# Eosinophil-Associated Comorbidities in Asthma: Prevalence, Burden, and Clinical implications

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# Eosinophilic comorbidities in asthma

- Allergic rhinitis (AR)
- Chronic rhinosinusitis with nasal polyps (CRSwNP)
- Atopic dermatitis (AD)
- Eosinophilic esophagitis (EoE)
- NSAID-exacerbated respiratory disease (N-ERD)

  / Aspirin-exacerbated respiratory disease (AERD)

✓ Type 2-high phenotype driven by IL-4, IL-5 and IL-13: 50-70% of asthma patients

✓ Systemic type 2 disease: same axis across the airways, skin, and gut, not lung alone

- Local disease reflects a broader inflammatory burden

# Prevalence of T2 comorbidities among asthma patients

| Comorbidity | Prevalence with asthma | Key clinical point |
|---|---|---|
| **AR** | Up to **~75%** of asthma | Most common upper-airway comorbidity |
| **CRSwNP** | Mild asthma **10–30%**<br>→ severe asthma **70–90%** | Strong link to severity & poor control<br>Tissue eosinophilia |
| **AD** | Up to **~20%** of asthma<br>**~70%** of severe AD develop asthma | Start of the atopic march<br>Reflects systemic type 2 burden |
| **EoE** | Up to **~5%** of asthma<br>**20–50%** of EoE patients have asthma | Late manifestation of the atopic march<br>Dysphagia, food impaction |
| **N-ERD/AERD** | **~7%** of asthma (up to **~21%** on challenge)<br>**~15%** of severe asthma | Samter triad (asthma + CRSwNP + NSAID hypersensitivity); eicosanoid imbalance |

# Unified airway hypothesis

## Epidemiologic evidence

In patients with CRSwNP:

Up to 70% have comorbid asthma

Up to 10% have comorbid NSAID-ERD

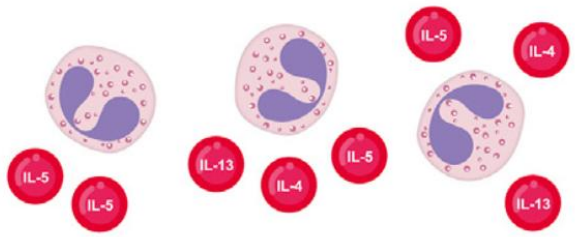
In patients with asthma:

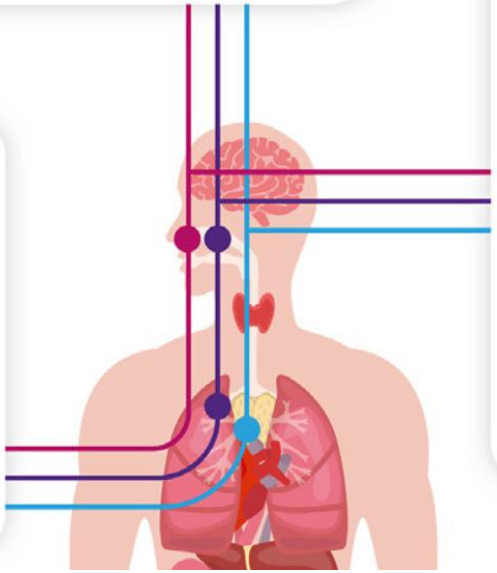
40% have comorbid CRSwNP

Up to 15% have comorbid NSAID-ERD

## Pathophysiologic evidence

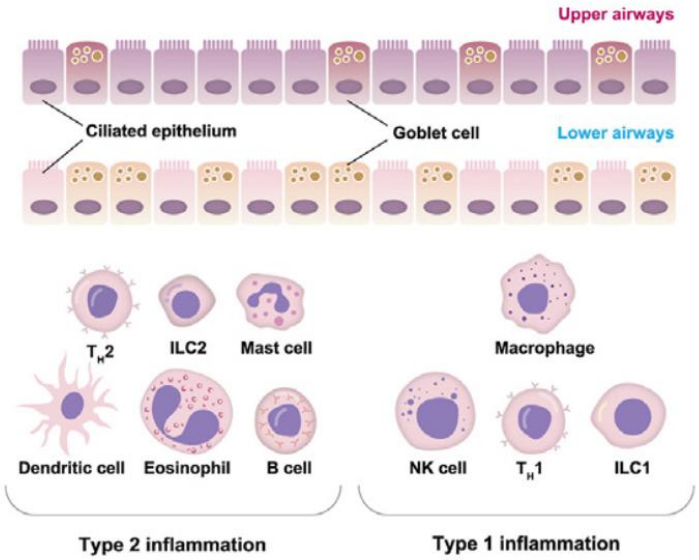
**Severe eosinophilic asthma, CRSwNP, N-ERD are commonly characterized by eosinophils and elevated levels of IL-5, IL-4, and IL-13**



## Functional and pathophysiologic evidence

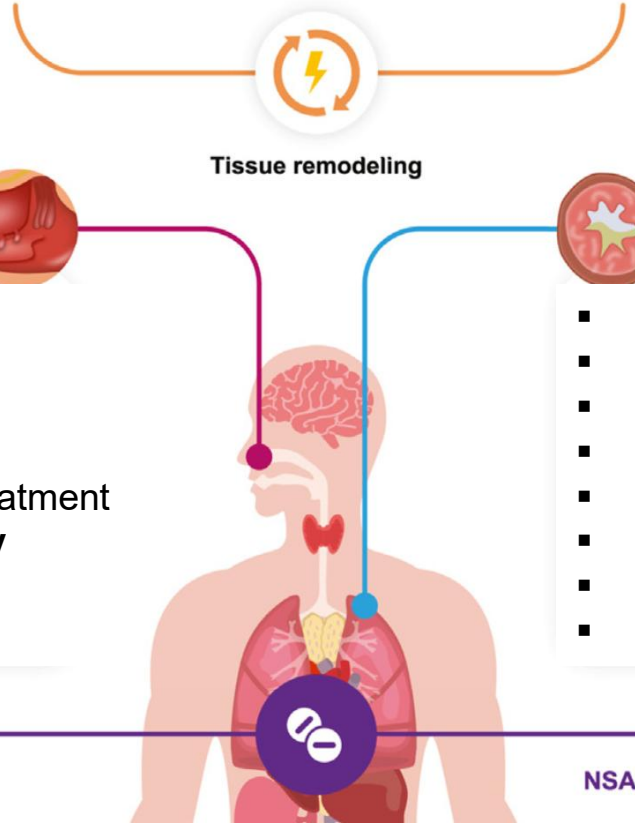
**The upper and lower airways share common cell types and immune interactions**




Bachert CC, et al. J Allergy Clin Immunol Pract 2023;11(9):2630-2641.

# Role of eosinophil in tissue remodeling

- Epithelial damage
- Edema
- Fibrosis
- Angiogenesis
- Neural plasticity
- Aberrant tissue repair
- Smooth muscle hypertrophy

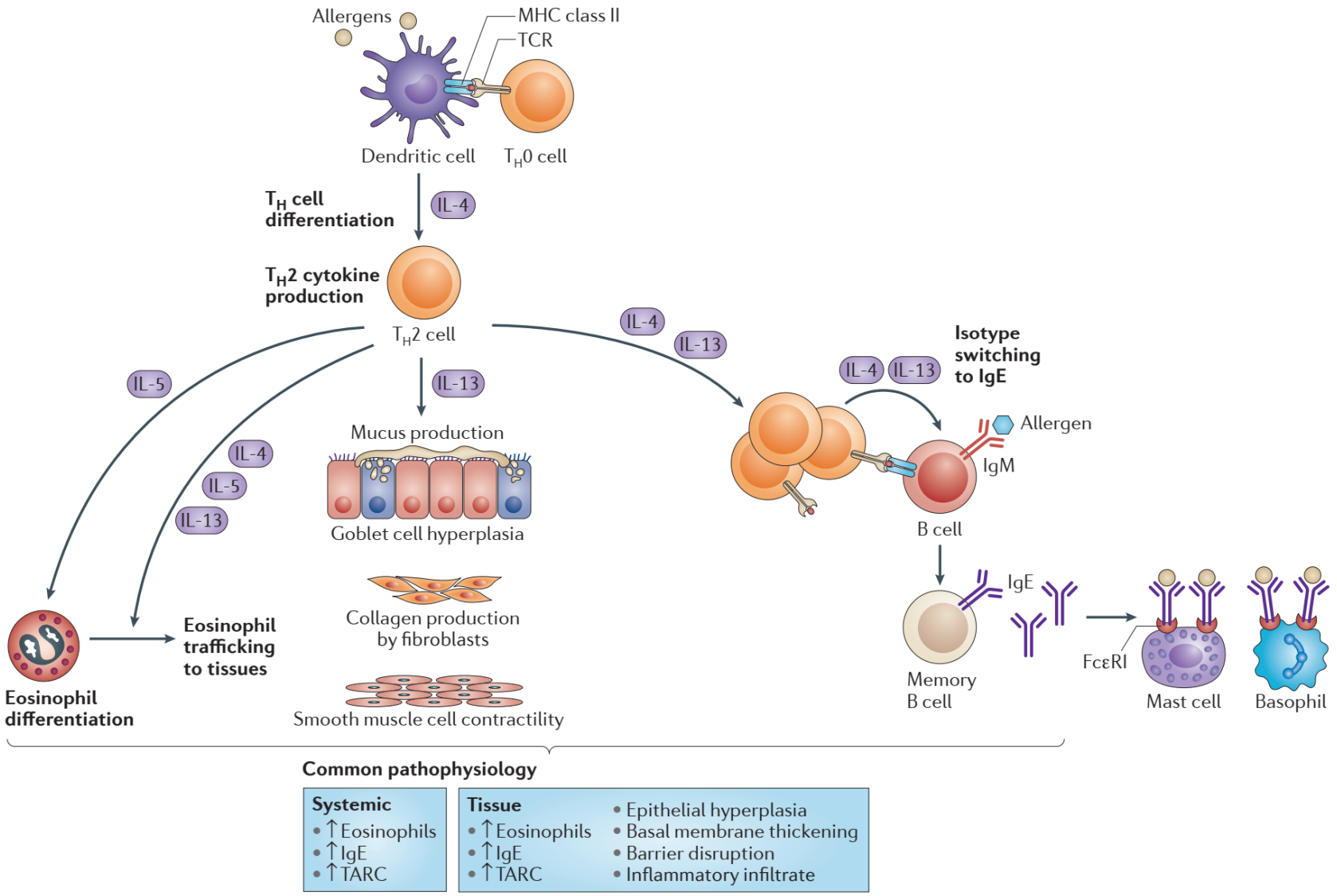



- NP formation and recurrence
- Increased comorbidities
- **Impaired host defense**
- **Mucus hypersecretion**
- Reduced response to SoC treatment
- **Increased symptom severity**
- Loss of smell
- **Poorer disease prognosis**

- Airway hyperresponsiveness
- Airway narrowing
- **Impaired host defense**
- **Mucus hypersecretion**
- Increased sputum production
- **Increased symptom severity**
- **Poorer disease prognosis**
- Increased risk of exacerbation

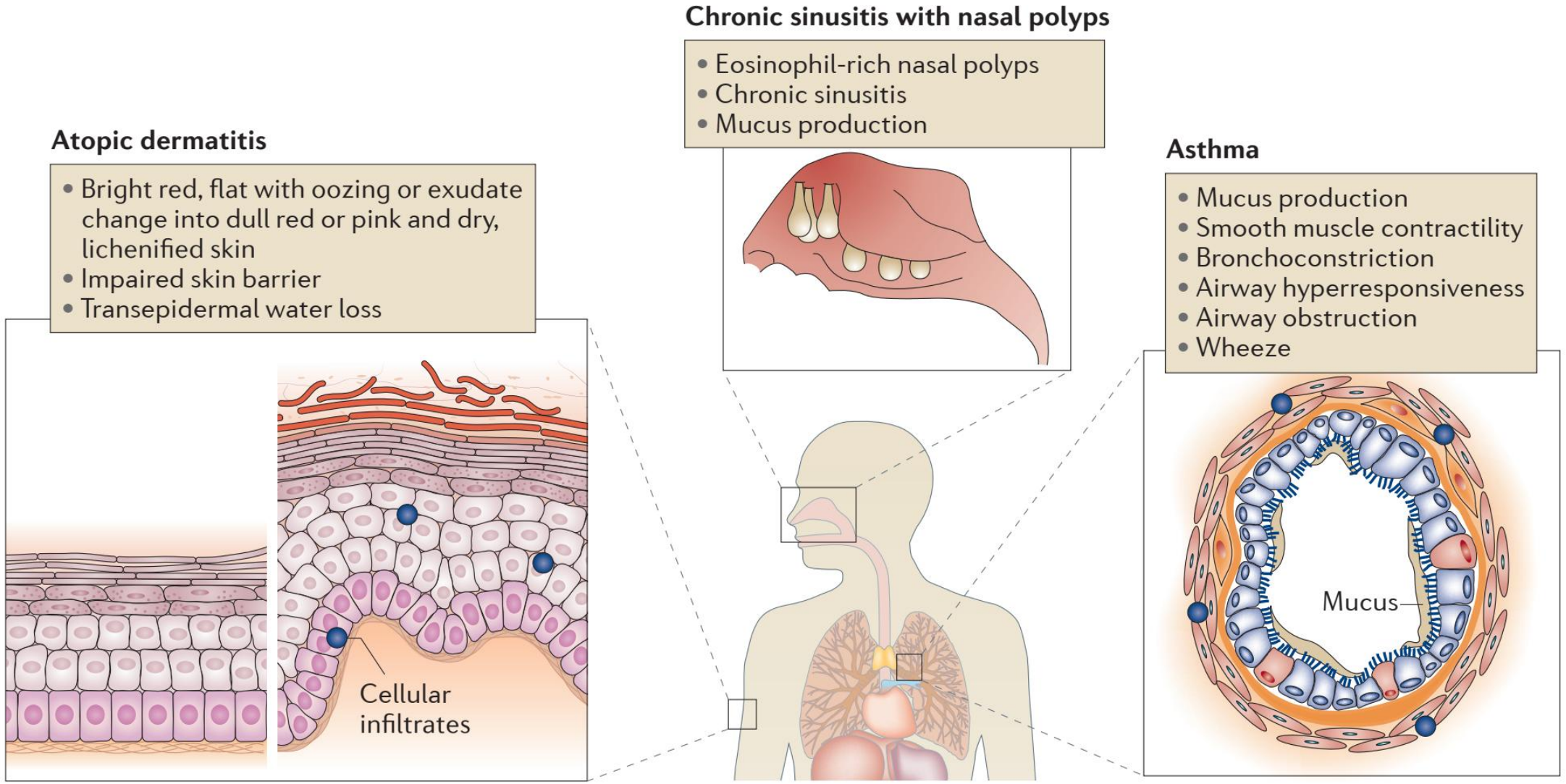
Bachert CC, et al. J Allergy Clin Immunol Pract 2023;11(9):2630-2641.

# Shared type 2 inflammatory pathway across allergic diseases



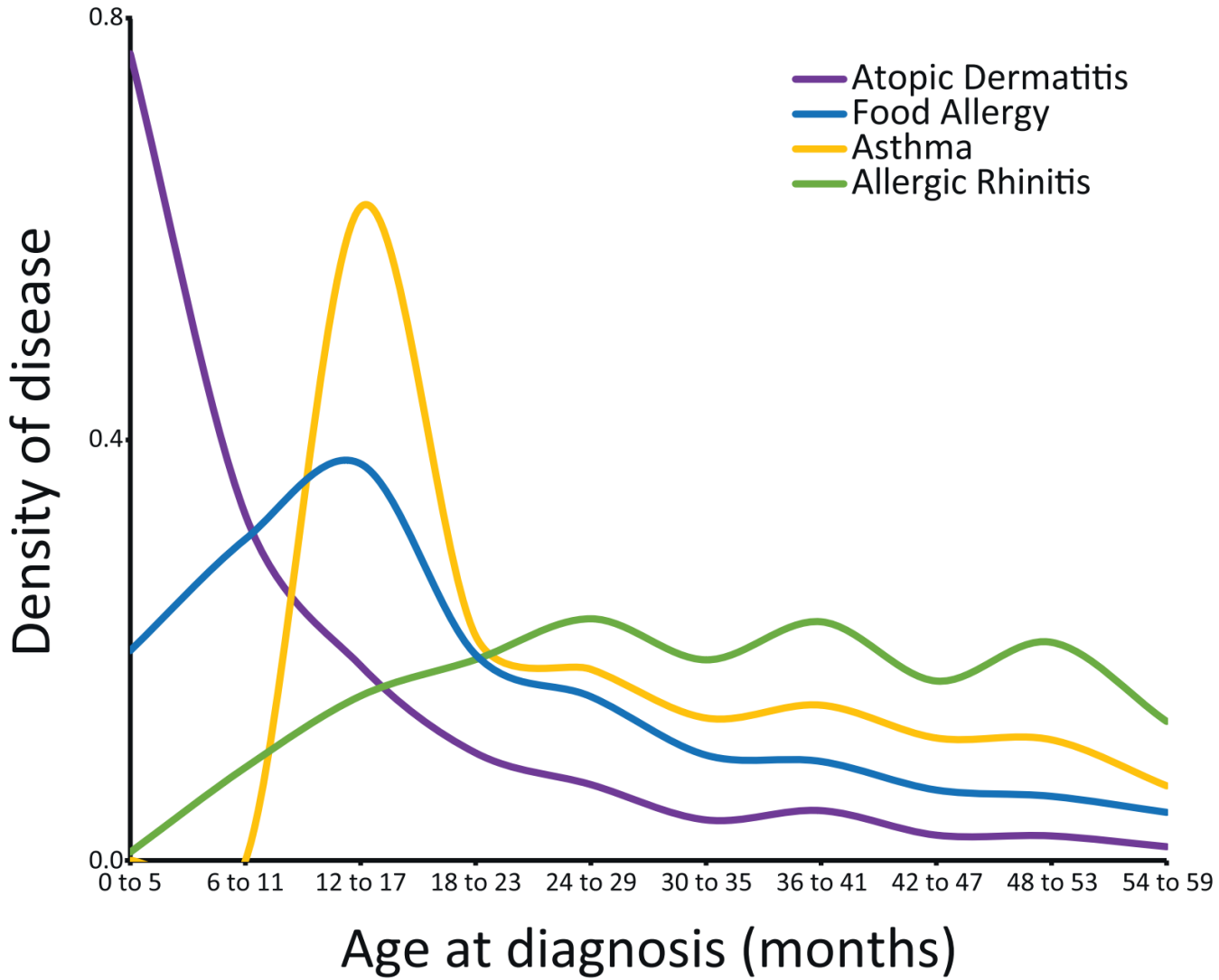

Bachert CC, et al. J Allergy Clin Immunol Pract 2023;11(9):2630-2641.

# Common pathological type 2 pathway activation in different allergic diseases



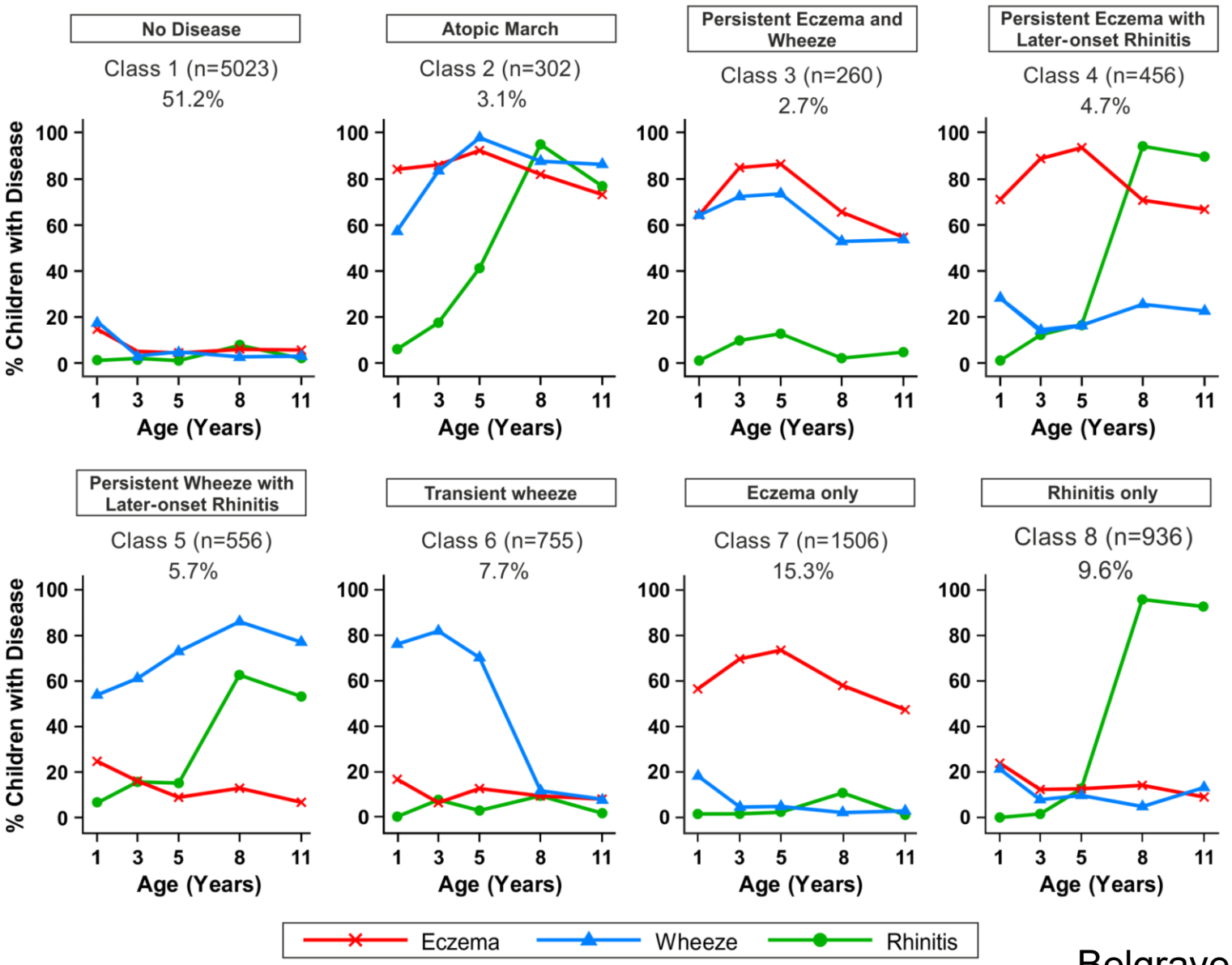

Gandhi NA, et al. Nat Rev Drug Discov 2016;15(1):35-50.D

# Atopic march




Hill DA, et al. Ann Allergy Asthma Immunol 2018;120(2):131-137.

# Atopic march in birth cohort studies

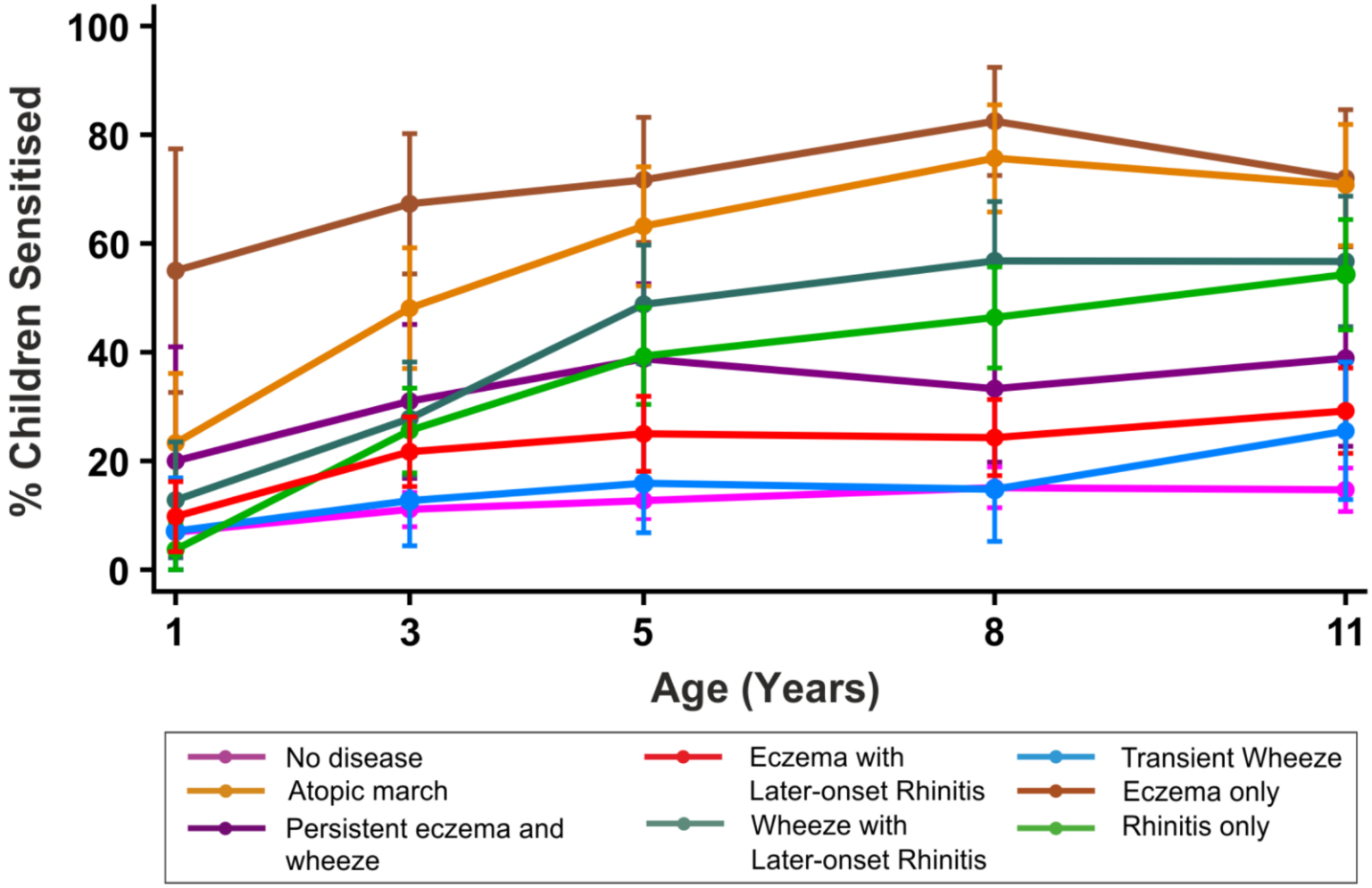



- 9,801 children in UK population-based birth cohorts (MAAS, ALSPAC)
- Parent-reported eczema, wheeze, rhinitis at ages 1, 3, 5, 8, and 11 year

Belgrave DC, et al. PLoS Med 2014;11(10):e1001748.

# Atopic march as a high-risk phenotype

- Atopic sensitization: via skin prick test to inhaled and food allergens

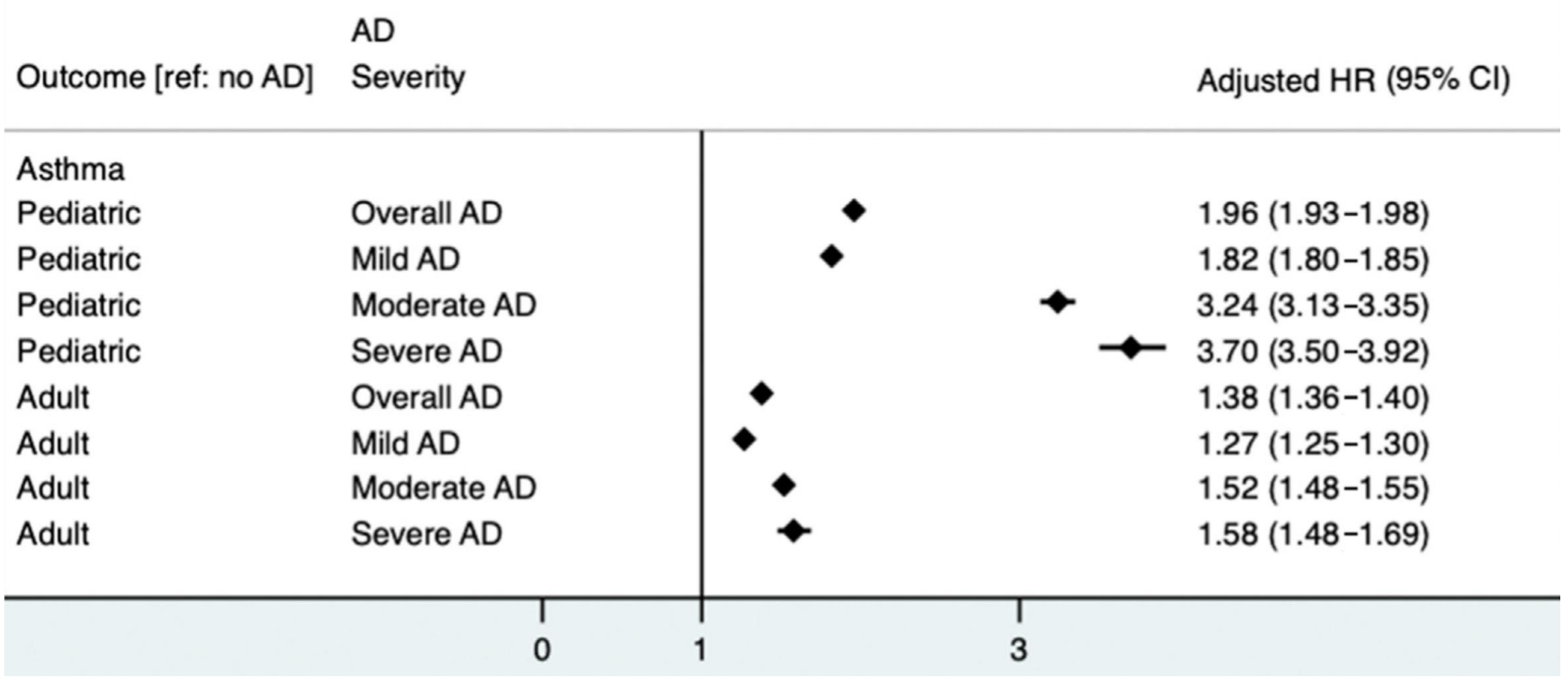



Belgrave DC, et al. PLoS Med 2014;11(10):e1001748.

# Risk of incident asthma in atopic dermatitis

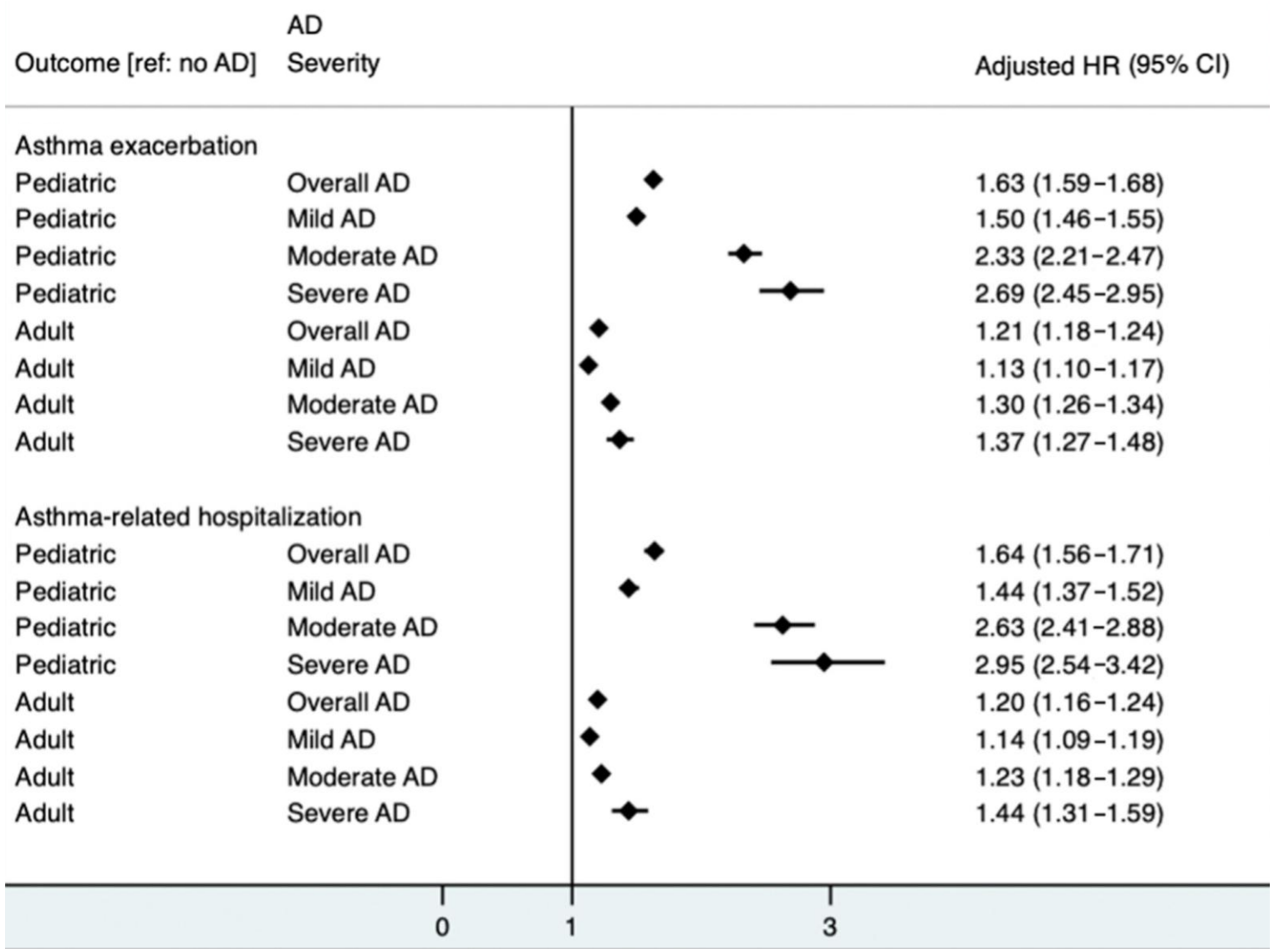
- Pediatric cohort (409,341 children with AD vs. 1,809,029 non-AD) and adult cohort (625,083 AD vs. 2,678,888 non-AD), UK THIN database, 1994-2015

Risk of incident asthma




Wan J, et al. J Allergy Clin Immunol Pract 2024;12(2):421-430.

# Risk of asthma exacerbation in atopic dermatitis




Wan J, et al. J Allergy Clin Immunol Pract 2024;12(2):421-430.

# Clinical characteristics according to CRS in asthma

- Mild-to-severe asthma without CRS (n=16) vs. with CRS (n=31; wNP 15, sNP 16)
- Observational cross-sectional study

| | As (*n* = 16) | As+CRS (*n* = 31) |
|---|---|---|
| Clinical data | | |
| Age, yr (mean ± SD) | 47.7 ± 12.1 | 53.2 ± 11.9 |
| Sex, M/F | 6/10 | 18/13 |
| BMI, kg/m$^2$ | 27.3 ± 5.9 | 26.3 ± 4.4 |
| Asthma onset, yr | 18.0 ± 13.4 | 31.0 ± 15.7* |
| Asthma duration, yr | 29.8 ± 16.0 | 22.2 ± 18.1 |
| Atopy, yes/no | 8/8 | 20/11 |
| Persistent rhinitis, yes/no | 9/7 | 20/11 |
| Nonsmokers | 13/16 (81.3) | 19/31 (61.3) |
| Current smokers§ | 2/16 (12.5) | 0/31 (0.0) |
| Former smokers§ | 1/16 (6.3) | 10/31 (32.2)‖ |
| Exacerbations/yr | 1.0 ± 1.5 | 1.9 ± 1.8‖ |
| ICS/d (μg fluticasone propionate HFA eq) | 378.9 ± 281.8 | 534.7 ± 407.9 |

Carriero V, et al. Ann Am Thorac Soc 2026;23(1):70-79.

# Clinical characteristics according to CRS in asthma

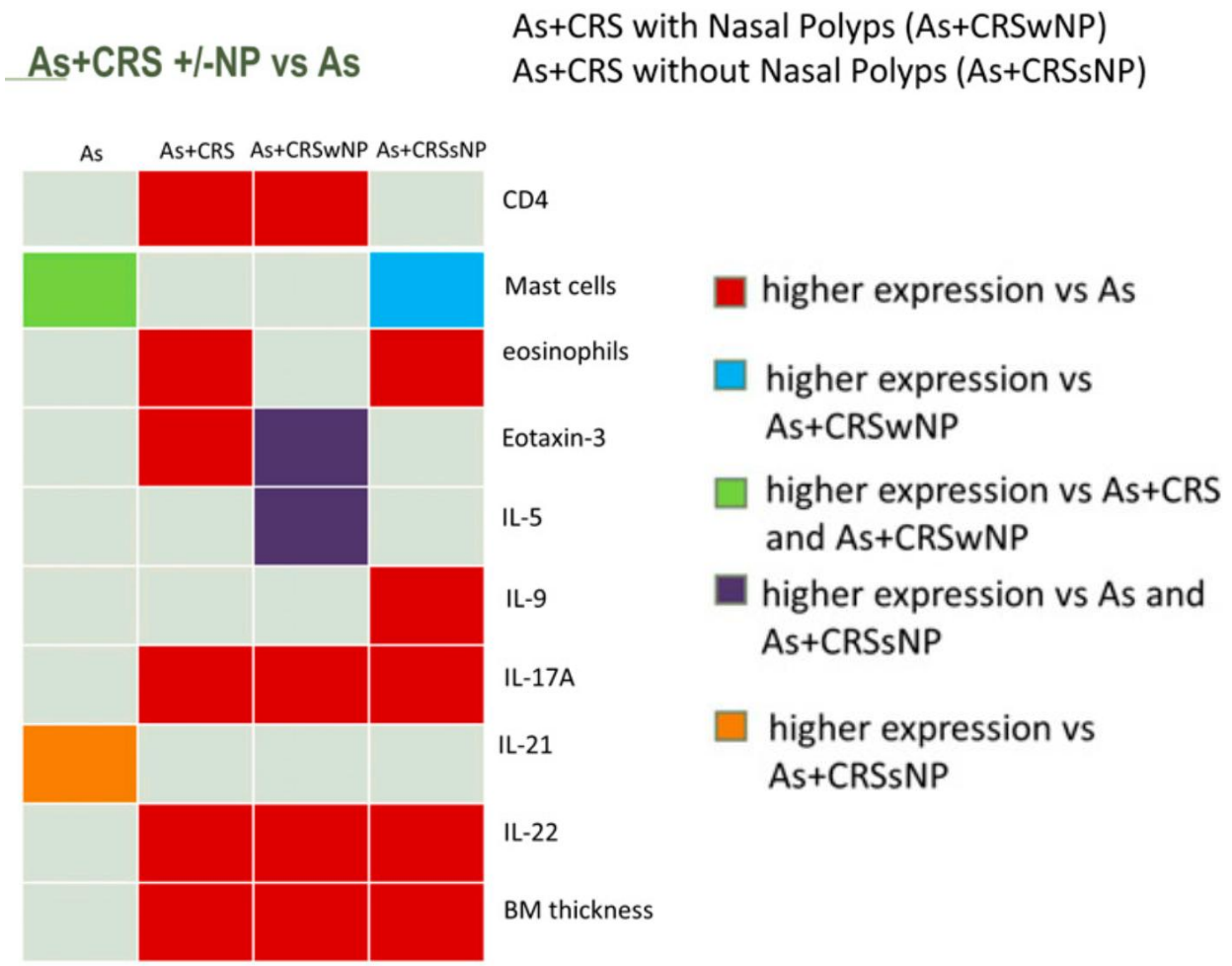
- Mild-to-severe asthma without CRS (n=16) vs. with CRS (n=31; wNP 15, sNP 16)

| | As (*n* = 16) | As+CRS (*n* = 31) |
|---|---|---|
| Functional markers | | |
| $FEV_1$% predicted | 86.6 ± 20.4 | 79.4 ± 26.0 |
| FVC% predicted | 103.7 ± 14.0 | 100.2 ± 24.1 |
| $FEV_1$/FVC% predicted | 0.7 ± 0.1 | 0.6 ± 0.2 |
| RV% predicted | 98.5 ± 15.6 | 137.5 ± 47.6†† |
| Mild-to-moderate asthma | 11/16 (68.75) | 15/31 (48.39) |
| Severe asthma | 5/16 (31.25) | 16/31 (51.61) |
| Limited/extensive CRS§§ | 0/0 | 17/14 |
| OCS use‖‖ | 0/16 (0.0) | 5/31 (16.1) |
| Good/poor control | 13/3 | 25/6 |
| T2 clinical phenotype¶¶ | 5/16 (31) | 21/31 (68)‖ |

Carriero V, et al. Ann Am Thorac Soc 2026;23(1):70-79.

# Endotyping with immunohistochemical data

- Expression of T2, T3, and remodeling biomarkers in the bronchial mucosa
- Bronchial biopsies by immunohistochemistry and immunofluorescence



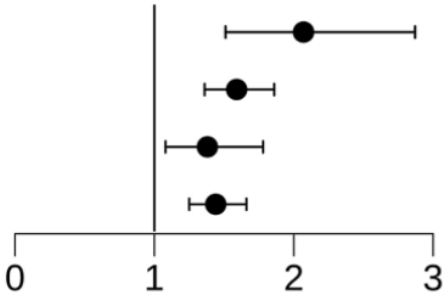

| Biomarker | Asthma severity | CRS comorbidity | Interaction |
|---|---|---|---|
| **CD4** | **0.01** | 0.37 | 0.18 |
| **Eotaxin-3** | **0.002** | 0.94 | 0.80 |
| **IL-13** | **0.01** | **0.008** | **0.007** |
| **IL-17A** | **0.02** | **0.03** | 0.96 |
| **IL-17F** | **0.004** | 0.87 | 0.77 |

Carriero V, et al. Ann Am Thorac Soc 2026;23(1):70-79.

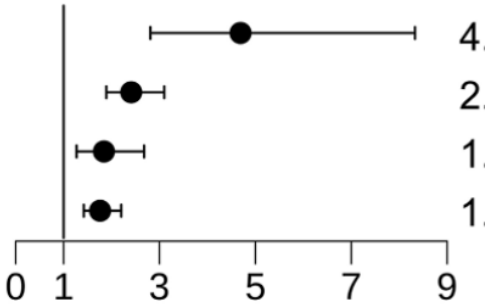
# Impact of CRS on long-term outcomes in asthma

- 16,153 adults with asthma, Ajou University Medical Center, 10-year follow-up
- CRS group (n=2,143): stratified into T2-high and T2-low based on BEC

| Outcomes | Incidence rate (/1,000 PYs) | | HR [95% CI] | *P*-value |
|---|---|---|---|---|
| | CRS group | non-CRS group | | |
| Severe asthma exacerbation | 12.5 | 6.7 | 2.07 [1.51-2.87] | <.001 |
| Asthma exacerbation | 54.8 | 35.2 | 1.59 [1.36-1.86] | <.001 |
| ED visit or hospitalization | 16.1 | 12.1 | 1.38 [1.08-1.78] | .01 |
| Systemic CS prescription | 74.6 | 54.0 | 1.44 [1.25-1.66] | <.001 |




| Outcomes | Incidence rate (/1,000 PYs) | | HR [95% CI] | *P*-value |
|---|---|---|---|---|
| | T2-high group | T2-low group | | |
| Severe asthma exacerbation | 20.7 | 5.6 | 4.69 [2.81-8.33] | <.001 |
| Asthma exacerbation | 79.4 | 32.2 | 2.41 [1.89-3.10] | <.001 |
| ED visit or hospitalization | 21.1 | 12.1 | 1.84 [1.27-2.68] | <.001 |
| Systemic CS prescription | 98.7 | 54.8 | 1.76 [1.42-2.20] | <.001 |

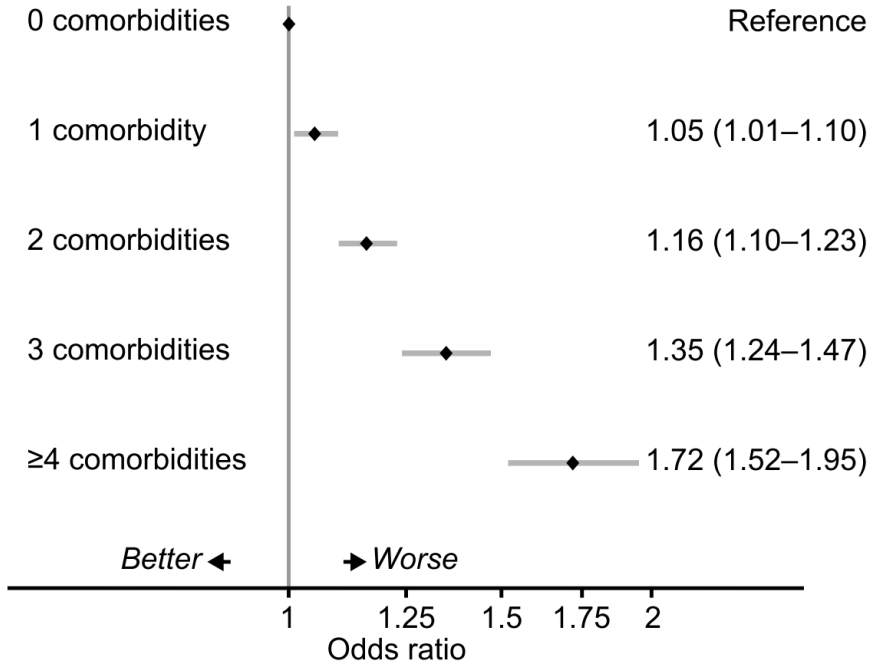



Woo SD, et al. J Allergy Clin Immunol Pract 2026;14:704-712.

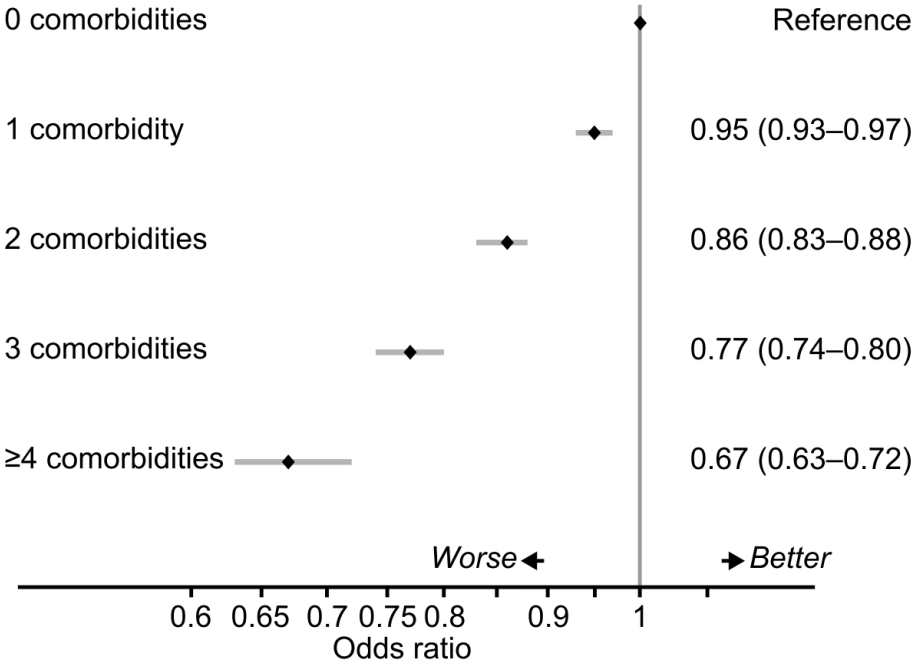
# Impact of CRS on long-term outcomes in asthma

- 245,893 asthma patients from UK primary care practices
- T2 comorbidity (allergic conjunctivitis, AR, anaphylaxis, eczema/AD, CRS, NP, EoE, food allergy, or urticaria)
  - ✓ ≥1 comorbidity, 66% ; ≥2 comorbidities, 24%

## Asthma exacerbation




## Asthma control




Price D, et al. J Asthma Allergy 2021;14:1173-1183.

# Screening for comorbidities in difficult-to-treat asthma

- Frequent under-diagnosis of comorbidities
- Multidisciplinary, protocolized evaluation for uncovering conditions missed by self-report

| | |
|---|---|
| **Upper airway** | Nasal symptoms, hyposmia; nasal endoscopy and sinus CT |
| **Skin** | History and examination for atopic dermatitis |
| **Esophagus** | Dysphagia and food-impaction history |
| **Drug reactivity** | NSAID / aspirin reaction history |

# Disease-specific diagnostic criteria

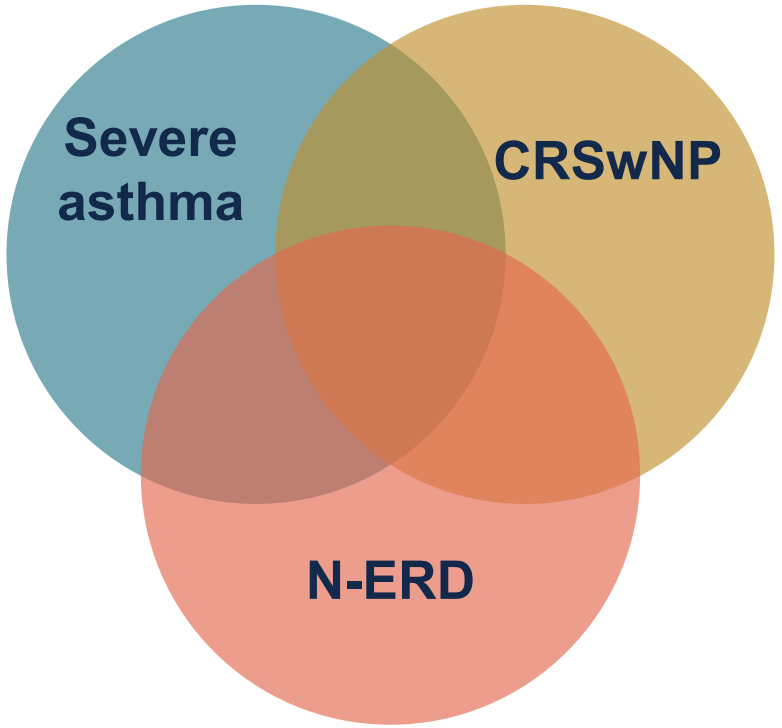
| Condition | Core diagnostic criteria |
|---|---|
| **AR** | Typical symptoms<br>+ demonstrated sensitization (skin-prick test or specific IgE) |
| **CRSwNP** | ≥12 wk of ≥2 symptoms (obstruction, discharge, facial pain, hyposmia)<br>+ bilateral polyps on endoscopy/CT |
| **AD** | Chronic, relapsing, pruritic eczematous dermatitis in a typical distribution |
| **EoE** | Esophageal dysfunction (dysphagia, food impaction)<br>+ ≥15 eosinophils/hpf on biopsy + exclusion of other causes |
| **N-ERD** | Asthma + CRSwNP + NSAID hypersensitivity<br>Confirmed by aspirin provocation (oral/bronchial/nasal) |

# Endotype clustering and eosinophilic multimorbid phenotype

- Clustering on T2 biomarkers (BEC, FeNO, IgE)
  → T2-high, multimorbid phenotype

- Late-onset, non-atopic eosinophilic asthma clusters
  - ✓ tightly with CRSwNP and N-ERD
  - ✓ the most severe, hardest-to-control group

- Asthma is best approached not as a diagnostic label but by shared endotype
  - ✓ Basis of precision medicine



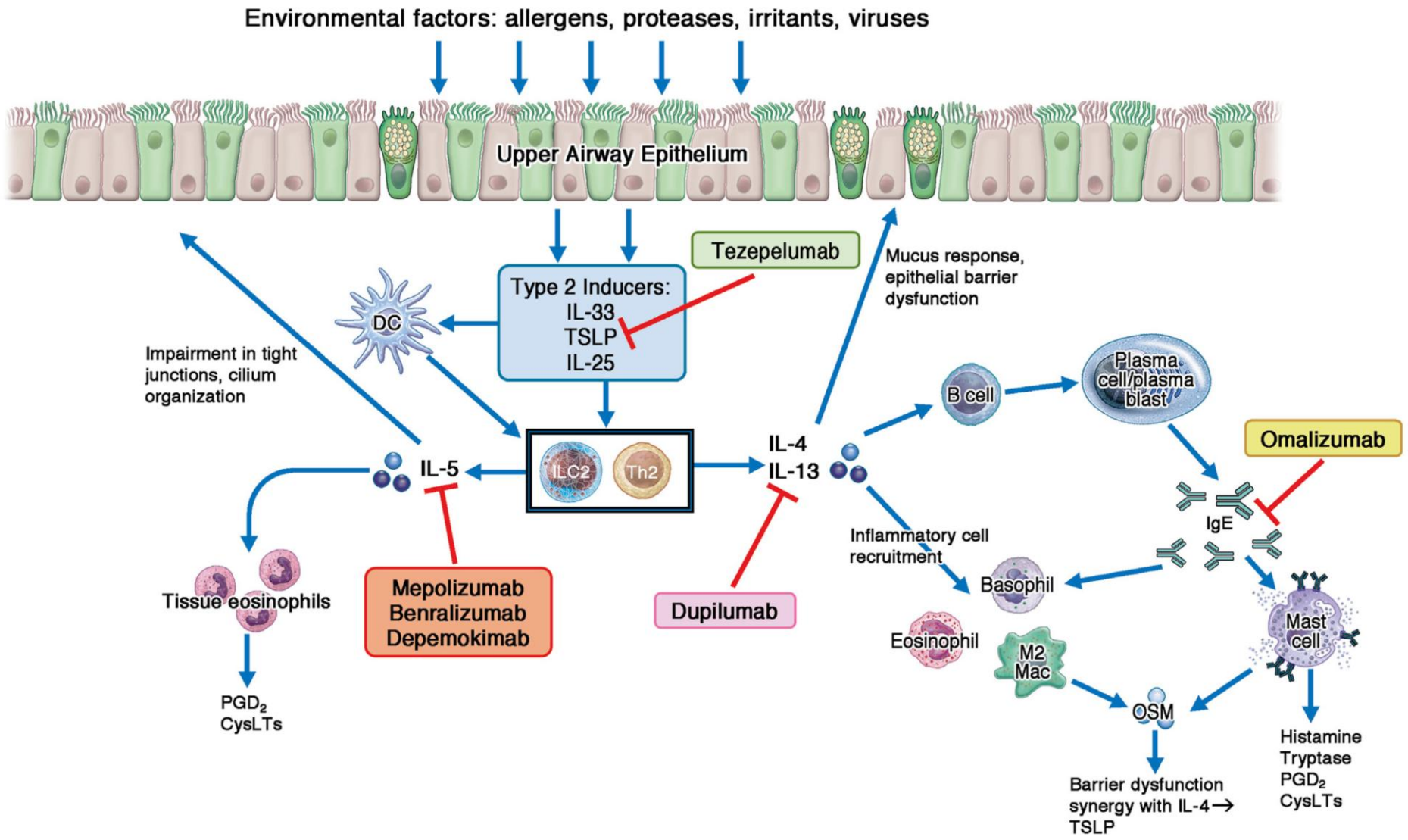

*Severe T2 phenotype*

Denton E, et al. J Allergy Clin Immunol Pract 2021;9(7):2680-2688.e7.

# Standard of care therapy across T2 comorbidities

| Condition | Mainstay pharmacotherapy | Adjunct · procedural |
|---|---|---|
| **AR** | Intranasal corticosteroids<br>2nd-generation oral/intranasal antihistamines<br>LTRA as alternative | Allergen avoidance; Saline irrigation;<br>Allergen immunotherapy (SCIT/SLIT) |
| **CRSwNP** | Intranasal corticosteroids (including steroid nasal irrigation)<br>Saline rinses; Short-course oral corticosteroids | Endoscopic sinus surgery |
| **AD** | Emollients & optimized skin care<br>Topical corticosteroids<br>Topical calcineurin inhibitors<br>Topical PDE4/JAK (crisaborole, ruxolitinib) | Phototherapy (NB-UVB); systemic immunos uppressants (cyclosporin, methotrexate, aza thioprine, MMF);<br>oral JAK inhibitors (upadacitinib, abrocitinib) |
| **EoE** | PPI (first-line); Food elimination diet<br>Swallowed topical corticosteroids (budesonide, fluticasone) | Esophageal dilation for strictures |
| **N-ERD/ AERD** | Strict NSAID avoidance; LTRA<br>Optimize asthma and CRSwNP therapy | Aspirin desensitization |

# Mechanism of T2 inflammation and biologic treatment




Kratchmarov R, et al. J Allergy Clin Immunol 2025;155:1401-1410.

# Efficacy of biologic agents in asthma and CRSwNP

- 16 RCTs, 3598 patients aged >12 years old with moderate-to-severe, severe, uncontrolled/inadequately controlled asthma and concurrent CRSwNP, meta-analysis
- Biologics (anti-IL-5/5Rα, anti-IL-4Rα, anti-TSLP) vs. standard-of-care treatment

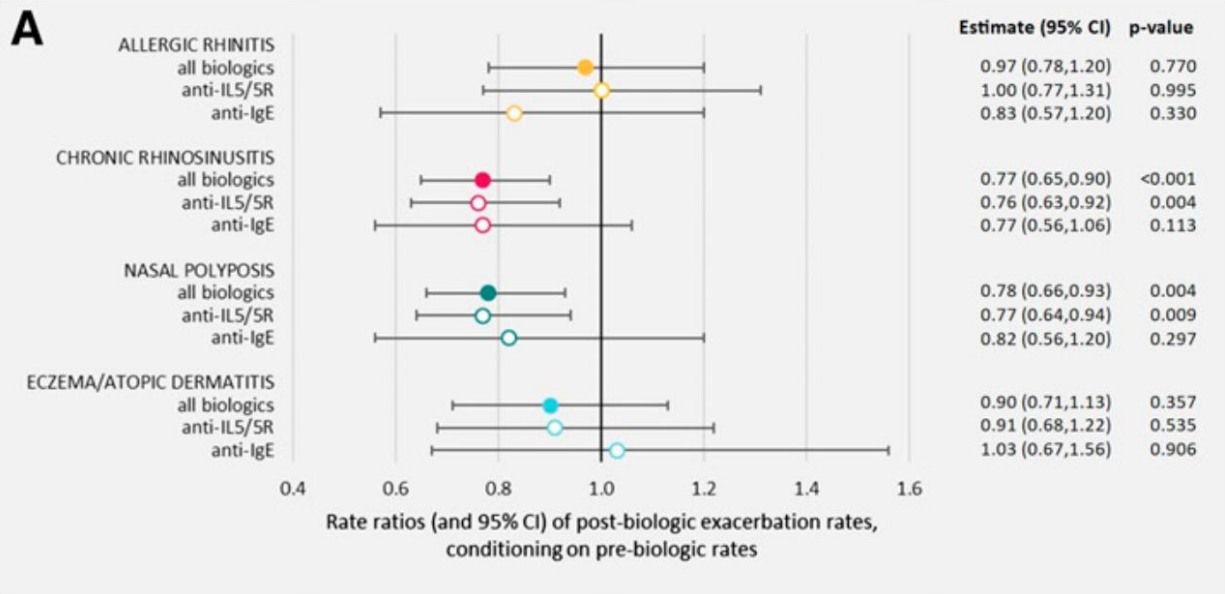
| Asthma exacerbations | Lung function | Asthma control | Quality of life | Sino-nasal outcomes |
|---|---|---|---|---|
| Significant reduction in risk of exacerbations | Improvement in $FEV_1$ | Improvement in ACQ | Improvement in AQLQ | Improvement in SNOT-22, NPS, LMK-CT, and NC scores |
| Exacerbations rate ratio 0.27 (95% CI 0.21–0.34) | $FEV_1$ MD 0.21 L (95% CI 0.11–0.30) | ACQ MD −0.70 (95% CI−0.83–−0.56) | AQLQ MD 0.71 (95% CI 0.49–0.93) | SNOT-22 score MD −15.15 (95% CI −19.64–−10.66) NPS score MD −1.39 (95% CI −1.88–−0.89) LMK-CT score MD −6.64 (95% CI −8.88–−4.40) NC score MD −0.84 (95% CI −1.13–−0.54) |

Kyriakopoulos C, et al. Eur Respir Rev 2026;35(179):250186.

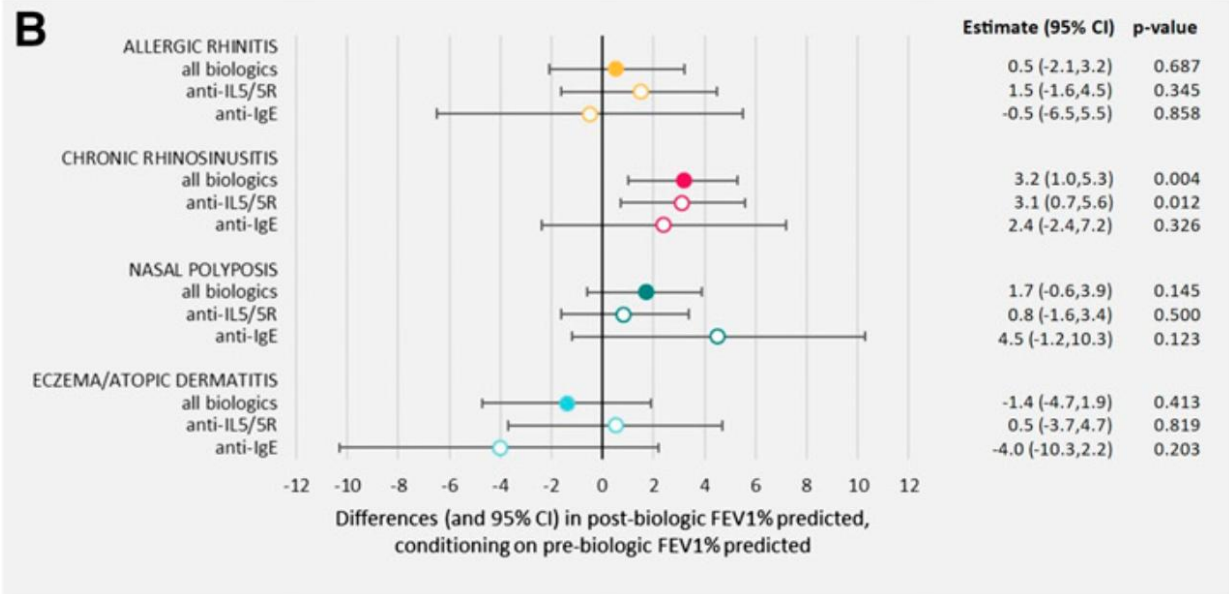
# T2 comorbidities and biologics effectiveness in severe asthma

- 1,765 patients with severe asthma, from 21 countries, ISAR cohort, 2017-2022
- Anti-IL-5/5R (n=1,257), anti-IgE (n=421), anti-IL4/13 (n=87)
- Post-biologic asthma outcomes according to AR, CRSwNP/sNP, NP, or eczema/AD

## Exacerbation rates




## Lung functions

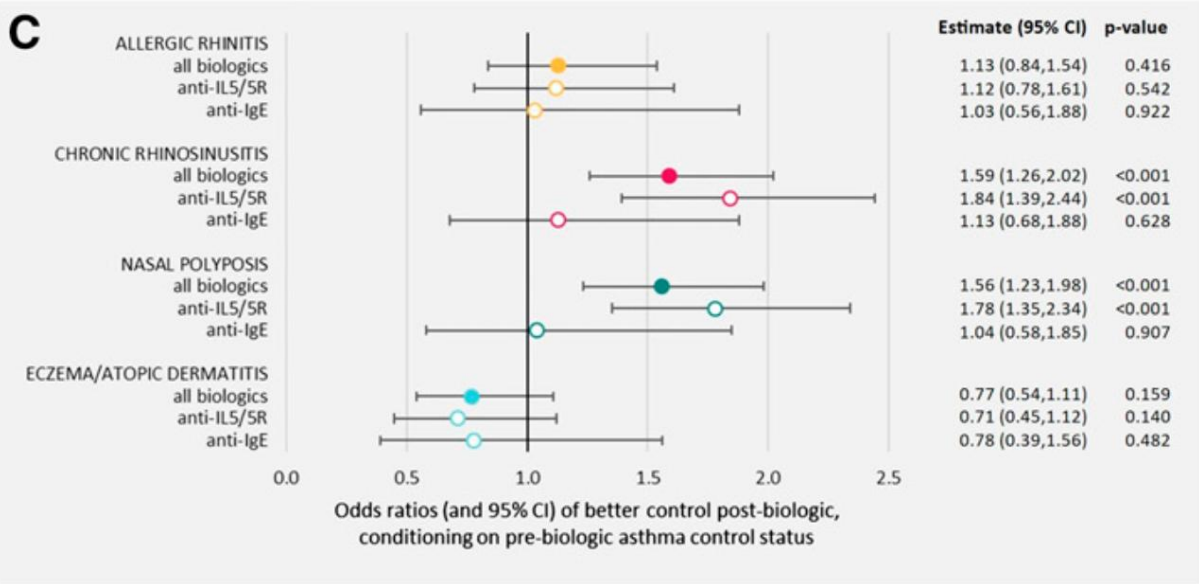


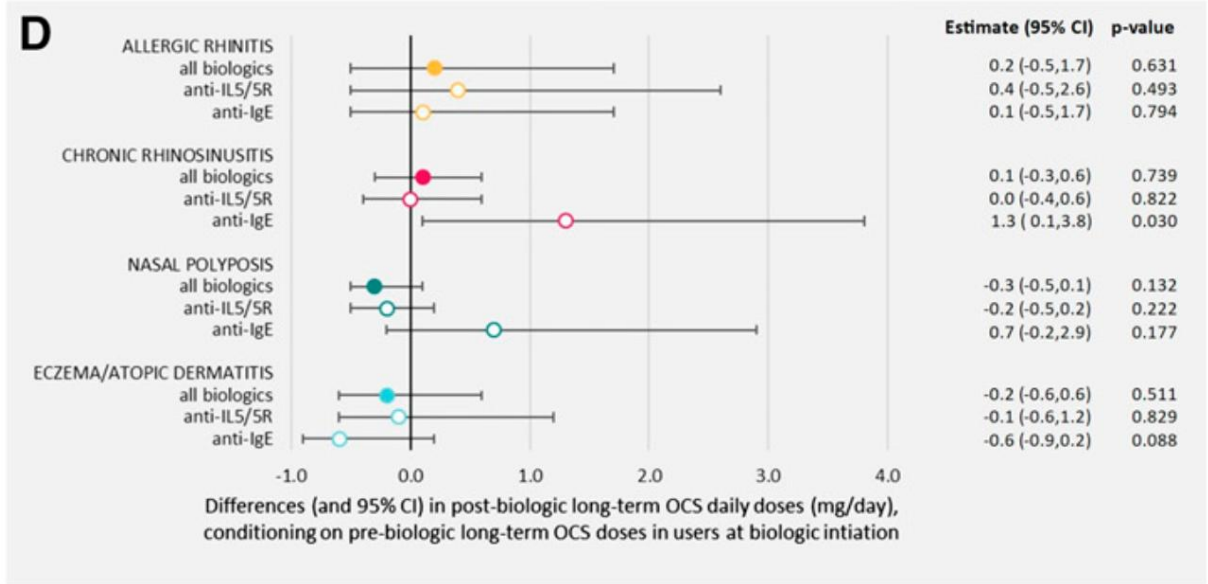

Wechsker ME, et al. Am J Resp Crit Care Med 2024;262-272.

# T2 comorbidities and biologics effectiveness in severe asthma

Asthma control

Long-term OCS daily dose

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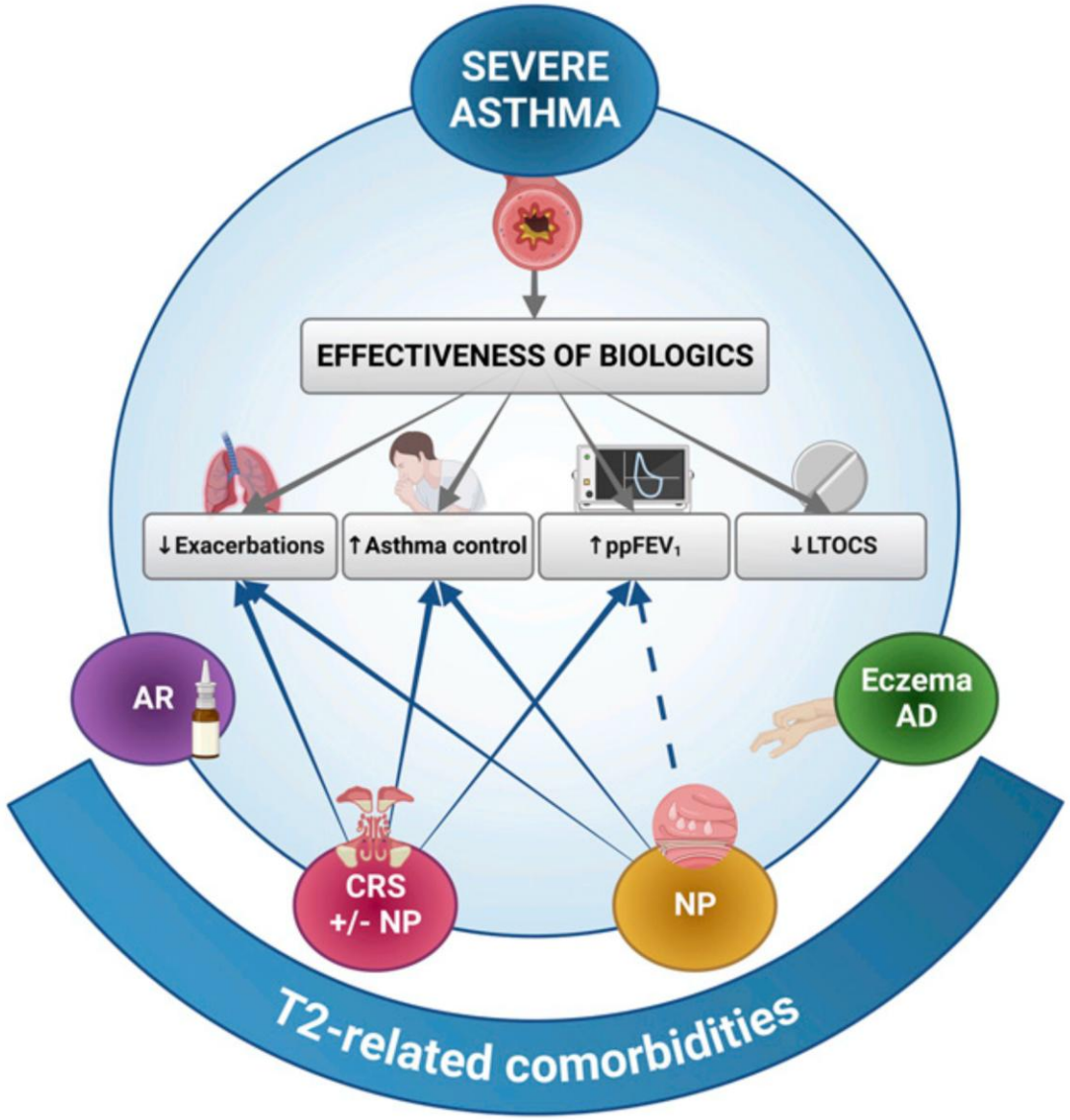
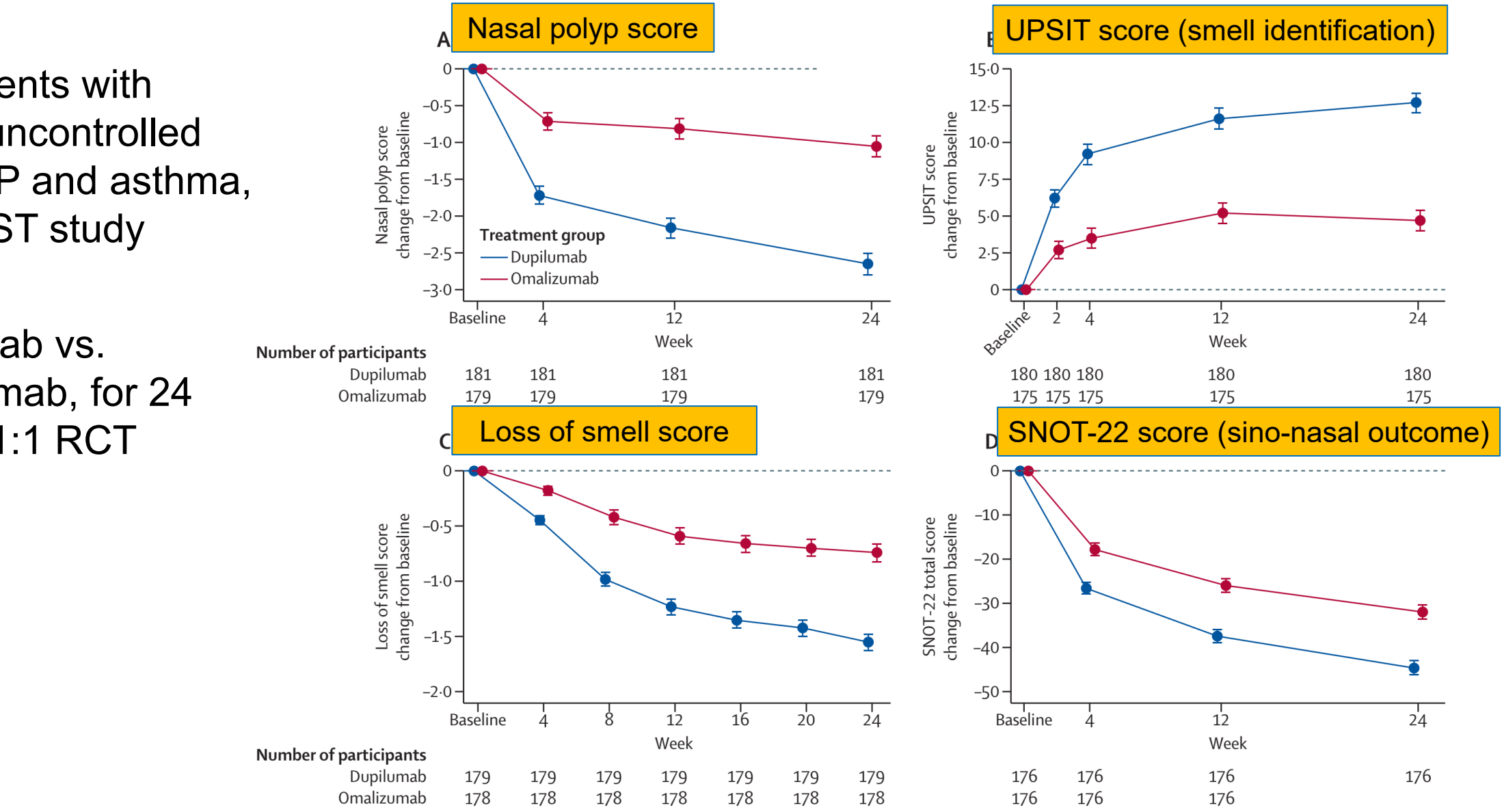



Pelaia C, et al. Am J Respir Crit Care Med 2024;209(3):233-235.

# Dupilumab vs. omalizumab in CRSwNP and asthma

- 360 patients with severe uncontrolled CRSwNP and asthma, EVEREST study
- Dupilumab vs. omalizumab, for 24 weeks, 1:1 RCT




Corso ED, et al. Lancet Respir Med 2025;13:1067-1077.

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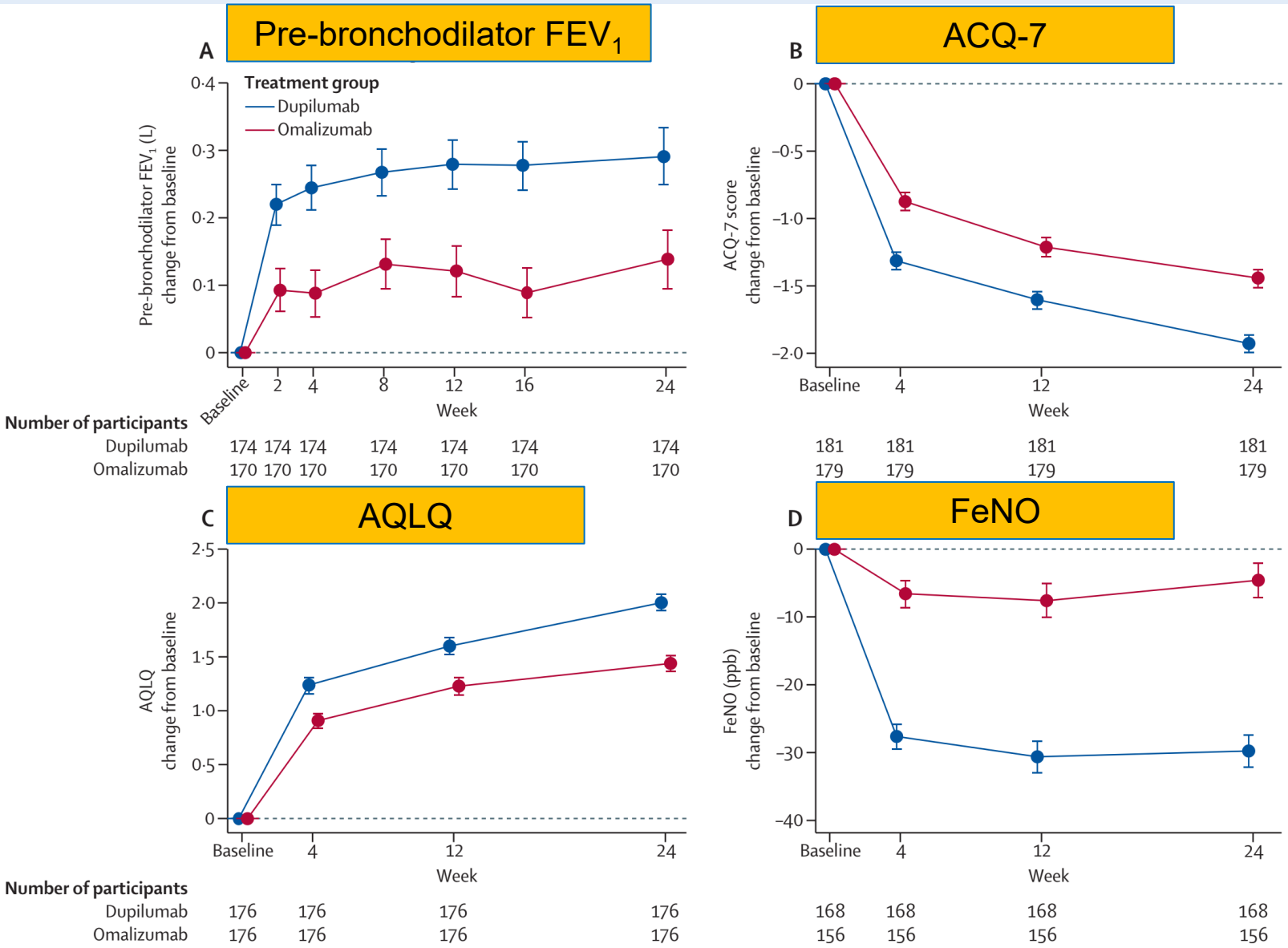
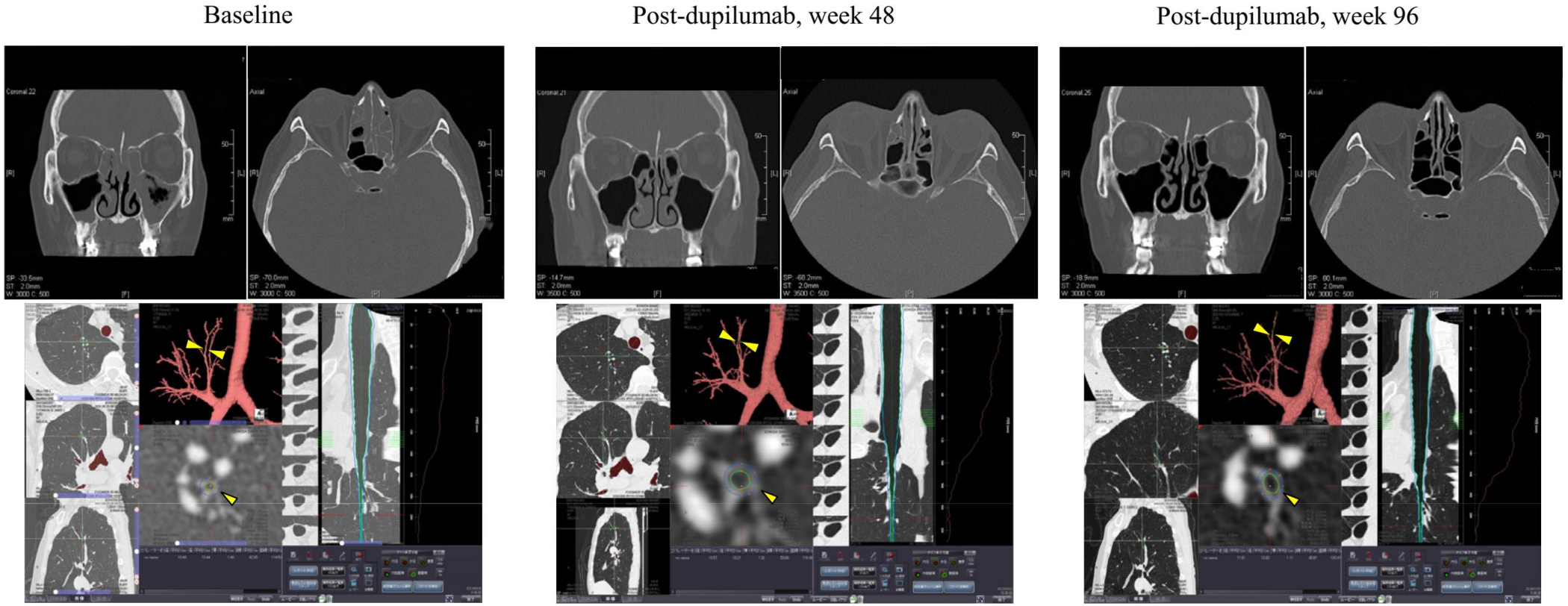



Corso ED, et al. Lancet Respir Med 2025;13:1067-1077.

# Dupilumab effect on respiratory outcome in severe CRSwNP and asthma

- 38 adult patients with severe uncontrolled CRSwNP and moderate-to-severe asthma
- Dupilumab, for 96 weeks; sinus and chest CT follow-up (0, 24, 48, 96-week)
- Sinusitis extent and severity: Lund-Mackey (LMK) score (sinus opacification, mucosal thickening)




Hoshino M, et al. J Allergy Clin Immunol Pract 2025;13(7):1849-1851.

# Dupilumab effect on respiratory outcome in severe CRSwNP and asthma

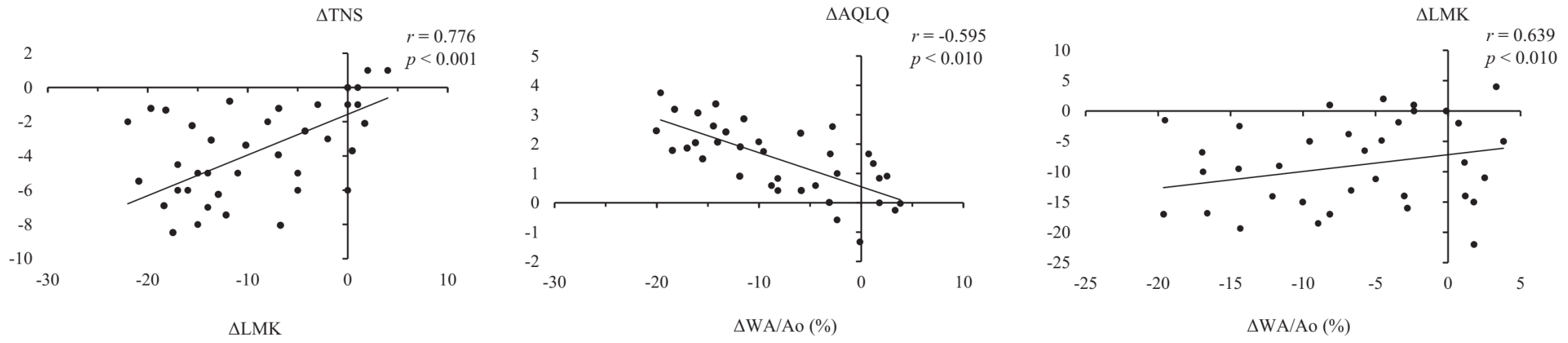



**FIGURE 1.** Relationship between changes in Lund-Mackey (LMK) and total nasal symptom (TNS) scores, airway wall area/total area of the airway (WA/Ao%) and Asthma Quality of Life Questionnaire (AQLQ) scores, and LMK and WA/Ao% after treatment with dupilumab. $\Delta$, week 96 to baseline.

Hoshino M, et al. J Allergy Clin Immunol Pract 2025;13(7):1849-1851.

# Tezepelumab in severe CRSwNP

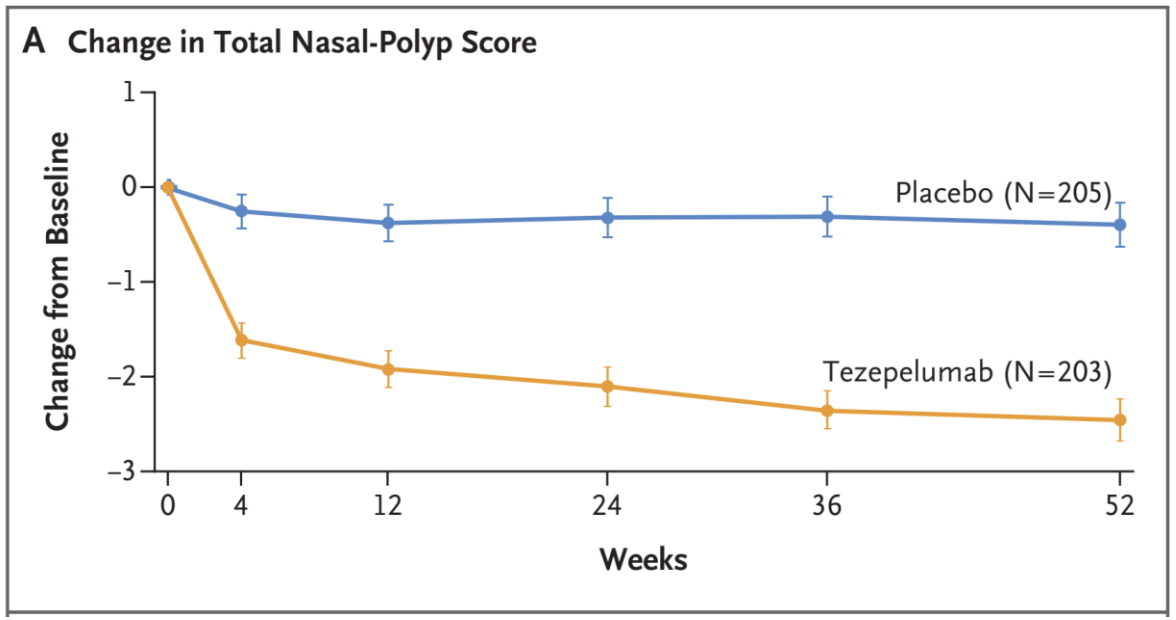
- 203 adult patients with symptomatic, severe CRSwNP
- Tezepelumab vs. standard care, for 52 weeks, WAYPOINT study

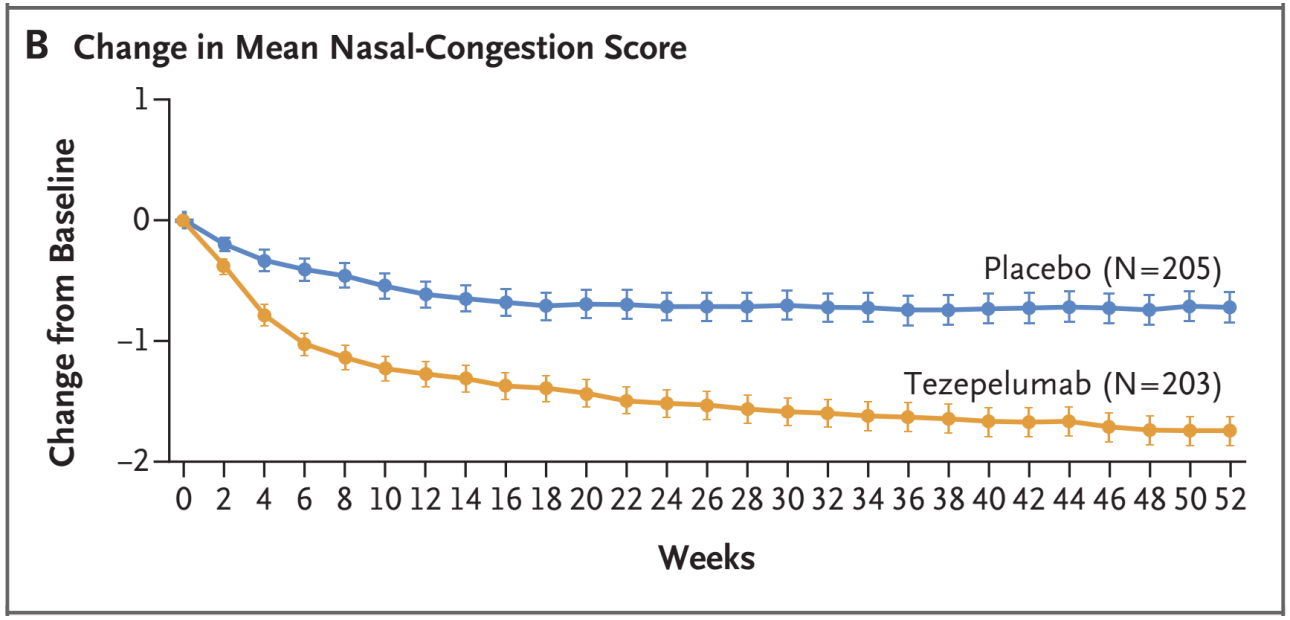
**Table 1. Demographic and Clinical Characteristics of the Patients at Baseline.***

| Characteristic | Tezepelumab (N=203) | Placebo (N=205) | Overall (N=408) |
|---|---|---|---|
| Age — yr | 50.1±13.6 | 49.4±13.7 | 49.7±13.6 |
| Male sex — no. (%) | 126 (62.1) | 140 (68.3) | 266 (65.2) |
| Time since diagnosis of CRS with nasal polyps — yr | 12.71±10.43 | 12.80±0.34 | 12.75±10.37 |
| Previous nasal-polyp surgery — no. (%) | 144 (70.9) | 147 (71.7) | 291 (71.3) |
| Time since last nasal-polyp surgery — yr† | 7.71±6.54 | 7.68±6.24 | 7.70±6.38 |
| Previous systemic glucocorticoid treatment for CRS with nasal polyps — no. (%) | 130 (64.0) | 137 (66.8) | 267 (65.4) |
| Coexisting asthma — no. (%)‡ | 122 (60.1) | 126 (61.5) | 248 (60.8) |
| Coexisting NSAID-exacerbated respiratory disease — no. (%) | 34 (16.7) | 37 (18.0) | 71 (17.4) |
| Blood eosinophil count | | | |
| Mean — cells/μl | 357±229 | 359±248 | 358±238 |
| Prebronchodilator $FEV_1$ — % of predicted normal value§§ | 87.2±16.7 | 84.4±16.1 | 85.8±16.4 |
| UPSIT score¶¶ | 13.1±7.3 | 11.9±5.9 | 12.5±6.6 |
| ACQ-6 score§§‖‖ | 1.85±1.23 | 1.80±1.10 | 1.82±1.16 |

Lipworth BJ, et al. N Engl J Med 2025;392:1178-1188.

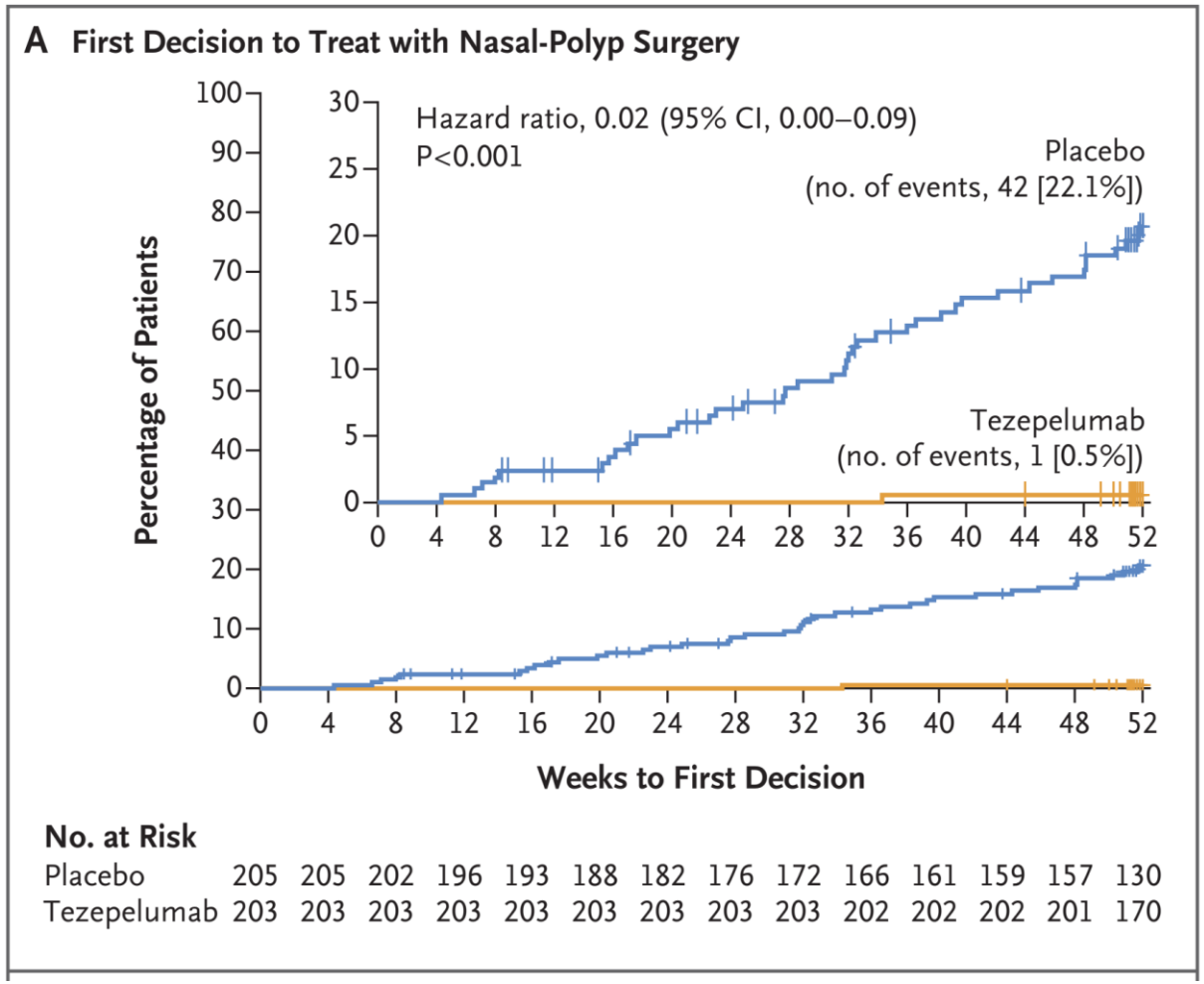
# Tezepelumab in severe CRSwNP

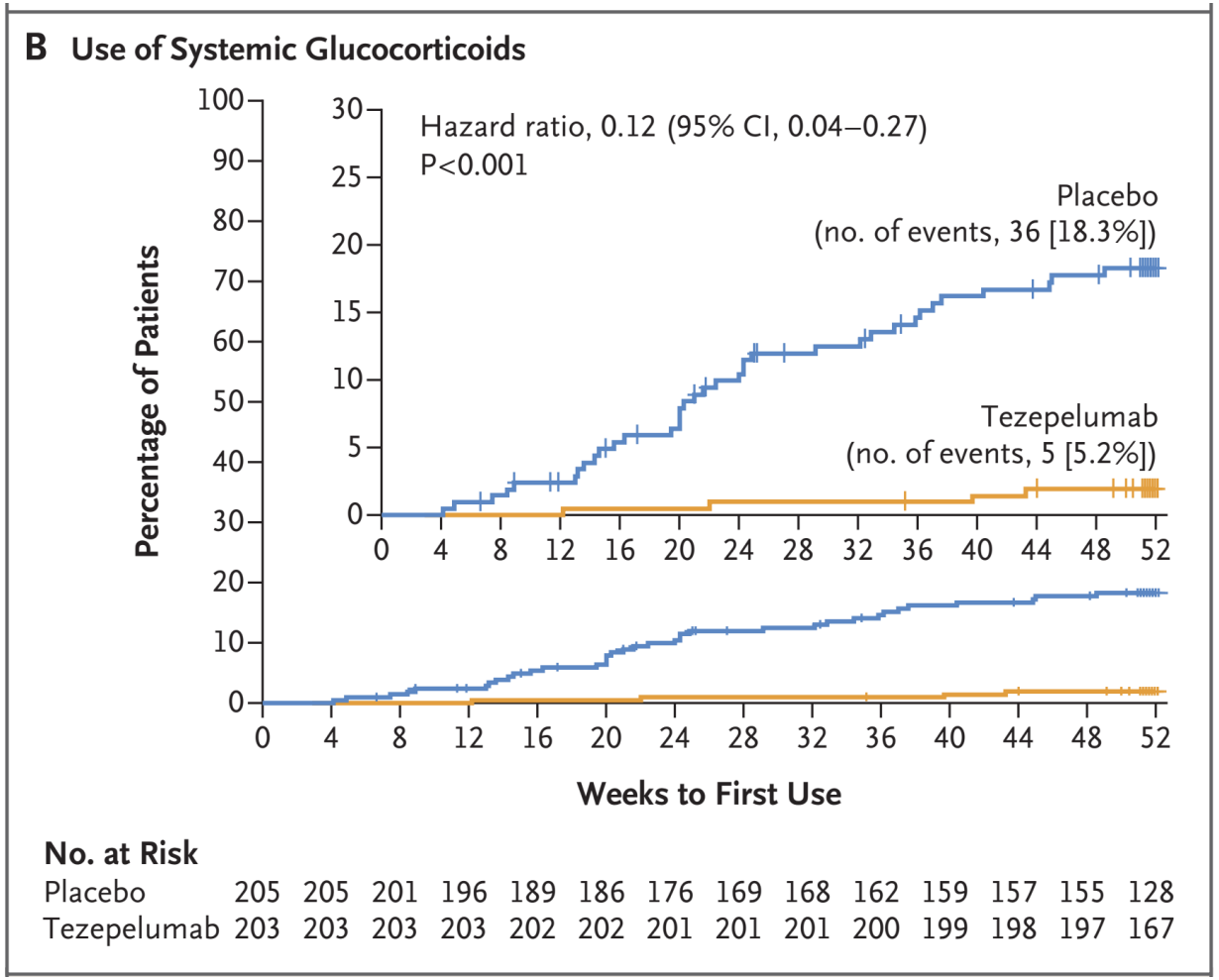



Lipworth BJ, et al. N Engl J Med 2025;392:1178-1188.

# Tezepelumab in severe CRSwNP

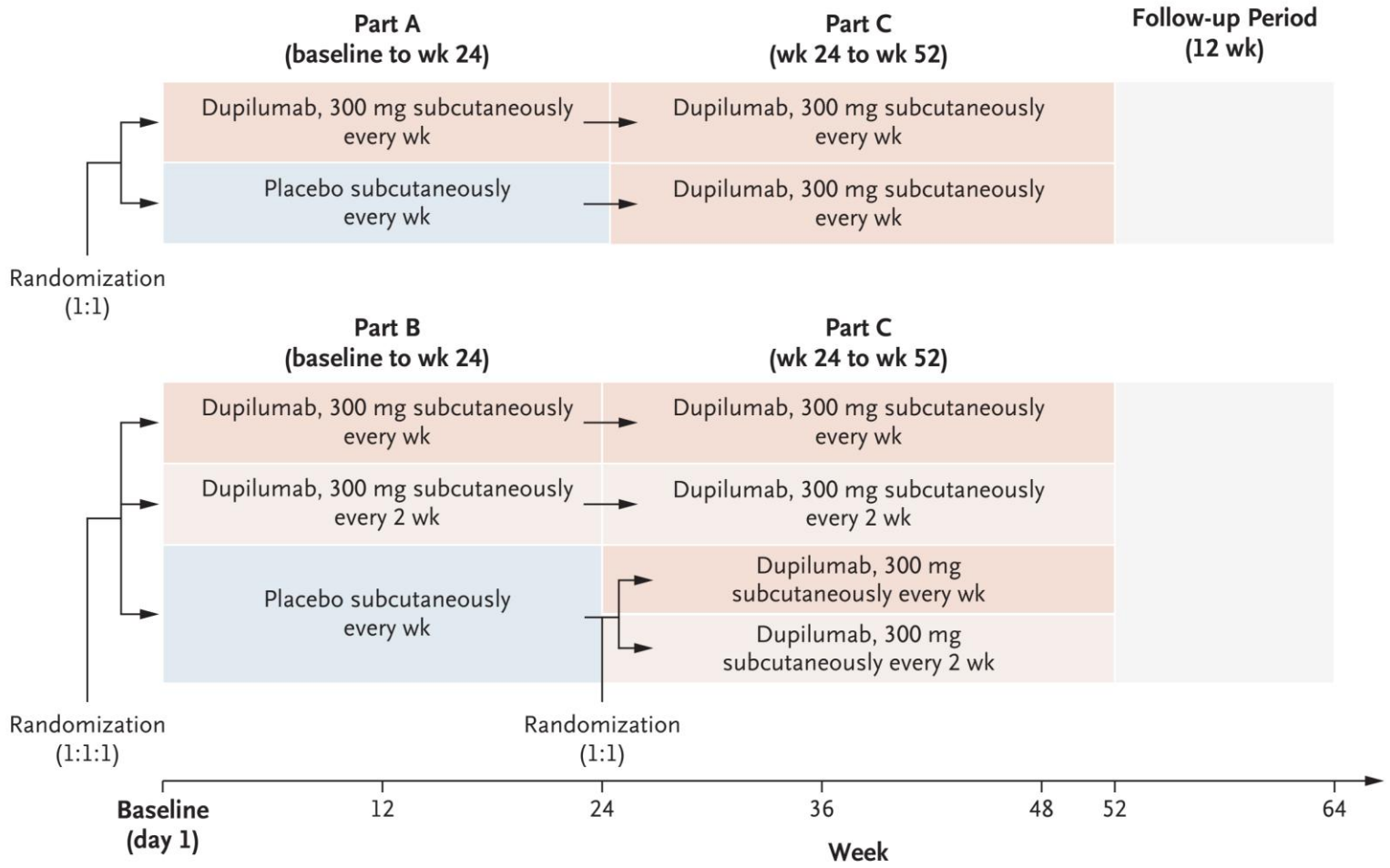



Lipworth BJ, et al. N Engl J Med 2025;392:1178-1188.

# Dupilumab in eosinophilic esophagitis

- 42 patients with eosinophilic esophagitis, diagnosed by endoscopic biopsy despite 8 weeks of high-dose PPI therapy




Dellon ES, et al. N Engl J Med 2022;387:2317-2330.

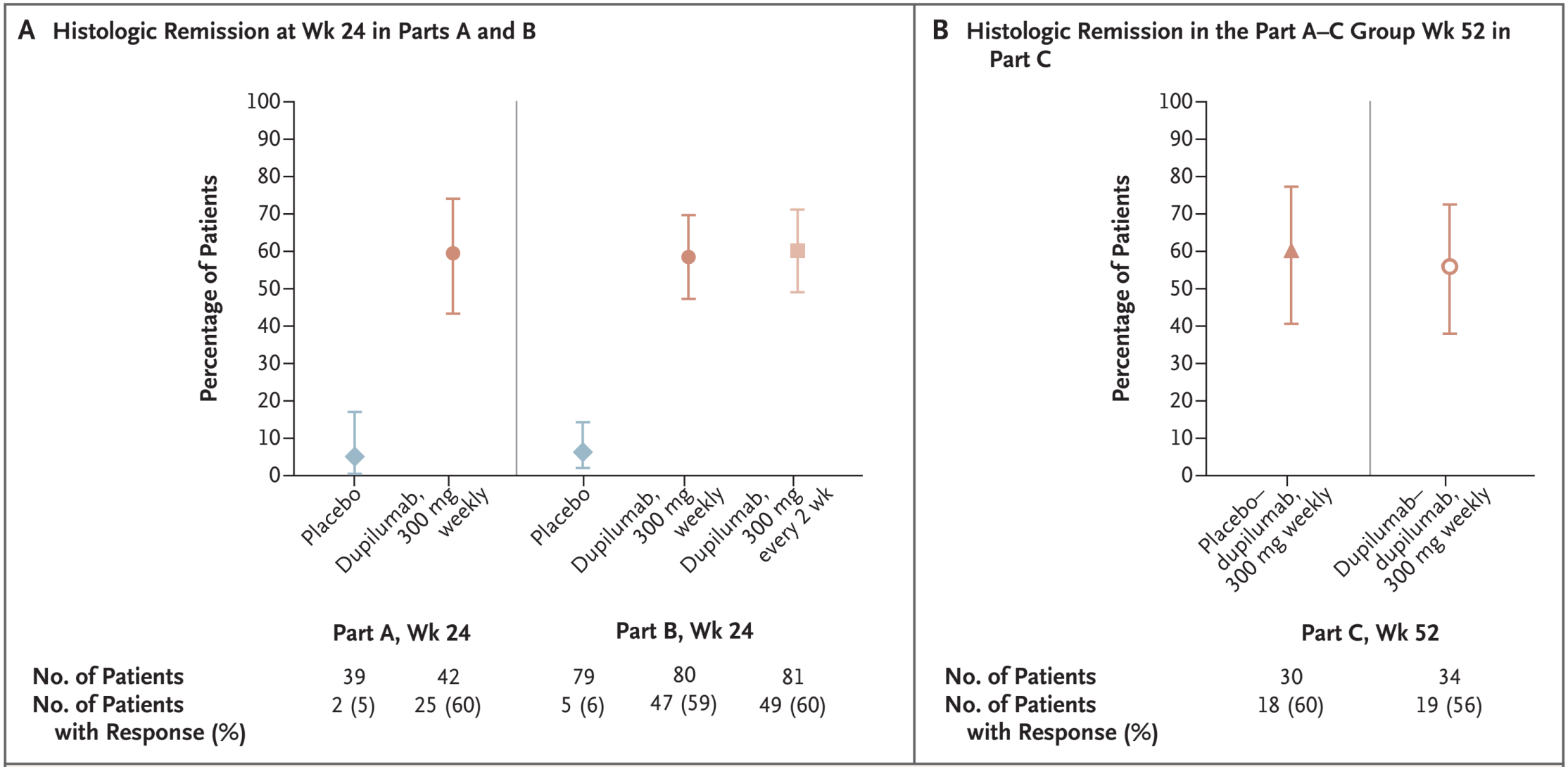
# Dupilumab in eosinophilic esophagitis

**Table 1. Selected Demographic and Clinical Characteristics of the Patients at Baseline (Full Analysis Set).***

| Characteristic | Part A | | | Part B | | | |
|---|---|---|---|---|---|---|---|
| | Dupilumab, 300 mg weekly (N=42) | Placebo (N=39) | Total (N=81) | Dupilumab, 300 mg weekly (N=80) | Dupilumab, 300 mg every 2 wk (N=81) | Placebo (N=79) | Total (N=240) |
| Age — yr | 33.9±15.53 | 28.8±12.53 | 31.5±14.31 | 28.7±13.72 | 27.8±13.21 | 27.9±12.56 | 28.1±13.12 |
| Female sex — no. (%) | 14 (33) | 18 (46) | 32 (40) | 30 (38) | 36 (44) | 21 (27) | 87 (36) |
| Duration of eosinophilic esophagitis — yr† | 5.23±4.18 | 4.77±4.55 | 5.01±4.34 | 5.89±4.66 | 5.92±5.18 | 4.88±4.48 | 5.57±4.79 |
| Previous use of topical glucocorticoids for eosinophilic esophagitis — no. (%) | 29 (69) | 31 (79) | 60 (74) | 55 (69) | 65 (80) | 56 (71) | 176 (73) |
| Refractory to previous therapy — no. (% of patients with previous use) | 23 (79) | 21 (68) | 44 (73) | 32 (58) | 38 (58) | 34 (61) | 104 (59) |
| Inadequate response to or unacceptable side effects from previous therapy or current contraindication — no. (%)‡ | — | — | — | 38 (48) | 41 (51) | 39 (49) | 118 (49) |
| Presence of concurrent type 2 inflammatory disease — no. (%) | 33 (79) | 35 (90) | 68 (84) | 71 (89) | 74 (91) | 69 (87) | 214 (89) |
| Allergic rhinitis | 26 (62) | 22 (56) | 48 (59) | 48 (60) | 49 (60) | 52 (66) | 149 (62) |
| Food allergy | 19 (45) | 17 (44) | 36 (44) | 46 (58) | 42 (52) | 41 (52) | 129 (54) |
| Asthma | 10 (24) | 15 (38) | 25 (31) | 32 (40) | 31 (38) | 27 (34) | 90 (38) |
| Atopic dermatitis | 6 (14) | 9 (23) | 15 (19) | 12 (15) | 17 (21) | 19 (24) | 48 (20) |
| Peak eosinophil count per high-power field** | 82.6±41.02 | 96.5±54.69 | 89.3±48.29 | 89.2±46.67 | 87.7±49.37 | 84.3±41.20 | 87.1±45.76 |
| Median blood peripheral eosinophils (IQR) — IU/ml | 430 (260–600) | 450 (270–680) | 440 (270–610) | 420 (280–520) | 380 (250–510) | 430 (270–530) | 400 (270–520) |
| Median IgE (IQR) — IU/ml | 110 (51–463) | 100 (47–294) | 107 (50–306) | 134 (48–302) | 134 (47–362) | 126 (52–416) | 134 (48–330) |

Dellon ES, et al. N Engl J Med 2022;387:2317-2330.

# Dupilumab in eosinophilic esophagitis

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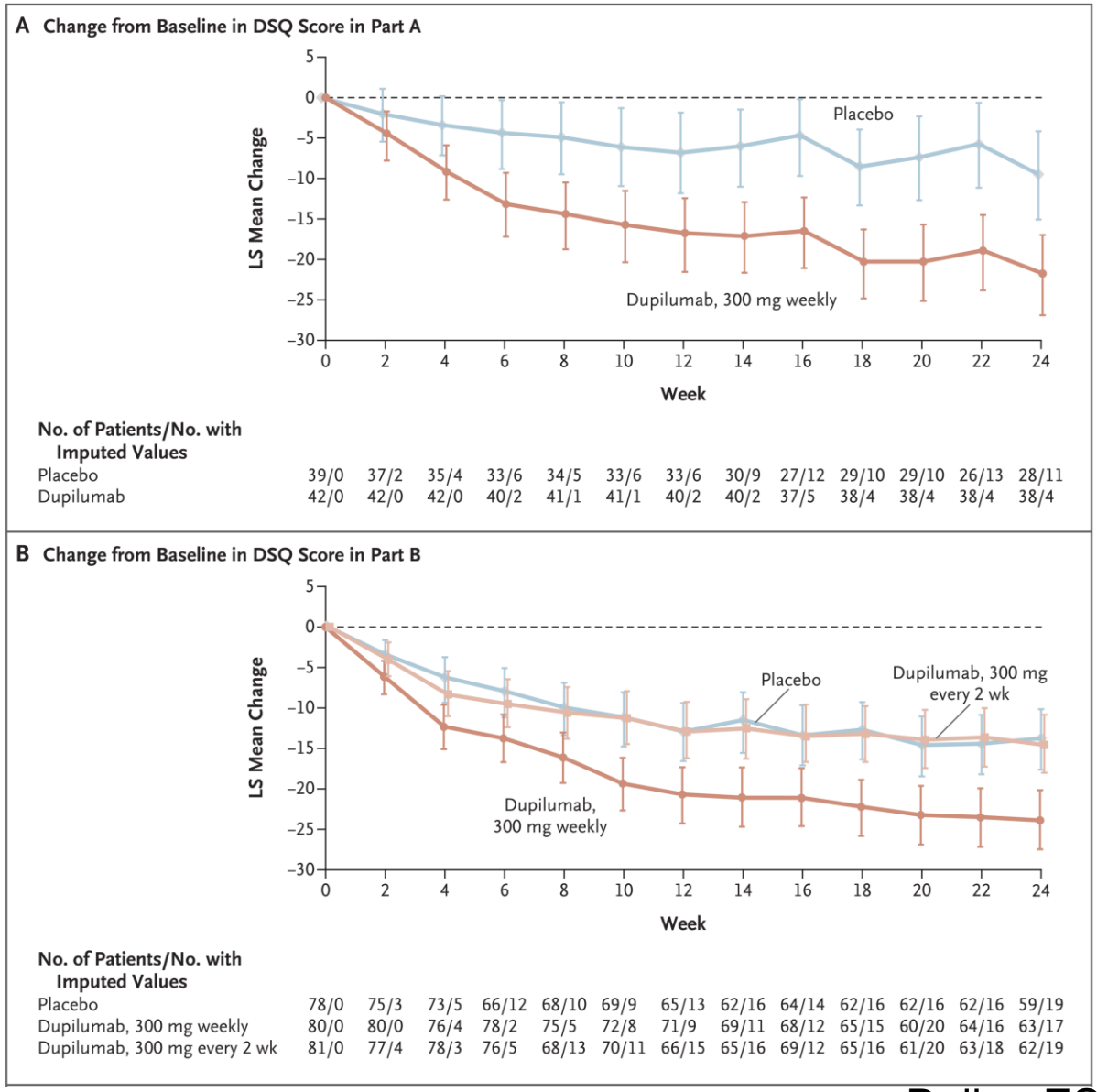
# Dupilumab in eosinophilic esophagitis




***DSQ score**
Dysphagia Symptom Questionnaire score

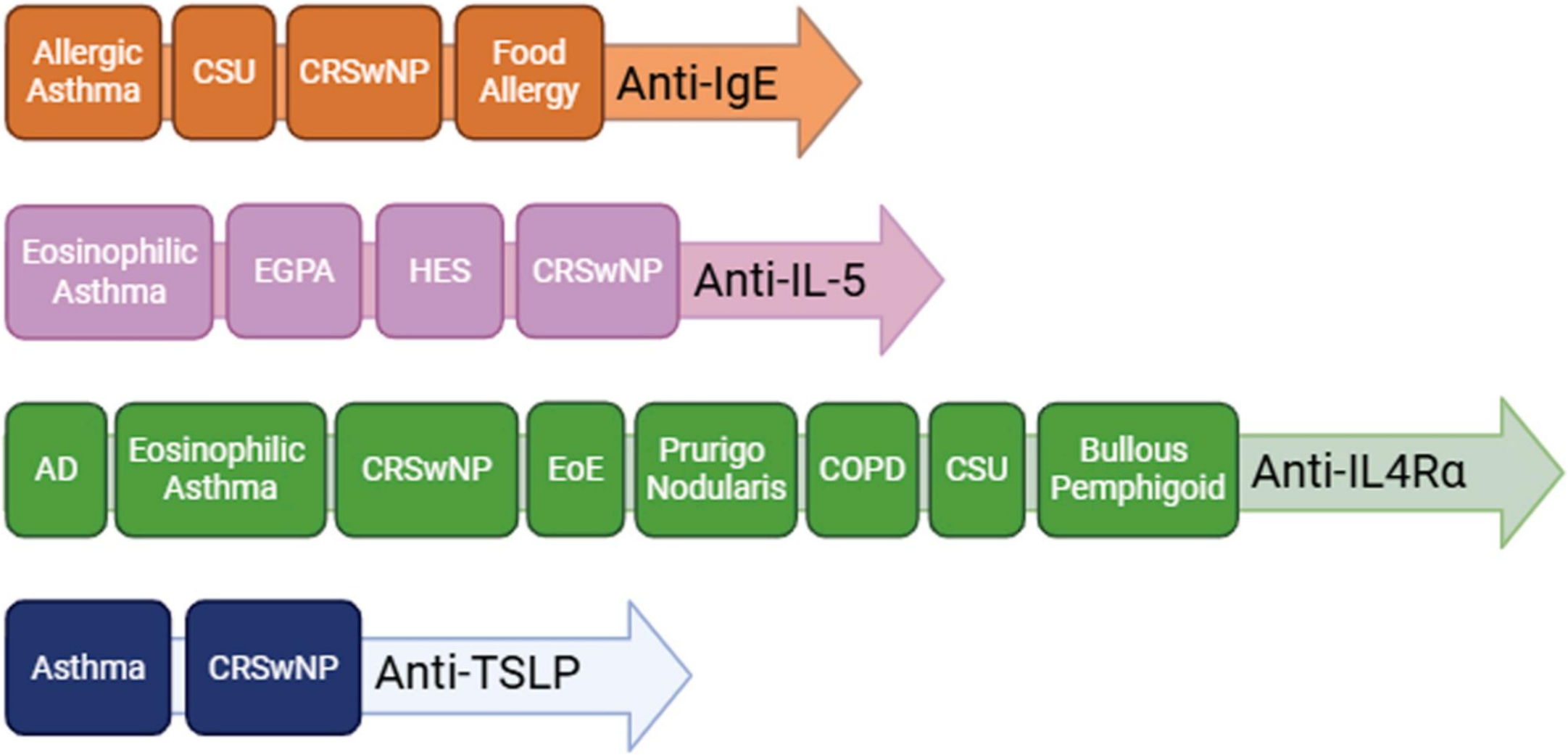
Dellon ES, et al. N Engl J Med 2022;387:2317-2330.

# Biologics selection according to multiple comorbidities

| Biologics | Target | Asthma | CRSwNP | AD | EoE |
|---|---|---|---|---|---|
| Omalizumab | IgE | ✓ | ✓ | – | – |
| Mepolizumab | IL-5 | ✓ | ✓ | – | – |
| Benralizumab | IL-5Rα | ✓ | (+) | – | – |
| Dupilumab | IL-4Rα | ✓ | ✓ | ✓ | ✓ |
| Tezepelumab | TSLP | ✓ | (+) | – | – |

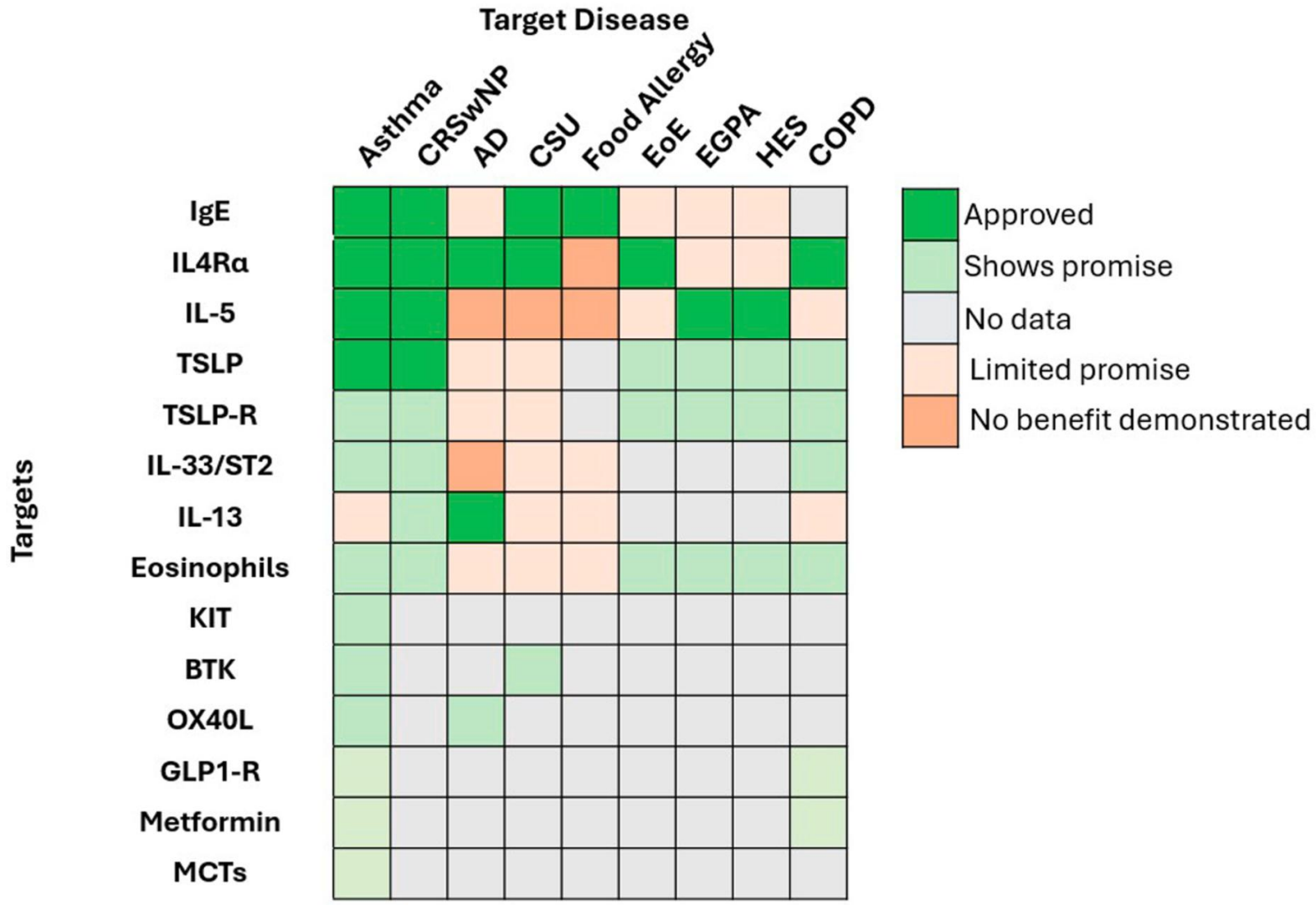
✓ Approved indication (+) Positive trial evidence – Not established

# Indication expansion and therapeutic repurposing for biologics




Tomasello A, et al. J Allergy Clin Immunol Pract 2026;14:325-335.

# Cross-disease mapping of therapeutic targets for treatment expansion




Tomasello A, et al. J Allergy Clin Immunol Pract 2026;14:325-335.

# Summary

- **Shared type 2 disease**: Asthma coexists with T2 comorbidities via a unified-airway, systemic mechanism
- **Higher burden**: Comorbidities drive greater severity, more exacerbations, and worse long-term outcomes
- **High-risk phenotypes**: Atopic march and CRSwNP mark the most severe, eosinophilic multimorbid endotype
- **Systematic work-up**: Screen, diagnose, and phenotyping of comorbidities in difficult-to-treat asthma
- **One target, multiple benefits**: T2-targeted biologics control several comorbidities at once, guided by the comorbidity profile

# Thank You for Your Attention