Safety and efficacy of eblasakimab, an interleukin 13 receptor α1 monoclonal antibody, in adults with moderate-to-severe atopic dermatitis: A phase 1b, multiple-ascending dose study

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Background: Eblasakimab, an interleukin (IL)-13 receptor α1 antagonist, blocks IL-4 and IL-13 signaling through the type 2 receptor.

Objective: The safety and efficacy of eblasakimab was evaluated in adults with moderate-to-severe atopic dermatitis (AD).

Methods: In this phase 1b randomized, double-blinded study, 52 patients with moderate-to-severe AD received weekly subcutaneous injections of eblasakimab 200, 400, or 600 mg, or placebo for 8 weeks. Primary outcome was the incidence of treatment-emergent adverse events. Secondary outcomes included percentage change in the Eczema Area and Severity Index from baseline; Eczema Area and Severity Index improvement of at least 50%, 75%, or 90% from baseline; and percentage change in the peak-pruritus numeric rating scale score from baseline.

Results: Treatment-emergent adverse events were reported in 47% placebo and 71% eblasakimab patients; most were considered mild or moderate and did not lead to study discontinuation. At week 8 eblasakimab 600 mg showed statistically significant improvement in mean percentage change in Eczema Area and Severity Index versus placebo (−65% vs −27%, P = .014). Other key secondary physician- and patient-reported end points were met.

Limitations: Longer studies are required to confirm eblasakimab safety and efficacy in AD patients.

Conclusions: Treatment of adults with moderate-to-severe AD with eblasakimab was well-tolerated and associated with significant clinical improvements versus placebo. (J Am Acad Dermatol 2024;90:504-11.)

Key words: atopic dermatitis; EASI; eblasakimab; IL-4; IL-13; moderate-to-severe AD; pruritus; type 2 inflammation.

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OVERVIEW

Atopic dermatitis (AD), the most common chronic inflammatory skin disease,[1] is associated with a heavy burden on life.[2] Type 2 inflammation is a key feature of AD, driven in part by dysregulation of helper T cell type 2 (T_H2) cells and type 2 innate lymphoid cells. In turn, activation of these cells leads to increased levels of interleukin (IL)-4 and IL-13 in the skin and blood.[1]

Moderate-to-severe AD often requires systemic therapy and despite recent approvals, therapeutic options remain limited. Long-term use of corticosteroids and immunosuppressive drugs is not recommended due to their poor tolerability and unfavorable adverse event profiles.[3] The launch of dupilumab, a fully human monoclonal antibody, offered a new treatment approach targeting the IL-4 receptor α (IL-4Rα), thereby blocking IL-4 signaling through the type 1 receptor and both IL-4 and IL-13 signaling through the type 2 receptor.[4-6] Although dupilumab was a treatment advance, not all patients respond sufficiently to therapy, others lose response with continued treatment,[7] and injection site reactions and conjunctivitis occur in some patients,[8,9] possibly influenced by inhibition of the type 1 receptor.[10] Notably, efficacy of other drugs inhibiting the action of IL-13 on the type 2 receptor, such as tralokinumab[11] and lebrikizumab,[10,12] elucidated a key role for IL-13 in AD pathogenesis.[11-15]

Recently, several oral Janus kinase (JAK) inhibitors were approved for treatment of moderate-to-severe AD. These drugs target and block signaling of multiple cytokines mediated by the JAK-signal transducer and activator of transcription pathway, that is upregulated in a number of immune-mediated inflammatory diseases.[16-18] Upadacitinib and abrocitinib were the first oral JAK-1 inhibitors approved by the FDA for moderate-to-severe AD. However, concerns that there may be an increased risk of serious adverse events[16] with these medicines led to recommended measures including multiple boxed warnings in the United States prescribing information and guidance from the European Medicines Agency. These safety concerns limit their use, especially in the elderly population. Indeed, despite recent advances in treatment for patients with AD, there remain unmet needs for efficacious systemic treatments with a favorable benefit-risk profile.

CAPSULE SUMMARY

- New systemic treatments for atopic dermatitis are needed.
- Eblasakimab, a fully human monoclonal antibody that binds the interleukin 13 receptor α1 subunit with high affinity, significantly improved physician- and patient-reported measures of atopic dermatitis severity versus placebo over 8 weeks of treatment in this study, warranting further investigation.

Eblasakimab is a novel, fully human monoclonal antibody that binds IL-13Rα1 with high affinity (K_D 453 ± 20.7 pM,[19] assessed using Biacore 4000[20]) and prevents the formation of the IL-13Rα1/IL-4Rα heterodimer receptor signaling complex; this leads to blockade of both IL-4 and IL-13 signaling exclusively via the type 2 receptor.[21] Eblasakimab's mechanism of action is illustrated in Supplementary Information #1 (available via Mendeley at https://data.mendeley.com/datasets/g6m6884znc/1). Here, results of a phase 1b multiple-ascending dose, proof-of-concept study assessing the safety and efficacy of weekly eblasakimab for 8 weeks in adults with moderate-to-severe AD are reported.

METHODS

The study was undertaken in accordance with the Declaration of Helsinki and/or all relevant regulations of Title 21 of the United States Code of Federal Regulations, in compliance with International Council for Harmonization (ICH) and Good Clinical Practice guidelines and according to the appropriate regulatory requirements in the countries where the study was conducted. Consolidated Standards of Reporting Trials (CONSORT) reporting guidelines were followed.

Study design and patients

The study was designed as a multiple-ascending dose escalation in 3 cohorts of patients, followed by a cohort expansion to further confirm the safety and tolerability of the selected dose before investigation in phase 2 studies. The study enrolled patients across 10 sites in the United States, Australia, and Singapore and was conducted from September 9, 2019, to October 28, 2021. The study protocol is presented in Supplementary Information #2 (available via Mendeley at https://data.mendeley.com/datasets/g6m6884znc/1).

Following screening, eligible patients were given a unique randomization number via the Randomization and Trial Supply Management system and randomized 3:1 in 3 cohorts to receive subcutaneous injections of eblasakimab in ascending doses (200, 400, or 600 mg), or placebo, every week for 8 weeks. Following interim analysis

Abbreviations used:

AD:	atopic dermatitis
BSA:	body surface area
EASI:	Eczema Area and Severity Index
IGA:	Investigator's Global Assessment
IL:	interleukin
JAK:	Janus kinase
NRS:	Numeric Rating Scale
POEM:	Patient-Oriented Eczema Measure
PP-NRS:	peak-pruritus numeric rating scale
TEAE:	treatment-emergent adverse event

of the 8-week safety data, an expansion cohort (2:1; eblasakimab 600 mg: placebo) was also conducted. At the end of treatment, there was a 12-week safety follow-up period. Patients, investigators, and the sponsor were blinded to study treatment.

Eligible patients were 18 years or older with a clinical diagnosis of chronic AD[18,19] for at least 3 years before the screening visit and moderate-to-severe AD at the screening and baseline visits (Investigator's Global Assessment [IGA] score ≥3; Eczema Area and Severity Index [EASI] ≥16; and affected body surface area [BSA] ≥10%). Detailed inclusion and exclusion criteria and specifics concerning rescue medications can be found in the study protocol (Supplementary Information #2). Rescue medication before day 29 was not allowed. Participants who used rescue medication (from day 29 forward) were considered nonresponders.

Outcome measures

The primary outcome measure was incidence of treatment-emergent adverse events (TEAEs) throughout the study. Secondary outcome measures reported herein for each week up to week 8 included the following: percentage change in the EASI from baseline; proportions of patients achieving an EASI improvement of at least 50%, 75%, and 90% from baseline (EASI50, EASI75, and EASI90); proportions of patients achieving an IGA score of 0 (clear) or 1 (almost clear) (IGA 0/1) (score range: 0-4); percentage change from baseline in BSA (%BSA) affected; percentage change in peak-pruritus numeric rating scale (PP-NRS) score from baseline; proportions of patients with ≥4-point improvement in PP-NRS score; and improvements in Patient-Oriented Eczema Measure (POEM) score.

Statistical analysis

Statistical analysis was performed on a modified intent-to-treat data set, that was comprised all randomized participants who had received at least once dose of treatment, excluding 3 patients who discontinued prematurely due to COVID restrictions and a further 9 patients from 1 site who were prespecified before unblinding as they did not have disease characteristics consistent with moderate-to-severe AD; thus, their diagnoses could not be verified. These patients were all from a single research site and without exception had baseline characteristics markedly different from the rest of the cohort, including substantially lower biomarkers of AD, lower EASIs, and lower IGA, BSA, and POEM scores. Other notable differences included older patient age with older age at the onset of the disease. Participants in this site had no atopic disease history, but reported higher incidence of other comorbidities, including diabetes and hypertension. Baseline characteristics of the excluded site can be found in Supplementary Information #3 (available via Mendeley at https://data.mendeley.com/datasets/g6m6884znc/1). Several sensitivity analyses were also performed. In all analyses, last observation carried forward imputation was applied to account for missing data, including for participants who used rescue medication, that was prohibited before day 29. The study was designed to have 80% power to detect a true mean difference of 39% in percentage change from baseline EASI between eblasakimab 600 mg compared with placebo only. No multiplicity adjustments were made, and a single placebo group served as a control for all arms in the study.

Due to the small exploratory nature of the study, significance was tested at 1-sided $P = .05$, as per the Statistical Analysis Plan. Statistical analyses were conducted with SAS (version 9.4; SAS Institute).

RESULTS

Patients

Of the 52 patients randomized to weekly treatment, 40 were included in the modified intent-to-treat analysis set (mean [SD] age, 33.1 [11.30] years; 30 male [65.3%]): placebo (n = 13), eblasakimab 200 mg (n = 4), 400 mg (n = 7), or 600 mg (n = 16) (Fig 1). Demographics and baseline characteristics were balanced across treatment groups (Table I).

Safety

Fifty-two participants included in the safety analysis received at least 1 dose of study drug. TEAEs were reported in 8/17 (47%) patients in the placebo group and in 25/35 (71%) patients receiving eblasakimab (Table II). Most TEAEs were considered mild or moderate and did not lead to study discontinuation. Mild-to-moderate injection site reactions were reported in 9/35 (26%) patients receiving eblasakimab and 2/17 (12%) in the placebo group. In 2 patients receiving active treatment, conjunctivitis developed (6%). One serious adverse event was reported in the eblasakimab 400 mg group (a

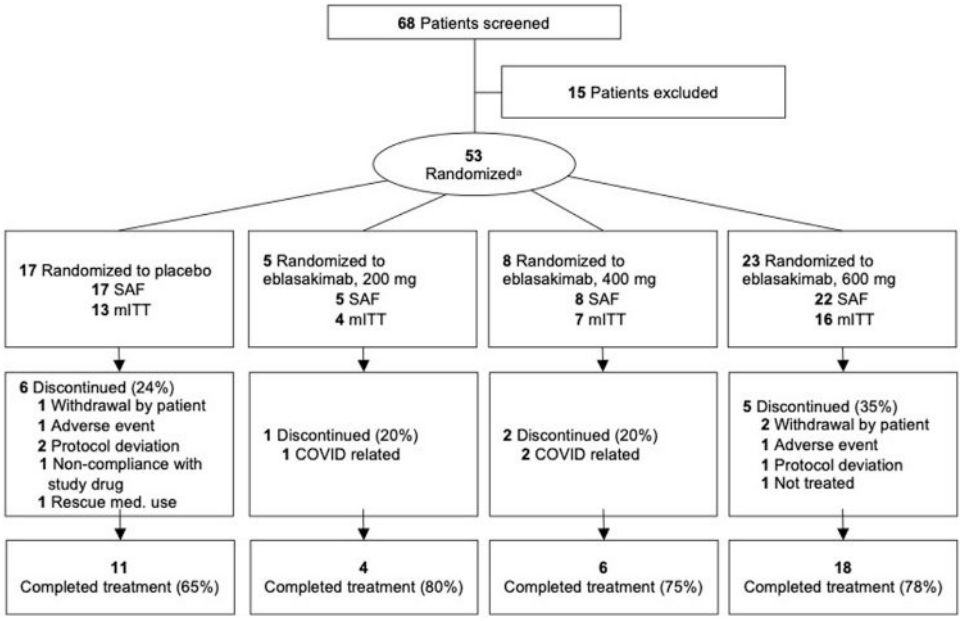

Fig 1. Patient disposition. *mITT*, Modified intent-to-treat analysis set; *SAF*, safety analysis set. [a]One randomized patient did not receive study treatment and was not included in the safety or efficacy analyses.

hospital visit due to mild abdominal pain); the causality is unlikely related to treatment. No deaths were reported during the study.

Efficacy outcomes

Improvements in EASI were seen early and progressed over the trial duration with eblasakimab treatment compared with placebo, with the 400 and 600 mg doses producing greater clinical responses than the 200 mg dose (Fig 2). Key secondary efficacy outcomes are presented in Table III and Supplementary Information #4 and #5 (available via Mendeley at https://data.mendeley.com/datasets/g6m6884znc/1).

Compared with placebo at week 8, eblasakimab 600 mg showed significant improvement in mean percentage change in EASI versus placebo (−65% vs −27%, $P = .014$) and proportion of patients achieving EASI50 (81% vs 31%, $P = .008$), and EASI75 (69% vs 15%, $P = .005$).

At week 8, the trend was for a higher percentage of patients for eblasakimab 600 mg versus placebo to achieve EASI90 (38% vs 15%, $P = .183$) and an IGA 0/1 (44% vs 15%, $P = .107$). Mean percentage change from baseline in %BSA affected at week 8 was −51% for eblasakimab 600 mg versus −13% for placebo.

Improvements in percentage change from baseline in PP-NRS for median worst itch and median average itch were apparent over time and at week 8 with 600 mg eblasakimab treatment versus placebo (worst itch: −48% vs −13%; average itch: −49% vs −6%, respectively). A ≥4-point improvement in PP-NRS was seen in the 600 mg eblasakimab group versus placebo (worst itch: 39% vs 13%; average itch: 31% vs 15%).

POEM score improved over time with eblasakimab treatment versus placebo, with the 400 and 600 mg doses producing numerically higher clinical responses than the 200 mg dose. At week 8, median POEM change from baseline for 600 mg eblasakimab was −9 versus −1 for placebo. A higher 4-point improvement in POEM score was observed at week 8 for eblasakimab 600 mg versus placebo (81% vs 23%). There was also greater improvement in POEM sleep scores with eblasakimab versus placebo. A 2-point mean improvement in sleep loss was observed at week 8 for 600 mg eblasakimab versus placebo (56% vs 15%). Patients had sleep scores of 3 or 4 at baseline. Most patients (75% [12/16]) in the 600 mg treatment group reported >5 nights of sleep disturbance at baseline versus placebo (54% [7/13]). More (63% [10/16]) of eblasakimab-treated patients reported "no days" or "1 to 2 days" of sleep disturbance at week 8 versus placebo (38% [5/13]). For all outcomes assessed, eblasakimab 200 mg failed to reach significance compared with placebo.

Rescue medication use was low throughout the study, but higher in the placebo group (Supplementary Information #6, available via Mendeley at https://data.mendeley.com/datasets/g6m6884znc/1).

Table I. Baseline demographics and disease characteristics in the modified intent-to-treat population*

	No. (%)				
	Eblasakimab				
	200 mg (*n* = 4)	400 mg (*n* = 7)	600 mg (*n* = 16)	All doses (*n* = 27)	Placebo (*n* = 13)
Baseline demographics					
Age					
Mean (SD), y	32.5 (5.3)	29.4 (4.9)	34.0 (14.4)	32.6 (11.5)	34.2 (11.3)
Min, Max	28, 38	23, 36	18, 68	18, 68	20, 61
Male	3 (75.0)	5 (71.4)	12 (75.0)	20 (74.1)	10 (76.9)
Age of onset					
Mean (SD), y	11.3 (8.2)	6.6 (7.6)	7.9 (10.5)	8.1 (9.3)	13.2 (14.7)
Race/ethnicity					
Asian	4 (100.0)	7 (100.0)	7 (43.8)	18 (66.7)	8 (61.5)
Black	0	0	1 (6.2)	1 (3.7)	0
White	0	0	8 (50.0)	8 (29.6)	3 (23.1)
Other	0	0	0	0	2 (15.4)
Weight (kg)					
Mean (SD)	75.3 (13.2)	72.2 (14.2)	74.7 (20.7)	74.1 (17.8)	75.2 (12.7)
Baseline disease characteristics					
EASI					
Mean (SD)	32.9 (14.3)	31.3 (12.3)	30.5 (14.2)	31.1 (13.2)	31.5 (10.1)
Median	30.4	33.3	25.1	27.3	33.0
IGA					
3, moderate	2 (50.0)	5 (71.4)	10 (62.5)	17 (63.0)	7 (53.8)
4, severe	2 (50.0)	2 (28.6)	6 (37.5)	10 (37.0)	6 (46.2)
BSA involvement					
Mean (SD)	55.5 (34.6)	62.3 (28.5)	45.82 (24.4)	51.5 (26.9)	50.1 (28.6)
Median	54.5	74.0	37.5	39.0	43.0
Pruritus NRS score (worst)					
Mean (SD)	7.4 (2.2)	7.7 (1.6)	7.5 (1.3)	7.6 (1.5)	7.7 (2.0)
Median	7.7	7.7	6.9†	7.7‡	8.0
TARC/CCL17 (pg/mL)					
Mean (SD)	6097 (6247)	18,310 (40,556)	4223 (5186)	8201§ (21,292)	5056 (6842)
Median	5556	2262	2128	2186	2398
Total IgE (kU/L)					
Mean (SD)	15,891 (14,993)	23,297 (28,508)	8660 (7718)	13,570§ (17,107)	8706 (8175)
Median	12,278	10,660	6468	6913	77,173
Medical history/comorbidities					
Any	1 (25.0)	6 (85.7)	16 (100.0)	23 (85.2)	11 (84.6)
General					
Diabetes	0	0	0	0	0
Anxiety/depression	0	0	3 (18.8)	3 (11.1)	1 (7.7)
Other					
Asthma	0	3 (42.9)	8 (50.0)	11 (40.7)	6 (46.2)
Allergy (dust, pet, seasonal, etc)	0	1 (14.3)	9 (56.3)	10 (37.0)	4 (30.8)
Allergic rhinitis	1 (25.0)	1 (14.3)	5 (31.3)	7 (25.9)	1 (7.7)
Allergic conjunctivitis/dry eye	0	2 (28.6)	0	2 (7.4)	0
Drug hypersensitivity	0	1 (14.3)	4 (25.0)	5 (18.5)	3 (23.1)
Psoriasiform dermatitis	0	0	1 (6.3)	1 (3.7)	1 (7.7)
Eczema herpeticum	0	0	0	0	1 (7.7)
Other	0	3 (42.9)	12 (75.0)	15 (55.6)	6 (46.2)
None documented	3 (75.0)	1 (14.3)	0	4 (14.8)	2 (15.4)

BSA, Body surface area; *EASI*, Eczema Area and Severity Index; *IGA*, Investigator's Global Assessment (5-point scale); *IgE*, immunoglobulin E; *NRS*, numeric rating scale; *SD*, standard deviation; *TARC/CCL17*, thymus- and activation-regulated chemokine/CCL17.

*Percentages are based on the number of patients in the modified intent-to-treat population with a nonmissing response.

†Sample size is (*n* = 13).

‡Sample size is (*n* = 23).

§Sample size is (*n* = 26).

Table II. Incidence of treatment-emergent adverse events occurring in ≥2 patients in any group characteristics in the modified intent-to-treat population

	No. (%)				
	Eblasakimab				
TEAE preferred term	200 mg (*n* = 4)	400 mg (*n* = 7)	600 mg (*n* = 16)	All doses (*n* = 27)	Placebo (*n* = 13)
Eye pruritus	0	3 (42.9)	0	3 (11.1)	1 (7.7)
Conjunctivitis allergic	0	0	2 (12.5)	2 (7.4)	0
Dry eye	0	2 (28.6)	0	2 (7.4)	0
Injection site erythema	1 (25.0)	3 (42.9)	0	4 (14.8)	1 (7.7)
Injection site swelling	1 (25.0)	0	3 (18.8)	4 (14.8)	0
Injection site pain	0	0	2 (12.5)	2 (7.4)	0
Injection site pruritus	0	1 (14.3)	2 (12.5)	3 (11.1)	0
Pyrexia	2 (50.0)	0	0	2 (7.4)	0
Pruritus	1 (25.0)	1 (14.3)	3 (18.8)	5 (20.8)	1 (7.7)
Headache	0	0	3 (18.8)	3 (11.1)	0
Lethargy	2 (50.0)	1 (14.3)	0	3 (11.1)	0
Rhinorrhea	2 (50.0)	1 (14.3)	0	3 (11.1)	1 (7.7)

TEAE, Treatment-emergent adverse event.

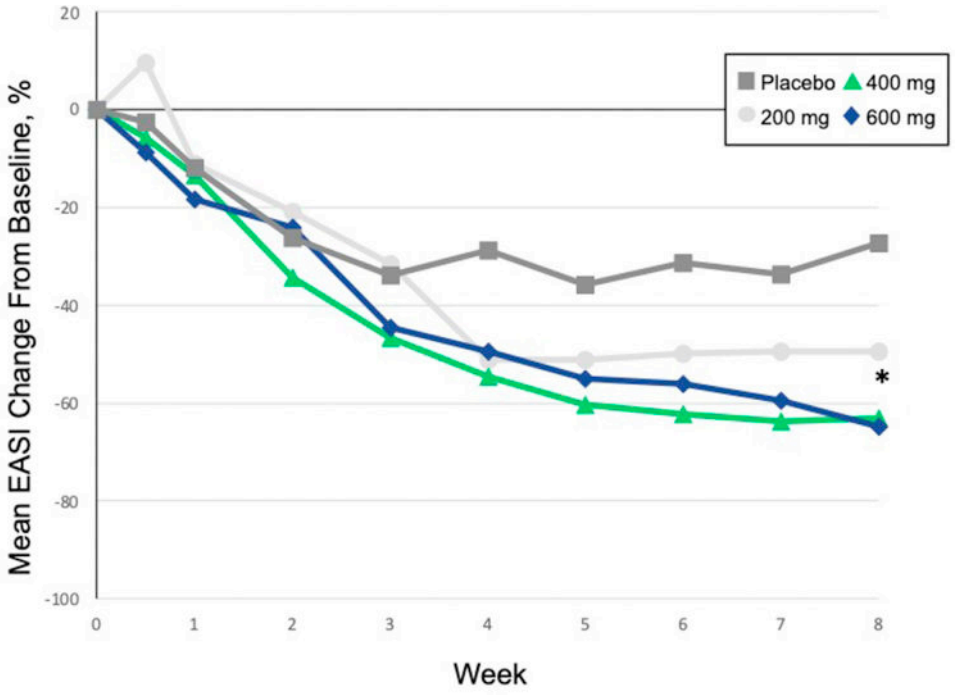

Fig 2. Eczema Area and Severity Index mean percentage change from baseline in patients with moderate-to-severe atopic dermatitis treated with eblasakimab. *P = .014, 600 mg eblasakimab versus placebo.

DISCUSSION

This phase 1b randomized, placebo-controlled multiple-ascending dose escalation study, with cohort expansion, demonstrated proof-of-concept for the safety and efficacy of eblasakimab in patients with moderate-to-severe AD. Weekly doses of eblasakimab for 8 weeks were well-tolerated, with rates of moderate-to-severe adverse events comparable between eblasakimab-treated patients and the placebo group. TEAEs were similar between groups, with those leading to discontinuation higher in the placebo group. Eblasakimab also demonstrated clinical benefits in moderate-to-severe AD. Indeed, proof-of-concept was established, with eblasakimab showing significant improvements versus placebo across a range of end points, including EASI

Table III. Key secondary outcome measures at week 8

Outcome measure	Eblasakimab 200 mg (*n* = 4)	Eblasakimab 400 mg (*n* = 7)	Eblasakimab 600 mg (*n* = 16)	Placebo (*n* = 13)
Physician reported				
EASI50, %	50	71	81*	31
EASI75, %	50	57	69†	15
EASI90, %	0	57	38	15
IGA 0/1 response, %	0	14	44	15
BSA % change from baseline	−50	−60	−51	−12
Patient reported				
PP-NRS % change from baseline (worst)	−40	−38	−48	−13
PP-NRS % change from baseline (average)	−39	−39	−49	−6
Improvement in POEM score (mean)	−5	−12	−10	−3

BSA, Body surface area; *EASI*, Eczema Area and Severity Index (indicating 50%, 75%, or 90% improvement from baseline); *IGA 0/1*, Investigator's Global Assessment (5-point scale, with 0 indicating clear and 1 indicating almost clear); *POEM*, Patient-Oriented Eczema Measure (range, 0 [clear] to 28 [very severe]); *PP-NRS*, peak-pruritus numeric rating scale.
*P = .008 versus placebo.
†P = .005 versus placebo.

percentage change from baseline, proportion of patients achieving at least EASI50 and EASI75, and PP-NRS percentage change from baseline.

With the aim of reducing the placebo response and standardizing skin care, patients were required to apply a bland moisturizer at least twice daily for at least 7 days before randomization and continue throughout the study. Additionally, excessive use of bathing/chlorinated baths was not allowed during study participation.

Although greater than for the placebo group, there was a low incidence of conjunctivitis, a TEAE of clinical interest in the eblasakimab group. Following its regulatory approval, dupilumab-induced conjunctivitis was widely reported as a tolerability issue in real-world clinical practice,[20,21] in comparison with published studies for IL-13 selective agents lebrikizumab and tralokinumab that reported conjunctivitis rates closer to those seen with placebo.[11,12] These data for eblasakimab suggest that inhibiting IL-13Rα1 may carry a lower risk of conjunctivitis. Injection site reactions were reported in 26% of eblasakimab-treated patients (vs 12% placebo), all of which were mild to moderate, transient, and did not lead to discontinuation. The emerging safety profile from this study provides confidence to further investigate eblasakimab's safety up to weekly doses of 600 mg in a longer, larger study.

Importantly, the patient-reported outcomes corroborated the physician-reported outcomes, lending weight to the potential utility of eblasakimab in treating AD. That the trend of these improvements was seen after 8 weeks of treatment is notable, given that traditional AD study designs typically employ 12- or 16-week primary efficacy end points. The data demonstrate continued improvement over the 8-week treatment duration. Continued treatment beyond 8 weeks may lead to additional benefit and is being explored in the ongoing phase 2 study with this drug.

The significant outcomes for the primary efficacy variable in this study, EASI percentage change from baseline, and other key secondary end points, were substantiated by sensitivity analyses, showing the robustness of the results.

LIMITATIONS

Although this study has demonstrated a positive effect on moderate-to-severe AD for eblasakimab, there are a several study limitations. First, the use of a small sample size limits the power of the study and the generalizability of the findings. It also contributes to the disparity of ethnicity in the different cohorts. Second, treatment for AD is typically long-term, consistent with the chronic nature of the disease; consequently, there is a need for long-term studies to assess safety and efficacy of eblasakimab beyond 8 weeks.

CONCLUSIONS

In summary, these phase 1b data demonstrate that treatment of adults with moderate-to-severe AD with eblasakimab was well-tolerated and was associated with significant clinical improvements versus placebo as measured by both physician- and patient-reported outcomes. Although highly significant results were observed in this 8-week study, additional benefit beyond this timepoint, and in a larger patient cohort, is warranted. A phase 2b clinical trial is ongoing to address these points.

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Conflicts of interest

Dr Blauvelt has served as a research investigator, consultant, and/or paid speaker for ASLAN Pharmaceuticals, AbbVie, Abcentra, Aligos, Almirall, Arcutis, Arena, Athenex, Boehringer Ingelheim, Bristol Myers Squibb, Dermavant, EcoR1, Eli Lilly and Company, Evommune, Forte, Galderma, Incyte, Janssen, Landos, LEO Pharma, Novartis, Pfizer, Rapt, Regeneron, Sanofi Genzyme, Sun Pharma, UCB Pharma, Vibliome, and Xencor. Drs Veverka, Menezes, and Kaoukhov are employees of ASLAN Pharmaceuticals Pte Ltd. Dr Thng has received funding and holds stock options from ASLAN Pharmaceuticals Pte Ltd and served as a member of the Data Safety Monitoring Board for this study. Dr Silverberg has served as a research investigator, consultant, and/or paid speaker for ASLAN Pharmaceuticals Pte Ltd, Celgene Corporation, Eli Lilly and Company, F. Hoffmann-LaRoche, Menlo Therapeutics Inc, Realm Therapeutics PLC, Regeneron Pharmaceuticals, Inc, Sanofi SA, Pfizer Inc, AbbVie, Inc, Anacor Pharmaceuticals, AnaptysBio, Inc, Arena Pharmaceuticals, Inc, Dermira, Inc, Dermavant Sciences, Galderma SA, GlaxoSmithKline, Glenmark Pharmaceuticals, Incyte Corporation, Kiniksa Pharmaceuticals Ltd, LEO Pharma A/S, and Novartis International AG. Dr Armstrong has served as a research investigator, consultant, and/or paid speaker for ASLAN Pharmaceuticals, AbbVie, Almirall, Arcutis, Boehringer Ingelheim, Bristol Myers Squibb, Dermavant, Dermira, Inc, Janssen Pharmaceuticals, Kyowa Hakko Kirin, LEO Pharma, Eli Lilly, Modernizing Medicine, Nimbus, Novartis, Ortho Dermatologics, Parexel, Pfizer, Regeneron, Sanofi Genzyme, Sun, and UCB.

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