithelial cells prior to environmental stimulation, but it is also produced by eosinophils, fibroblasts, group 2 innate lymphoid cells (ILC-2s), macrophages, mast cells, type 2 helper cells (TH2), while various cell types such as airway smooth muscle cells, eosinophils, basophils, dendritic cells, eosinophils, mast cells, hematopoietic progenitor cells, ILC-2s, lymphocytes, macrophages, and monocytes possess receptors for TSLP [27]. In response to the inhalation of environmental stimuli such as allergens, bacteria, cigarette smoke, fungi, or viruses, the airway epithelial cells begin producing TSLP along with IL-25 and IL-33, which leads to the activation of ILC-2s to release IL-5 and IL-13, resulting in eosinophilia, airway hyperresponsiveness (AHR), and mucus hypersecretion [Figure 4] [11, 16]. Additionally, TSLP has been found to play a role in initiating pathways in both allergic and non-allergic eosinophilic inflammation [38]. In allergic eosinophilic inflammation, TSLP triggers pathways that involve basophils, mast cells, and Th2 lymphocytes, leading to an increase in eosinophils (eosinophilia) in the airways; in contrast, during non-allergic eosinophilic inflammation, TSLP stimulates innate lymphocytes like ILC-2s, which also play a role in promoting eosinophilia in the airways [38]. TSLP has also been associated with airway remodeling by enhancing fibroblast-mediated collagen synthesis and promoting the proliferation of airway smooth muscle cells [14].

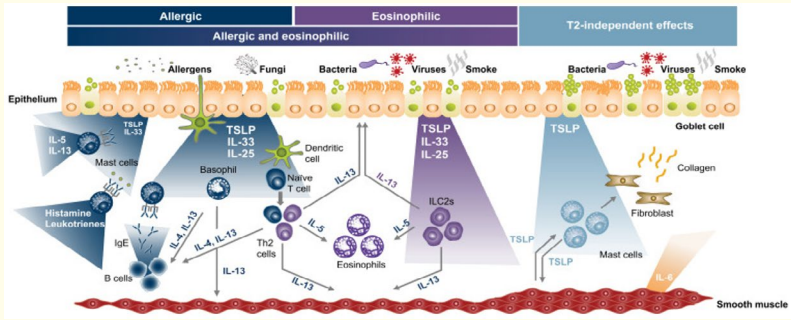


***Figure 4:*** Cytokines, cell types, and environmental triggers involved in the asthma inflammatory cascade [31].

Inhaled environmental triggers, allergens, fungi, bacteria, viruses, and cigarette smoke stimulate airway epithelial cells to release TSLP, IL-25, and IL-33. These cytokines activate dendritic cells, ILC-2s, and Th2 cells, promoting the production of IL-5 and IL-13. This inflammatory cascade leads to eosinophilic inflammation, goblet cell hyperplasia, mucus hypersecretion, and AHR.

### *Tezepelumab (Tezspire)*

Tezepelumab (Tezspire) is the first fully human monoclonal antibody (IgG2λ), approved by the FDA in 2021, that selectively binds to TSLP, preventing its interaction with the heterodimeric TSLP receptor (TSLPR), thereby inhibiting downstream inflammatory signaling [14]. Tezepelumab inhibits TSLP, leading to a downregulation in the asthma inflammatory cascade, leading to a decrease in airway

eosinophil counts, FeNO levels, IL-4, IL-5, IL-13, serum IgE levels, and mucus production, while also decreasing AHR [31]. Treatment with tezepelumab has been shown to lower both blood and sputum eosinophil levels, both before and after an allergen challenge, as well as to reduce FeNO levels [33].

In their phase II clinical trials, the CASCADE trial evaluated tezepelumab in patients receiving inhaled corticosteroids and one or more asthma control medications [14]. At the conclusion of the trial, the number of eosinophils in the bronchoscopy biopsy specimens decreased significantly by 89% in the tezepelumab treatment group, compared to only a 25% reduction in the placebo group; this indicated that tezepelumab effectively suppressed eosinophil levels in the lungs, particularly within the airway submucosa [14, 22]. In phase III clinical trials, the NAVIGATOR trial found that patients receiving tezepelumab experienced a significantly lower annual asthma exacerbation rate (AAER) and improved forced expiratory volume in one second (FEV1); additionally, among patients with a baseline blood eosinophil count of 300 cells/μl or higher, tezepelumab reduced the AAER by 70% more than the placebo at Week 52 [38].

Similar to the majority of the other FDA-approved asthma biologics, aside from reslizumab, which utilizes an intravenous (IV) infusion, tezepelumab also utilizes an accessible subcutaneous (SC) delivery system available in glass vials, pre-filled syringes or pre-filled injector pens for home use [6, 40]. However, in comparison to the other asthma biologics that only target and suppress one or two cytokines, an IgE antibody, or a cytokine receptor, tezepelumab offers a broader range of inhibition for multiple pathways involved in type 2 inflammation (see Figure 5), and it effectively reduces asthma exacerbations in patients with poorly controlled, moderate-to-severe asthma, regardless of their blood eosinophil counts, FeNO levels, or the presence of sensitization to allergens [22].

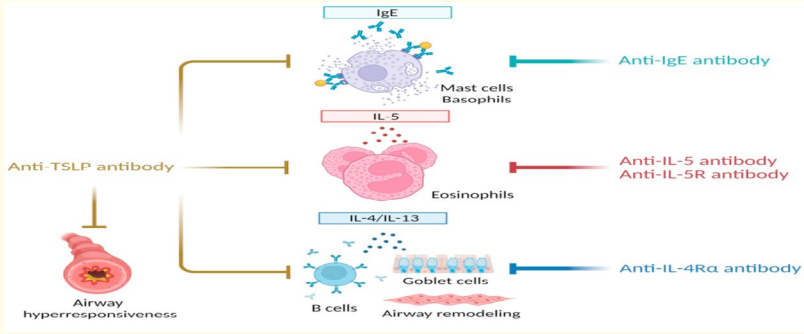


***Figure 5:*** Comparison of current biologic targets for the treatment of asthma [22].

This schematic outlines the key inflammatory mediators involved in asthma management and the corresponding biologic therapies. Anti-IgE antibodies target the activation of mast cells and basophils, while anti-IL-5 and anti-IL-5R antibodies help to reduce eosinophilic inflammation. Anti-IL-4Rα antibodies block IL-4 and IL-13 signaling, which impact B cells, goblet cell hyperplasia, and airway remodeling. The anti-TSLP antibodies broadly inhibit multiple upstream inflammatory signaling pathways, which helps reduce AHR and decrease the activity of downstream cytokines.

### *Ongoing Clinical Trials - Emerging TSLP Inhibitor for Asthma: Ecleralimab (CSJ117)*

Novartis is currently developing an anti-TSLP biologic called ecleralimab (CSJ117), which is in clinical phases 1 and 2 of development [15, 30]. Unlike previous monoclonal antibodies therapies such as tezepelumab, which require subcutaneous (SC) delivery, ecleralimab (CSJ117) is the first inhaled monoclonal antibody fragment designed to be delivered directly to the airways [30]. It is a highly potent neutralizing antibody fragment (Fab) of the IgG1/λ isotype, lacking an Fc region and possessing a significantly smaller molecular weight (46.6 kDa) compared to a full-length antibody (approximately 150 kDa) [Figure 6] [16, 30]. Due to its reduced size

and localized administration, ecleralimab (CSJ117)'s inhaled delivery enables enhanced lung distribution and tissue penetration, and the absence of an Fc region allows the Fab fragment to penetrate airway tissue while preserving antigen-binding specificity effectively [30]. The antibody fragment binds to TSLP and inhibits TSLPR activation, allowing for a targeted approach to TSLP inhibition [15, 30].

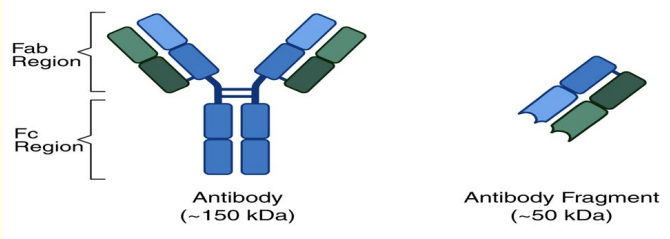


***Figure 6:*** Comparison of a full-size monoclonal antibody and a monoclonal antibody fragment [16, 30].

Full-length monoclonal antibodies (~150 kDa) consist of both the Fab and Fc regions. This structure enables various effector functions, such as recruiting immune cells. In contrast, antibody fragments like (CSJ117) ecleralimab (~46.6 kDa) contain only the Fab region and lack the Fc portion. This structural difference limits immune activation while maintaining antigen-binding ability, allowing for targeted therapeutic effects with potentially reduced systemic side effects.

Preclinical studies have demonstrated that ecleralimab (CSJ117) effectively reduces TSLP-driven inflammation in the lungs [15, 30]. Furthermore, ecleralimab (CSJ117) has been found to significantly reduce allergen-induced bronchoconstriction and airway inflammation in subjects with mild atopic asthma [15]. An inhaled anti-TSLP treatment may also provide additional advantages over subcutaneous (SC) injections, as direct delivery to the airways at lower dosages could minimize systemic impacts and reduce effects from TSLP signaling beyond the airways [16, 30].

### *Safety and Adverse Effects of TSLP Inhibitors*

Clinical trials involving TSLP inhibitors, such as tezepelumab, have generally reported favorable tolerability among patients; the most frequently observed adverse effects included nasopharyngitis, headache, and bronchitis, and these side effects are typically mild to moderate in severity and aligned with the safety profiles observed in other biologic therapies for asthma [6, 24]. In the Phase III clinical trial, the DESTINATION study, tezepelumab treatment was found to be well tolerated for up to two years and resulted in significant, sustained reductions of asthma exacerbations and improved lung function in participants with severe, uncontrolled asthma, demonstrating both long-term safety and sustained efficacy [26]. In the Phase III NAVIGATOR study, 29 out of 601 patients (5%) who received the recommended dosing regimen of tezepelumab developed anti-drug antibodies, with 11 patients (2%) experiencing treatment-emergent antibodies and one patient (<1%) developing neutralizing antibodies, yet there was no evidence that these antibodies impacted pharmacokinetics, pharmacodynamics, efficacy, or safety [6].

## Conclusion and Future Considerations

Asthma remains a prevalent and complex chronic inflammatory airway disease that contributes to the global disease burden and requires a multifaceted treatment approach to mitigate exacerbations and improve patient outcomes [9, 32, 41]. Although conventional therapies, such as corticosteroids and bronchodilators, have effectively controlled asthma symptoms in the majority of patients, a significant subset continues to experience persistent disease despite adherence to these standard treatments [23]. The introduction of biologics as additional add-on maintenance therapies to the standard treatments has significantly transformed asthma management

by offering targeted inhibition of key inflammatory pathways, including IL-4, IL-5, IL-13, and IgE, leading to improved lung function and reduced exacerbation rates [10, 37].

TSLP has emerged as a crucial upstream regulator of type 2 inflammation in asthma. It connects innate and adaptive immune responses, promoting airway hyperresponsiveness (AHR), eosinophilic inflammation, and mucus hypersecretion [33, 38]. The development of tezepelumab, the first FDA-approved TSLP inhibitor, marks a significant milestone in asthma therapeutics; it has demonstrated broad efficacy in reducing exacerbations, irrespective of eosinophil levels or FeNO biomarkers [14, 22]. Clinical trials have shown that tezepelumab can reduce airway eosinophilia, suppress type 2 cytokine responses, and improve lung function in patients with moderate-to-severe asthma [31, 33].

Emerging therapies, such as ecleralimab (CSJ117), offer promising options for managing asthma, mainly through innovative inhaled monoclonal antibody delivery systems [15, 30]. By targeting TSLP at the site of airway inflammation, ecleralimab (CSJ117) may offer a more precise and convenient alternative to systemic monoclonal antibodies by utilizing localized delivery, which may enhance lung-specific targeting while minimizing systemic exposure, potentially improving patient adherence and accessibility while also alleviating treatment-related burdens [30]. Future studies should optimize dosing regimens, evaluate long-term safety profiles, and explore potential combination therapies to personalize asthma treatment further [15].

Furthermore, while inhibiting TSLP has demonstrated significant therapeutic benefits, its role beyond type 2 inflammation requires further exploration. TSLP has been implicated in airway remodeling and non-eosinophilic asthma subtypes, indicating that its inhibition might have broader implications for asthma pathophysiology [14, 38]. Future research should investigate the interactions between TSLP inhibitors and other inflammatory pathways and their potential use in treating chronic airway diseases like chronic obstructive pulmonary disease [COPD] [27]. Notably, tezepelumab is undergoing clinical trials for several inflammatory conditions, including chronic spontaneous urticaria (hives), atopic dermatitis (eczema), and COPD [1, 2, 39].

As the global market for asthma biologics continues to grow, cost-effectiveness and accessibility will be critical factors in determining their widespread clinical adoption [4, 5]. Given the significant financial burden associated with biological therapies, healthcare systems must find a balance between innovation and affordability to ensure equitable access for patients worldwide [37].

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There is no funding to report on this study.

### *Conflict of Interest*

The authors declare that there is no conflict of interest.

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