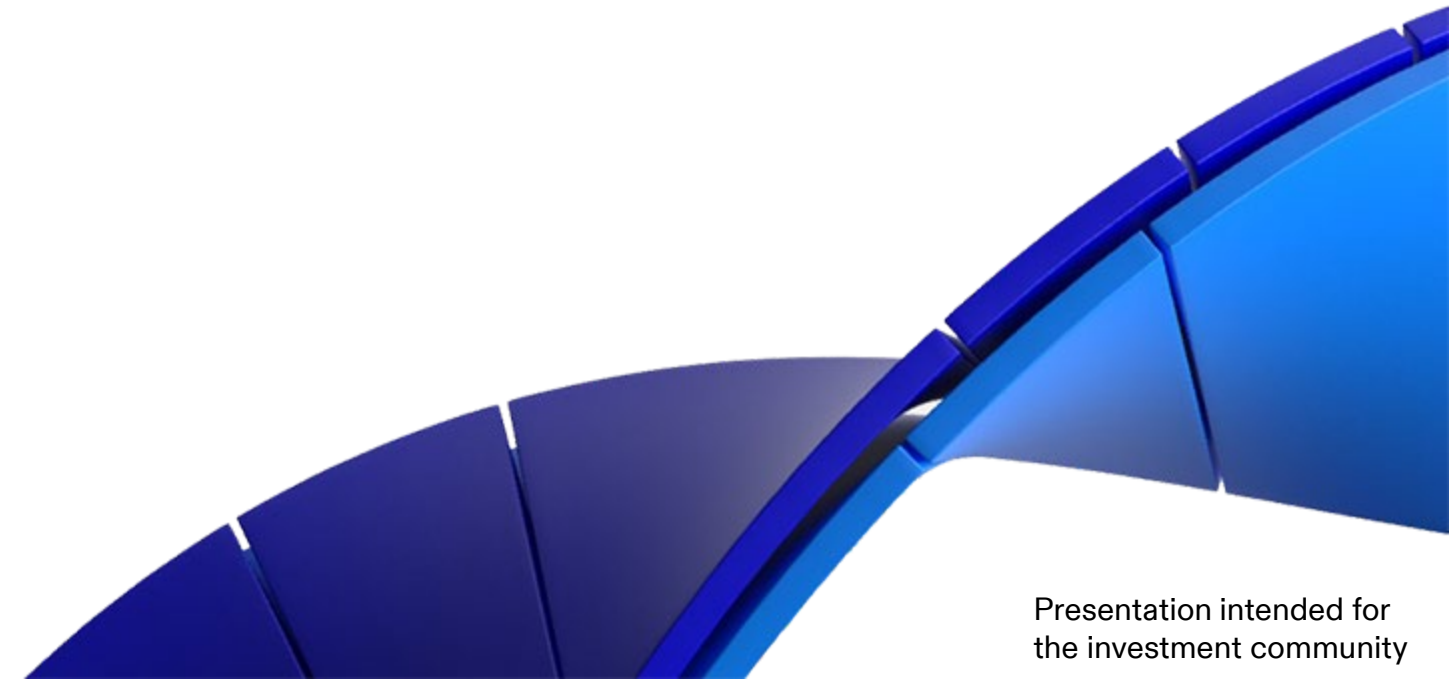
Pfizer Pflash: A Spotlight on the Trispecific Antibody Tilrekimig

November 12, 2026



Presentation intended for
the investment community

Forward-Looking Statements and Other Notices

Our discussions during this presentation will include forward-looking statements about, among other topics, Pfizer's investigational inflammation and immunology portfolio, including tilrekimig (PF-07275315), an investigational potential first-in-class trispecific antibody, and its potential benefits, the development program for tilrekimig, results from a Phase 2 trial for tilrekimig in patients with moderate-to-severe atopic dermatitis, ongoing Phase 3 trials of tilrekimig in atopic dermatitis and asthma and an ongoing Phase 2/3 study in COPD, the inflammation and immunology market, including anticipated market size, and Pfizer's ongoing commitment to the inflammation and immunology therapeutic area and efforts to address conditions with high unmet need and large patient populations, that involves substantial risks and uncertainties that could cause actual results to differ materially from those expressed or implied by such statements. Risks and uncertainties include, among other things, the uncertainties inherent in research and development, including the ability to meet anticipated clinical endpoints, commencement and/or completion dates for our clinical trials, regulatory submission dates, regulatory approval dates and/or launch dates, as well as the possibility of unfavorable new clinical data and further analyses of existing clinical data; the risks associated with initial, preliminary or interim data; the risk that clinical trial data are subject to differing interpretations and assessments by regulatory authorities; whether regulatory authorities will be satisfied with the design of and results from our clinical studies; whether and when applications for tilrekimig may be filed in particular jurisdictions for any potential indications; whether and when any such applications for tilrekimig that may be filed may be approved by regulatory authorities, which will depend on myriad factors, including making a determination as to whether the product's benefits outweigh its known risks and determination of the product's efficacy and, if approved, whether tilrekimig will be commercially successful; decisions by regulatory authorities impacting labeling, manufacturing processes, safety and/or other matters that could affect the availability or commercial potential of tilrekimig; risks and uncertainties related to issued or future executive orders or other new, or changes in, laws or regulations; uncertainties regarding the impact of COVID-19 on Pfizer's business, operations and financial results; and competitive developments. Such statements are forward-looking and are estimates that are subject to change and subject to, among other risks, assumptions and uncertainties, clinical trial, regulatory and commercial success, demand, availability of supply, excess inventory write-offs, product recalls, withdrawals, competitive and market dynamics, the regulatory and commercial landscape, and recent changes, and potential changes to economic, trade and foreign policy in the U.S. and globally, including, without limitation, tariffs, trade restrictions, retaliatory trade measures or other changes in laws, regulations or policy regarding trade, potential changes to U.S. federal or state legislation or regulatory action and/or policy efforts affecting, among other things, pharmaceutical product pricing, changes to healthcare policy in the U.S. and our ability to successfully integrate and scale our AI initiatives.

These statements may be affected by underlying assumptions that may prove inaccurate or incomplete, and are subject to risks, uncertainties and other factors that may cause actual results to differ materially from past results, future plans and projected future results. As forward-looking statements involve significant risks and uncertainties, caution should be exercised against placing undue reliance on such statements. Additional information regarding these and other factors can be found in Pfizer's Annual Report on Form 10-K for the fiscal year ended December 31, 2025 and its subsequent reports on Form 10-Q, including in the sections thereof captioned "Risk Factors" and "Forward-Looking Information and Factors That May Affect Future Results", as well as in Pfizer's subsequent reports on Form 8-K, all of which are filed with the U.S. Securities and Exchange Commission and available at http://www.sec.gov/ and http://www.pfizer.com/. Potential risks and uncertainties also include global economic and/or geopolitical instability, foreign exchange rate fluctuations and inflationary pressures and the uncertainties regarding the impact of COVID-19. The forward-looking statements in this presentation speak only as of the original date of this presentation and we undertake no obligation to update or revise any of these statements.

Today's discussions and presentation are intended for the investor community only; they are not intended to promote the products referenced herein or otherwise influence healthcare prescribing decisions. Definitive conclusions cannot be drawn from cross-trial comparisons or anticipated data as they may be confounded by various factors and should be interpreted with caution. All trademarks in this presentation are the property of their respective owners.

Certain of the products and product candidates that may be discussed during this conference call are being co-researched, co-developed and/or co-promoted in collaboration with other companies for which Pfizer's rights vary by market or are the subject of agreements pursuant to which Pfizer has commercialization rights in certain markets.

Speakers

Host



Francesca DeMartino

Chief Investor Relations Officer



Mike Vincent

Chief Inflammation & Immunology (I&I) Officer

Joining for Q&A



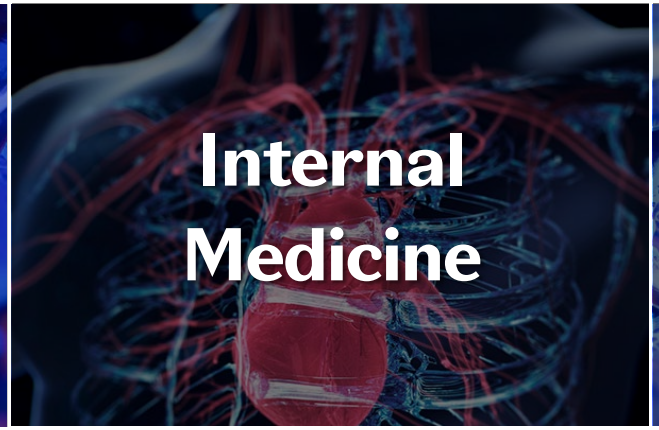
Christian Antoni

Chief Development Officer, I&I

R&D Strategy

To Discover and Develop the Next Wave of Breakthroughs That Change Patients' Lives









Supported by Expertise Across Three Core Modalities. Transforming with AI.

Small Molecules

Inhibitors, Activators, Degraders

Biologics

ADCs, Multispecifics, Peptides

Vaccines

Conjugate, Subunit and mRNA Platforms

End-to-End AI

Target Discovery to Real World Evidence

Pfizer

ADC: Antibody-drug conjugate

Diverse Inflammation & Immunology Portfolio

Select mid-to-late-stage inflammatory programs

Targeting Molecular Drivers of Dysfunction to Promote Restoration of the Immune System

Phase 2	Phase 3 / Registration[5]	Approved
LITFULO[1] CSU	**Dazukibart** DM and PM	CIBINQO™ (abrocitinib) tablets
LITFULO[1] Hidradenitis Suppurativa	**LITFULO**[1] Moderate Alopecia Areata	Enbrel® etanercept[6]
Ompekimig Atopic Dermatitis	**LITFULO**[1] Vitiligo	Litfulo® (ritlecitinib) capsules 50mg[1]
p40 x TL1A Bispecific[2] Ulcerative Colitis	**Tilrekimig** Atopic Dermatitis	Velsipity™ (etrasimod) tablets
STAT6 Inhibitor[3] Atopic Dermatitis	**Tilrekimig** Asthma	XELJANZ® [tofacitinib]
Tilrekimig COPD[4]		

● Small Molecule ● Biologic

1. LITFULO is approved for severe alopecia areata. The LITFULO development program spans multiple conditions. **2.** Pfizer and Roche have a global collaboration for p40 x TL1A bispecific antibody (PF-07261271); **3.** PF-08049820; **4.** Currently in a Phase 2/3 trial; **5.** Programs shown are in Phase 3 development, except LITFULO in vitiligo, which is in registration phase; **6.** Pfizer markets ENBREL outside the U.S. and Canada; **CSU:** Chronic spontaneous urticaria; **COPD:** Chronic obstructive pulmonary disease; **DM:** Dermatomyositis; **PM:** Polymyositis

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Tilrekimig Phase 2 Atopic Dermatitis (AD) Data: The Key Takeaways[1]

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Potentially superior to dupilumab[2†] with ongoing Phase 3 trial evaluating head-to-head

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Lower frequency of conjunctivitis and injection site reactions in Stage 2* compared to historical rates with dupilumab[3†]

Pipeline-in-a-Product Opportunity

Strong Phase 2 data in AD support development in other type 2 inflammatory disorders

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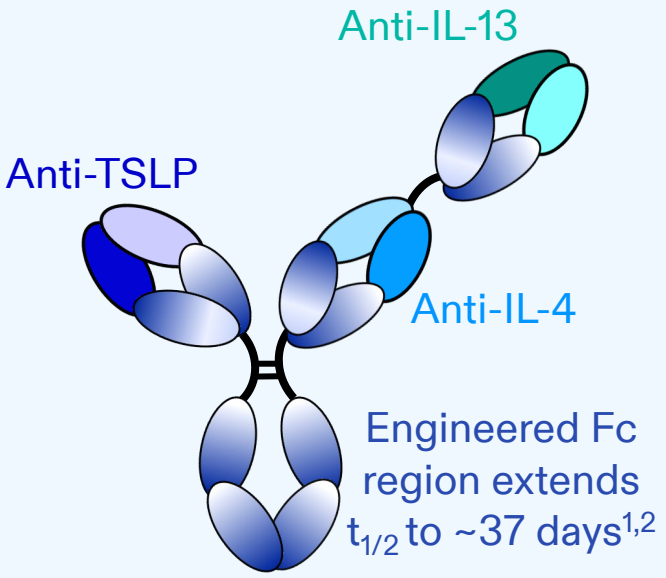
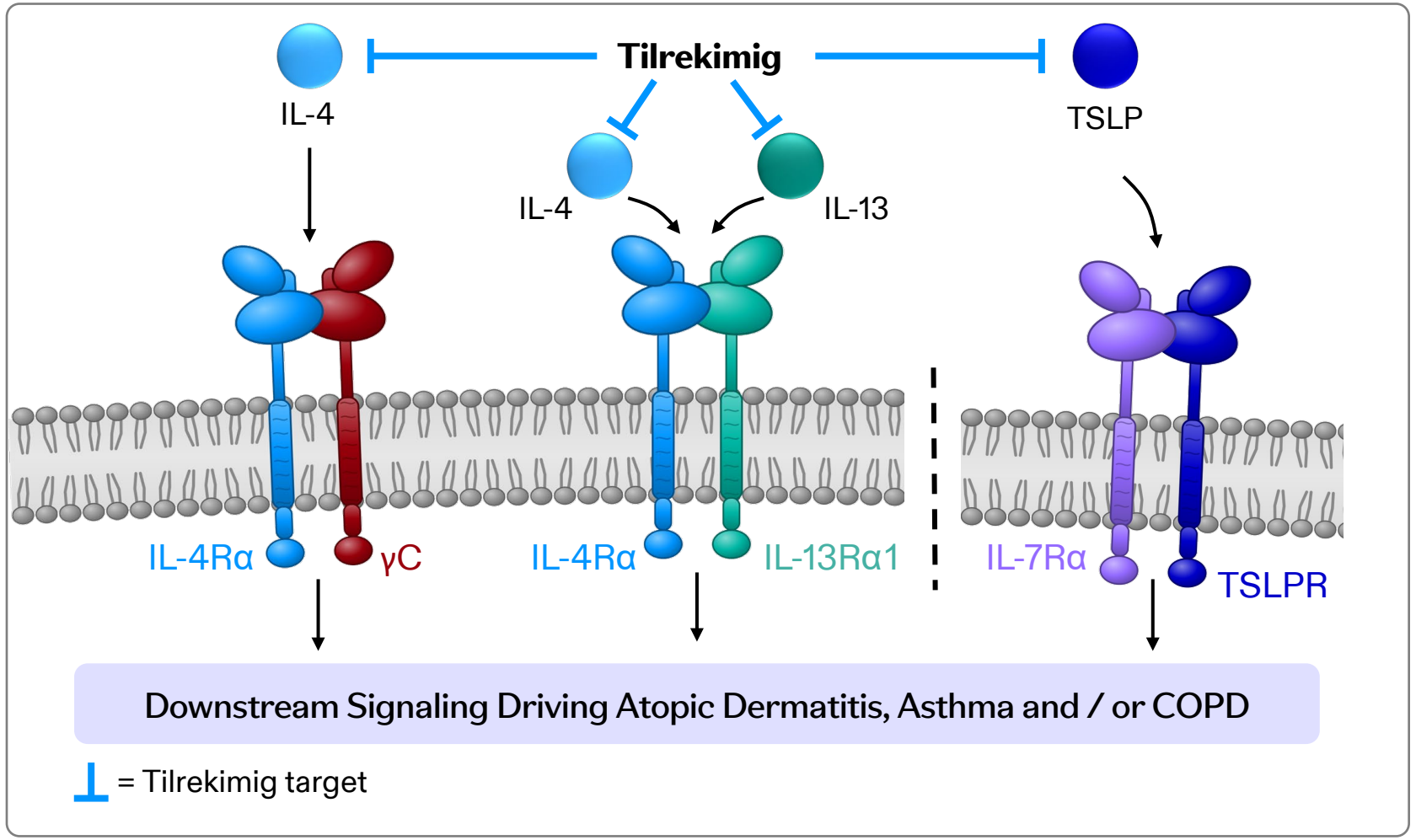
Tilrekimig is Directed Against Validated & Complementary Targets

First-in-class approach directly targets IL-4, IL-13, and TSLP ligands for potentially additive effects



Tilrekimig

Internally discovered trispecific exhibits potent binding to all three targets without interference[2]



1. Bloom L. Festival of Biologics. 2024; **2.** Data on file; **COPD:** Chronic obstructive pulmonary disease; **IL-4:** Interleukin-4; **IL-4Rα:** IL-4 receptor alpha; **IL-7Rα:** IL-7 receptor alpha; **IL-13:** Interleukin-13; **IL-13Rα1:** IL-13 receptor alpha-1; **$t_{1/2}$:** Half-life; **TSLP:** Thymic stromal lymphopoietin; **TSLPR:** TSLP receptor; Schematics represent generalized representations.

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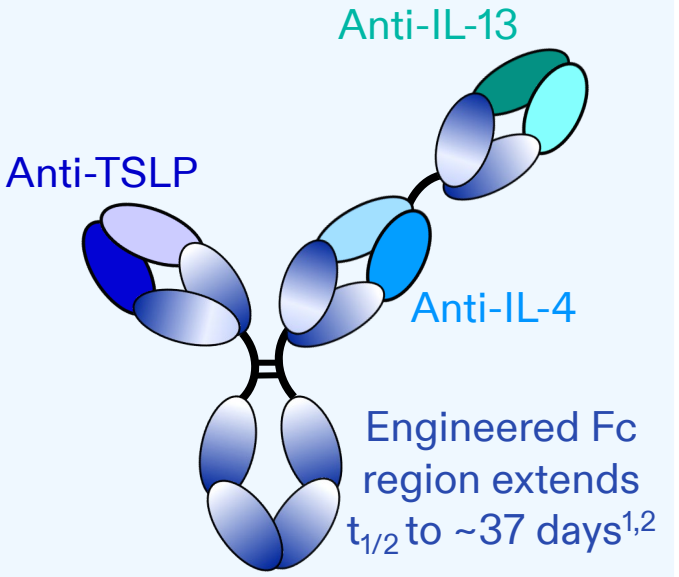

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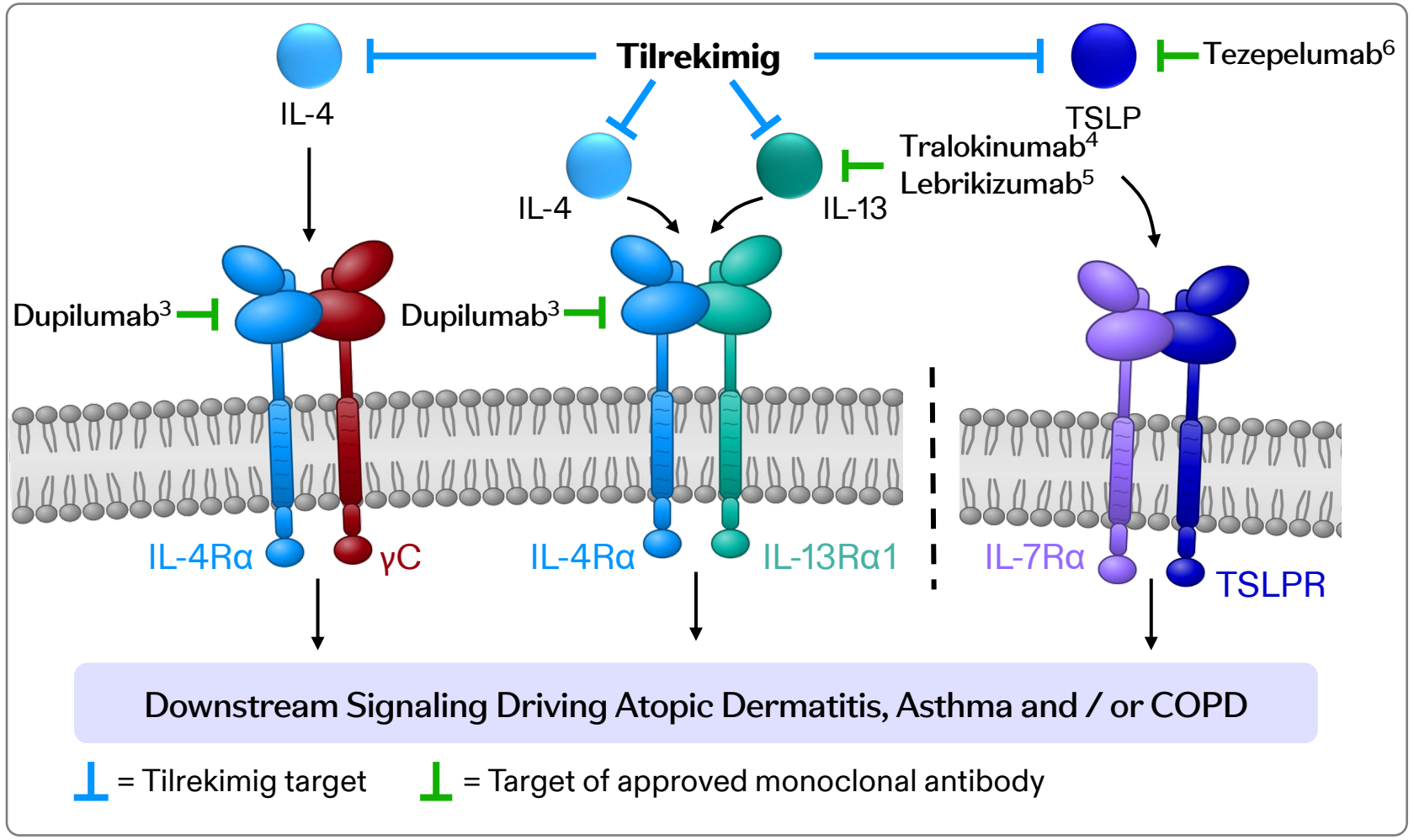


1. Bloom L. Festival of Biologics. 2024; **2.** Data on file; **3.** Dupilumab is a human monoclonal IgG4 antibody that inhibits IL-4 and IL-13 signaling by specifically binding to the IL-4Rα subunit shared by the IL-4 and IL-13 receptor complexes; **4.** Tralokinumab-ldrm: a human IgG4 monoclonal antibody that specifically binds to IL-13 and inhibits its interaction with the IL-13 receptor α1 and α2 subunits (IL-13Rα2 subunit not shown); **5.** Lebrikizumab-lbkz: an IgG4 monoclonal antibody that binds with high affinity and slow off-rate to IL-13 and allows IL-13 to bind to IL-13Rα1 but inhibits human IL-13 signaling through the IL-4Rα/IL-13Rα1 receptor complex; **6.** Tezepelumab-ekko: a TSLP blocker, human monoclonal antibody IgG2λ that binds to human TSLP and blocks its interaction with the heterodimeric TSLP receptor; **COPD:** Chronic obstructive pulmonary disease; **IL-4:** Interleukin-4; **IL-4Rα:** IL-4 receptor alpha; **IL-7Rα:** IL-7 receptor alpha; **IL-13:** Interleukin-13; **IL-13Rα1:** IL-13 receptor alpha-1; **IL-13Rα2:** IL-13 receptor alpha-2; **$t_{1/2}$:** Half-life; **TSLP:** Thymic stromal lymphopoietin; **TSLPR:** TSLP receptor; Schematics represent generalized representations.

Atopic Dermatitis is the Leading Contributor to Skin Disease Burden[1-6]

Moderate-to-severe atopic dermatitis affects ~9.8M people in the U.S.[7]



Photos enhanced to illustrate moderate-to-severe atopic dermatitis

Atopic dermatitis is a chronic, relapsing inflammatory skin disease **with substantial symptomatic, psychosocial and economic burden**

Many Patients with Moderate-to-Severe Atopic Dermatitis Receiving Advanced Systemic Therapies Do Not Achieve Treatment Targets[8]

Skin Clearance

82%
Missed **optimal** treatment target

37%
Missed **moderate** treatment target

Itch

79%
Missed **optimal** treatment target

67%
Missed **moderate** treatment target

No Biologic has Demonstrated Superiority vs. the Current SOC Biologic, Dupilumab

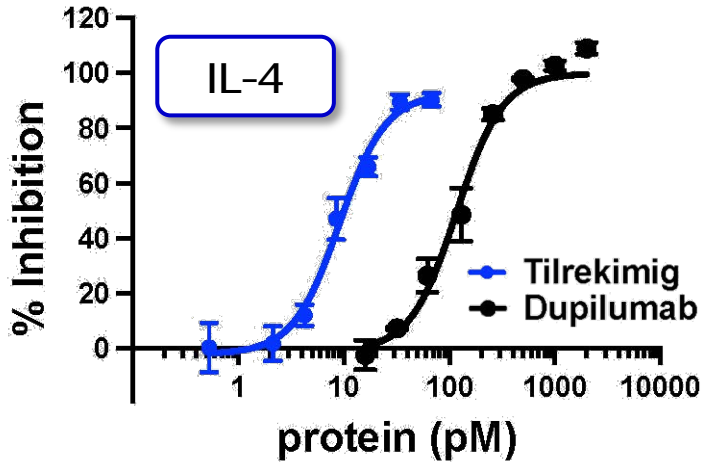
Pfizer

1. As measured by disability-adjusted life years; **2.** Tian et al. Br J Dermatol. 2023 Dec 20;190(1):55-61; **3.** Laughter et al. Br J Dermatol. 2021 Feb;184(2):304-309; **4.** Langan et al. Lancet. 2020 Aug 1;396(10247):345-360; **5.** Hay et al. J Invest Dermatol. 2014 Jun;134(6):1527-1534; **6.** Ständer, N Engl J Med. 2021 Mar 25;384(12):1136-1143; **7.** National Eczema Association (accessed November 9, 2026); **8.** At 6 months, as reported in Eichenfield et al. SKIN J Cutan Med. 2024;8(6):s462; **SOC:** Standard of care; **AD:** Atopic dermatitis

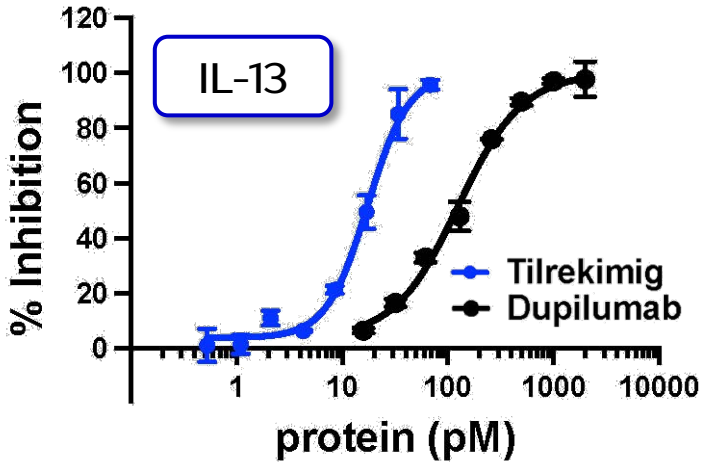
Tilrekimig Shows Improved *In Vitro* Functional Potency vs. Dupilumab, Supporting its Potential to Deliver Enhanced Efficacy

Inhibition of IL-4, IL-13 or TSLP-Stimulated Signaling in a Monocyte CD23 Expression Assay[1*]

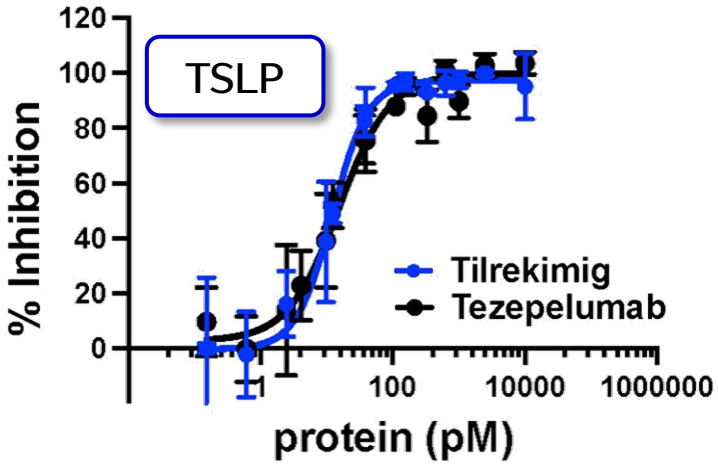
~13x Higher Functional Potency vs. IL-4 with Tilrekimig vs. Dupilumab



~7x Higher Functional Potency vs. IL-13 with Tilrekimig vs. Dupilumab



Comparable Functional Potency vs. TSLP with Tilrekimig vs. Tezepelumab



*Not intended to compare efficacy or safety across these molecules

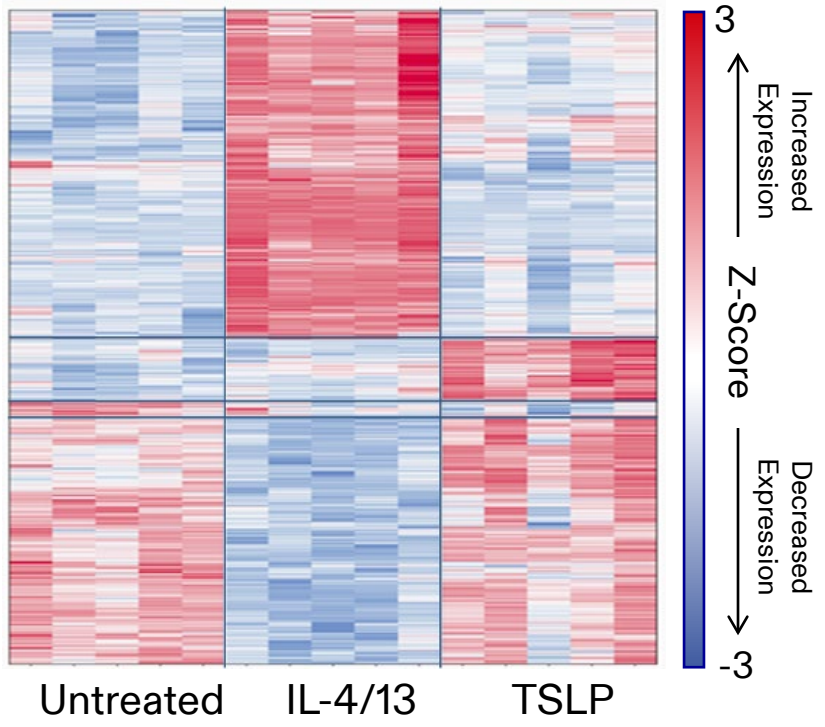
Tilrekimig's Improved Functional Potency for IL-4 and IL-13, Together with its Additional Mechanism of Action Targeting TSLP, May Drive Enhanced Efficacy vs. Dupilumab (Current SOC Biologic)

1. Data on file; **IL-4:** Interleukin-4; **IL-13:** Interleukin-13; **TSLP:** Thymic stromal lymphopoietin; **SOC:** Standard of care

Distinct Inflammatory Pathways Driven by IL-4/13 and TSLP Suggest Combined Blockade May Drive Additive Efficacy in Atopic Dermatitis

Human Skin Explant Data: IL-4/13 and TSLP Drive Both Shared and Distinct Inflammatory Pathways

Heatmap of Gene Expression Changes with IL-4/13 and TSLP Stimulation[1]



IL-4/13 Induced >2-fold Expression Changes for 910 Genes

TSLP Induced >2-fold Expression Changes for 179 Genes

Only 44 Genes with >2-fold Expression Changes in Response to Both IL-4/13 and TSLP

1. Zhang and Gieseck III, European Academy of Dermatology and Venereology Congress 2026 (Poster: P0145); **IL-4:** Interleukin-4, **IL-13:** Interleukin-13, **TSLP:** Thymic stromal lymphopoietin

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Phase 2 Trial of Tilrekimig in Atopic Dermatitis[1]

Stage 1

Tilrekimig 450 mg Q2W[2] in biologic-naïve participants[3]

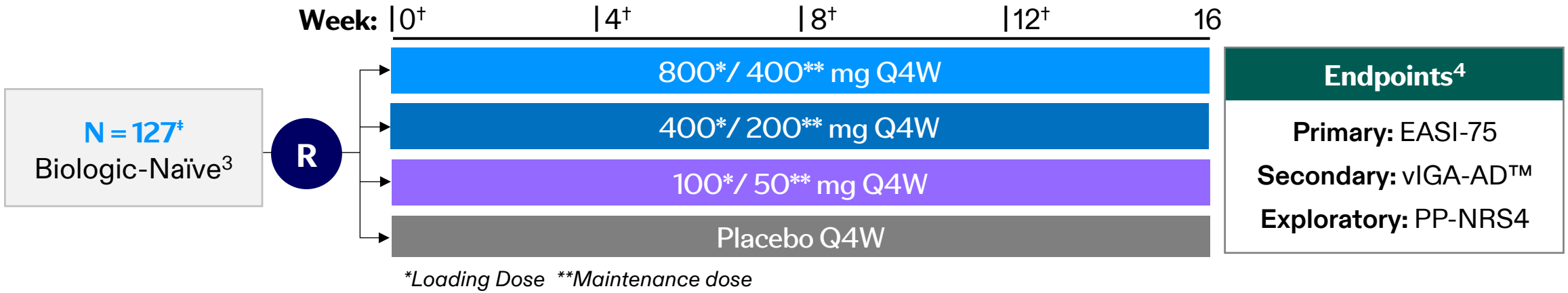
Stage 2

Tilrekimig **dose ranging** in biologic-naïve participants[3]

Stage 3

Tilrekimig in **biologic-experienced** population[3]

Today's Focus is Stage 2: Dose-Ranging Study with 16-Week Treatment Period



Endpoints[4]

Primary: EASI-75

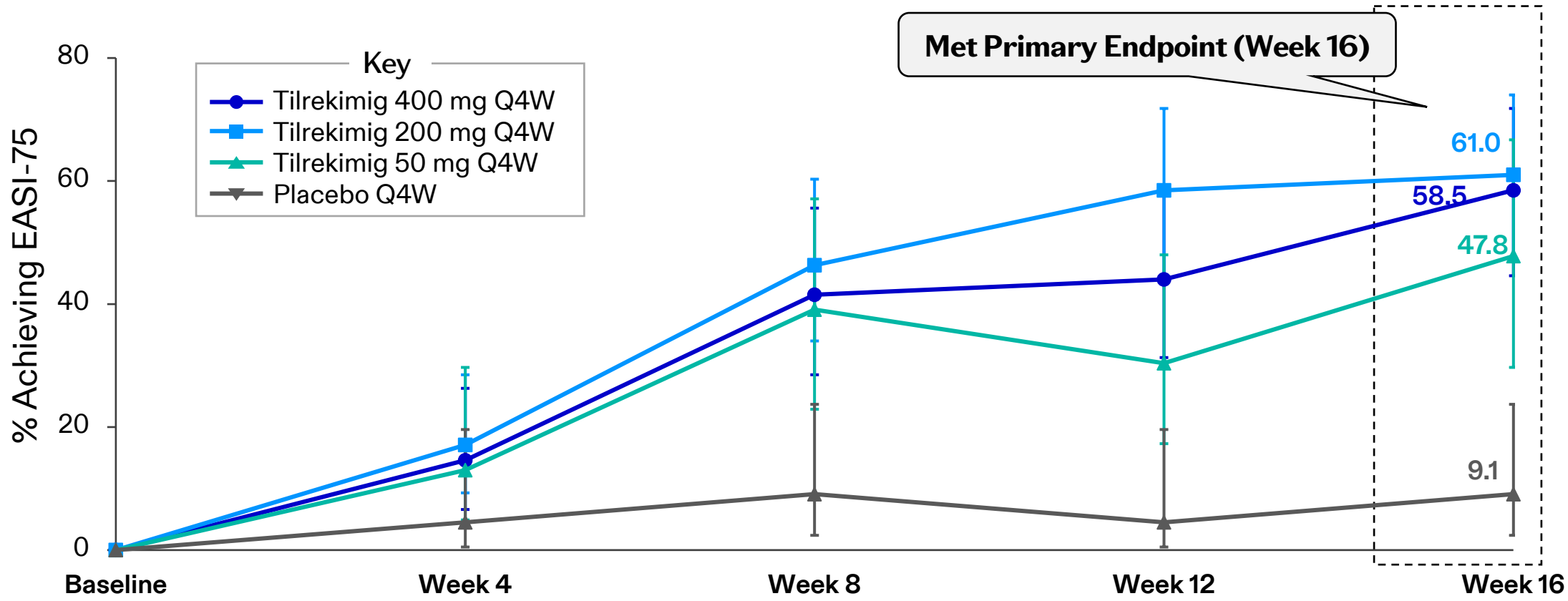
Secondary: vIGA-AD™

Exploratory: PP-NRS4

†Denotes time of administration of tilrekimig or placebo; ‡Denotes number of participants in the Stage 2 modified full analysis set (mFAS2), which includes all patients randomly assigned to study intervention who took ≥1 dose of study intervention meeting moderate to severe AD eligibility and excluded patients who were discontinued from the treatment and dosed at Day 1. For more information see Simpson et al. European Academy of Dermatology and Venereology Congress 2026 (AS-1730); **1.** Schema shows select tilrekimig arms only and represents a subset of overall trial design; **2.** Follows a 1200 mg loading dose; **3.** Biologic-naïve and biologic–experienced refer to patient history of treatment with a biologic agent, e.g., dupilumab; **4.** Not exhaustive; **EASI-75:** ≥75% improvement from baseline in Eczema Area and Severity Index; **AD:** Atopic dermatitis; **Q2W:** Every two weeks; **Q4W:** Every four weeks; **R:** Randomization; **vIGA-AD™:** Validated Investigator Global Assessment for Atopic Dermatitis. Endpoint measures the proportion of participants achieving a score of (0/1) (Clear/Almost Clear skin) with ≥2-point improvement from baseline; **PP-NRS4:** ≥4 points of reduction from baseline in weekly averages of Peak Pruritus Numerical Rating Scale

Tilrekimig Demonstrated Robust Efficacy on Primary Endpoint in Ph 2

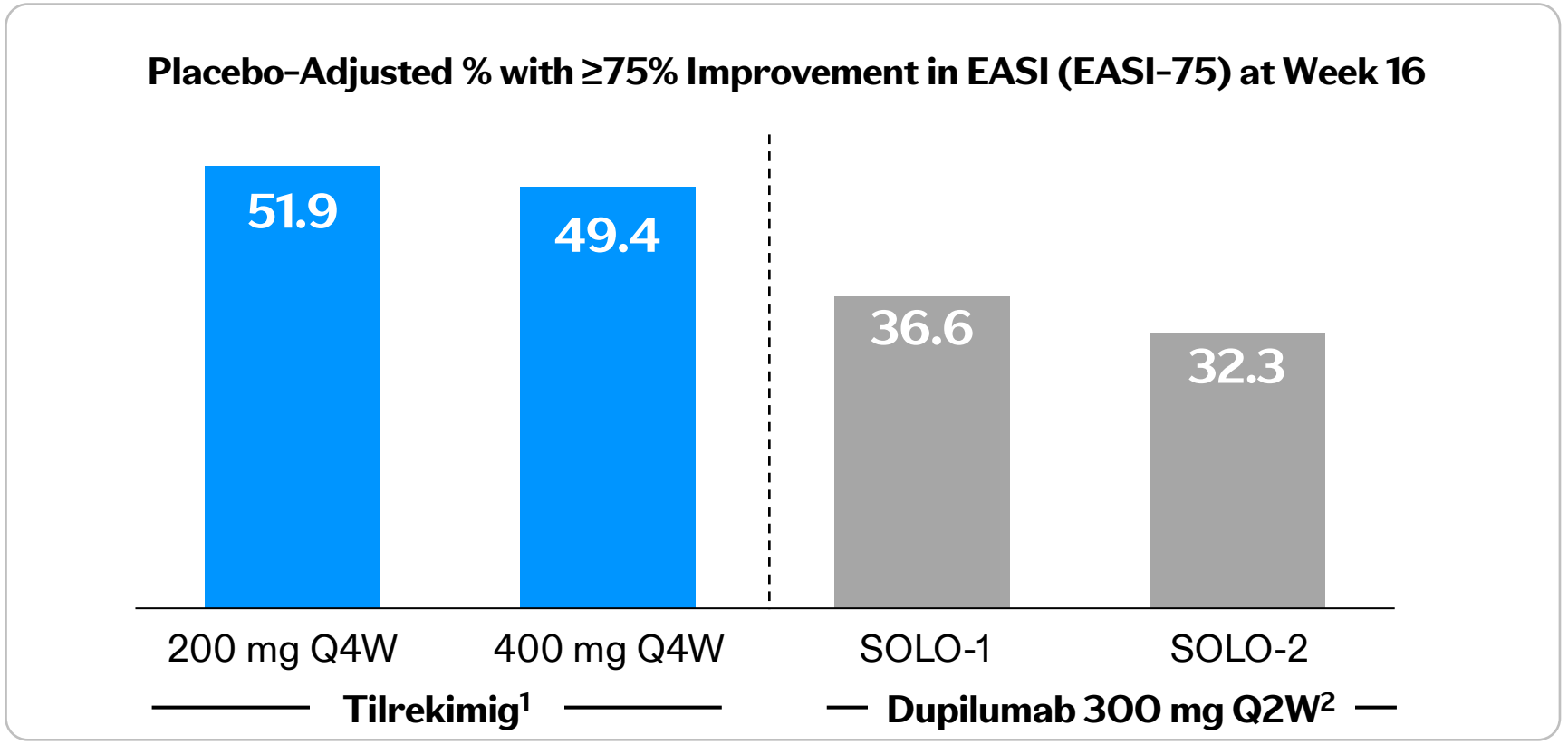
Percentage of Tilrekimig Participants Achieving a ≥75% Improvement in Atopic Dermatitis Disease Score (EASI-75) Increased Over Time with Significant Improvements for All Doses vs. Placebo at Week 16*



*For more information see Simpson et al. European Academy of Dermatology and Venereology Congress 2026 (AS-1730). Error bars represent 90% confidence intervals; **EASI-75:** At least a 75% improvement from baseline in Eczema Area and Severity Index score; **Ph:** Phase; **Q4W:** Every four weeks. Data from Stage 2 of Phase 2 trial.

Phase 2 Primary Endpoint Data Support Potential to Demonstrate Superior Efficacy vs. Dupilumab in Ongoing Phase 3 Trial

Cross-Trial Comparison: Tilrekimig Phase 2 Trial[1] vs. Pivotal Trials of Dupilumab (SOLO-1 and SOLO-2)[2]



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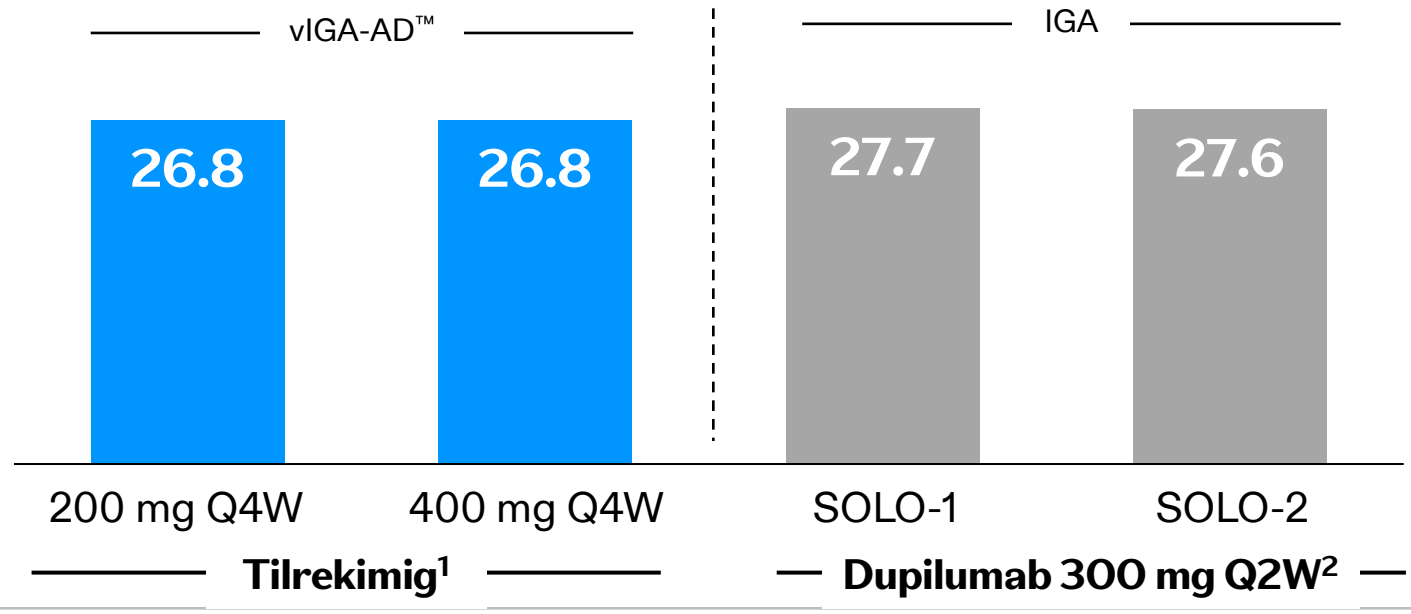
Tilrekimig Skin Clearance Similar to SOC Benchmarks at Week 16

Anticipate tilrekimig skin clearance scores to improve and potentially differentiate from SOC with continued dosing

Cross-Trial Comparison: Tilrekimig Phase 2 Trial[1] vs. Pivotal Trials of Dupilumab (SOLO-1 and SOLO-2)[2]

Placebo-Adjusted % with IGA 0/1 and ≥2-point Improvement from Baseline at Week 16



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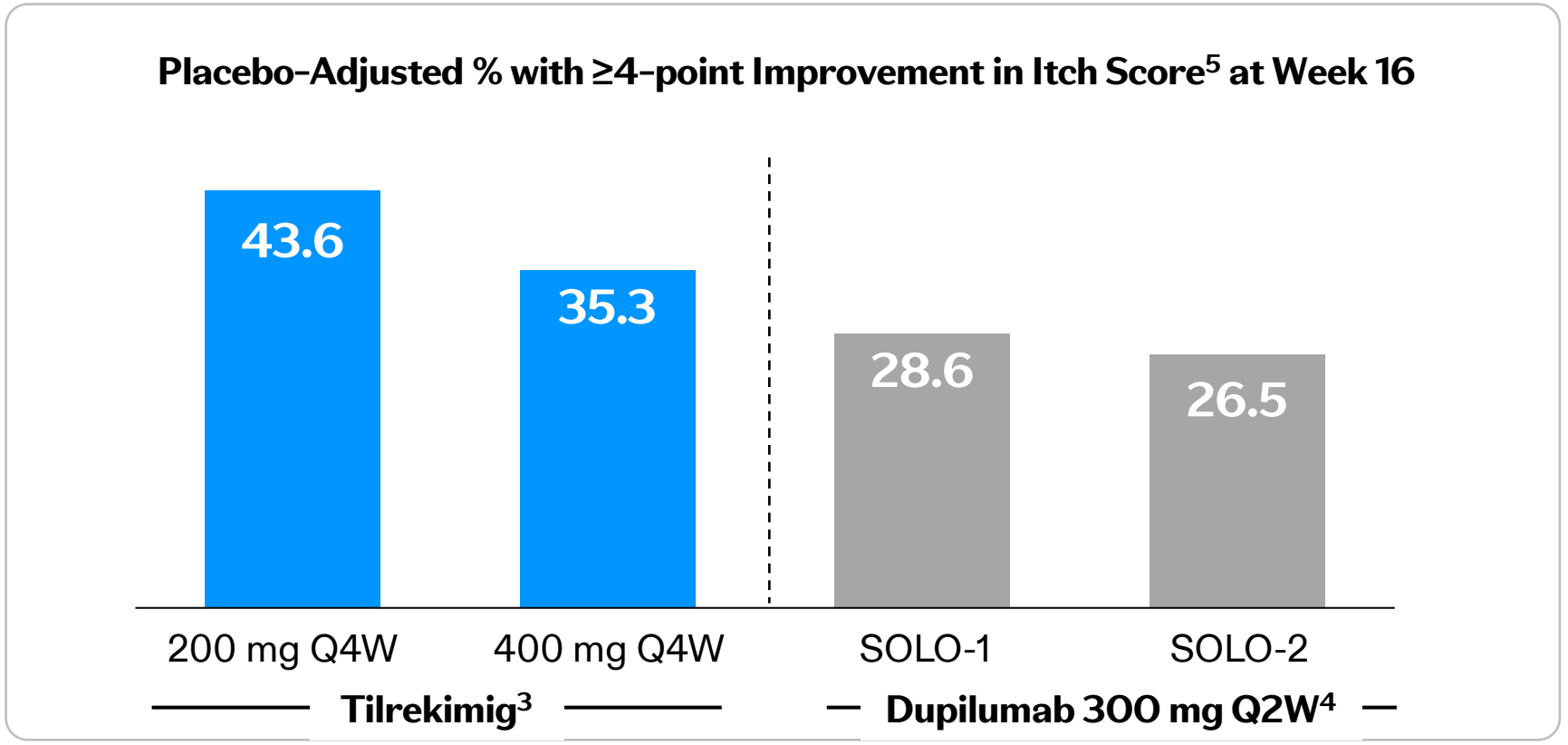
Tilrekimig: Improved Itch Relief Relative to SOC Benchmark at Week 16

Itch is the most prevalent and burdensome symptom of atopic dermatitis and the main reason for HCP visits[1,2]

Cross-Trial Comparison: Tilrekimig Phase 2 Trial[3] vs. Pivotal Trials of Dupilumab (SOLO-1 and SOLO-2)[4]



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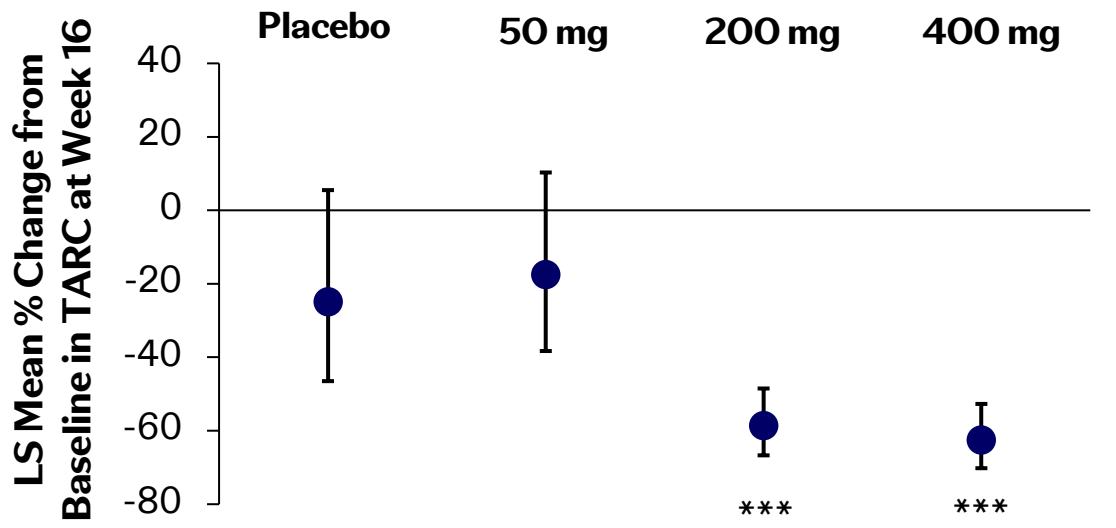
1. Silverberg et al. Dermatitis. 2023 Mar-Apr;34(2):135-144; **2.** Begolka et al. Dermatol Clin. 2024 Oct;42(4):537-548; **3.** For more information see Simpson et al. European Academy of Dermatology and Venereology Congress 2026 (AS-1730). Data are from Stage 2 of the trial; **4.** Difference between dupilumab 300 mg Q2W and placebo values from SOLO-1 and -2 trials reported in Simpson et al. N Engl J Med. 2016 Dec 15;375(24):2335-2348; **5.** The proportion of patients with an improvement of at least 4 points at week 16 in the weekly average of peak scores for pruritus was a key secondary endpoint in SOLO-1 and SOLO-2. The proportion with ≥4 points of improvement from baseline in weekly averages of Peak Pruritus Numerical Rating Scale (PP-NRS4) over time was an exploratory endpoint for tilrekimig; **Q4W:** Every four weeks; **Q2W:** Every two weeks; **HCP**: Health care provider; **SOC**: Standard of care; Doses listed in figure represent maintenance doses, which followed loading doses

Biomarker Data Support Tilrekimig's Potential Efficacy & Differentiation

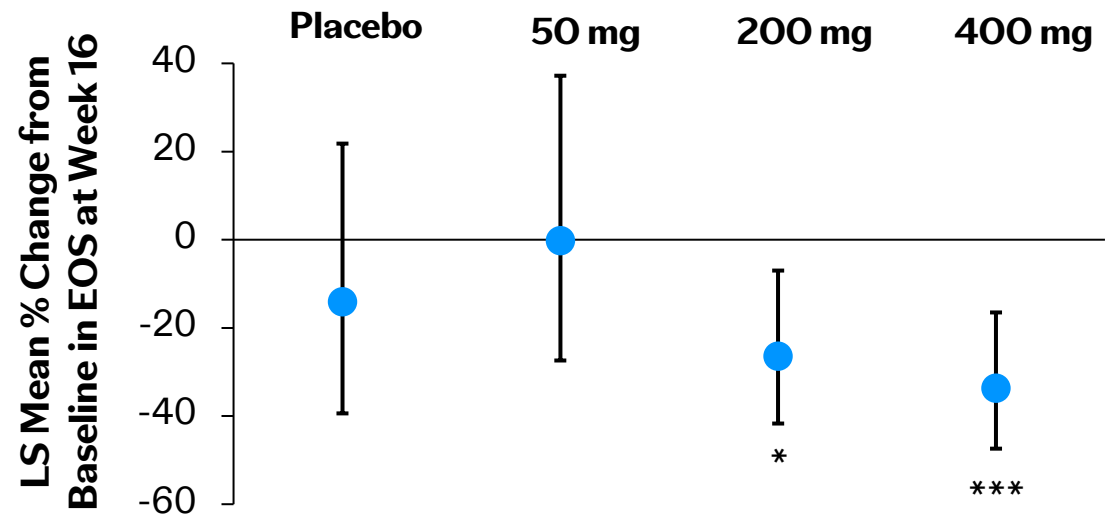
Observed significant reductions across key type 2 inflammatory markers, consistent with tilrekimig's MOA

Significant Reductions in TARC at Week 16 in 200 mg and 400 mg Dose Groups in Phase 2[1]



TARC is a well-established biomarker of atopic dermatitis disease activity[2].

Significant Reductions in Blood Eosinophils at Week 16 in 200 mg and 400 mg Dose Groups in Phase 2[1]



Eosinophils are effector cells in type 2 disease. Lowering eosinophils can differentiate tilrekimig vs. dupilumab[3].

*p<0.05; ***p<0.001; **1.** Guttman-Yassky et al. European Academy of Dermatology and Venereology Congress 2026 (P1659). Data are from Stage 2 of the trial. Error bars represent 95% confidence intervals. Statistical significance reflects within-treatment change from baseline; **2.** Kataoka, Front Allergy. Jan 23, 2025;5:1473902; **3.** Simpson et al. N Engl J Med. 2016 Dec 15;375(24):2335-2348; **EOS:** Eosinophils; **LS:** Least squares; **MOA:** Mechanism of action; **TARC:** Thymus and activation-regulated chemokine

Tilrekimig Phase 2 Atopic Dermatitis (AD) Data: The Key Takeaways[1]

Robust Efficacy

Potentially superior to dupilumab[2†] with ongoing Phase 3 trial evaluating head-to-head

Favorable Safety Profile

Lower frequency of conjunctivitis and injection site reactions in Stage 2* compared to historical rates with dupilumab[3†]

Pipeline-in-a-Product Opportunity

Strong Phase 2 data in AD support development in other type 2 inflammatory disorders

Tilrekimig is a Potential First-in-Class Trispecific Antibody Targeting Validated Pathways that Drive Type 2 Inflammatory Disorders Such as Atopic Dermatitis, Asthma and COPD

†Based on a cross-trial comparison, **not** data from a head-to-head study. Definitive conclusions cannot be drawn from results across different clinical trials.

1. Simpson et al. European Academy of Dermatology and Venereology Congress 2026 (AS-1730); **2.** Simpson et al. N Engl J Med. 2016 Dec 15;375(24):2335-2348; **3.** DUPIXENT prescribing information; *Stage 2 evaluated maintenance doses up to 400 mg every four weeks; **COPD:** Chronic obstructive pulmonary disease; **AD**: Atopic dermatitis; Third party trademarks are the property of their respective owners and any references are for identification purposes only.

Tilrekimig was Well-tolerated with a Favorable Safety Profile in Phase 2*

No dose-dependent safety signals or serious adverse events related to tilrekimig[1]

Comparable TEAE rates
between tilrekimig and placebo groups

Numerically lower injection site reaction
AEs for tilrekimig 400 mg Q4W vs. placebo Q4W

No eosinophilia
AEs in tilrekimig or placebo groups

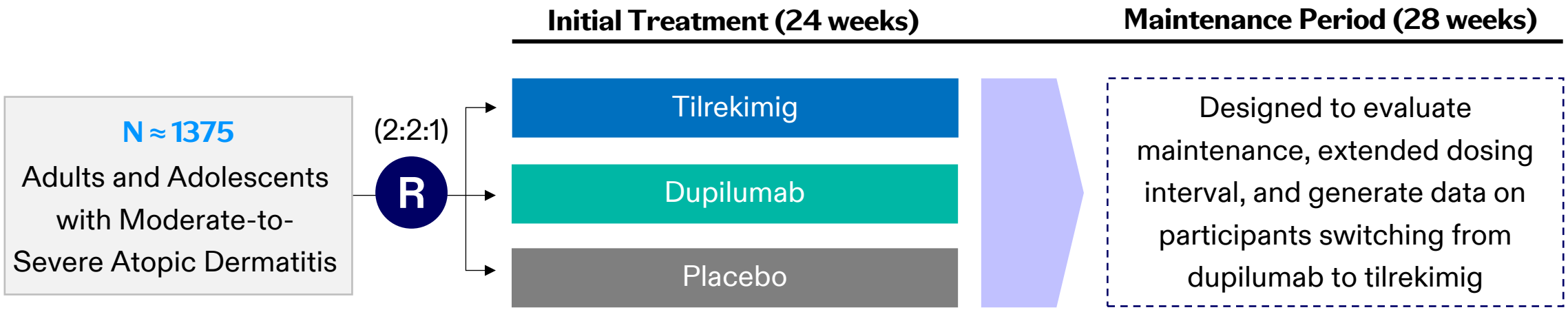
Similar rates of conjunctivitis
AEs between tilrekimig and placebo groups

Potential for Placebo-Like Conjunctivitis and Injection Site Reaction Rates Highlights Additional Opportunity for Potential Differentiation vs. Dupilumab

*Simpson et al. European Academy of Dermatology and Venereology Congress 2026 (AS-1730). Findings from Stages 1 and 2 of trial, treatment-emergent adverse events reported up to Week 16 or Day 128; **1.** Two Stage 2 patients had treatment-emergent serious adverse events, unrelated to treatment; **AE:** Adverse event; **Q4W:** Every four weeks; **TEAE:** Treatment-emergent adverse event; Doses listed above represent maintenance doses, which followed loading doses

Two Phase 3 Trials of Tilrekimig in Atopic Dermatitis Ongoing

Tilrekimig vs. Placebo and vs. Dupilumab[1]



Co-primary Endpoints: Tilrekimig vs. Placebo - EASI-75 and vIGA-AD™ Response at **Week 16**

Select Key Secondary Endpoints: Tilrekimig vs. Dupilumab - EASI-75, PP-NRS4, and vIGA-AD™ Response

Additional Phase 3 Trial: Tilrekimig vs. Placebo in Atopic Dermatitis[2]
Designed To Evaluate Tilrekimig on Background Topical Medication (N ≈ 375)

1. For more information see clinicaltrials.gov: NCT07783425; **2.** For more information see clinicaltrials.gov: NCT07765888; **AD:** Atopic dermatitis; **EASI-75:** At least a 75% improvement from baseline in Eczema Area and Severity Index score; **PP-NRS4:** ≥4 points of reduction from baseline in weekly averages of Peak Pruritus Numerical Rating Scale; **R:** Randomization; **vIGA-AD™:** Validated Investigator Global Assessment for Atopic Dermatitis

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Tilrekimig Embodies Go-Forward I&I Research & Development Strategy

Enabled by capabilities and expertise in small molecules, multispecific antibodies and systems immunology



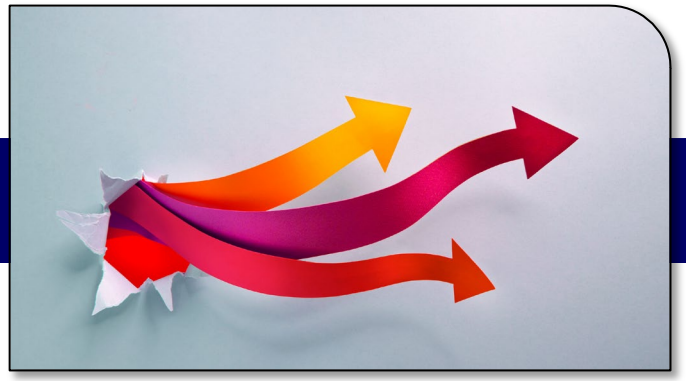
Identify Biological Axis

That drives multiple diseases with substantial unmet need



Demonstrate Clinical POC

In high-value anchor indication(s)



Strategically Expand

To additional indications driven by biological axis

I&I: Inflammation and Immunology; **POC:** Proof-of-concept

IL-4, IL-13 and TSLP are Key Drivers of Airway Inflammation

TSLP
Upstream epithelial alarmin

Released by the airway lining in response to environmental triggers[1]

Activates multiple immune cells that initiate type 2 inflammation[1]

Contributes to non-type 2 inflammation[2]

IL-4
Programs the type 2 immune response

Helps establish and amplify type 2 inflammation[3]

Promotes IgE production[4,5]

Promotes recruitment of eosinophils to airways[4,5]

IL-13
Drives effects within the airway

Drives mucus production[5,6]

Promotes tissue remodeling[5]

Makes airways more sensitive and prone to constriction[6,7]

1. Parnes et al. J Asthma Allergy. 2022 Jun 3;15:749–765; **2.** Calderon et al. Eur Respir Rev. 2023 Jan 25;32(167):220144; **3.** Hammad & Lambrecht. Cell. 2021 Mar 18;184(6):1469-1485; **4.** Maspero et al. ERJ Open Res. 2022 Aug 1;8(3):00576-2021; **5.** Sahnoon et al. Clin Rev Allergy Immunol. 2025 Apr 21;68(1):44; **6.** Rael & Lockey. World Allergy Organ J. 2011 Mar 15;4(3):54–64; **7.** Risse et al. Am J Physiol Lung Cell Mol Physiol. 2011 Jun;300(6):L958-66

IL-4/13 & TSLP Pathways are Validated Targets in Airway Diseases

Approved antibodies target IL-4/13 & TSLP pathways in airway diseases, yet substantial unmet need persists[1-3]

Asthma

IL-4/13 Signaling is a Validated Target in Type 2-High Asthma[4]

TSLP is a Validated Target in Asthma Across Blood Eosinophil Levels[5]

~3M People Ages 12+ in the U.S. Diagnosed with Severe Asthma[6]

COPD

IL-4/13 Signaling is a Validated Target in COPD with Blood Eosinophils ≥300 cells/μL[7]

Emerging Validation of Potential Role for TSLP in COPD[8]

~2.5M Adults in the U.S. Diagnosed with COPD that Remain Uncontrolled[6]

Pfizer

1. Approved antibodies targeting IL-4/13 and TSLP signaling in airway disease include dupilumab and tezepelumab; **2.** Gates et al. Lancet Respir Med. 2026 Oct;14(10):e58-e59; **3.** García Morales et al. Respir Med. 2026 May;256:108815; **4.** Castro et al. N Engl J Med. 2018 Jun 28;378(26):2486-2496; **5.** Menzies-Gow et al. N Engl J Med. 2021 May 13;384(19):1800-1809; **6.** Estimated patient numbers (2026) - Pfizer Analysis; **7.** Bhatt et al. N Engl J Med. 2023 Jul 20;389(3):205-214; **8.** Singh et al. Lancet Respir Med. 2025 Jan;13(1):47-58; **COPD:** Chronic obstructive pulmonary disease

Tilrekimig's Potential First Wave: Atopic Dermatitis, Asthma and COPD*

	Select Late-Stage Trials*	Differentiation Goal in Phase 3
Phase 3 Atopic Dermatitis	**Tilrekimig vs. Placebo and vs. Dupilumab**[1]	**Aim to Differentiate with Superior Efficacy vs. Dupilumab**
	Tilrekimig + Topical Medicine vs. Placebo + Topical Medicine[2]	
Phase 3 Asthma	**Tilrekimig vs. Placebo**[3] *Enrolls across blood eosinophil counts*	**Aim to Differentiate with Efficacy in a Broad Range of Blood Eosinophil Counts**
	Tilrekimig vs. Placebo[4] *Enrolls across blood eosinophil counts*	
Phase 2/3 COPD	**Tilrekimig vs. Placebo**[5] *Enrolls blood eosinophil counts ≥150 cells/µL*	

Aim to Differentiate Tilrekimig from Dupilumab Across Atopic Dermatitis, Asthma and COPD

*Key trials shown (not exhaustive); **1.** NCT07783425; **2.** NCT07765888; **3.** NCT07772921; **4.** NCT07869043; **5.** NCT07363694; **COPD:** Chronic obstructive pulmonary disease

Ph 2 Data Support Tilrekimig's Potential to be a First-in-Class Trispecific and Leading Biologic Therapy for Type 2 Inflammatory Disorders

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Ongoing Late-Stage Studies of Tilrekimig in Atopic Dermatitis, Asthma, and COPD, Diseases Collectively Projected to Represent a >$55B Global Market in 2030[4]

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Pfizer

1. Demonstrated in Phase 2. For more information see Simpson et al. European Academy of Dermatology and Venereology Congress 2026 (AS-1730); **2.** Simpson et al. N Engl J Med. 2016 Dec 15;375(24):2335-2348; **3.** DUPIXENT prescribing information; **4.** 2030 estimates. Source: *Evaluate Pharma*; *Stage 2 evaluated maintenance doses up to 400 mg every four weeks; **COPD:** Chronic obstructive pulmonary disease; **AD**: Atopic dermatitis; **Ph:** Phase; Third party trademarks are the property of their respective owners and any references are for identification purposes only.

Q&A

Host



Francesca DeMartino

Chief Investor Relations Officer



Mike Vincent

Chief Inflammation & Immunology (I&I) Officer



Christian Antoni

Chief Development Officer, I&I