



Corporate Presentation

May 2026

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Evommune (EVMN) is Addressing Chronic Inflammation, a Global Healthcare Crisis



Chronic Inflammation Destroys Lives

- Contributes to 3 out of 5 deaths worldwide¹



Substantial Burden on the Healthcare System

- Annual direct cost of at least \$90B²



Existing Treatment Options Have Critical Limitations

- Current therapies fail to deliver efficacy and safety suitable for the majority of patients

Evommune is Delivering Next Generation Therapies



Experienced Team



Distinct Mechanisms



Portfolio Approach

Our Mission-Driven Approach to Treating Immune-Mediated Diseases



Address critical gaps in care...



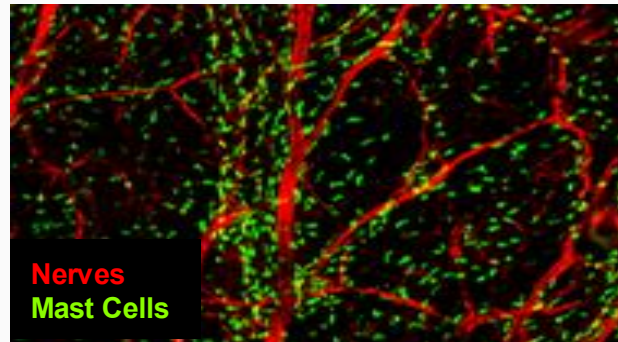
...Strategically select mechanisms with strong probability of success...



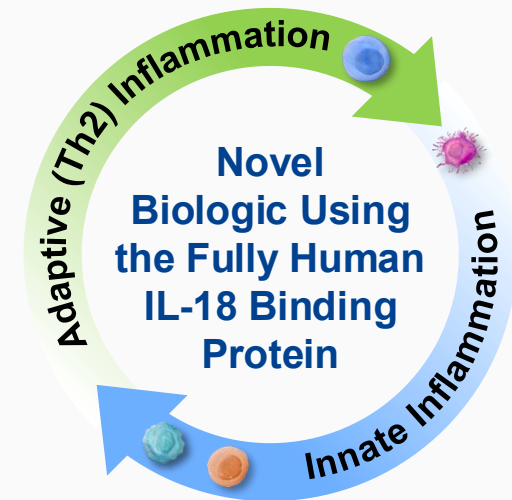
... Steady cadence of new programs entering the clinic

Two Phase 2 Programs with Novel Approaches to Targeting Heterogeneous Diseases

EVO756: Oral Therapy Targeting Mast Cells and Sensory Neurons



EVO301: IL-18 Blockade for Multi-Pathway Immunomodulation



Expansive Portfolio of Preclinical Programs

EVO756: Oral MRGPRX2 Antagonist

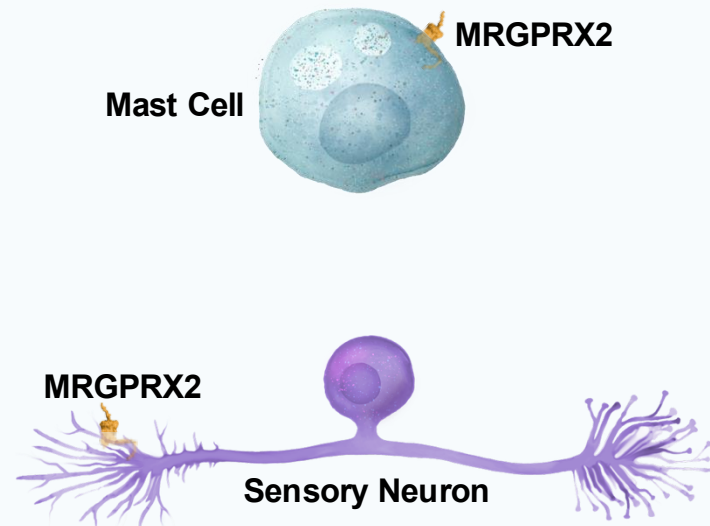
Targeted Approach to Controlling Mast Cell Mediated Diseases and Neuroinflammation

EVO756: Broad Spectrum Oral Anti-Inflammatory Potential

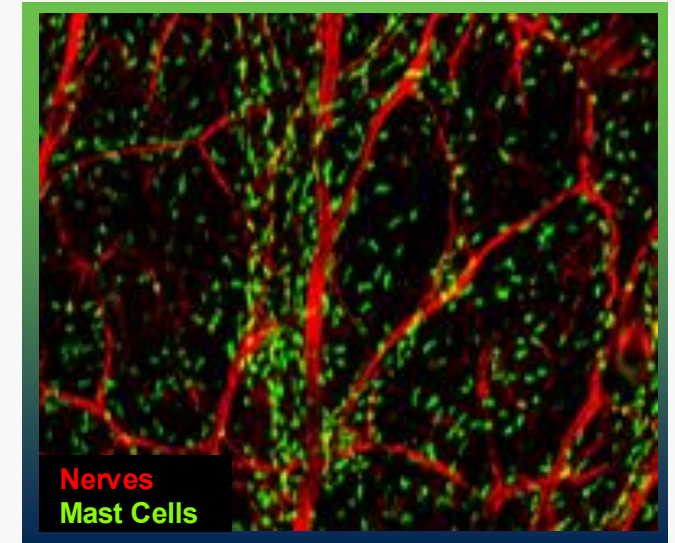
Potential First-Line Oral Across Several Specialties

- Potent and highly selective small molecule
- Oral convenience could drive adoption across multiple indications
- Anticipate favorable safety and tolerability profile

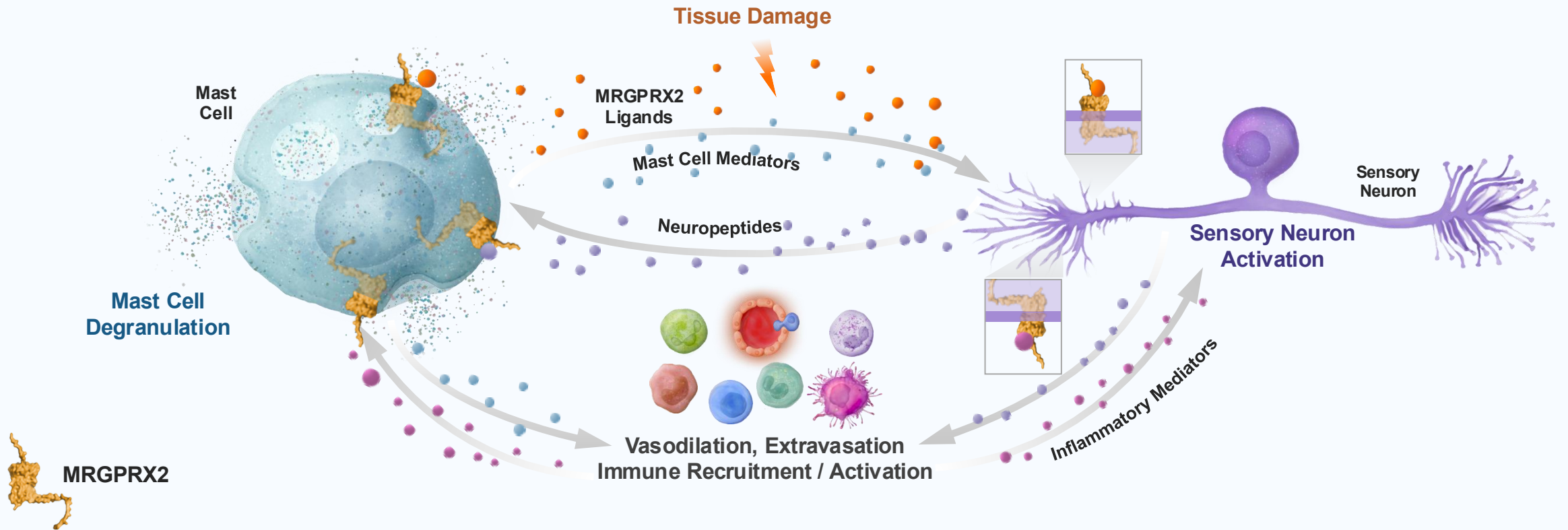
MRGPRX2 Expressed on Both Mast Cells and Sensory Neurons



Mast Cells and Sensory Neurons Are Found in Close Proximity



MRGPRX2 in Mast Cell Activation and Neuroinflammation







Tissue Pathophysiology

- Neuronal Sensitivity
- Inflammatory Infiltrates
- Increased Mast Cell Numbers
- Innate Immunity
- Adaptive Immunity
- Tissue Remodeling
- Vascular Leak

Clinical Manifestations

- Itch / Pain / Cough
- Chronic Inflammation
- Erythema
- Hives
- Barrier Dysfunction
- Airflow Limitation
- Edema Angioedema
- Sensitivity to Chemicals / Foods

EVO756 Development Roadmap: Demonstrate Proof-of-Concept and Expand into Additional Indications

 Cutaneous	 Neurological	 Respiratory	 Other
<input checked="" type="checkbox"/> Chronic Urticarias <input checked="" type="checkbox"/> Atopic Dermatitis ¹	<input checked="" type="checkbox"/> Migraine ²	<input type="checkbox"/> Asthma	<input type="checkbox"/> Irritable Bowel Syndrome <input type="checkbox"/> Interstitial Cystitis

EVO756 Development Strategy

Initially pursue inflammatory diseases with:

- Underserved patient population
- Economic viability
- Well-defined clinical and regulatory development pathway

Footnotes:

- 1) Proof-of-concept based on skin challenge test in healthy volunteers
- 2) Proof-of-concept supported by positive clinical data for PACAP (MRGPRX2 ligand) inhibition in migraine prophylaxis (Lundbeck)

EVO756 Clinical Data

Dual Mechanism Modulates Both Mast Cells and Peripheral Sensory Neurons

EVO756: Encouraging Results in Two Clinical Trials Support Initial Development in CSU and AD

EVO756 Clinical Development Summary

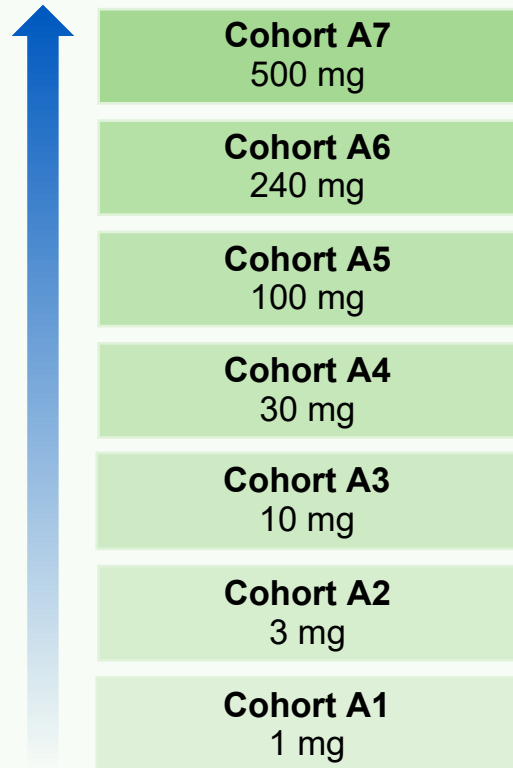
Trial	Phase 1 Proof-of-Concept	Phase 2	Phase 2b	Phase 2b
N	132	30	~160	~120
Indication	Healthy Volunteers	CIndU	CSU	AD
Key Takeaways	<ul style="list-style-type: none"> Well-tolerated across all doses Clear target engagement in skin challenge Concentration dose proportional and linear 	<ul style="list-style-type: none"> Well-tolerated across all doses Complete responses as early as week 1 POC achieved after just 4 weeks of dosing 	Topline Data Expected Q2 2026	Topline Data Expected Q3 2026

EVO756: Phase 1 Proof-of-Concept Trial Design and Summary

Inclusion of Skin Challenge in MAD Portion Allowed Early PD Assessment and Trial Showed Positive Pharmacokinetics and Pharmacodynamics

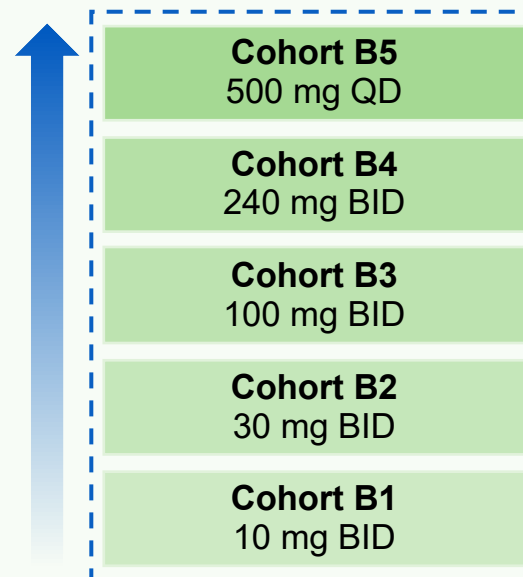
PART A: SAD Dosing

N = 55 (41 active / 14 placebo)



PART B: MAD Dosing

N = 77 (58 active / 19 placebo)



*Included Skin Challenge
at All Doses*

Pharmacokinetics

- Concentration dose proportional and linear
- Half-life ranges from 8 - 12 hours
- T_{max} : 1 - 4 hours
- Support QD and BID dosing

Pharmacodynamics – Icatibant Skin Challenge Test

- Clear target engagement
- Dose dependent activity
- All doses associated with response

Safety

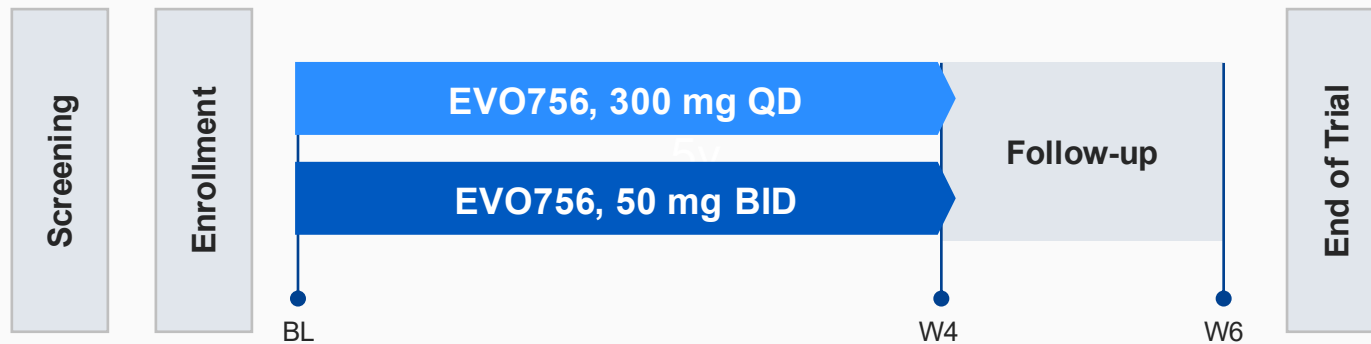
- Well-tolerated across all doses
- No severe or serious adverse events
- No clinically significant abnormal lab values
- No clinically significant ECG abnormalities

EVO756: Phase 2 Trial in Chronic Inducible Urticaria (CIndU) Data

Phase 2 Chronic Inducible Urticaria (CIndU) Trial Design

Adults with Chronic Inducible Urticaria (N = 30)

Open Label, Within-Patient Controlled Trial (All with SD)



Patient Population

- Symptomatic dermographism, Total FricTest Score ≥ 2

Primary Endpoint

- Safety as assessed by incidence of TEAEs

Efficacy Measures

- Complete response, change from baseline in provocation test
 - Total FricTest Score
- Change from baseline in Pruritus-NRS at provocation site

Biomarker Data

- Patient subtyping (e.g., IgE high and low)
- Pharmacodynamics and disease severity

Both Doses Demonstrated Robust Clinical Activity

At Just 4 Weeks, 70% ≥ 1 Pt Improvement, 41% ≥ 2 Pt Improvement, 30% Complete Response Rate

FricTest



- Standardized provocation test
- 4 calibrated tines

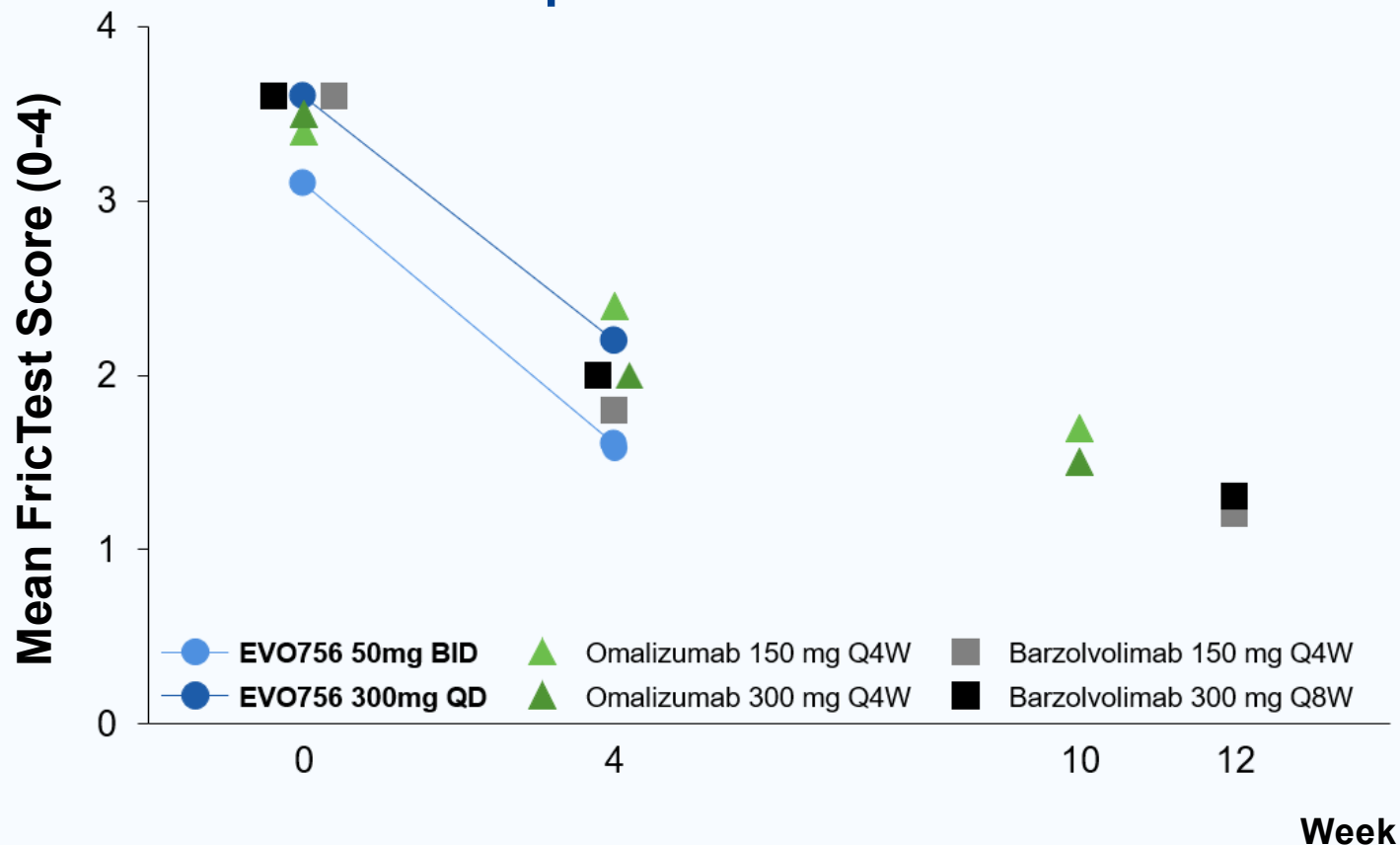
FricTest Response at Week 4

	300 mg QD	50 mg BID	All
N (efficacy)	10	17	27
Complete Response	3 (30%)	5 (29%)	8 (30%) ¹
Partial Response			
≥ 2-point Decrease	1 (10%)	2 (12%)	3 (11%)
1-point Decrease	2 (20%)	6 (35%)	8 (30%)
No Response	4 (40%)	4 (24%)	8 (30%) ²

EVO756 Potential for Increased Response with Longer Dosing

Omalizumab and Barzolvolimab Activity Continued to Improve Over Time

Clinical Improvements Over Time



Observations

- ✓ Comparable baseline disease severity shown between EVO756 and other SD trials
- ✓ At week 4, 300 mg QD patients (N=10) saw a **1.4 point** reduction and 50 mg BID patients (N=17) saw a **1.5 point** reduction
- ✓ Clinical response may continue to improve past week 4 similar to other agents

For illustrative purposes only. Not a head-to-head comparison. Differences exist between trial designs and study characteristics, and caution should be exercised when comparing across trials. Sources: Evommune clinical data (observed), competitor data from Maurer et al. (2017), Maurer et al., ACAAI (2024)



Safety Summary

Well Tolerated Across All Evaluated Dosing Levels

Summary of Treatment Emergent Adverse Events Occurring in >1 Patient

	300 mg QD N = 11	50 mg BID N = 19
ALT/AST Increased	2 (18%) ¹	–
Gastroenteritis	1 (9%)	1 (5%)
Pruritus	1 (9%)	1 (5%)

EVO756 was Generally Well Tolerated

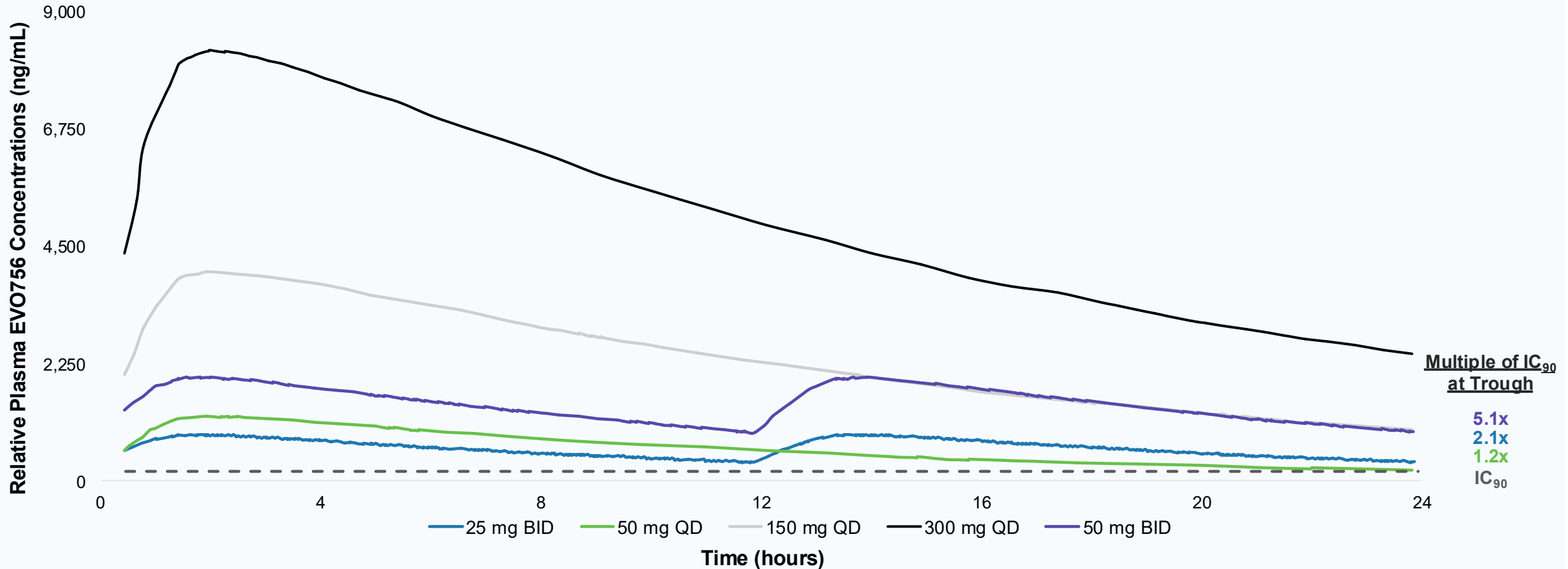
-  No serious adverse events
-  No treatment discontinuations due to adverse events

EVO756: Pharmacokinetic (PK) Data and Molecular Properties

Pharmacokinetic Modeling Based on Clinical Data to Date

IC₉₀ Coverage Across All Dose Levels Supports Potential QD and BID Dosing Regimens

Day 10 Median Concentrations



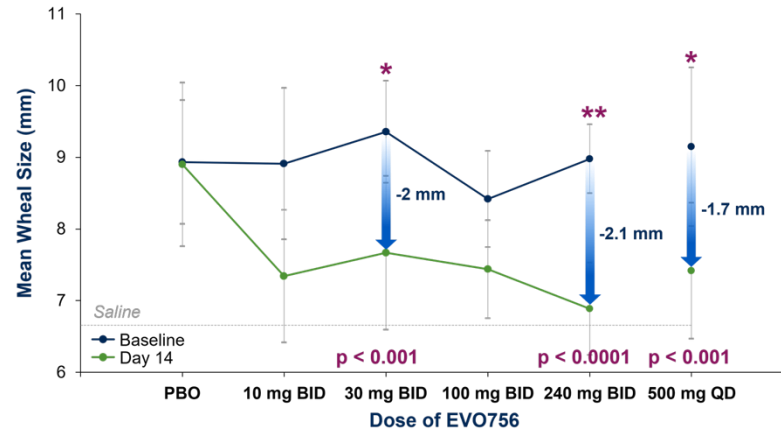
Footnotes: IC₉₀ Primary Mast Cells = 180 ng/mL

EVO756 Phase 2b Dose Selection Rationale

Understanding of Dose Response Evolved During CIndU Study, Guiding Phase 2b Trial Doses

HV Icatibant Skin Challenge

- Active across all icatibant doses
- **10 µg/mL icatibant dose is most relevant comparison** based on patient biopsies
- Suggests **potential activity as low as 10 mg BID**



PK/PD Modeling

- Refined model to predict IC₉₀ coverage at trough
- Suggested **complete coverage as low as 25 mg BID**
- **High tissue penetration** in human skin (~70%)

EVO756 Phase 2 CIndU Results

- Strong activity in 300 mg QD dose **provided confidence to explore lower doses**
- 50 mg BID dose had similar activity

Selection of Phase 2b Doses

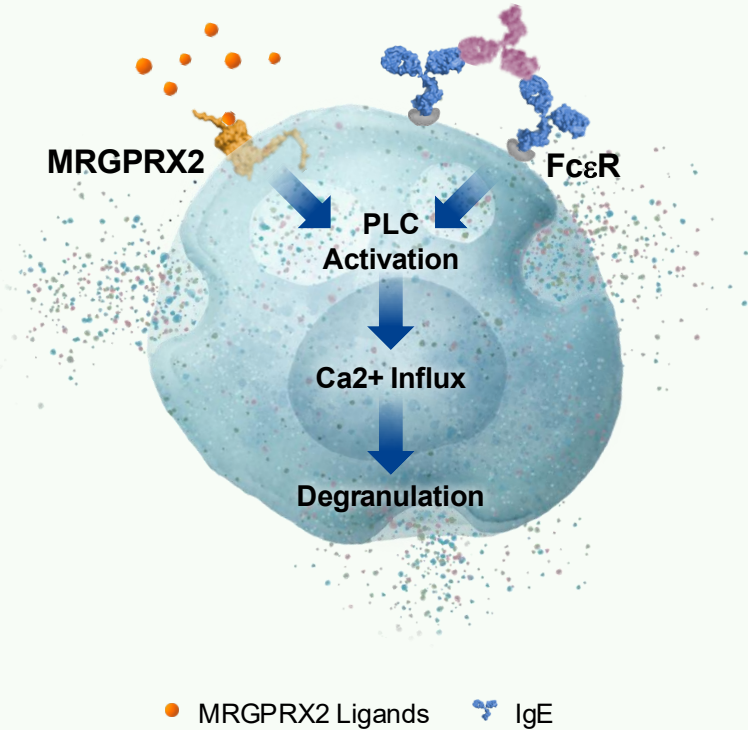
Potential for large therapeutic window; driving approach to dose-ranging trials

EVO756: Phase 2b Trial in Chronic Spontaneous Urticaria (CSU)

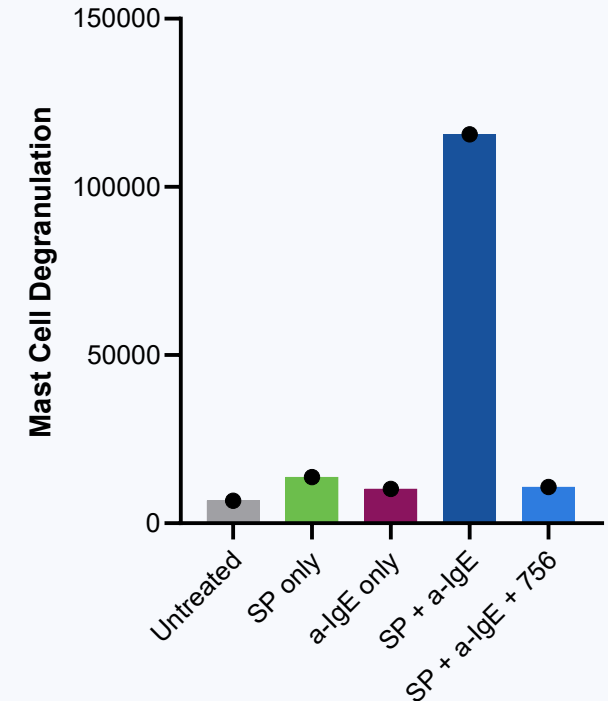
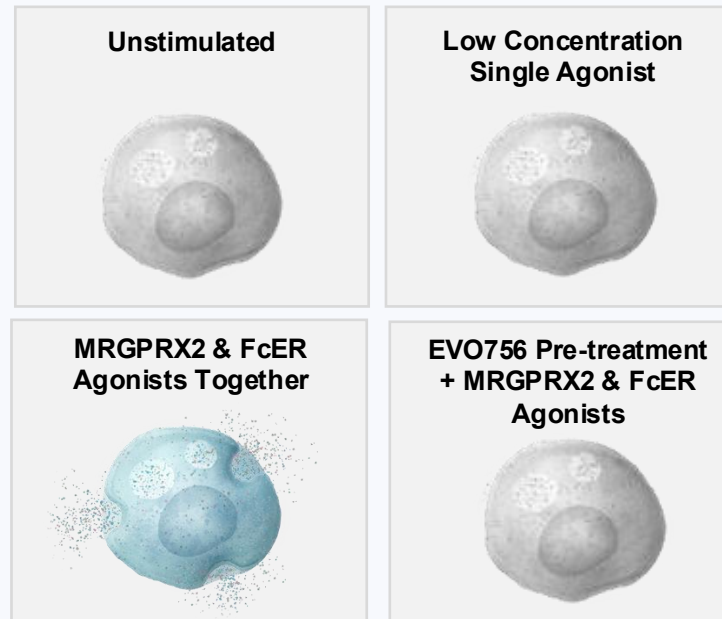
EVO756 Blocks MRGPRX2 & IgE Mast Cell Degranulation

Suggests benefit of MRGPRX2 inhibition for all CSU endotypes

In Mast Cells, MRGPRX2 and FcεR Pathways Converge



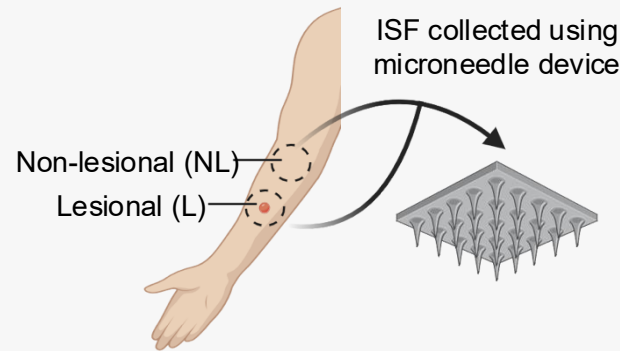
Dual Activation of MRGPRX2 and IgE Potently Drives Degranulation, and EVO756 Blocks this Effect



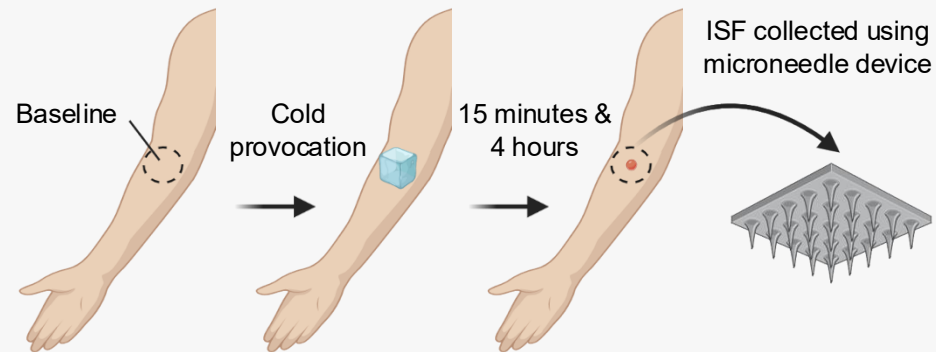
Strong Evidence of Overlap between CIndU and CSU Pathogenesis

Interstitial Fluid (ISF) Collection

Chronic Spontaneous Urticaria (CSU)

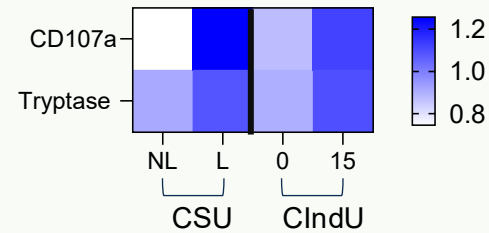


Chronic Inducible Urticaria (CIndU)

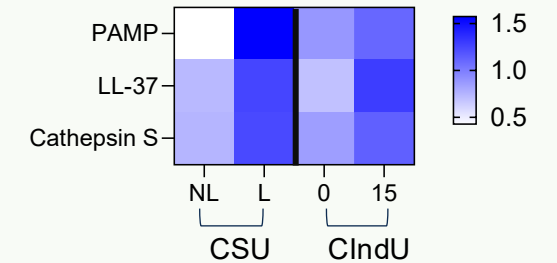


Mass Spectrometry Results

Increased Mast Cell Activation in CSU and CIndU ISF

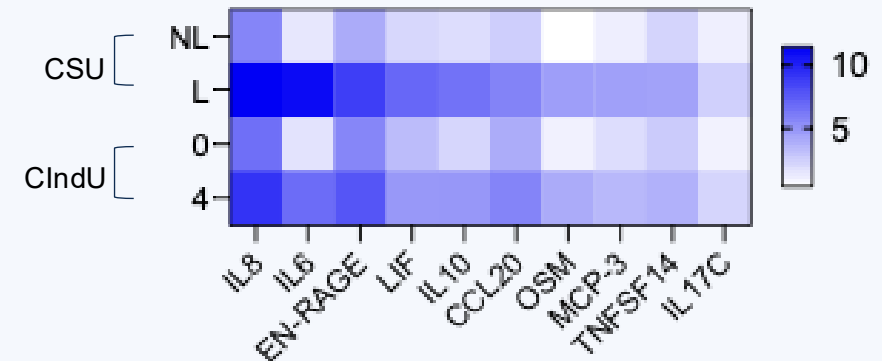


MRGPRX2 Ligands Upregulated in CSU and CIndU



Inflammation Panel









Shared Set of Top 10 Secreted Proteins Enriched in CSU and CIndU ISF



Relationship Between CIndU Efficacy and Impact on CSU

Phase 2 EVO756 Results in CIndU Provide Early Support for Potential CSU Clinical Profile

Correlation Between CIndU Success and CSU Benefit

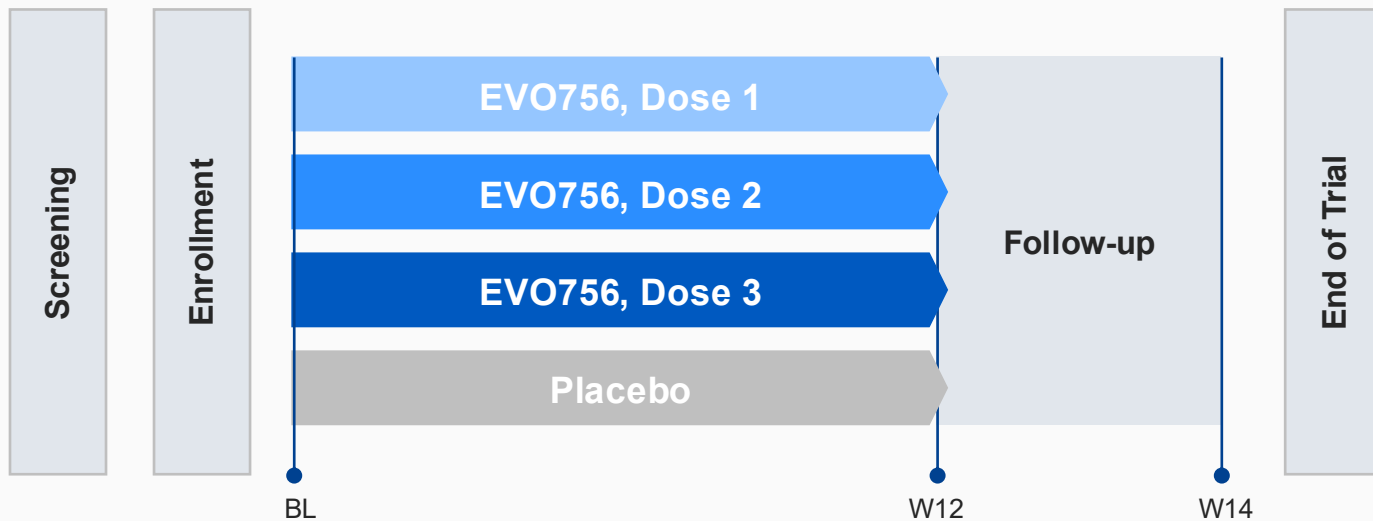
MOA	Cell Target	Drug	CIndU	CSU
IgE	Mast Cells Basophils Eosinophils	omalizumab		
KIT	Mast Cells Hematopoietic Stem Cells Germ Cells Melanocytes	barzolvolimab		
BTK	Mast Cells B Cells Basophils Myeloid Cells	remibrutinib		
IL-4 / IL-13	Th2 Cells Epithelial Cells Macrophages	dupilumab		

Phase 2b Dose-Ranging Trial in CSU

Top-Line Data Expected Q2 2026

Adults with Mod-to-Sev CSU, Refractory to H1 Antihistamines (N = 160)

Randomized, Double-Blind, Placebo-Controlled Trial



Primary Endpoint

- Mean change from baseline in UAS7 at Week 12

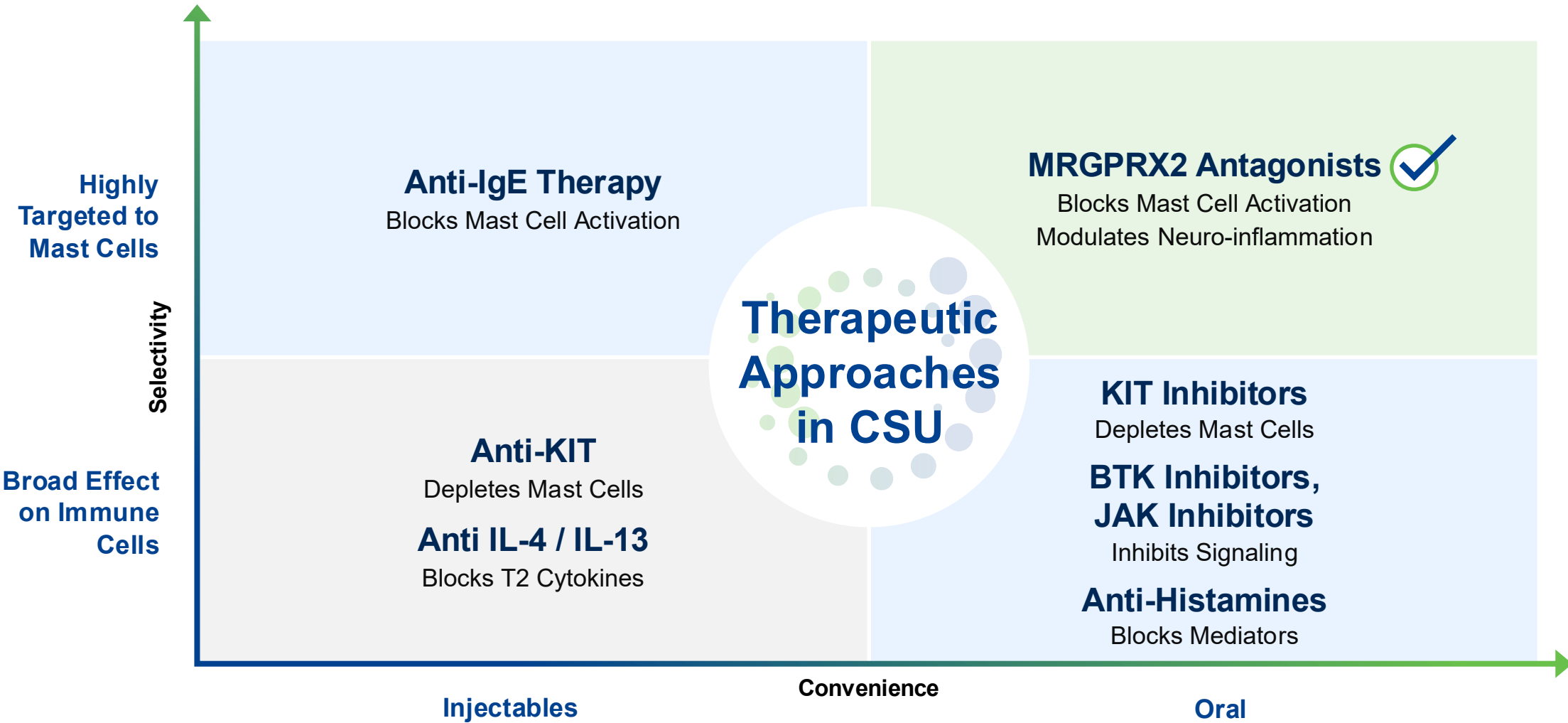
Key Secondary Endpoints

- UAS7 \leq 6 and UAS7 = 0
- Change in ISS7
- Change in HSS7
- Change in AAS7

Exploratory Biomarkers

- Patient subtyping (e.g., IgE high and low)
- Pharmacodynamics and disease severity
- Prior exposure to omalizumab allowed

EVO756: Potential to Address an Urticaria Market With Significant Therapeutic Opportunity



CSU is an Underserved Market with Limited Treatment Options

Estimated CSU Prevalence¹

~3M

Eligible CSU Patients²

~450K

Untreated by Advanced Therapies³

~400K

EVO756 Market Opportunity in Patients R/R to Antihistamines, Majority Currently Off Treatment



EVO756 Profile Potentially Amenable to First Line Treatment



Oral dosing



Potential for differentiated clinical activity profile



Well tolerated

Footnotes:

1) In the US; Maurer et al. (2011)

2) In the US; "Eligible" defined as CSU patients with incomplete response to OTC H1-antihistamines and eligible for targeted therapy

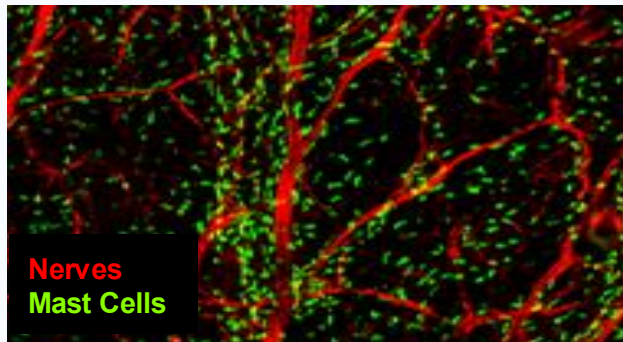
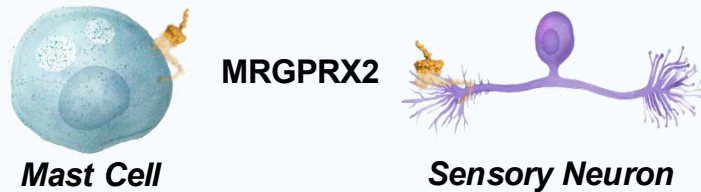
3) Approximately 50K patients currently treated with a biologic



EVO756 in Atopic Dermatitis (AD)

MRGPRX2 is Only Dual MOA: Targeting Inflammatory Lesions and Neuroinflammation

Expect Benefit on Mast Cell and Neuroinflammation Aspects of AD



Strong Scientific Rationale for EVO756 in AD



Mast cell and neuroinflammatory disease

Dual mechanism impacting key inflammatory pathways



Rapid impact on itch

Direct effect on sensory neurons



Broad therapeutic potential

MOA likely effective across patient endotypes



Strong translational validation

Pathway activation in disease and preclinical evidence of Mrgprb2/X2 involvement

CytoReason Collaboration: *in silico* AD Model Identifies and Validates Opportunities Beyond IL-4/IL-13

MRGPRX2 Signature Generation



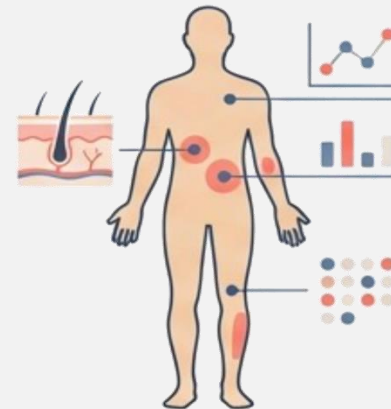
From RNAseq of *in vitro* human mast cells activated with MRGPRX2 ligands with/without EVO756

Computational Engine



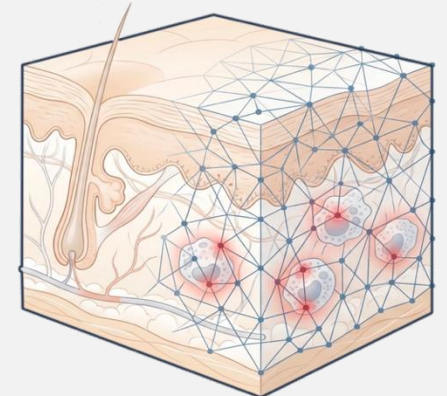
Integration of MRGPRX2 signature, 24 RNAseq datasets, >2,000 of human skin biopsies

Clinical Anchoring



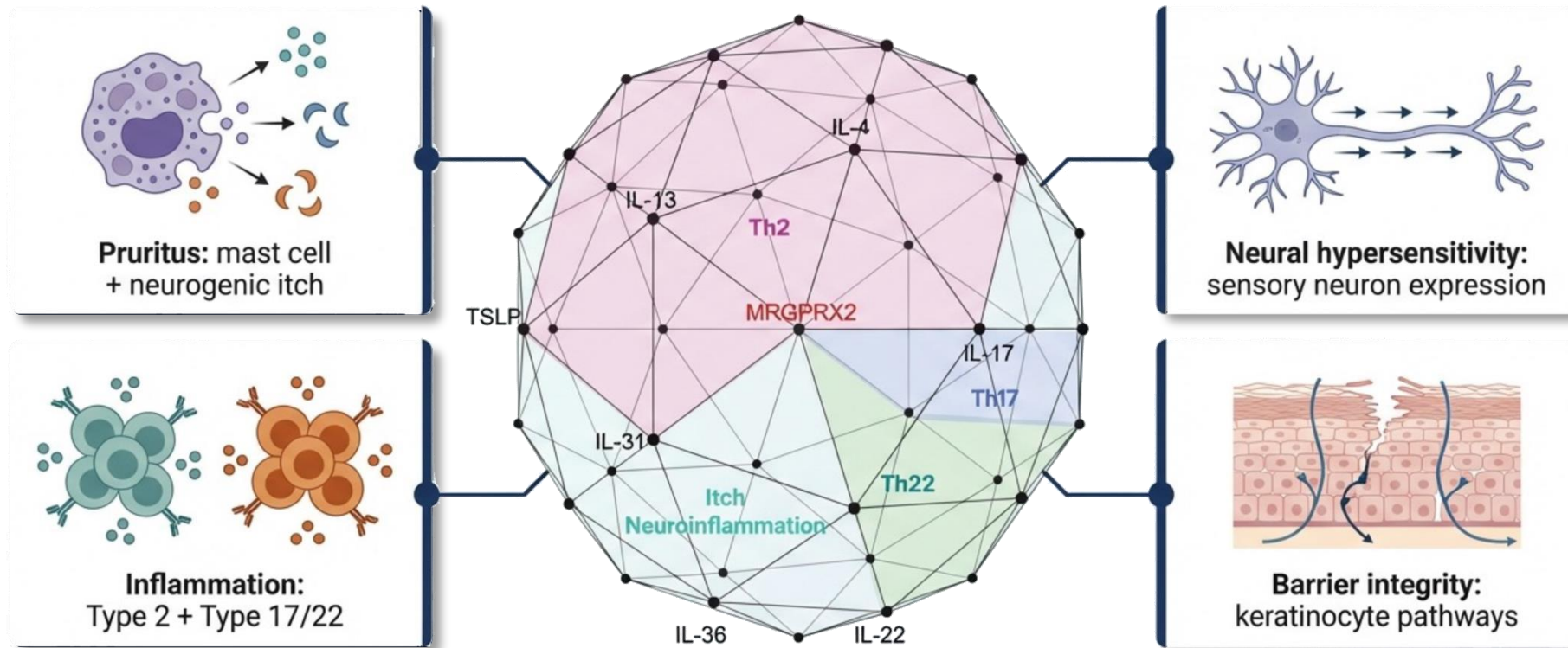
Model calibration against EASI scores and Dupilumab response data

in silico AD Model



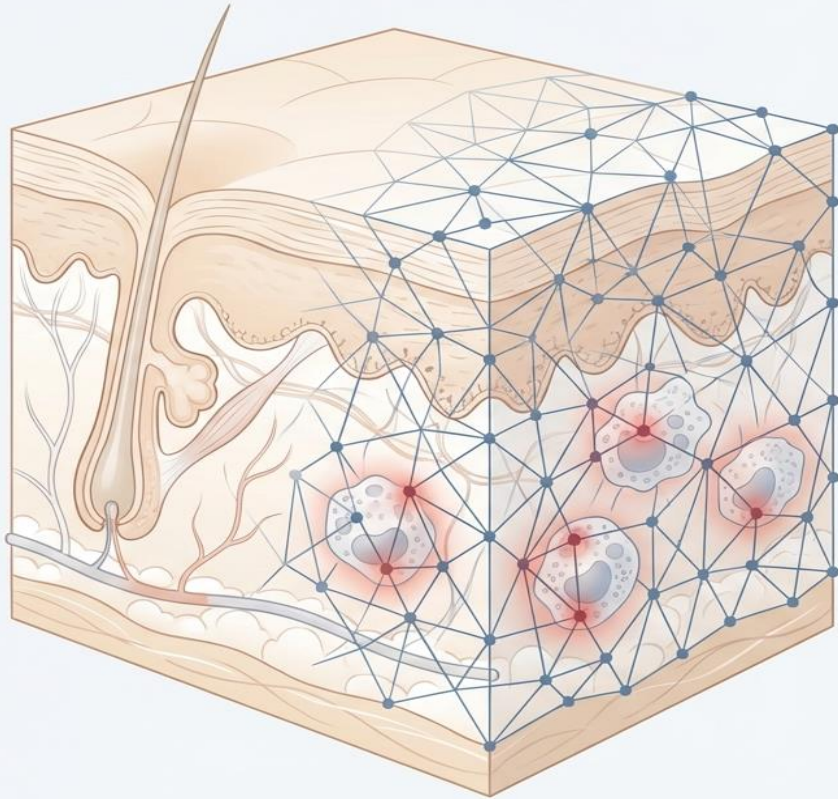
Acts as digital twin by integrating high-dimensional molecular data with real world clinical endpoints

Predictive Modeling Reveals MRGPRX2 as a Core to AD Pathophysiology



Broad reach may enable EVO756 to potentially outperform IL-4/-13–restricted approaches

MRGPRX2 Captures Core IL-4/-13 Biology and Broader Disease Drivers



Shared Biology

- MRGPRX2 and IL-4/-13 signatures capture both Th2 inflammation and neuroinflammation

Differentiated Coverage

- MRGPRX2 uniquely captures “white space” biology, including cellular proliferation and barrier pathways
- MRGPRX2 molecular signature remains expressed in non-responders to IL-4/-13 standard of care

Implications

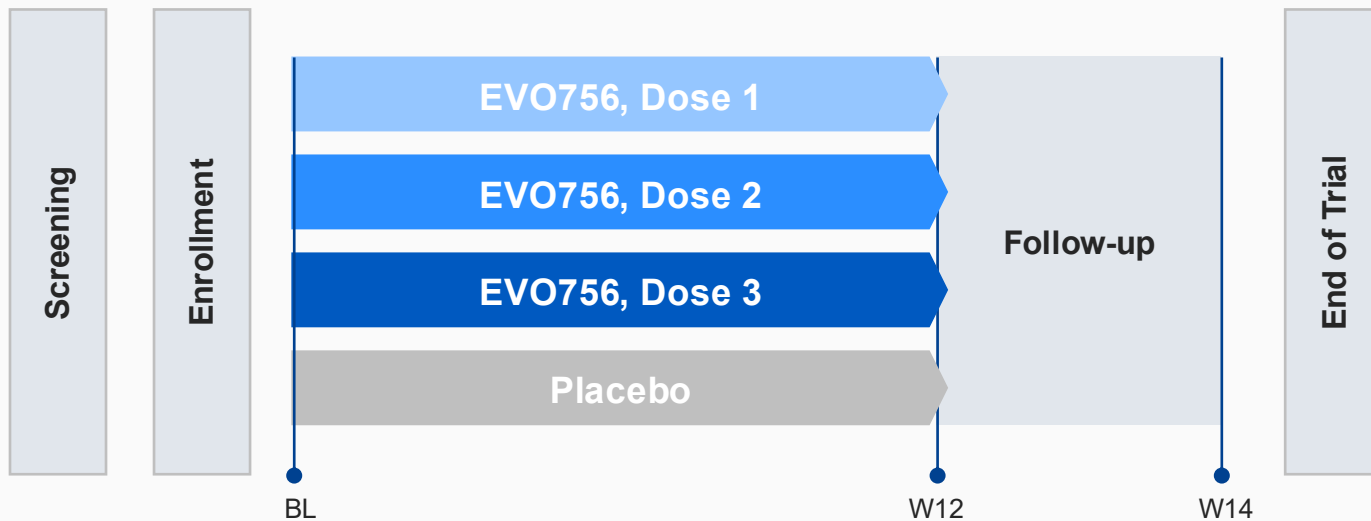
- Complementary—not redundant—mechanism
- Potential to expand efficacy beyond current standard of care

Phase 2b Dose-Ranging Trial in AD

Top-Line Data Expected Q3 2026

Adults with Moderate-to-Severe Atopic Dermatitis (N = 120)

Randomized, Double-Blind, Placebo-Controlled Trial



Primary Endpoint

- Percent change from EASI at Week 12

Key Secondary Endpoints

- EASI-50, EASI-75, and EASI-90
- Change in vIGA
- Change in Pruritus-NRS
- Proportion of patients achieving ≥ 4 point reduction in Pruritus-NRS
- Change in BSA affected

Exploratory Biomarkers

- Patient subtyping
- Pharmacodynamics & disease severity

AD is an Underserved Market Lacking a First-Line Oral Option

Current Therapies Fail to Deliver Both Lesion Control and Itch Relief, While Also Being Well-Tolerated

Estimated Adult AD Prevalence¹

~16M

Patients with Moderate-to-Severe Disease

~6M-8M

Patients Eligible for Targeted Therapy

~1.4M-1.8M

EVO756 Market Opportunity Across Patient Populations within AD



EVO756 Profile Potentially Amenable to First Line Treatment



Oral dosing



Potential for differentiated clinical activity profile on lesions and itch



Well tolerated

Footnotes:

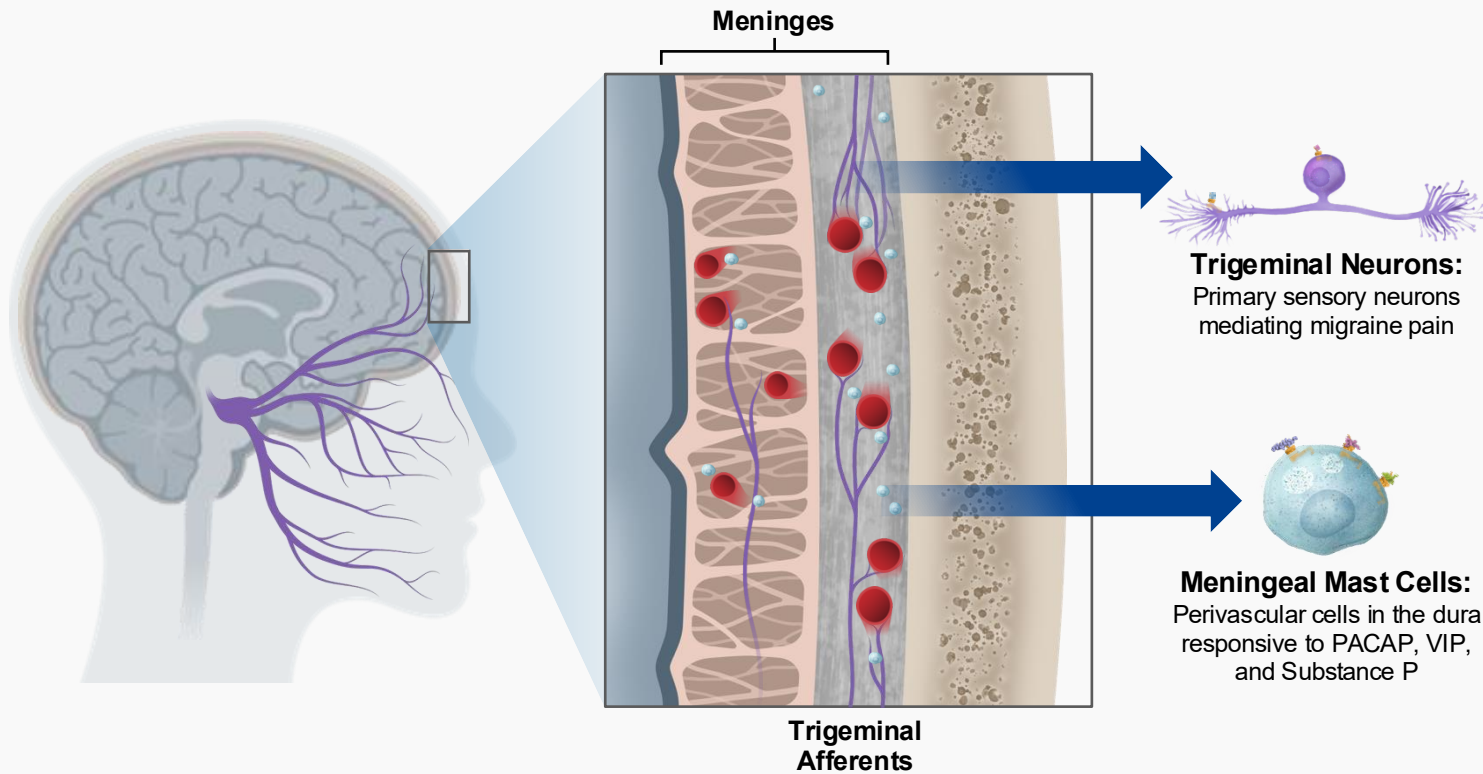
1) In the US; Atopic dermatitis impacts >100M adults and more than 102M children worldwide. Global epidemiology of atopic dermatitis: a comprehensive systematic analysis and modelling study. British Journal of Dermatology 2023 Dec 20; 190(1):55-61.



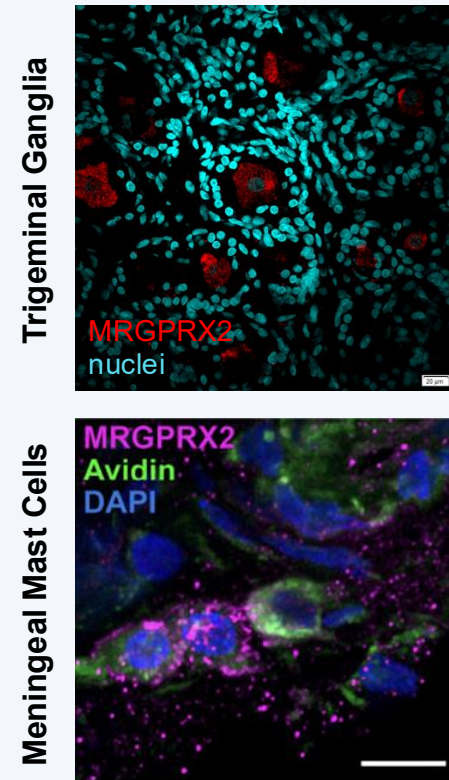
EVO756 in Migraine

MRGPRX2: Positioned to Address Neuronal and Mast Cell Drivers of Migraine

MRGPRX2 Mediates Neuropeptide Signaling Associated with Migraine (PACAP, VIP, Substance P)

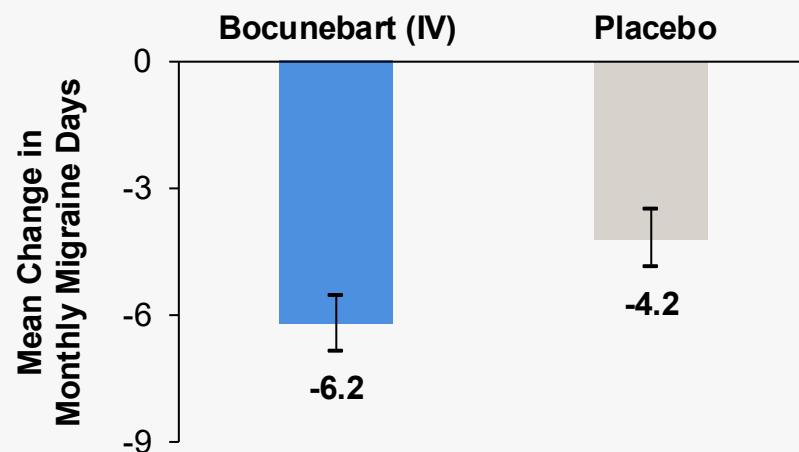


Expression Confirmed in Disease-Relevant Tissues



Inhibition of MRGPRX2 Ligand PACAP Achieves CGRP-Like Efficacy in Migraine Prophylaxis

Lundbeck's Bocunebart Reduced Monthly Migraine Days by ~2



- Magnitude of benefit consistent with marketed CGRP inhibitors

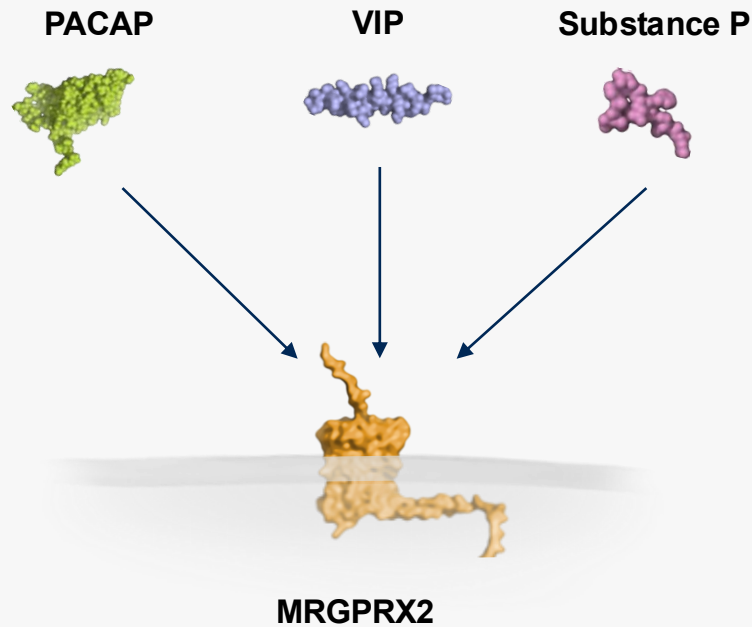
Second Neuropeptide Axis Validated in Migraine Prevention

- ✓ PACAP likely acts through MRGPRX2 as a key neuropeptide trigger of migraine attacks
- ✓ Antibody blockade reduced migraine frequency in controlled clinical study
- ✓ Effect size falls within range observed for approved CGRP therapies

Note: Lundbeck's bocunebart is a humanized mAb that neutralizes PACAP. Results above from Phase 2 a study in migraine prophylaxis (HOPE; N=237; single IV administration of bocunebart in patients that were a mix of episodic and chronic migraineurs). Source: Clinicaltrials.gov NCT05133323. Direct comparisons cannot be made in the absence of head-to-head trials because of differences in trial design, patient population and other factors.

MRGPRX2 Inhibition May Offer Broader Migraine Benefit than Targeting PACAP Alone

3 Neuropeptides that Trigger Migraine Signal Through MRGPRX2



Neuropeptide	Preclinical Evidence	Induced Headache in Humans	Clinical Validation
PACAP	✓	✓	✓
VIP	✓	✓	TBD
Substance P	✓	✓	TBD

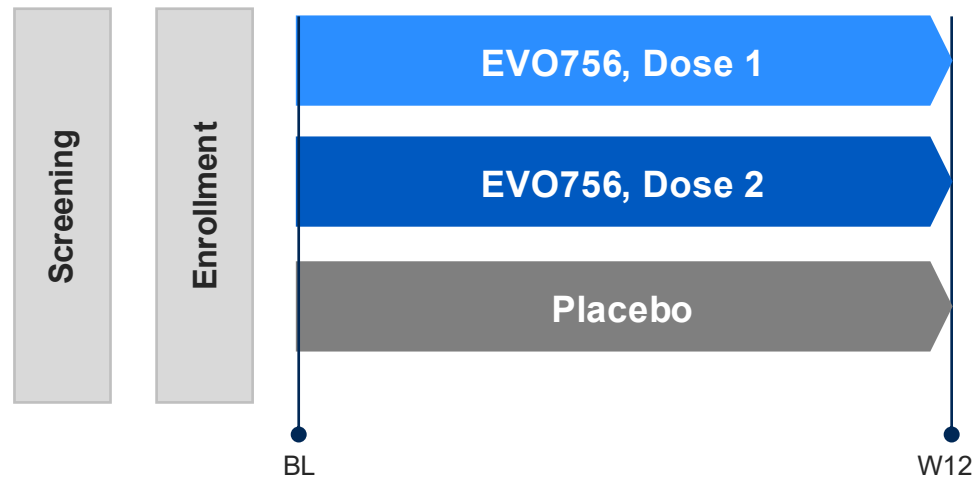
Planned Phase 2b Dose-Ranging Trial in Migraine Prophylaxis

Initiation Expected Mid-2026

Adults with Refractory Migraine ≥ 6 Days/Month

(N \approx 330)

Randomized, Double-Blind, Placebo-Controlled Trial



Exploring daily doses up to 100 mg

Primary Endpoint

- Mean CFB in MMD

Key Secondary Endpoints

- $\geq 50\%$, $\geq 75\%$ reduction in MMD
- CFB in MHDs and MMD
- CFB in monthly acute migraine medication use

Exploratory Endpoints

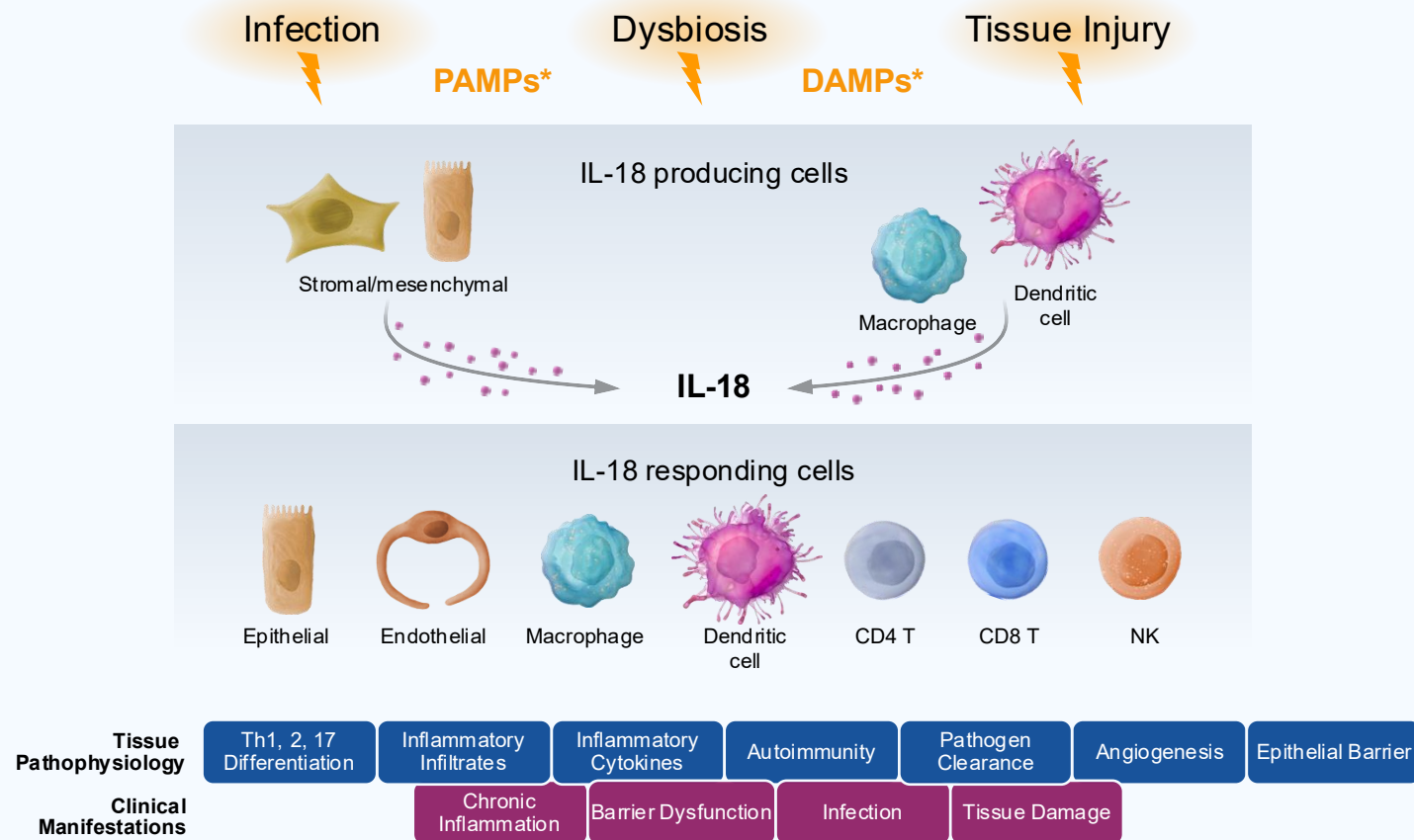
- Patient subtyping
- Changes in biomarkers
- Change in migraine-specific QoL

EVO301: IL-18BP Fusion Protein

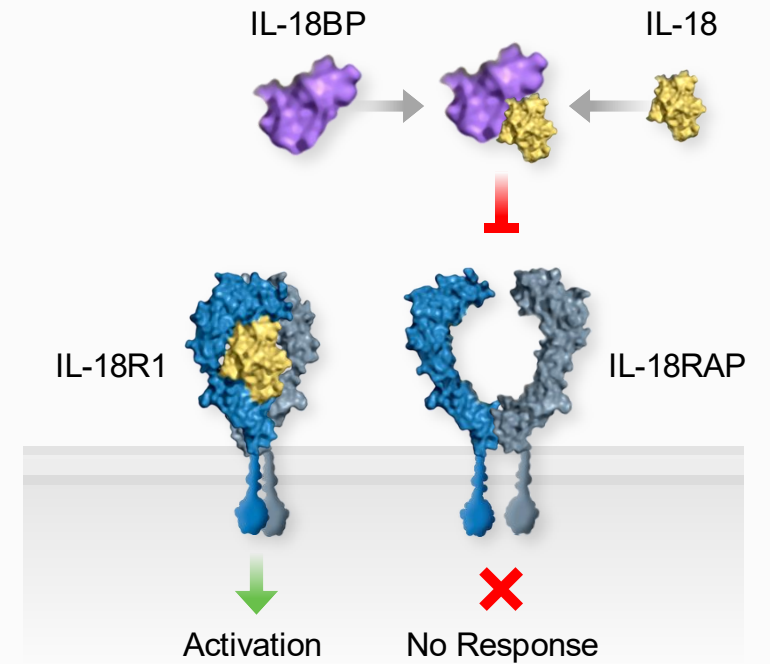
Long-Acting Serum Albumin-Binding Injectable Therapeutic Fusion Protein Designed to Neutralize IL-18 Signaling

IL-18 Immune Rebalancing: Modulate Innate and Adaptive Inflammation for Potential Disease Remission

Involved in Innate and Adaptive Immune Processes



IL-18BP Therapeutic Approach



EVO301: Long-Acting IL-18 Neutralizer Designed for Tissue Targeting

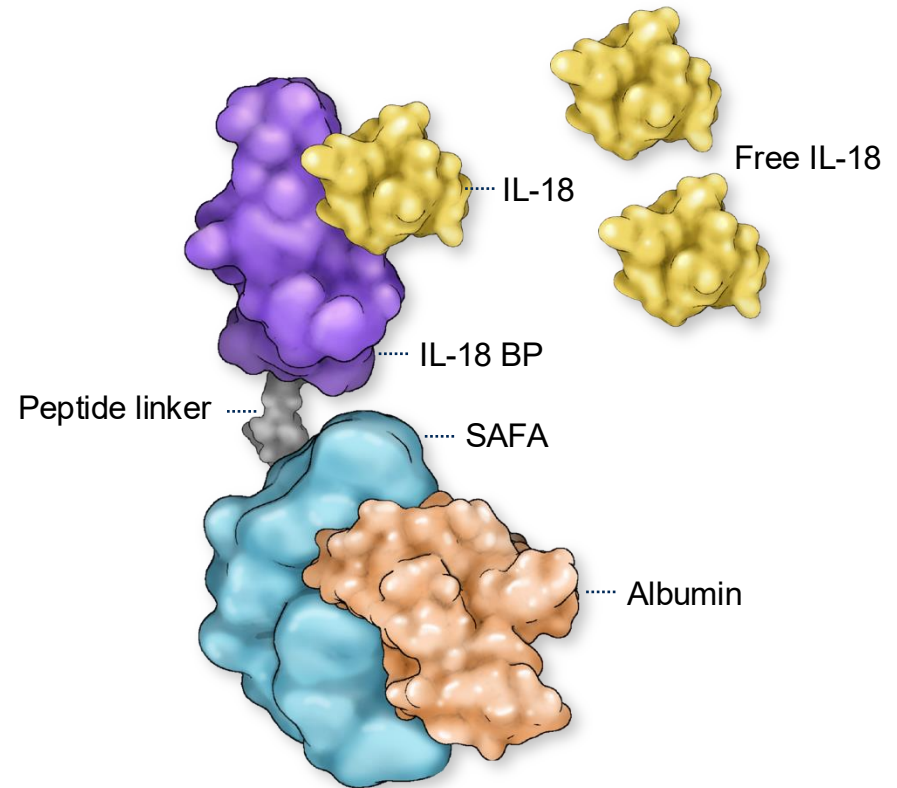
SAFA and IL-18BP fused via peptide linker for extended neutralization of IL-18 activity

SAFABody™ Platform Technology

- $T_{1/2}$ extension: FcRn-mediated recycling of HSA
- Efficient tissue distribution:
 - Smaller size (MW ~65 kD) and HSA binding

IL-18 Binding Protein (IL-18BP)

- High binding affinity and specificity
- Native fully human sequence



EVO301 Addresses Limitations of Existing Biologics; Demonstrating Ability to Impact Multiple Drivers of AD, while being Well Tolerated

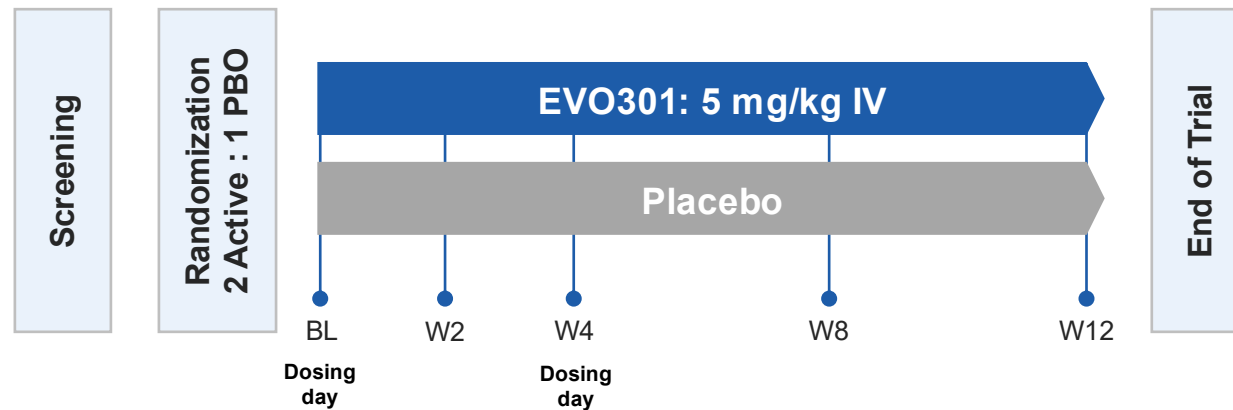
Biologic Pathway	Adaptive Inflammation			Innate Inflammation	Skin Barrier (IL-22)
	TH2	TH1	TH17		
IL-18	✓	✓	✓	✓	✓
DUPIXENT®	✓	✗		✓	✓
EBGLYSS®	✓	✗		✓	✓
ADBRY®	✓	✗		✓	✓
NEMLUVIO®	✓				✓

Broader inflammatory signaling of IL-18 can address endotypes not fully captured by Th2-targeted therapies — enabling potential for broader patient coverage and efficacy

EVO301 Phase 2a Proof of Concept Trial Design

Adults with Moderate-to-Severe Atopic Dermatitis (N = 70)

Randomized, Double-Blind, Parallel Group, Placebo-Controlled Trial



AD Population

- EASI ≥ 16
- vIGA ≥ 3
- BSA $\geq 10\%$

Primary Endpoint

- Percent change from EASI at Week 12 (Bayesian)

Pharmacokinetics

Target Engagement

EVO301 Achieved the Primary Endpoint

Phase 2a Proof-of-Concept Trial in Moderate-to-Severe Atopic Dermatitis

- **Highly statistically significant EASI reductions at weeks 4, 8, and 12 versus placebo**
- **34% and 33% placebo adjusted improvement in EASI at week 8 and 12, respectively**
- **23% of patients achieved IGA 0/1 at week 12 versus 0% placebo**
- **Well-tolerated, with no treatment related serious or severe adverse events reported**
- **Corresponding reductions in secondary endpoints, as well as key Th2 and non Th2 cytokines**
- **Pharmacokinetics (PK) continues to support a Q4 week dosing regimen**

Clinical Data Supports Continued Development, with Phase 2b Planning Underway

Disposition, Baseline Demographics and Disease Characteristics

Trial well-balanced across cohorts

	EVO301	Placebo
N (treated)	48	22
N (completed)	45 ¹	20 ²
Age	30.5 (11.1)	33.1 (11.8)
Gender (female, %)	29 (60.4%)	13 (59.1%)
Weight (kg)	78.5 (18.7)	76.4 (17.5)
BMI (kg/m²)	27.4 (6.0)	28.1 (6.3)
EASI	30.0 (11.8)	29.8 (10.5)
IGA	3.3 (0.5)	3.5 (0.5)
Pruritus-NRS	6.3 (1.5)	6.7 (2.1)
% BSA	47.1 (21.2)	49.3 (16.1)

Note: Numbers in parentheses are standard deviations. BMI: body mass index, EASI: Eczema Area and Severity Index, IGA: Investigator's global assessment, NRS: numeric rating score, BSA: body surface area, SD: standard deviation.

Subjects who were early terminations were 1. Lost to follow-up. 2. Lost to follow-up and subject withdrawal

Phase 2a Trial in AD Met Primary Outcome Measure (Bayesian)

% Change in EASI at Week 12: Protocol Success Criterion Met

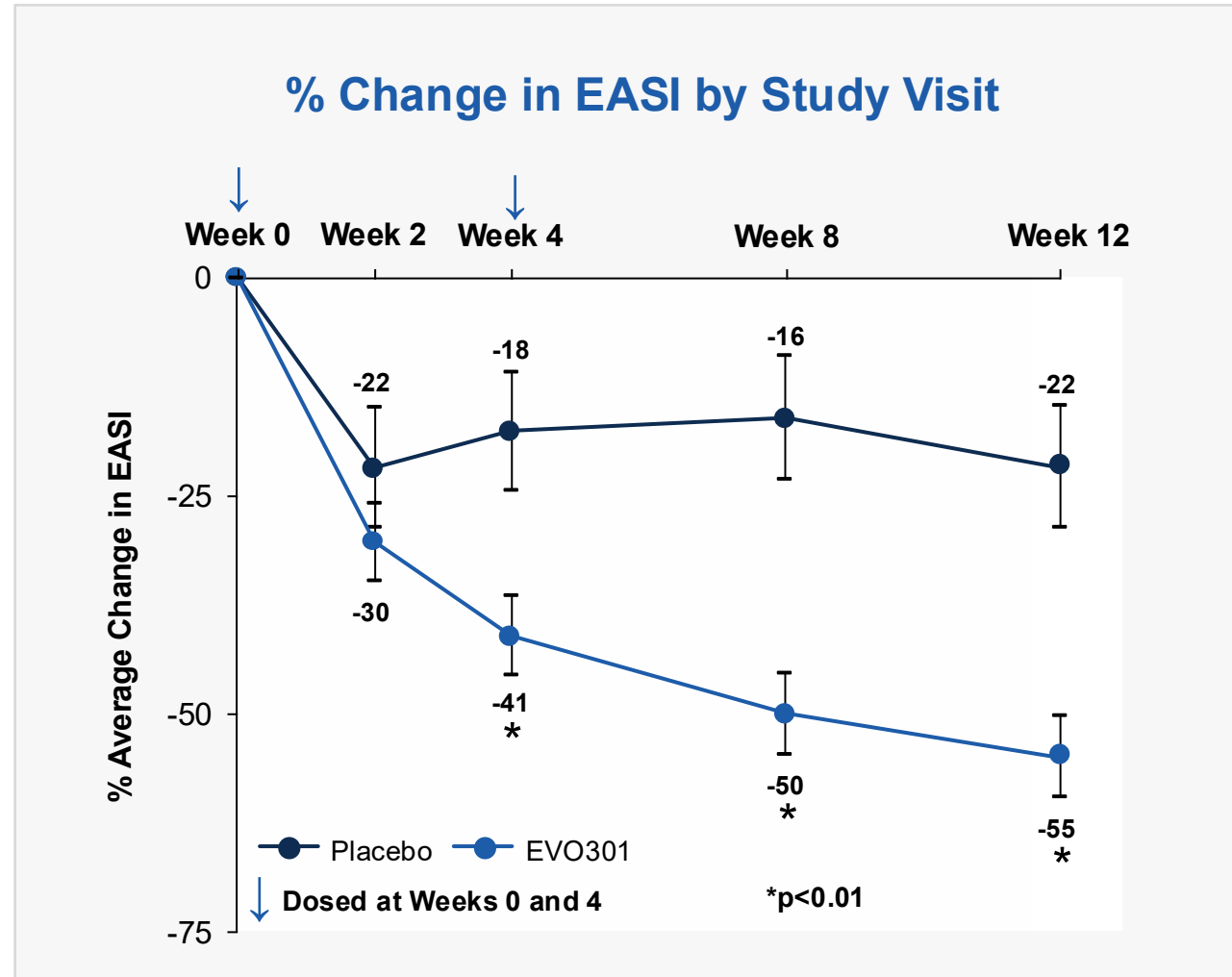
Statistic	EVO301 versus Placebo
Success Criterion: Posterior Probability of Difference < -8%	75%
Trial Results: Posterior Probability of Difference < -8%	99.8%
Posterior Mean Difference	-32
95% HPD Interval for Difference in Mean	-47, -15

Phase 2a Trial in AD Demonstrated Statistically Significant Efficacy Across Time Points

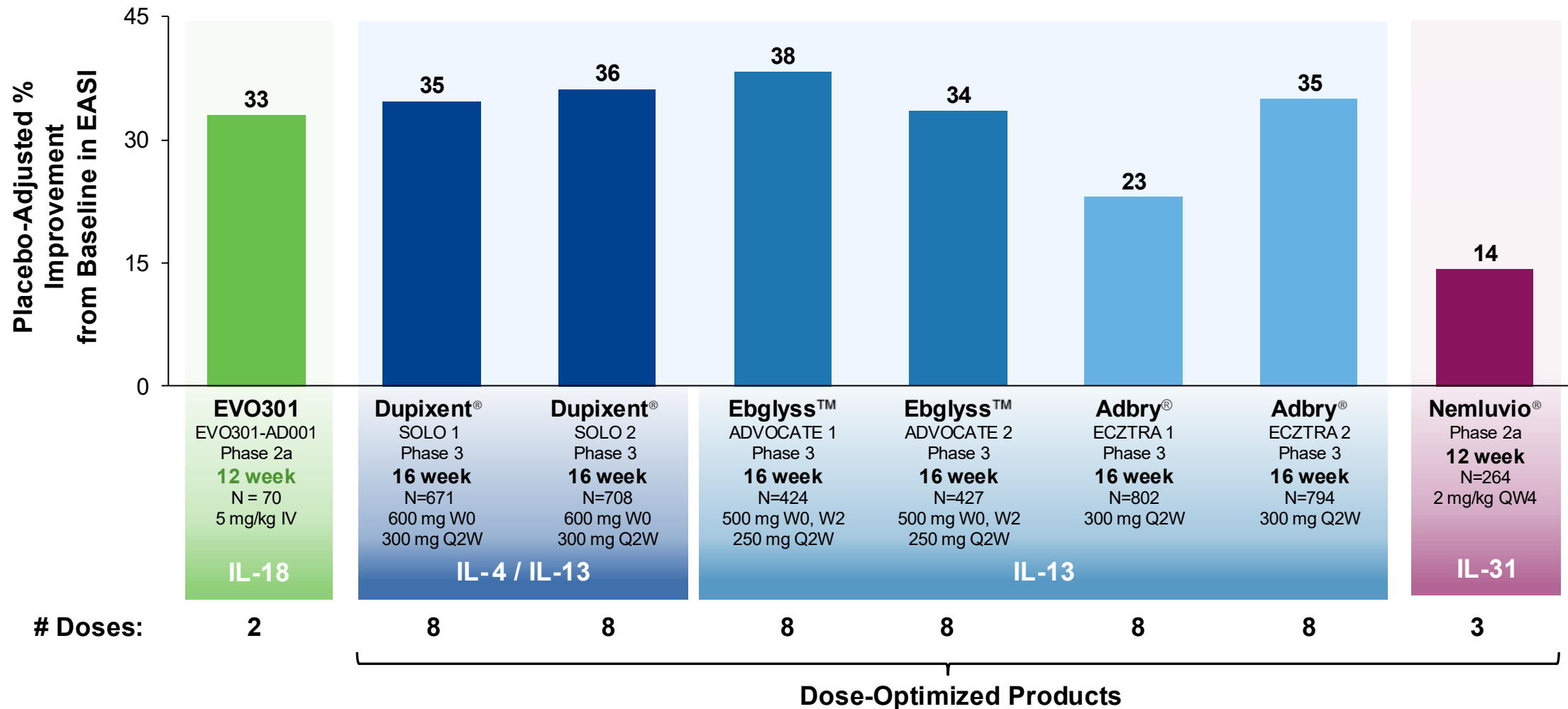
% Change in EASI by Study Visit

Visit	EVO301	Placebo	Placebo-adjusted Change	p-value
Week 4	-41	-18	-23	<0.01
Week 8	-50	-16	-34	<0.01
Week 12	-55	-22	-33	<0.01

Phase 2a Trial in AD Demonstrated Statistically Significant Efficacy Across Time Points

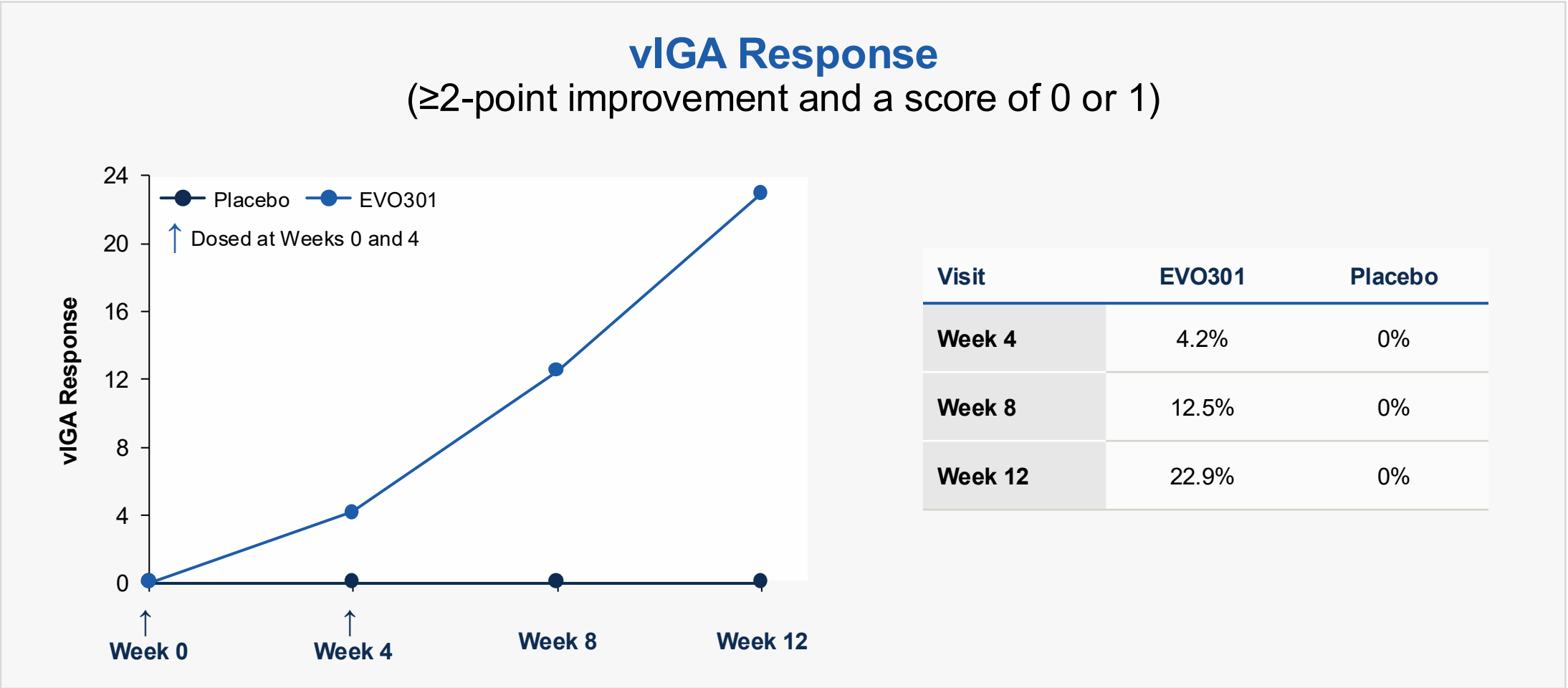


Two Doses of EVO301 Demonstrated Comparable Activity at 12 Weeks to Dose-Optimized Marketed Biologics at 16 Weeks



For illustrative purposes only. Not a head-to-head comparison. Differences exist between trial designs and study characteristics, and caution should be exercised when comparing across trials. Sources: Silverberg *et al.* (2016), Silverberg *et al.* (2023), Wollenberg *et al.* (2020), Ruzicka *et al.* (2017).

Phase 2a Trial in AD: Early Clinical Signal in vIGA 0/1 Response



Safety Summary Over 12 Week Trial Period

EVO301 Was Well Tolerated

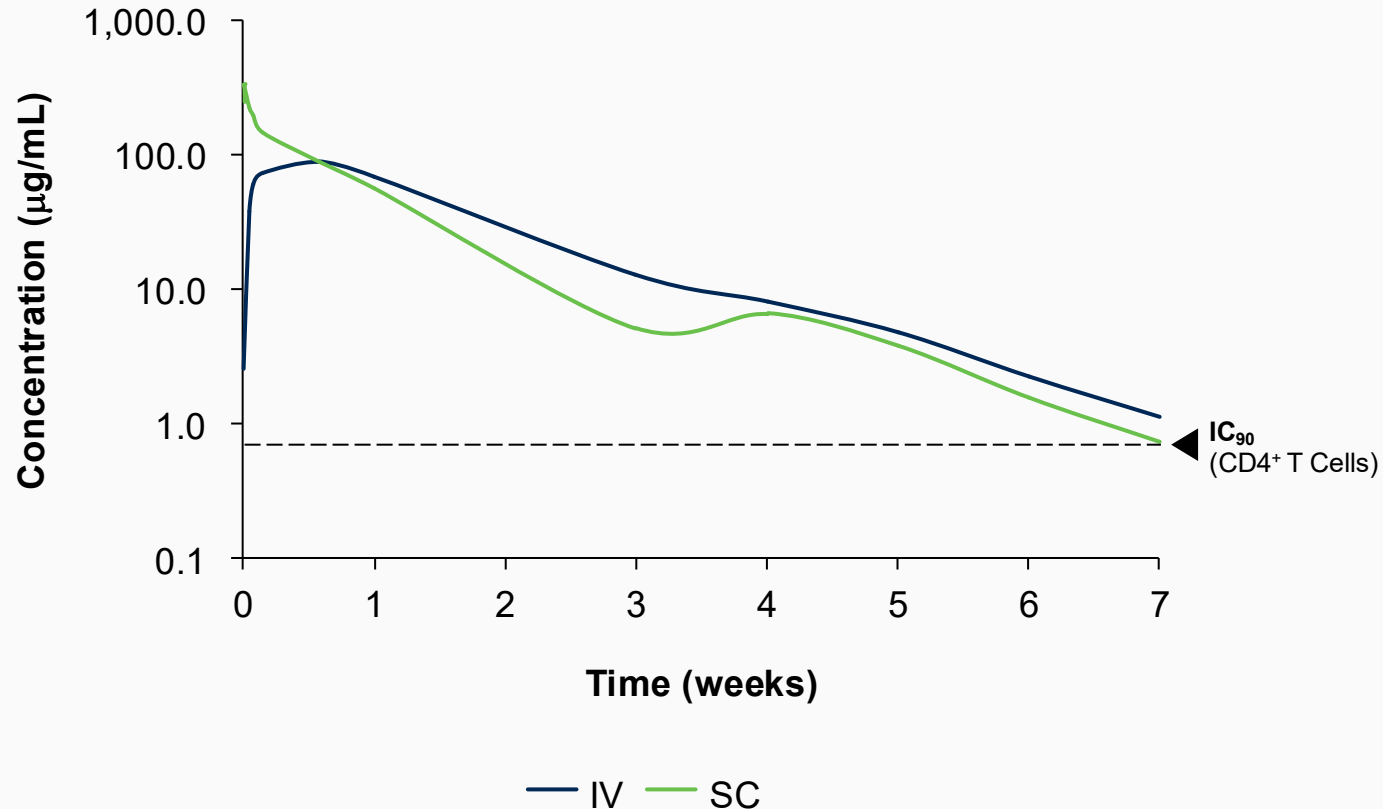
	EVO301	Placebo	Total
	N=48	N=22	N=70
Patients with ≥1 Adverse Event (AE)	30 (62.5%)	16 (72.7%)	46 (65.7%)
Patients with ≥1 Treatment Related AE	5 (10.4%)	3 (13.6%)	8 (11.4%)
Patients with a Related Serious or Severe AE	0	0	0
AEs Leading to Study Discontinuation	0	0	0

AEs > 5% in Either Arm	EVO301	Placebo	Total
Upper respiratory tract infection	10 (20.8%)	4 (18.2%)	14 (20.0%)
Atopic dermatitis	10 (20.8%)	9 (40.9%)	19 (27.1%)
Headache	8 (16.7%)	3 (13.6%)	11 (15.7%)
Nasopharyngitis	4 (8.3%)	0	4 (5.7%)
Viral upper respiratory tract infection	3 (6.3%)	2 (9.1%)	5 (7.1%)
Dizziness	3 (6.3%)	1 (4.5%)	4 (5.7%)
Fatigue	3 (6.3%)	0	3 (4.3%)

**No Clinically Significant Lab Abnormalities.
No Conjunctivitis Reported (as is Common with Other Biologics in AD)**

EVO301 Subcutaneous Formulation Exposure Consistent with IV

Comparable Nonclinical Serum Exposure



Key PK Parameters

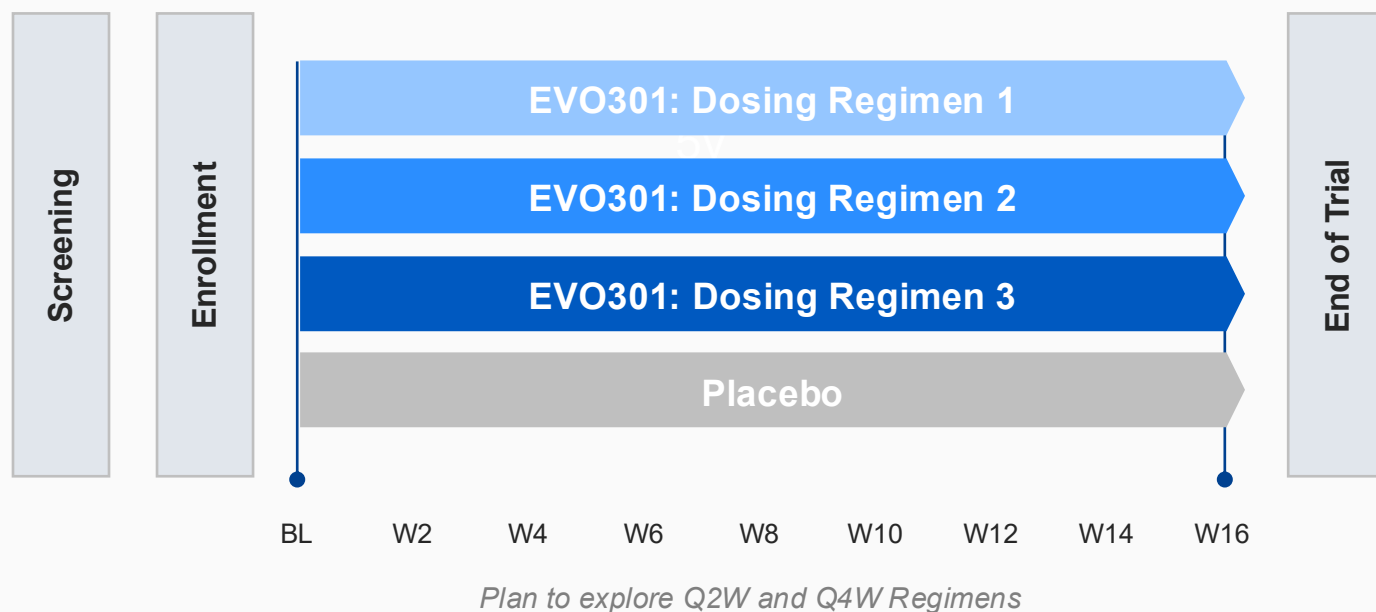
Parameters	10 mg/kg (N = 3/group)	
	Intravenous (IV)	Subcutaneous (SC)
C_{max} (µg/mL)	343	90
AUC (µg·hr/mL)	~27,000	~27,000
T_{max} (hr)	0.83	72
$T_{1/2}$ (hr)	101	168

Planned EVO301 Phase 2b Dose-Ranging Trial in AD

Trial Initiation Expected Mid-2027

Adults with Moderate-to-Severe Atopic Dermatitis (N ≈ 180)

Randomized, Double-Blind, Placebo-Controlled Trial



Primary Endpoint

- % CFB in EASI

Key Secondary Endpoints

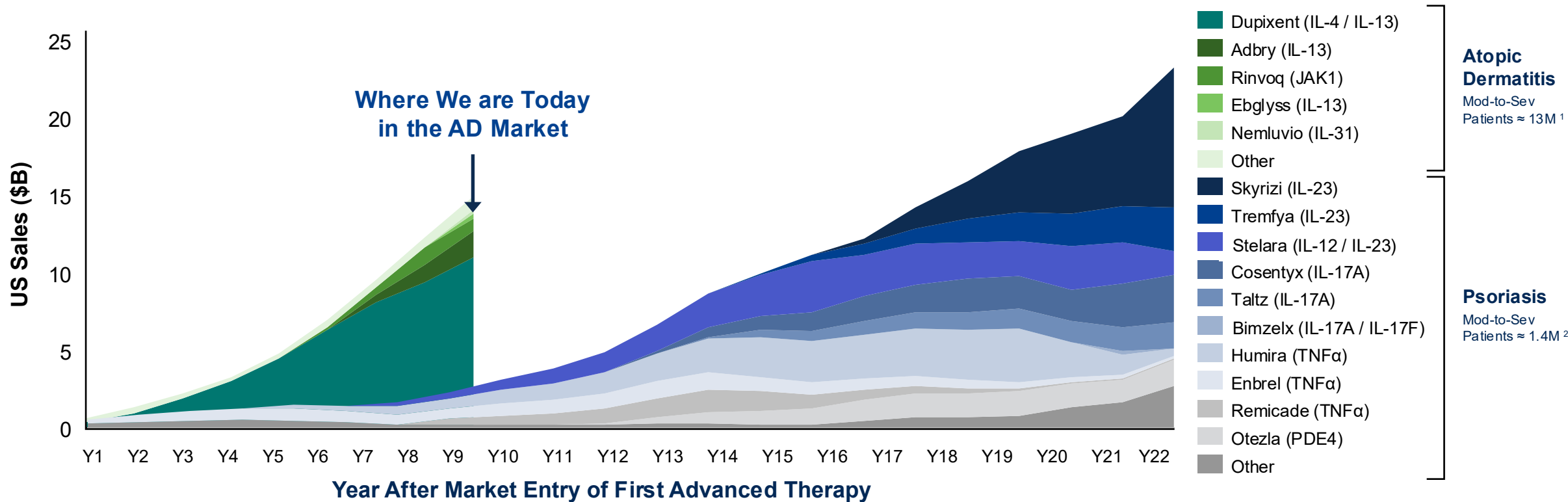
- EASI-50, EASI-75, and EASI-90
- Change in vIGA
- Change in Pruritus-NRS
- Proportion of patients achieving ≥ 4 point reduction in Pruritus-NRS
- Change in BSA affected

Exploratory & Biomarkers

- QoL
- Biomarkers
- Target Engagement

Evommune Could Reshape the Future of AD: The Largest I&I Market


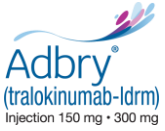


Expansion of AD Market Outpacing That of Psoriasis



Psoriasis Growth Driven by Nine Blockbusters Spanning Six MOAs. AD Remains a Concentrated Market with Clear Opportunity for New MOAs and Better Benefit-Risk Ratio.

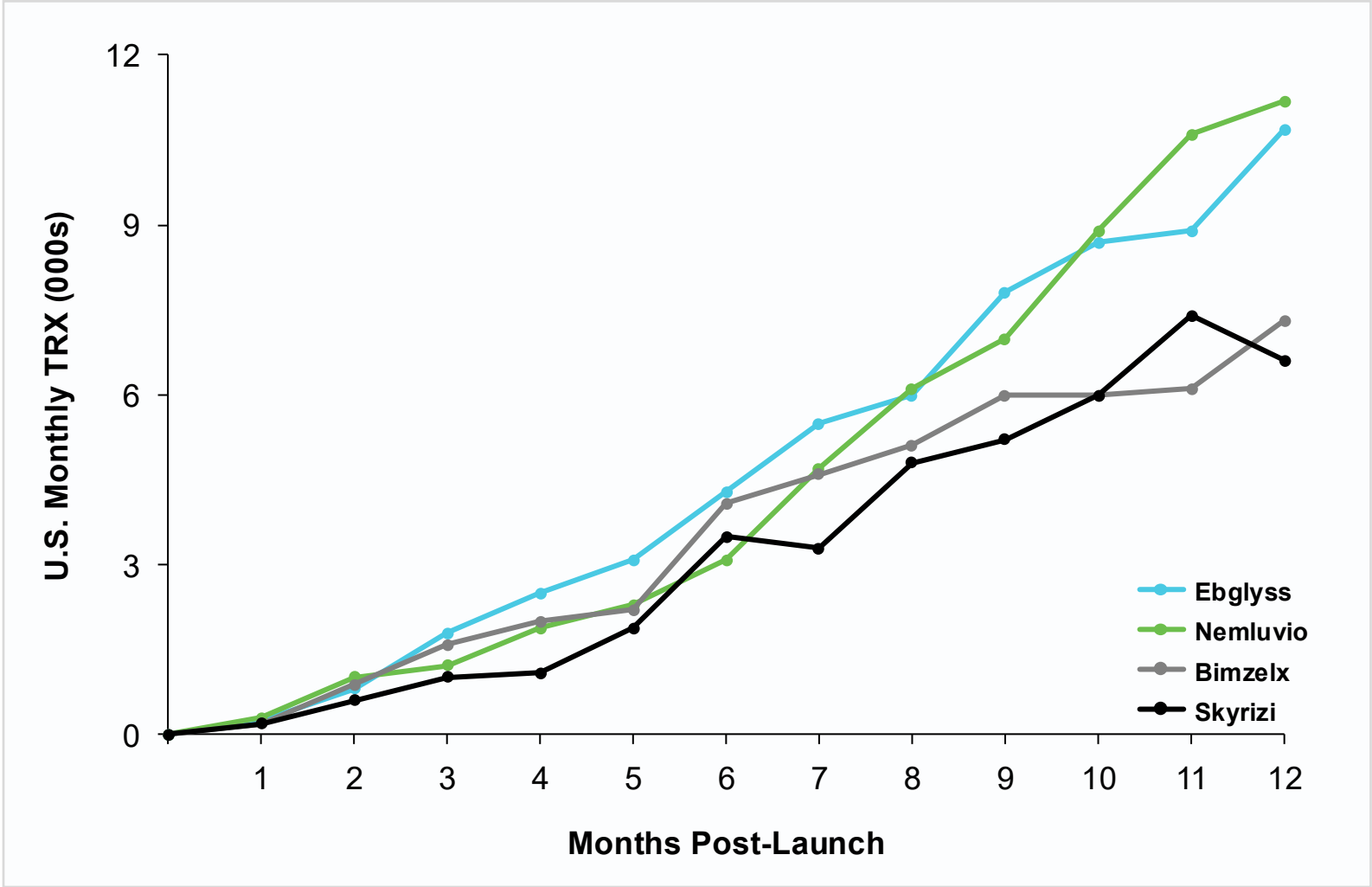
Per Evaluate Pharma (May represent projections and not actual sales); "Year 1" for AD represents 2017 (year of Dupixent launch); "Year 1" for Psoriasis represents 2004 (year of Enbrel launch in plaque psoriasis); 1. Total estimated prevalence in adult and pediatric populations; Estimated per Allergy & Asthma Network, Hanafin & Reed (2007), AAFA, Fuxench *et al.* (2019); 2. Total estimated prevalence in adult and pediatric populations; Estimated per psoriasis.org, datacenter.aecf.org, Armstrong *et al.* (2021), Paller *et al.* (2018), Tannenbaum *et al.* (2022), Helmick *et al.* (2014), Rosario-Jansen *et al.* (2025). Note that slide contains registered trademarks not owned by Evommune

EVO301 Could Command Substantial Market Share in the Potentially \$50B+ AD Market as a Clearly Differentiated Biologic

Sales in \$M	Class	Route of Administration ¹	Launch Year	2025 WW Sales	2025 US Sales	Projected Growth ²	Projected Peak WW Sales in AD
 <i>(dupilumab)</i>	IL-4/-13	Q2W SubQ	2017	12,496	9,234	+9%	17,423 (2030)
 <i>(tralokinumab-ldrm)</i> Injection 150 mg • 300 mg	IL-13	Q2W SubQ	2021	1,508	1,421	+22%	2,469 (2030)
 <i>(lebrikizumab-lbkz)</i>	IL-13	Q4W SubQ	2024	409	274	+72%	2,625 (2032)
 <i>(nemolizumab-ilto) for injection</i> 30 mg	IL-31	Q8W SubQ	2024	268	172	+91%	2,759 (2032)

Four Marketed AD Biologics Currently ~\$15B, Projected to be ~\$25B by 2032

EBGLYSS and NEMLUVIO Outpacing Psoriasis Launches, Highlighting Need for New Options in AD

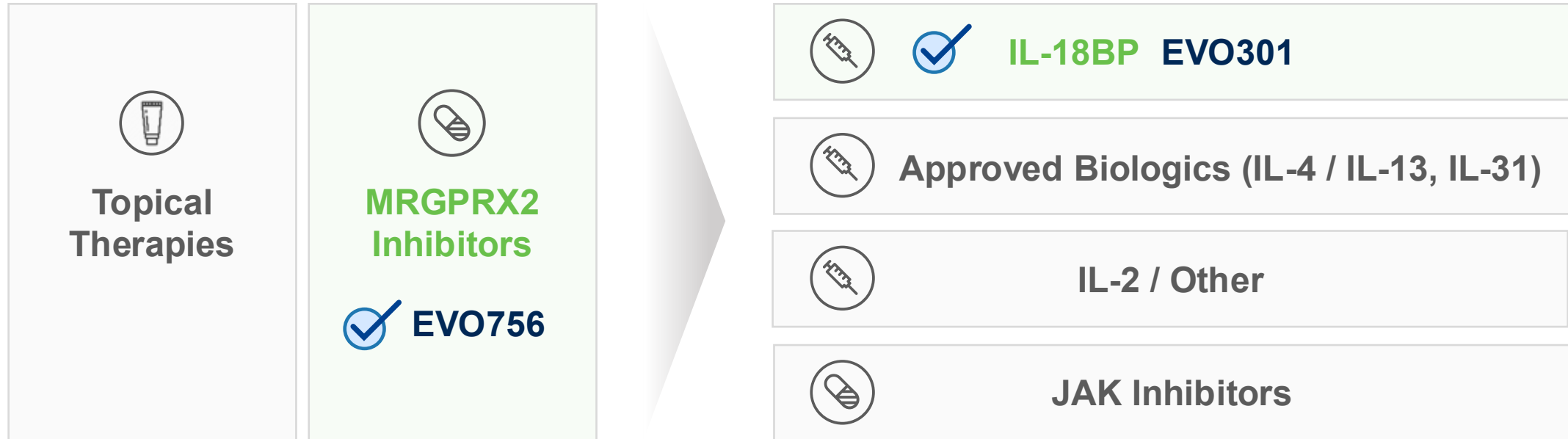


Both EBGLYSS and NEMLUVIO, Launched in 2024, Projected for \$2.5B+ Global Sales

Evommune Could Reshape the Future of AD: The Largest I&I Market

Novel MoAs Enable Treatment Across AD Patient Journey and Severity Spectrum

An AD Patient's Treatment Journey



EVO756 and EVO301 Have Synergistic Potential to Address Different Segments of the AD Landscape

- ✓ EVO756 as a first-line oral treatment post topical therapies
- ✓ EVO301 as a preferred biologic for moderate-to-severe patients

Company Overview

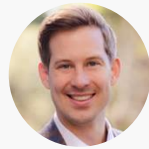
Proven and Experienced Leadership Team Has Delivered Almost 30 NDAs and BLAs



Luis Peña
Founder, President & CEO



Eugene Bauer, MD
Founder, CMO



Kyle Carver, MBA
CFO



Jeegar Patel, PhD
CSO



Greg Moss, Esq
CBO & CLO



Janice Drew, MPH
Chief of Development Operations



Daniel Burge, MD
SVP, Clinical Development



Lou Sehl, PhD
SVP, Technical Operations



Mark Jackson, MD
SVP, Clinical Development

Leadership in >25 Companies

Dermira
(Acquired by Eli Lilly for \$1.1B)



Genentech
A Member of the Roche Group



(Acquired by GlaxoSmithKline for \$2.9B)



(Acquired by LEO Pharma for \$288M)



connetics
(Acquired by Stiefel for \$930M)



Kadmon
(Acquired by Sanofi for \$1.9B)



MYOKARDIA
(Acquired by Bristol Myers Squibb for \$13.1B)



(Acquired by Eli Lilly for \$6.5B)

COHESION
(Acquired by Angiotech for ~\$50M)



Key Roles in Almost 30 NDA / BLAs

Ebglyss
(tebrikizumab-tkz)
250mg/2mL injection

REZUROCK
(belumosudil) tablets

Xolair
Omalizumab
FOR SUBCUTANEOUS USE 75 mg + 150 mg

cimzia
(certolizumab pegol)

BOTOX
OAB

eucrisa
crisaborole ointment 2%

TNKase
Tenecteplase For Fast Lytic Delivery in AMI

ACTIVASE
ALTEPLASE
A RECOMBINANT TISSUE PLASMINOGEN ACTIVATOR

Qbrexza
(glycopyrronium) cloth

Kyprolis
(carfilzomib) powder for injection

Lartuvo
(OLARATUMAB) injection 10 mg/mL

Portrazza
necitumumab injection 800 mg/50 mL vial

Picato
(ingenol mebutate) gel 0.05%, 0.05%

SORIATANE
(acitretin) Capsules

CYRAMZA
(ramucirumab)

extina
(ketconazole) Foam, 2%

CAMZYOS
(mavacamten) capsules

CLOVIQUE
Trentine Hydrochloride Capsules, USP

Coseal
Surgical Sealant

CellCept
(mycophenolate mofetil)

Liletta
hormonal-releasing intrauterine system 52mg

Kerydin
(TAVABOROLE) TOPICAL SOLUTION, 5%

veltin
(clindamycin phosphate and tretinoin) Gel 1.2%/0.025%

Vitagel

evoclin
(clindamycin phosphate) Foam, 1%

FABIOR
(tazarotene) Foam, 0.1%

Footnotes: Acquisition prices from press releases

Strong Cash Position with Multiple Clinical Milestones in 2026

✓ **Mid-stage clinical company** developing novel therapeutics for immune-mediated chronic inflammatory diseases

✓ **Two programs in Phase 2:**

- EVO756 (oral MRGPRX2 antagonist) in chronic spontaneous urticaria and atopic dermatitis, with migraine trial planned for mid-2026
- EVO301 (long-acting IL-18bp fusion protein) in atopic dermatitis

✓ **Three clinical data readouts in 2026:**

- EVO301 reported positive data in a Phase 2a in AD (Feb 2026), moving to Phase 2b with subcutaneous formulation
- EVO756 Phase 2b in CSU expected in June 2026
- EVO756 Phase 2b in AD expected in Q3 2026

✓ **Proven and experienced leadership team has played key roles in almost 30 NDAs and BLAs**

✓ **Steady cadence of new programs entering the clinic in a broad range of inflammatory diseases**

\$173 million IPO in
Nov 2025

\$125 million Private
Placement in Feb 2026

~\$307 million of
cash & investments as of
March 31, 2026

Thank You!