Company presentation

June 2023

NASDAQ: ASLN

ASLAN PHARMACEUTICALS

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ASLAN Pharmaceuticals is a clinical-stage, immunology-focused biopharmaceutical company developing innovative therapies to treat inflammatory disease, transforming the lives of patients

Company highlights

- **Targeting major inflammatory disease markets with significant unmet need**

- *Eblasakimab*, is a potential **first-in-class antibody targeting the IL-13 receptor that has the potential to improve upon current biologics** used to treat allergic disease
  - There are few safe and effective treatments for moderate-to-severe atopic dermatitis (AD), expected to be a $24B market by 2029[1]. Despite *dupilumab* advancing the standard of care, physicians / patients still seek additional options
  - Topline data from completed multiple ascending dose (MAD) study established proof of concept for *eblasakimab* in AD, and supports a potentially differentiated safety and efficacy profile
  - Ongoing Ph2 studies in biologic naïve and *dupilumab* experienced patients, readouts in early July 2023 and 1Q 2024

- *Farudodstat* is a novel **DHODH inhibitor with the potential to be first-in-class** for alopecia areata (AA)
  - Stronger *in vitro* potency and lower potential for hepatotoxicity compared to other DHODH inhibitors
  - Phase 2 proof-of-concept study in AA initiated, interim topline readout expected 1Q 2024

- **Strong cash position $57.5M** as of March 31, 2023 (includes $20M BVF-led private placement in Feb)
- Additional **$12M upfront payment** received from *eblasakimab* licensing deal in Japan, closed in June 2023

1 Decision Resources Group, June 2021

Developing innovative therapies to treat inflammatory disease

Program	Target	Indication	Preclinical	Phase 1	Phase 2	Phase 3	Anticipated milestones
Eblasakimab	IL-13Rα1	Atopic dermatitis	Biologic naïve				Phase 2b topline data in early July 2023
			Dupilumab experienced				Phase 2 topline data in 1Q 2024
		Type 2-driven disease					
Farudodstat	DHODH	Alopecia areata					Phase 2a interim topline data 1Q 2024

Management and advisory team with global development experience in dermatology

Management team

Dr Carl Firth
CEO

Stephen Doyle
Chief Business Officer

Dr Alex Kaoukhov
Chief Medical Officer

Dr Karen Veverka
VP Medical

Dr Ferda Cevikbas
Head Translational Sciences

Board of Directors

Andrew Howden
Chairman

Dr Neil Graham
Board Director

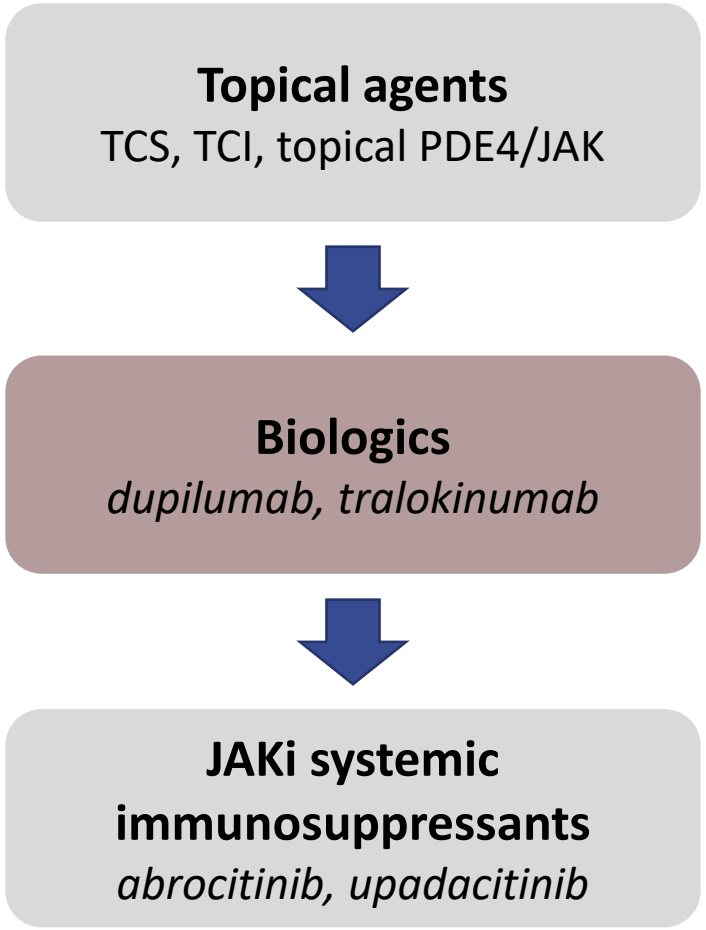
Robert Hoffman
Board Director

Dr Kathleen Metters
Board Director

(Acquired by Equillium)

Eblasakimab
A new mechanism for treatment of AD

Dupilumab has advanced the standard of care for atopic dermatitis but a significant unmet need remains



- There are few safe and effective treatments for moderate-to-severe AD
- Treatment is traditionally focused on topical corticosteroids but steroid use can be associated with safety risks
- *Dupilumab* has established dual blockade of IL-4/IL-13 biologic therapy as the new standard of care [1]
  - Launch of *dupilumab* in 2017 helped drive a large market for systemic AD therapy with 2022 sales of $8.8B
  - Sanofi expects to grow sales to over $14B
- However, only 8% of eligible patients receive *dupilumab* today [1] and there remains a significant unmet need:
  - Only 30-40% of patients treated with *dupilumab* achieved an optimal response [2,3]
  - Conjunctivitis is common and can lead to treatment discontinuations
  - Opportunity to improve upon biweekly dosing regimen

1 Sanofi's quarterly financials and annual reports
2 Spherix (2018) Atopic dermatitis ATU study
3 IGA 0/1 response rate at week 16, Simpson et al (2016) NEJM 375(24):2334-2348

The number of biologics in development is limited due to several notable failures in recent years

Topical agents
TCS, TCI, topical PDE4/JAK

Biologics
dupilumab, tralokinumab

JAKi systemic immunosuppressants
abrocitinib, upadacitinib

Target	Biologic	Current status
IL-4Rα	*Dupilumab*	Approved
IL-13	*Tralokinumab* *Lebrikizumab*	Suboptimal efficacy [1] (approved) Phase 3 completed
IL-31	*Nemolizumab*	Suboptimal efficacy [1] (phase 3)
OX-40 OX-40L	*Rocatinlimab* *Amlitelimab*	Phase 3 trial ongoing

Discontinued programs (or no activity reported) in AD:

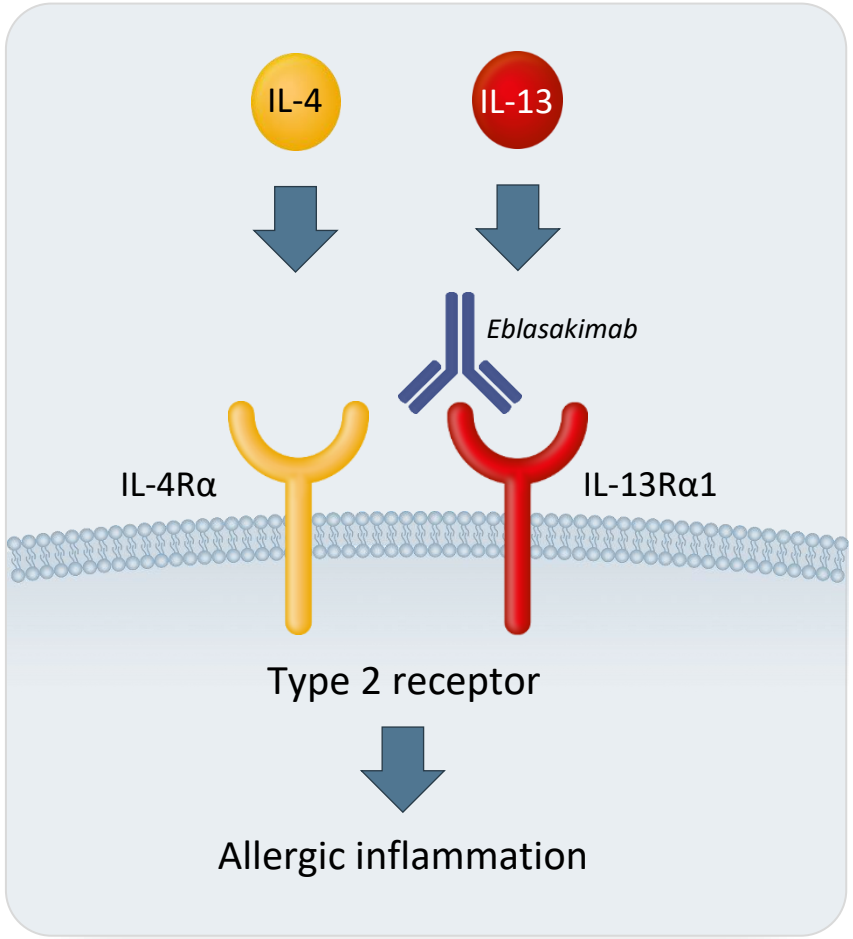
Berekimab (IL-1α)	*Mepolizumab* (IL-5)	*Tezepelumab* (TSLP)
MOR16 (IL-17C)	*Fezakinumab* (IL-22)	*Secukinumab* (IL-17)
Itepekimab (IL-33)	*Sposolimab* (IL-36)	*Etokimab* (IL-33)
		Ligelizumab (IgE)

1 Suboptimal efficacy defined as 20% or less placebo-adjusted IGA 0/1 score (in comparison *dupilumab* achieved 30% placebo-adjusted IGA 0/1 score in Phase 3 trials)

Significant unmet needs exist despite existing and soon-to-be-approved therapies

		Ideal therapy	*Dupilumab*	*Tralokinumab*	*Lebrikizumab*	Oral JAKi
Efficacy	Efficacy comparable or better than *dupilumab*	✓	✓		✓	✓
	Rapid efficacy onset within 2 weeks of treatment	✓				✓
	Complete inhibition of Type 2 receptor without affecting Type 1 receptor	✓				
	Proven to block sensitization of itch neurons to IL-13 and IL-4	✓	?			?
Safety	Safe and well-tolerated (no boxed warning or monitoring)	✓	✓	✓	✓	
	Low rates of conjunctivitis and Type 1 driven effects	✓		✓	?	
Dosing and Convenience	Convenient dosing: monthly injection from start of treatment or oral	✓				✓
	Potential for flexible dosing	✓				✓
	Stable at room temperature (no refrigeration required)	✓				✓
Treats comorbidities	Effective in other atopic diseases	✓	✓			?

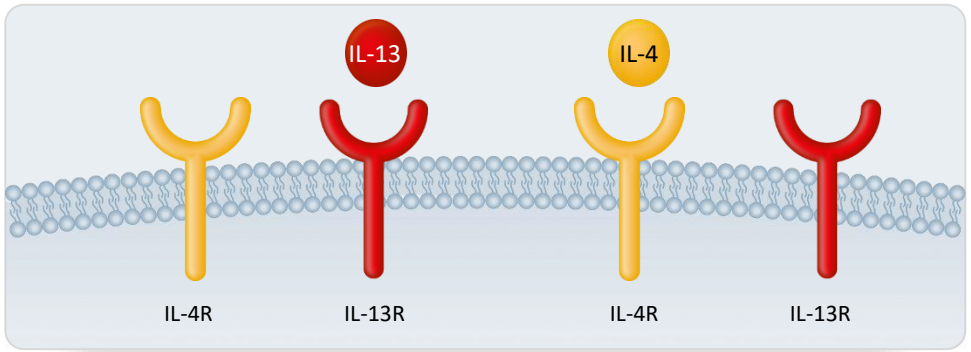
Eblasakimab is the only monoclonal antibody in the clinic targeting the IL-13 receptor



IL-4 and IL-13 are central to triggering allergy and symptoms of atopic dermatitis.

By targeting the IL-13 receptor, *eblasakimab* blocks the Type 2 receptor complex, preventing signaling through **both** IL-4 and IL-13.

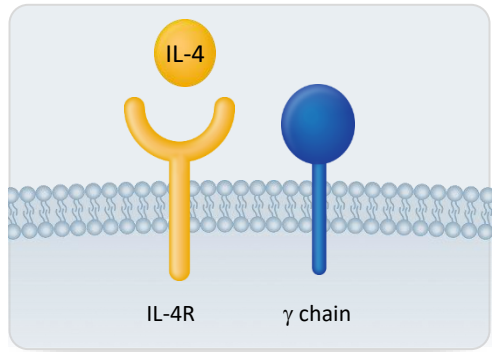
Eblasakimab selectively blocks the Type 2 receptor



Type 2 receptor

Blocks IL-13 signalling

Blocks IL-4 signalling



Type 1 receptor

Blocks IL-4 signalling

Eblasakimab

Specific and complete blockade of Type 2 receptor

Lebrikizumab

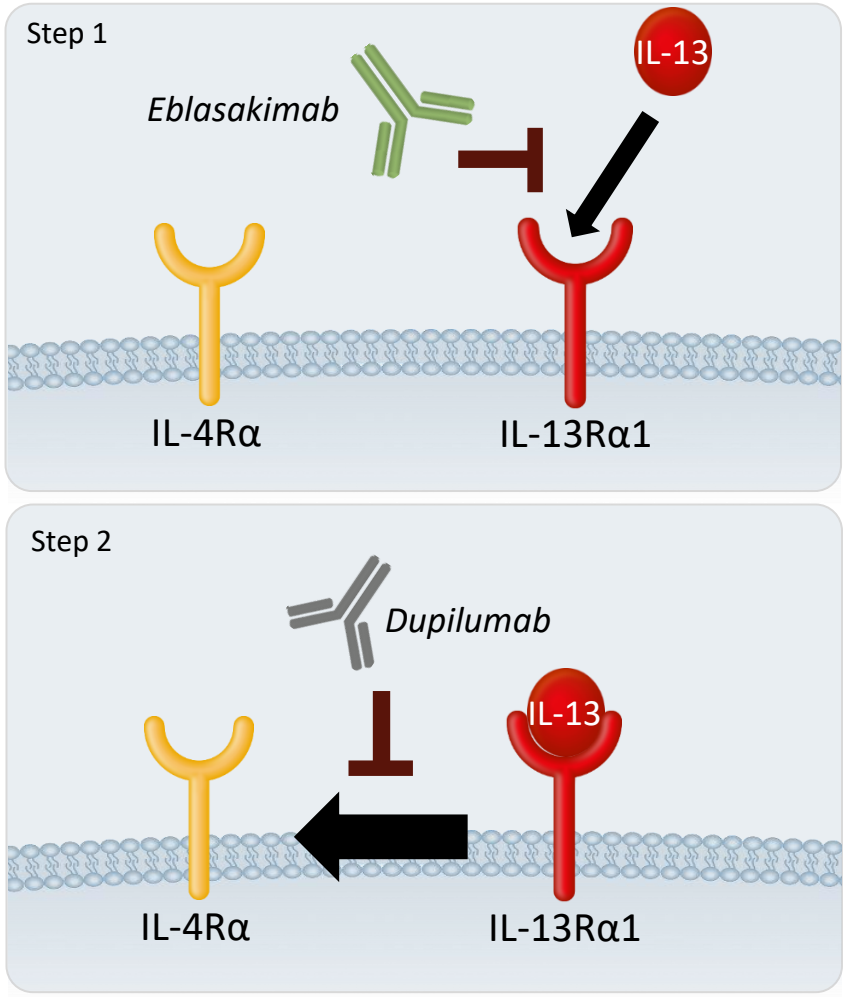
Partial blockade of Type 2 receptor signalling

Dupilumab

Broad blockade of Type 1 and Type 2 receptors

What makes *eblasakimab* different?

Binding IL-13Rα1 directly has the potential for more efficient blockade of the receptor



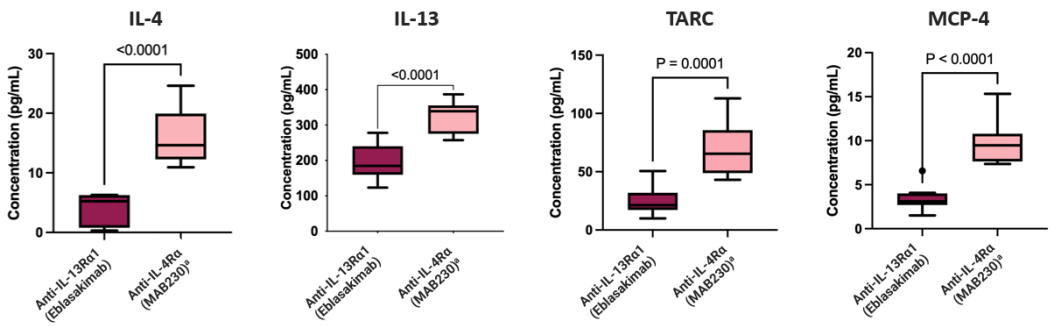
- Formation of the Type 2 receptor complex occurs in 2 steps:
  1. Ligand binding to its receptor (IL-13 to IL-13Rα1)
  2. Bound receptor binding to the partner receptor (IL-4Rα)
- Step 1 is a weaker, lower affinity interaction and a rate limiting step while Step 2 is a high affinity interaction
- By directly blocking the rate limiting step, *eblasakimab* has the potential to provide more efficient blockade of IL-13 signaling versus *dupilumab* which interferes with Step 2, a high affinity interaction
- This may translate to lower required concentration *in vivo* and may provide improved dosing frequency and efficacy

1 Ito et al (2009) JBC 284(36): 24289-24296
2 Andrews et al (2002) JBC 277(48):46073-46078.

Recent translational data highlights advantages of targeting the IL-13Rα1 subunit over IL-4Rα in AD patient cells

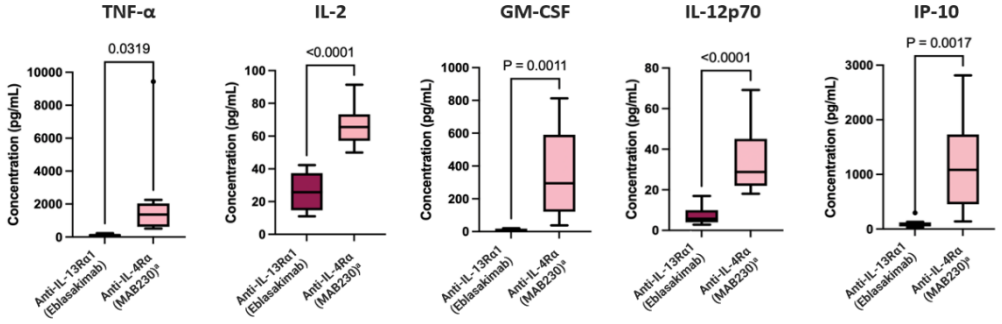
Th2 cytokines

IL-13Rα1 blockade resulted in lower levels of key cytokines implicated in Th2-driven inflammation compared to IL-4Rα blockade



Th1 cytokines

Levels of Th1 cytokines were lower with IL-13Rα1 blockade compared to IL-4Rα blockade



Selective blockade of IL-13Rα1 offers a potentially differentiated approach:

- More efficient reduction of Th2 inflammation
- No increase in Th1 cytokines, compared to IL-4Rα blockade

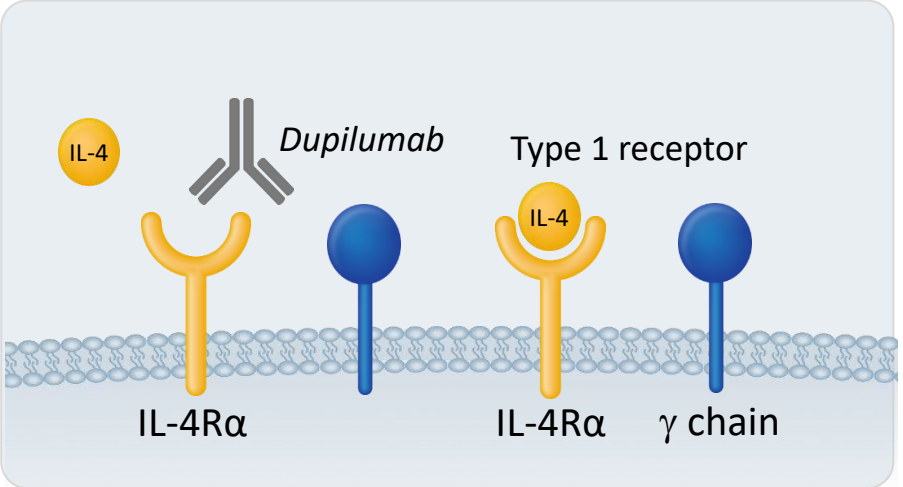
Data from *In vitro* studies conducted in PBMCs of moderate-to-severe AD patients, cells were cultured with anti-IL-13Rα1 (*eblasakimab*) or anti-IL-4R α (R&D Systems antibody) and supernatants assayed for cytokine panel using electrochemiluminescence.

Data presented at the 1st International Society of Investigative Dermatology Meeting, May 10-14, 2023, in Toyko, Japan, in late-breaker minisymposium "Downstream effects of IL-13α1 blockade on Type 2 inflammation and Th1 immune axis activation in atopic dermatitis" (Cevikbas et al)

Certain side effects, such as conjunctivitis, may be driven by inhibition of Type 1 receptor, which *eblasakimab* does not bind

Dupilumab study	Rate of conjunctivitis
Phase 3 mono [1]	**8%** (placebo-adjusted)
Open label extension [1]	**20%**
Real world experience [2]	**26%**

- Rates of conjunctivitis are higher in *dupilumab* treated patients [1]
- *Lebrikizumab,* which targets IL-13 and does not block the Type 1 receptor, may have a lower rate of conjunctivitis (5% placebo-adjusted) [3]

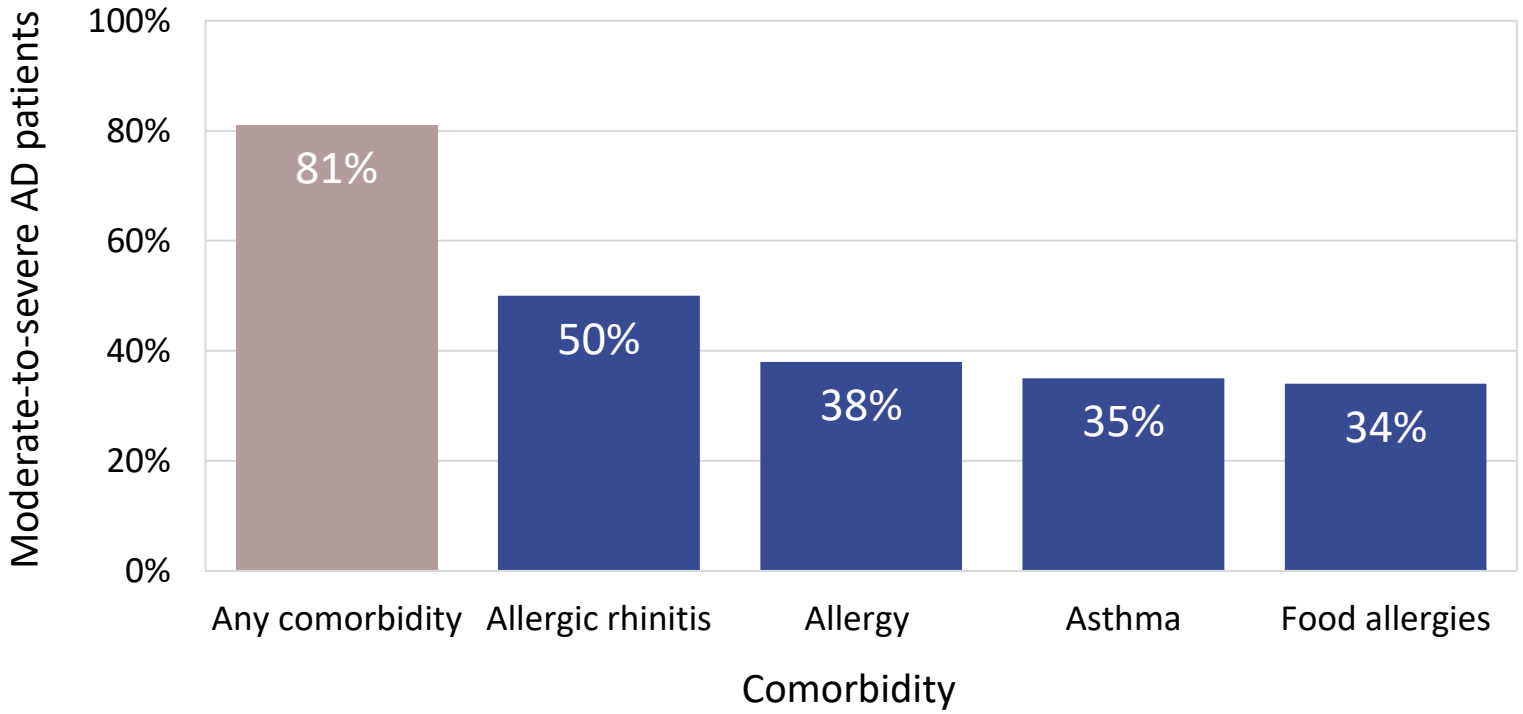


- Blockade of the Type 1 receptor may drive T-cells to a pro-inflammatory T_H1 phenotype
- This may lead to unwanted side effects, such as conjunctivitis

1 Dupixent full prescribing information
2 Halling et al (2021) JAAD 84:139-147
3 Simpson et al (2022) AAD Annual Meeting, 25-29 March 2022.

Eblasakimab may be efficacious against a wide range of AD comorbidities, such as asthma and allergy

81% of moderate-to-severe AD patients have Type 2 inflammatory comorbidities:



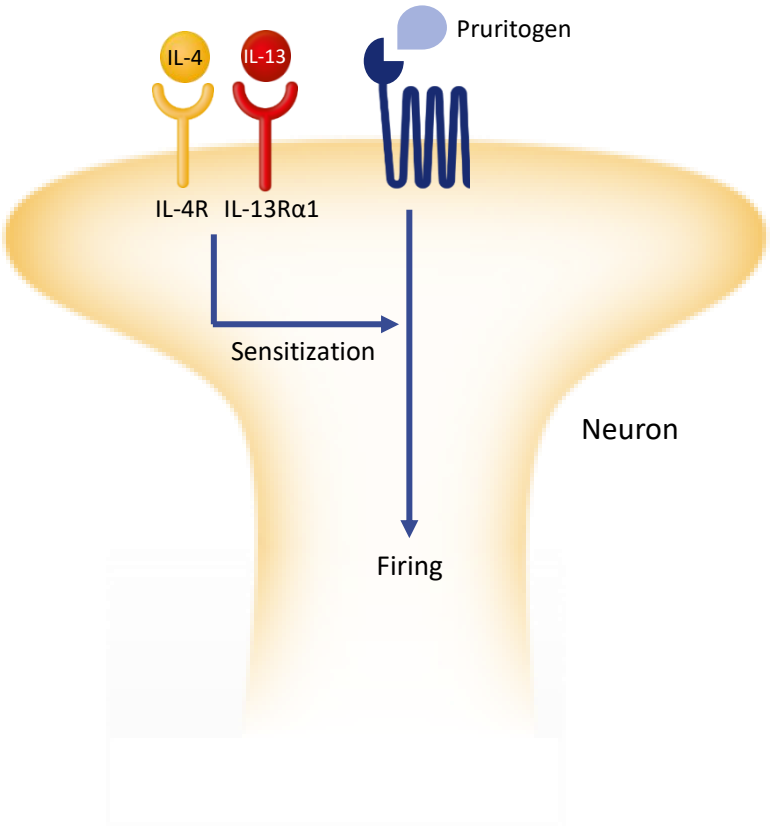
Blockade of IL-4 and IL-13 signaling through the Type 2 receptor will be important to address both IL-4 and IL-13 driven comorbidities in AD patients.

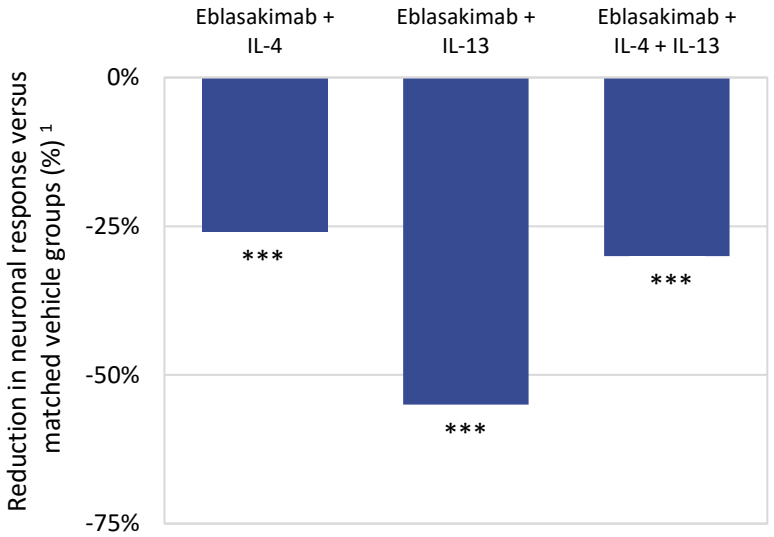
Physicians would prefer treatment options that can address these other conditions.

Calzavara-Pinton et al (2022) AAD Annual Meeting poster: Baseline patient demographics and comorbidities in patients with atopic dermatitis from the GLOBOSTAD registry (237 patients)

Eblasakimab blocks the Type 2 receptor on itch neurons supporting the potential for rapid itch relief

The Type 2 receptor is expressed on certain itch-specific neurons. IL-13 and IL-4 believed to amplify itch responses.





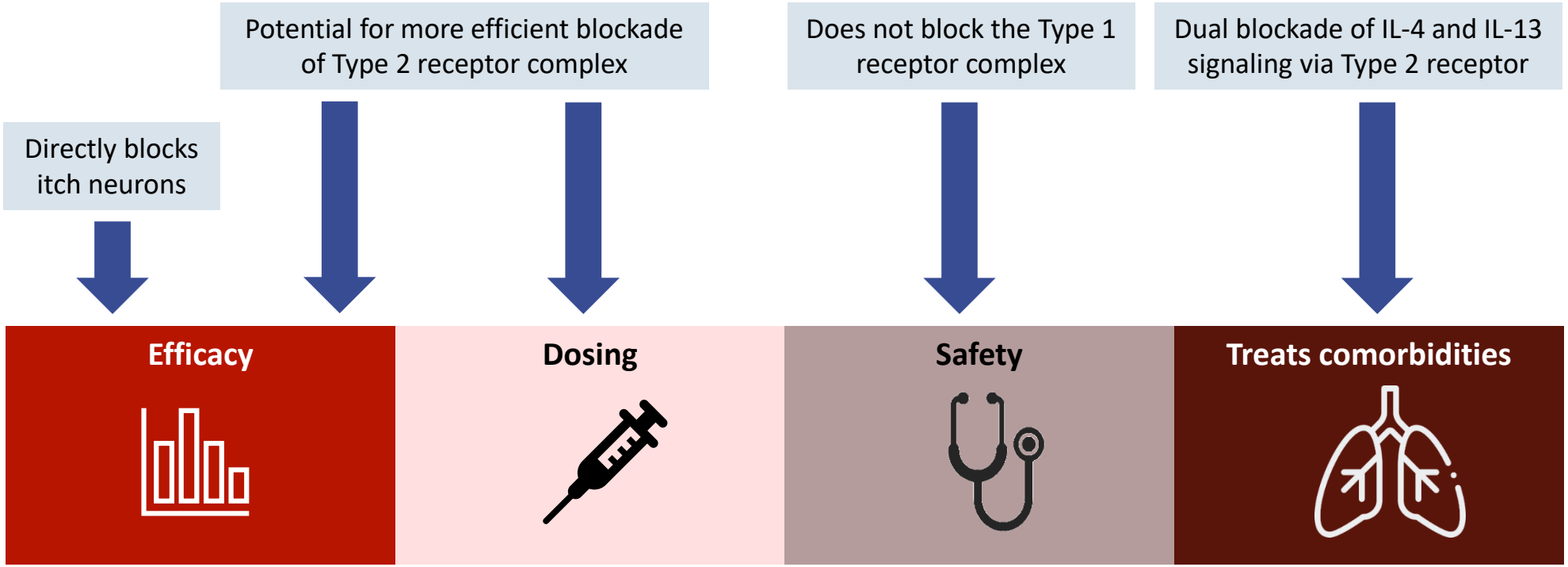
- *Ex vivo* studies performed on sensory neurons from human donors
- IL-4 and IL-13 enhanced the neuronal itch response via the Type 2 receptor
- *Eblasakimab* significantly reduced neuronal responses to IL-4, IL-13, and their combination by an average of 40% (p<0.0001)

These results suggest a molecular basis for the significant reduction of pruritus scores observed in *eblasakimab* treated moderate-to-severe AD patients in the Phase 1b clinical trial

1 Total N= 41-134 across tested conditions from 3 donors, at 24hr timepoint
*** p <0.001, p values are one-sided

Eblasakimab has the potential to be a differentiated therapy in AD

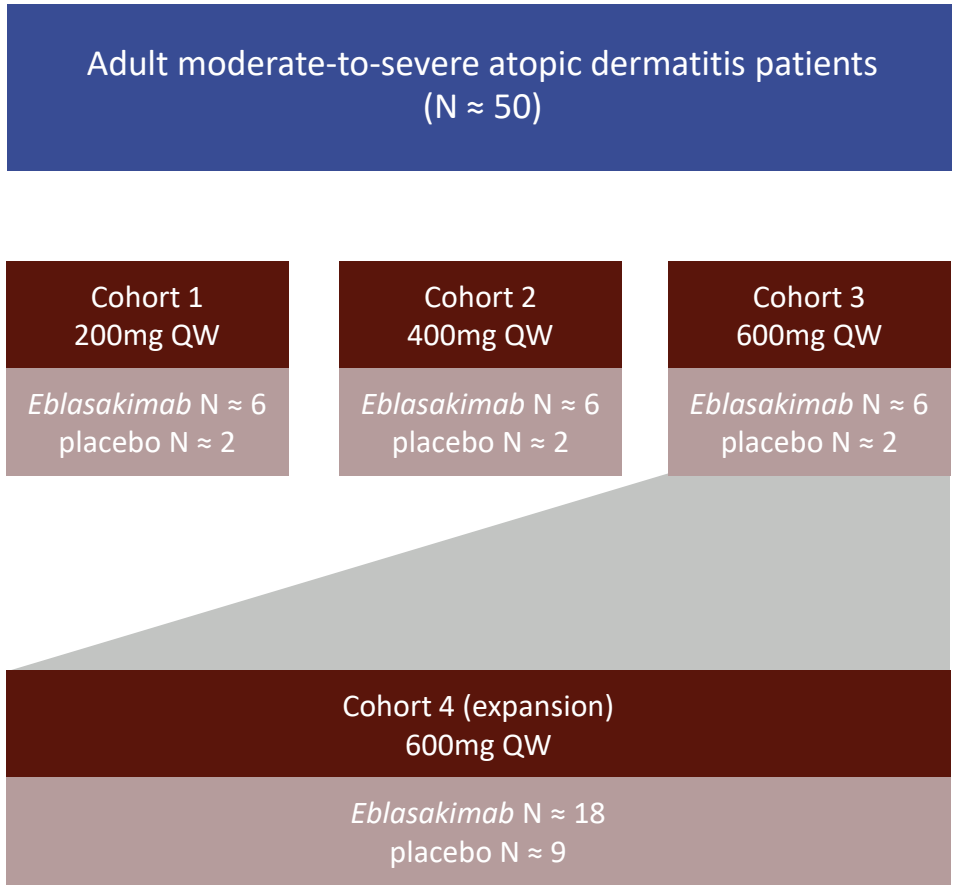
What makes *eblasakimab* different?



Ideal target product profile				
	Better efficacy over current standard-of-care with rapid control of itch	Less frequent and more convenient dose regimen	Addresses physician concerns on safety with lower rate of discontinuation	Able to address allergic comorbidities such as asthma and rhinitis

Eblasakimab
Completed proof of concept study

Completed Proof of Concept study in moderate-to-severe AD



- Double-blind, randomized, placebo-controlled Phase 1 MAD study
- Patients dosed weekly via subcutaneous injection for 8 weeks with a 12-week recovery period
- Positive interim data from dose escalation (cohorts 1 to 3) announced in March 2021
- Positive data from full study (cohorts 1 to 4) presented at AAD (2022)

Primary endpoints are safety and tolerability

Secondary endpoints include percentage change from baseline in EASI (Eczema Area and Severity Index) score, pruritus score (numeric rating scale, NRS) and IGA (Investigator Global Assessment), and biomarkers TARC and IgE

Key inclusion criteria:

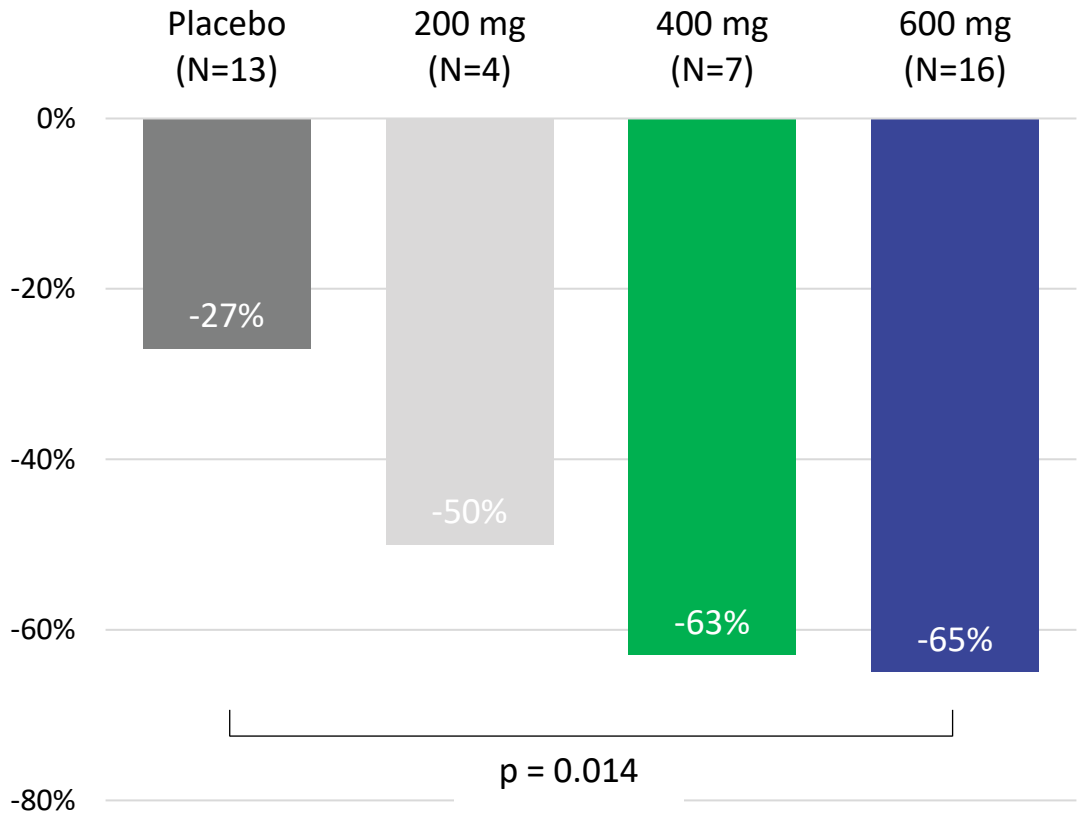
- Chronic AD present for ≥3 years before screening visit
- EASI score ≥16 at screening and baseline
- IGA score ≥3 (scale of 0 to 4) at screening and baseline
- ≥10% BSA (Body Surface Area) of AD involvement at screening and baseline

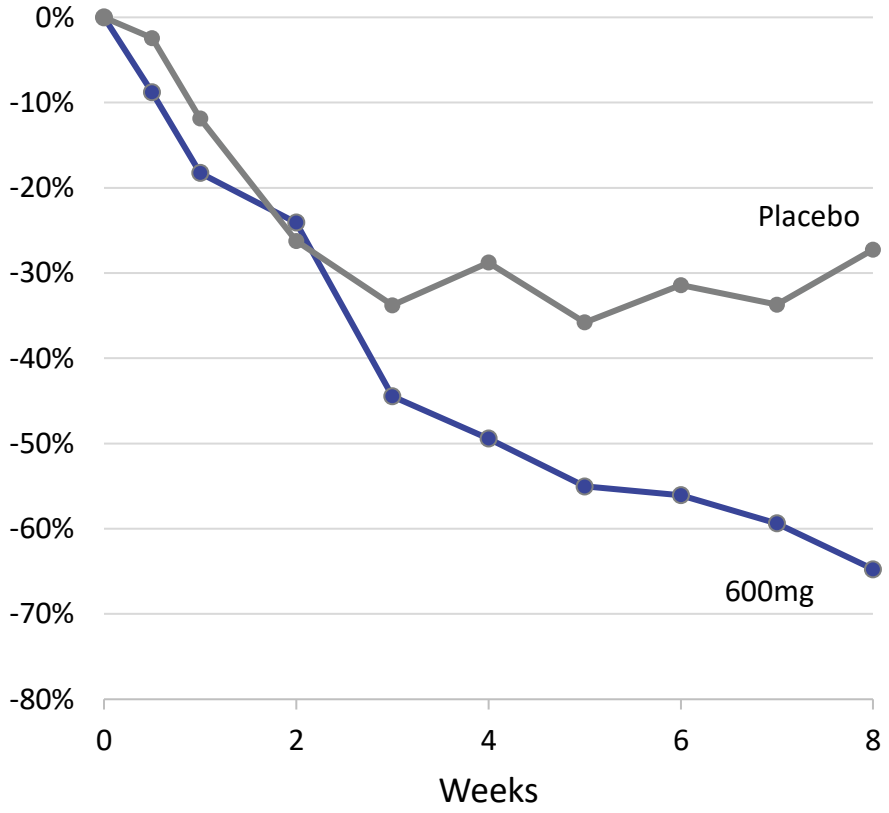
Selected baseline patient characteristics

	mITT[1] (n=40)			
	Placebo (N=13)	200mg (N=4)	400mg (N=7)	600mg (N=16)
Age (years)	37.8	30.4	29.4	40.2
Mean EASI score	28.3	29.6	30.5	27.6
Patients with IGA 3 / IGA 4	65% / 35%	60% / 40%	75% / 25%	68% / 32%
Mean BSA	44.8%	47.8%	59.9%	41.0%
Mean peak pruritus NRS score	7.7	7.4	7.7	7.9
Median TARC (pg/ml)	2,398	5,556	2,262	2,128
Median IgE (kU/l)	419	429	687	306

1 Efficacy analysis was performed on a modified population, excluding 9 patients from a single site because their eligibility could not be confirmed and was pre-specified and defined prior to unblinding

Primary efficacy endpoint: change in EASI from baseline (week 8)

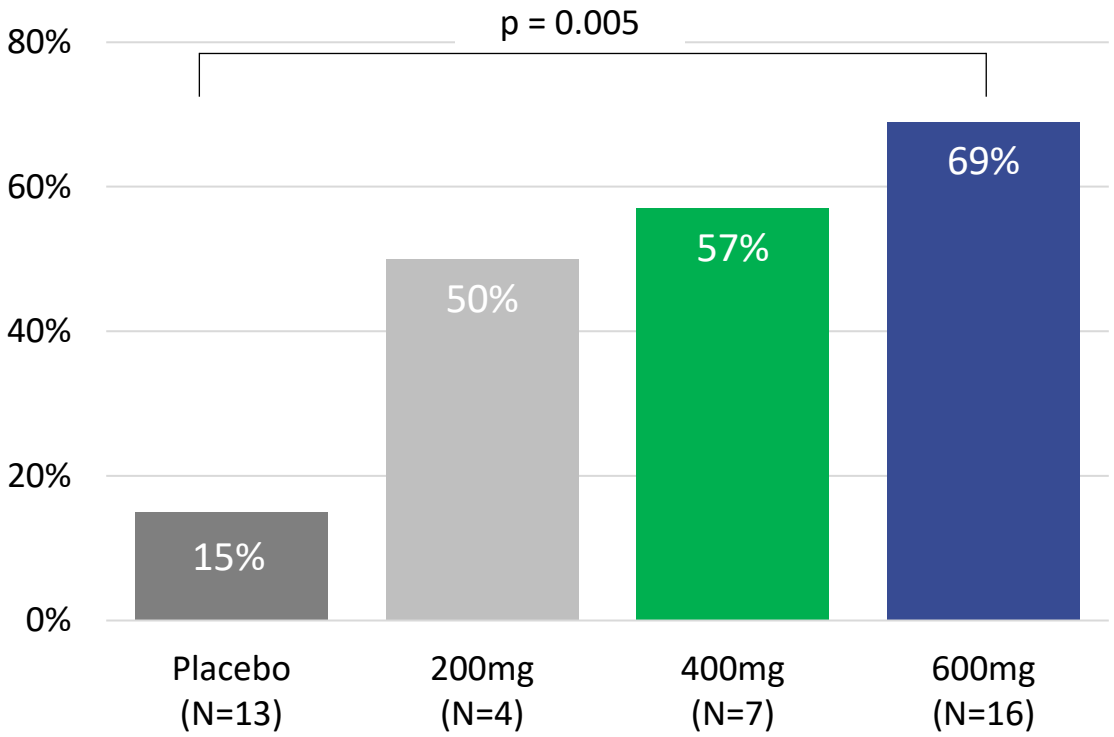




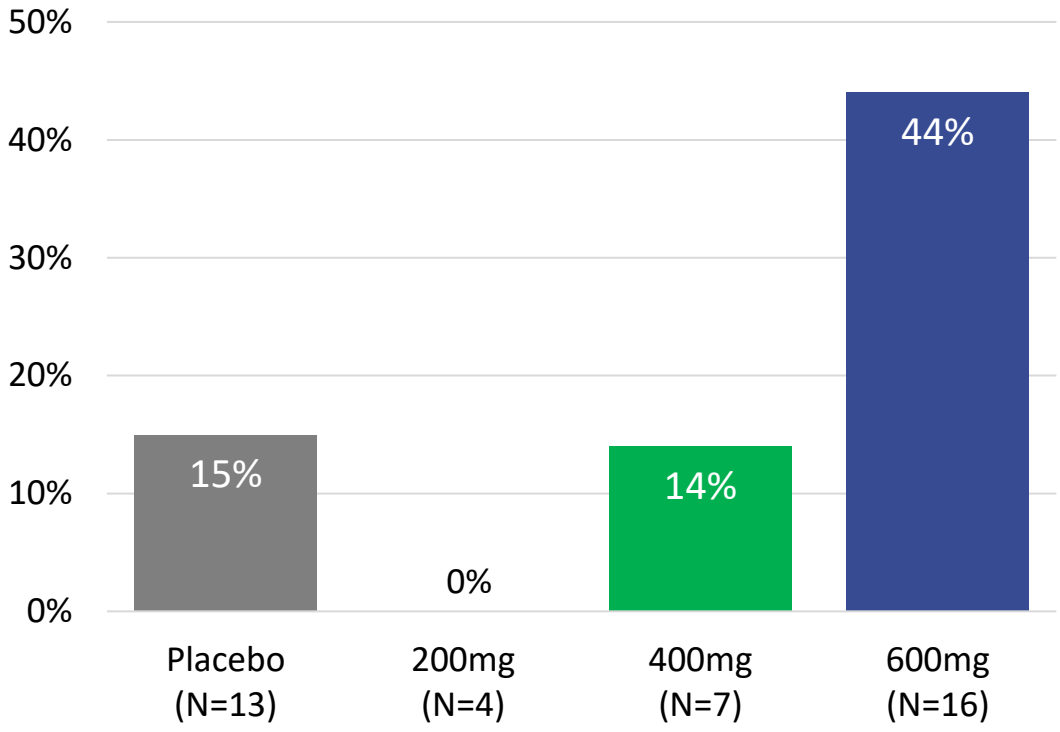
Data from mITT population, mean values, presented at 31st EADV Congress, September 7-10, 2022
p-values are one-sided

Other efficacy endpoints (week 8)

EASI-75



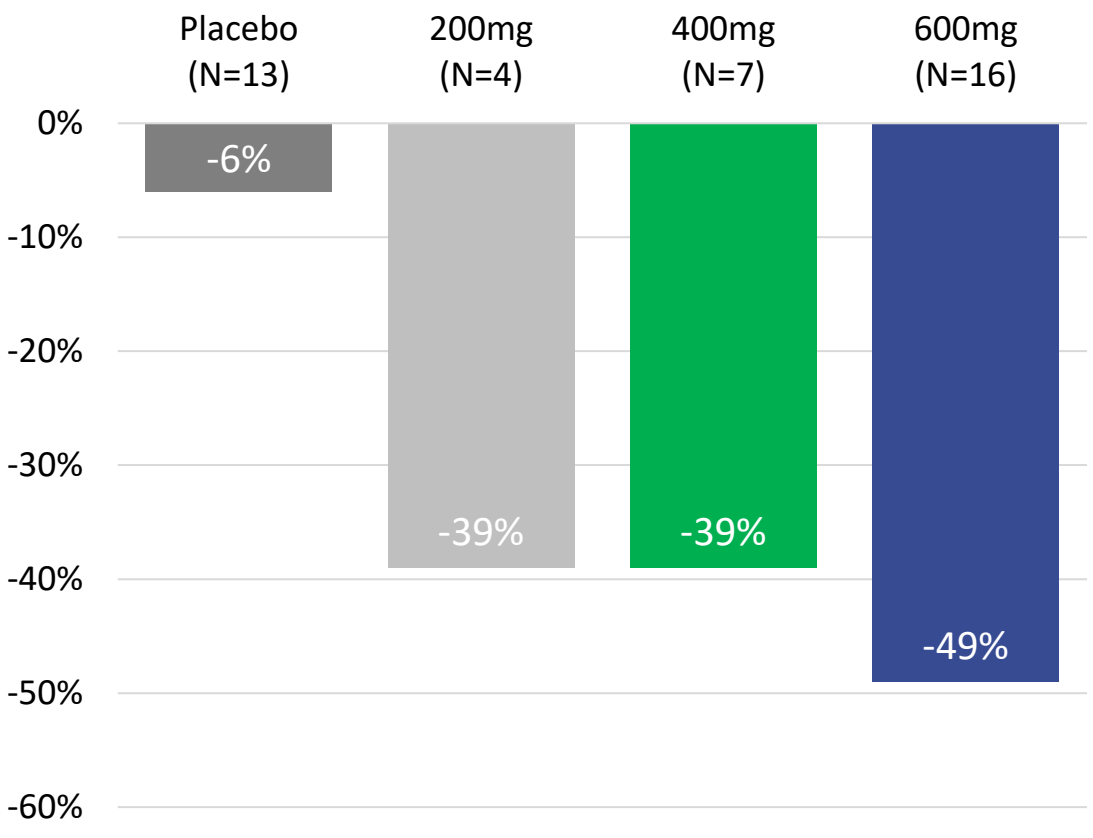
Patients achieving IGA 0/1



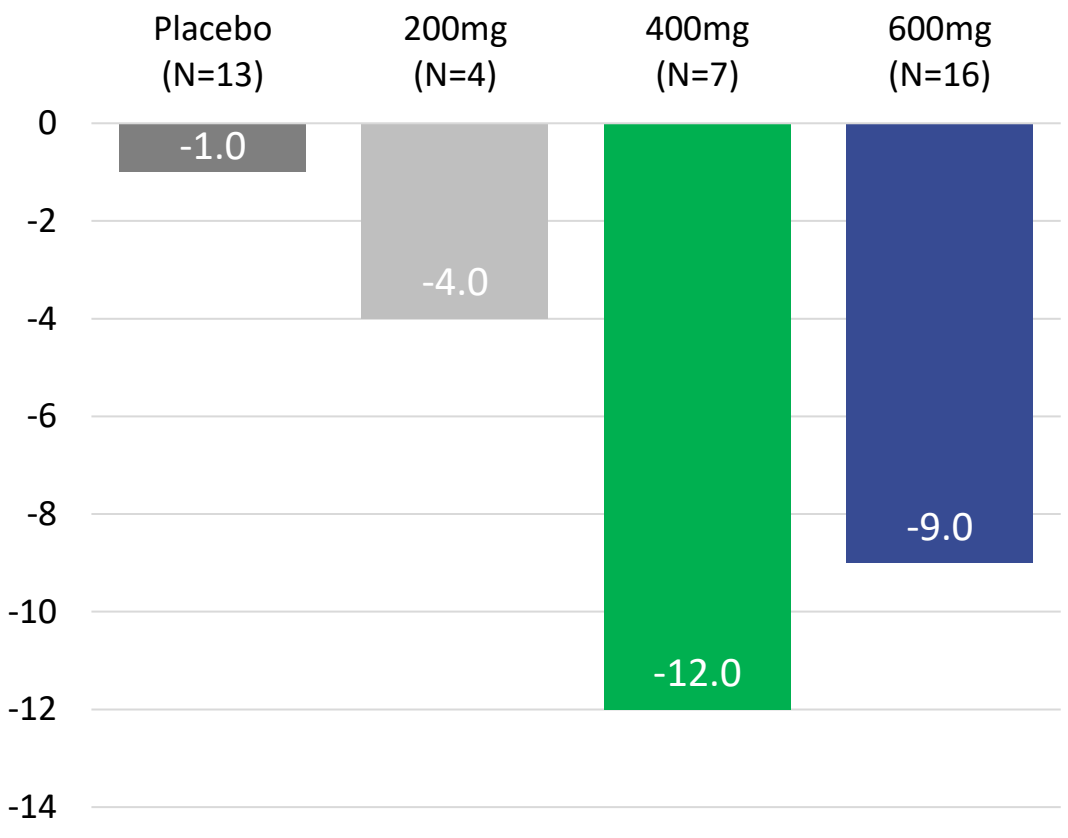
Data from mITT population, presented at 31st EADV Congress, September 7-10, 2022
p-values are one-sided

Patient reported endpoints (week 8)

Peak pruritus (P-NRS)

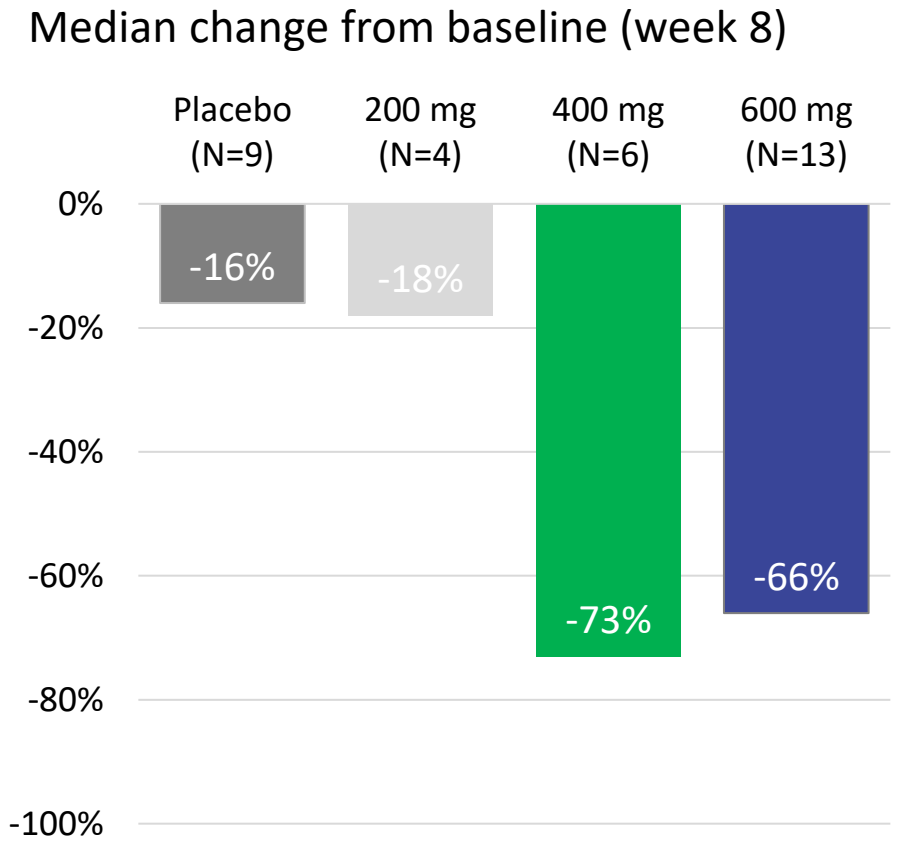


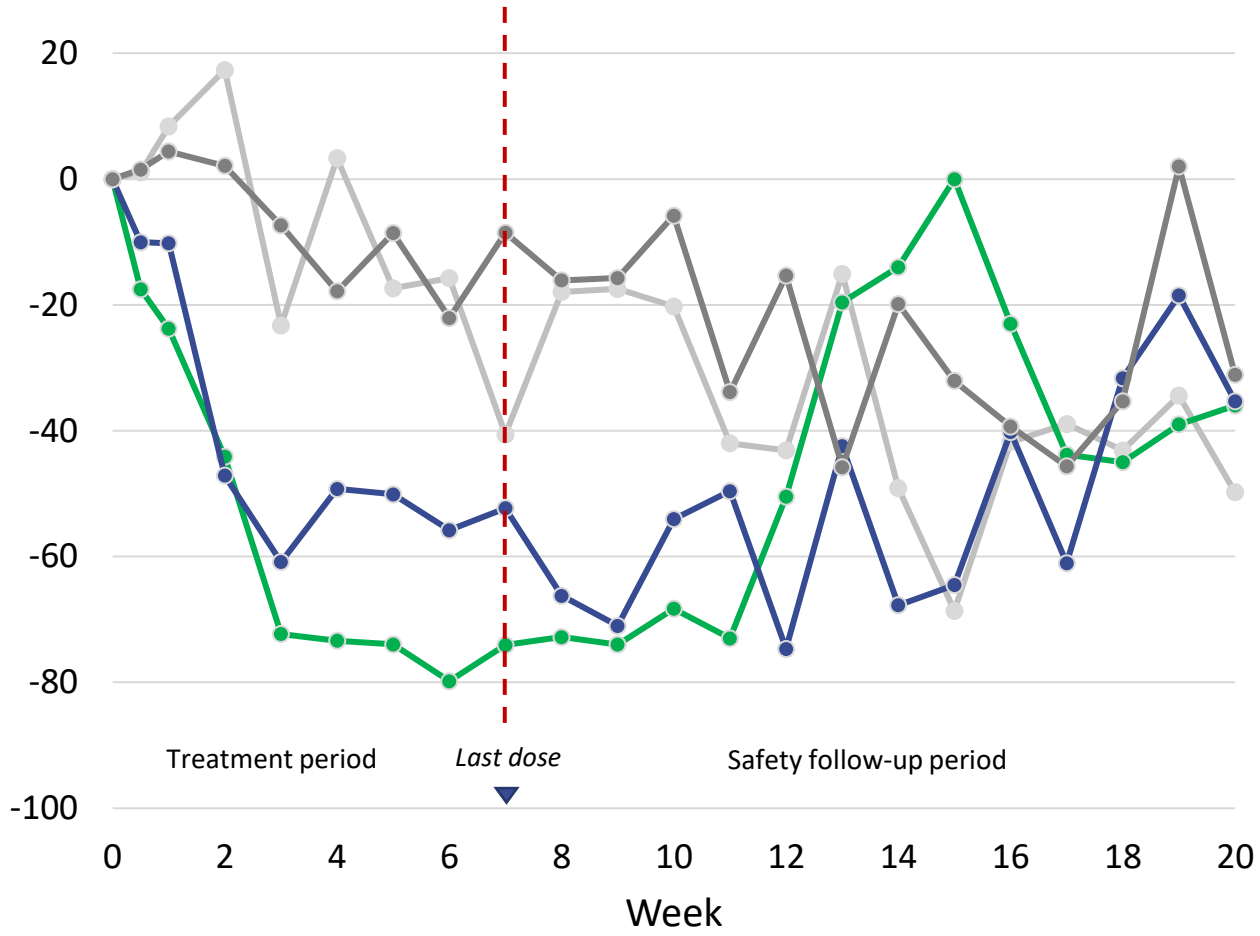
POEM



Data from mITT population, median values, presented at 31st EADV Congress, September 7-10, 2022

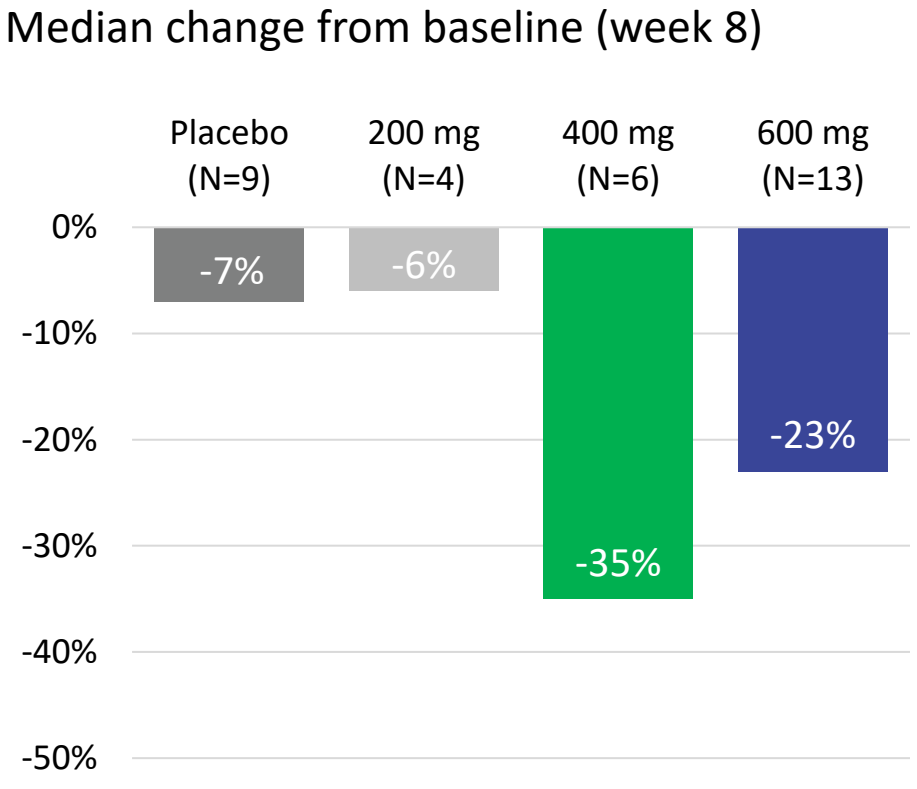
TARC biomarker

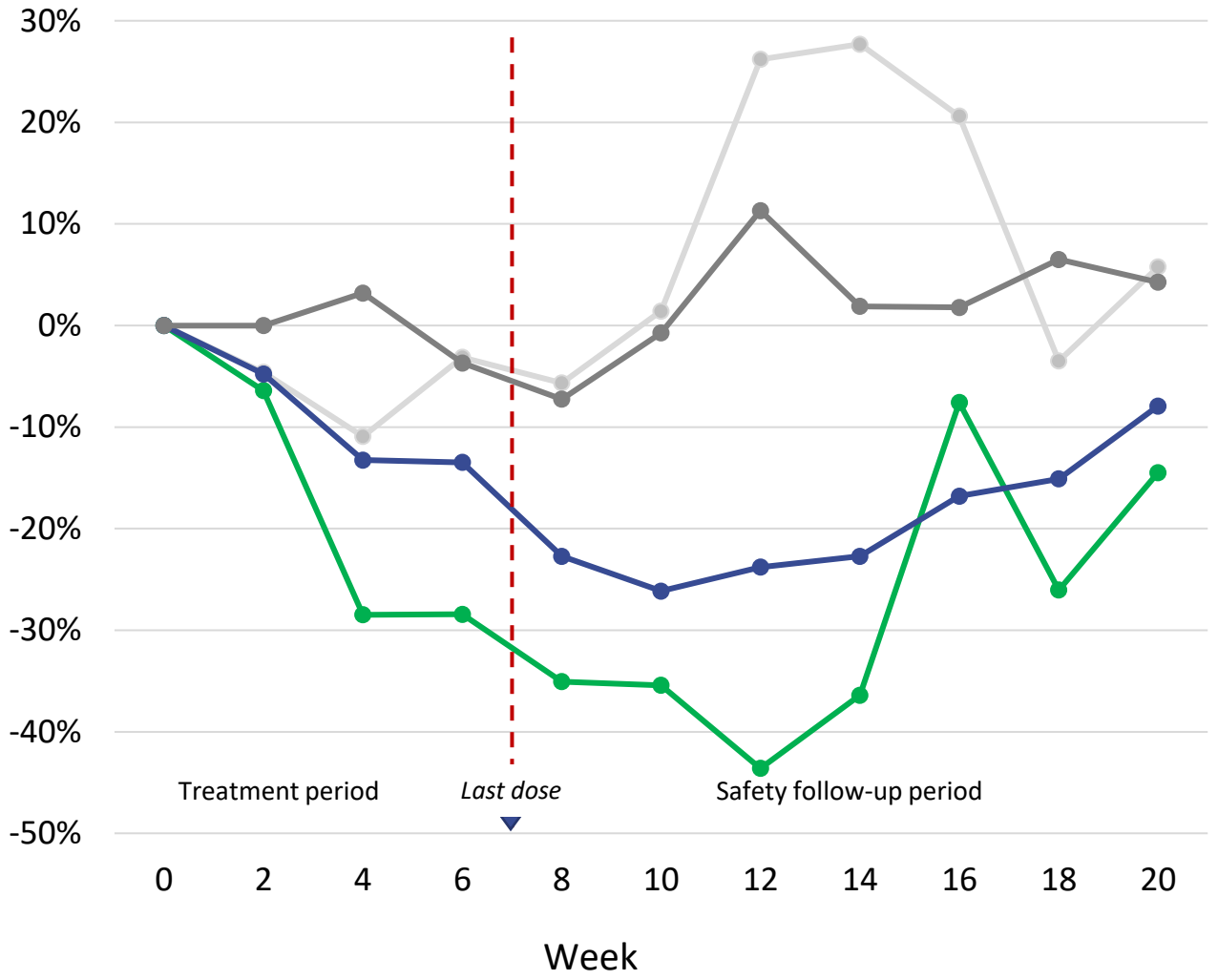




Data from mITT population, presented at 31st EADV Congress, September 7-10, 2022

IgE biomarker





Data from mITT population, presented at 31st EADV Congress, September 7-10, 2022

Eblasakimab well-tolerated with low incidence of conjunctivitis

Treatment Emergent Adverse Event (TEAE) by category [1]	All patients dosed (N=52)		
	600mg (N=22)	200-600mg (N=35)	Placebo (N=17)
Any	12 (55%)	25 (71%)	8 (47%)
Related	8 (36%)	19 (54%)	7 (41%)
Moderate/severe	6 (27%)	11 (31%)	5 (29%)
Serious adverse event (SAE)	0 (0%)	1 (3%)	0 (0%)
Drug-related AEs of interest [2] :			
• Injection site reaction	5 (23%)	9 (26%)	2 (12%)
• Conjunctivitis	1 (5%)	2 (6%)	0 (0%)

- Conjunctivitis was classified as mild. Patients had prior history of allergy or allergic conjunctivitis
- Rescue medication use: 3 patients on placebo arm, 1 patient on 600mg arm

1 Presented at AAD Annual Meeting, 25-29 March 2022
2 Drug-related AEs defined by the investigators as definitely related, probably related or possibly related

Eblasakimab
Ongoing development program

High unmet need in biologic eligible and experienced population

AD patients → Moderate-to-severe →

TARGET MARKET

Biologic eligible
- Biologic naïve population seeking safe long-term treatment
- *Dupilumab* is standard of care

↓

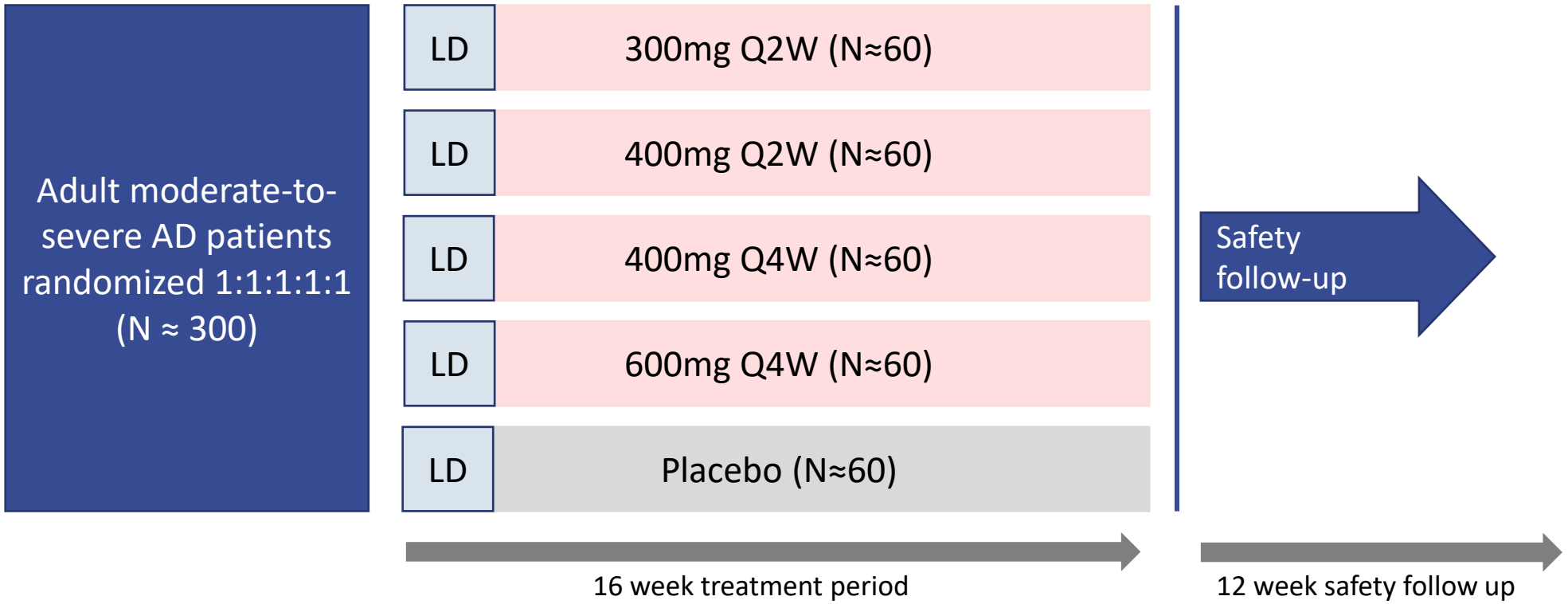
Dupilumab treated

↓

Dupilumab experienced
- Patients who show sub-optimal response
- Response declines over time
- Discontinuations due to conjunctivitis

- Our clinical studies address the target market in AD, comprised of biologic naïve and experienced patients
- Positive clinical outcomes will increase preference for *eblasakimab* as the first choice of biologic for AD in both patient populations in the target market

TREK-AD: Phase 2b in biologic naïve patients
Enrollment complete, topline data expected early July 2023



- Loading dose of 600mg for the Q2W dose groups at week 1 and week 2
- Loading dose of 600mg for the Q4W dose groups at week 1, week 2 and week 3

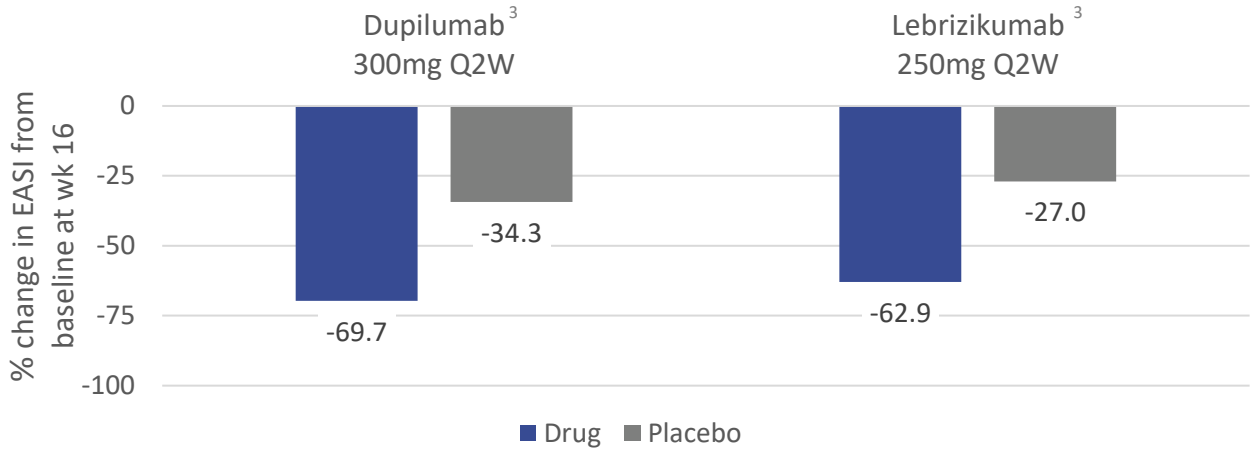
TREK-AD:
Primary endpoint is % change in EASI from baseline at wk 16

Primary endpoint:

- EASI: Eczema Area Severity Index
- Validated in 2004 [1]
- Recommended as the core outcome measure for clinical signs of eczema [2]

Other key secondary endpoints:

- EASI-75, EASI-90
- vIGA-AD 0/1
- PP-NRS: % change from baseline

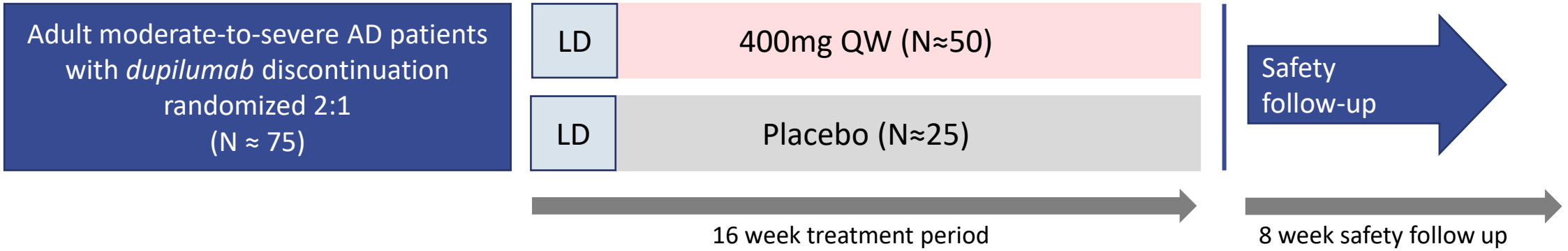


vIGA-AD vs IGA

- Validated IGA-Atopic Dermatitis (vIGA-AD) measure established in 2020 [4]
- Previous trials with *dupilumab* and *lebrikizumab* used non-validated IGA scores
- vIGA-AD more stringent measure, includes skin lichenification in assessment matrix

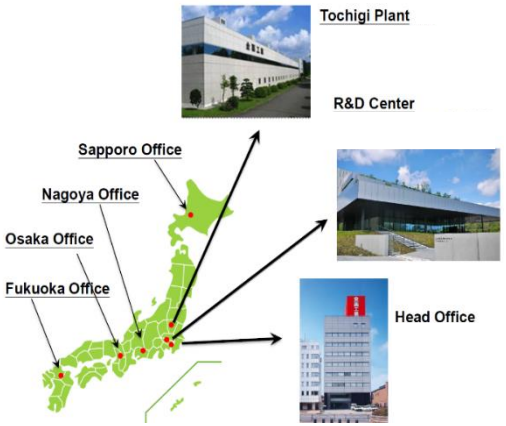
1 Barbier et al (2004) BJD 150(1):96-102
2 Chalmers et al (2014) BJD:171(6):1318-25, report from Harmonizing Outcome Measures for Eczema (HOME)
3 Represented values are averages pooled from respective Phase 3 trials
4 Simpson et al (2020) JAAD 83(3):839-846

TREK-DX: Phase 2 study in *dupilumab* experienced patients
Topline data expected 1Q 2024



- Loading dose of 600mg at week 1 and week 2
- Randomization stratified by
  - Reason for *dupilumab* discontinuation (failure or non-failure)
  - Baseline vIGA score (3 or 4)
- The TREK-DX study is crucial for testing *eblasakimab* in a patient population with few safe treatment options
- A positive outcome of TREK-DX will strengthen the positioning of *eblasakimab* as the biologic of first choice for AD treatment

Eblasakimab licensed in Japan to leading local pharma ZK



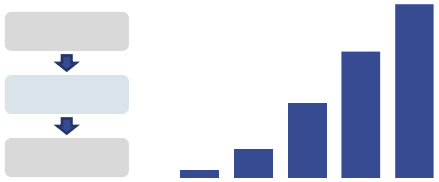
Zenyaku Kogyo

- Leading Japanese biopharmaceutical company marketing prescription and OTC drugs in dermatology
- Markets anti-CD20 biologic Rituxan(*rituximab*) in the Japanese market
- Successfully obtained regulatory approvals in Japan for Rituxan in 11 additional indications, including several oncology and dermatology indications, 7 of which are unique to the Japanese market

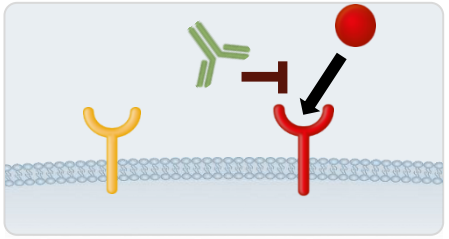
Deal terms

- $12M in upfront and $3M in near-term payments
- Up to $123.5M on development and commercial milestones
- Tiered royalties on sales, ranging up to low twenties percentages
- ASLAN retains option to reacquire rights to *eblasakimab* in Japan in the future, exercisable at any time
- Zenyaku plans to initiate a Phase 1 study of *eblasakimab* for the treatment of moderate-to-severe AD in Japan in the second half of 2023

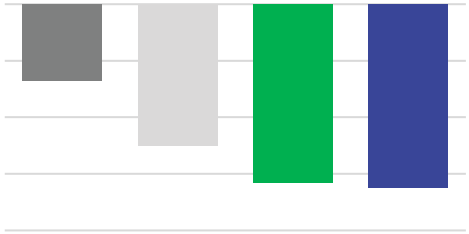
Eblasakimab has the potential to be an important new therapy for the treatment of AD and other Type 2 inflammatory diseases



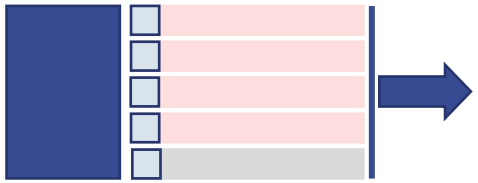
Estimated $24B market with only 2 approved biologics



Unique mechanism of action that provides the potential for improved efficacy, safety, dosing



Positive POC - met primary efficacy endpoint (p=0.01) with significant changes in other efficacy and biomarker endpoints

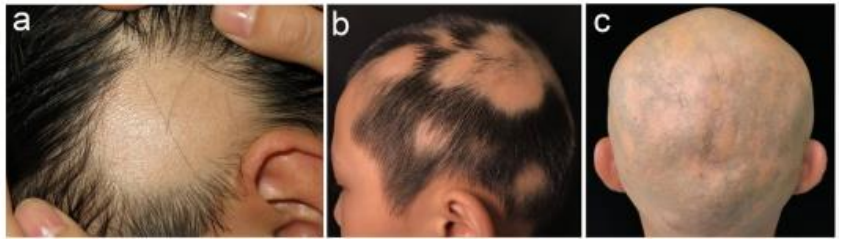


Phase 2b readout in early July 2023 in biologic naive patients with data in *dupilumab* experienced patients to follow

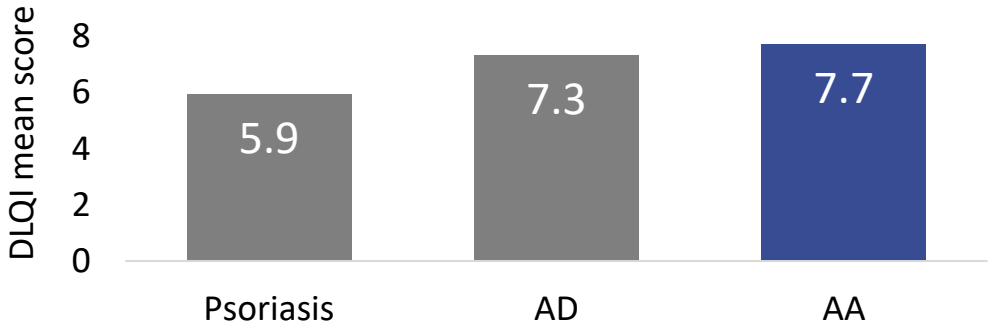
Farudodstat

High burden of disease – around 700,000 patients in the US alone

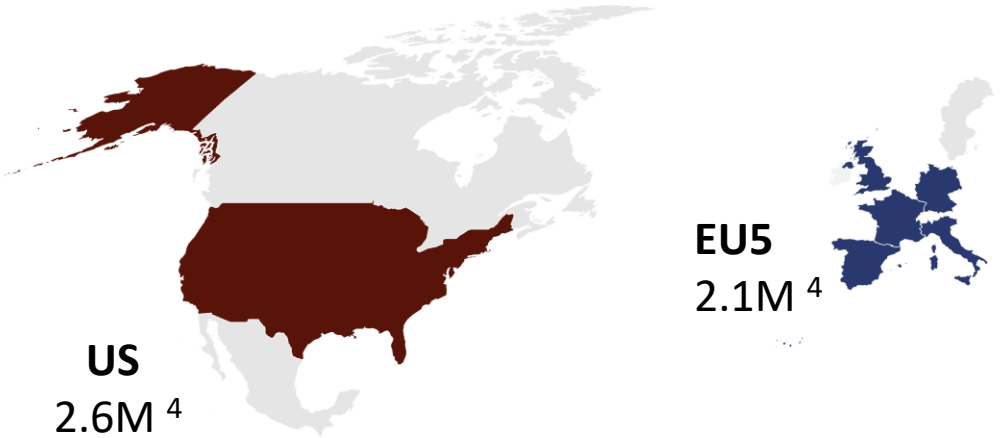
AA is a common autoimmune disease characterised by complete or partial hair loss [1]



AA has profound negative impact on quality-of-life scores, similar or worse than other dermatologic diseases [2,3]



Total diagnosed lifetime prevalence AA cases

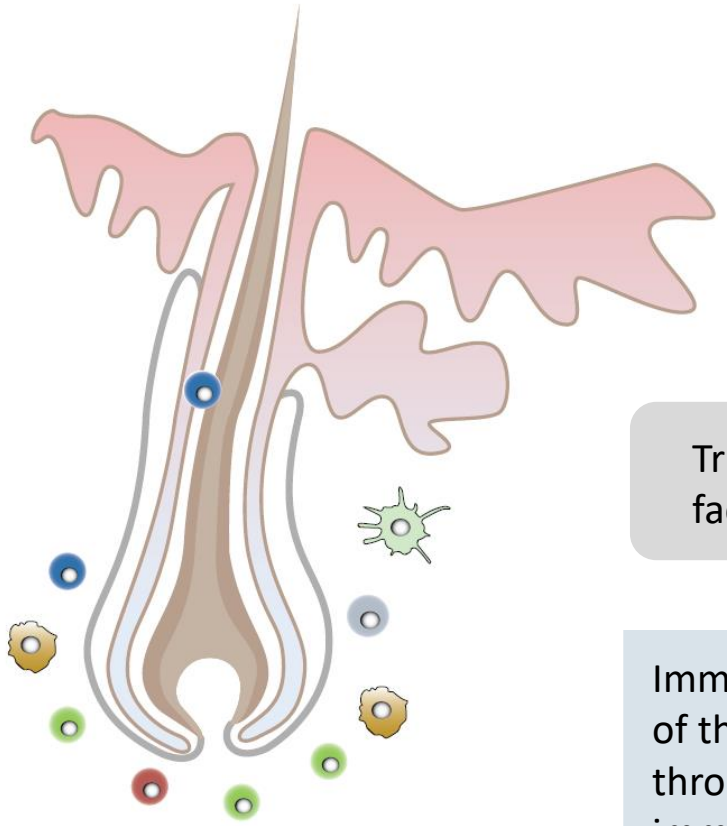


- **2.1%** of the population can develop AA at some point in their lifetime[5]
- **700k** patients in the US in 2020 [6,7]
- **25%** of patients have **severe** disease
- **62%** of AA patients **receive drug** treatment [4]

1. Zhou et al (2021) Clin Rev All Imm 61:403-423
2. Liu et al (2018) JAAD 79(3):556-558
3. Lundberg et al (2000) Acta Derm Venereol 80(6):430-434
4. DRG Alopecia Areata Disease Landscape and Forecast report 2023
5. Mirzoyev et al (2014) J Inv Derm 134(4):1141-1142
6. Benigno et al (2020) Clin, Cos & Invest Derm 13:259-266
7. Mostaghimi et al (2023) JAMA Derm, published online

AA results from collapse of immune privilege of the hair follicle

Healthy hair follicle

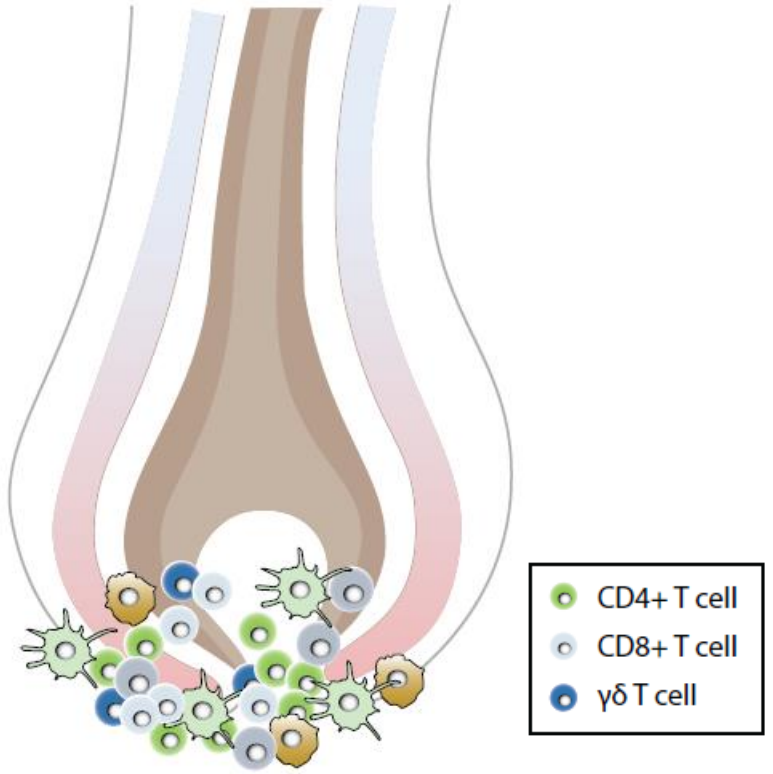


Immune privilege: intact

Trigger factors → Activated T cells + IFNγ → MHC I & II expression →

Immune privilege (IP) describes the protection of the hair follicle from the immune system through absence of MHC I expression and an immunosuppressive environment

Alopecia areata affected hair follicle



Immune privilege: collapse

Moderate-to-severe AA patients have limited treatment options

Mild
<30% hair loss

→ Intralesional Corticosteroids (ILCS)[1]

- ILCS can be effective with high spontaneous remission rate but can only be used for patients with <50% hair loss

Moderate
30-50% hair loss

→ Corticosteroids: Intralesional, topical or oral

- TCS and oral corticosteroids have limited effectiveness
- High frequency of ILCS is painful

Severe
>50% hair loss

→ JAK inhibitors

- JAKi have several boxed warnings and are effective in only 30-40% of patients[2]
- Discontinuation can result in rapid relapse

1. Oral Minoxidil is also used to encourage hair growth but is not approved and has limited efficacy
2. % patients achieving primary endpoint of SALT score ≤20 in published Phase 3 studies of JAK inhibitors in AA

AA pipeline is dominated by JAKi, novel mechanisms are needed

MoA	Company	Product	Stage
JAK inhibitors	Lilly	Olumiant *(baricitinib)*	Approved
	Pfizer	*Ritlecitinib*	Phase 3
	CoNCERT Pharmaceuticals Inc.	*Deuruxolitinib*	Phase 3
	Bristol Myers Squibb	*Deucravacitinib*	Phase 2
	Reistone Biopharma	SHR0302	Phase 2
S1P Inhibitor	Pfizer	*Etrasimod*	Phase 2
IL2/9/15 inhibitor	equillium	EQ101 (exBNZ-1)	Phase 2 open label
Anti-ILT7	HORIZON	*Daxdilimab*	Phase 2 open label

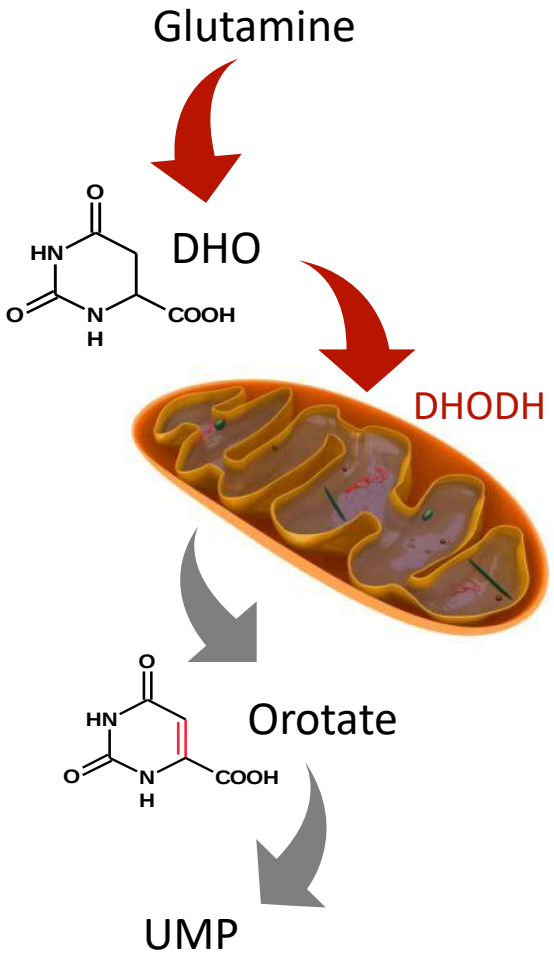
WARNING: SERIOUS INFECTIONS, MORTALITY, MALIGNANCY, MAJOR ADVERSE CARDIOVASCULAR EVENTS (MACE), and THROMBOSIS

See full prescribing information for complete boxed warning.

- **Increased risk of serious bacterial, fungal, viral and opportunistic infections leading to hospitalization or death, including tuberculosis (TB). Interrupt treatment with OLUMIANT if serious infection occurs until the infection is controlled. OLUMIANT should not be given to patients with active tuberculosis. Test for latent TB before and during therapy, except for COVID-19; treat latent TB prior to use. Monitor all patients for active TB during treatment, even patients with initial negative, latent TB test. (5.1)**
- **Higher rate of all-cause mortality, including sudden cardiovascular death with another Janus kinase inhibitor (JAK) vs. TNF blockers in rheumatoid arthritis (RA) patients. (5.2)**
- **Malignancies have occurred in patients treated with OLUMIANT. Higher rate of lymphomas and lung cancers with another JAK inhibitor vs. TNF blockers in RA patients. (5.3)**
- **Higher rate of MACE (defined as cardiovascular death, myocardial infarction, and stroke) with another JAK inhibitor vs. TNF blockers in RA patients. (5.4)**
- **Thrombosis has occurred in patients treated with OLUMIANT. Increased incidence of pulmonary embolism, venous and arterial thrombosis with another JAK inhibitor vs. TNF blockers. (5.5)**

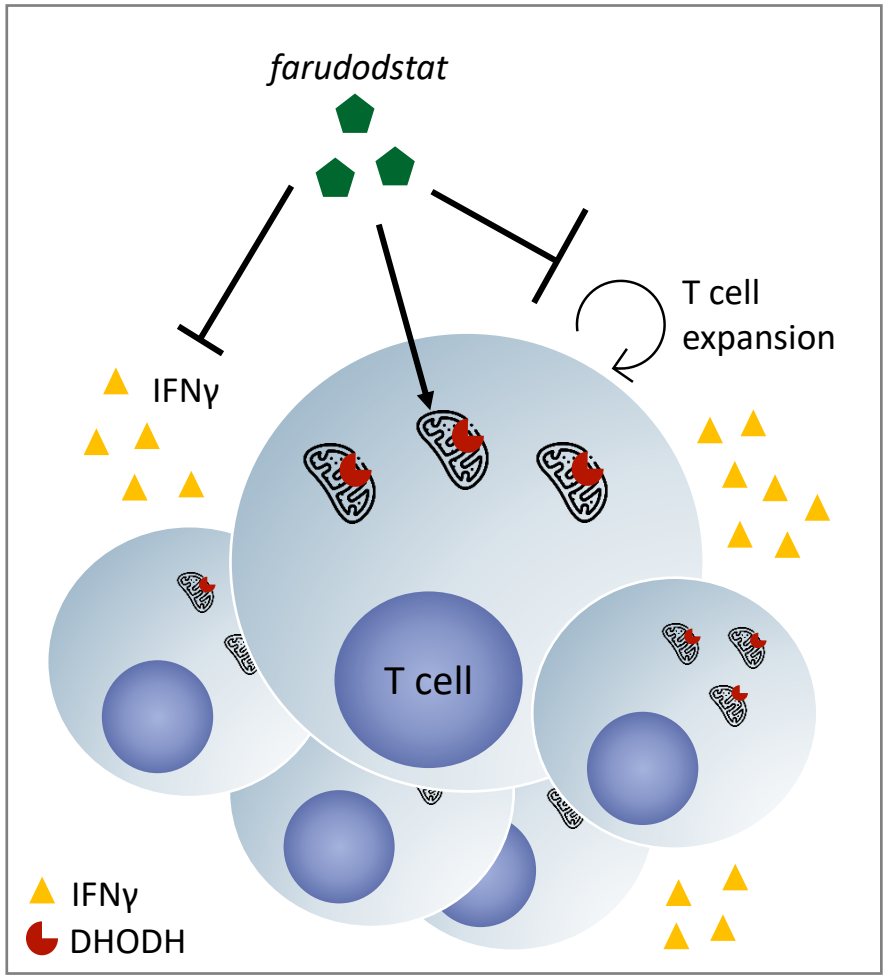
Proof-of-concept not yet established

DHODH is a validated target for autoimmune disease



- Cells synthesise pyrimidines via
  - *De novo* pathway (DHODH dependent)
  - Salvage pathway (DHODH independent)
- DHODH inhibition will block *de novo* synthesis, impacting rapidly dividing cells eg lymphocytes
- Other cells can continue to use the salvage pathway and are unaffected
- First generation inhibitors are approved in MS (Aubagio) and RA (Arava), however they have limited potency and significant safety liabilities
- *Farudodstat* was designed to be more potent and to address the toxicities associated with first generation inhibitors

Farudodstat potently blocks IFNγ secretion from immune cells, supporting its potential as a treatment for AA



Farudodstat potently inhibits key drivers of AA pathophysiology

- IFNγ secretion
- T cell expansion

Farudodstat has the potential to be first-in-class for AA

Highly selective and 30-fold more potent than first generation DHODH inhibitors

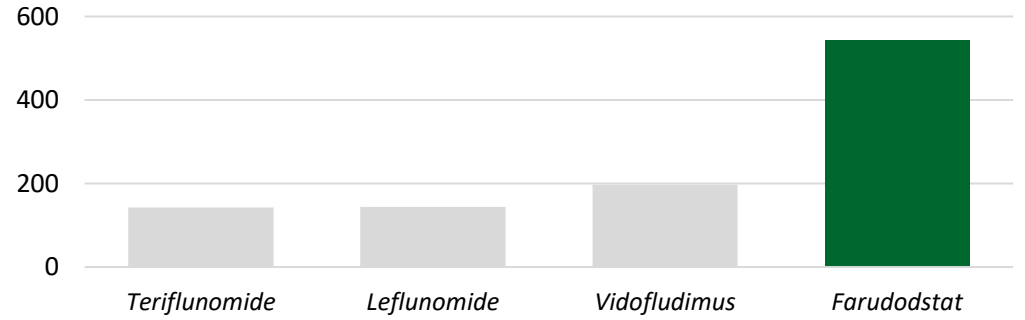
- Stronger *in vitro* potency as compared with other DHODH inhibitors
- Selective against a panel of 195 enzymes, ion channels and receptor binding assays

IC_{50} inhibition (µM)	DHODH	Human PBMC proliferation	IFNγ secretion
Farudodstat	0.035	1.4	2.5
Teriflunomide	1.1	46	259
Relative potency	> 30-fold	> 30-fold	> 100-fold

No evidence of hepatotoxicity in non-clinical studies

- Rodent liver function models: No hepatotoxicity caused by *farudodstat* (in contrast to *teriflunomide* which was hepatotoxic at lower doses)
- *In vitro* studies demonstrated *farudodstat* has lowest potential for hepatotoxicity out of 6 approved and clinical stage DHODH inhibitors [1]

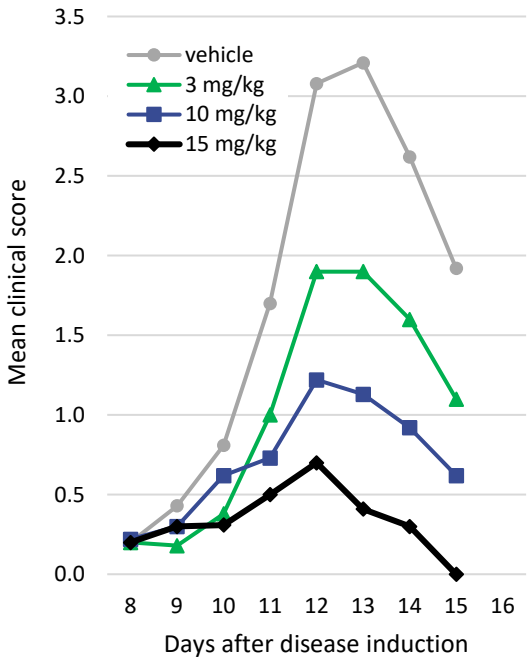
Concentration (µM IC_{50}) required to induce mitochondrial toxicity in HepaRG cells at 24 hrs[1]



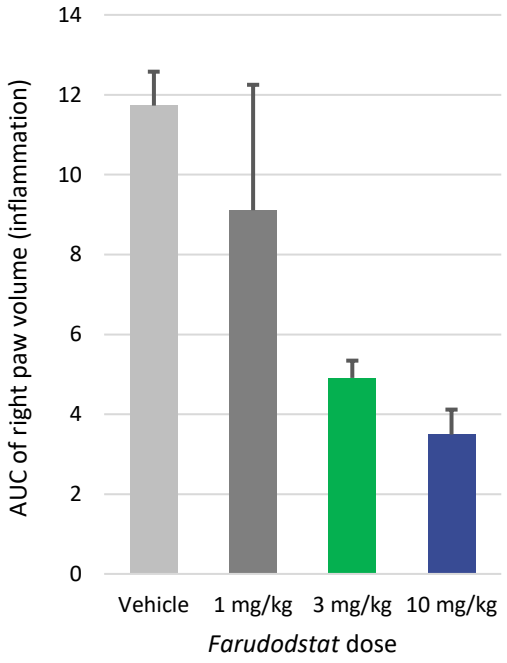
1 Jones et al (2021) Toxicology in Vitro 72:105096

Farudodstat has the potential to be first-in-class for AA

- Active in the multiple sclerosis EAE model and rheumatoid arthritis AIA model

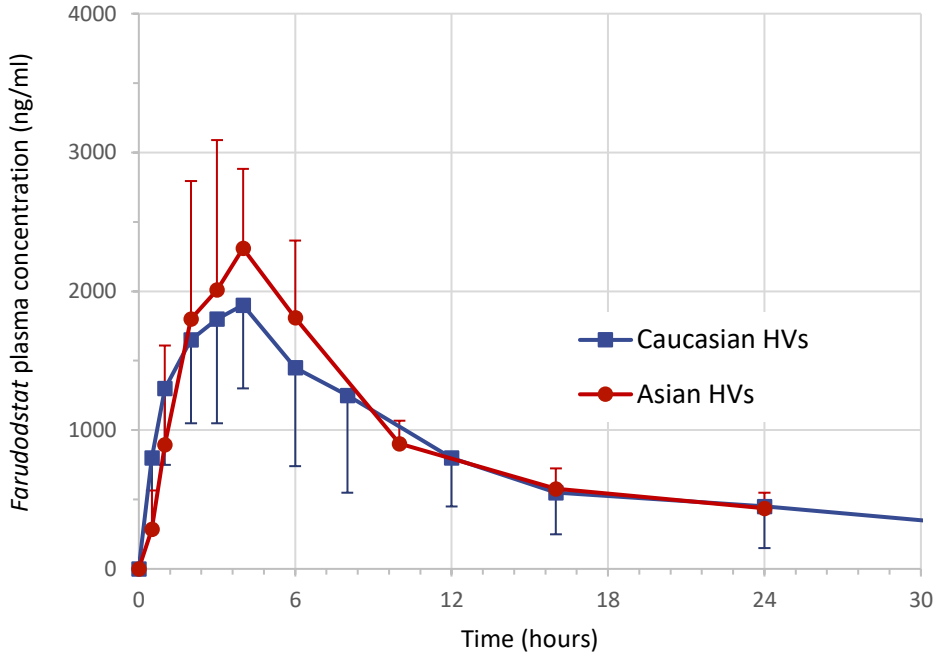


MS model in rat (EAE)



RA model in rat (AIA)

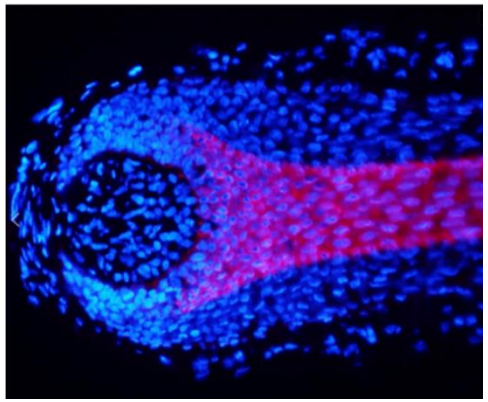
- Well-tolerated in 119 subjects in Phase 1 and Phase 2 clinical trials
- PK profile suitable for once-daily dosing

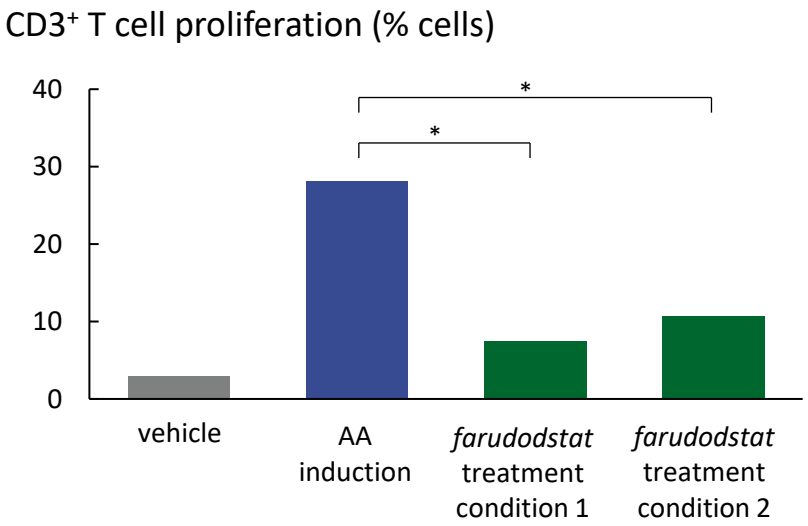


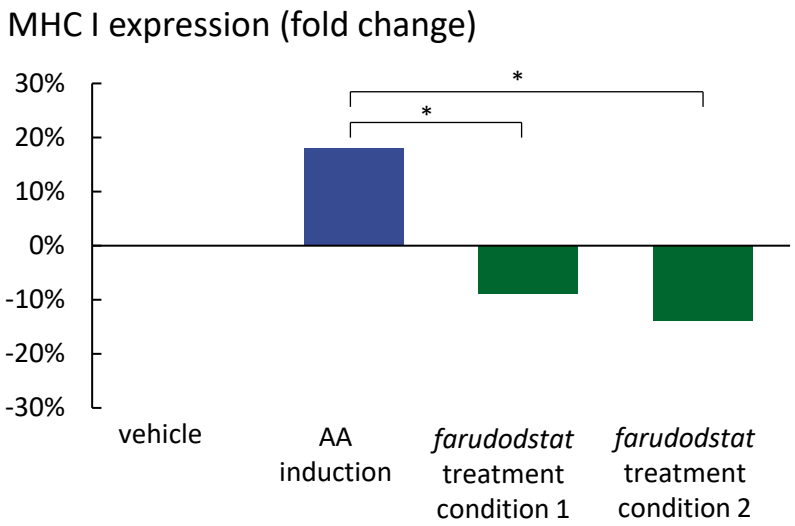
Single dose pharmacokinetics

Ex vivo human model indicates *farudodstat* has potential to restore IP collapse and inhibit key drivers of AA

- Microdissected hair follicles from healthy human scalp cultured *ex vivo* [1-4]
- Stimulation by anti-CD3/CD28 led to IP collapse and AA induction
- *Farudodstat* was tested at clinically relevant doses and reduced key drivers of IP collapse, including T cell proliferation and MHC expression



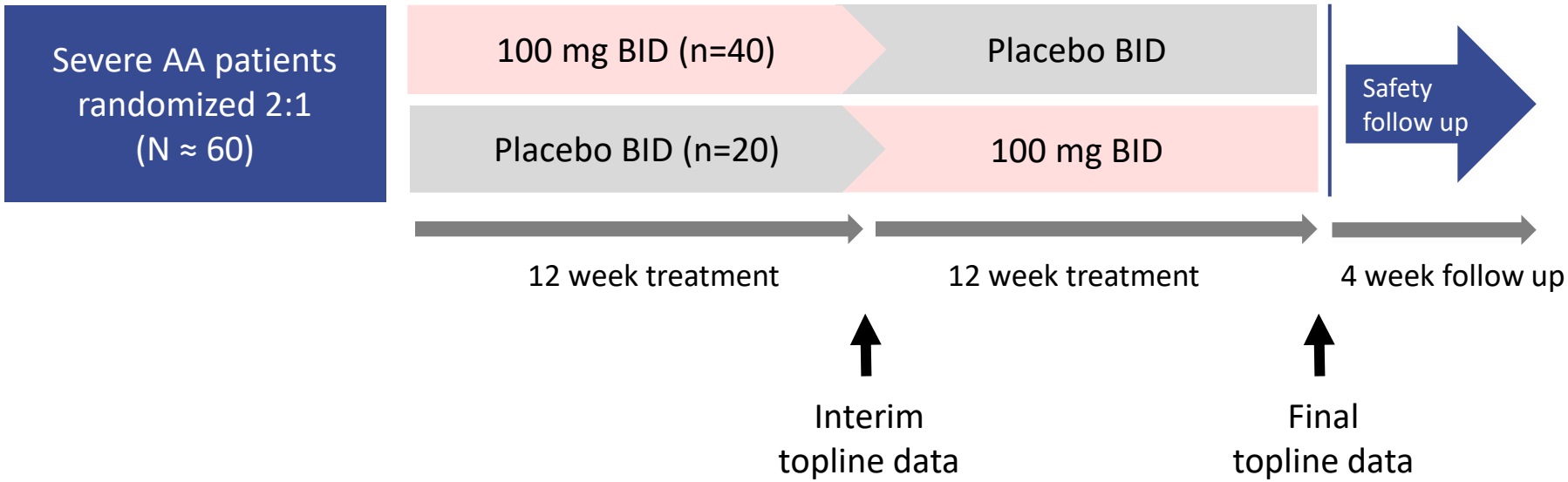




1. Bertolini et al (2016) Br J Derm 175(3):531-41
2. Fehrholz & Bertolini (2020) Methods Mol Biol 2154:133-141
3. Bertolini et al (2021) Br J Dermatol 184(2):371-373
4. Uchida et al (2021) J Autoimmun 124:102711

* $p<0.05$; n=7 for each condition tested

Phase 2a: Proof-of-concept trial in AA, topline expected 1Q 2024

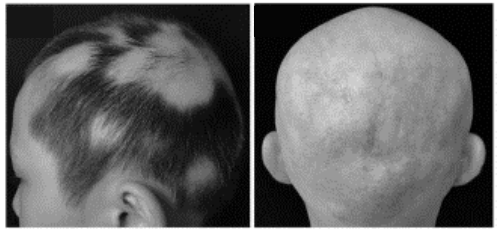


Primary efficacy endpoint: % change from baseline in SALT score

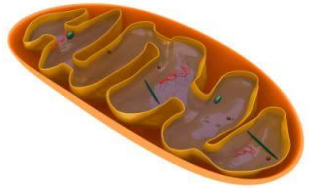
Select inclusion criteria:

- Adults with at least 50% scalp hair loss (SALT score ≥ 50)
- Current episode of hair loss duration between 6 months to 7 years

Farudodstat has the potential to be an effective, novel approach in the treatment of AA



High burden of disease and unmet need in AA with few effective treatments.



Farudodstat is 30-fold **more potent** at inhibiting DHODH, a validated target, than first-generation inhibitors with **less potential for hepatotoxicity**

***Farudodstat* inhibits the key drivers of AA pathophysiology:** IFNγ secretion, T cell expansion and MHC expression

Phase 2a proof-of-concept study in AA initiated, interim **topline readout expected 1Q 2024**

Financials

Financials and milestones

Ticker	NASDAQ: ASLN
Net operating cash used	$ 19.3M (1Q 2023)
Cash balance	$ 57.5M (as of March 31, 2023)
	Additional $12M upfront payment from *eblasakimab* licensing deal closed in June 2023

Upcoming expected milestones:

1Q 2023	2Q 2023	3Q 2023	4Q 2023	1Q 2024
$20 million financing led by BVF	*Farudodstat* Phase 2a PoC study in AA initiation	*Eblasakimab* licensing deal for Japan signed	*Eblasakimab* Phase 2b TREK-AD topline data readout	*Eblasakimab* Phase 2 TREK-DX topline readout
Eblasakimab TREK-AD completion of enrollment				*Farudodstat* Phase 2a interim topline readout