



Positive Top-line Data from Phase 2a Proof-of-Concept Trial of EVO301, a Novel IL-18 Binding Protein, in Patients with Moderate-to-Severe Atopic Dermatitis

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Affiliations and Disclosures

Affiliations

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- Dan Burge, M.D. — Evommune, Inc., Palo Alto, CA, USA
- Janice Drew, M.P.H. — Evommune, Inc., Palo Alto, CA, USA
- Polina Bukshpun — Evommune, Inc., Palo Alto, CA, USA
- Amanda Jacobsen Ph.D. — Evommune, Inc., Palo Alto, CA, USA
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- Jamie Harden, Ph.D. — Evommune, Inc., Palo Alto, CA, USA
- Mark Jackson, M.D. — Evommune, Inc., Palo Alto, CA, USA

Disclosures

- Mark Lebwohl has served as a key advisor on the trial to Evommune, Inc.
- Dan Burge, Janice Drew, Polina Bukshpun, Amanda Jacobsen, Jamie Harden, Mark Jackson are employees and shareholders of Evommune, Inc.
- Lorena Riol-Blanco is a former employee and shareholder of Evommune, Inc.

Funding and registration

- This study was sponsored by Evommune, Inc. Trial registration: NCT06723405.

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

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

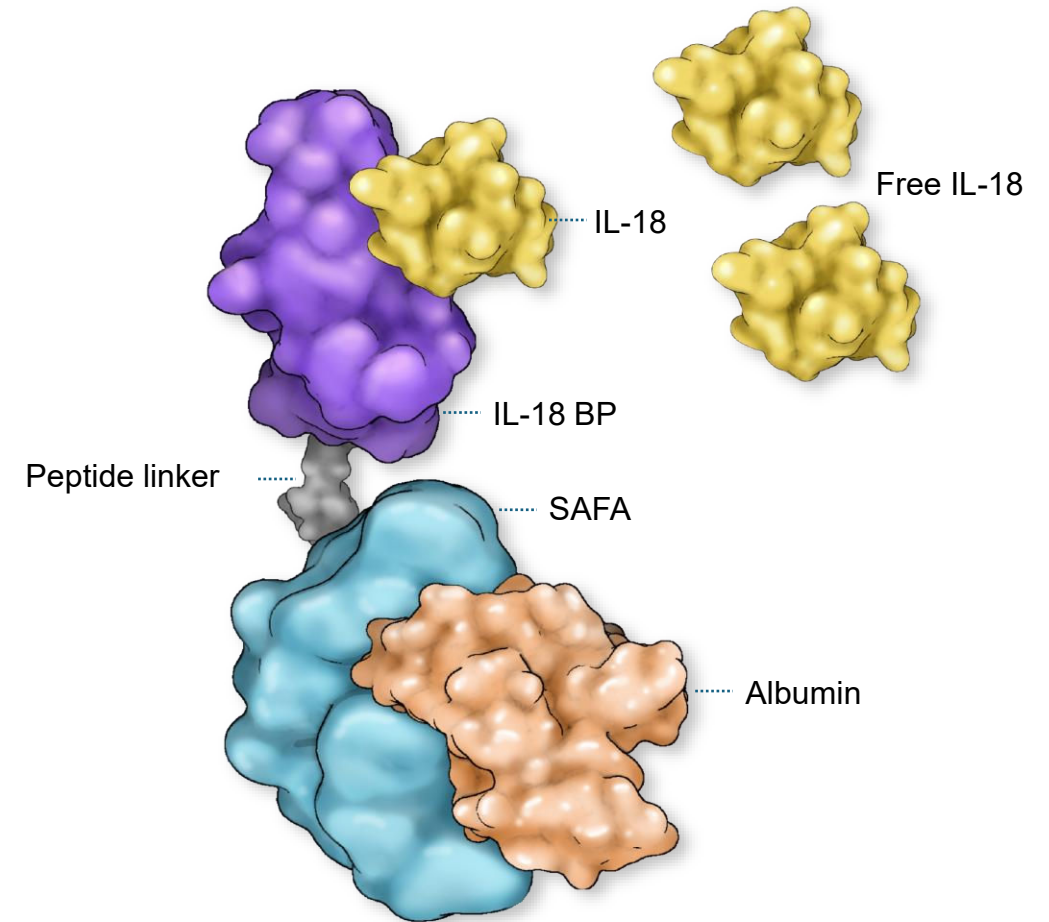


SAFAbody™ Platform Technology

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

IL-18 Binding Protein (IL-18BP)

- High binding affinity and specificity
- Native fully human sequence

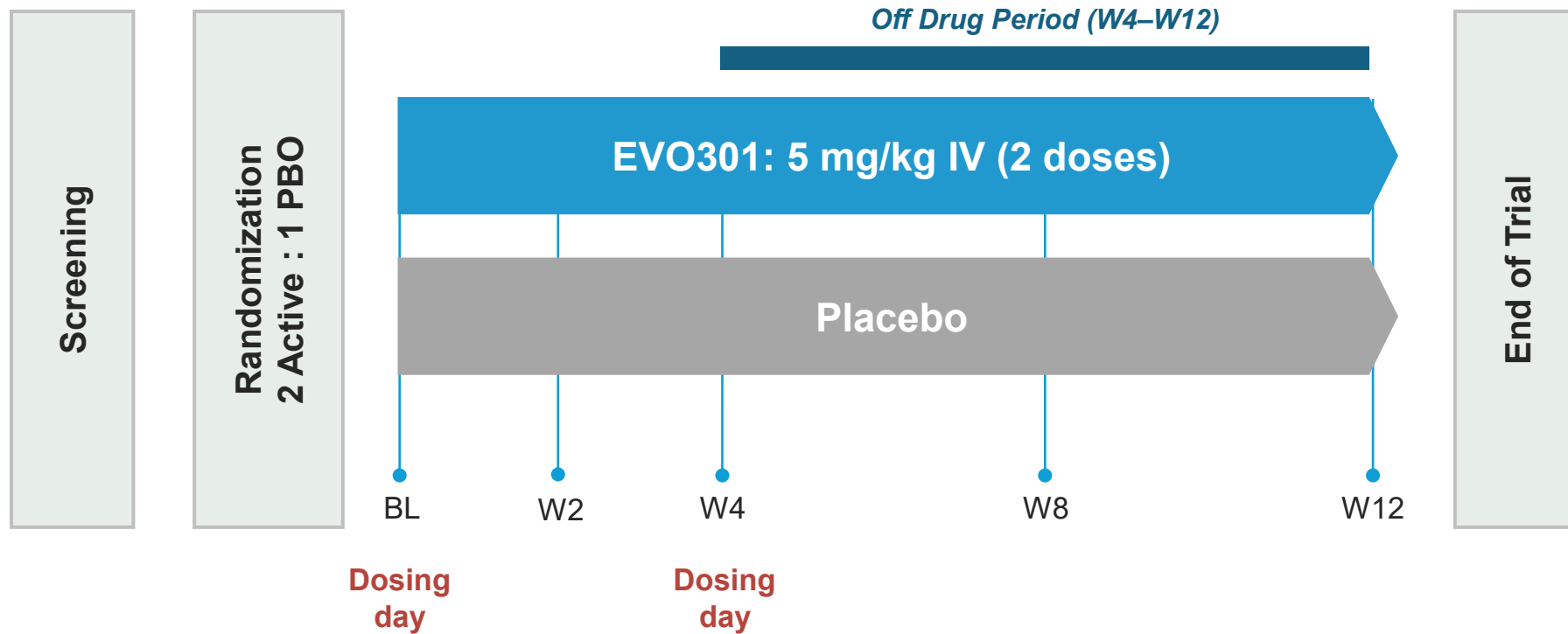


EVO301 Phase 2a Proof of Concept Trial Design

Patients received single dose administered twice of EVO301 or placebo, at Baseline and Week 4

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

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



AD Population

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

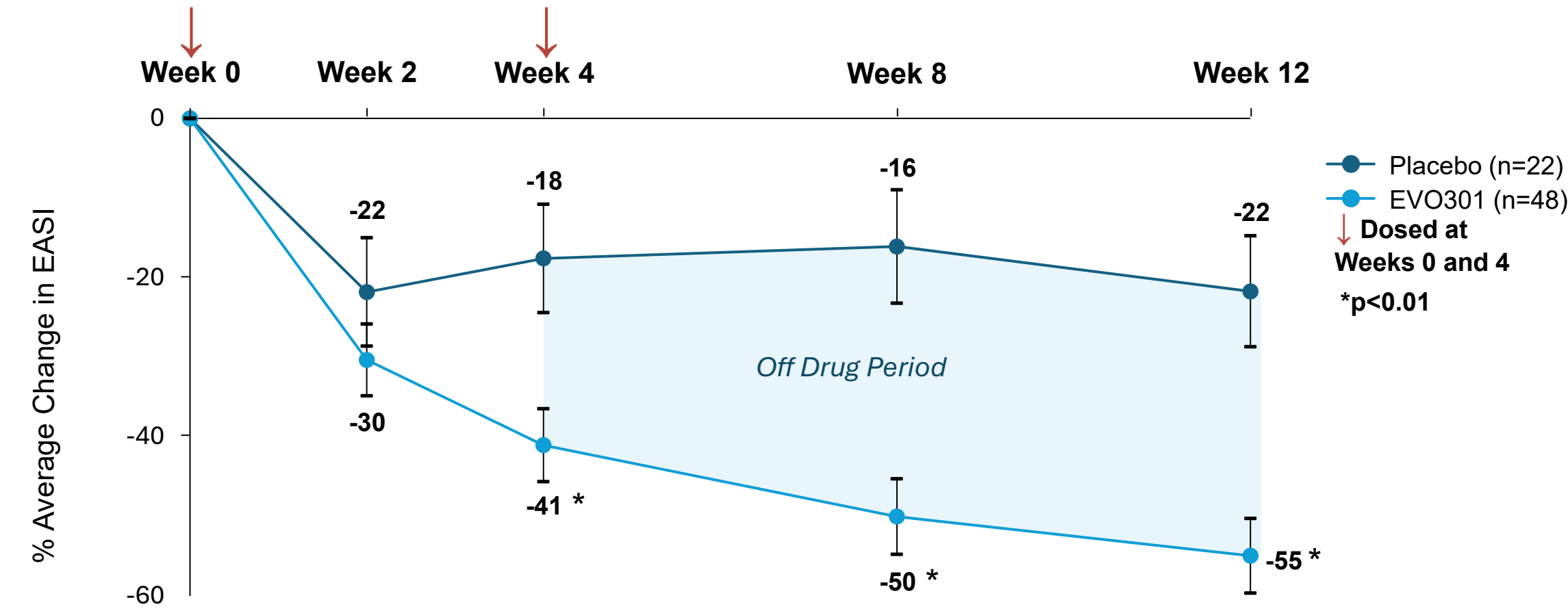
Primary Endpoint

- Percent change from EASI at Week 12 (Bayesian)

Pharmacokinetics

Target Engagement

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



EVO301 Demonstrated Comparable Activity at 12 Weeks with Marketed Biologics at 16 Weeks

Phase 2 POC Trial in AD: Efficacy (Frequentist)

- EASI-50 and EASI-75 on par with and/or trending to commercially available products with single dose administered twice
- Phase 2a signal finding study successful in meeting primary endpoint; secondary endpoints trending positive in line with the primary efficacy readout
- Supports further Phase 2b development

EASI-50 Response

	Visit		EVO301	Placebo	p-Value
	Week 2		25.0%	18.2%	0.5297
	Week 4		39.6%	13.6%	0.0291
	Week 8		43.8%	22.7%	0.0824
	Week 12		62.5%	22.7%	0.0014

EASI-75 Response

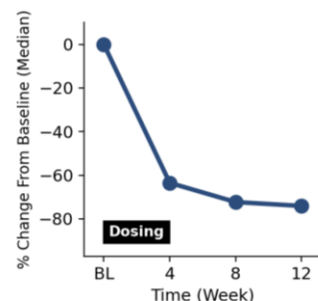
	Visit	EVO301	Placebo	p-Value
	Week 2	8.3%	0%	NE
	Week 4	18.8%	0%	NE
	Week 8	18.8%	4.5%	0.1481
	Week 12	29.2%	9.1%	0.0686

EVO301 reduction in Th2, Th1, Th17, and Th22 biomarkers reflects broad anti-inflammatory MoA

Type 2 Inflammation

TARC¹ (CCL-17)

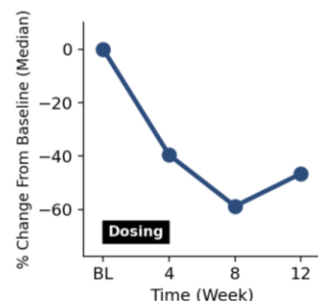
Validated biomarker for Th2 inflammation and disease severity



74%

CCL-22¹

Key cytokine responsible for Th2 cell activation and amplification of inflammation

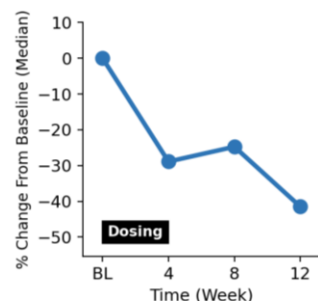


59%

Type 1 Inflammation

CXCL9²

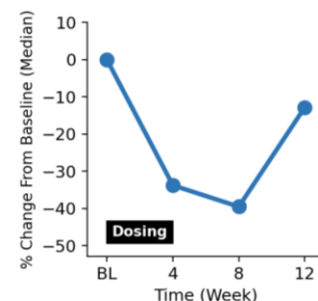
Highly specific cytokine upregulated by immune cells in response to IFN-γ



41%

CXCL10²

Validated biomarker for Th1 inflammation, disease severity and treatment responses

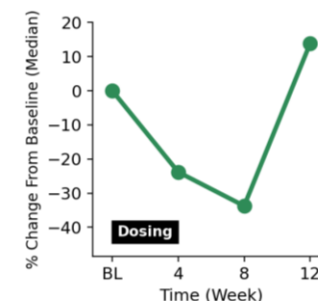


39%

Type 17/22 Inflammation

S100A12³

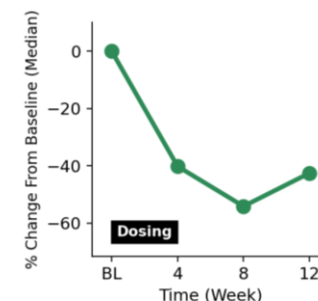
Highly specific alarmin downstream of IL-17 signaling



34%

IL-22⁴

Key cytokine involved in AD skin barrier dysfunction and keratinocyte proliferation



54%

Maximum inhibition post-EVO301 treatment

¹Th2-high subjects (n=11): CCL-17 ≥ 929 pg/mL, CCL-22 ≥ 2,348 pg/mL; ²Th1-high subjects (n=9): CXCL9 ≥ 56 pg/mL, CXCL10 ≥ 205 pg/mL; ³Th17-high subjects (n=12): S100A12 ≥ 159 pg/mL mL;

⁴all EVO301 – treated subjects (n = 47); Maximum cytokine reduction represents median % change from baseline at relevant time point.

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 (Common with Other Biologics in AD)**

Summary

Efficacy	• EVO301 demonstrated comparable activity at 12 weeks (after only dosing at week 0 and 4) to marketed biologics at 16 weeks • Primary endpoint met statistical significance as early as week 4 • Data support dose optimization in Phase 2b
Pharmacokinetics	• PK/PD data supports Q4 week dosing regimen, with Q8-Q12 week maintenance dose potential
Biomarkers	• Total IL-18 levels demonstrated robust target engagement • Decrease in Th2, Th1, Th17 and Th22 biomarkers reflects broad anti-inflammatory MoA • Robust decrease in Th2 biomarkers highlights IL-18 mediating of Th2 inflammation in AD
Safety	• Well-tolerated and no safety signals were observed • No conjunctivitis

Thank you

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