Corporate Presentation

July 2026

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

3 of 5

Deaths Worldwide[1]

Chronic Inflammation Destroys Lives

$90B

Annual Direct Cost[2]

Substantial Burden on the Healthcare System

>50%

I&I Therapies Fail Patients

Existing Treatment Options Have Critical Limitations

Evommune is Delivering Next Generation Therapies

Experienced Team

Distinct Mechanisms

Portfolio Approach

1. https://www.ncbi.nlm.nih.gov/books/NBK493173/. 2. Wylezinski LS, *et al*. PMID: 30979036.

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Our Mission-Driven Approach to Treating Immune-Mediated Diseases

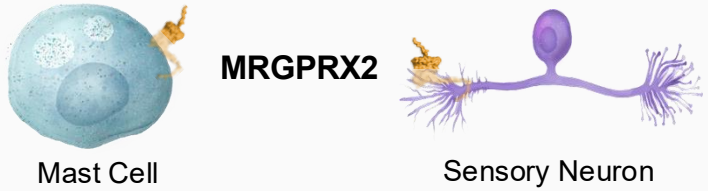
Address critical gaps in care…

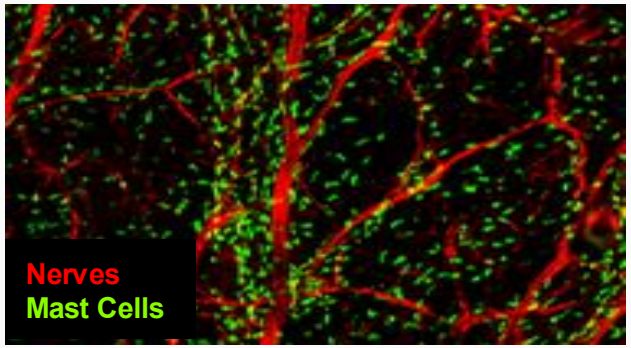
…Strategically select novel mechanisms with strong probability of success…

… Steady cadence of new programs entering the clinic

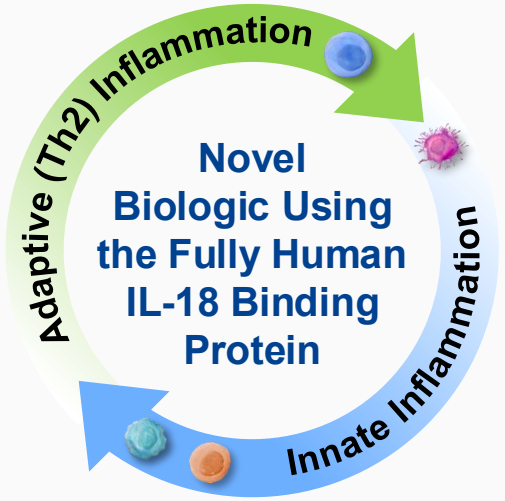
Two Phase 2 Programs with Novel Approaches to Targeting Heterogeneous Diseases

EVO756: Oral Therapy Targeting Mast Cells and Sensory Neurons





EVO301: IL-18 Blockade for Multi-Pathway Immunomodulation



Expansive Portfolio of Preclinical Programs

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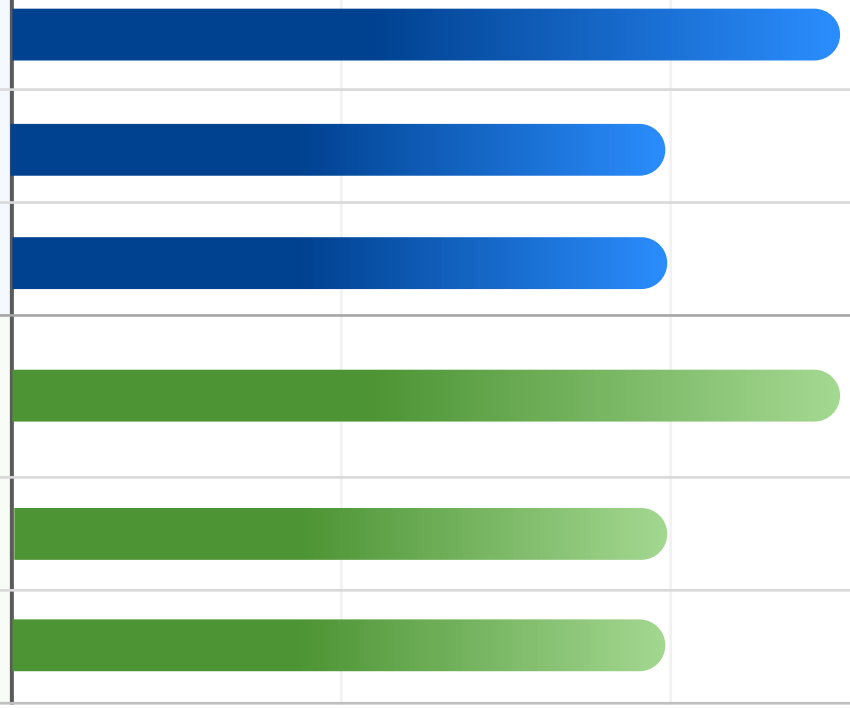
Our Inflammation Portfolio

Program / Target	Indication	Preclinical	Phase 1	Phase 2	Phase 3	Next Anticipated Milestone
EVO756 MRGPRX2	Atopic Dermatitis					• Phase 2b top-line data (Q3 2026)
	Migraine					• Phase 2b trial initiation (mid-2026); top-line data (2027)
	Other Indications					• Phase 2 trial planning underway
EVO301 IL-18	Atopic Dermatitis					• Positive Phase 2a POC: Full data to be presented at an upcoming medical meeting • Phase 2b trial planning underway
	Ulcerative Colitis					• Phase 2 trial planning underway
	Other Indications					• Phase 2 trial planning underway



Advancing Multiple Preclinical Programs Toward Clinical Proof-of-Concept

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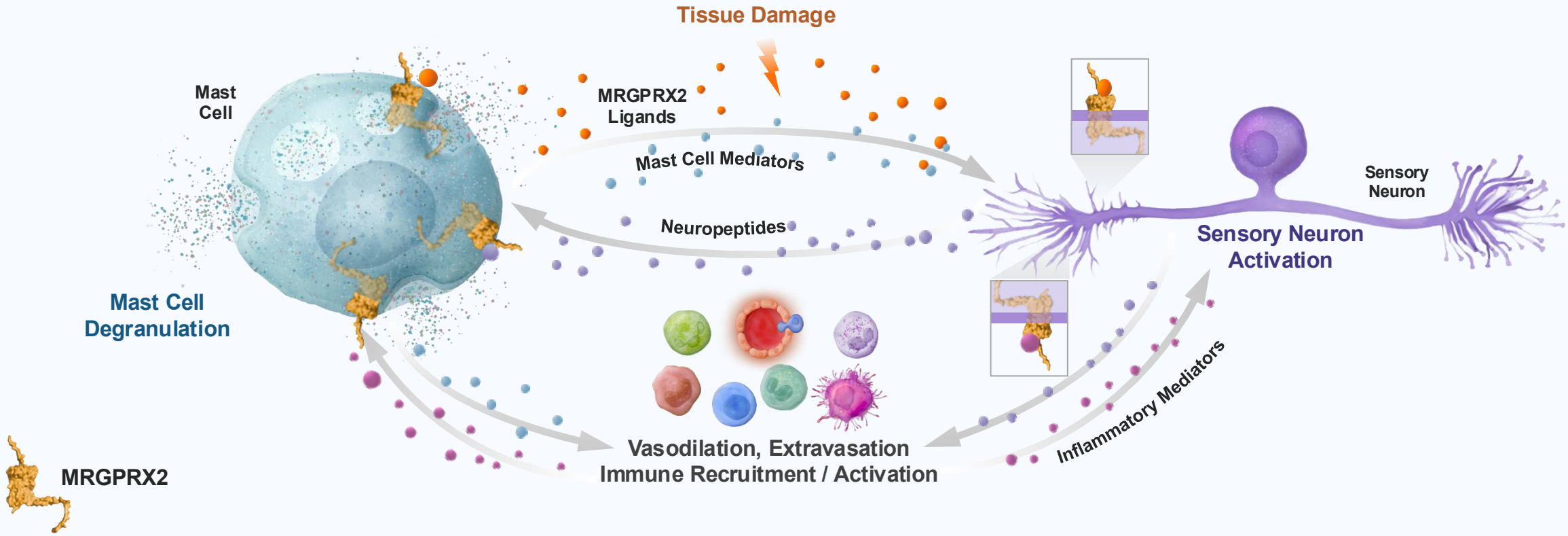
EVO756: Oral MRGPRX2 Antagonist

First and Best-in-Class Dual Mechanism Modulates Both Peripheral Sensory Neurons and Mast Cells

MRGPRX2 in Neuroinflammation and Mast Cell Activation



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

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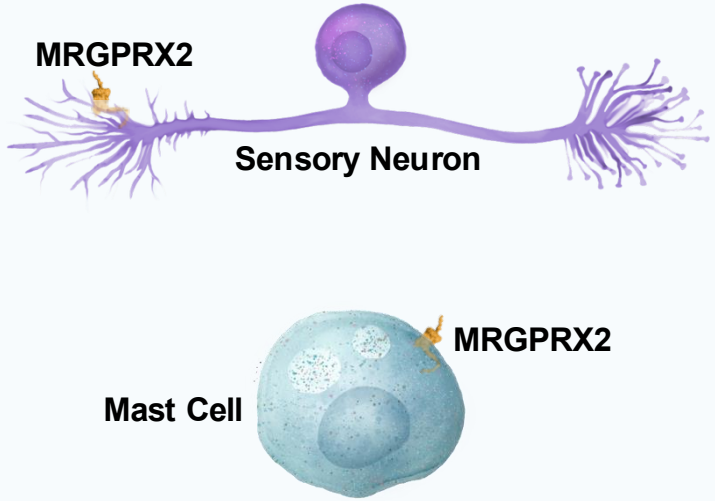
EVO756: Broad Spectrum Oral Anti-Inflammatory Potential

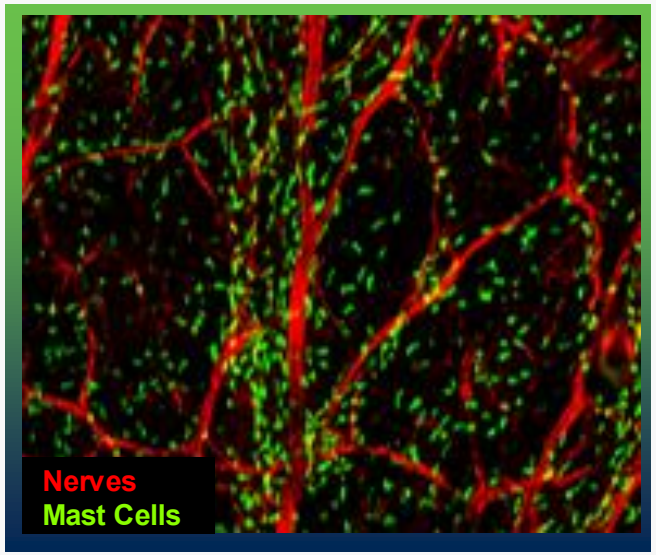
Potential First-Line Oral Across Several Specialties

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

MRGPRX2 Expressed on Both Sensory Neurons and Mast Cells



Sensory Neurons and Mast Cells Are Found in Close Proximity



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EVO756: Differentiated Mechanism Across Multi-Billion-Dollar Markets

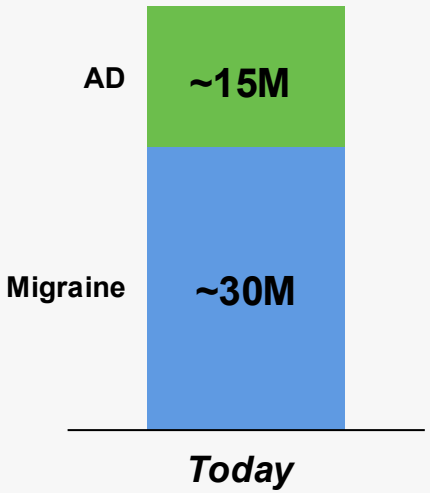
Sequenced Expansion Across Underserved, Blockbuster Markets

Initial Target Population

Selected for unmet need, market size, and defined regulatory path

~45M

Eligible Patients

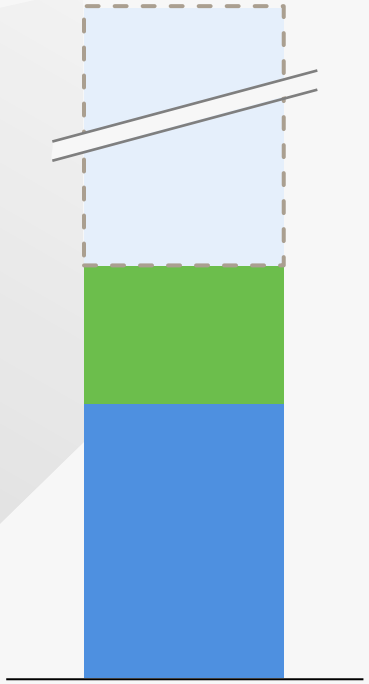


Future Target Population

>95M Patients

Expanding into High-Potential Indications Where Neuroinflammation Drives Disease

>3M Chronic Pruritus	**~25M** Asthma	**~10M** IC
~1M CIndU	**~7M** Food Allergy	**~7M** IBS-D



G7 prevalence estimates. CIndU = chronic inducible urticaria; AD = atopic dermatitis, IC = interstitial cystitis, IBS-D = diarrhea-predominant irritable bowel syndrome. Sources: Decision Resources DL&F; https://pmc.ncbi.nlm.nih.gov/articles/PMC9677261/, Novartis Investor Presentation (2026) treatment and eligibility figures per multiple published references. Future market estimates to be confirmed. LCM indications sized by moderate-to-severe populations.

EVO756 Clinical Development Summary

Trial	Phase 1 Proof-of-Concept	Phase 2	Phase 2b	Phase 2b
N	132	30	~120	~330
Indication	Healthy Volunteers	CIndU	AD	Migraine
Key Takeaways	• Well-tolerated across all doses • Clear target engagement in skin challenge • Concentration dose proportional and linear	• Well-tolerated across all doses • Complete responses as early as week 1 • POC achieved after just 4 weeks of dosing	**Top-line Data Expected Q3 2026**	**Trial Initiation Mid-2026**

CIndU = Chronic inducible urticaria; AD = Atopic dermatitis

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

Inclusion of Icatibant Skin Challenge Demonstrated EVO756 Target Engagement in Humans

PART A: SAD Dosing

N = 55 (41 active / 14 placebo)

- **Cohort A7** 500 mg
- **Cohort A6** 240 mg
- **Cohort A5** 100 mg
- **Cohort A4** 30 mg
- **Cohort A3** 10 mg
- **Cohort A2** 3 mg
- **Cohort A1** 1 mg

PART B: MAD Dosing

N = 77 (58 active / 19 placebo)

- **Cohort B5** 500 mg QD
- **Cohort B4** 240 mg BID
- **Cohort B3** 100 mg BID
- **Cohort B2** 30 mg BID
- **Cohort B1** 10 mg BID

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

ECG = Electrocardiogram

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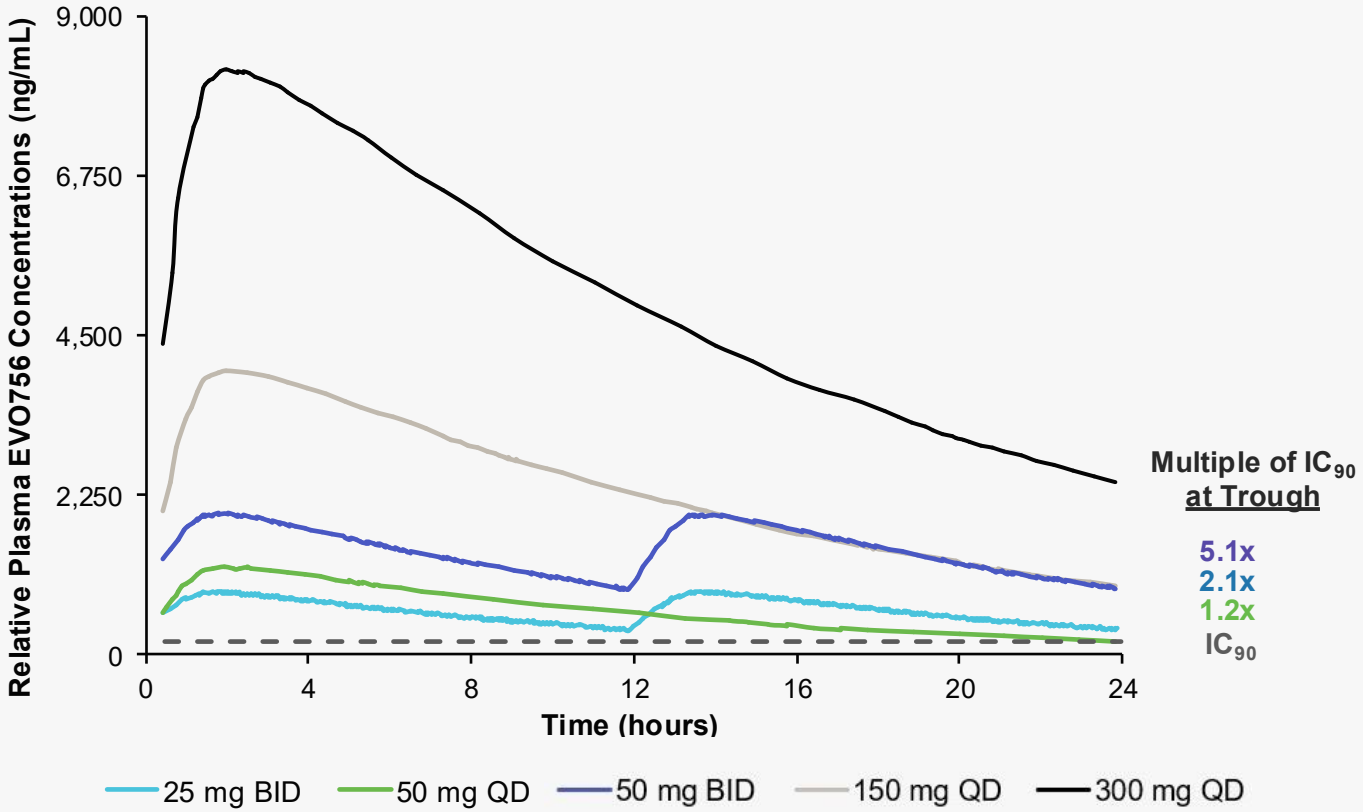
EVO756 PK Supports Full Target Coverage as Low as 25mg BID

Day 10 Median Concentrations



Dose-Proportional

- Linear concentration-dose relationship across SAD and MAD cohorts

Half-Life: 8-12 Hours

- Tmax 1–4 hours · supported both QD and BID dosing

High Skin Penetration

- ~70% tissue:plasma ratio in human skin 2-fold accumulation
- 17% free drug ratio

IC_{90} (mast cells) = 180 ng/mL

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Positive Phase 2 CIndU Data Validated EVO756 Activity

Robust Efficacy at Week 4 Competitive with Omalizumab and Barzolvolimab

30% **Complete Response**

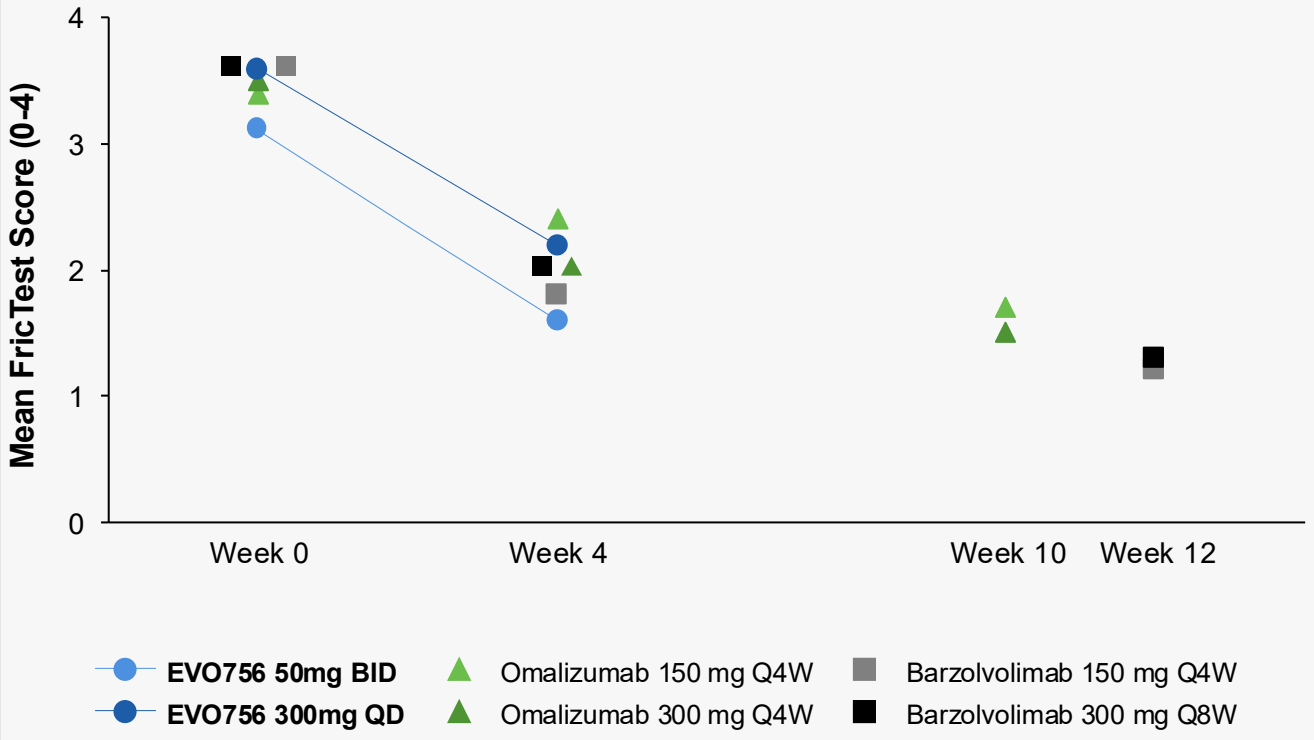
at Week 4 across both doses
(8 of 27 patients · FricTest score = 0)

70% **FricTest Response**

≥1-point improvement at Week 4
(19 of 27 patients)

- Symptomatic dermographism (N=30)
- Open-label, within-patient controlled
- 50 mg BID and 300 mg QD over 4 weeks
- FricTest: standardized friction provocation test (0–4)

Clinical Improvements Over Time



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

Evommune clinical data; ALT = alanine aminotransferase; AST = aspartate aminotransferase; 1. The two subjects in the 300 mg QD cohort with AEs of increased ALT and AST had asymptomatic transaminase elevations that were greater than ten times the upper limit of normal at four weeks, which were notpresent at baseline, week 1 or week 2 and later returned to baseline. Other liver tests, including bilirubin and alkaline phosphatase were within normal limits. Both of these subjects had confounding factors that may have contributed to these elevations.

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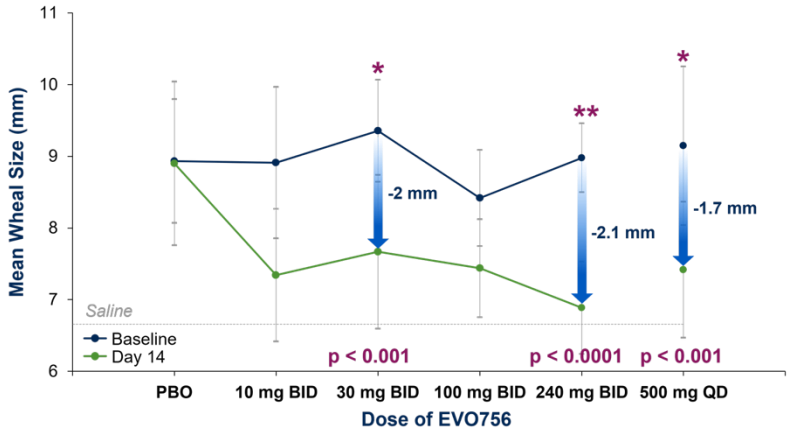
EVO756 Phase 2b Dose Selection Rationale

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 IC_{90} 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

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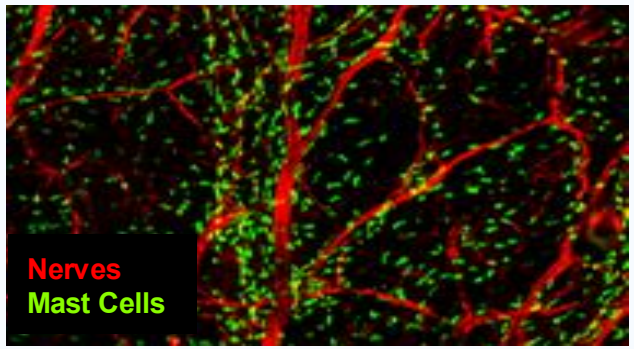
EVO756 in Atopic Dermatitis (AD)

MRGPRX2 is Only Dual MOA: Targeting Inflammatory Lesions and Neuroinflammation

Expect Benefit on Neuroinflammation and Mast Cell Aspects of AD



Strong Scientific Rationale for EVO756 in AD

Neuroinflammatory and mast cell 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

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AD Still Has No First-Line Oral Option

No Current Oral Therapy Combines Lesion Control, Itch Relief, and Tolerability

~15M

Targeted-Therapy Eligible Patients

EVO756 – Promising First-Line Candidate Designed to Deliver on All Three:

1

Oral Dosing

Preferred ROA

2

Differentiated Activity

MOA Targets Lesions + Itch

3

Well-Tolerated

Profile Supports Broad Use

G7 prevalence estimates. Sources: Decision Resources DL&F; treatment and eligibility figures per published references. Future market estimates to be confirmed.

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

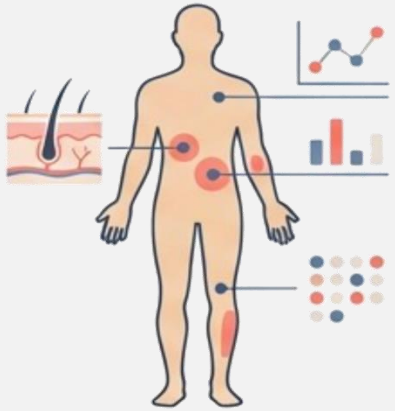
MRGPRX2 Signature Generation

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

Computational Engine

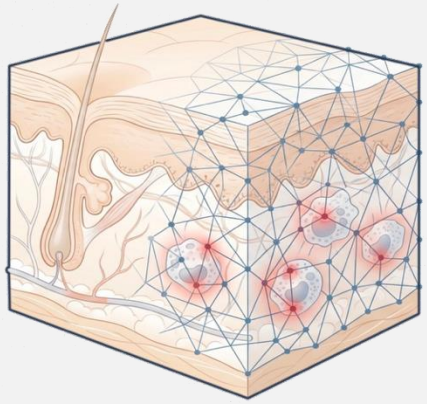
Integration of MRGPRX2 signature, 24 RNAseq datasets, >2,000 of human skin biopsies

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

In collaboration with CytoReason

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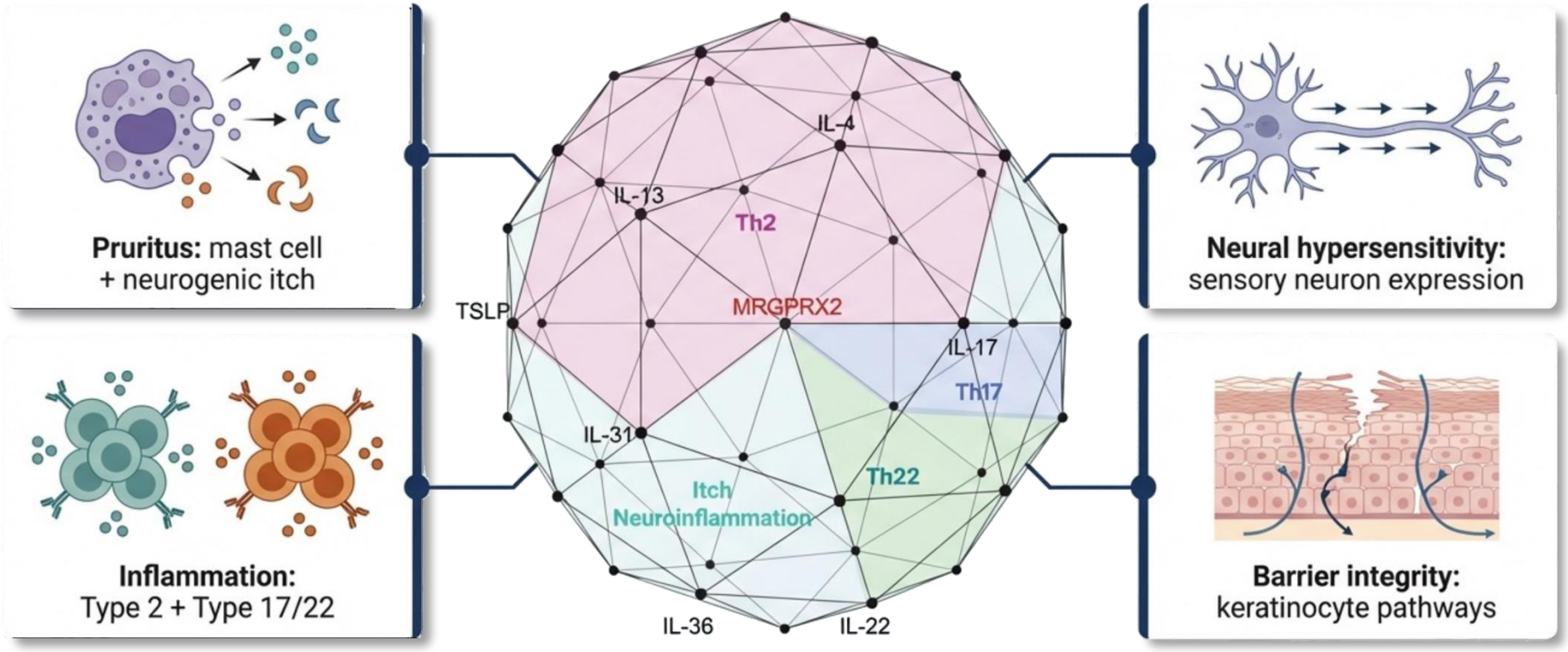
Predictive Modeling Reveals MRGPRX2 as Core to AD Pathophysiology

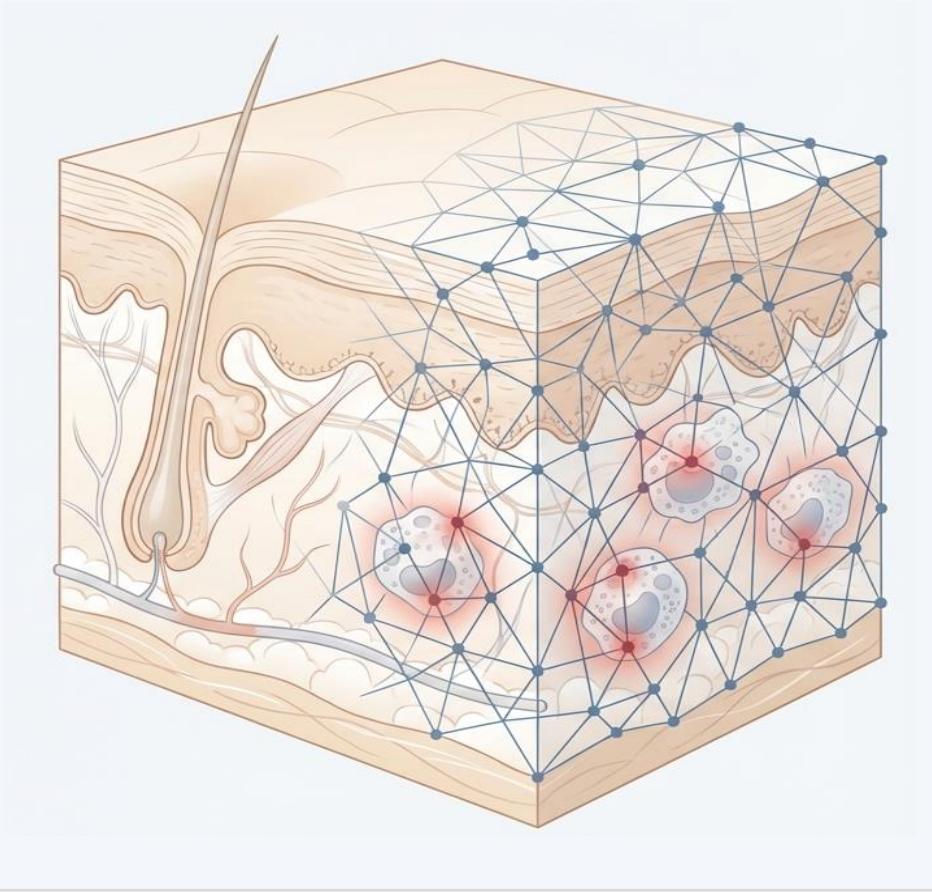


Broad reach may enable EVO756 to potentially outperform IL-4/-13–restricted approaches

Magenta = Th2 signatures; **Teal** = Itch / Neuroinflammation signatures, **Green** = Th22 signatures, **Blue** = Th17 signatures

In collaboration with CytoReason evommune

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

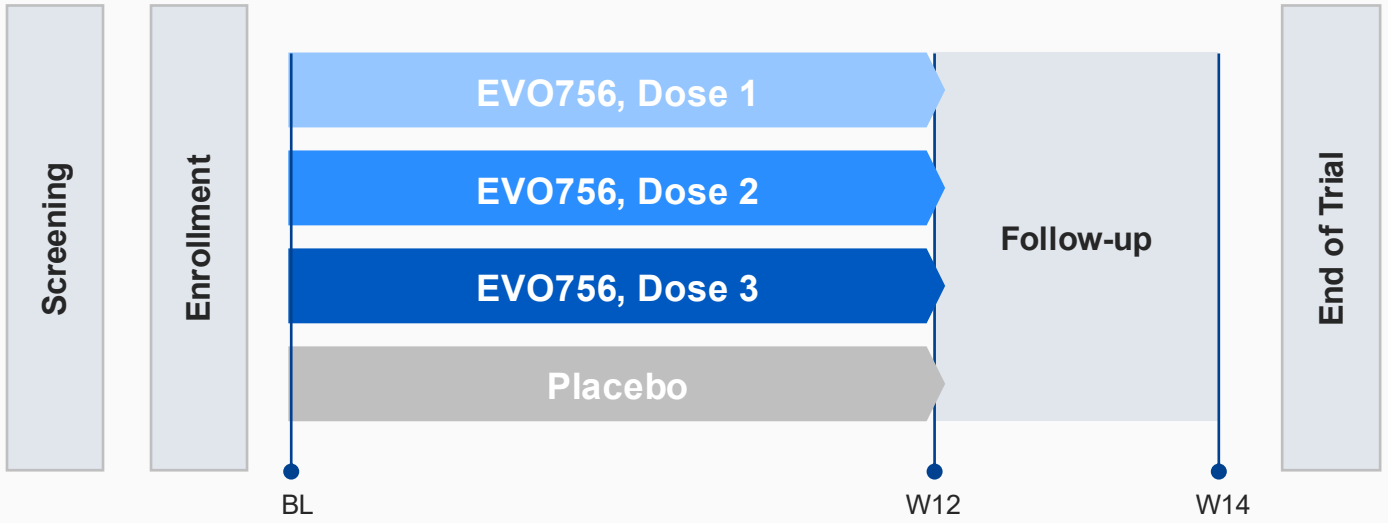
In collaboration with CytoReason

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

- % change in EASI from BL 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

EASI = Eczema Area and Severity Index; vIGA = Validated Investigator Global Assessment; Pruritus-NRS = Pruritus Numerical Rating Scale; BSA = Body Surface Area; BL = Baseline. Trial identifier: NCT07150845

EVO756 in Migraine

MRGPRX2 is a Novel Migraine Target with Potential to Address Neuroinflammatory and Mast Cell Drivers of Migraine

Strong Scientific Rationale for EVO756 in Migraine

Disease-Relevant Expression	MRGPRX2 is expressed in human trigeminal neurons and meningeal mast cells
Preclinical Validation	*in vivo* headache models support pathogenic role for MRGPRX2
Translational Insights	Multiple MRGPRX2 ligands induce migraine in humans
Clinical Proof-of-Concept	mAb inhibition of MRGPRX2 ligand (PACAP) shows clinical benefit

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EVO756: Targeting Major Unmet Need in Migraine Prevention

~30M

G7 Patients Eligible for Preventative Therapy

EVO756 – Defining the Next Wave of Preventatives

1

Oral Dosing

Preferred ROA

2

Novel Dual Mechanism

Targets neurons + mast cells;
Blocks 3 migraine triggers
(PACAP, VIP, Substance P)

3

First-Line Potential

AHS now recommends targeted therapies first-line

G7 eligible patients estimated. Sources: Clarivate "Migraine: Disease Landscape and Forecast" (2026), AHS guidelines for migraine prevention eligibility, Buse *et al*. (2024), Silberstein *et al*. (2015), Cohen *et al*. (2024), Coppola *et al*. (2025), Sakai *et al*. (2022).

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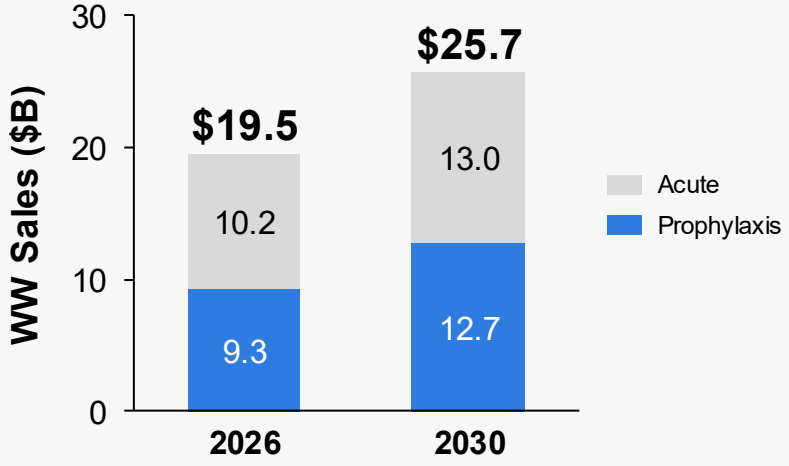
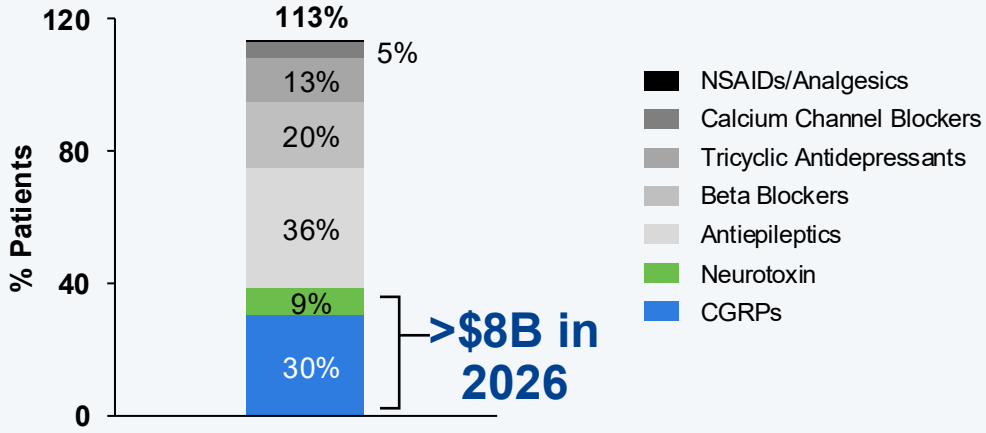
High Demand for Preventative Migraine Therapy

Prevention Drives ~50% of $25B Migraine Market



>75M People Living with Migraine Worldwide

Most Patients Remain on Legacy Preventives — Targeted Therapies Drive Sales



~30M Global Patients Eligible for Preventative Therapy

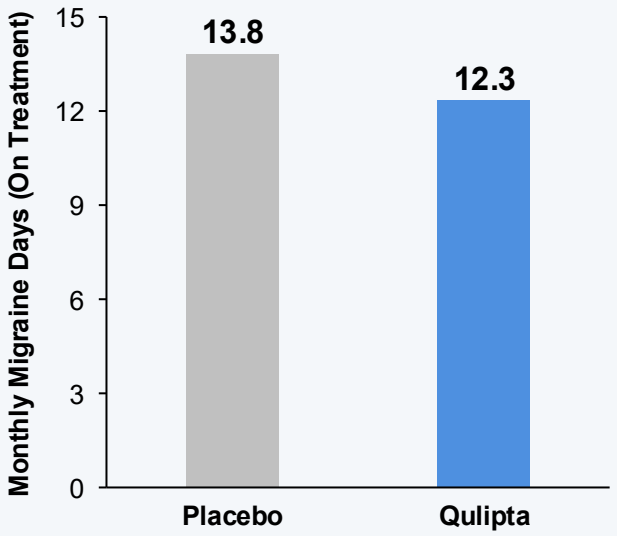
Sources: Clarivate "Migraine: Disease Landscape and Forecast" (2026), AHS guidelines for migraine prevention eligibility, Buse *et al.* (2024), Silberstein *et al.* (2015), Cohen *et al.* (2024), Coppola *et al.* (2025), Sakai *et al.* (2022). Note: Sales represent 7 Major Markets (US, EU5, Japan); Percent of patients by therapy type exceeds 100% due to co-prescribing; % patient numbers by therapy reflect US patient breakdown.

Innovation in Migraine Prevention Has Been Limited to CGRPs

Preventive Innovation has Clustered Around a Single Target (CGRP)

Advanced Treatments	Class	Route of Administration
QULIPTA (atogepant) tablets	CGRP	Oral QD
Nurtec ODT		Oral EOD
vyepti		IV
Emgality		SC
aimovig erenumab		SC
AJOVY		SC
BOTOX onabotulinumtoxinA	Neurotoxin	IM

Migraine Burden Persists Despite Oral CGRP Therapy



- Many chronic patients still experience >12 monthly migraine days on treatment

High Unmet Need in Migraine Prevention

Limited Therapeutic Diversity

- Only CGRP inhibitors and neurotoxin

Inadequate Efficacy

- ~45% of patients do not achieve 50% improvement

Tolerability Challenges Remain

- CGRPs associated with constipation, hypertension, Raynaud's, nausea, allergic and injection site reactions

Source: Qulipta Prescribing Information from pivotal trial in chronic migraine patients.

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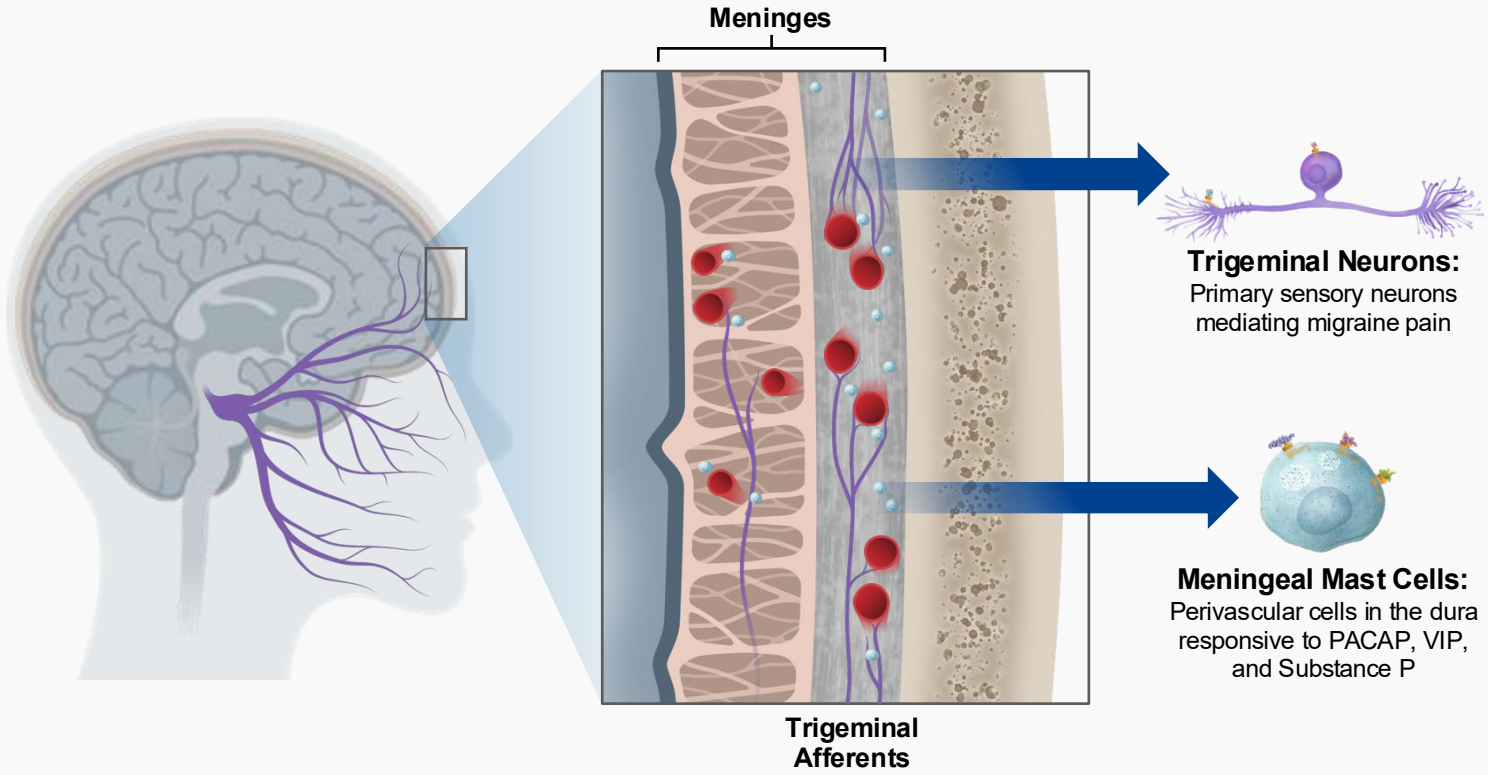
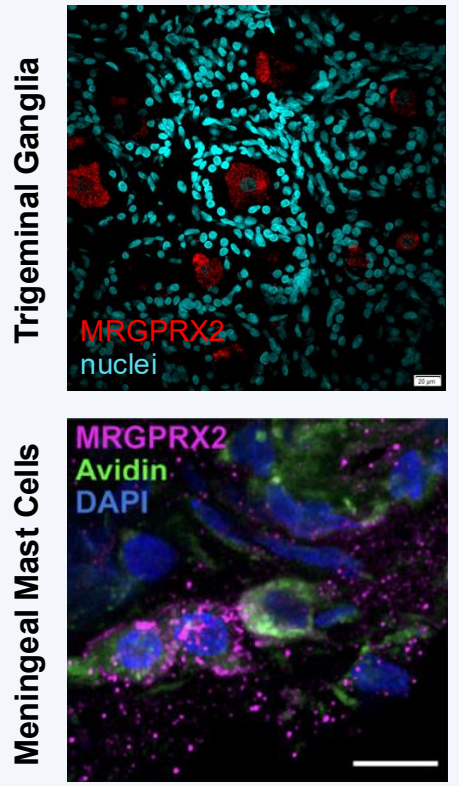
MRGPRX2: Positioned to Address Neuronal and Mast Cell Drivers of Migraine

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



Expression Confirmed in Disease-Relevant Tissues



Source: Evommune internal data (trigeminal neurons; *in situ* hybridization on human tissue samples), PMID: 40712576 (meningeal mast cells)

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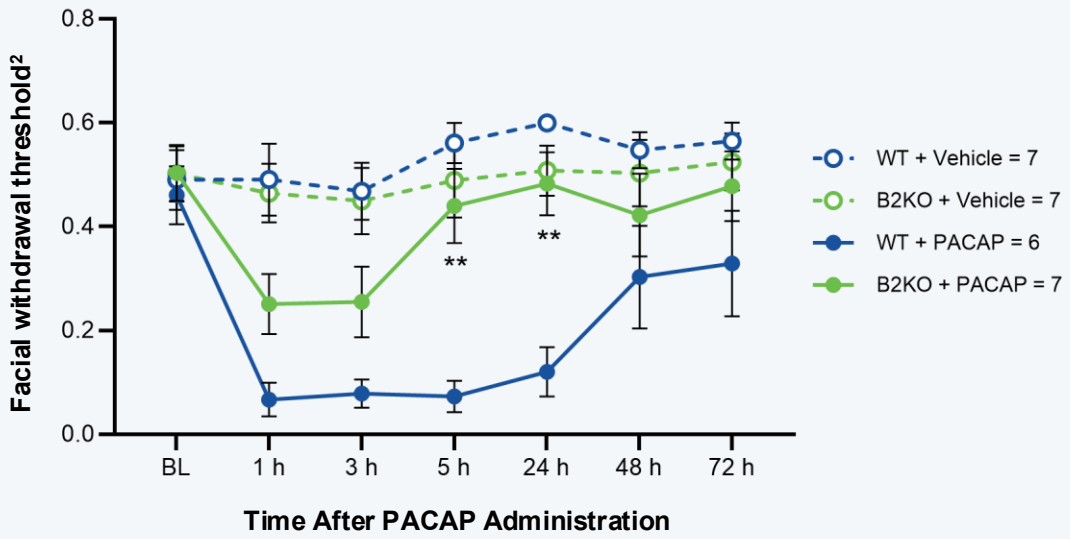
PACAP Triggers Migraine via MRGPRX2[1] as Primary Receptor *in vivo*

MRGPRX2 Ligand PACAP Induces Headache *in vivo*

1. PACAP injected directly to meninges of wild type and knockout models
2. Facial withdrawal threshold used as functional pain readout

Knockout of MRGPRX2[1] Reduced PACAP-Induced Migraine Symptoms

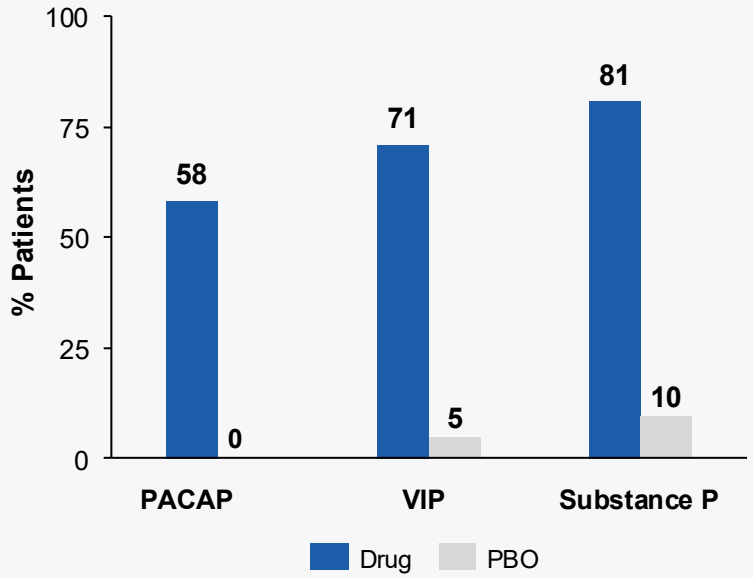


- *in vivo* data support functional role of MRGPRX2[1] signaling in migraine

1. Mrgprb2 is rodent homologue. 2. In grams, measured via Orbital von Frey assessment. Source: Internal Evommune data. Confirms data published in PMID: 37516794.

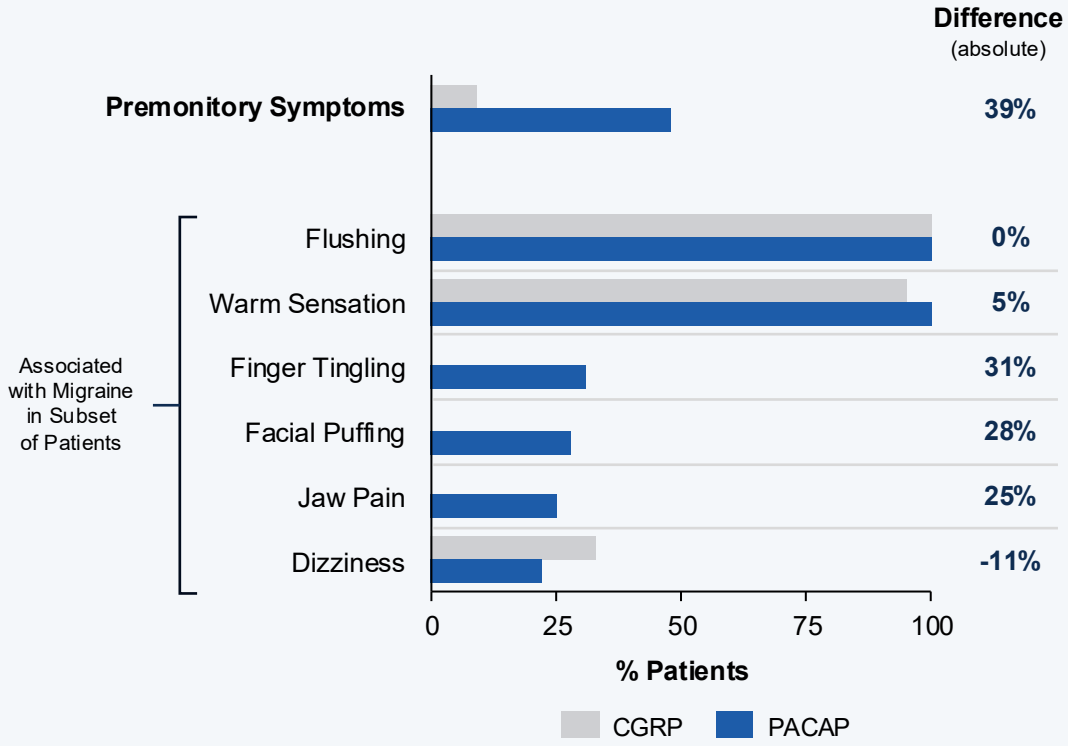
MRGPRX2 Ligands Induce Migraine in Humans
PACAP, VIP, Substance P are Known MRGPRX2 Agonists

PACAP, VIP, Substance P Infusion all Induce Migraine-Like Headache in Migraineurs



- Similar to CGRP, which induces migraine in ~2/3 patients

PACAP-Induced Headaches More Closely Recapitulate Migraine Features vs. CGRP



Note: Data shown from PACAP-38; PACAP-27 also induces migraine. Sources: Adapted from PMID19052139, PMID34357396, PMID37009867, Al-Khazali *et al.*, (2026). Note that there have been multiple studies inducing headache/migraine with ligands and experimental paradigms / esults differed. Data shown represent cumulative observations pooled across multiple trials and cannot support definitive conclusions.

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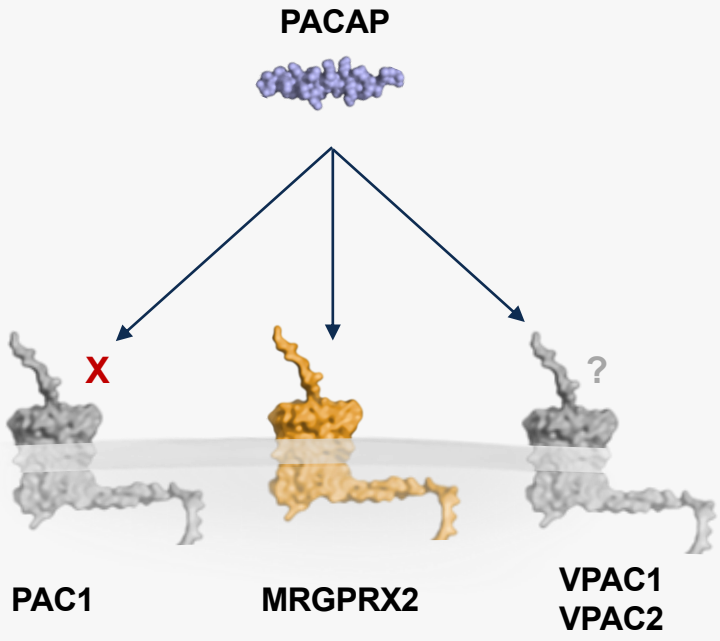
PACAP Likely Induces Migraine in Humans Through MRGPRX2 as Primary Receptor

PACAP Impact in Migraine Is Primarily through MRGPRX2



PACAP Receptor	Relevant Tissue Expression	Preclinical Evidence?	Clinical Validation In Migraine?
PAC1	**Neurons**[1]	Limited	✗
VPAC1 **VPAC2**	**Cranial vessels** Neurons (?)	Limited	*Not evaluated*
MRGPRX2	**Mast Cells** **Sensory Neurons**	✓	**TBD**

1. Trigeminal sensory neurons, brainstem pain circuits, hypothalamus, cortex, thalamus. Note: In addition to MRGPRX2 PACAP binds PAC1, VPAC1, VPAC2, but PAC1 inhibition (Amgen's AMG301: PAC1 blocking mAb.) does not show therapeutic benefit in migraine. Sources: PMID: 33231489, PMID: 39085771, PMID: 37706270.

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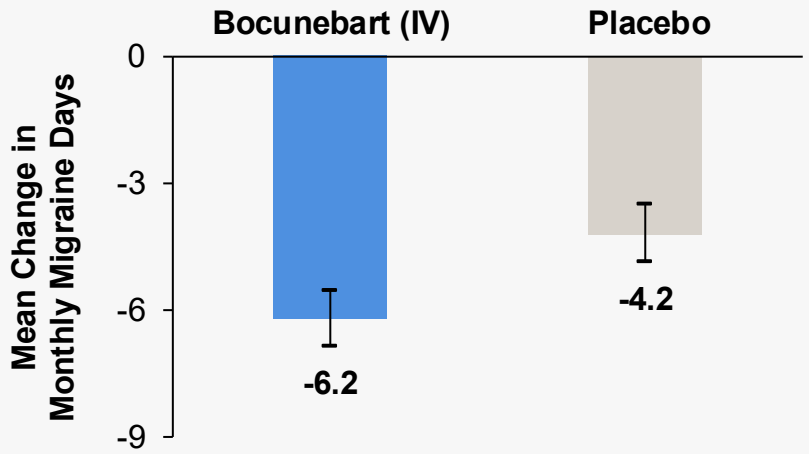
Inhibition of MRGPRX2 Ligand PACAP Achieves CGRP-Like Efficacy in Migraine Prophylaxis

Lundbeck's Bocunebart Reduced Monthly Migraine Days by ~2



- Magnitude of benefit consistent with marketed CGRP inhibitors

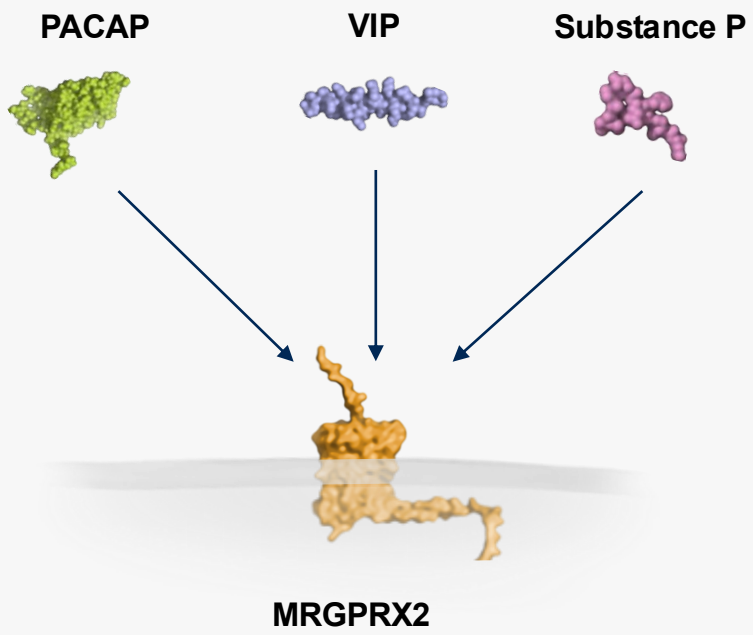
Second Neuropeptide Axis Validated in Migraine Prevention

- 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

Note that PACAP also binds PAC1, VPAC1, VPAC2, but PAC1 inhibition does not show therapeutic benefit in migraine.

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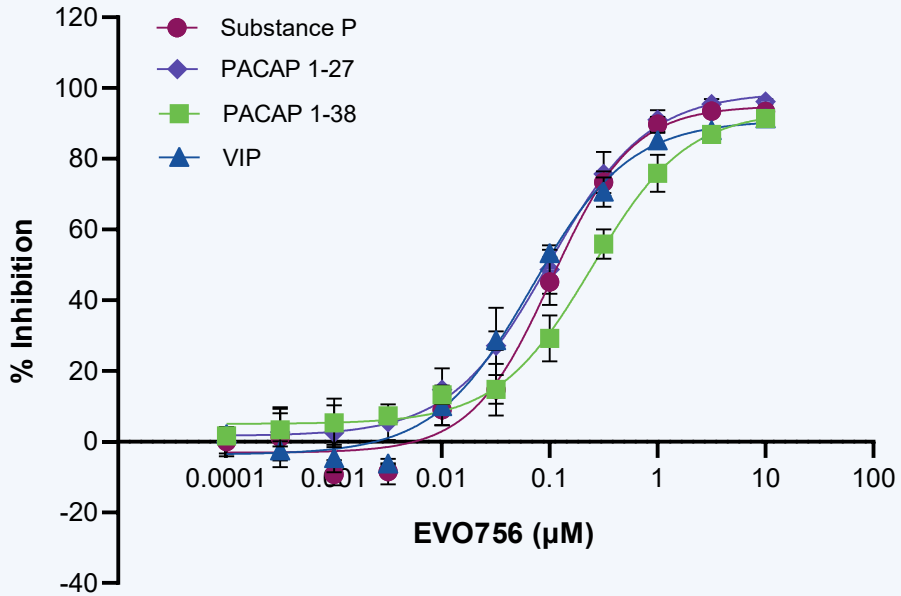
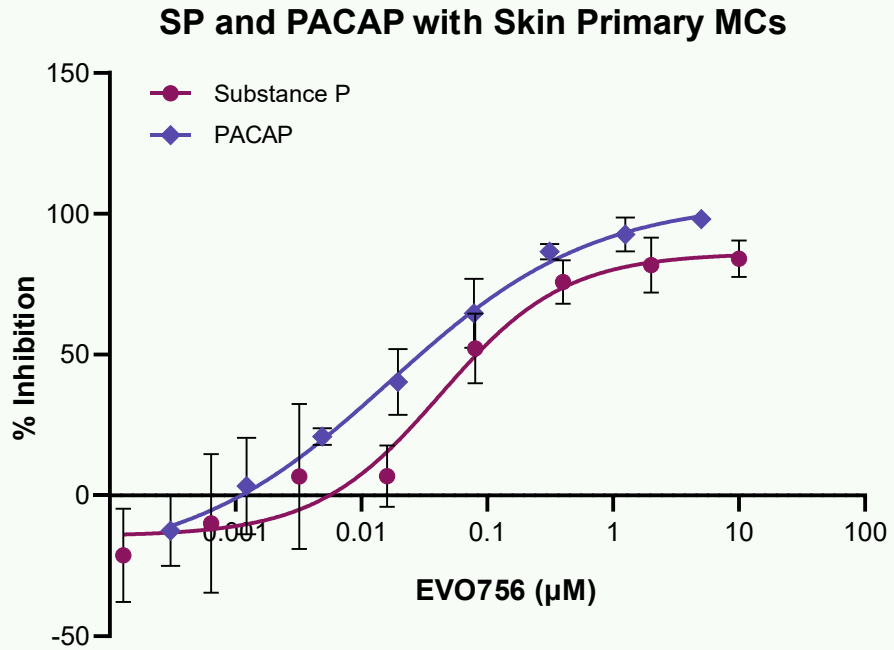
EVO756 Potently (low nM) Inhibits PACAP, Substance P and VIP-Induced MRGPRX2 Activation *in vitro*

EVO756 Inhibits Migraine Relevant Endogenous Ligands in X2-CHO Cells



EVO756 Inhibits PACAP and SP-Induced Primary Human Mast Cell Activation *in vitro*



All assays (agonists) run at ≥EC_{80}. Primary skin MC: CD63; similar experiment run on primary skin mast cells with CD107a with similar results.

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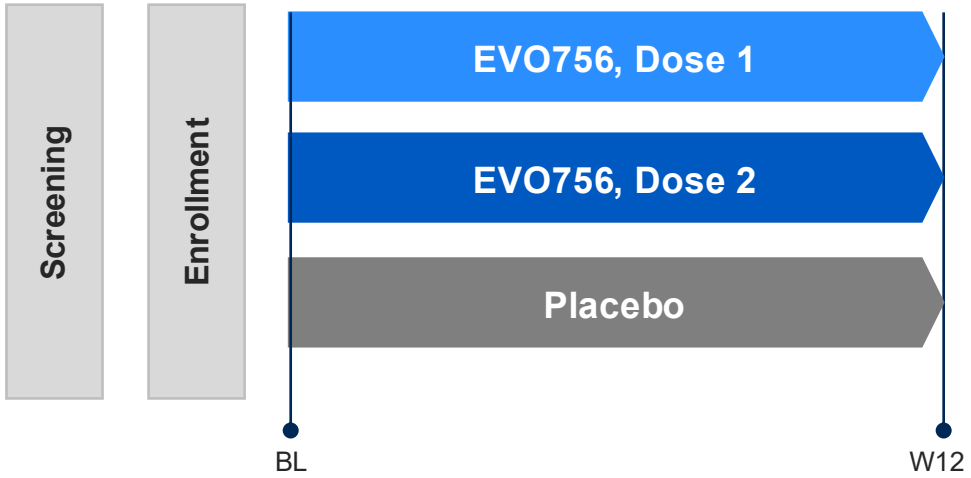
Planned Phase 2b Dose-Ranging Trial in Migraine Prophylaxis

Initiation Expected Mid-2026, Top-line Data Expected in 2027

Adults with Refractory Migraine ≥6 Days/Month

(N ≈ 330)

Randomized, Double-Blind, Placebo-Controlled Trial



Exploring daily doses up to100 mg

Primary Endpoint

- Mean CFB in MMD

Key Secondary Endpoints

- ≥50%, ≥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

BL = Baseline; CFB = change from baseline; MMD = monthly migraine days; MHD = monthly headache days; QoL = quality of life

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EVO301: Best-in-Class IL-18BP Fusion Protein

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

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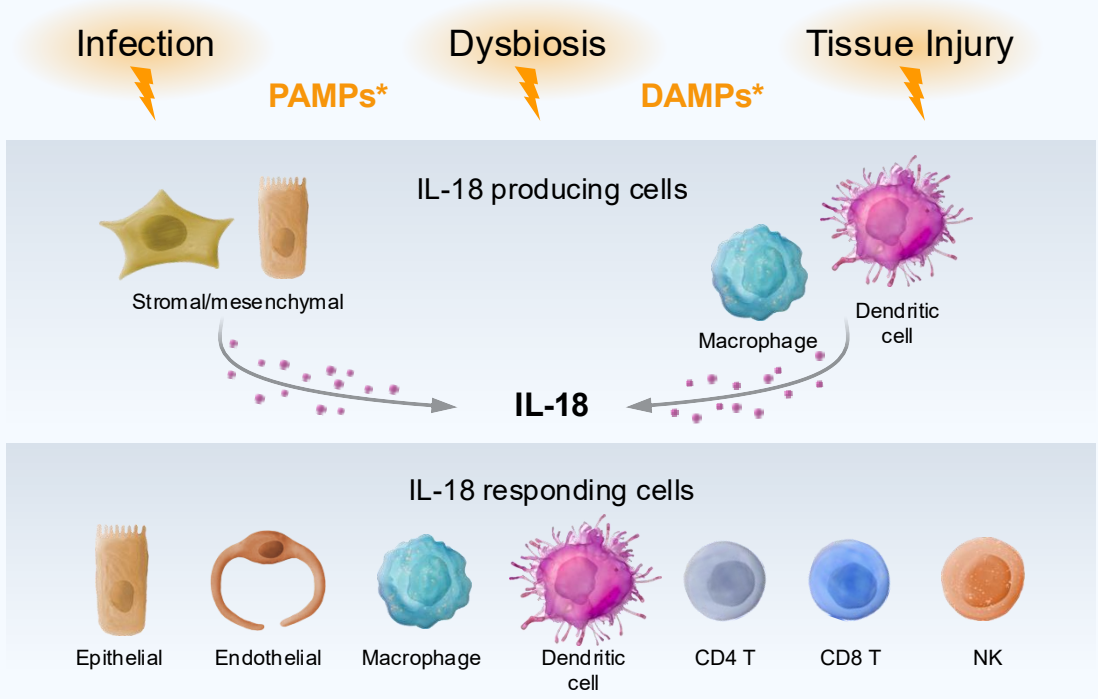
IL-18 Immune Rebalancing: Modulate Innate and Adaptive Inflammation for Potential Disease Remission

Involved in Innate and Adaptive Immune Processes



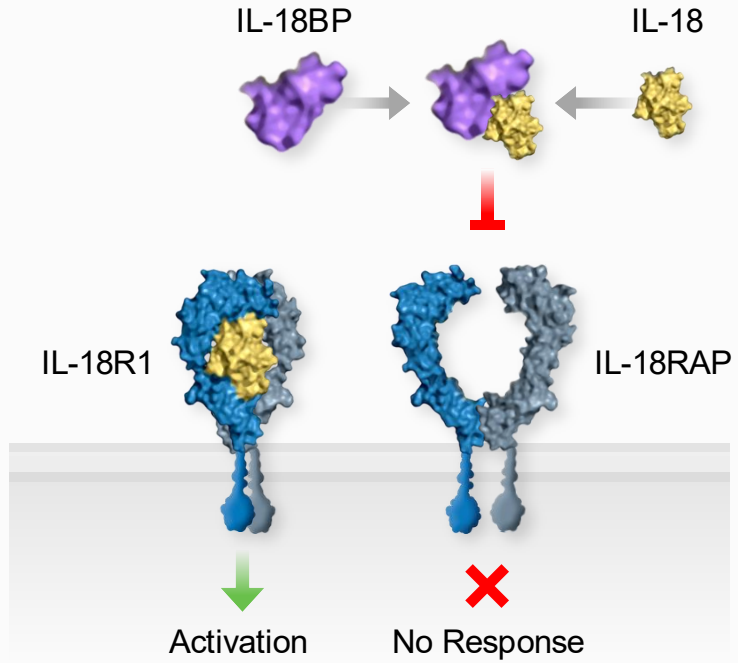
Tissue Pathophysiology

Th1, 2, 17 Differentiation | Inflammatory Infiltrates | Inflammatory Cytokines | Autoimmunity | Pathogen Clearance | Angiogenesis | Epithelial Barrier

Clinical Manifestations

Chronic Inflammation | Barrier Dysfunction | Infection | Tissue Damage

IL-18BP Therapeutic Approach



PAMPs: Pathogen-associated molecular patterns; DAMPs: Damage-associated molecular patterns

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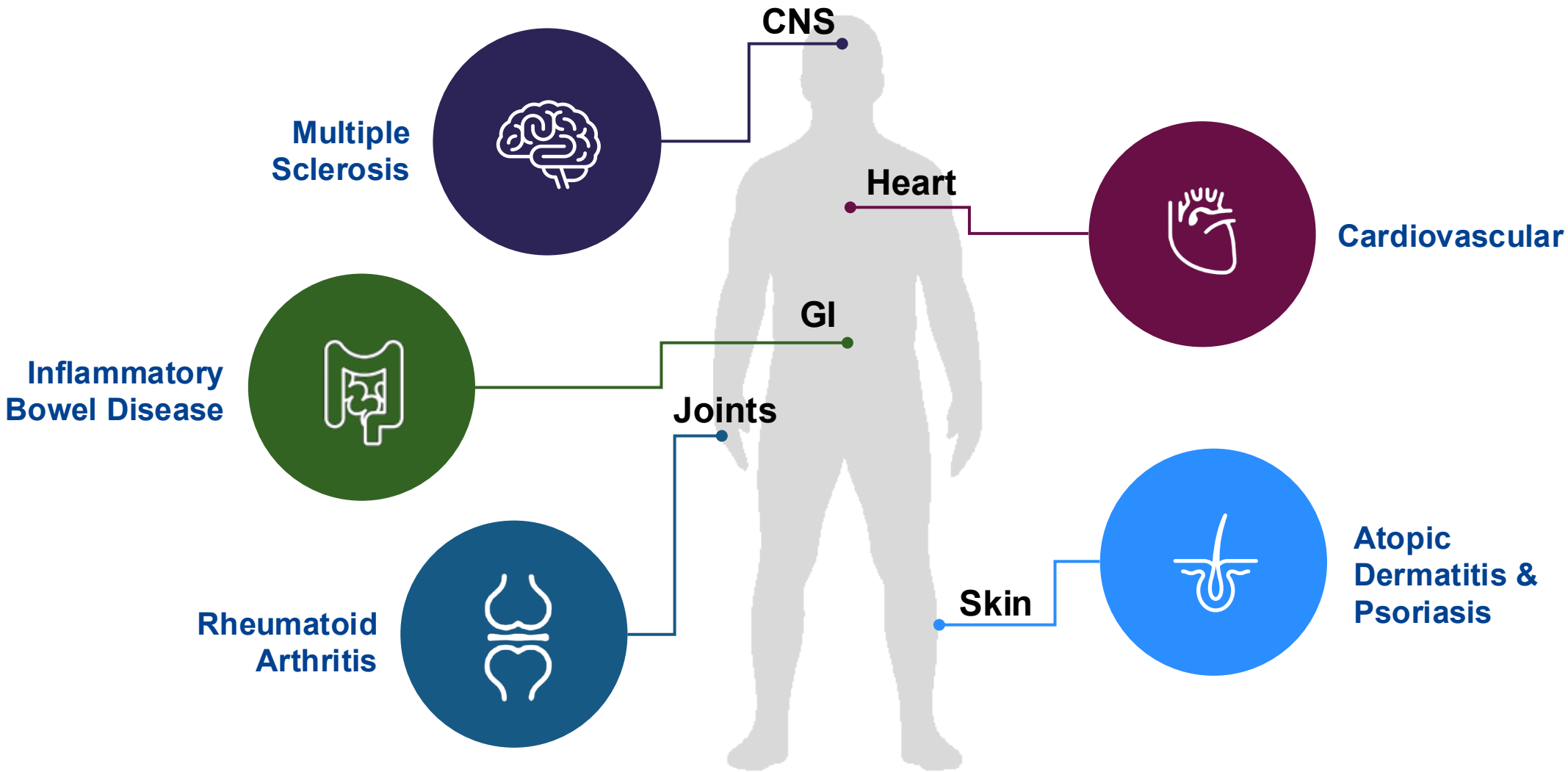
IL-18's Broad Footprint Across Inflammatory and Immune Disease



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

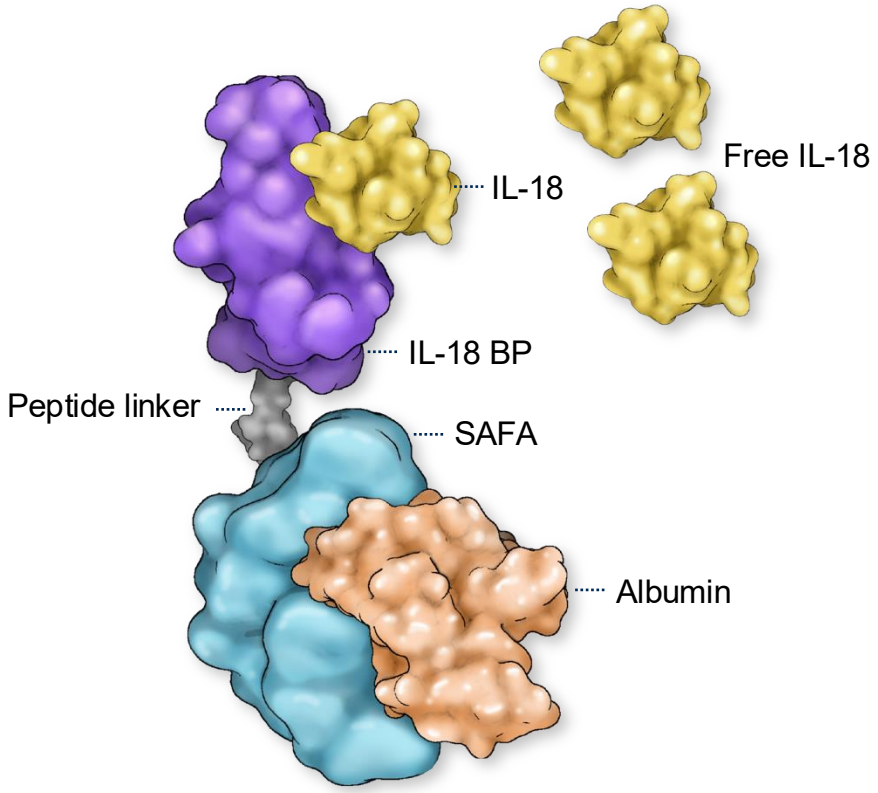
SAFA and IL-18BP Fused Via Peptide Linker for Extended Neutralization of IL-18 Activity

SAFAbody™ Platform Technology

- $T_{½}$ 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



SAFA - Anti-Serum Albumin Fab-Associated. HSA – Human Serum Albumin SAFABODY™ is a trademark of AprilBio Co., Ltd.

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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

Green = impacts biological pathway; **Red** = negative effect.

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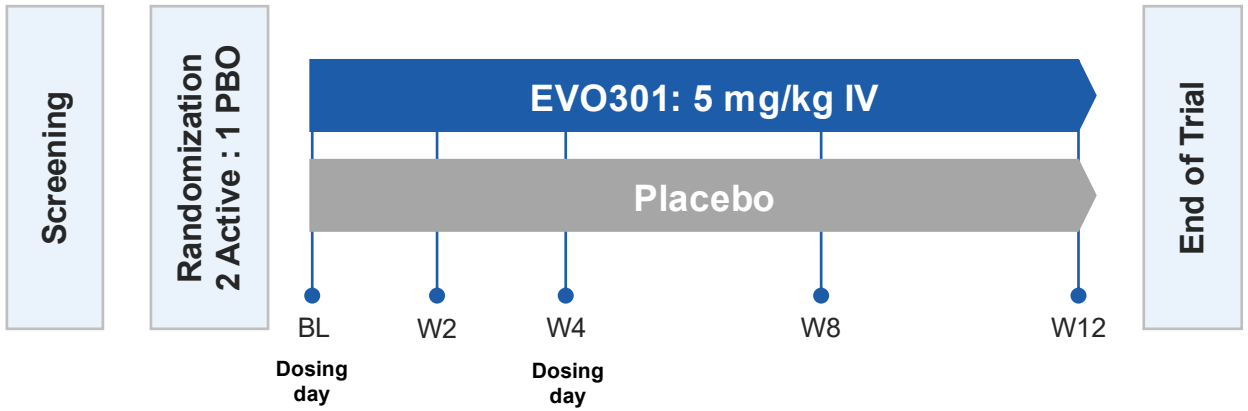
EVO301 Phase 2a Proof of Concept Trial Design

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

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



AD Population

- EASI ≥16
- vIGA ≥3
- BSA ≥10%

Primary Endpoint

- Percent change from EASI at Week 12 (Bayesian)

Pharmacokinetics

Target Engagement

EASI = Eczema Area and Severity Index; vIGA = Validated Investigator Global Assessment; BSA = Body Surface Area; BL = Baseline; PBO = Placebo. Trial identifier: NCT06723405

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

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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/m^2)	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

EASI: Eczema Area and Severity Index, SD: standard deviation, HPD: high posterior density

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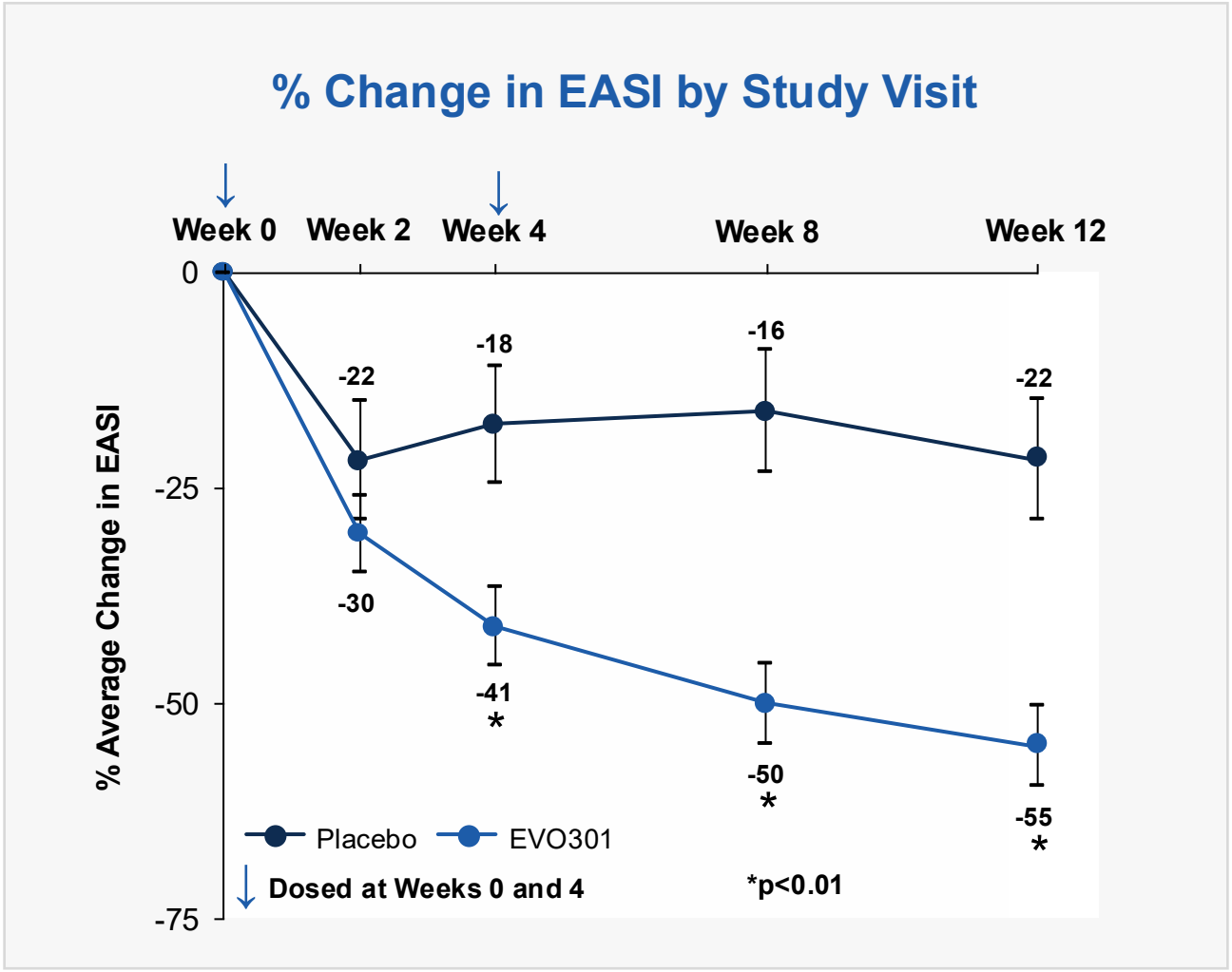
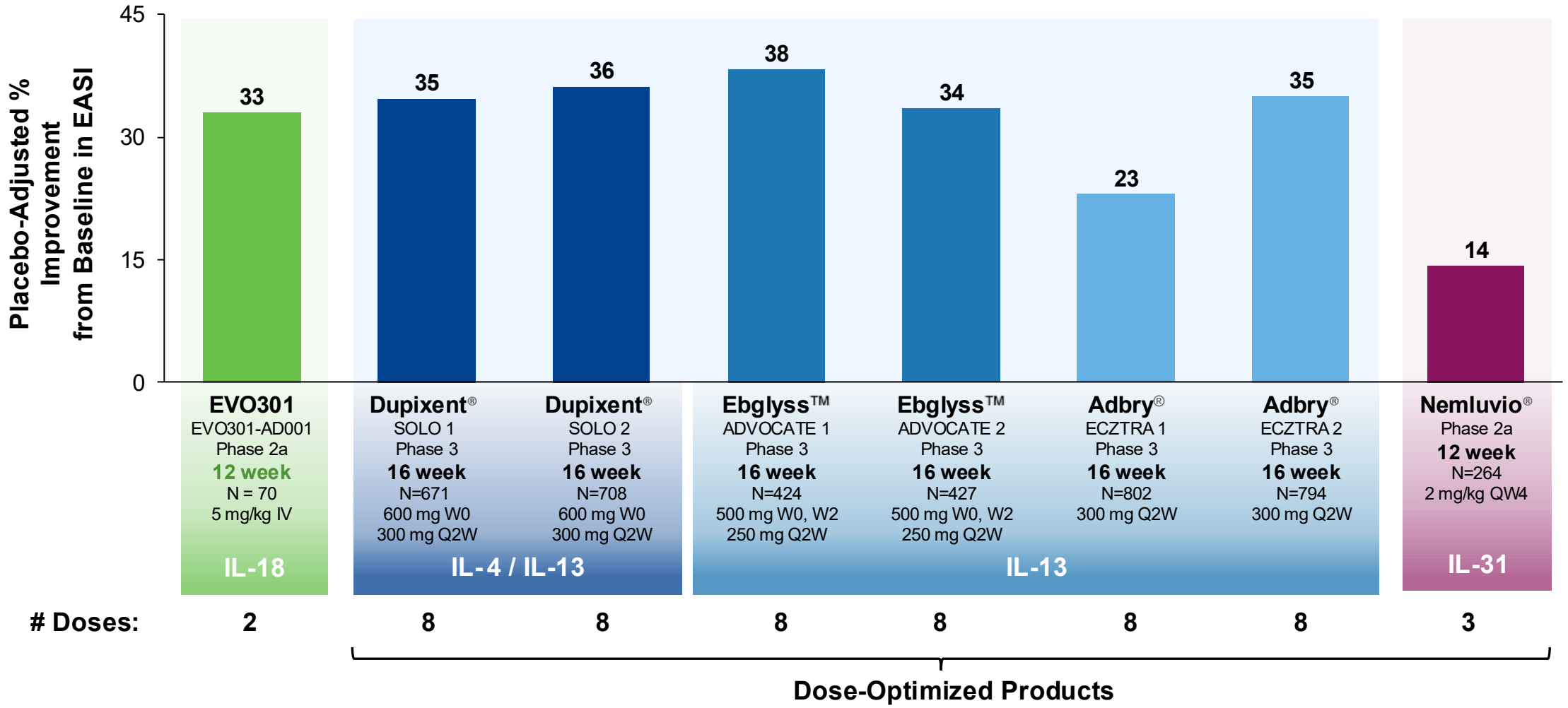
Phase 2a Trial in AD Demonstrated Statistically Significant Efficacy Across Time Points



% Average change in EASI is LS Mean

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

vIGA Response

(≥2-point improvement and a score of 0 or 1)

Visit	EVO301	Placebo
Week 4	4.2%	0%
Week 8	12.5%	0%
Week 12	22.9%	0%

vIGA: Validated Investigator's Global Assessment

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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)

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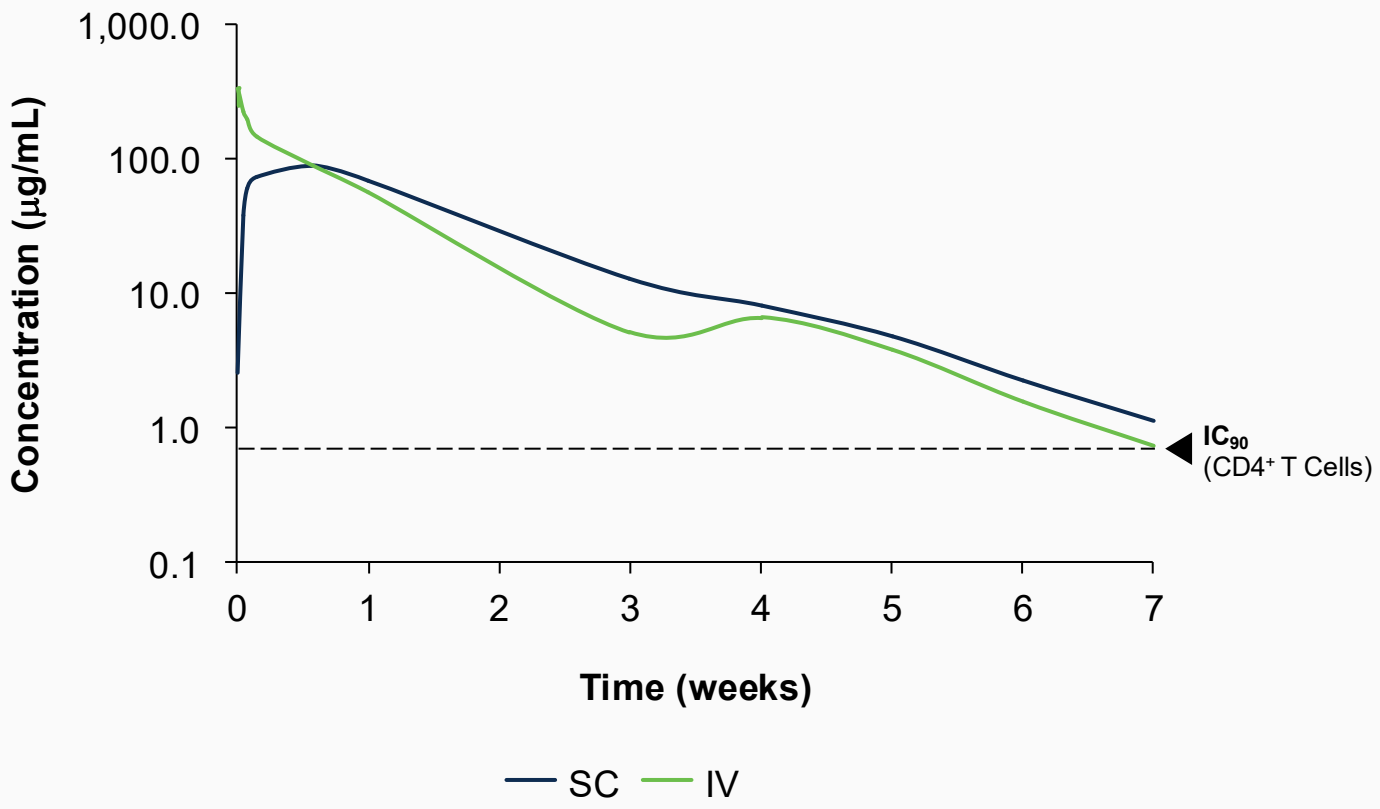
EVO301 Subcutaneous Formulation Exposure Consistent with IV

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

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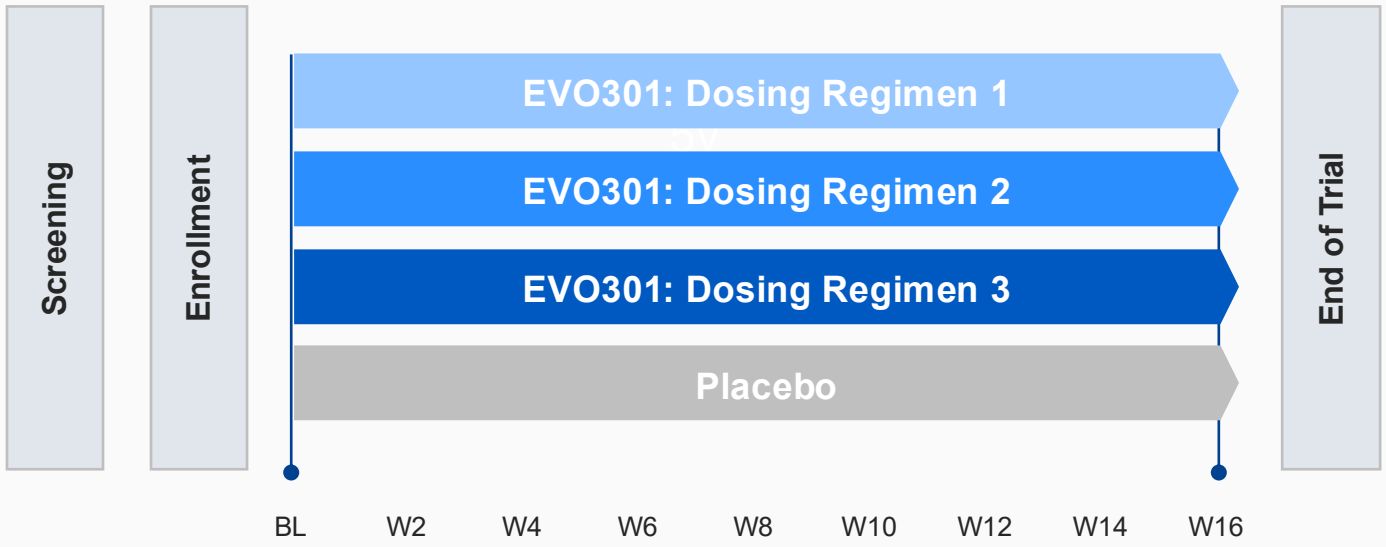
Planned EVO301 Phase 2b Dose-Ranging Trial in AD

Trial Initiation Expected Mid-2027

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

Randomized, Double-Blind, Placebo-Controlled Trial



Plan to explore Q2W and Q4W Regimens

Primary Endpoint

- % CFB in EASI at Week 16

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

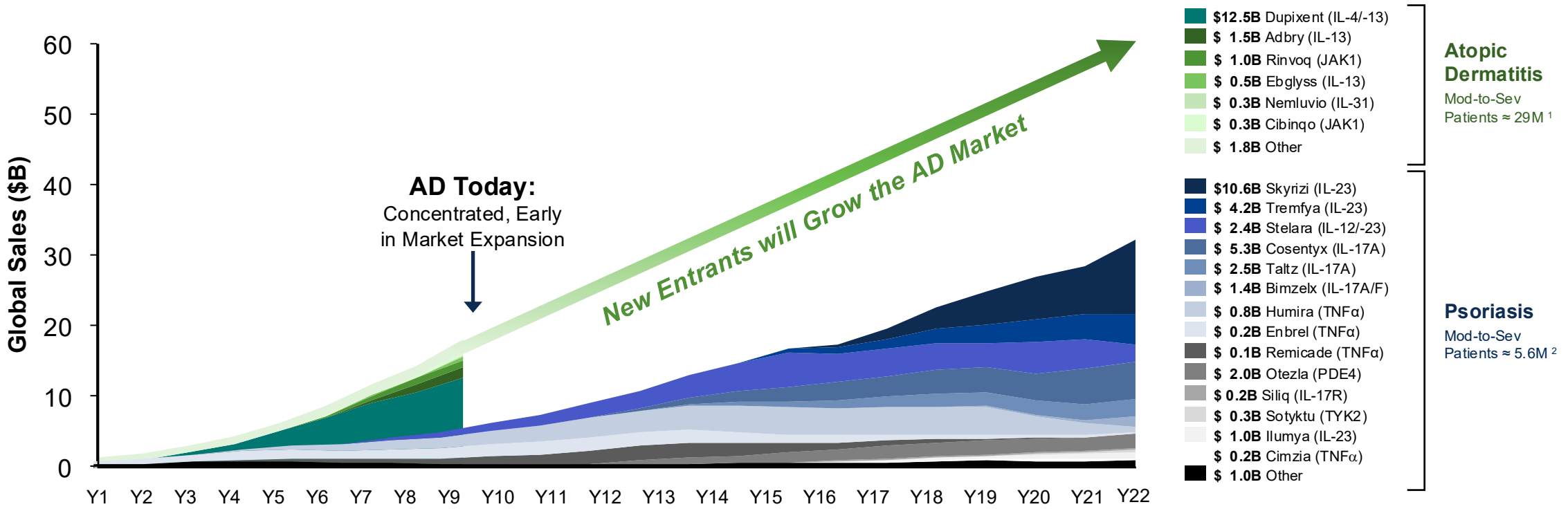
Exploratory & Biomarkers

- QoL
- Biomarkers
- Target Engagement

EASI = Eczema Area and Severity Index; vIGA = Validated Investigator Global Assessment; Pruritus-NRS = Pruritus Numerical Rating Scale; BSA = Body Surface Area; BL = Baseline, QoL = Quality of Life Q4W = Every four week dosing, Q2W = Every two week dosing, LD = Loading Dose.

Proven Playbook, Larger Market: AD Projected to Reach ~$60B

10 of 14 Psoriasis Advanced Therapies Became Blockbusters — And AD Has ~5x the Patients



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 from Decision Resources DL&F; 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 this slide contains registered trademarks not owned by Evommune. AD market size from Evaluate and internal analyses.

EVO301 Could Command Substantial Market Share in the Potentially $60B+ 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 AD Sales in $M
DUPIXENT® (dupilumab)	IL-4/-13	Q2W SubQ	2017	$12,496	$9,234	+9%	$17,423 (2030)
Adbry® (tralokinumab-ldrm) Injection 150 mg • 300 mg	IL-13	Q2W SubQ	2021	$1,508	$1,421	+22%	$2,469 (2030)
Ebglyss™ (lebrikizumab-lbkz)	IL-13	Q4W or Q8W SubQ	2024	$533	$274	+72%	$2,625 (2032)
nemluvio™ (nemolizumab-ilto) for injection 30 mg	IL-31	Q8W SubQ	2024	$339	$172	+91%	$2,759 (2032)

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

Sales from Evaluate Pharma may represent projections. 1. At maintenance. 2. From '25-'26.

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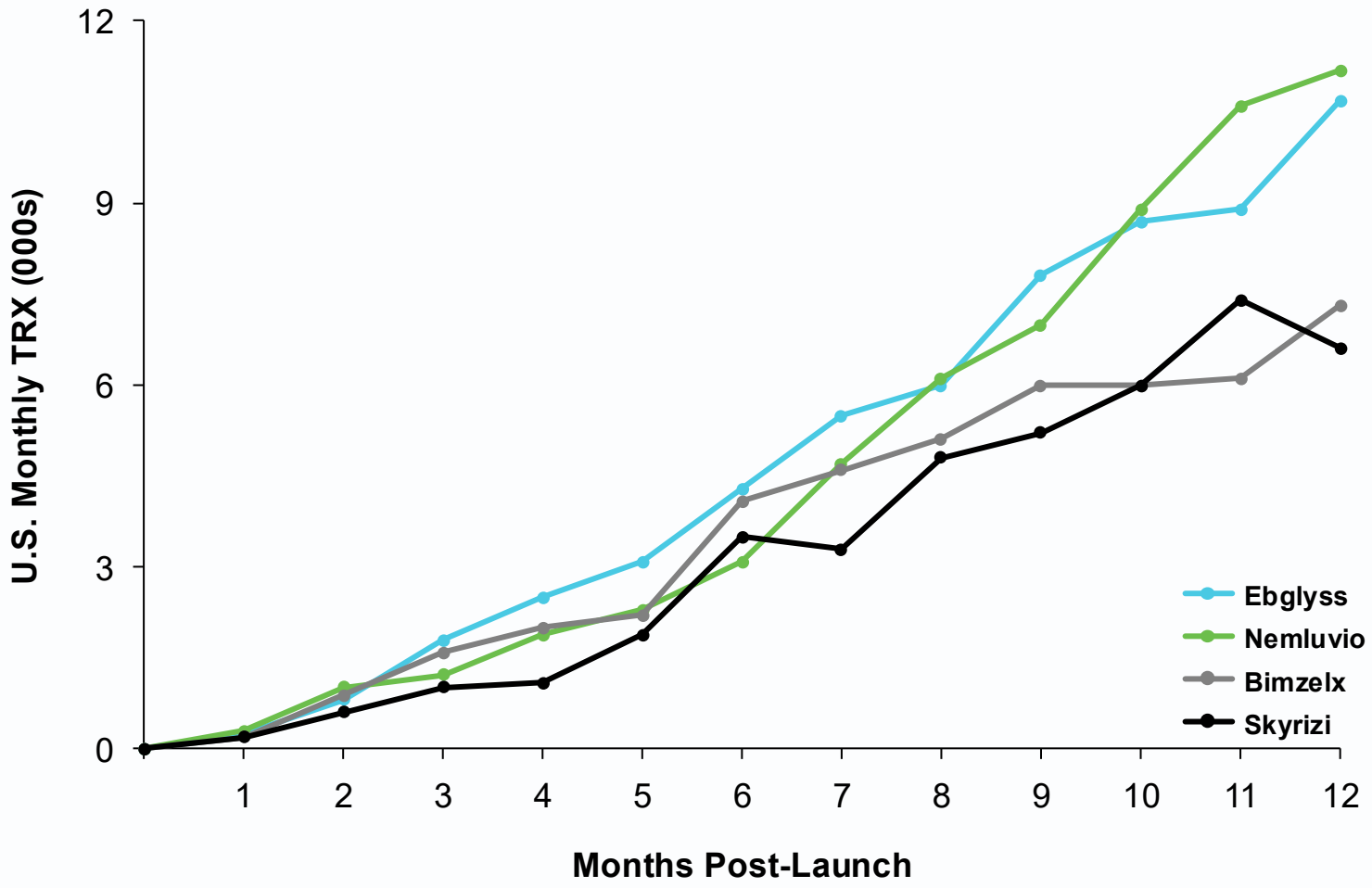
EBGLYSS and NEMLUVIO Launches in AD Outpacing Historical Psoriasis Launches, Highlighting Need for New Options in AD



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

Sources: IQVIA, based on New to Brand prescriptions.

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

Topical Therapies

MRGPRX2 Inhibitors

EVO756

IL-18BP EVO301

Approved Biologics (IL-4 / IL-13, IL-31)

IL-2 / Other

JAK Inhibitors

Topical Oral Injection

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

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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)

Lilly

Genentech
A Member of the Roche Group

STIEFEL
(Acquired by GlaxoSmithKline for $2.9B)

Peplin
Innovation in Dermatology
(Acquired by LEO Pharma for $288M)

PROTEOLIX

connetics
(Acquired by Stiefel for $930M)

GSK

Kadmon
(Acquired by Sanofi for $1.9B)

sanofi

Stanford MEDICINE

Allergan

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

ImClone Systems
(Acquired by Eli Lilly for $6.5B)

COHESION
(Acquired by Angiotech for ~$50M)

Roche

Key Roles in Almost 30 NDA / BLAs

Ebglyss (lebrikizumab-lbkz) 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

Qbrexza (glycopyrronium) cloth

Kyprolis (carfilzomib)

Lartruvo (OLARATUMAB) Injection 10 mg/mL

Portrazza necitumumab injection 800 mg/50 mL vial

Picato (ingenol mebutate) gel 0.015%, 0.05%

SORIATANE® (acitretin) Capsules

CYRAMZA (ramucirumab)

extina (ketoconazole) Foam, 2%

CAMZYOS (mavacamten) capsules

CLOVIQUE Trientine Hydrochloride Capsules, USP

Coseal Surgical Sealant

CellCept (mycophenolate mofetil)

Liletta (levonorgestrel-releasing intrauterine system) 52 mg

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

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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 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 & 2027:**
  - EVO301 reported positive data in a Phase 2a in AD (Feb 2026), moving to Phase 2b with subcutaneous formulation
  - EVO756 Phase 2b top-line data in AD expected in Q3 2026
  - EVO756 Phase 2b trial initiation in migraine expected mid-2026, top-line data in 2027
- **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**

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

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Thank You!