Company presentation

April 2022

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*, also known as ASLAN004, 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 recently completed multiple ascending dose (MAD) study conclusively establishes proof of concept for *eblasakimab* in AD, and supports a potentially differentiated safety and efficacy profile
  - Phase 2b study initiated in January 2022, evaluating 2-weekly and 4-weekly regimens.

- *Farudodstat*, also known as ASLAN003, is a second generation **DHODH inhibitor with the potential to be best-in-class** for autoimmune disease
  - Stronger *in vitro* potency and lower potential for hepatotoxicity compared to other DHODH inhibitors
  - Expecting to initiate phase 2 in IBD in 1H 2022. Planning future studies in autoimmune skin diseases

- **Strong cash position ($90M[2]) with runway to late 2023**

1 Decision Resources Group, June 2021
2 As of Q4 ending December 31, 2021

Developing innovative therapies to treat inflammatory disease

Program	Target	Discovery	Preclinical	Phase 1	Phase 2	Anticipated milestones
Eblasakimab (ASLAN004)	IL-13Rα1	Atopic dermatitis (AD)				• Phase 1b biomarker and PRO data in 2H 2022 • Phase 2b topline data in 1H 2023
		Type 2-driven disease				
Farudodstat (ASLAN003)	DHODH	Inflammatory bowel disease				• Initiate Phase 2 in 1H 2022
		Autoimmune skin disease				

Eblasakimab (ASLAN004)

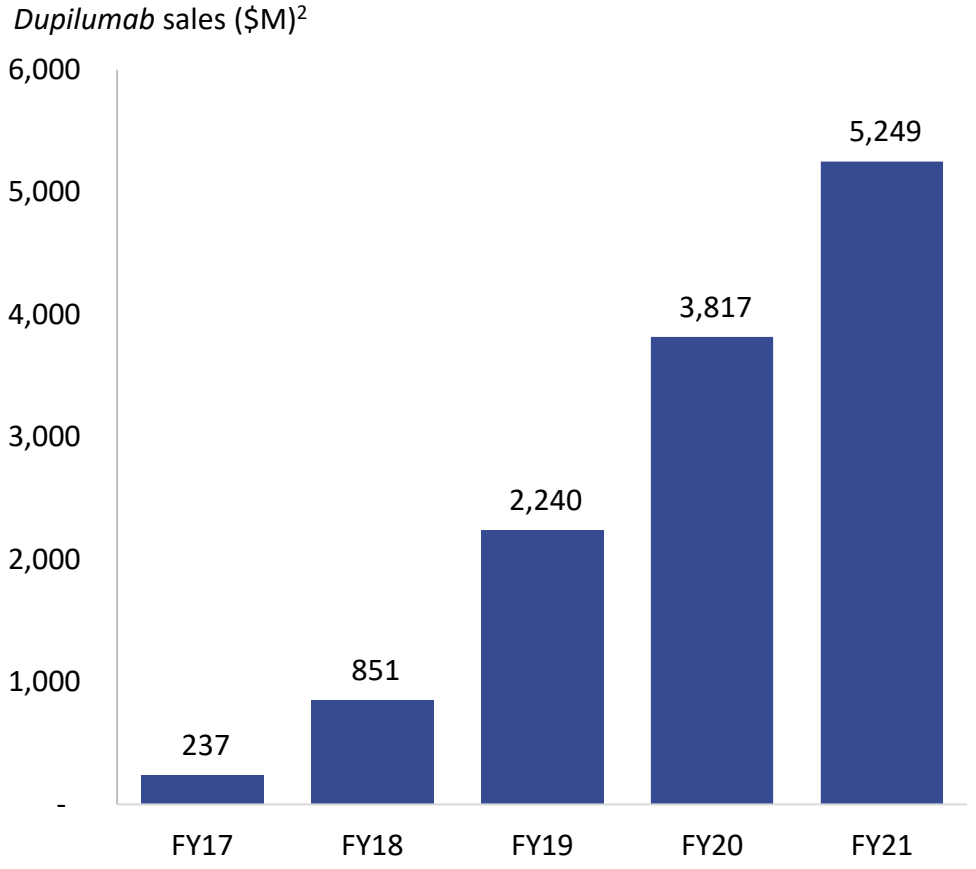
Eblasakimab: potential first-in-class IL-13 receptor antibody

- Novel antibody targeting IL-13 receptor (IL-13R), blocking both IL-4 and IL-13 signaling through the Type 2 receptor
- Topline data from recently completed MAD study conclusively establishes proof of concept for *eblasakimab* in AD, and supports a potentially differentiated safety and efficacy profile
  - *Eblasakimab* demonstrated a statistically significant improvement versus placebo in the primary efficacy endpoint of percent change from baseline in EASI (at 8 weeks)
  - *Eblasakimab* also showed statistically significant improvements in other key efficacy endpoints: EASI-50, EASI-75, peak pruritus, POEM
  - Well-tolerated with no emerging safety concerns
- Phase 2b study initiated in January 2022, evaluating 2-weekly and 4-weekly regimens.

Topline data demonstrate a potential best-in-class profile in terms of efficacy and safety

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
  - Launch of *dupilumab* in 2017 helped drive a large market for systemic AD therapy
  - Sanofi expects to grow sales to over $14B
- However, there remains a significant unmet need:
  - Only 35% of patients treated with *dupilumab* achieved an optimal response[1]
  - Conjunctivitis is common and can lead to treatment discontinuations
  - Opportunity to improve upon biweekly dosing regimen



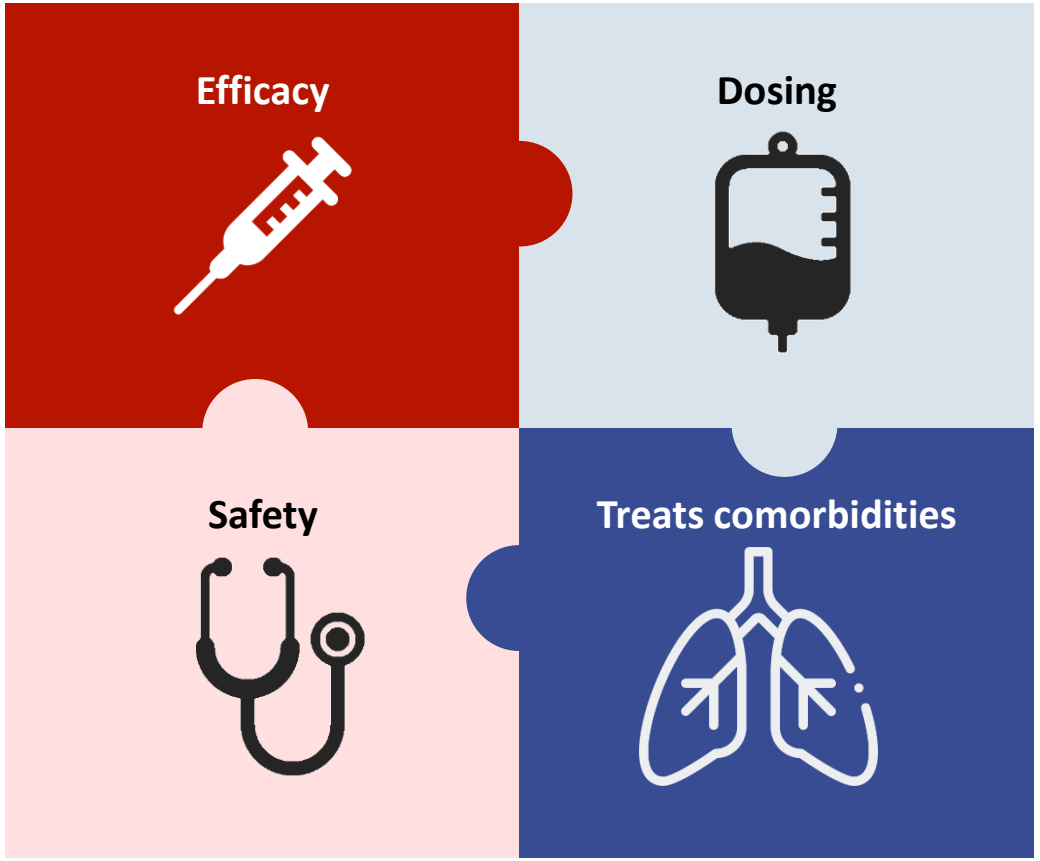
1 Spherix (2018) Atopic dermatitis ATU study

2 Sanofi's quarterly financials and annual reports

Eblasakimab has the potential to be a differentiated therapy in AD

Ideal target product profile

Better efficacy over current standard-of-care with rapid control of itch

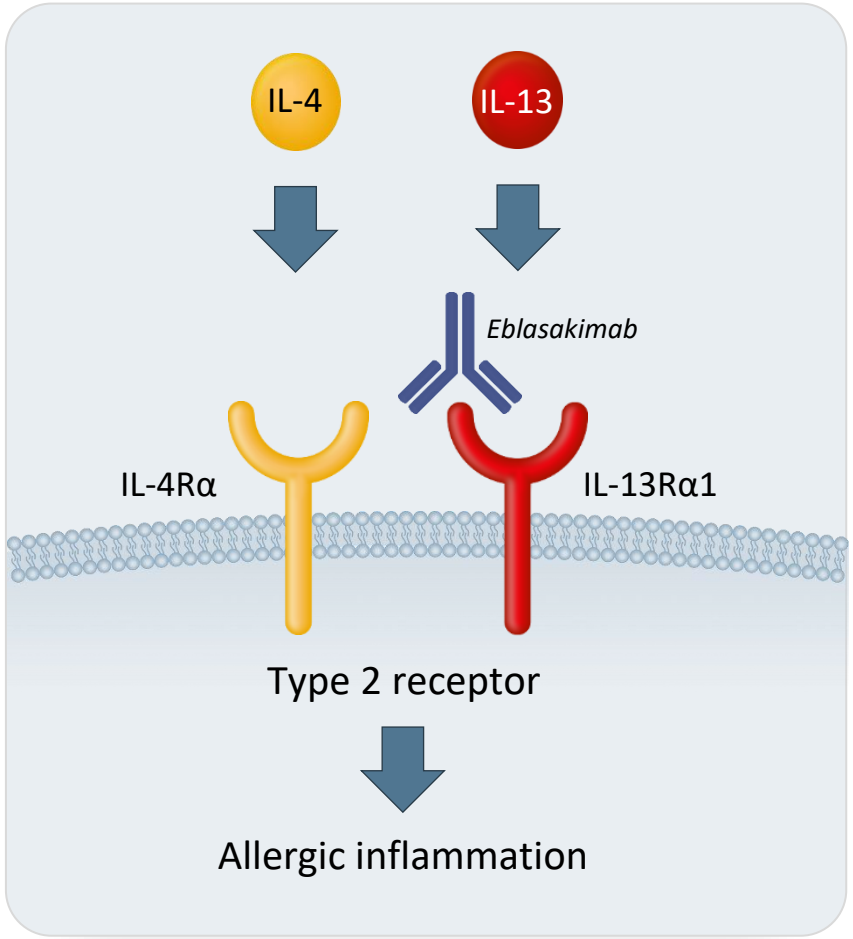


Monthly dosing, improving convenience and compliance

Addresses physician concerns on safety with lower rate of discontinuation

Able to address allergic comorbidities such as asthma and rhinitis

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

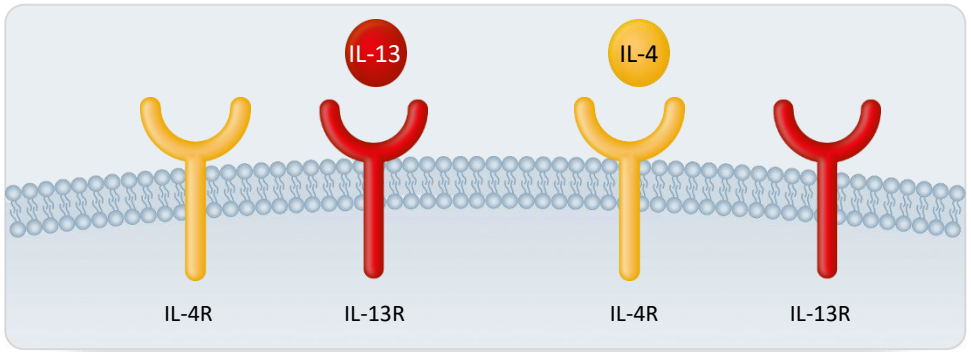


- IL-4 and IL-13 are central to triggering allergy and symptoms of atopic dermatitis
- By targeting the IL-13 receptor, *eblasakimab* blocks the Type 2 receptor complex, preventing signaling through **both** IL-4 and IL-13

Potential for improved efficacy, safety and dose regimen:

- Selectively targets the Type 2 receptor. Blocking the Type 1 receptor may lead to unwanted effects
- Stronger binding to receptor than *dupilumab* relative to its respective ligand

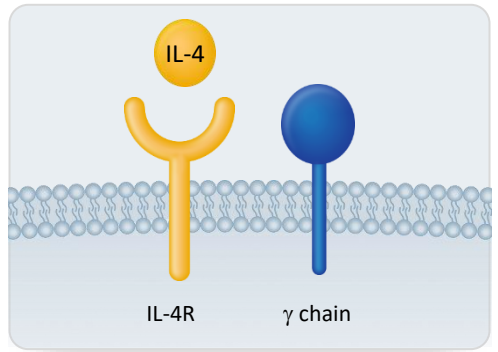
Eblasakimab selectively blocks the Type 2 receptor



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

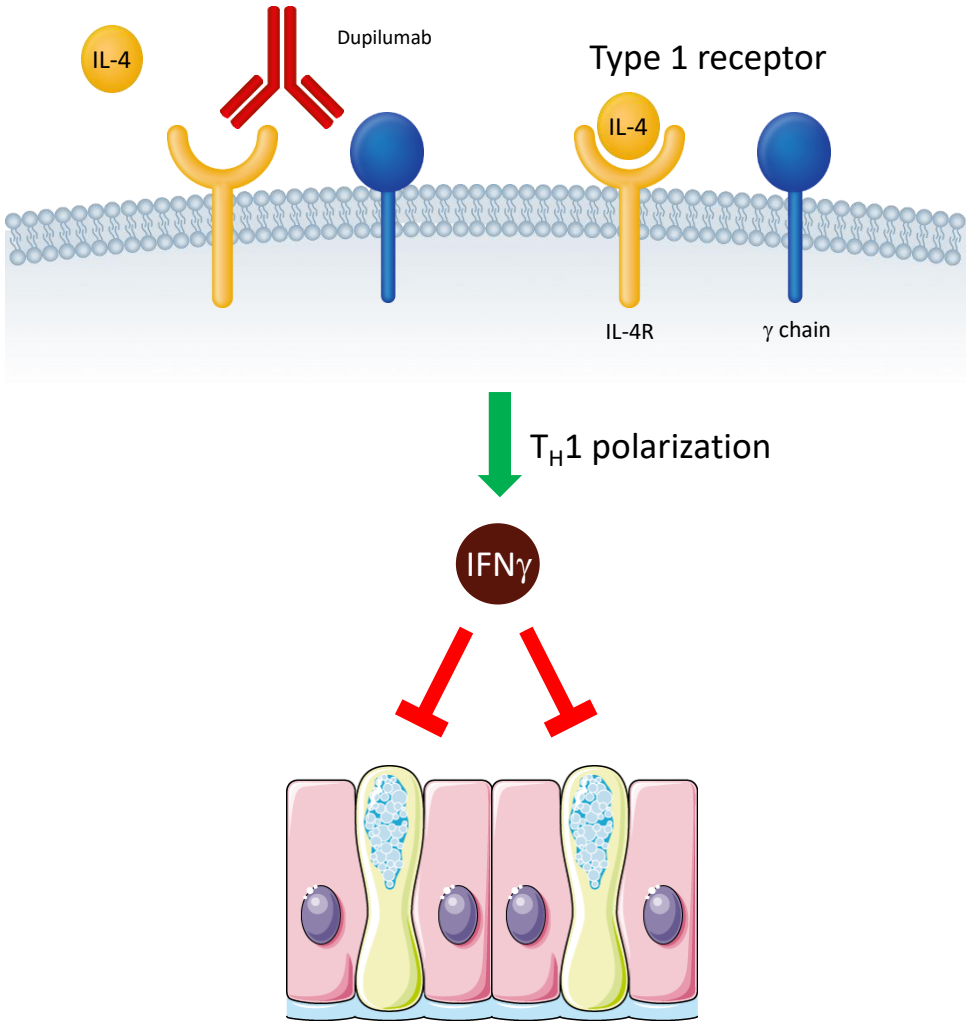
Lebrikizumab

Partial blockade of Type 2 receptor signalling

Dupilumab

Broad blockade of Type 1 and Type 2 receptors

Dupilumab-associated conjunctivitis may be driven by inhibition of the Type 1 receptor, which *eblasakimab* does not bind



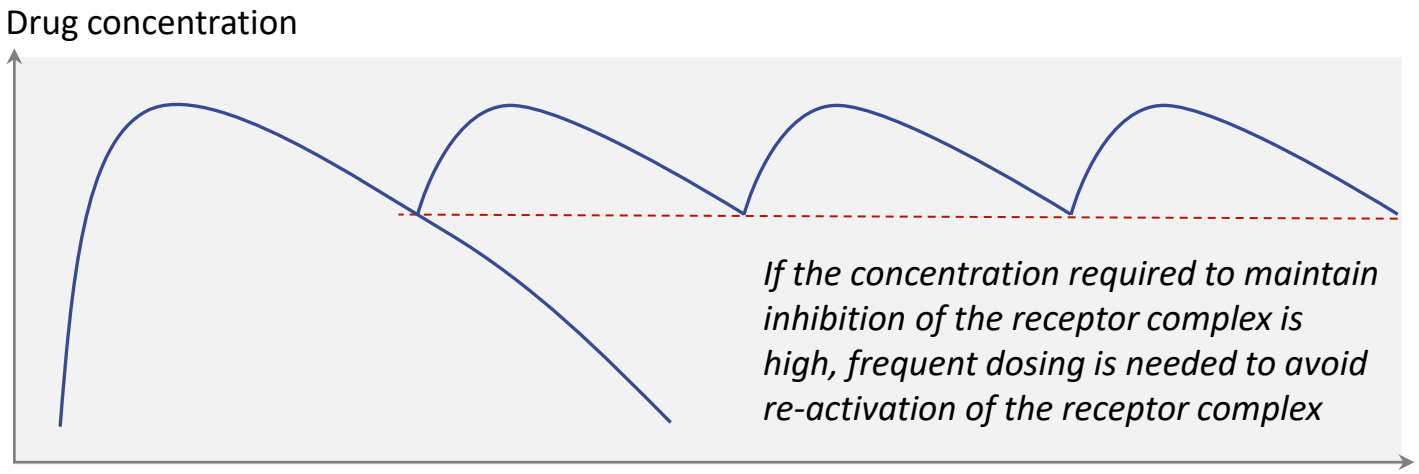
Guttman-Yassky et al (2020) JAMA Derm 156:411

- *Dupilumab* blocks the Type 1 receptor
- This may drive T_H2 to T_H1 polarization
- T_H1 cells product interferon gamma, which can lead to apoptosis of goblet cells
- This could lower the production of mucin and lead to development of dry eye and conjunctivitis

Eblasakimab binds more strongly to receptor than *dupilumab* relative to its respective ligand

Affinity of ligand and antibody to their respective target receptors:

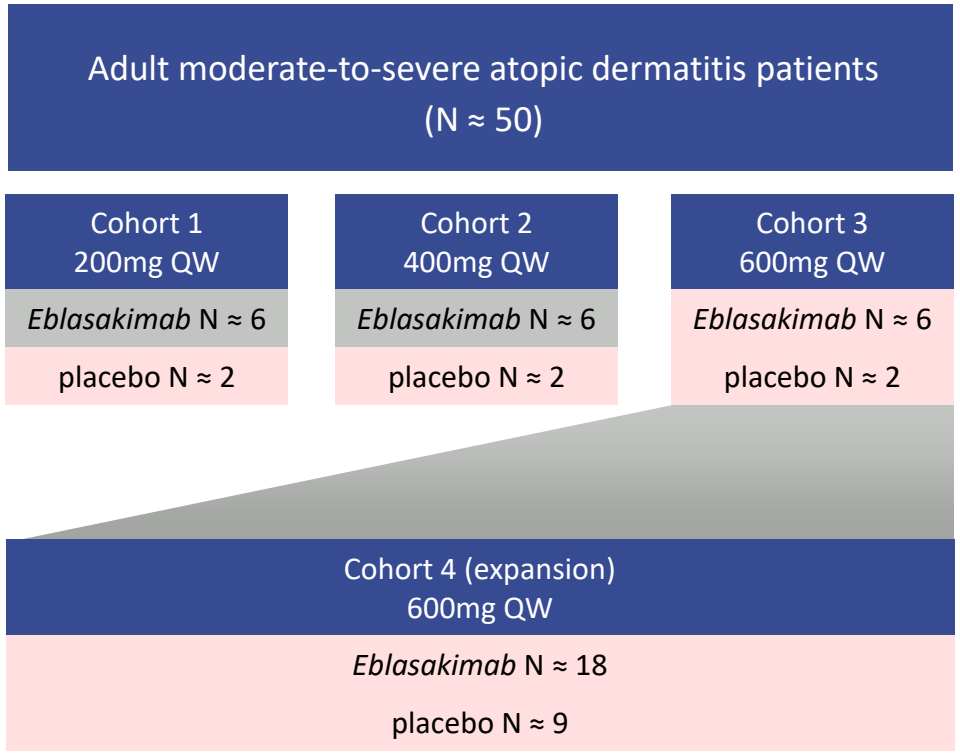
Receptor	Ligand	K_D (nM)	Comments
IL-13Rα1	IL-13	30[1]	*Eblasakimab* has a **60-fold** higher affinity for receptor versus IL-13
IL-13Rα1	*Eblasakimab*	0.5	
IL-4Rα	IL-4	0.1[1]	*Dupilumab* only has a **3-fold** higher affinity for receptor versus IL-4
IL-4Rα	*Dupilumab*	0.03	



Eblasakimab offers a greater affinity difference between ligand and receptor binding which may translate to lower required concentration *in vivo* and may provide improved dosing frequency and efficacy

1 Kraich et al (2006) BMC Biology 4:13

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



Study has 80% power to detect 39% improvement in EASI from baseline, compared to placebo, based on a one-sided 5% significance level

Pink box denotes patients included in the analysis in this presentation

- 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
- Cohort 4 (expansion) recruited additional patients dosed with 600mg QW
- Subsequent analysis compares patients in cohorts 3 and 4 dosed with 600mg QW against all placebos

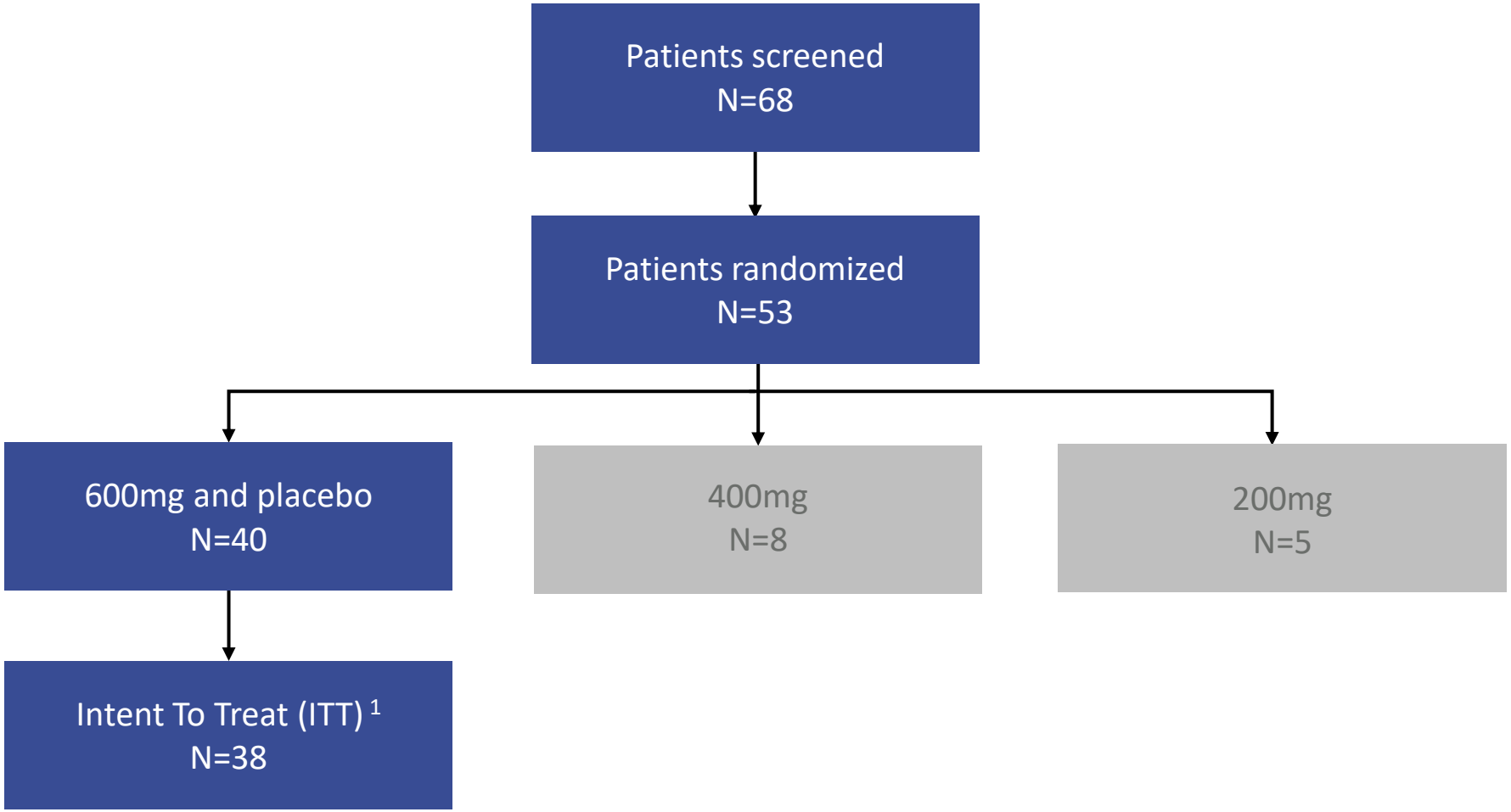
Primary endpoints are safety and tolerability

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

Patients recruited from 10 sites in US, Australia and Singapore



1 ITT represents all patients dosed excluding one patient discontinued from study prematurely due to COVID-19 restrictions and one patient randomized but not dosed

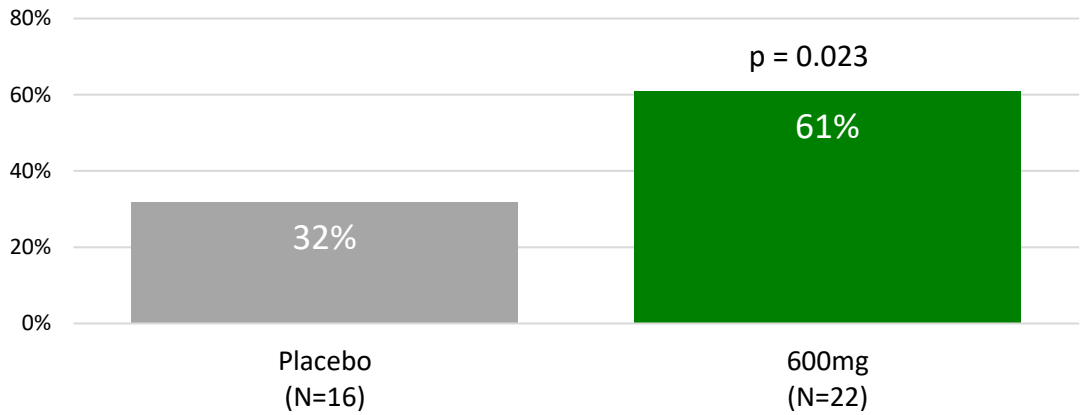
Selected baseline patient characteristics

	ITT	
	600mg (N=22)	Placebo (N=16)
Age (years)	40.2	38.8
Mean EASI score	27.6	29.0
Mean BMI	25.5	26.7
Patients with IGA 3 / IGA 4	68% / 32%	63% / 38%
Mean BSA	41.0%	46.1%
Mean peak pruritus NRS score	7.9[1]	7.9

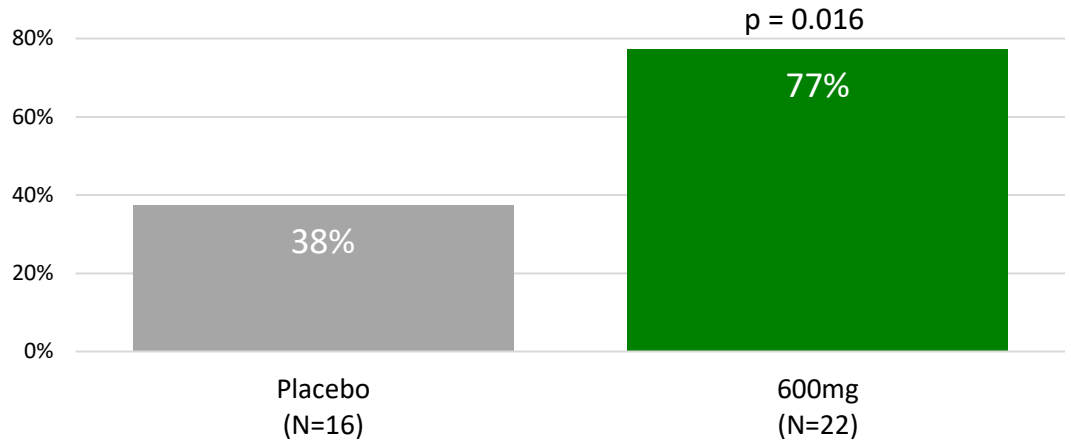
1 N=19 as 3 patients did not have a baseline value

Key efficacy endpoints

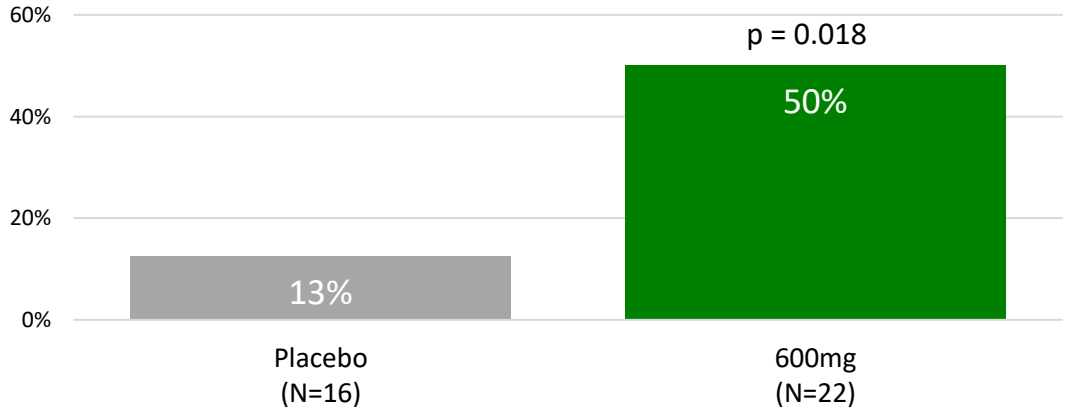
Mean reduction in EASI from baseline



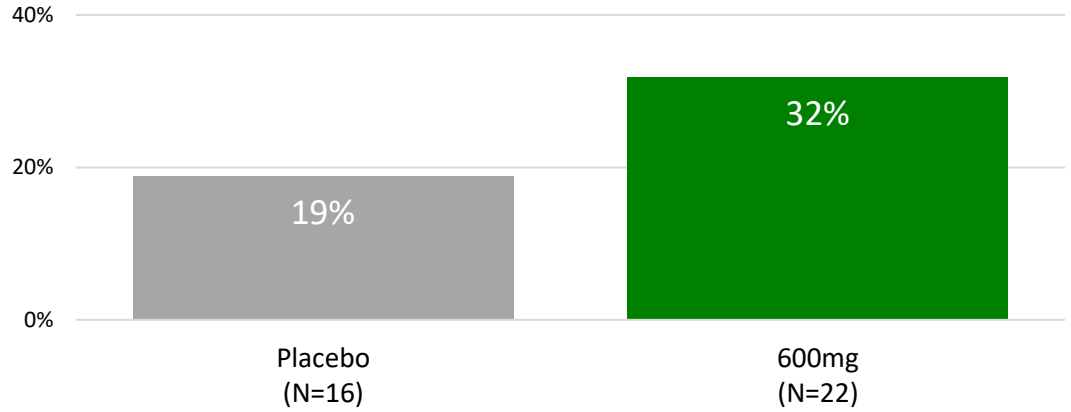
EASI-50



EASI-75



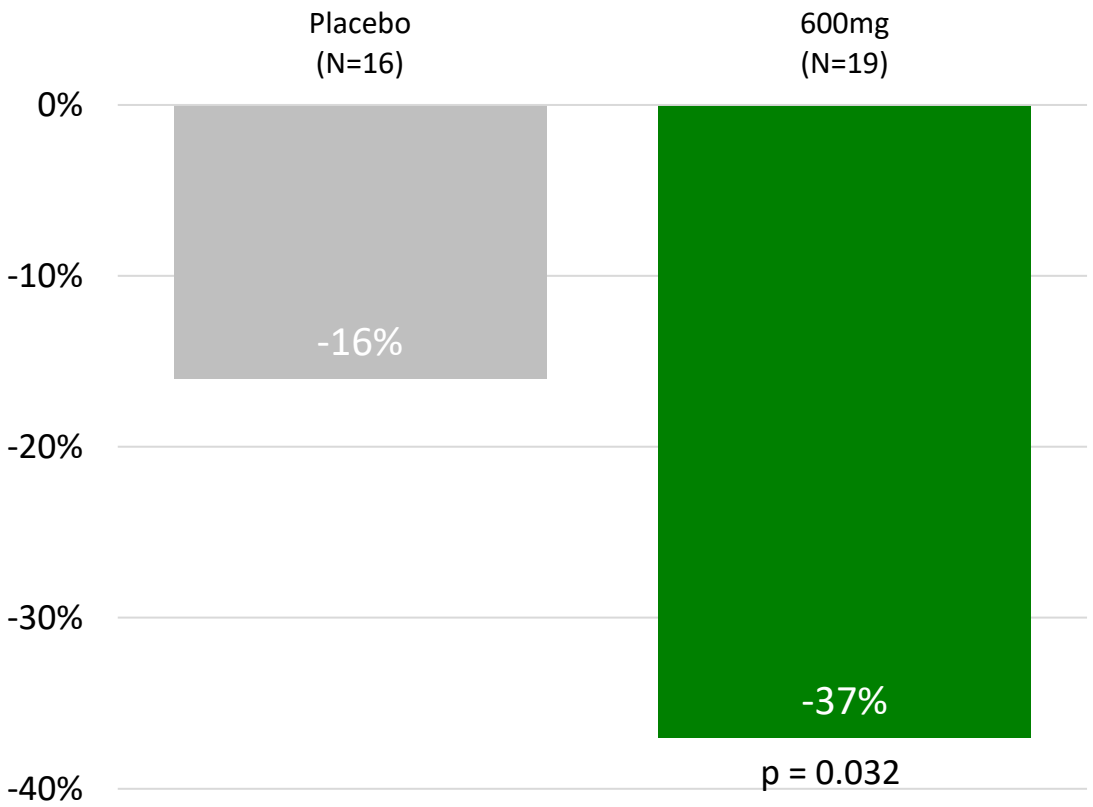
Patients achieving IGA 0/1



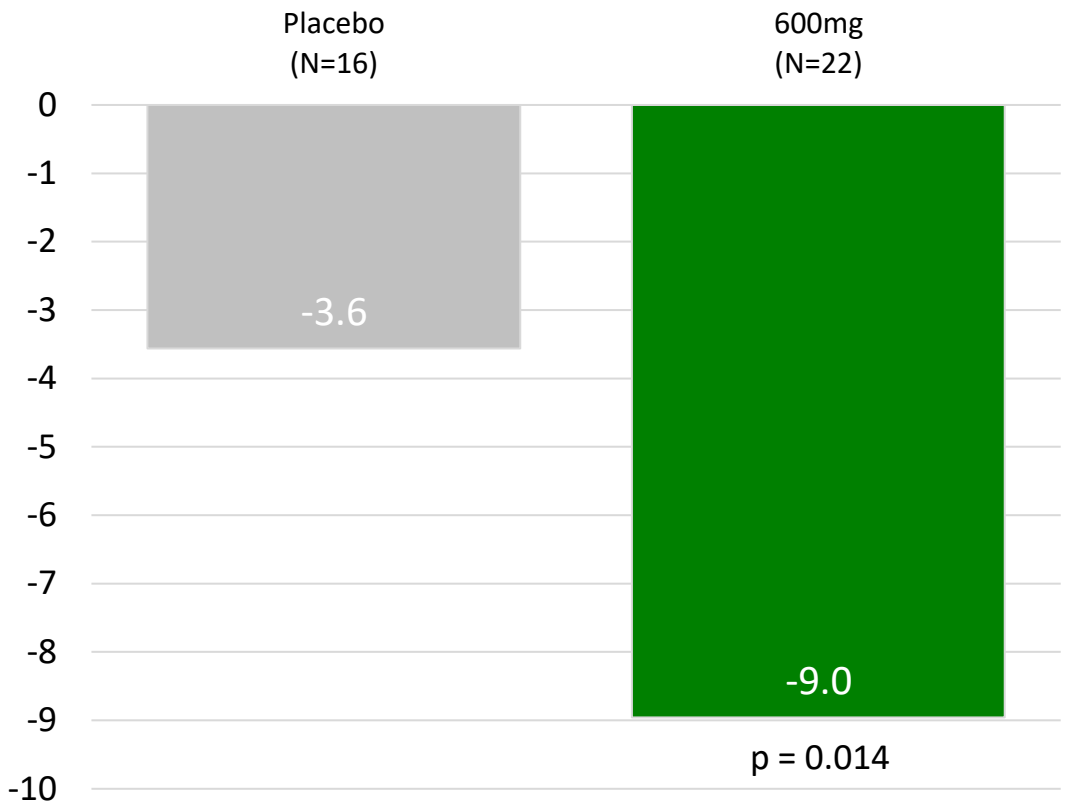
Data from ITT population
p-values are one-sided

Patient reported endpoints

Mean change in peak P-NRS from baseline



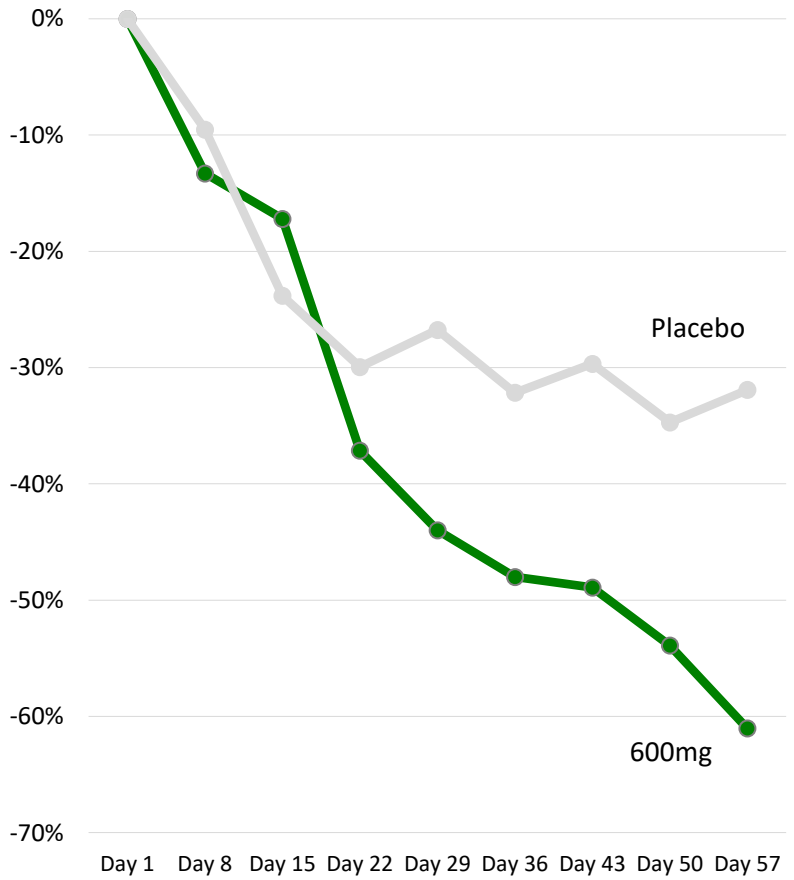
Mean change in POEM from baseline



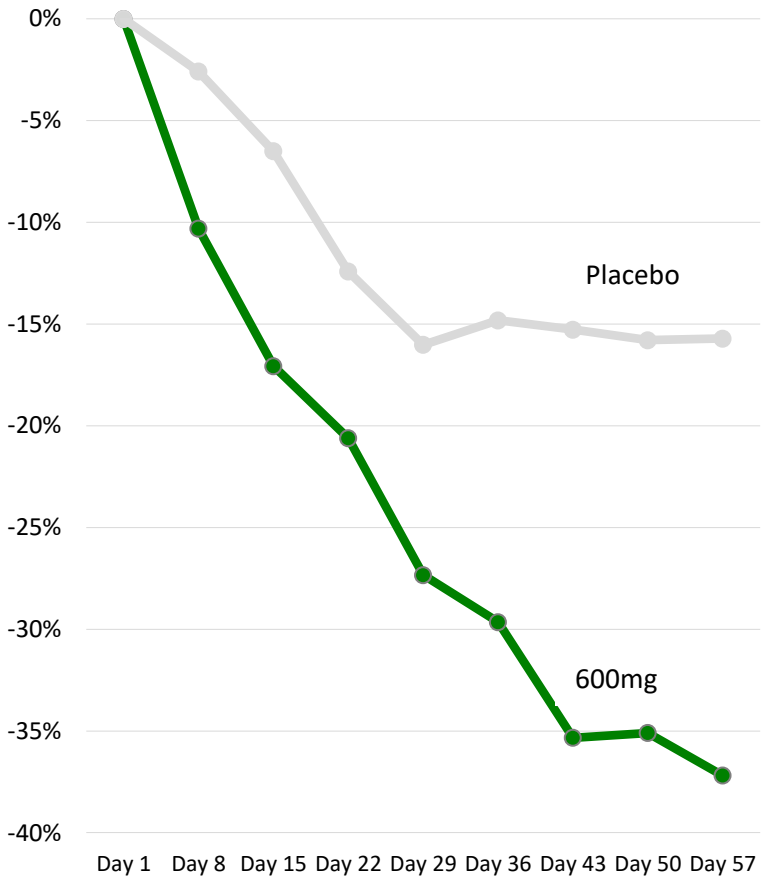
Data from ITT population
p-values are one-sided

Time course (mean change from baseline)

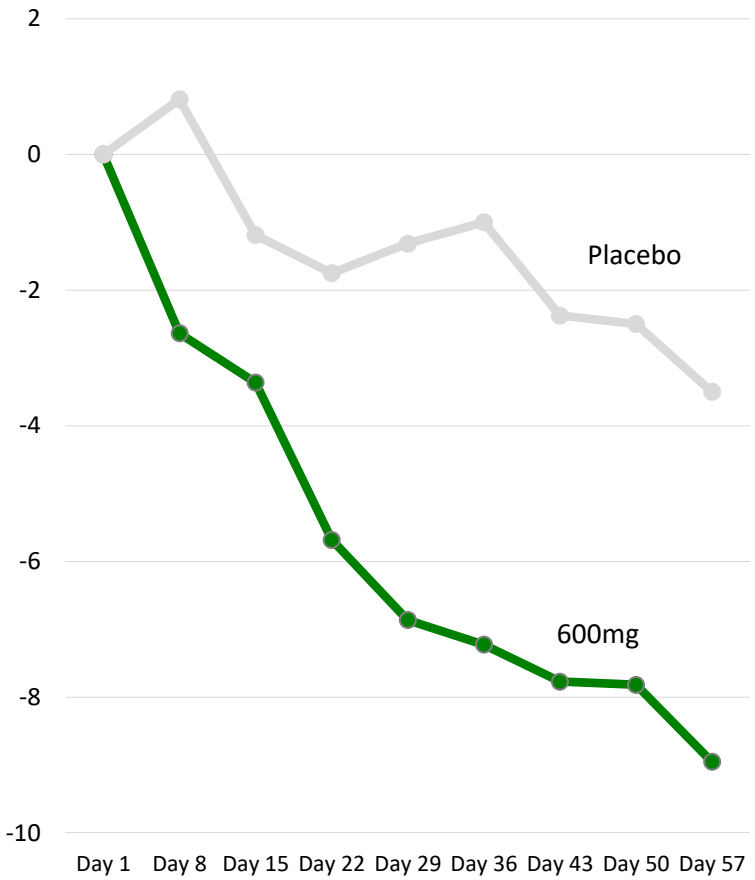
EASI



Peak P-NRS



POEM



Data from ITT population

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 Safety data cutoff as of September 1, 2021, at which time all patients had completed at least 4 weeks of safety monitoring period.
2 Drug-related defined as definitely related, probably related or possibly related

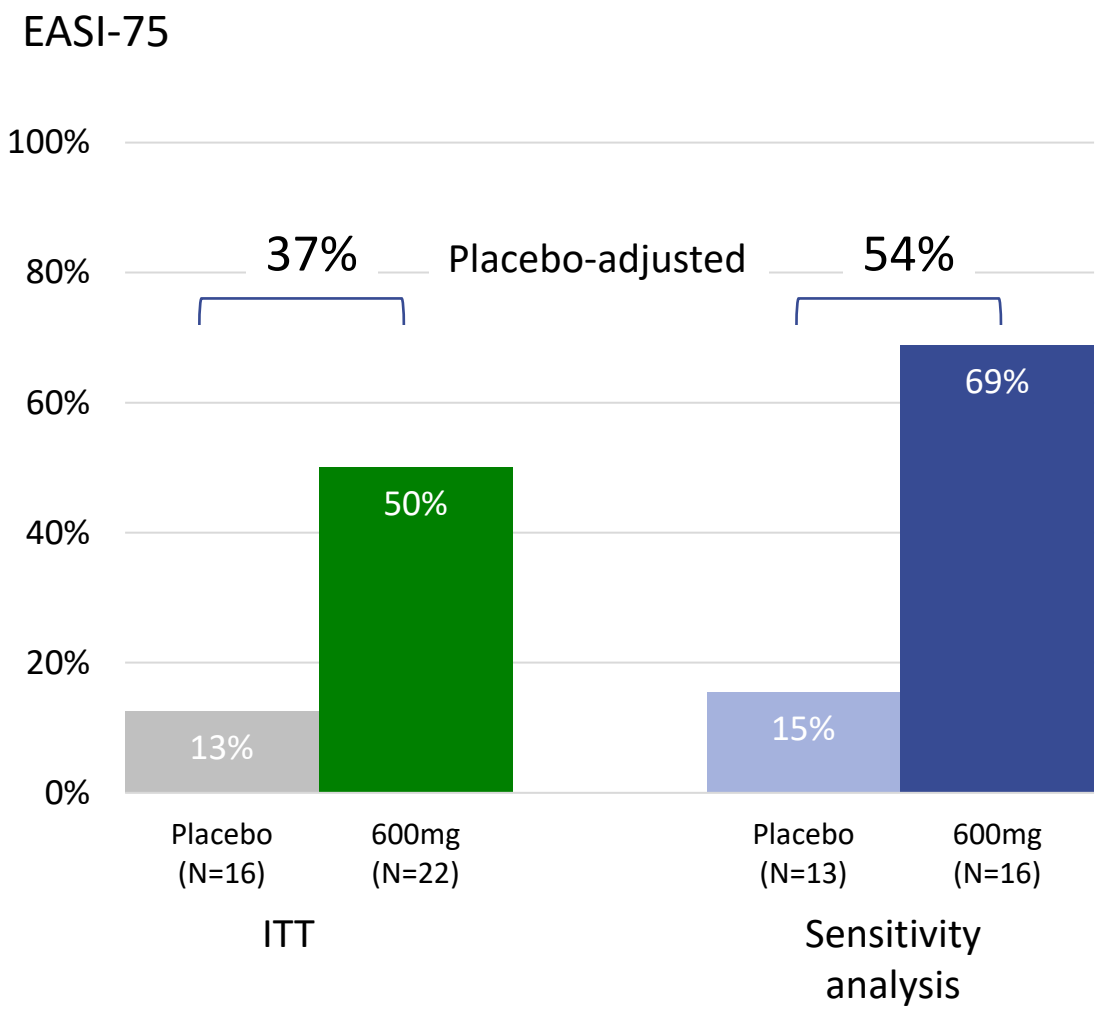
Sensitivity analysis was performed to analyze patient population more representative of mod/sev AD patients seen in other studies

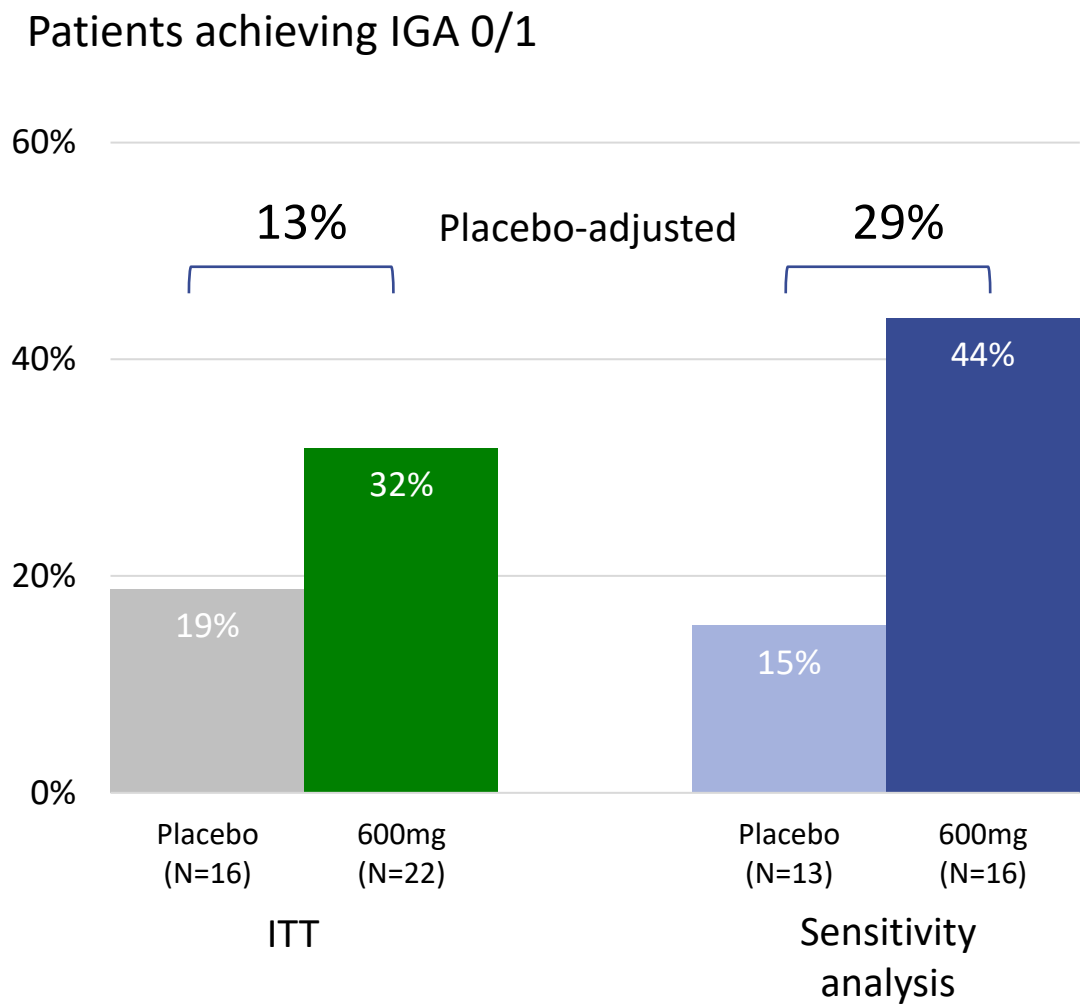
- Sensitivity analysis was pre-specified and defined prior to unblinding
- All 9 patients at one clinical site appeared atypical of moderate-to-severe AD patient population and were excluded in sensitivity analysis
  - In a heterogeneous population, some patients may exhibit one or more of these elements, however unusual to see 9 out of 9 patients exhibit all elements at the same time

Low TARC	Average baseline TARC level at this site was 0.5 ng/ml (compared to average at other sites of 4.7 ng/ml and published range from *dupilumab* studies[1,2] of 4.8 to 6.2 ng/ml). No patients at this site had TARC levels over 1.1 ng/ml
Low eosinophils	Baseline eosinophil levels were an order of magnitude lower than other sites and other comparable AD studies
Low IgE	Mean baseline IgE levels were over an order of magnitude lower than other sites and *dupilumab* studies[1,2]
Few allergic co-morbidities	Almost all patients (89%) had no allergic co-morbidities (compared to 13% at other sites)

1 Beck et al (2014) N Engl J Med 2014;371:130-9
2 Hamilton et al (2019), 49th Annual ESDR Meeting Sep 18-21, 2019

Sensitivity analysis suggests in a more typical moderate-to-severe AD patient population, efficacy may be further improved





Comparison of Proof of Concept studies in AD

Drug	Study	Target	Patients	Efficacy assessment at	Reached statistical significance?		
					ΔEASI score (%)	EASI-75	IGA 0/1
Eblasakimab	Phase 1b (ITT)	IL-13R	38	8 weeks	✓	✓	
Dupilumab	Phase 1b (M4A+ M4B)[1]	IL-4R	67	4 weeks	✓		
	Phase 2a (M12)[1]	IL-4R	109	4 weeks			
				12 weeks	✓		✓
CBP201	Phase 1b[2]	IL-4R	31	4 weeks			
KHK4083	Phase 1[3]	OX-40	20	6 weeks			

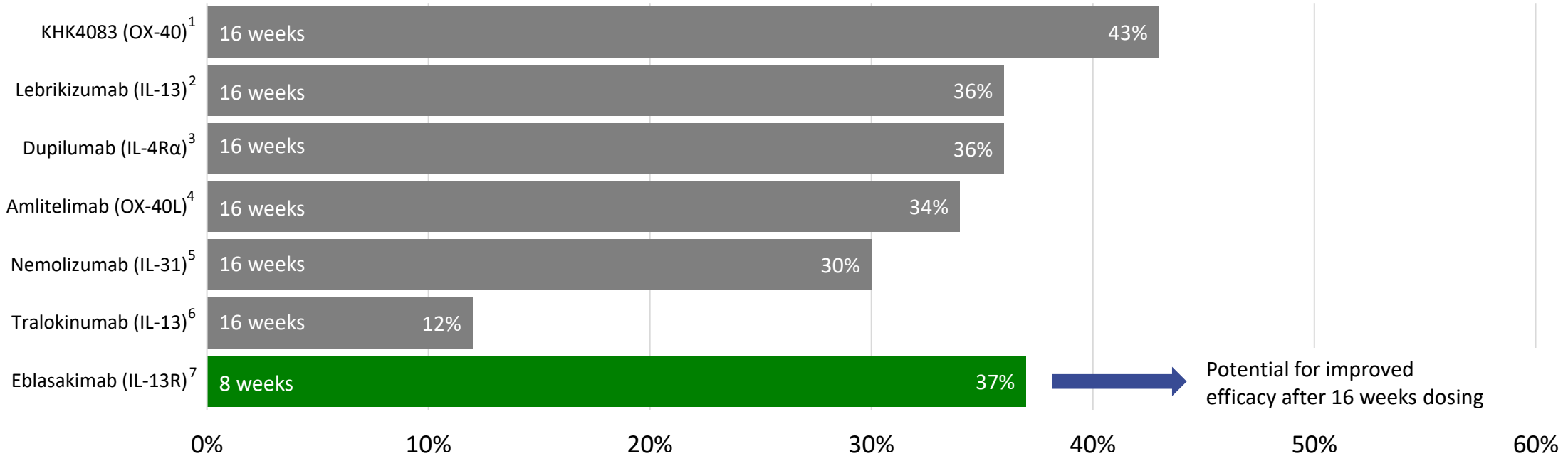
Data from Phase 1 studies of *lebrikizumab* and *tralokinumab* were not published

✓ represents two-sided p-value <0.05

1 Beck et al (2014) NEJM 371(2):130-139
2 Wang et al (2020), 29th EADV Congress, Oct 28- Nov 1, 2020, p-value not disclosed
3 Nakagawa et al (2020) J Derm Sci 99:82-89, p-value not applicable (single-arm study)

The evolving landscape in AD

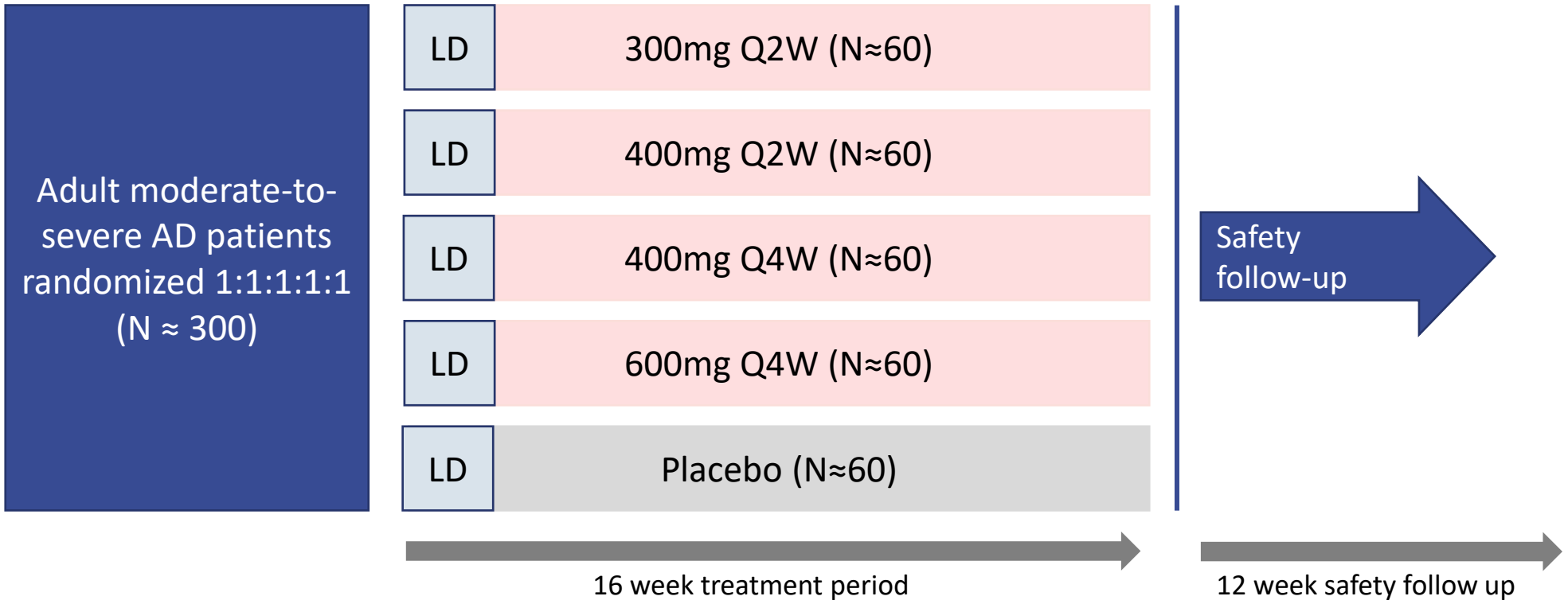
Efficacy of selected drugs in atopic dermatitis (placebo-adjusted EASI-75) at 16 weeks:



For illustrative purposes only. Not a head-to-head comparison. Caution should be exercised when comparing data across trials of different products and product candidates. Differences exist between trial designs and patient populations and characteristics. The results across such trials may not have interpretative value on our existing or future results.

1 Phase 2: Guttman-Yassky et al (2021), 30th EADV Congress, Sep 29- Oct 2, 2021
2 Phase 2b: Guttman et al (2020) JAMA Derm 156(4):411-420
3 Phase 3: Simpson et al (2016) NEJM 375(24):2335
4 Phase 2a: Weidinger et al (2021), 30th EADV Congress, Sep 29- Oct 2, 2021
5 Phase 2b: Silverberg et al (2020) JACI 145:173-182
6 Phase 3: Wollenberg et al (2021) Br J Derm 184(3):437-449
7 Phase 1b: ITT population

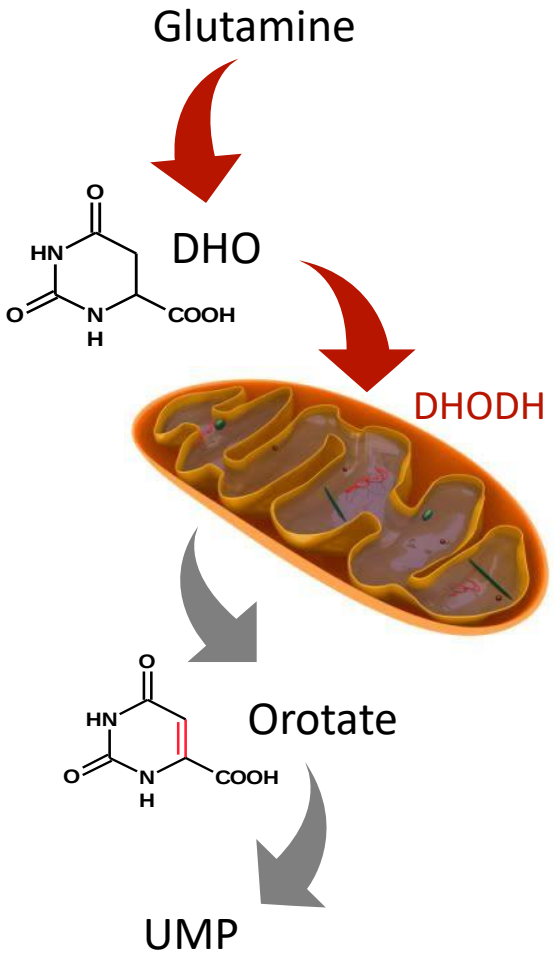
Phase 2b (TREK-AD): initiated Jan 2022, topline expected 1H 2023



- 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

Farudodstat (ASLAN003)

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

DHODH is an attractive target for IBD

Leflunomide

- 2nd line treatment in Crohns' disease (CD) patients refractory or intolerant to *azathioprine*[1]
  - In 24 patients steroid free remission was 42% by week 16
  - Crohn's Disease Activity Index (CDAI) decreased from 219 to 87
  - Steroid intake decreased from 25 to 3 mg/day
  - Adverse side effects were frequent
- CD patients intolerant to *azathioprine*/6-MP[2]
  - *Leflunomide* well tolerated with significant reduction in clinical score in 8/12 patients

Vidofludimus

- Phase 2a ENTRANCE study in steroid-dependent CD and ulcerative colitis (UC)[3]
  - After 12 weeks of treatment, 8 out of 14 (57%) patients with CD and 6 out of 12 (50%) patients with UC were in steroid-free remission (complete responders)

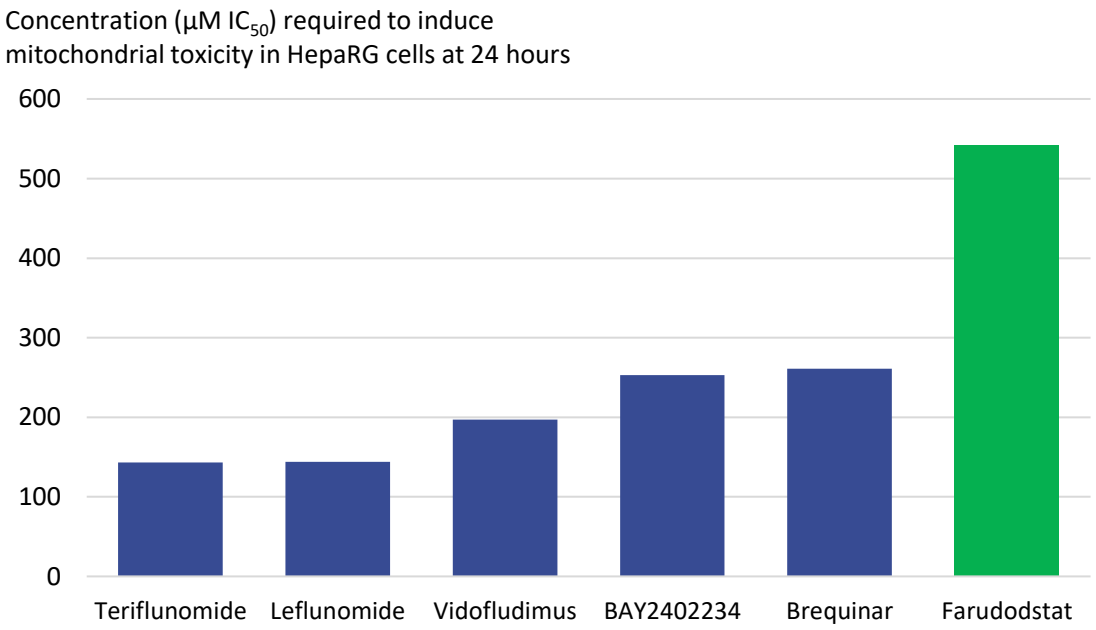
1 Holtmann et al (2008) Dig Dis Sci 53:1025-1032
2 Prajapati et al (2003) J Clin Gastroenterol 37(2):125-128
3 Herrlinger et al (2013) J Crohns Colitis 7:636-643

Farudodstat has the potential to be best-in-class for autoimmune disease

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

Assay used to measure IC_{50}	*Farudodstat* (µM)	*Teriflunomide* (µM)
Enzymatic DHODH inhibition	0.035	1.1
Human PBMC proliferation inhibition	1.4	46
IFN_{γ} inhibition in human whole blood	2.5	259

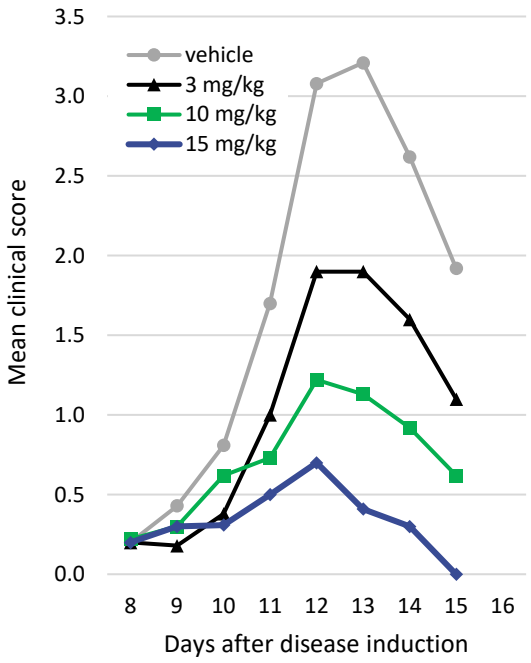
- *In vitro* studies demonstrated *farudodstat* has lowest potential for hepatotoxicity out of 6 approved and clinical stage DHODH inhibitors



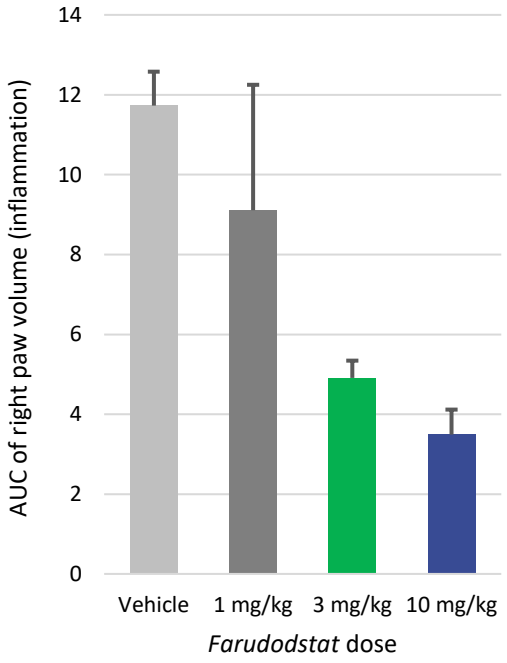
Jones et al (2021) Toxicology in Vitro 72:105096

Farudodstat has the potential to be best-in-class for autoimmune disease

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

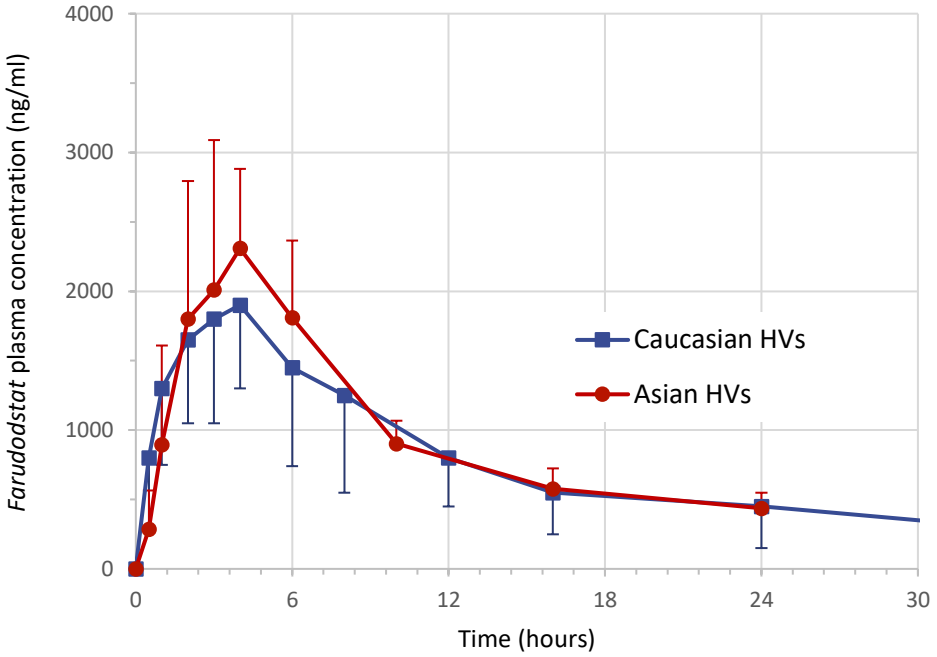


MS model in rat (EAE)



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

Summary of *farudodstat*

Farudodstat has the potential to be best-in-class for autoimmune disease

- Stronger *in vitro* potency as compared with other DHODH inhibitors
- Selective against a panel of 195 enzymes, ion channels and receptor binding assays
- *In vitro* studies demonstrated *farudodstat* has lowest potential for hepatotoxicity out of 6 approved and clinical stage DHODH inhibitors
- Well-tolerated in 119 subjects in Phase 1 and Phase 2 clinical trials
- PK profile suitable for once-daily dosing

Next steps

- Expecting to initiate phase 2 in IBD in 1H 2022
- Planning future studies in autoimmune skin diseases

Financials

Financials

Ticker	NASDAQ: ASLN	
Shares outstanding[1]	69.6M[1]	(as of December 31, 2021)
Net operating cash used	US$ 11.9M	(4Q 2021)
Cash balance	US$ 90.2M	(as of December 31, 2021)
	Runway through to late 2023	

1 American Depositary Shares