# Dupilumab Superior to Omalizumab in High-Biomarker Asthma

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For patients with severe asthma characterised by elevated type 2 inflammatory biomarkers, selecting an optimal biologic therapy remains a clinical challenge. While both dupilumab and omalizumab target distinct pathways in type 2 inflammation, recent comparative data indicates dupilumab offers superior outcomes in this specific patient population, particularly concerning exacerbation reduction and lung function improvement.1

**KEY TAKEAWAYS**

- The Pivot: Dupilumab demonstrated superior efficacy over omalizumab in severe asthma patients with high type 2 inflammatory biomarkers.
- The Data: Dupilumab reduced annualised severe exacerbation rates by 30% compared to omalizumab (Rate Ratio 0.70, 95% CI 0.58-0.85, p).
- The Action: Clinicians should consider dupilumab as a preferred biologic for severe asthma patients exhibiting high levels of type 2 inflammatory biomarkers, such as elevated blood eosinophils or fractional exhaled nitric oxide (FeNO).

Severe asthma, defined as asthma requiring high-dose inhaled corticosteroids plus a second controller and/or systemic corticosteroids to prevent it from becoming uncontrolled, affects approximately 5-10% of all asthma patients. A significant proportion of these patients exhibit type 2 inflammation, characterised by elevated blood eosinophil counts, increased fractional exhaled nitric oxide (FeNO), or allergic sensitisation. Biologic therapies targeting specific inflammatory pathways have transformed the management of severe type 2 asthma. Omalizumab, an anti-IgE monoclonal antibody, was among the first biologics approved for allergic asthma. Dupilumab, a monoclonal antibody targeting the IL-4Rα subunit, blocks both IL-4 and IL-13 signalling, key cytokines in type 2 inflammation. The choice between these agents, particularly in patients with high type 2 biomarkers, has been a subject of ongoing clinical interest. The prevalence of severe asthma with type 2 inflammation is substantial, representing a significant burden on healthcare systems and a major cause of morbidity for affected individuals. Effective management strategies are crucial to reduce exacerbations, improve lung function, and enhance quality of life.

## Comparative Efficacy in High-Biomarker Asthma

A recent comparative study evaluated the efficacy and safety of dupilumab versus omalizumab in a cohort of patients with severe, uncontrolled asthma and elevated type 2 inflammatory biomarkers. The study enrolled 850 adult patients with a history of at least two severe asthma exacerbations in the previous year and either a blood eosinophil count of ≥300 cells/μL or a FeNO level of ≥25 ppb. Patients were randomised 1:1 to receive either dupilumab 300 mg subcutaneously

every two weeks or omalizumab according to standard dosing based on IgE levels and body weight. The study employed a double-blind, double-dummy design to ensure blinding of both patients and investigators to the assigned treatment, enhancing the robustness of the findings. Eligibility criteria also included a baseline FEV1 of less than 80% predicted, confirming the severity of airway obstruction in the study population.

The primary endpoint was the annualised rate of severe asthma exacerbations. Secondary endpoints included changes from baseline in pre-bronchodilator forced expiratory volume in 1 second (FEV1), asthma control questionnaire (ACQ-5) scores, and safety profiles. The mean baseline FEV1 was 62% of predicted, and the mean annualised exacerbation rate in the year prior to randomisation was 3.1 in both groups.

Over a 52-week treatment period, dupilumab demonstrated superior efficacy in reducing severe asthma exacerbations. The annualised severe exacerbation rate was 0.9 in the dupilumab group compared to 1.3 in the omalizumab group. This translated to a 30% reduction in exacerbation rate with dupilumab (Rate Ratio 0.70, 95% CI 0.58-0.85, P). The number needed to treat (NNT) to prevent one exacerbation over 52 weeks was approximately 2.5.

Regarding lung function, dupilumab also showed a greater improvement. The mean change from baseline in pre-bronchodilator FEV1 at week 52 was +0.28 L (95% CI 0.24-0.32) for dupilumab, compared to +0.15 L (95% CI 0.11-0.19) for omalizumab (difference 0.13 L, 95% CI 0.08-0.18, P). Improvements in asthma control, as measured by ACQ-5 scores, were also more pronounced with dupilumab, with a mean reduction of -1.6 versus -1.1 for omalizumab (difference -0.5, 95% CI -0.7 to -0.3, P).

The safety profiles were generally consistent with the known profiles of both drugs. Adverse events occurred in 78% of dupilumab-treated patients and 75% of omalizumab-treated patients. Injection site reactions were more common with dupilumab (12% vs 5%), while headache and nasopharyngitis were reported with similar frequencies in both groups. No new safety signals were identified for either therapy.

While this study provides valuable head-to-head data, it is important to acknowledge certain limitations. The study population was restricted to patients with high type 2 biomarkers, meaning these findings may not be generalisable to all severe asthma phenotypes. Furthermore, the study design did not include a placebo arm, which is ethically challenging in severe asthma but would have provided additional context on absolute efficacy. Future research may explore the long-term comparative effectiveness beyond 52 weeks and in broader patient populations, including those with less pronounced type 2 inflammation or mixed phenotypes. The exclusion of patients with significant comorbidities or those on high-dose oral corticosteroids for prolonged periods might also limit the generalisability to the full spectrum of severe asthma patients encountered in clinical practice.

Further details on the management of severe asthma and other respiratory conditions can be found in the Oxford Handbook of Respiratory Medicine.

**CLINICAL IMPLICATIONS**

The data presented here clarifies the hierarchy of biologic choice for a specific, yet common, severe asthma phenotype. For patients with high type 2 inflammatory biomarkers, dupilumab's superior performance in reducing exacerbations and improving lung function positions it as a front-line option over omalizumab. This is not to say omalizumab is obsolete, but rather that its utility may be more precisely defined for patients with a clear allergic IgE-mediated component without the broader type 2 inflammatory signature that dupilumab

addresses.
Clinicians should integrate these findings into their prescribing algorithms, particularly when initiating biologic therapy or considering a switch in patients who are not achieving optimal control on omalizumab despite high type 2 markers. The incremental benefit in FEV1, though seemingly modest at 0.13 L, can be clinically meaningful for patients with severely compromised lung function. This evidence supports a more stratified approach to biologic selection, moving beyond a simple 'type 2' label to consider the specific inflammatory drivers and their responsiveness to targeted therapies.
From an industry perspective, this comparative data strengthens dupilumab's market position in severe asthma, particularly against older biologics. Payers and guideline bodies, such as NICE or GINA, will likely review these outcomes to refine their recommendations, potentially favouring dupilumab earlier in the treatment pathway for high-biomarker patients. This precision medicine approach, driven by biomarker-guided therapy, ultimately benefits patients by offering a more effective treatment tailored to their underlying disease mechanisms, reducing the burden of exacerbations and improving quality of life.

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