evommune

Corporate Presentation

November 2026

© Evommune, Inc.

Disclaimers

This presentation has been prepared by Evommune, Inc. (“we”, “us” or “our”) and contains forward-looking statements, including: statements about our expectations regarding the potential benefits, clinical activity and tolerability of our product candidates; our expectations with regard to the results of our clinical trials, preclinical studies and research and development programs, including the potential therapeutic benefit of EVO756, EVO301, EVO646 and EVO570, the design, objectives, initiation, timing, progress and results of current and future preclinical studies and clinical trials of our product candidates, including the ongoing and planned Phase 2 clinical trials for EVO756, EVO301, EVO646 and EVO570; anticipated cash runway; and continued advancement of our portfolio. These statements involve substantial known and unknown risks, uncertainties and other factors that may cause our actual results, levels of activity, performance or achievements to be materially different from the information expressed or implied by these forward-looking statements. We may not actually achieve the plans, intentions or expectations disclosed in our forward-looking statements, and you should not place undue reliance on our forward-looking statements. Actual results or events could differ materially from the plans, intentions and expectations disclosed in the forward-looking statements we make.

These and other risks are described more fully in our Annual Report on Form 10-K for the year ended December 31, 2025 and our other filings with the Securities and Exchange Commission (the “SEC”) and our other documents subsequently filed with or furnished to the SEC. All forward-looking statements represent our views as of the date of this presentation. All forward-looking statements contained in this presentation speak only as of the date on which they were made. Except to the extent required by law, we undertake no obligation to update such statements to reflect events that occur or circumstances that exist after the date on which they were made.

This presentation also contains estimates made by independent parties relating to industry market size and other data. These estimates involve a number of assumptions and limitations, and you are cautioned not to give undue weight to such estimates. We have not independently verified the accuracy or completeness of such information and we do not take any responsibility for the accuracy or completeness of such information.

The trademarks included in this presentation are the property of the owners thereof and are used for reference purposes only.

© Evommune, Inc.

Chronic Inflammation is 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

Treatments Have Critical Limitations

234M

Patients Diagnosed with Immunological Disease[3]



Advanced Therapies >$100B Sales[4]

Evommune (EVMN) is Doing Something About It

Experienced Team

Leaders behind
~30 approved NDAs and BLAs

Distinct Mechanisms

Novel approaches to
heterogeneous diseases

Portfolio Approach

Multiple assets across
multiple indications

© Evommune, Inc.

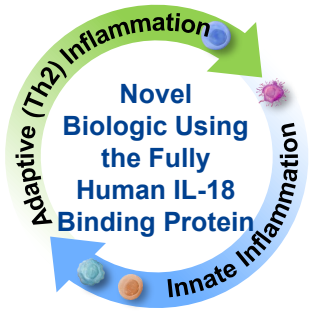
1. https://www.ncbi.nlm.nih.gov/books/NBK493173/. 2. Wylezinski LS, et al. PMID: 30979036. 3. GlobalData, 2023. 4. GlobalData, 2023 (US/EU5/JP forecasts).

evommune

A Broad Portfolio Progressing Multiple Opportunities

EVO301 | IL-18 Biologic

IL-18 Blockade for Multi-Pathway Immunomodulation



Target Population: 6M[1]

EVO756 | Oral MRGPRX2

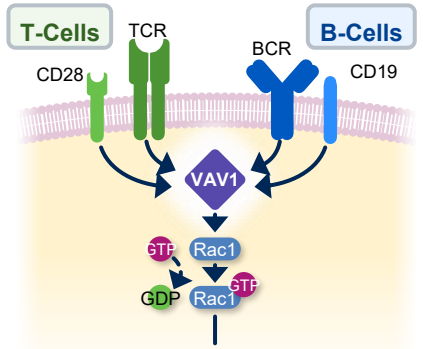
Targeting Sensory Neurons and Mast Cells in Migraine



Target Population: 30M[2]

EVO570 | VAV1 Degrader

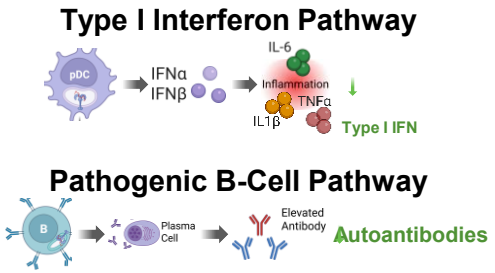
Oral Molecular Glue Degrader for T- and B-Cell Disease



Target Population: ~18M[3]

EVO646 | Oral TLR7/8

Dual Mechanism for Autoimmune Disease



Target Population: ~6M[4]

Focused on Innovative Targets with a High Probability of Success

1. Moderate-to-severe atopic dermatitis, US adults; Chiesa Fuxench ZC, et al. J Invest Dermatol 2019. 2. US/EU5/JP 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). 3. Rheumatoid arthritis, global prevalence (17.6M); GBD 2021, Lancet Rheumatol 2023. 4. Evommune internal estimates based on initial potential indications.

© Evommune, Inc.

Pipeline of Potential Best-in-Class and/or First-in-Class Assets



Advancing Multiple Programs Toward Clinical Proof-of-Concept

© Evommune, Inc.
evommune

Key Milestones Ahead For Evommune

Championing Innovation for Chronic Inflammation

EVO301 IL-18BP	• Plan to initiate Phase 2b in AD with subcutaneous formulation in mid-2027 • Plan to initiate Phase 2a in UC in 1H 2027
EVO756 MRGPRX2 Antagonist	• Expect top-line data from Phase 2b migraine trial in 2027
EVO570 VAV1 MGD	• Expect Phase 1 HV biomarker data mid-2027 • Plan to initiate Phase 2 program 2H 2027
EVO646 TLR7/8 Antagonist	• Plan to initiate Phase 1 in 1H 2027 • Expect Phase 1 HV biomarker data in 2H 2027

~$290m Cash and Investments[1], Current Runway Into 2H 2029

© Evommune, Inc.

1. Pro Forma: $262.3M cash, cash equivalents and investments as of September 30, 2026, plus net proceeds of approximately $28.0 million from the November PIPE financing.

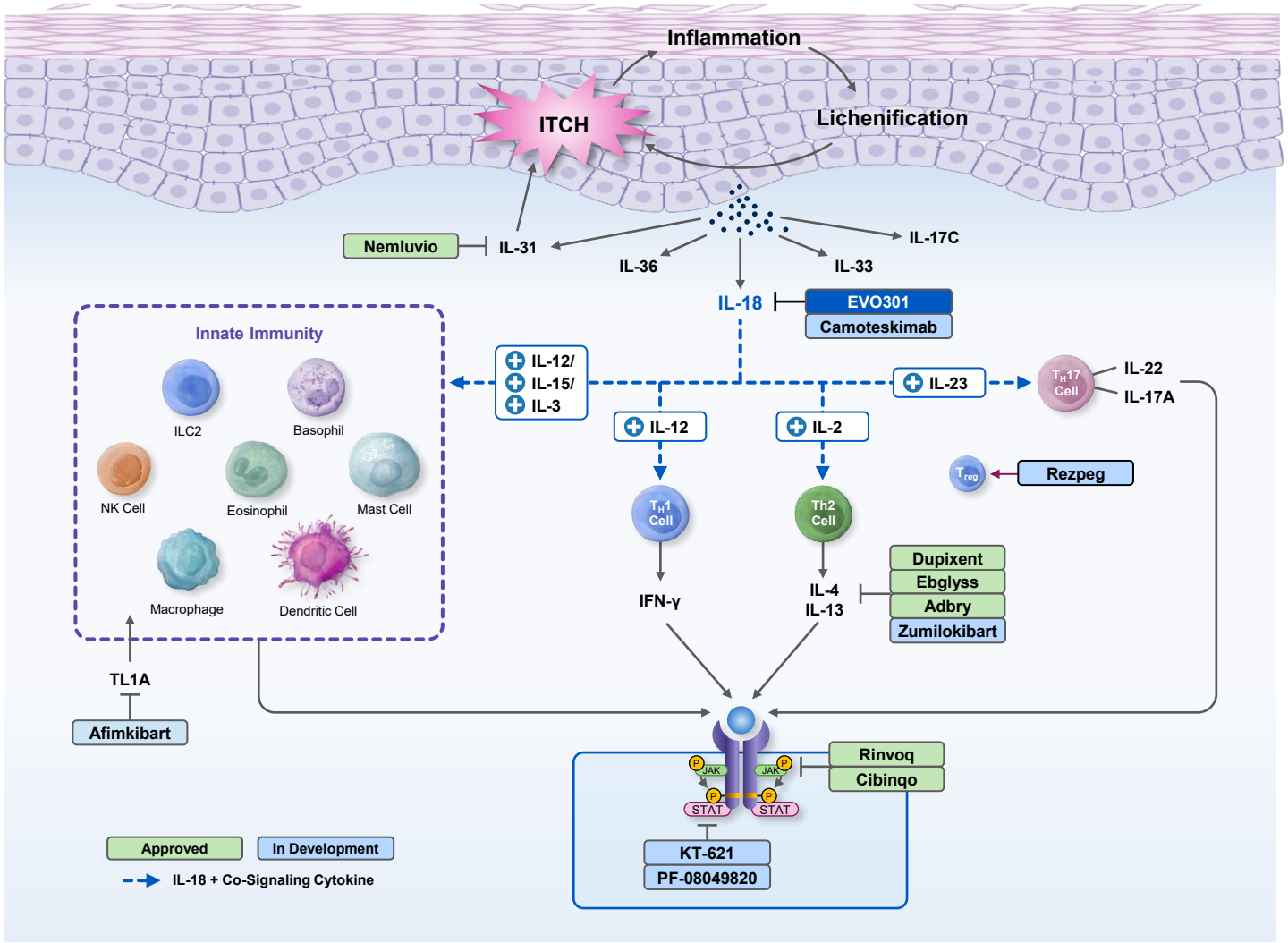
evommune

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

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

© Evommune, Inc.
evommune

EVO301 Mechanism of Action



EVO301 Blocks IL-18, a Cytokine Active Across Multiple AD Inflammatory Pathways

- **IL-18 is released from the skin barrier** and, alongside other cytokines, contributes to innate immunity and Th1 (IFN-γ), Th2 (IL-4, IL-13) and Th17 (IL-17) responses
- **EVO301 and camoteskimab target IL-18**, which plays a role across several AD inflammatory pathways at once
- Approved agents block single pathways: **IL-31, IL-4/IL-13 or JAK signaling**

© Evommune, Inc.
evommune

IL-18 Blockade Clinically Validated: Safe, Long-Term & Effective

Human Data Support Long-Term IL-18 Inhibition

IL-18BP Is the Basis of EVO301

Mechanism

- IL-18BP is the body's natural IL-18 brake[1]
- Tolerated for years, incl. in children[2]

Human Genetics

- **No AD, infections or other disease** with IL-18 loss of function[3]
- People with **undetectable** IL-18 live normal lifespans[4]

Clinical Safety

- No treatment-related safety signals
- **No conjunctivitis** in 4 IL-18 programs[5,6]

Sources: [1]Dinarello et al., Front Immunol (2013). [2]Canna et al., J Allergy Clin Immunol (2017); Gabay et al., Ann Rheum Dis (2018). [3]Belkaya et al., J Exp Med (2019). [4]Dominy et al., J Allergy Clin Immunol (2026). [5]Ellis et al., Allergy (2025). [6]Evommune EVO301 top-line results (2026).

Efficacy Shown Across 3 IL-18 Programs

Tadekinig alfa IL-18BP, AB2 Bio

- Still's Ph2: 11 of 22 responded by Week 3[2]
- Ph3 ongoing in NLRC4/XIAP[2]

Camoteskimab anti-IL-18, Apollo

- AD Ph2a: met primary EASI endpoint[7]
- ~80% EASI reduction, open-label[7]

Aletekitug anti-IL-18, GSK

- AD Ph1b: EASI −68% vs. −33% placebo[5]
- Single IV dose, Week 12[5]

Data from separate studies, not head-to-head. Source: [7]Apollo Therapeutics camoteskimab Ph2a press release (Sept 2025).

© Evommune, Inc.
evommune

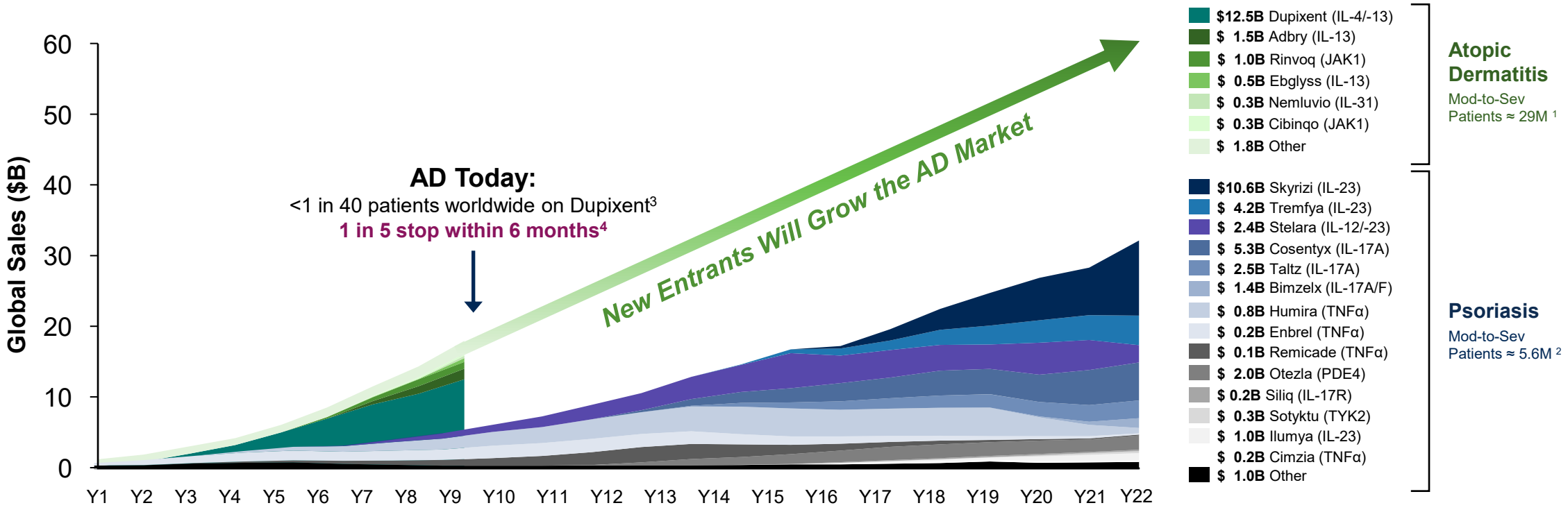
EVO301 in Atopic Dermatitis (AD)

Neutralizing IL-18 to address the Th2 and non-Th2 inflammation that drives AD

© Evommune, Inc.

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

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



40% of Psoriasis Patients Have Cycled Through 3+ Biologics, AD Needs Many More Options

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. 3. Kymera Therapeutics perspective piece: <1M of ~42M AD patients worldwide receive dupilumab. 4. Apogee Therapeutics 10-K: >20% of patients discontinue Dupixent within six months.

© Evommune, Inc.
evommune

EVO301 Has Potential to Command Substantial Market Share in AD Market

Sales in $M	Class	Route of Administration[1]	Launch Year	2025 WW Net Sales	2025 US Net Sales	Projected CAGR[2]	Projected Peak WW AD Net Sales in $M
DUPIXENT® (dupilumab)	IL-4/-13	Q2W SubQ	2017	$12,496	$9,234	+9%	$18,787 (2030)
Adbry® (tralokinumab-ldrm) Injection 150 mg • 300 mg	IL-13	Q2W SubQ	2021	$532	$422	+5%	$691 (2030)
Ebglyss™ (lebrikizumab-lbkz)	IL-13	Q4W or Q8W SubQ	2024	$408	$274	+24%	$2,318 (2033)
nemluvio™ (nemolizumab-ilto) for injection 30 mg	IL-31	Q8W SubQ	2024	$339	$172	+33%	$3,312 (2033)

Top AD Biologics Currently ~$17B Worldwide, Projected to be ~$30B by 2033[3]

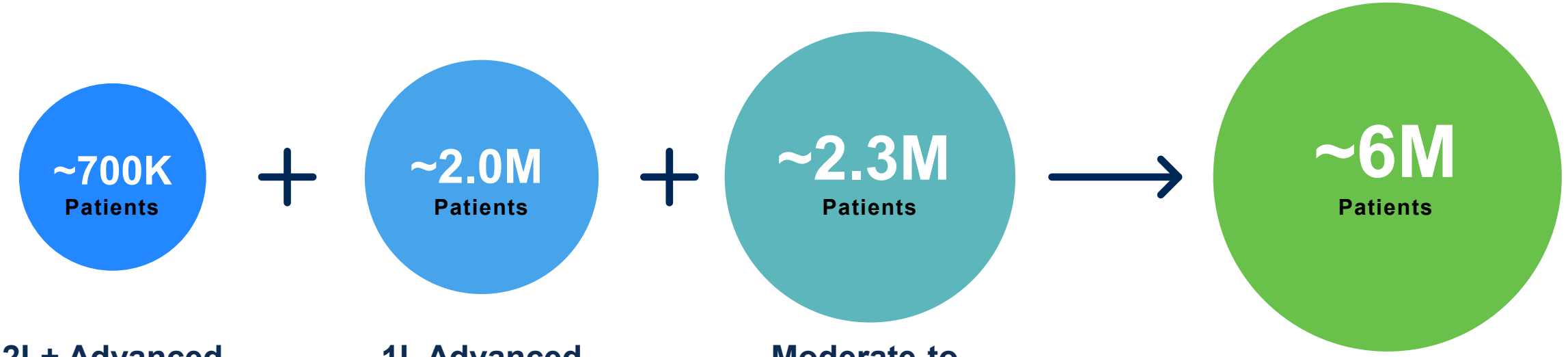
© Evommune, Inc.

Sales from Evaluate Pharma may represent projections (accessed Sept 2026). 1. At maintenance. 2. From '25-'26. 3. Global atopic dermatitis therapeutics market, projected at $29.5B by 2033 from $15.4B in 2024 (7.5% CAGR); Astute Analytica, Feb 2026.

evommune

EVO301 Has Multi-Billion Dollar Potential

Secure Access → **Advance to 1L** → **AD Market Expansion** → **Expand into IL-18 Diseases**



2L+ Advanced Therapy AD
Biologic-experienced / inadequate responders

1L Advanced Therapy AD
Initiating advanced systemic therapy

Moderate-to-Severe AD
Eligible for advanced therapies

~$60B Market

~$100B Market
IL-18-Driven Diseases
AD, UC, CD + adjacent pipeline expansion

EVO301 has the potential to become a foundational therapy in atopic dermatitis and other IL-18 driven diseases

© Evommune, Inc.
Source: Evommune 2036 estimates based on Evaluate Pharma (2032), Datamonitor (2033) data
evommune

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

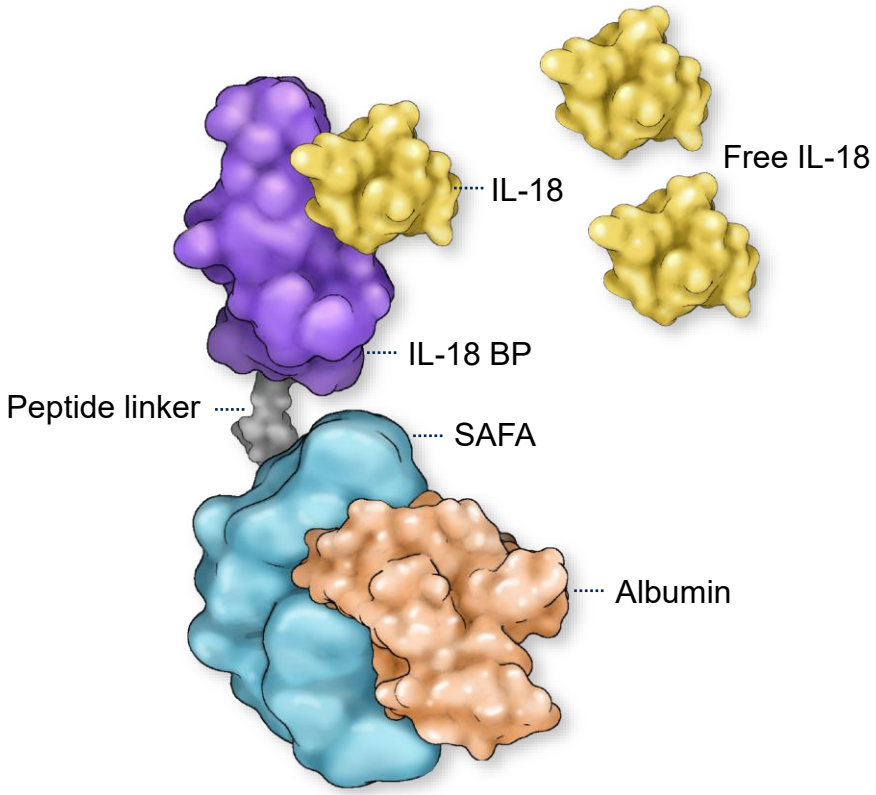
SAFA and IL-18BP Fused Via Peptide Linker for Extended Neutralization of IL-18 Activity

SAFAbody™ Platform Technology

- $T_{½}$ extension: FcRn-mediated recycling of HSA
- Efficient tissue distribution:
  - Smaller size (MW ~65 kDa) and HSA binding

IL-18 Binding Protein (IL-18BP)

- High binding affinity and specificity
- Native fully human sequence
- No binding to anti-inflammatory IL-37



Only Long-Acting IL-18BP in Development, Differentiated From IL-18 mAbs in Size, Binding Affinity and Targeting

© Evommune, Inc.

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

evommune

EVO301 Matches Biologics on Th2, Then Goes Beyond It

Clinically Validated Modulation of Th2 and Non-Th2 Inflammation

Demonstrated Ability to Impact Multiple Drivers of AD

Therapy	Adaptive Inflammation			Innate Inflammation	Skin Barrier (IL-22)	No Conjunctivitis Signal
	Th2	TH1	TH17			
EVO301	✓	✓	✓	✓	✓	✓
DUPIXENT®	✓	✗		✓	✓	✗
EBGLYSS®	✓	✗		✓	✓	✗
ADBRY®	✓	✗		✓	✓	✗
NEMLUVIO®	✓				✓	✓

Broader inflammatory signaling of IL-18 can safely address endotypes not fully captured by Th2-targeted therapies

Green = impacts biological pathway; **Red** = negative effect. EVO301 biomarker data: see slide 22

© Evommune, Inc.

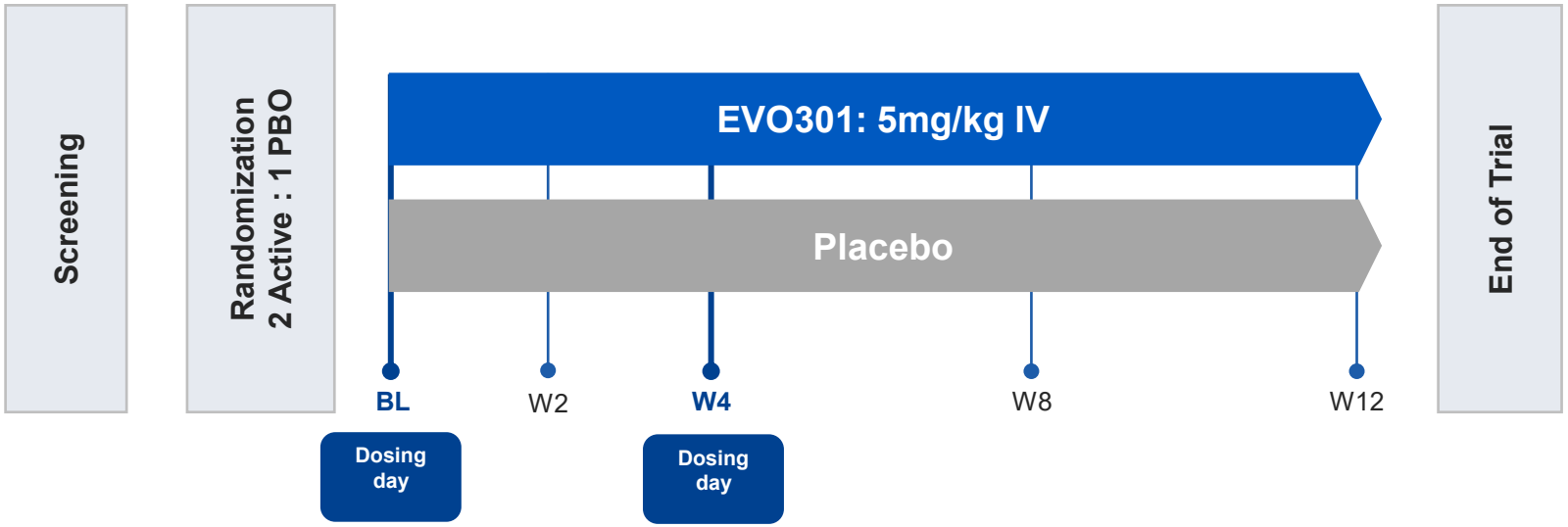
evommune

EVO301 Phase 2a Proof-of-Concept Trial Design

One dose, administered twice

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 baseline in 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

© Evommune, Inc.

EVO301 Achieved the Primary Endpoint

Phase 2a Proof-of-Concept Trial in Moderate-to-Severe Atopic Dermatitis (N=70)

With one dose, administered twice, comparable to dose-optimized marketed biologics

34%

placebo-adjusted EASI improvement at week 8 (33% at week 12)

23%

of patients achieved vIGA 0/1 at week 12 vs 0% on placebo

Highly significant EASI reductions vs placebo

at weeks 4, 8, and 12



No treatment-related serious or severe AEs
No conjunctivitis



Biomarkers and secondary endpoints moved with EASI



PK supports at least Q4-week dosing

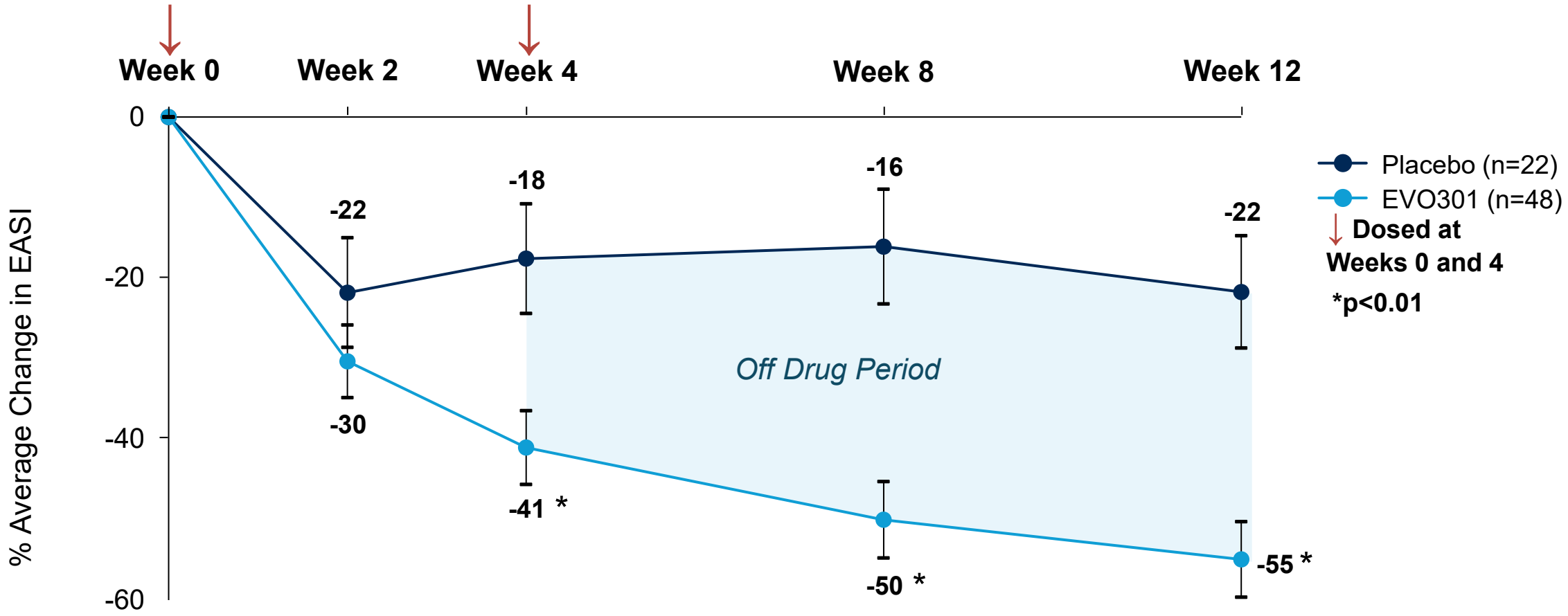
Phase 2a Profile Supports Potential Best-in-Class Monotherapy in Atopic Dermatitis

© Evommune, Inc.

evommune

Rapid, Robust and Durable Separation from Placebo

One dose, administered twice



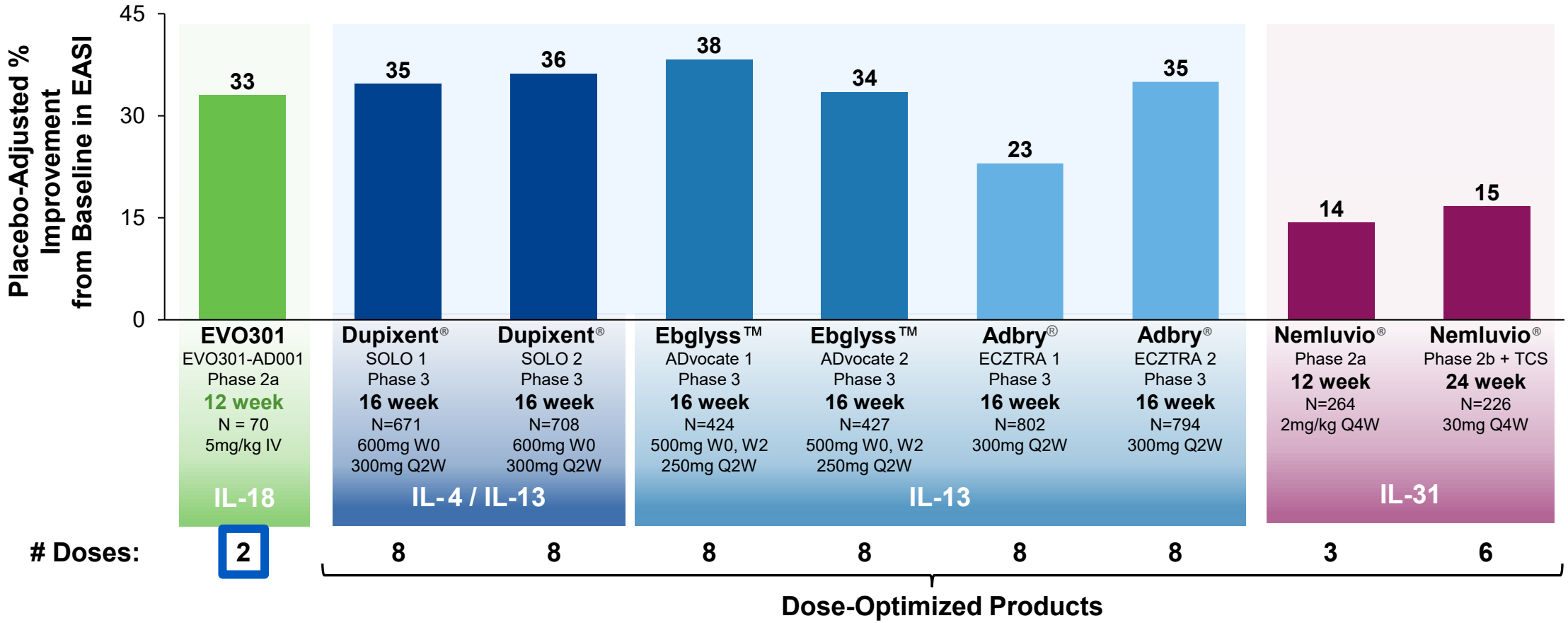
EASI Improvement Kept Deepening for 8 Weeks After the Last Dose (-41% to -55%)

© Evommune, Inc.

% Average change in EASI is LS Mean

evommune

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



EVO301: Single Dose Administered Twice, Already Comparable to Marketed Biologics

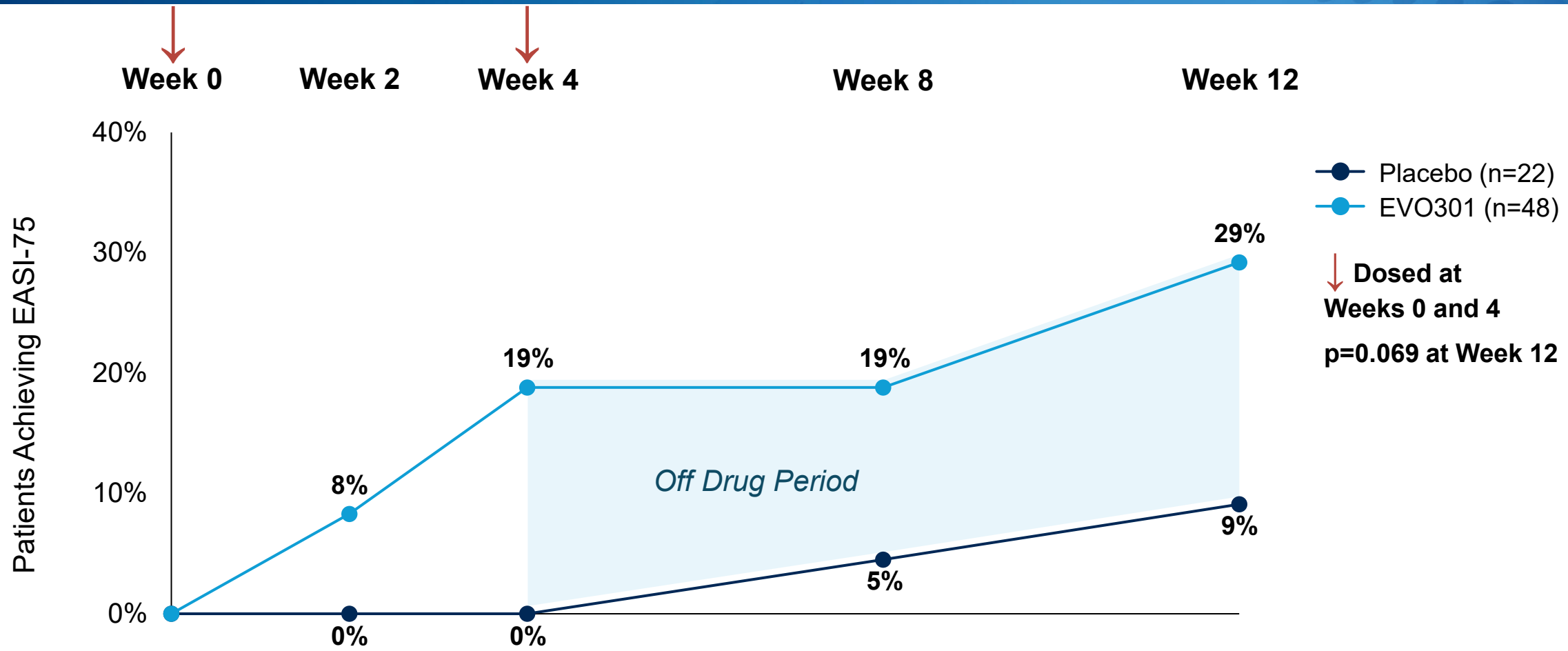
For illustrative purposes only. Not a head-to-head comparison. Differences exist between trial designs and trial characteristics, and caution should be exercised when comparing across trials. Sources: Silverberg *et* al. (2016), Silverberg *et* al. (2023), Wollenberg *et* al. (2020), Ruzicka *et al.* (2017); Nemluvio Ph2b + TCS: Silverberg et al., JACI (2020), 30mg arm, week 24.

© Evommune, Inc.

evommune

EASI-75 Response Continued to Rise After the Last Dose

Rapid, Durable and Increasing Responses with Just One Dose, Administered Twice



PBO-Adjusted EASI-75 Reached 20% at Week 12, 8 Weeks After the Last Dose

© Evommune, Inc.

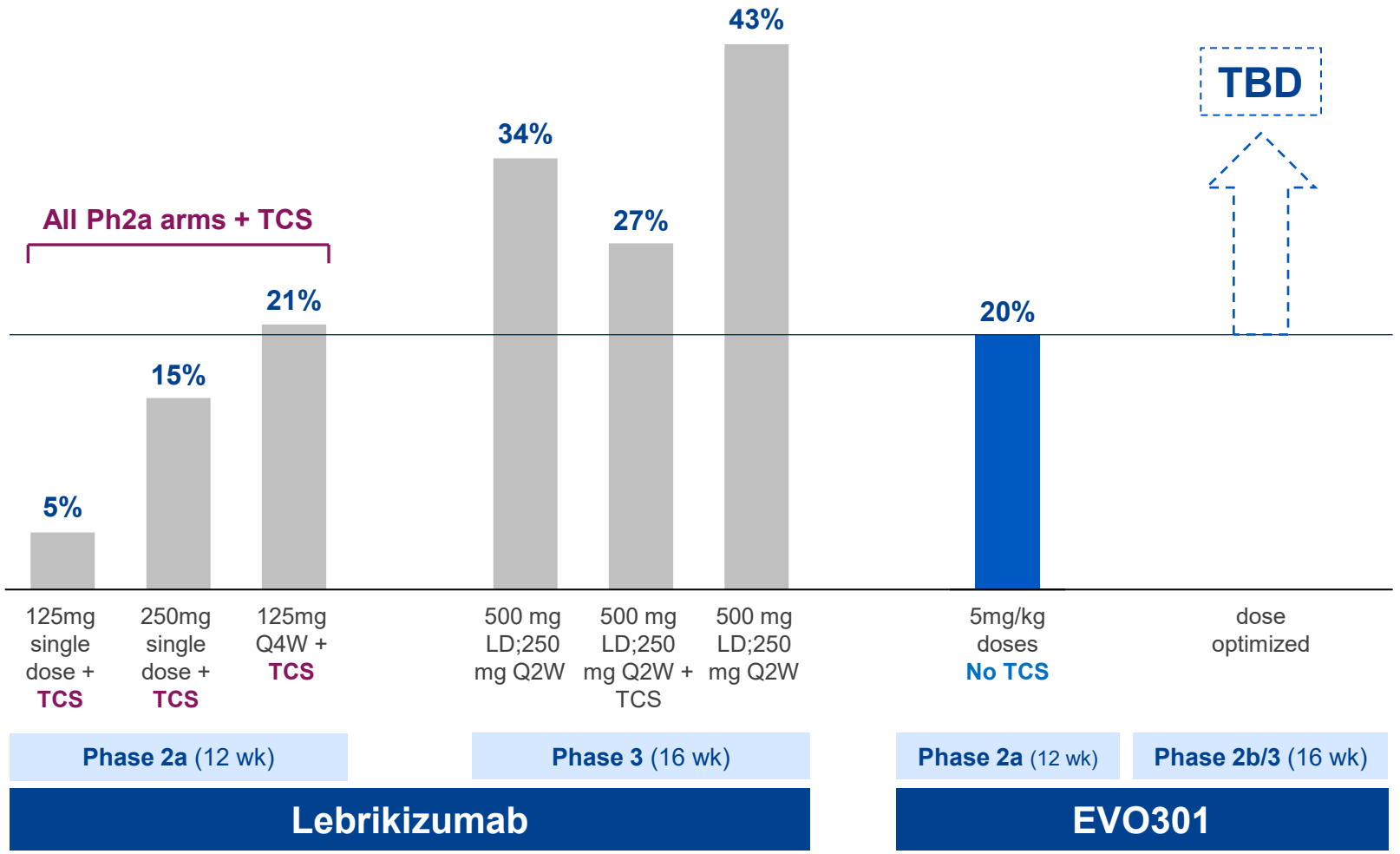
EASI-75 = ≥75% improvement in EASI. EVO301 vs placebo: p=0.148 (Wk 8), p=0.069 (Wk 12).

evommune

EVO301 Ph2a EASI-75 Matches Lebrikizumab Without TCS

Lebrikizumab Dose-Optimization After Ph2a Drove Substantial Improvements in EASI-75

PBO-Adjusted EASI-75, %



Lebrikizumab Precedent

EVO301 matched Lebri's Ph2a efficacy without TCS

- EVO301 reached **20% with no TCS**; Lebri's Ph2a arms reached 5-21%, **all with TCS**

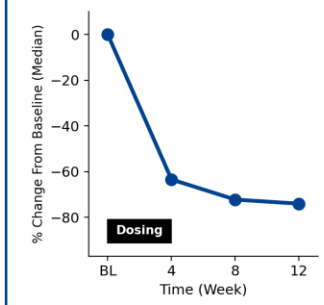
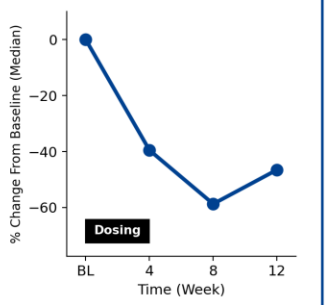
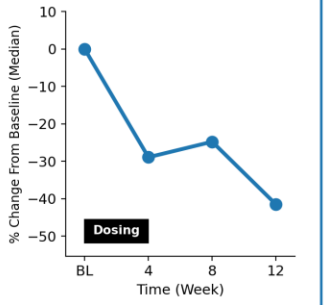
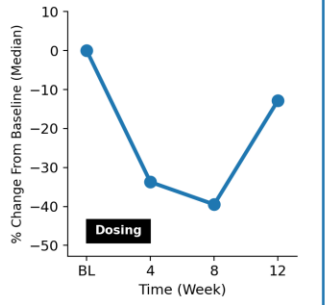
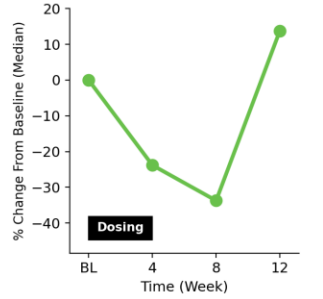
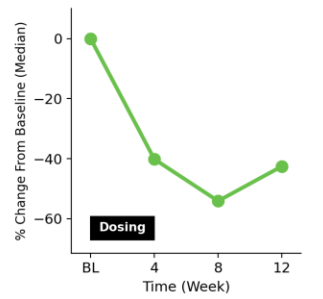
Phase 3 optimization drove a step-change in efficacy

- Dose, regimen and trial-design refinements increased EASI-75 from 5%-21% in Phase 2a to as high as 43% in pivotal studies

© Evommune, Inc.
evommune

EVO301 Impacts Biomarkers Th2 and Beyond

Matches IL-13 Therapies on Th2, and Impacts Th1 and Th17/22

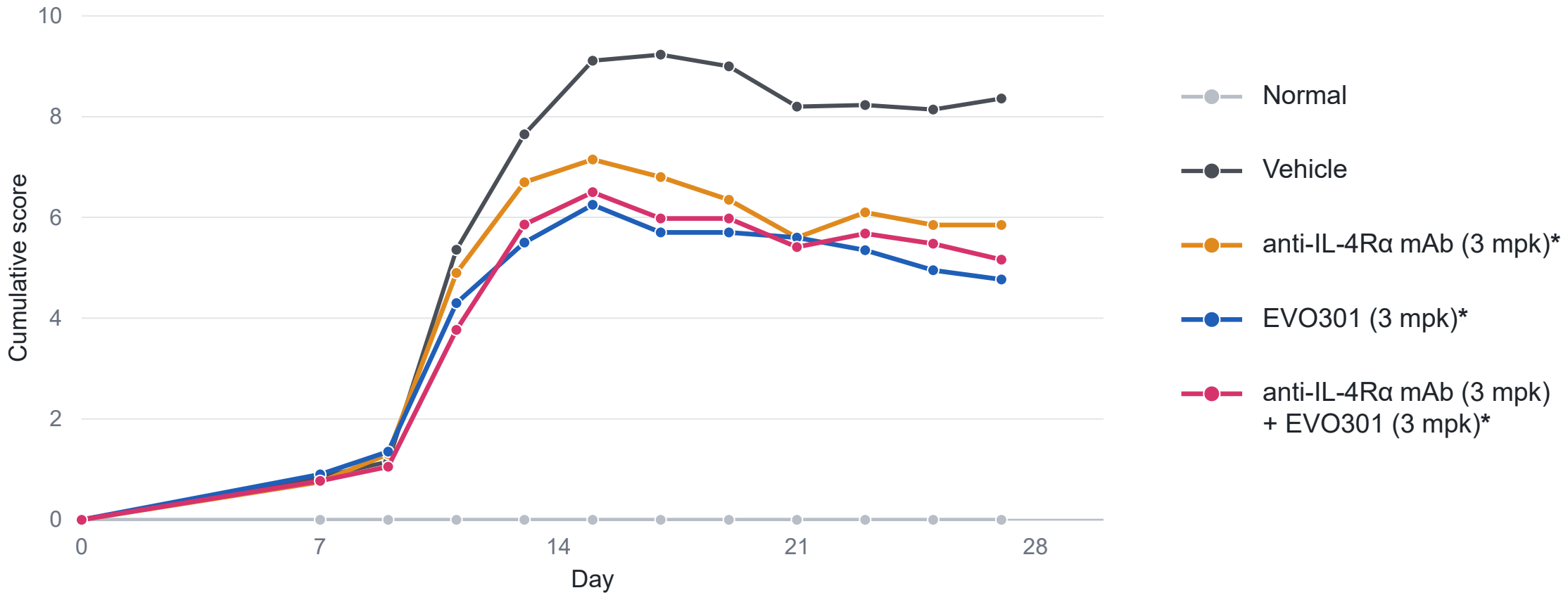
	Type 2 Inflammation		Type 1 Inflammation		Type 17/22 Inflammation	
	TARC[1] (CCL-17)	CCL-22[1]	CXCL9[2]	CXCL10[2]	S100A12[3]	IL-22[4]
	Validated biomarker for Th2 inflammation and disease severity	Key cytokine responsible for Th2 cell activation and amplification of inflammation	Highly specific cytokine upregulated by immune cells in response to IFN-γ	Validated biomarker for Th1 inflammation, disease severity and treatment responses	Highly specific alarmin downstream of IL-17 signaling	Key cytokine involved in skin barrier dysfunction and keratinocyte proliferation
						
EVO301 Maximum inhibition	**74%**	**59%**	**41%**	**39%**	**34%**	**54%**
Dupilumab	**79%[5]**	Not measured	Not measured	Not measured	Not measured	**60%[6]**
Lebrikizumab	**52-75%[7–8]**	**29%[7]**	Not measured	Not measured	Not measured	Not measured
Tralokizumab	**50%[9]**	Not measured	Not measured	**0%[9]**	Not measured	**20%[9]**
KT621	**74%[10]**	Not measured	Not measured	Not measured	Not measured	Not measured

[1]Th2-high subjects (n=11): TARC ≥ 929 pg/mL, CCL-22 ≥ 2,348 pg/mL; [2]Th1-high subjects (n=9): CXCL9 ≥ 56 pg/mL, CXCL10 ≥ 205 pg/mL; [3]Th17-high subjects (n=12): S100A12 ≥ 159 pg/mL; [4]all EVO301-treated subjects (n=47); [5]Week 16, SOLO1/2 trials, TARC ≥ 1,600 pg/mL; [6]Week 16, estimated from Ariens et al. 2020; [7]Week 16, estimated from ADvocate1/2 and [8]ADhere-J trials; [9]Week 28, estimated from Dekkers et al. 2024; [10]Week 4, Broaden trial, TARC; EVO301 maximum cytokine reduction represents median % change from baseline.

© Evommune, Inc.

IL-4/13 Co-administration with EVO301 Showed No Additive Benefit *in vivo*

Chronic oxazolone-induced atopic dermatitis preclinical model



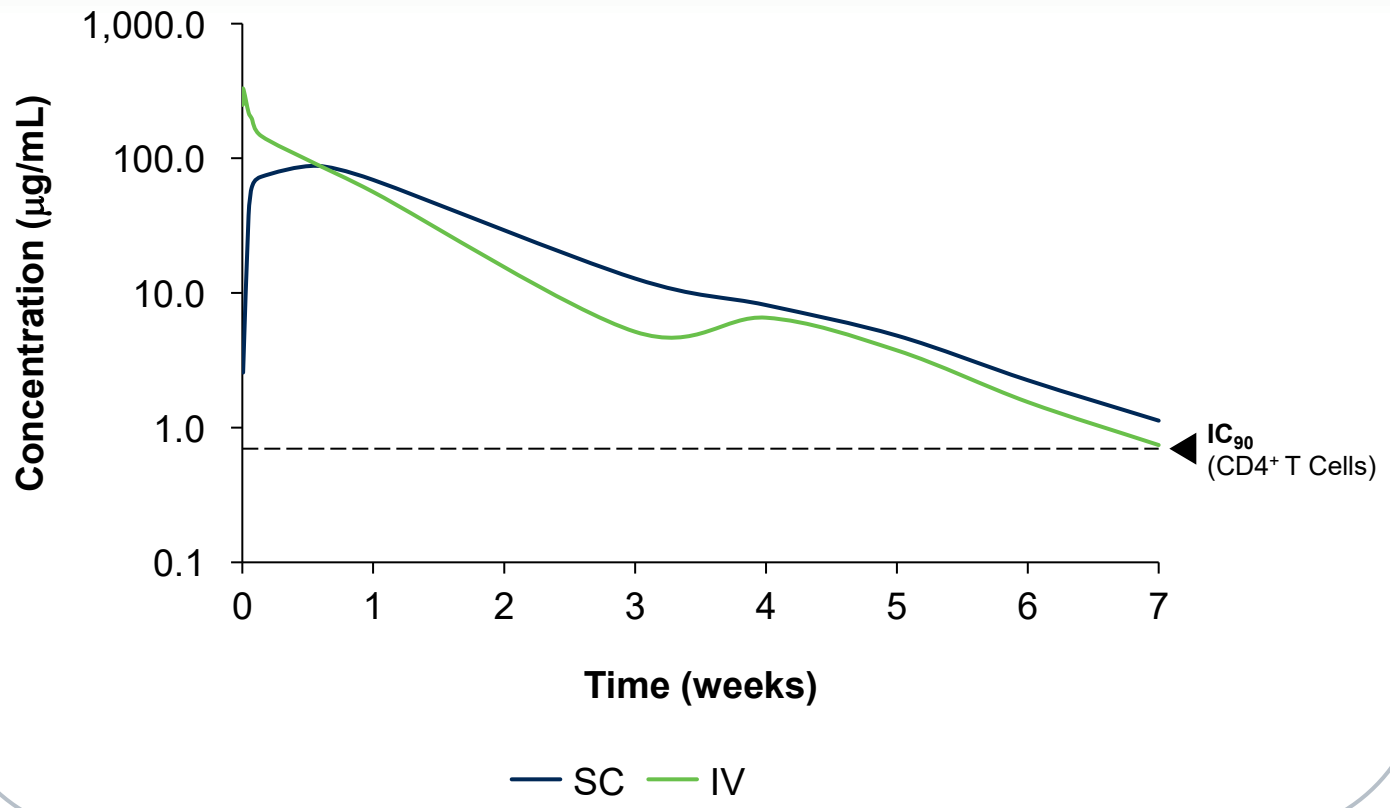
Mean cumulative score (erythema, scaling, thickness); error bars omitted for clarity. *Significant vs. vehicle at day 27 for each treatment arm.

© Evommune, Inc.
evommune

EVO301 Subcutaneous Formulation Exposure Consistent with IV

Subcutaneous Formulation Work Complete, Trial Initiation On-Track for Mid-2027

Comparable Nonclinical Serum Exposure



Key PK Parameters

Parameters	10mg/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

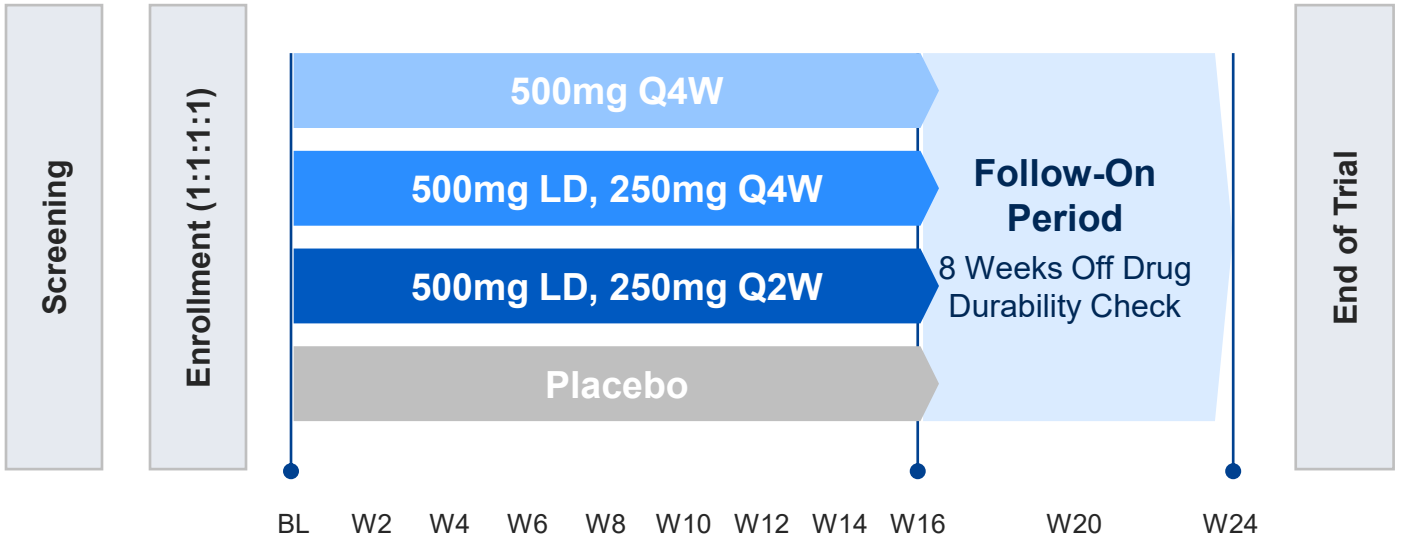
© Evommune, Inc.
evommune

Planned EVO301 Phase 2b Dose-Ranging Trial in AD

Trial Initiation Expected Mid-2027

Adults with Moderate-to-Severe Atopic Dermatitis (N ≈ 200)

Randomized, Double-Blind, Placebo-Controlled Trial



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

Planned Countries

- Poland, Germany, Czechia, Bulgaria, US, Canada, Australia, New Zealand

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, CFB = Change from Baseline.

© Evommune, Inc.

EVO301 in Inflammatory Bowel Disease

Extending IL-18BP into ulcerative colitis (UC), where epithelial barrier failure drives disease

© Evommune, Inc.

Current Therapies Stall at 20-30% Remission

1.25M

Americans living with UC[1]

20-30%

Remission rates in induction trials of advanced therapies[2]

1 in 5

Patients hospitalized within five years of diagnosis[3]

$6.4B

Skyrizi IBD sales in 2025, more than double 2024[4]

No approved therapy targets IL-18: EVO301 is the only IL-18BP program in IBD development[5]

1. Lewis JD et al., Gastroenterology 2023;165:1197-1205 (claims data extrapolated to 2020 US Census). 2. Alsoud D et al., Lancet Gastroenterol Hepatol 2021;6:589-595. 3. Frontiers in Gastroenterology, 2026 (~20% hospitalized, 7% colectomy within five years). 4. AbbVie 4Q25 earnings, Feb 2026 (Skyrizi total 2025 net revenues $17.6B). 5. Inflamm Bowel Dis 2024 (doi:10.1093/ibd/izad292); Evommune analysis of ClinicalTrials.gov, November 2026. IBD, inflammatory bowel disease; IL-18BP, IL-18 binding protein; UC, ulcerative colitis.

© Evommune, Inc.
evommune

UC is a Large and Expanding Market Where Innovation Can Win

>$21B by 2033, with Durable Remission Still Out of Reach for Most Patients

Market Scale and Momentum

>$21B

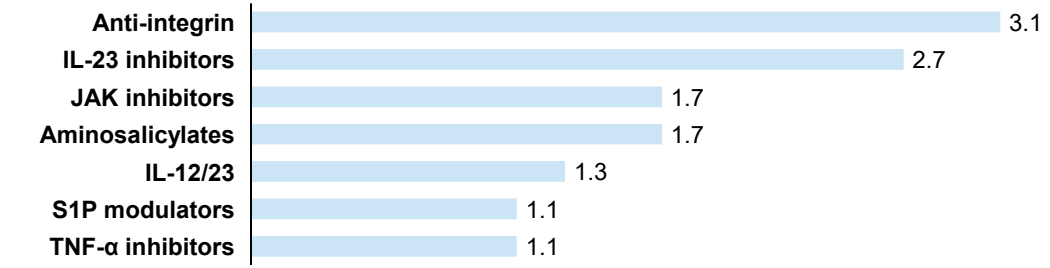
UC therapy sales in 2033[1]

+$10.4B (~92%) market growth expected

3.3M

Patients in 7MM[2]

Multiple Mechanisms Expected to Exceed $1B (2033 projections)[1]



- Recent launches suggest differentiated entrants can expand the category, not just redistribute the share

Clinical Opportunity

~10-30%

of patients do not respond to initial anti-TNF therapy[3]

~23-46%

of patients lose response over time[3]

Meaningful Clinical Gaps

Durable remission, efficacy in bio-experienced patients, convenience, and prevention of fibrosis

UC is a $21B+ market with room for multiple winners and headroom for better outcomes

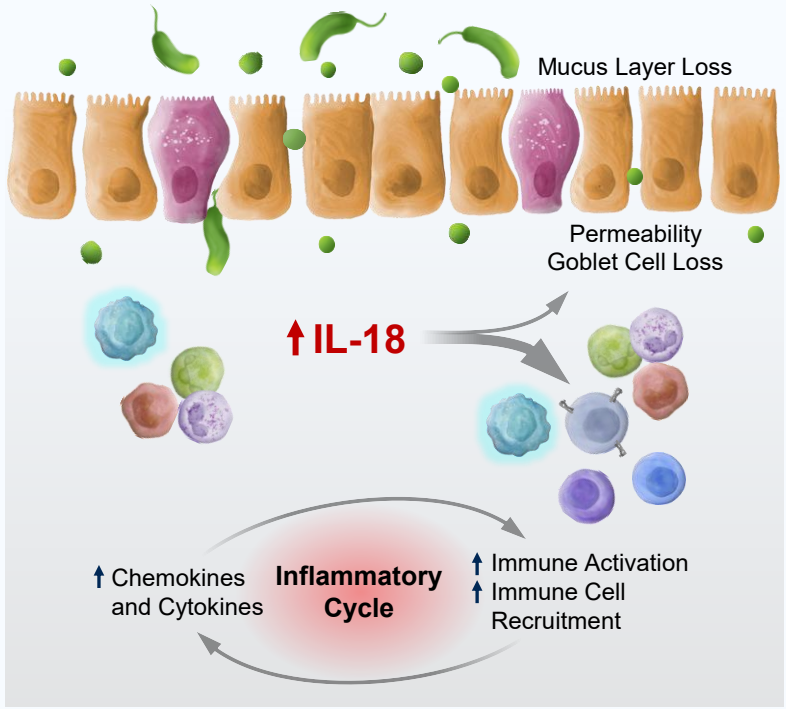
1. Clarivate, UC Disease Landscape & Forecast (G7: US, EU5, Japan), Oct 2025; Datamonitor, UC Disease Landscape & Forecast. 2. DelveInsight, UC Market Insight, Epidemiology and Market Forecast, 2026 (diagnosed prevalent cases, 2025). 3. Peyrin-Biroulet L, et al. Ther Adv Gastroenterol 2021;14:17562848211059954. Other sources: pivotal trials (GEMINI 1, UNIFI, LUCENT, INSPIRE, QUASAR, ELEVATE, U-ACHIEVE/ACCOMPLISH, ACT 1); CGH 2023. 7MM, seven major markets; TNF, tumor necrosis factor; UC, ulcerative colitis.

© Evommune, Inc.
evommune

EVO301 Targets Epithelial Barrier Failure, the First Trigger in IBD

Translates to Potential Barrier Repair

Key role for IL-18 signaling in IBD





Barrier failure is the first trigger in IBD; IL-18 blocks its repair

Human Genetics	IL18R1-IL18RAP variants raise IBD risk[1]
Patient Data	IL-18 elevated in inflamed IBD mucosa[1]
Preclinical Validation	Loss of IL-18BP drives severe colitis in mice[2]
Barrier Function	IL-18 blocks goblet cell and mucus barrier repair[2]
AD Clinical Data	EVO301 modulated Th22 (IL-22) biomarkers in the Phase 2a AD trial[3]
Safety	Long-term IL-18 inhibition tolerated; supports potential use in combination

[1] Zhernakova et al., Am J Hum Genet, 2008; Harrison et al., Mucosal Immunol, 2015.
[2] Nowarski et al., Cell, 2015 (PMID 26638073).
[3] EVO301 Phase 2a AD trial biomarker data, EADV 2026.

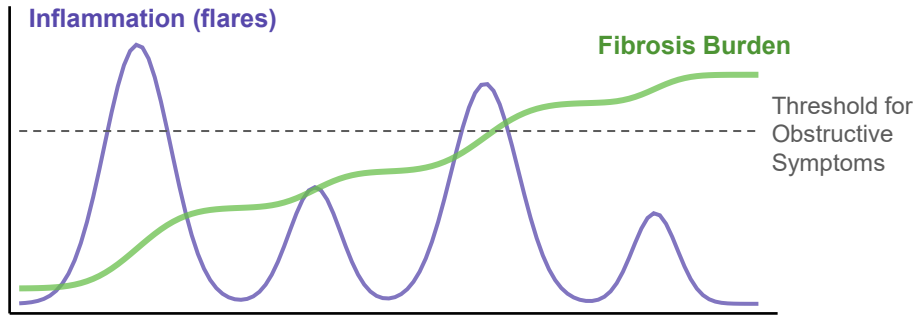
© Evommune, Inc.

EVO301 Reversed Established Fibrosis *in vivo*

A Differentiating Effect Not Observed with Current IBD Therapies

Fibrosis Remains a Major Unmet Need in IBD

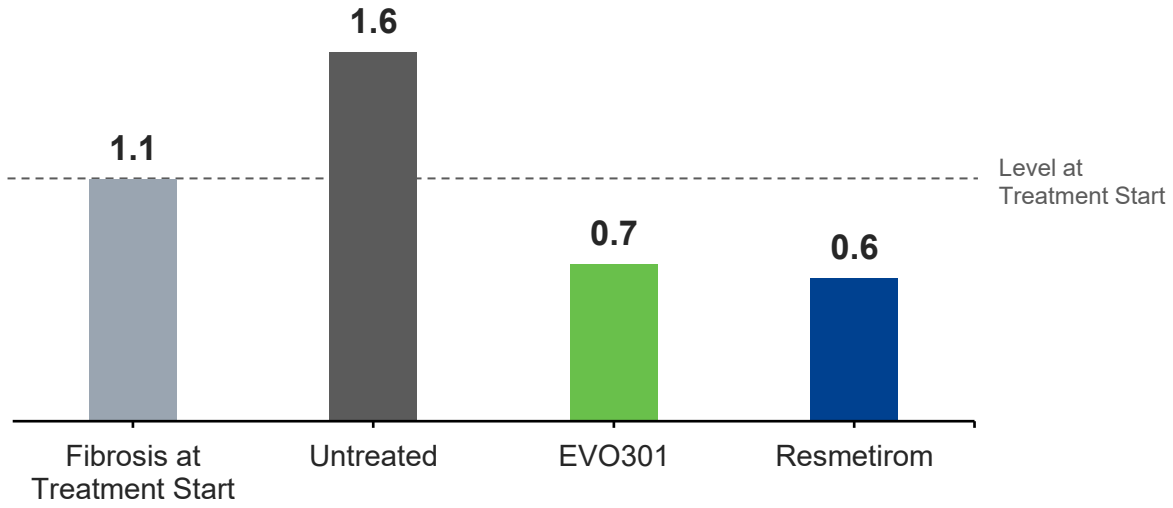
- ~50% of patients with Crohn's disease develop stricturing or penetrating complications within 10 years
- Fibrotic complications are a major driver of surgery and irreversible bowel damage
- Anti-TNF, IL-23, JAK and S1P agents treat inflammation. No approved IBD therapy is antifibrotic



Disease course. Flares come and go; fibrosis progresses over time

EVO301 Reduced Fibrosis Below Baseline, Matching Resmetirom in a Mouse MASH Model

Histological Fibrosis (Sirius red, % positive area)



- EVO301 matched resmetirom's antifibrotic effect, with fibrosis falling below pretreatment levels - consistent with true regression rather than prevention alone

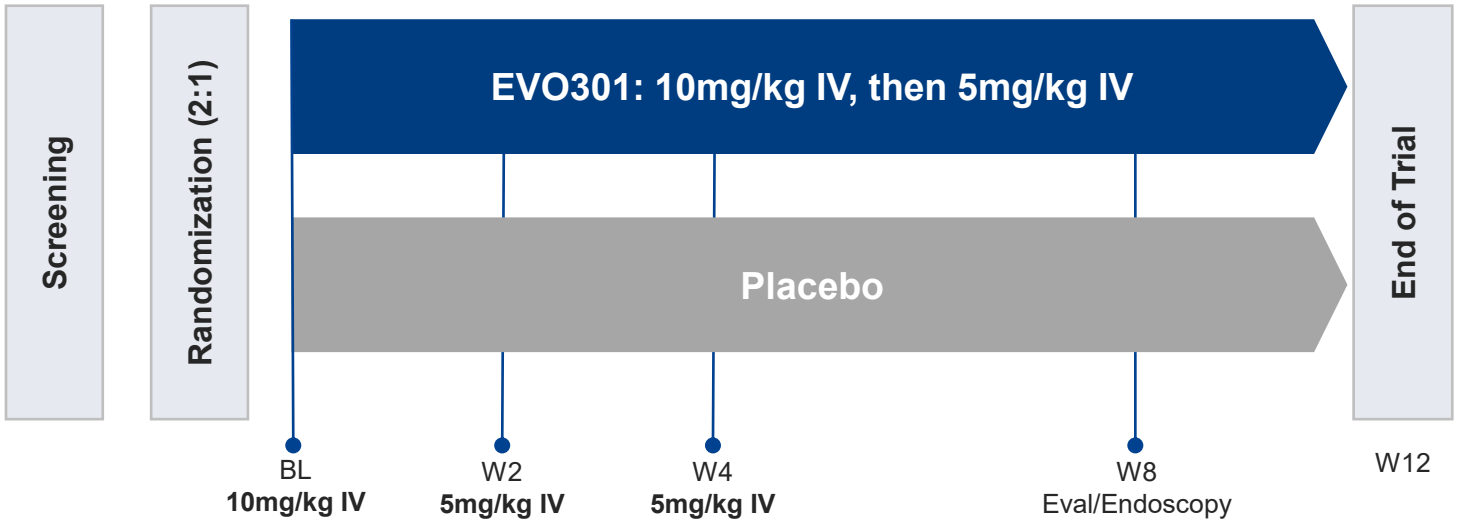
MASH: metabolic dysfunction-associated steatohepatitis. 1 Rieder et al., Gut, 2024;73:854-866. AprilBio 2025, PMID: 40239621, PMID: 27720839 Note: EVO301 vs resmetirom: not significantly different (p = 0.07). Hydroxyproline and Pro-C3 showed the same pattern. Treatment started at week 19; efficacy readout at week 27.

© Evommune, Inc.

Planned EVO301 Ulcerative Colitis Proof-of-Concept Phase 2a Trial

Adults with Moderate-to-Severe Ulcerative Colitis (N ~ 84)

Randomized, Double-Blind, Placebo-Controlled Trial
IV treatment at Baseline, Week 2, Week 4



Primary Endpoint

- Safety and tolerability

Secondary Endpoints (Weeks 2, 4, 8, 12)

- ΔmMCS vs placebo
- Endoscopic improvement (Week 8)
- Fecal calprotectin (Weeks 8 and 12)
- PROs rectal bleeding and stool frequency
- Partial and complete remission (weeks 8 and 12)

Pharmacokinetics and ADA

Exploratory Biomarkers

- Serum IL-18 and drug-bound IL-18 target engagement
- Serum and stool: Fcal, Lactoferrin, hsCRP, LRG, NGAL, immune cytokines
- Biopsy bulk RNA-seq: tissue engagement and MoA

UC: ulcerative colitis; mMCS: modified Mayo Clinical Score; PRO: patient reported outcomes; Fcal: fecal calprotectin, hsCRP: high sensitivity C-reactive protein; LRG: Leucine-rich α2-glycoprotein; NGAL: Neutrophil Gelatinase-Associated Lipocalin; MoA: mechanism of action.

© Evommune, Inc.

EVO756: Oral MRGPRX2 Antagonist

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

© Evommune, Inc.

EVO756: Targeting Major Unmet Need in Migraine Prevention

~30M

Patients Eligible for Preventive Therapy[1]

EVO756 - Defining the Next Wave of Preventives

1. **Oral Dosing**
2. **Novel Dual Mechanism**
3. **First-Line Potential**

1. US/EU5/JP 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).

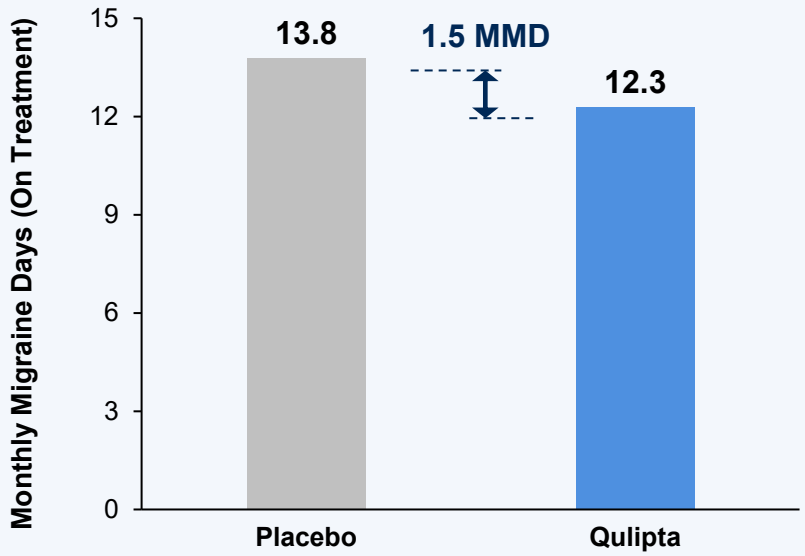
© Evommune, Inc.
evommune

Innovation in Migraine Prevention Has Been Largely Limited to CGRPs

Preventive Innovation Has Clustered Around a Single Target (CGRP)

Advanced Treatments	Class	Route of Admin.	MMDs on drug	Annual US Sales
QULIPTA (atogepant) tablets	CGRP	Oral QD	12.3	$1.5B
Nurtec ODT		Oral EOD	*	$2B
vyepti		IV	7.9	$1.3B
Emgality		SC	14.6	$0.3B
aimovig erenumab		SC	*11.2	$0.4B
AJOVY		SC	11.3	$0.6B
BOTOX onabotulinumtoxinA	Neuro-toxin	IM	*11.5	$2B

Migraine Burden Persists Despite Oral CGRP Therapy



Efficacy, Not Just Innovation

Best therapies beat placebo by only **~1-1.5 monthly migraine days**

MMD = Monthly Migraine Days. All MMDs are from chronic migraine trials across 12 weeks. *Nurtec ODT does not have trials in chronic patients. *Aimovig is across weeks 9-12. * Botox is across weeks 1-24. Sources: Qulipta - Prescribing Information, Vyepti 300mg - NCT02974153, Emgality - Prescribing Information, Aimovig 140mg - NCT02066415, Ajovy 675mg - NCT02621931, Botox - NCT00156910

© Evommune, Inc.
evommune

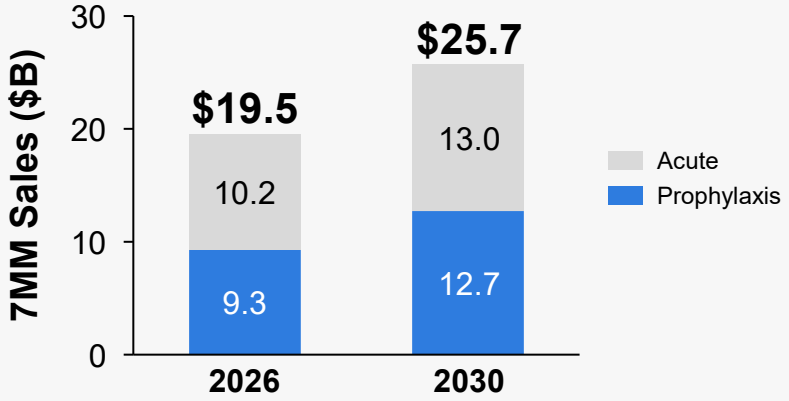
High Demand for Preventive Migraine Therapy

Limited Therapeutic Diversity
Only CGRP inhibitors and neurotoxin

Inadequate Efficacy
~45% of episodic and chronic patients do not achieve 50% improvement and have ≥8 monthly migraine days

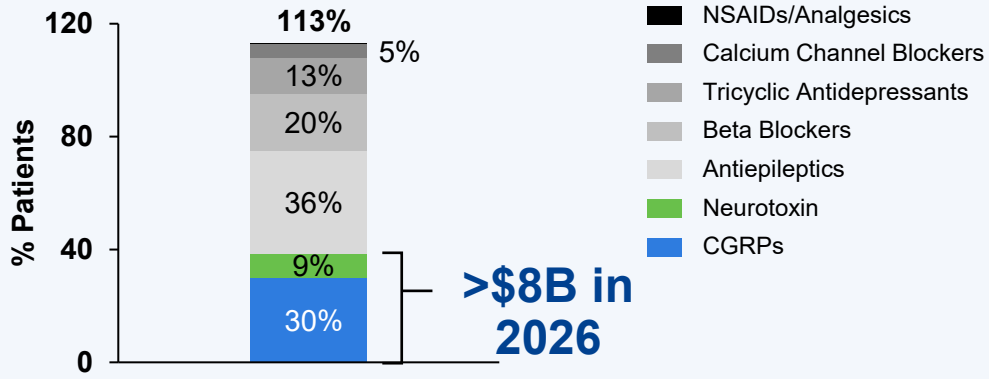
Tolerability Challenges Remain
CGRPs associated with constipation, hypertension, Raynaud's, nausea, allergic and injection site reactions

Prevention Drives ~50% of $25B Migraine Market



>75M People Living with Migraine in 7 Major Markets

Most Patients Remain on Legacy Preventives - Targeted Therapies Drive Sales



~30M Patients Eligible for Preventive Therapy in 7 Major Markets

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. AHS Consensus Statement

© Evommune, Inc.

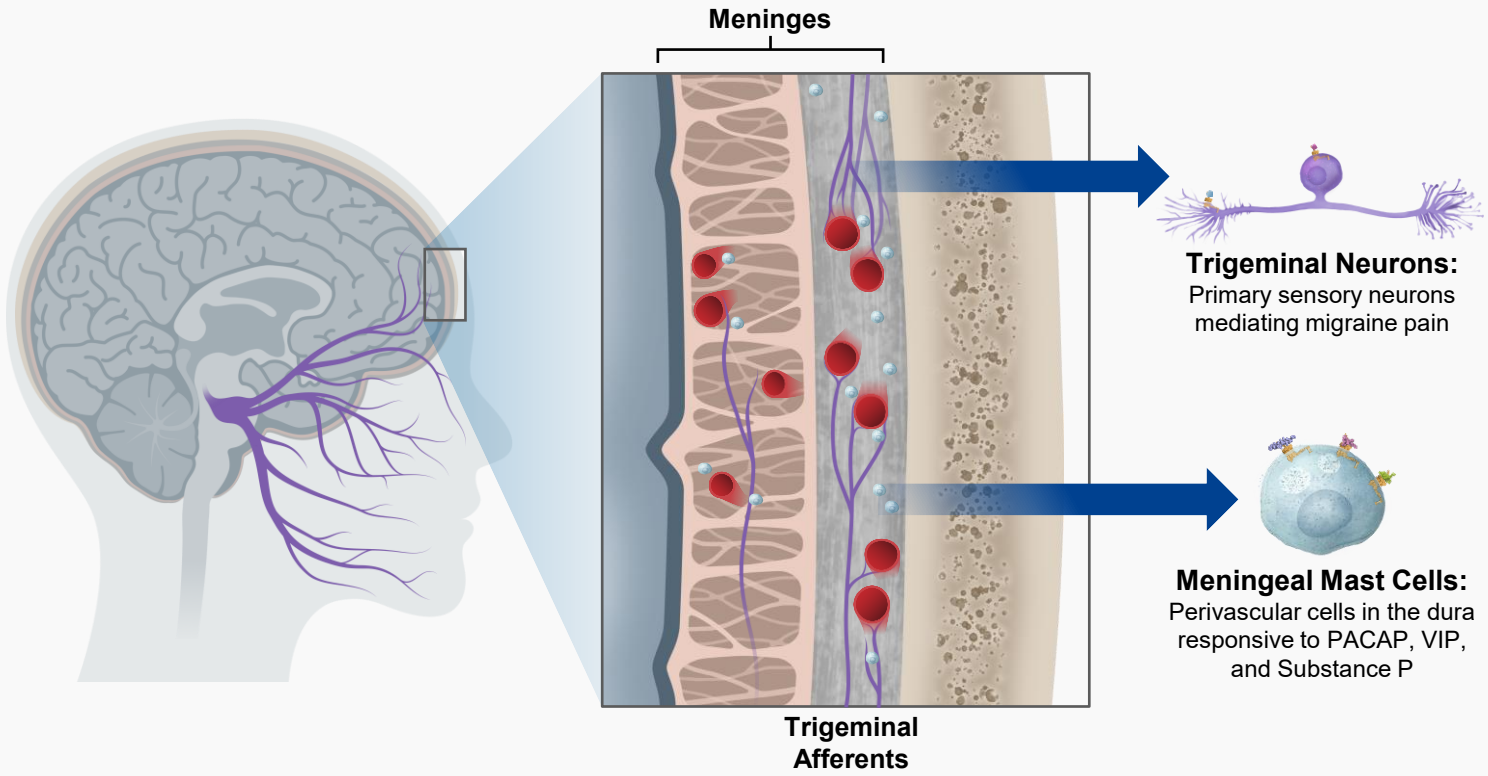
Strong Scientific Rationale for EVO756 in Migraine

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 Validation	mAb inhibition of MRGPRX2 ligand (PACAP) shows clinical benefit in-line with CGRPs

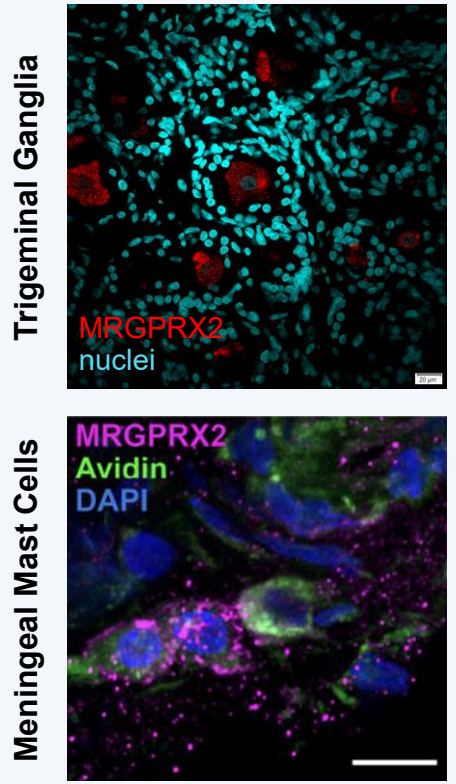
© Evommune, Inc.
evommune

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



© Evommune, Inc.

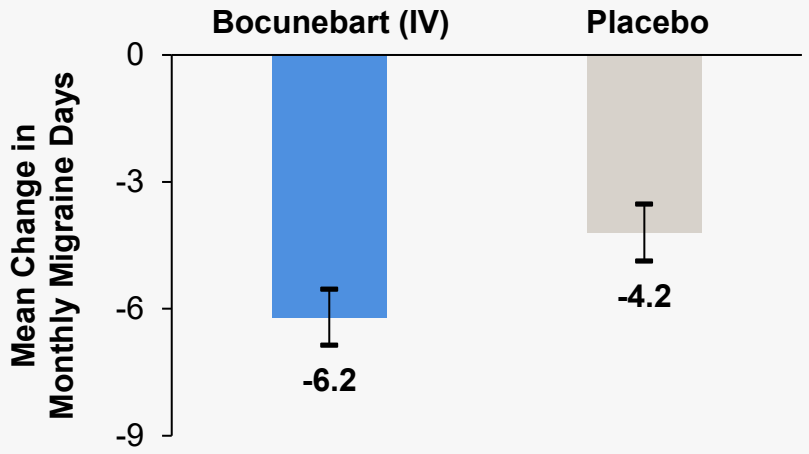
Source: Evommune internal data (trigeminal neurons; *in situ* hybridization on human tissue samples), PMID: 40712576 (meningeal mast cells)

evommune

PACAP Inhibition Achieves CGRP-Like Efficacy in Migraine Prophylaxis

Lundbeck's Bocunebart Reduced Monthly Migraine Days by ~2

in Patients Who Had Failed 2-4 Preventive Treatments



- ~2 MMD benefit vs. placebo, above the ~1-1.5 MMD of marketed CGRP inhibitors

Second Neuropeptide Axis Validated in Migraine Prevention

- PACAP is a key neuropeptide trigger of migraine attacks
- Antibody blockade reduced migraine frequency in controlled clinical trial
- Effect size exceeds the ~1-1.5 MMD benefit of approved CGRP therapies

Note: Lundbeck's bocunebart is a humanized mAb that neutralizes PACAP. Results above from Phase 2a trial in migraine prophylaxis (HOPE; N=237; single IV administration of bocunebart in patients who 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.

© Evommune, Inc.
evommune

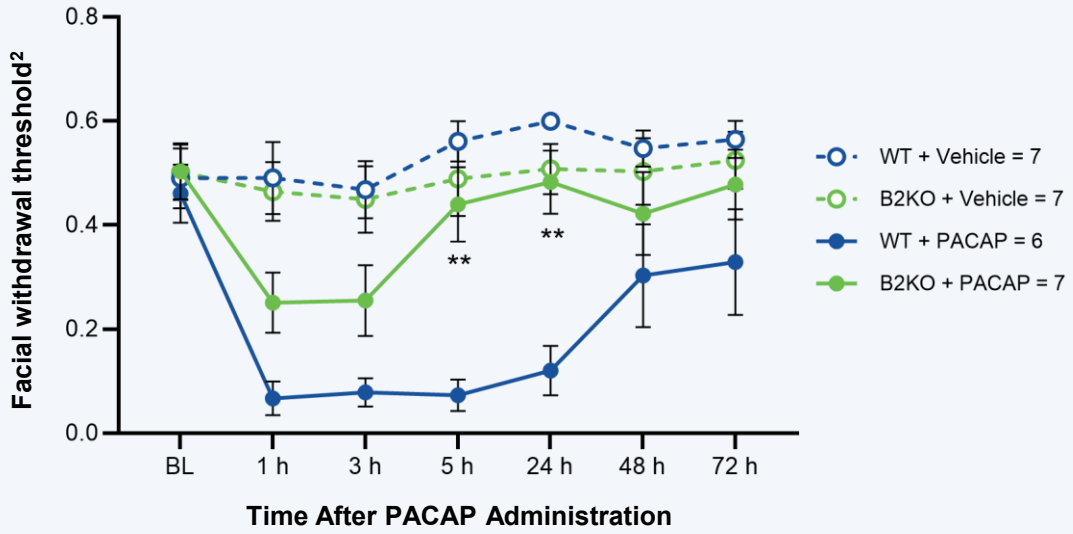
PACAP Triggers Migraine via MRGPRX2[1]

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

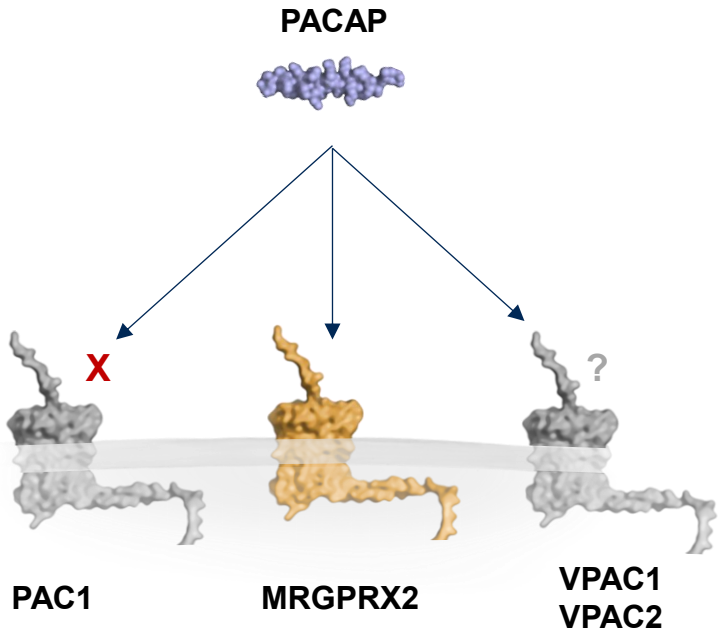


- *in vivo* data support functional role of MRGPRX2[1] signaling in migraine

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

© Evommune, Inc.

PACAP Impact in Migraine Is Primarily Through MRGPRX2

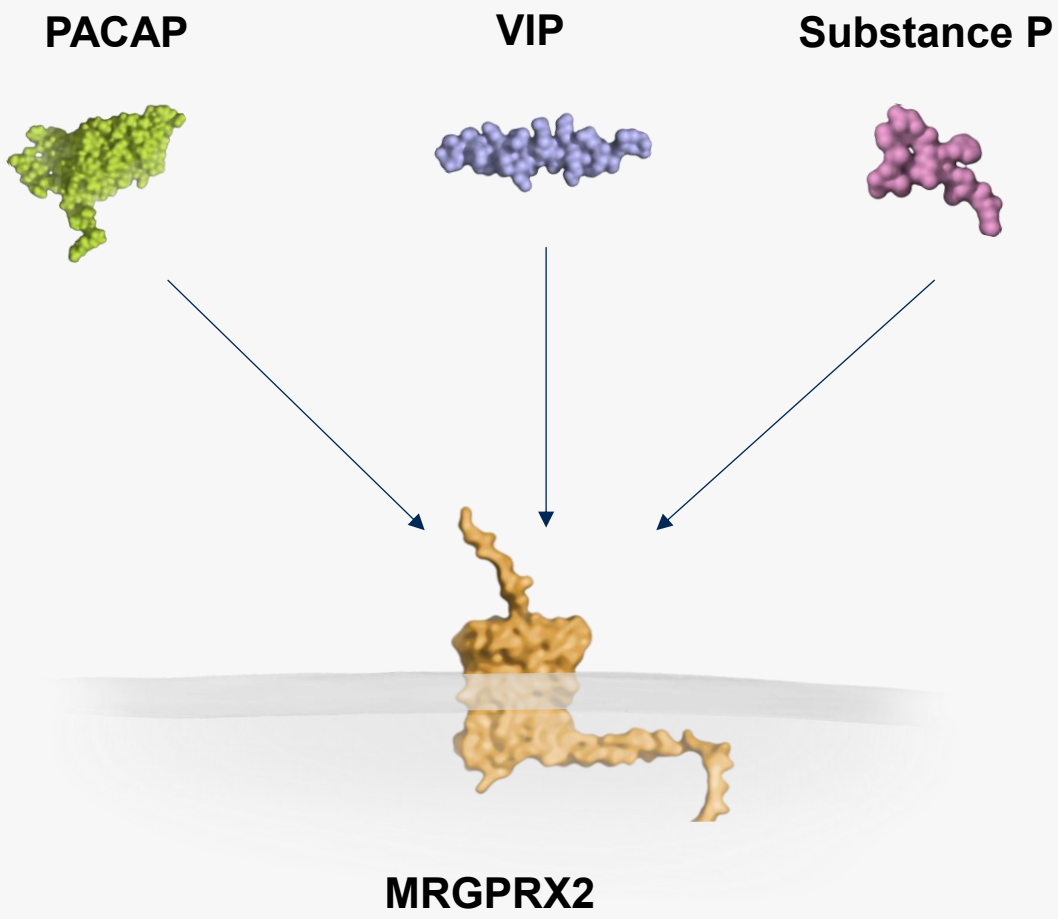


PACAP Receptor	Relevant Tissue Expression	Preclinical Evidence?	Clinical Validation In Migraine?
PAC1	**Neurons**[1]	Limited	✗
VPAC1 **VPAC2**	**Cranial vessels** Neurons (?)	Limited	*Not evaluated*
MRGPRX2	**Mast Cells** **Sensory Neurons**	✓	Top-line Data Expected 2027

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.

© Evommune, Inc.
evommune

3 Neuropeptides That Trigger Migraine Signal Through MRGPRX2



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

© Evommune, Inc.

Blocking MRGPRX2 Addresses Validated Migraine Triggers

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

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

© Evommune, Inc.
evommune

EVO756 Clinical Development Overview

Trial	Phase 1 Proof-of-Concept	Phase 2	Phase 2b
N	132	30	~330
Indication	Healthy Volunteers	CIndU	Migraine
Key Takeaways	• Well-tolerated • Clear target engagement • ~70% human skin penetration • PK supports full target coverage as low as 25mg BID	• Well-tolerated • Complete responses as early as week 1 • Clear POC achieved with 70% responders • Similar activity at 50mg BID and 300mg QD doses	**Trial Initiated in July 2026, Top-line Data Expected 2027**

Safety and Tolerability Established Across All Trials to Date including CSU and AD

© Evommune, Inc.

CIndU = Chronic inducible urticaria; CSU = Chronic Spontaneous Urticaria; AD = Atopic dermatitis

evommune

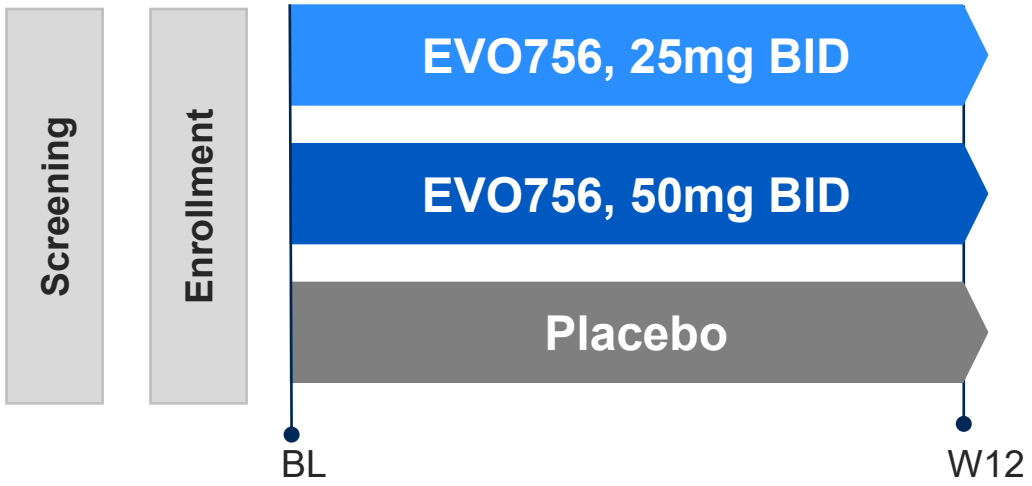
Phase 2b Dose-Ranging Trial in Migraine Prophylaxis

Top-line Data Expected in 2H 2027

Adults with Refractory Migraine ≥6 Days/Month

(N ≈ 330)

Randomized, Double-Blind, Placebo-Controlled Trial



Primary Endpoint

- Mean CFB in MMD

Key Secondary Endpoints

- ≥50%, ≥75% reduction in MMD
- CFB in MHDs
- CFB in monthly acute migraine medication use

Exploratory Endpoints

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

© Evommune, Inc.

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

evommune

EVO570: VAV1 Molecular Glue Degrader

JAK-like Oral Degrader with Potential for Improved Safety and a Best-in-Class Profile in Autoimmune Disease

© Evommune, Inc.
evommune

VAV1 Could Be the Next Major I&I Class

JAK-like oral convenience with immune-cell selectivity, starting in RA

JAK Inhibitors Today

~$12.5B[4]

2025 WW JAK class sales

- Oral, once-daily dosing
- Approved in 10+ I&I indications
- Boxed warning limits most use to 2L

How VAV1 Differs

Immune-cell restricted

VAV1 is expressed in immune cells; JAKs signal across most tissues

Targeted pathway

Acts downstream of T- and B-cell receptors, not broad cytokine signaling

Same oral convenience

Once-daily oral small molecule, like the JAKs

Where VAV1 can go

1 · START

~18M[1]

RA patients

Rheumatoid arthritis

Clear development and regulatory path

2 · SUPPLANT JAKS

200M+[2]

patients in JAK-approved diseases

10+ JAK-approved indications

Including Psoriasis · UC · Crohn's · PsA · AD

3 · GO BEYOND JAKS

6.5M+[3]

patients in diseases JAKs don't treat

MS · MG · Sjögren's · lupus

Novartis Ph2 underway in Sjögren's

All figures are global prevalence. 1. RA patients worldwide: 17.6M (GBD 2021; Lancet Rheumatol 2023). 2. Patients across JAK-approved I&I diseases, led by AD (2.54% global prevalence; GBD, JAAD Int 2024), plus IBD (UC and Crohn's), PsA, axSpA and alopecia areata. 3. Lupus 3.41M (Ann Rheum Dis 2023) and MS 3.1M (Atlas of MS 2024); excludes Sjögren's and MG. AD, atopic dermatitis; axSpA, axial spondyloarthritis; GCA, giant cell arteritis; GVHD, graft-versus-host disease; IBD, inflammatory bowel disease; I&I, immunology and inflammation; MG, myasthenia gravis; MS, multiple sclerosis; Ph2, Phase 2; PsA, psoriatic arthritis; RA, rheumatoid arthritis; UC, ulcerative colitis. 4. Evaluate Pharma, WW JAK class sales 2025 (incl. Rinvoq $8.3B per AbbVie FY2025; includes Jakafi heme indications). Boxed warning per FDA labels (2021 class update after ORAL Surveillance). Expression: Human Protein Atlas.

© Evommune, Inc.
evommune

VAV1 Biology Overlaps with Approved I&I Drug Mechanisms

Seven drug classes span nine indications; VAV1 has direct evidence in each

		T-cell mediated				T/B-cell mediated					
Drug class	**Example drugs**	**Psoriasis**	**UC**	**Crohn's**	**PsA**	**RA**	**MS**	**MG**	**SLE**	**Sjögren's**	**Route**
TNF	Humira, Enbrel	●	●	●	●	●					Inj.
IL-17A	Taltz, Cosentyx	●			●						Inj.
IL-6	Actemra, Kevzara					●		○			Inj.
FcRn	Vyvgart					○		●		○	Inj.
CD20	Ocrevus, Rituxan					●	●				Inj.
JAK	Rinvoq, Xeljanz, Olumiant		●	●	●	●			○		Oral
TYK2	Sotyktu	●		○	●				○	○	Oral
VAV1	**Direct evidence**	Genetics; expression	Colitis model	Colitis model	Ph2a/b posted	Arthritis model; genetics	EAE model; genetics	Genetics	Lupus model; patient cells	Lupus model; patient cells; Ph2a/b	**Oral**

● Approved in indication ○ Investigational

VAV1 row: direct evidence only (blank = none); Ph2a/b = trial underway / posted, no efficacy data. Source: Evaluate Pharma.

© Evommune CONFIDENTIAL

VAV1: JAK-Like with a More Selective Profile

	VAV1	JAK1	JAK2	JAK3	TYK2	T-cell depletion	B-cell depletion (Rituximab)
Expression is limited to immune cells	●	●	●	●	●	●	●
Modulates T-cell function	●	●	●	●	●	●	●
Modulates B-cell function	●	●	●	●	●	●	●
Phenotype of KO mouse	●	●	●	●	●	*N/A*	*N/A*
Modulates cytokine production	●	●	●	●	●	●	●
Immune cell chemotaxis	●	●	●	●	●	●	●
Safety	●	●	●	●	●	●	●

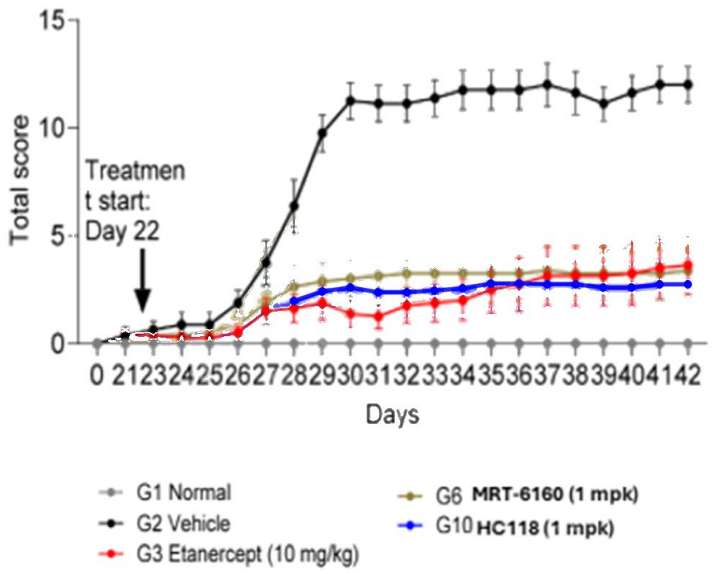
Key ● Strong yes ● Yes ● Somewhat ● Mostly no ● No

© Evommune CONFIDENTIAL

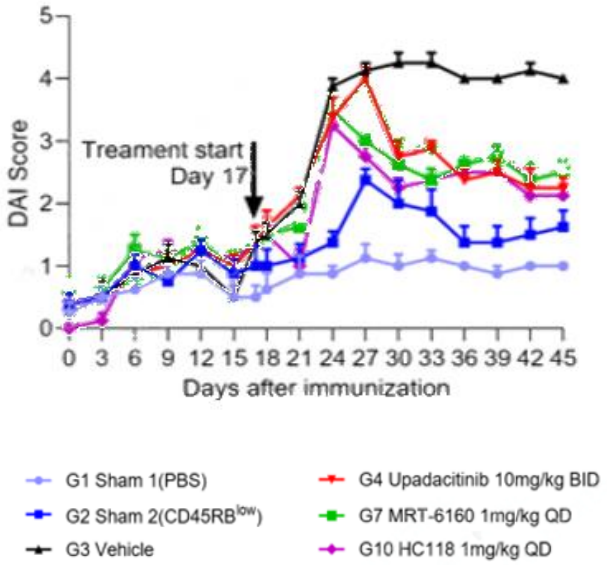
evommune

EVO570 Meets or Exceeds MRT-6160 and Standard of Care Across All Tested Disease Models

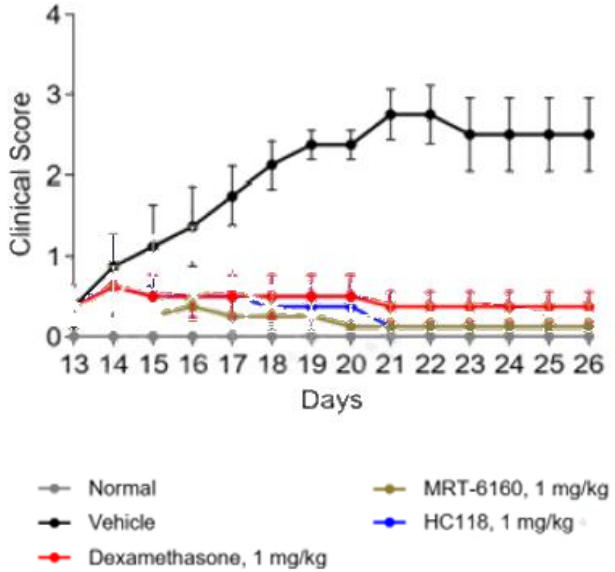
Rheumatoid Arthritis (RA)



Inflammatory Bowel Disease (IBD)



Multiple Sclerosis (MS)



EVO570 previously known as HC118

© Evommune CONFIDENTIAL
evommune

EVO570 Outperforms MRT-6160 in Preclinical Head-to-Head

~20× greater degradation potency with clean selectivity and a potential for low dosing

Property	MRT-6160 (Novartis)	EVO570	EVO570 Advantage
VAV1 DC50 (HiBiT, Jurkat)	7 nM	0.3 nM	~20× more potent
Max degradation (Dmax)	97%	99%	Deeper knockdown
CRBN binding IC50	1.4 µM	0.145 µM	~10× higher affinity
Ternary complex EC50	11 nM	2.7 nM	~4× stronger
T-cell IL-2 IC50	0.98 nM	0.08 nM	~12× more potent
B-cell IL-6 IC50	0.6 nM	0.06 nM	~10× more potent
Lymphoma REC-1 / LY10 IC50	18 / 19 nM	3 / 0.6 nM	~6-30× more potent
Durability of degradation	-	>85% at 7 days	QW-dosing potential
Selectivity / neo-substrates	Clean	Clean	Comparable
Safety window	~600-1,000×	>1,000×	Comparable / better

© Evommune, Inc.
evommune

Planned China Phase 1 Trial Design

Will support global Phase 2 program

Phase 1 FIH Randomized, Double-Blind, Pbo-Controlled SAD, MAD, and Food Effect Trial

N = 146

PART A: SAD Dosing

8 Cohorts: Oral dose of EVO570 or pbo (10 per group, 8:2)

- Cohort A8
- Cohort A7
- Cohort A6
- Cohort A5
- Cohort A4
- Cohort A3
- Cohort A2
- Cohort A1 (0.1mg)

PART B: MAD Dosing

4 Cohorts: Oral doses of EVO570 or pbo (12 per group, 10:2)

- Cohort B4
- Cohort B3
- Cohort B2
- Cohort B1

PART C: Food Effect

Oral dose of EVO570 in two-way crossover design (18 total, one dose fasted, one dose fed conditions)

Endpoints

Primary
Safety and tolerability of single and multiple oral doses, and food effect

Secondary
PK and PD: VAV1 degradation in T and B cells; ex vivo response to TCR and BCR stimulation (CD69, IL-2, IL-17A, IFNγ, IL-6)

Adaptive design
Safety Review Committee can adjust SAD and MAD doses based on prior cohorts

Projected timeline

China/US IND submission: Sep/Oct 2026, US opened Nov 2026

Phase 1 start: Jan 2027

Phase 1 data: ~Mid-2027

Phase 1b: exploring an RA patient cohort for early proof-of-concept

© Evommune, Inc.
evommune

EVO646: Oral TLR7/8 Antagonist

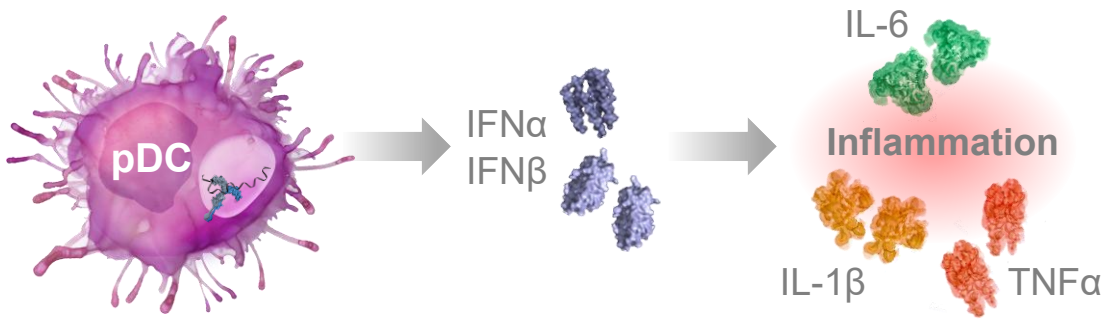
Potential Best-in-Class Dual Mechanism (Type I Interferon and Pathogenic B-Cell / Autoantibody) for Autoimmune Disease

© Evommune, Inc.

TLR7/8 Inhibition Suppresses Two Major Drivers of Autoimmune Disease

Type I Interferon Pathway

Suppresses pDC-driven type I interferon production

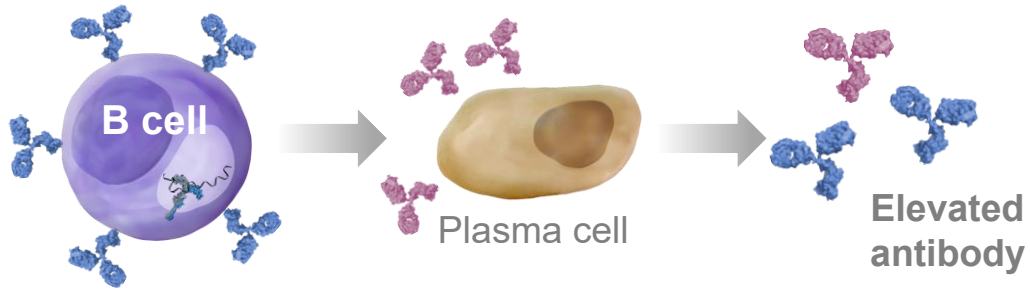


↓ **Type I IFN**

Pathway validated: anifrolumab (anti-IFNAR1), approved in SLE

Pathogenic B-Cell Pathway

Reduces B-cell activation, plasmablast differentiation, and autoantibody production



↓ **Autoantibodies**

Pathway validated: belimumab (anti-BLyS), approved in SLE

Single Target with Complementary Effects on Two Major Drivers of Autoimmune Disease

© Evommune, Inc.
evommune

Large Autoimmune Market Opportunity for TLR7/8

Strong Scientific Rationale for TLR7/8 in Autoimmune Diseases

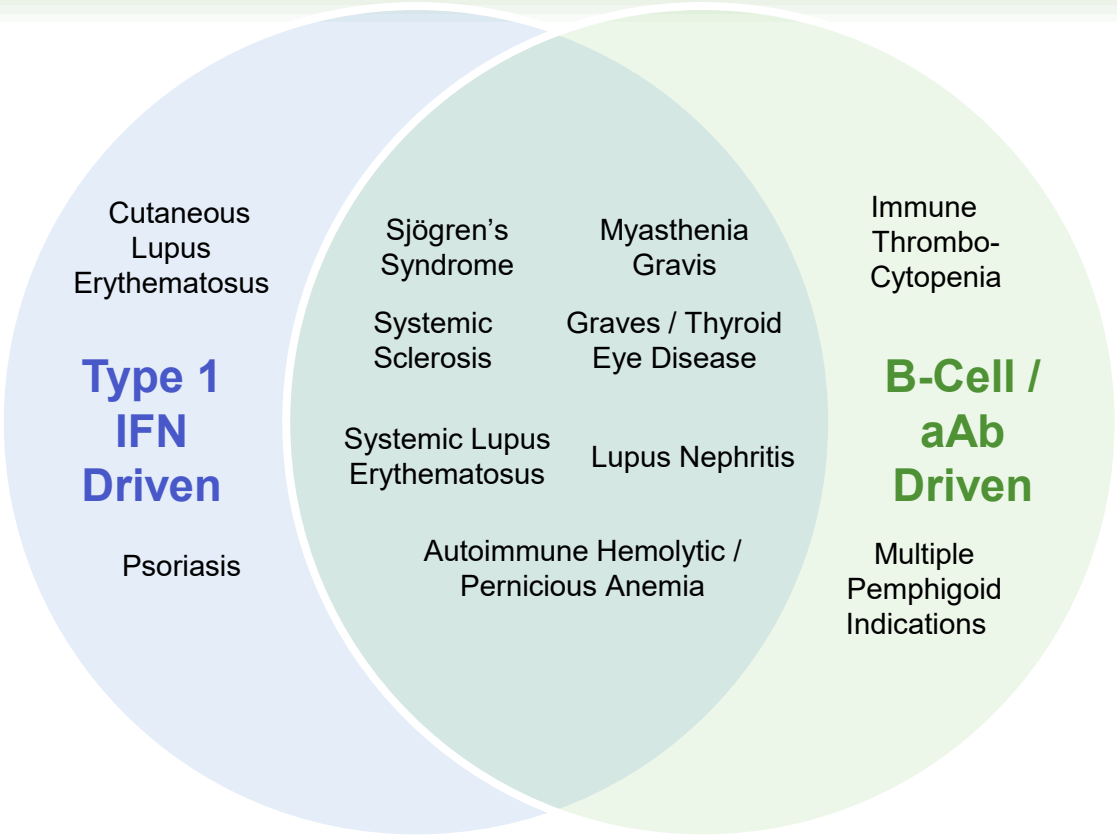
Human Genetics
Strong causal link of TLR7 to autoimmune disease

Preclinical Validation
TLR7-/- mice resistant to autoimmune/inflammatory models

Mechanistic Insights
Multimodal MoA: limiting type 1 interferons and pathogenic B-cells/auto-antibody production

Clinical POC
TLR7/8 inhibition demonstrated clinical benefit in CLE and SLE

TLR7/8 Potential Indications
6M Patients Initial Targeted Indications[1]



© Evommune, Inc.

1. Evommune internal estimates based on initial potential indications.

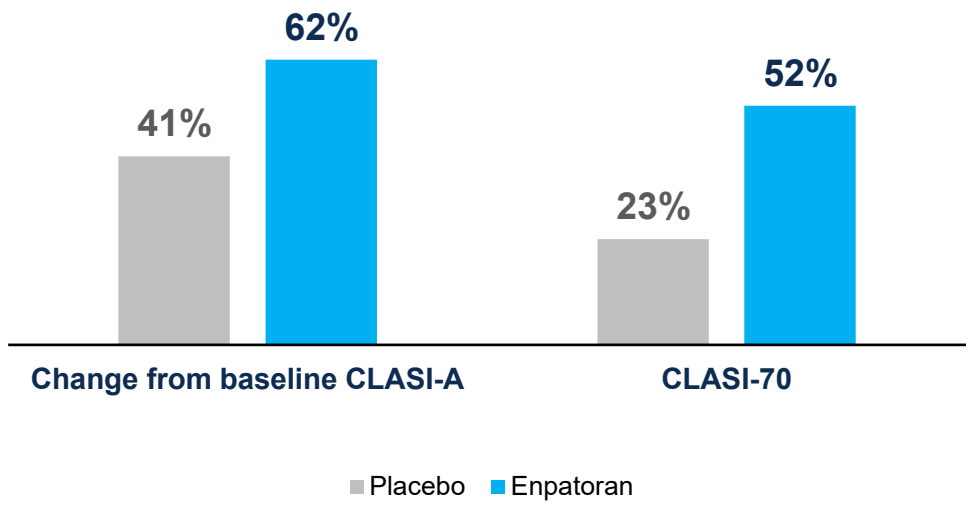
evommune

TLR7/8 Inhibition Is Clinically Validated in Lupus, with Oral Best-in-Disease Efficacy and Favorable Tolerability in Phase 2[5]

Phase 2 Proof-of-Concept Established in CLE[1]

Enpatoran Demonstrated Phase 2 Activity in CLE

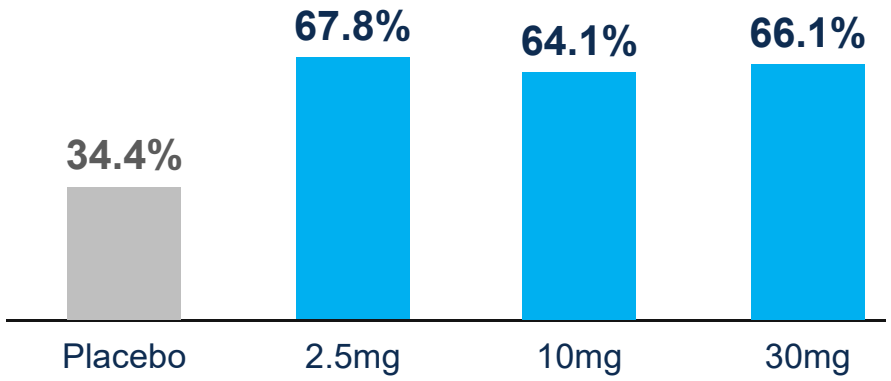
WILLOW Cohort A, Week 16[2]



~30-Point SRI-4 Benefit at Every Dose in Phase 2 SLE[3,4]

SRI-4 Response at Week 48

Randomized, placebo-controlled (N=248)



~30 pt SRI-4 benefit over placebo at every dose at Week 48

Broad Clinical Efficacy	Rapid IFN-GS Suppression	Favorable Tolerability
Skin, joints, steroid sparing	Near-maximal by Week 2	TEAEs: 5.9% vs. 12.9% PBO

© Evommune, Inc.

1. Enpatoran is an investigational product being developed by EMD Serono. 2. Enpatoran WILLOW Phase 2, Cohort A (ACR 2025). 3. Afimetoran is an investigational product being developed by Beeline Medicines. 4. Afimetoran Phase 2 in Systemic Lupus Erythematosus (Oct 2026). 5. Cross-trial comparison; no head-to-head data.

evommune

EVO646 Targets TLR7/8 and Spares TLR9

Genetics, disease models and two Phase 2 trials all point to TLR7/8 as the productive pair

HUMAN GENETICS

TLR7 gain of function causes lupus

- TLR7 variants drive disease in patients and in mice[1]
- No comparable gain-of-function case for TLR9
- EVO646 is designed for >100-fold selectivity over TLR9

DISEASE MODELS

TLR9 acts as a brake, not a driver

- Removing TLR7 protects lupus-prone mice; removing TLR9 worsens nephritis and shortens survival[2]
- That worsening disappears when TLR7 is also removed[3]

CLINICAL EVIDENCE

TLR7/8 blockade has already worked

- Afimetoran: Phase 2 SLE met SRI-4 across all three doses
- Enpatoran: Phase 2 activity in CLE
- Both programs block TLR7/8 only - no TLR9 engagement required for benefit

EVO646 blocks the two validated receptors and leaves TLR9 intact

1. Brown et al., Nature (2022); TLR7 gain-of-function variant causes human lupus. 2. Christensen et al., Immunity (2006). 3. Nickerson et al., PLoS One (2017).
Afimetoran (Beeline Medicines) and enpatoran (EMD Serono) are investigational and not developed by Evommune.

© Evommune, Inc.
evommune

EVO646: Selective Interferon and B-Cell Modulator in a Pill

Best-in-class potential

	EVO646 (Evommune)	Afimetoran (BMS/Beeline)	Enpatoran (Merck Serono)
Potency (nM) (HEK293T)	6.70	1.70	11.2
In vivo* activity Mouse PD	>80% inhibition @ 1mpk	>80% inhibition @ 1mpk	>80% inhibition @ 1mpk
Dosing	QD	QD	BID
Safety	>10 μM hERG 1/87 CEREP	9.29 uM hERG 6/87 CEREP	>10 μM hERG 2/87 CEREP

*acute R848 model → output highlights combined impact of potency, PPB, and bioavailability
CEREP hits = >50% inhibition @ 10 μM

© Evommune, Inc.

Key Milestones Ahead For Evommune

Championing Innovation for Chronic Inflammation

EVO301 IL-18BP	• Plan to initiate Phase 2b in AD with subcutaneous formulation in mid-2027 • Plan to initiate Phase 2a in UC in 1H 2027
EVO756 MRGPRX2 Antagonist	• Expect top-line data from Phase 2b migraine trial in 2027
EVO570 VAV1 MGD	• Expect Phase 1 HV biomarker data mid-2027 • Plan to initiate Phase 2 program 2H 2027
EVO646 TLR7/8 Antagonist	• Plan to initiate Phase 1 in 1H 2027 • Expect Phase 1 HV biomarker data in 2H 2027

~$290m Cash and Investments[1], Current Runway Into 2H 2029

© Evommune, Inc.

1. Pro Forma: $262.3M cash, cash equivalents and investments as of September 30, 2026, plus net proceeds of approximately $28.0 million from the November PIPE financing.

Thank You!

www.evommune.com

© Evommune, Inc.

evommune

Appendix

© Evommune, Inc.

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



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

cimzia (certolizumab pegol)

BOTOX OAB

eucrisa crisaborole ointment 2%

TNKase Tenecteplase

ACTIVASE ALTEPLASE

Qbrexza (glycopyrronium) cloth

Kyprolis (carfilzomib)

Lartruvo (OLARATUMAB) Injection 10 mg/mL

Portrazza necitumumab injection

Picato (ingenol mebutate) gel

SORIATANE (acitretin) Capsules

CYRAMZA (ramucirumab)

extina (ketoconazole) Foam, 2%

CAMZYOS (mavacamten) capsules

CLOVIQUE Trientine Hydrochloride Capsules, USP

Coseal Surgical Sealant

CellCept (mycophenolate mofetil)

Liletta

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%

© Evommune, Inc.

Footnotes: Acquisition prices from press releases

evommune