Eblasakimab Improves Multiple Disease Measures in Adult Patients with Moderate-to-severe Atopic Dermatitis in a Randomized, Double-blinded, Placebo-controlled, Phase 1 Study

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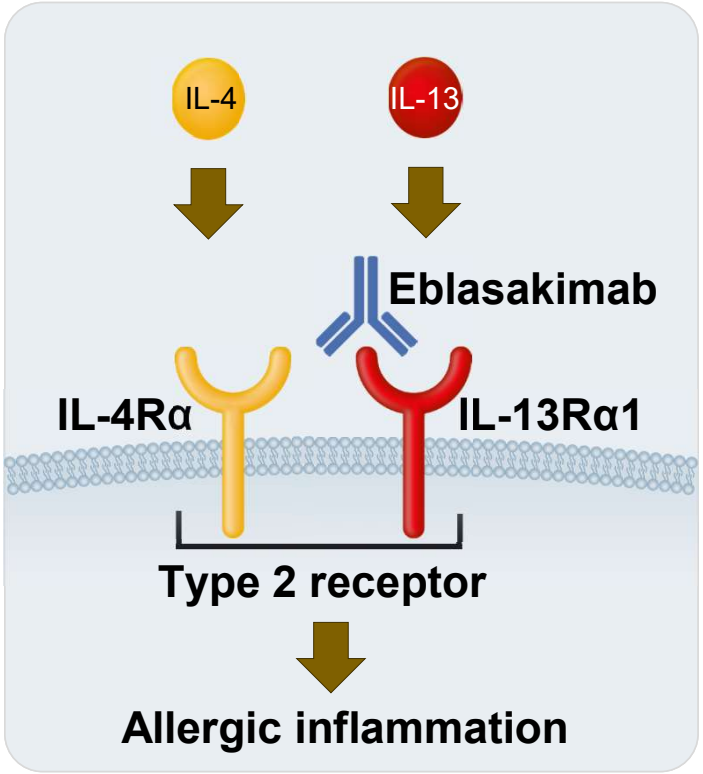
Disclosures

- ST has received honoraria as a consultant and/or advisory board member from ASLAN Pharmaceuticals
- MG has received grants, personal fees, and/or nonfinancial support from Pfizer Inc, AbbVie, Inc, Akros Pharma, Inc, Amgen, Inc, Arcutis Biopharmaceuticals, Inc, Bristol-Myers Squibb Company, Boehringer Ingelheim, Celgene Corporation, Coherus BioSciences, Inc, Dermira, Inc, Eli Lilly and Company, Galderma SA, GlaxoSmithKline, Glenmark Pharmaceuticals, Janssen Pharmaceutica, Kyowa Kirin Co, Ltd, LEO Pharma A/S, MedImmune, LLC, Merck & Co, Novartis International AG, Regeneron Pharmaceuticals, Inc, Roche Diagnostics, Sanofi Genzyme, UCB, and Bausch Health Companies, Inc.
- ES has consulted for Pfizer Inc, AbbVie, Inc, Celgene Corporation, Eli Lilly and Company, Galderma SA, GlaxoSmithKline, LEO Pharma A/S, Menlo Therapeutics, and Regeneron Pharmaceuticals, Inc, and served as principal investigator for AbbVie, Inc, GlaxoSmithKline, LEO Pharma A/S, Novartis International AG, Regeneron, Pharmaceuticals, Inc, Tioga Pharmaceuticals, and Vanda Pharmaceuticals, Inc.
- KAV & FC are employees of ASLAN Pharmaceuticals Ltd
- ASLAN Pharmaceuticals was the sponsor of this study (NCT04090229)

Rationale

- IL-4 and IL-13 are **pivotal cytokines** involved in the pathogenesis of allergic diseases, including atopic dermatitis (AD)
- However, **not all patients achieve optimal responses** with current treatment options and safety concerns exist for these therapies
- Eblasakimab is a first-in-class molecule with a novel MOA, **selectively targeting IL-13Rα1** and blocking signaling of both IL-4 and IL-13 through the Type 2 receptor
- By selectively targeting the Type 2 receptor and **sparing the Type 1 receptor** there is the potential to avoid unwanted effects

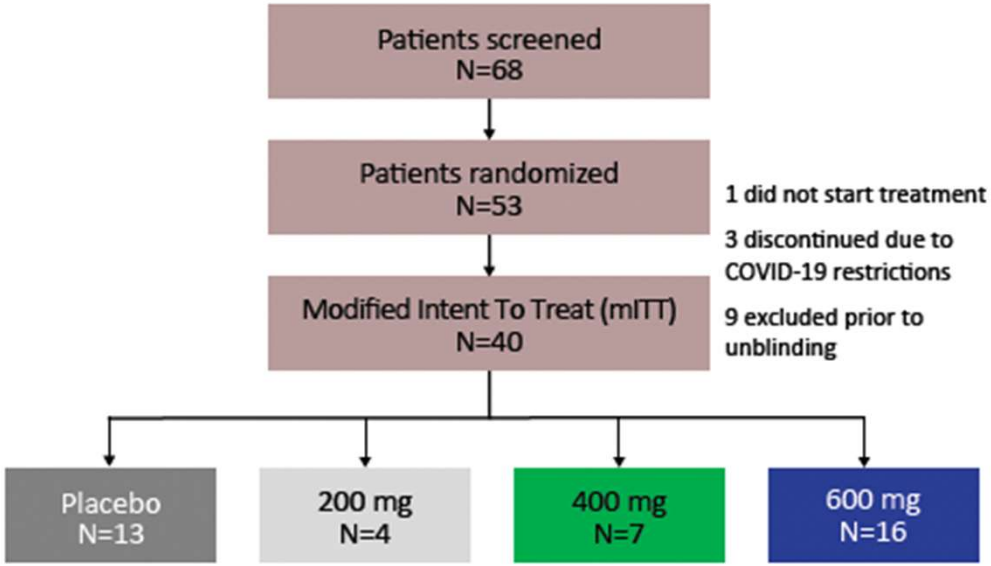


Study Design

- Double-blinded, randomized, placebo-controlled study
- Patients dosed weekly via subcutaneous injection for 8 weeks with a 12-week safety follow up period

Key inclusion criteria:

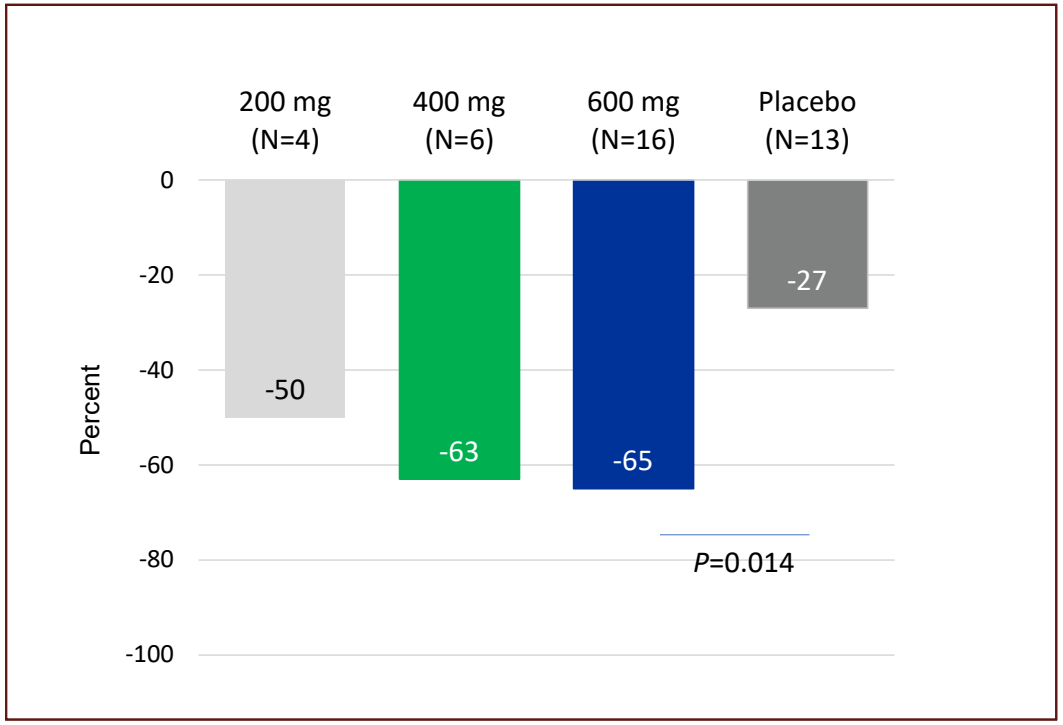
- Chronic AD present for ≥3 years before screening visit
- EASI ≥16 at screening and baseline
- IGA score ≥3 (scale of 0 to 4) at screening and baseline
- ≥10% body surface area (BSA) of AD involvement at screening and baseline



Statistical Considerations

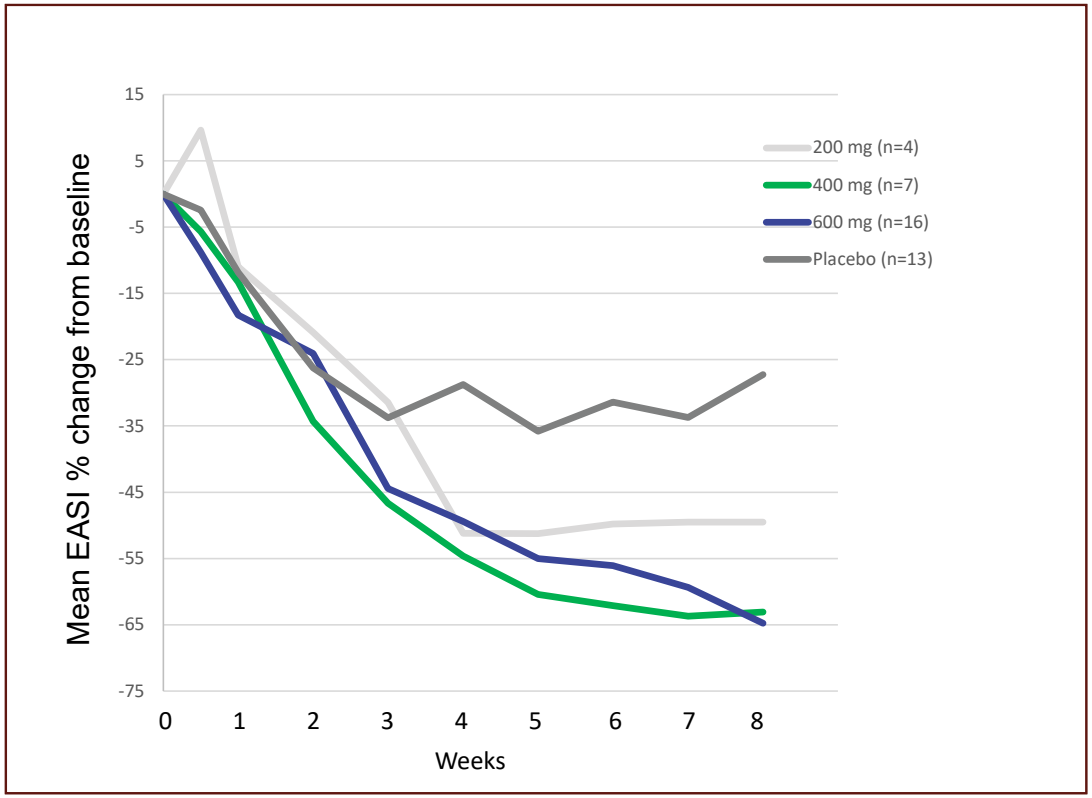
- mITT analysis set: all randomized and treated subjects, excluding 3 prematurely discontinued due to COVID protocols
- LOCF imputation applied to account for missing data
- Designed to have 80% power to detect a true mean difference of 39% in the %change from baseline in EASI score between eblasakimab 600 mg compared with placebo, based on a one-sided 5% significance level

Mean %CFBL: EASI Improvement at Week 8



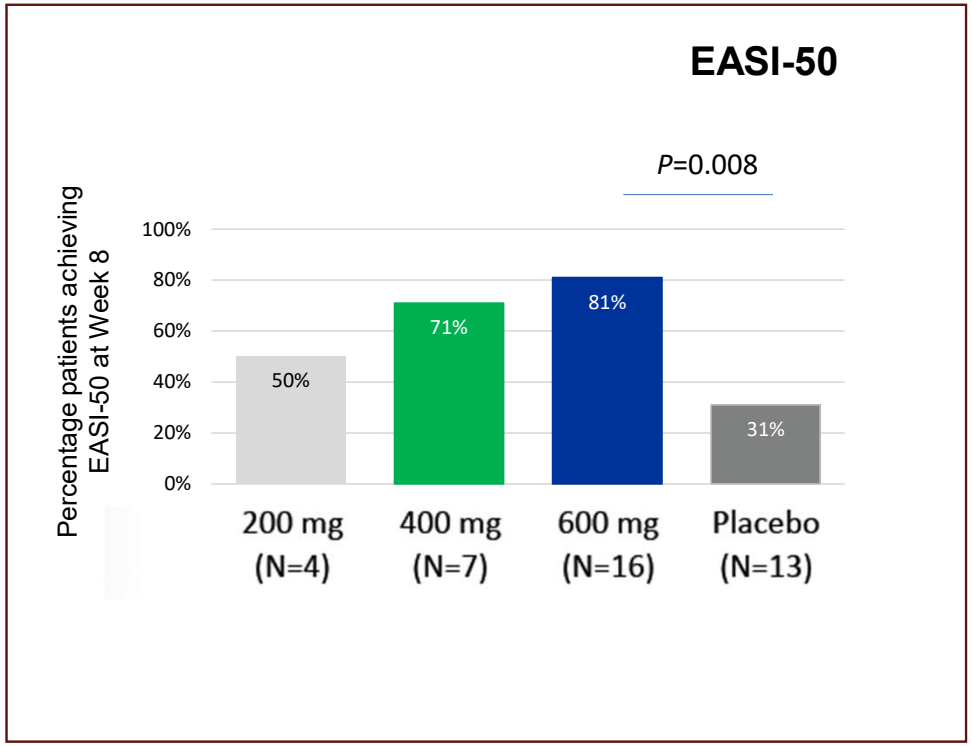
Response in the modified Intent-to-Treat Population (statistical comparison at Week 8 only)

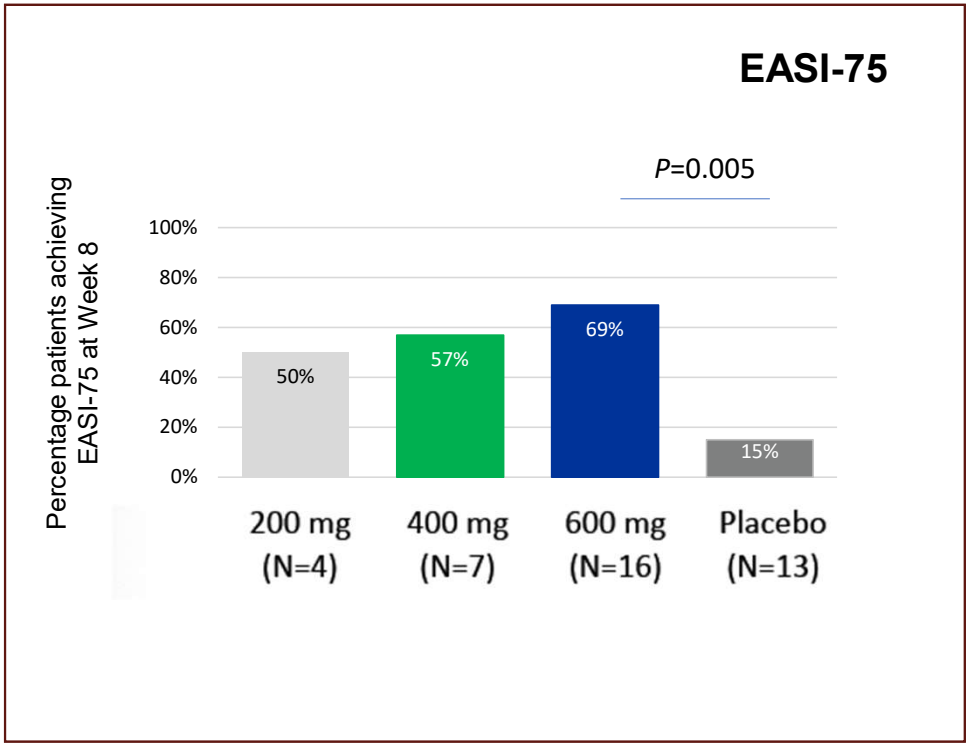
Mean %CFBL: EASI Improvement Over Time



Tine course response in the modified Intent-to-Treat Population (statistical comparison at Week 8 only)

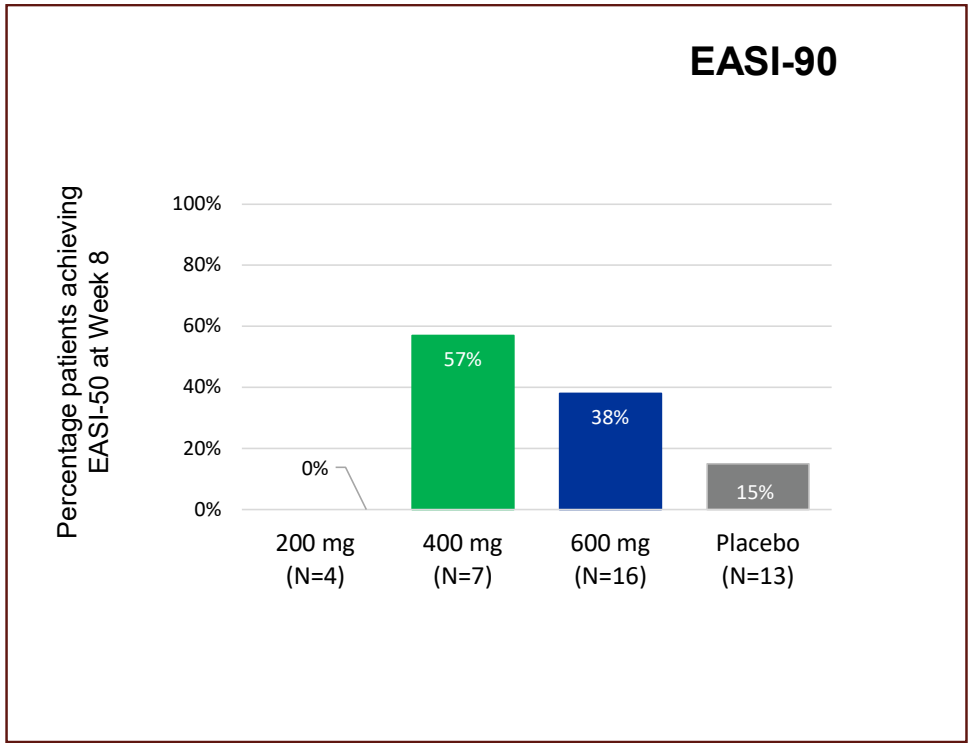
Key Secondary Data – EASI-50 and EASI-75 at Week 8





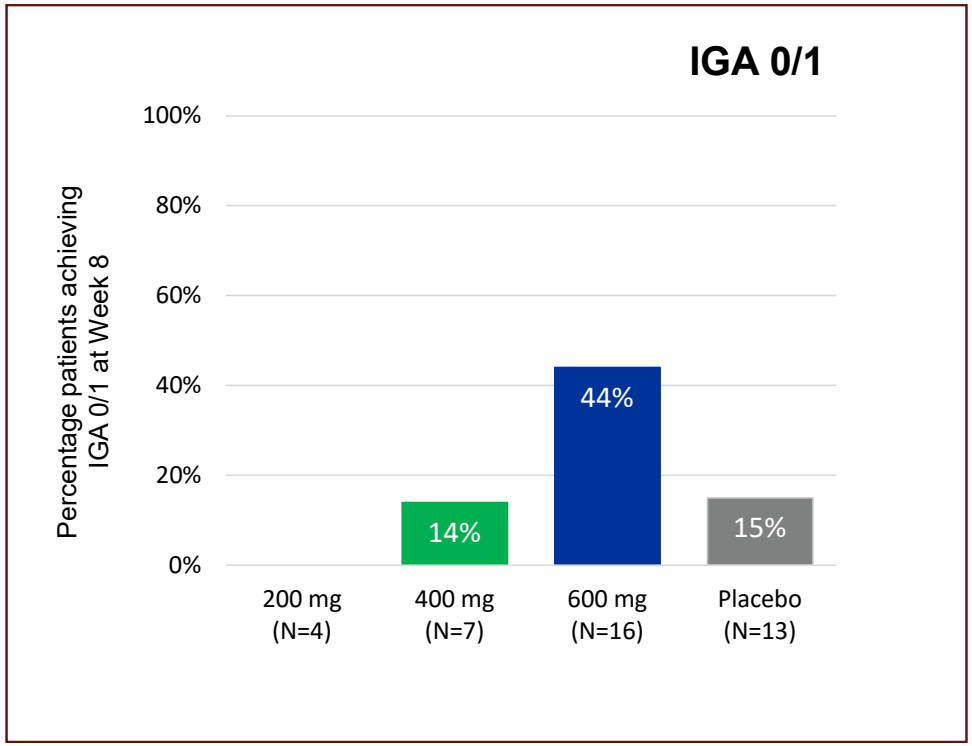
Response in the modified Intent-to-Treat Population (statistical comparison at Week 8 only)

Key Secondary Data – EASI-90 at Week 8



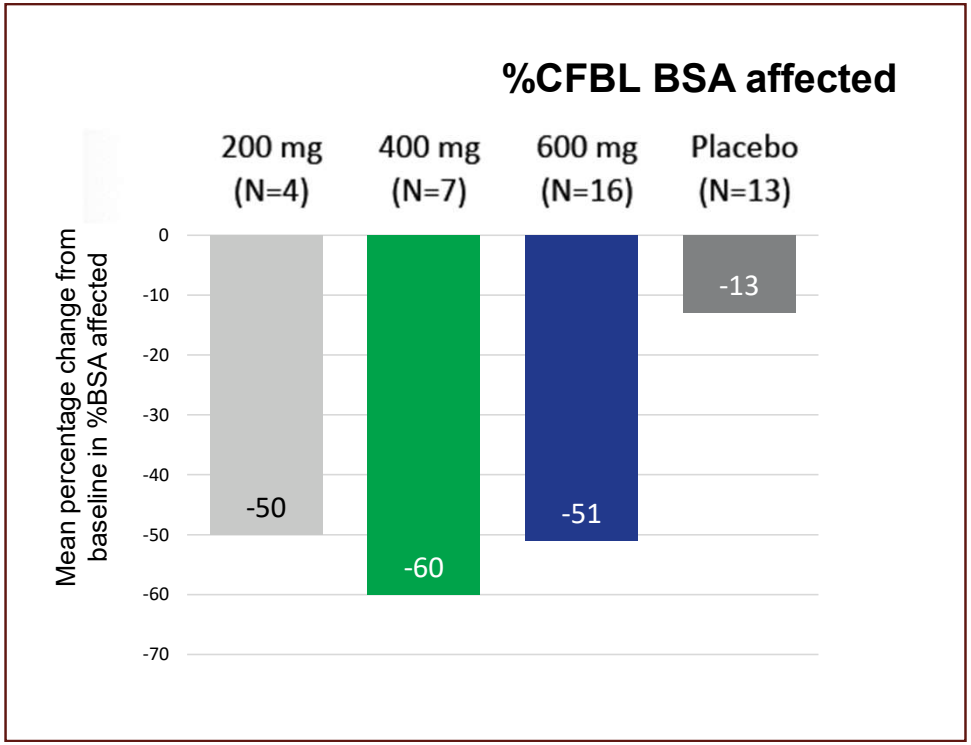
Response in the modified Intent-to-Treat Population (statistical comparison at Week 8 only)

Key Secondary Data – IGA 0/1 at Week 8



Response in the modified Intent-to-Treat Population (statistical comparison at Week 8 only)

Key Secondary Data – BSA Affected at Week 8



Response in the modified Intent-to-Treat Population (statistical comparison at Week 8 only)

Well-Tolerated Safety Profile

- Rates of moderate-to-severe AEs were **comparable** between 600 mg and placebo
- AEs related to treatment were **similar between groups**
- AEs leading to treatment discontinuation were **higher in the placebo group**
- 1 SAE reported in the study (mild abdominal pain, 400 mg); considered **unrelated to treatment**
- No deaths reported

	All treated patients minus excluded site (N=43)			
	200 mg (N=5)	400 mg (N=8)	600 mg (N=16)	PBO (N=14)
Any	5 (100%)	8 (100%)	9 (56%)	8 (57%)
Related	5 (100%)	6 (75%)	6 (38%)	7 (50%)
Moderate/ severe	2 (40%)	3 (38%)	4 (25%)	5 (36%)
Serious adverse event (SAE)	0 (0%)	1 (13%)	0 (0%)	0 (0%)
Leading to discontinuation	0 (0%)	1 (13%)	2 (13%)	3 (21%)
Drug-related AEs of interest:				
• Injection site reaction	4 (80%)	3 (38%)	3 (19%)	2 (14%)
• Allergic conjunctivitis	0 (0%)	1 (13%)	2 (13%)	0 (0%)

Conclusions

- Eblasakimab targets IL13Rα1, one half of the IL-13 receptor, offering a novel approach for blocking IL-13 signaling
- Eblasakimab also partially interferes with IL-4 signaling, since IL-4 can bind and signal through IL-4Rα/IL-13Rα1 (Type 2 receptor)
- **Statistically significant improvements versus placebo** across a range of endpoints demonstrate proof of concept for eblasakimab in AD
- That these significant improvements were seen within the 8-week study period offers the **potential for a greater magnitude of effect with prolonged treatment**
- Further data will be available following the **completion of a Phase 2b study** (NCT05158023)