Company presentation

May 2024

NASDAQ: ASLN



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Targeting major inflammatory disease markets with significant unmet need

Eblasakimab

Potential first-in-class antibody that targets the IL-13 receptor with potential to become a **leading therapy** in treating atopic dermatitis (AD) and other indications eg COPD

- AD expected to be a \$24B market by 2029 1, only 2 approved biologics in US to date
- Positive phase 2b study of eblasakimab in AD in July 2023, phase 3 preparation underway
- Positive phase 2 interim data in *dupilumab*-experienced patients: unprecedented efficacy data compared to other biologics after just 16 weeks 60.0% achieved EASI-90 and 66.7% vIGA score of 0 or 1
 - Initial positioning as therapy of choice for patients with inadequate response or intolerance to dupilumab
- Translational data indicates the potential for improved efficacy over dupilumab in COPD

Farudodstat

Novel DHODH inhibitor with the potential to be first-in-class for alopecia areata (AA)

Phase 2 proof-of-concept study in AA initiated, interim topline readout expected Q3 2024

Financials

\$18.4M cash as of March 31, 2024

Net operating cash used in Q1 2024: \$7.4M

S. C.

Multiple catalysts in upcoming 12 months

Program	Target	Indication	Preclinical	Phase 1	Phase 2	Phase 3	Anticipated milestones in 2024
Eblasakimab IL-13Rα1		Atopic	Biologic na	ïve		 Selection of partner to advance eblasakimab into Phase 3 	
	dermatitis	Dupilumab	experienced		 Topline readout from dupilumab- experienced trial end 2024 		
		COPD					
Farudodstat	DHODH	Alopecia areata					Phase 2a interim topline data Q3 2024

Management and advisory team with global development experience in dermatology

Management team













Board of Directors

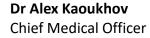


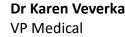


Dr Carl Firth CEO **Board Director**



Boehringer Ingelheim









Andrew Howden Dr Neil Graham **Board Director**

Robert Hoffman Board Director

Dr Kathleen Metters Board Director



Bank of America

Merrill Lynch





Allergan





























Eblasakimab

Targeting atopic dermatitis, a highly prevalent disease with only 2 approved biologics

AD is a devastating disease that causes massive suffering for both patient and family











- Chronic, incessant itch
- Long term changes in skin barrier
- Sleep deprivation
- Severe impact on quality of life
- Allergic comorbidities
- Rising prevalence

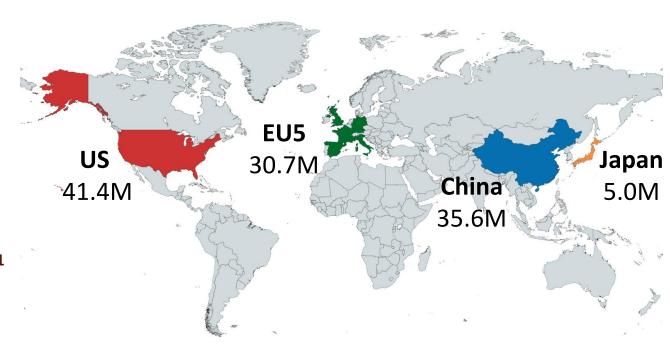
AD is a global disease, and affects up to 13% of children and 7% of adults in developed countries ¹

200M

AD patients worldwide ²

30%

with moderate-to-severe disease 1



Total AD prevalent cases, 2019 3,4

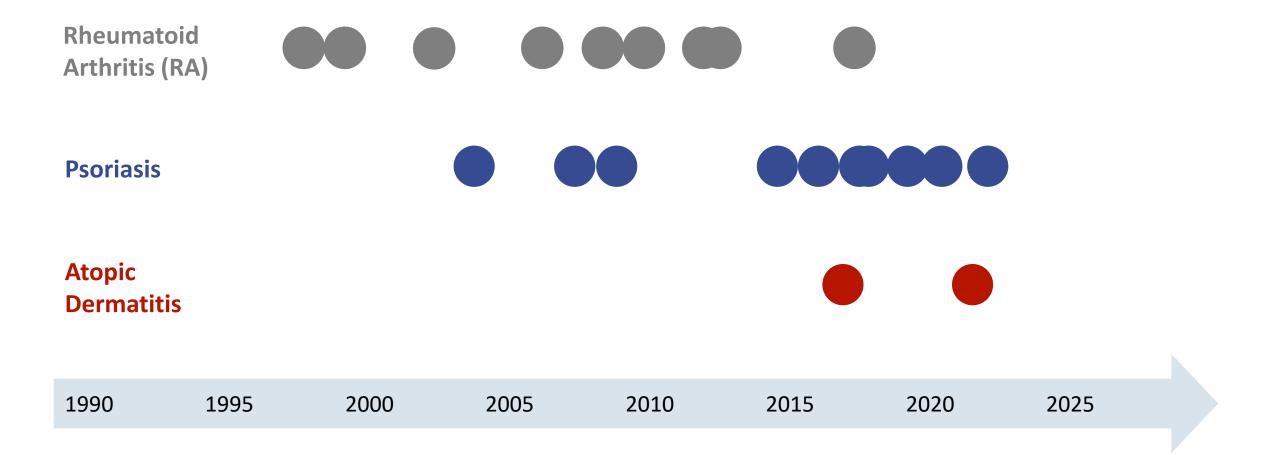
Silverberg (2017) Dermatol Clin 35: 283–289

Weidinger et al (2018) Nat Rev Dis Prim 4:1

³ Divekar et al (2021) Atopic Dermatitis/Atopic Eczema: Disease Landscape & Forecast. Decision Resources Group (DRG)

⁴ Wen-lan Dong et al (2021) WAO Journal 14(11):100604.

Only 2 biologics have been launched for AD, yet there are double the number of patients compared to psoriasis





There are few safe and effective treatments for moderate-to-severe AD

Topical agents

TCS, TCI, topical PDE4/JAK

 Treatment has been traditionally focused on topical corticosteroids but steroid use is associated with long term safety risks



Biologics

dupilumab, lebrikizumab

• *Dupilumab* was launched in 2017 as the first biologics and has established biologics as the cornerstone for AD treatment



JAKi systemic immunosuppressants abrocitinib, upadacitinib

- JAK inhibitors (JAKi) received recent approval in AD
- Whilst effective, they carry black box warnings for higher risk of: cardiovascular death, stroke, serious infections (including tuberculosis) and cancer

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

Topical agents

TCS, TCI, topical PDE4/JAK



Biologics

dupilumab, lebrikizumab



JAKi systemic immunosuppressants

abrocitinib, upadacitinib

- Launch of dupilumab in 2017 established new standard of care
 - 2023 sales of \$12B, dominated by AD ¹
 - Following phase 3 wins, analysts including COPD in forecasts, suggesting total \$20B peak sales ²
- However, market still nascent only 9% of eligible patients receive dupilumab today 1
- Patients looking for improved treatment options majority of dupilumab patients would switch to a biologic with an incrementally improved profile ³
 - Opportunity to improve upon biweekly dosing regimen
 - Only 30-40% of patients treated with dupilumab achieved an optimal response ^{4,5}
 - Conjunctivitis is common and can lead to treatment discontinuations
- Lebrikizumab approval in US delayed due to CRL from FDA, lack of meaningful differentiation and inability to address allergic comorbidities will likely position it behind dupilumab

¹ Sanofi's quarterly financials, annual reports and investor presentations

² FiercePharma article "Sanofi, Regeneron's Dupixent could hit \$20B in peak sales with COPD expansion: analyst" published 24 March 2023

³ Market research conducted by ASLAN from May-Aug 2023 with 83 AD patients in US (27% patients severe, 69% moderate, 5% mild), 32 patients current Dupixent users. 56% of current Dupixent users willing to switch

⁴ Spherix (2018) Atopic dermatitis ATU study

⁵ IGA 0/1 response rate at week 16, Simpson et al (2016) NEJM 375(24):2334-2348

Recent years have seen many disappointments with new mechanisms

Earlier	2018	2019	2020	2021	2022	2023	2024
JEE	1)_5	IL-22	IL 17	TSKP	IL-1α	OX <mark>?</mark> 40	Siglec-8
CRX(H2	2 NK1R	IL-17C	IL-33		IL-36	IL- 3 1	
NK 1R	CD40	IL- 3 3	H4-R				

Besides several unproven mechanisms, dupilumab and lebrikizumab remain the only significant competitors in late phase development

Eblasakimab Positioning in the rapidly evolving AD market

We believe *eblasakimab* can be initially positioned as the therapy of choice for patients with inadequate response or intolerance to *dupilumab*

Initially targeting \$10B second line market ¹

Eblasakimab is the first antibody to target the IL-13 receptor with potential to become a **leading** therapy in treating atopic dermatitis (AD) and other allergic disease

- Potential to be leading second line biologic therapy for patients with inadequate response to dupilumab
- Second line market is substantial with potential to be \$10B by 2029²
- Prescriber experience in second line could enhance use in first line treatment

Translational data supports positioning

Eblasakimab has a unique mechanism of action compared to dupilumab

- Translational data in AD skin biopsies demonstrates *eblasakimab* is more effective at downregulating inflammatory markers than *dupilumab*
- Eblasakimab's MoA has potential to be effective in dupilumab refractory patients

Only placebocontrolled trial in 2nd line

TREK-DX - Phase 2 study of *eblasakimab* in *dupilumab* experienced patients is currently ongoing First and only double-blind randomized study for patients with inadequate response to *dupilumab*

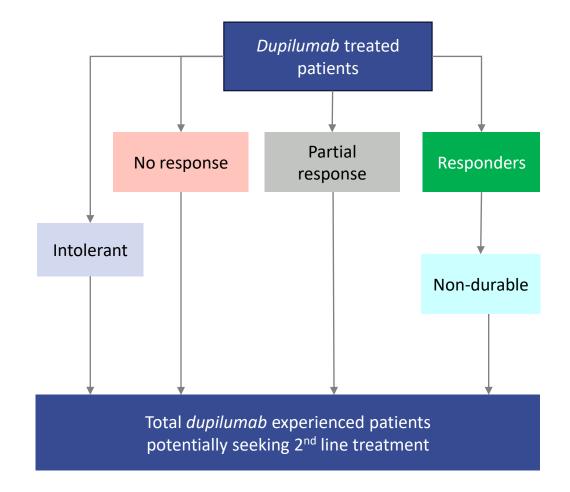
2 Decision Resources Group, December 2022

¹ Second line market here refers to a second systemic therapy following inadequate response to dupilumab

Patients in need of second line biologics ¹ treatment lack safe long-term options

- Dupilumab has established standard-of-care for AD patients
- Around 270,000 AD patients are being treated with dupilumab ²
- However, 63% of *dupilumab*-treated patients do not achieve IGA 0/1 ³ within 16 weeks and of those that do, only 54% maintain the response at week 52 4
- Patients can discontinue due to intolerance and real-world data shows conjunctivitis prevalent in over 26% of dupilumab users ⁵
- Many of these patients may respond but may not be satisfied with their response and will seek alternative treatments
- In market research survey, 56% of current dupilumab users and 56% of lapsed dupilumab users are willing or very willing to switch to a treatment with *eblasakimab*'s target profile ⁵

Based on market research 5, we believe around 150,000 patients who are currently using or have used dupilumab could switch to an alternative biologic treatment



- Second line market here refers to a second systemic therapy following inadequate response to dupilumab
- Sanofi investor presentations (Dec 2023), based on prevalence numbers of uncontrolled moderate-to-severe AD patients in US, EU and JP markets, and 9% penetration rate of Dupixent

from 1-7, where 1= very unwilling and 7= very willing, their willingness to switch from current treatment to a treatment with eblasakimab's target profile, % of patients selecting rating of 6 or 7 shown above

- Thaci et al (2019) J Dermatol Sci 94(2):266-275
- Worm et al (2020) JAMA Derm 156(2):131-143
- Halling et al (2021) JAAD 84(1):139-147 Market research conducted by ASLAN from May-August 2023 with 83 AD patients in the US (27% patients severe, 69% moderate, 5% mild) in different treatment cohorts. Patients were asked to rate on a scale



What do patients with inadequate response to dupilumab look for?



Safe for long-term use



Effective in patients with inadequate response to dupilumab



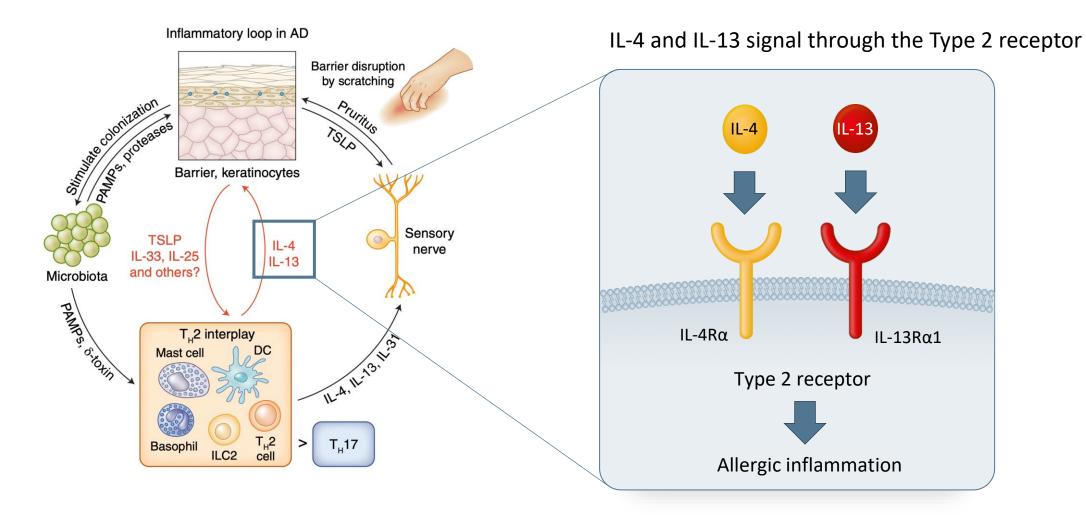
Potential to treat comorbidities



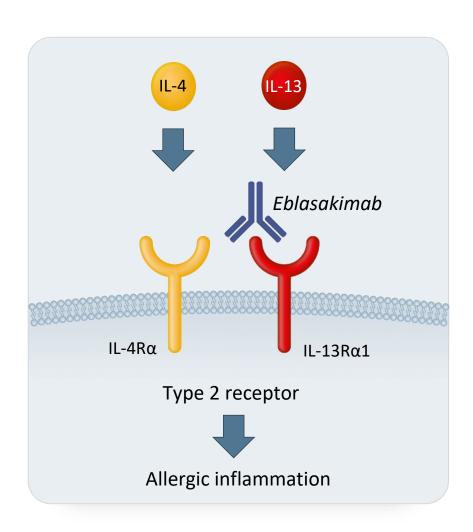
Rapid speed of onset

Eblasakimab A novel mechanism for treatment of AD

IL-4 and IL-13 are the central drivers of the itch-scratch cycle in AD



Eblasakimab is the only monoclonal antibody in the clinic targeting the IL-13 receptor¹



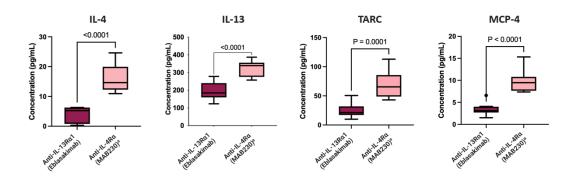
IL-4 and IL-13 are central to triggering allergy and symptoms of AD

By targeting the IL-13 receptor, *eblasakimab*'s novel approach efficiently blocks the Type 2 receptor, preventing signaling through **both** IL-4 and IL-13, while sparing the Type 1 receptor

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

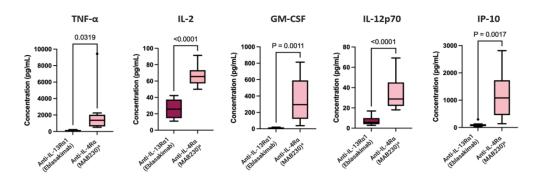
Th2 cytokines

IL-13R blockade resulted in lower levels of key cytokines implicated in Th2-driven (allergic) inflammation compared to IL-4R blockade



Th1 cytokines

Levels of pro-inflammatory Th1 cytokines were lower with IL-13R blockade compared to IL-4R blockade

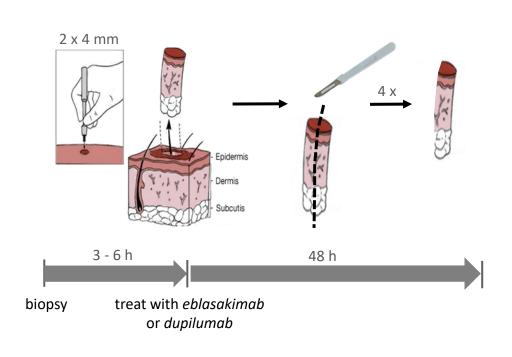


Selective blockade of IL-13R offers a potentially differentiated approach:

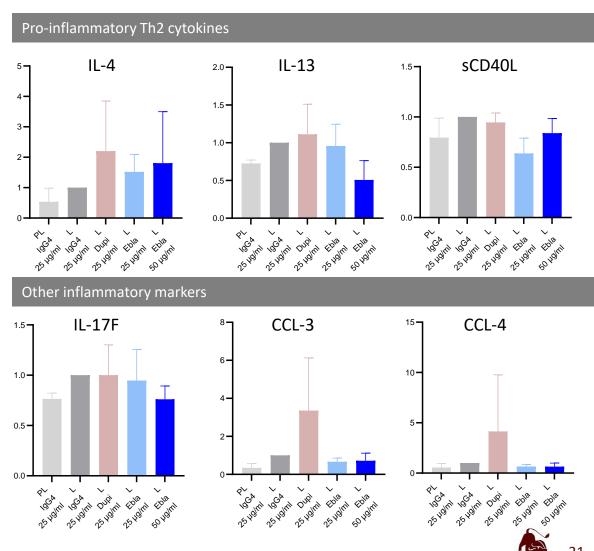
- More efficient reduction of Th2 inflammation
- No increase in Th1 cytokines, compared to IL-4R blockade

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-4Rα (R&D Systems antibody) and supernatants assayed for cytokine panel using electrochemiluminescence.

Head-to-head study between *eblasakimab* and *dupilumab* in skin biopsies confirm differentiated effects of targeting IL-13R vs IL-4R



In AD lesional skin biopsies, *eblasakimab* reduced secretion of pro-inflammatory Th2 cytokines as well as other AD relevant mediators more efficiently than *dupilumab*



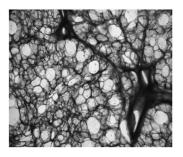
New translational work in COPD: Eblasakimab outperformed dupilumab in restoring lung function in COPD derived PCLS

COPD is a heterogeneous lung condition characterized by chronic respiratory symptoms due to abnormalities of the airways and/or alveoli which causes persistent, often progressive, airflow obstruction

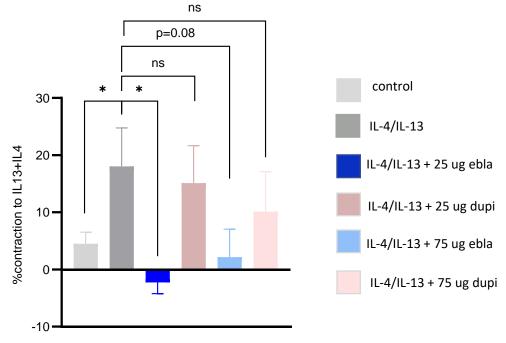
Precision Cut Lung Slices (PCLS): human ex vivo model of COPD¹



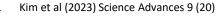




Human PCLS were treated for 48 hours with cytokines IL-4, IL-13 and/or *eblasakimab* and *dupilumab*. Airway responsiveness was also tested with increasing doses of methacholine (MCh), followed by a single dose of formoterol (induces dilation)

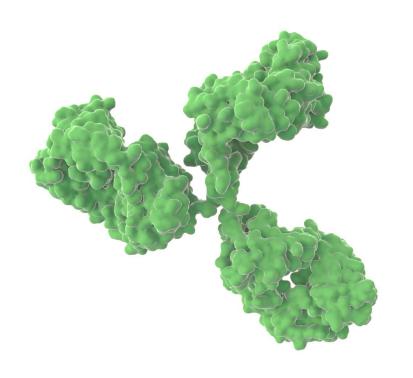


Eblasakimab showed statistically significant improvement across all measured bronchial outcomes whereas dupilumab did not achieve statistical significance relative to placebo for multiple measures, suggesting that eblasakimab has the potential to significantly improve IL-4 and IL-13-induced bronchial airway constriction





Eblasakimab's unique approach, supported by translational data, may deliver a differentiated clinical profile



Unique mechanism of action targeting IL-13R

Efficient inhibition of IL-4 and IL-13 signalling through the Type 2 receptor while sparing the Type 1 receptor

Differentiated cytokine profile compared to dupilumab

- More efficient reduction of Th2 inflammation
- No increase in Th1 cytokines
 Effective in models of AD and COPD



Potential to be effective even when there is inadequate response to *dupilumab*

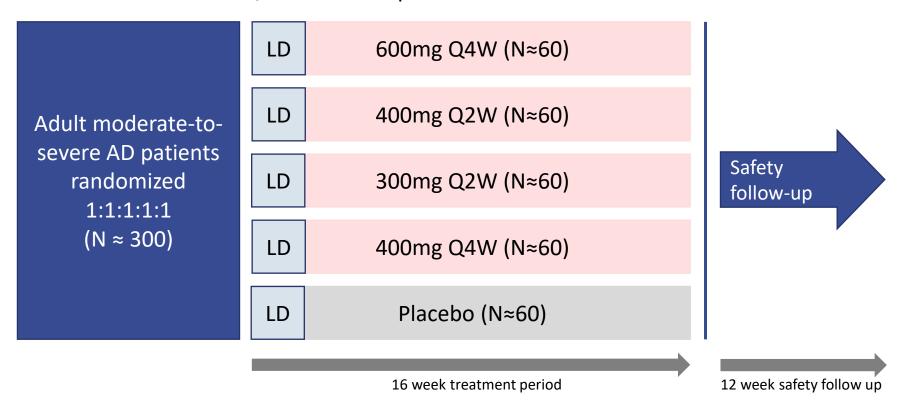
Eblasakimab Positive readout from phase 2b TREK-AD

Phase 2b TREK-AD demonstrated monthly dosing regimen without compromising on efficacy

- TREK-AD is a **global dose-ranging study** testing *eblasakimab* conducted across 8 countries with around 300 moderate-to-severe AD patients
- The study was positive and demonstrated potential for monthly dosing
 - The study met the primary endpoint and key secondary endpoints in the ITT population in the 3 key doses ¹
 - The 600mg Q4W arm was numerically the best performing arm (73% reduction in EASI score, p=0.001)
 - Eblasakimab showed a rapid onset of action in the first few weeks of treatment and was generally well tolerated with low rates of conjunctivitis and injection site reactions
- Post-hoc analyses demonstrated the possibility for further widening in the placebo-adjusted scores
 - In keeping with several other recent studies, the placebo response was higher than dupilumab studies conducted a decade ago
 - High proportion of milder patients in the US contributed to the high placebo response (over a third of patients in the US had an EASI score less than 18)
 - Eblasakimab performed equally well in more severe patients, however placebo scores greatly reduced

TREK-AD: Phase 2b in biologic naïve patients

90 sites from 8 countries, over half the patients enrolled in North America



- Loading dose of 600mg for the Q2W dose groups at week 0 and week 1
- Loading dose of 600mg for the Q4W dose groups at week 0, week 1 and week 2

Key inclusion criteria

- Chronic AD present for ≥3 years
- EASI score ≥16
- vIGA-AD score ≥3
- BSA (Body Surface Area) ≥10%

Primary endpoint

% change in EASI from baseline

Key secondary endpoints

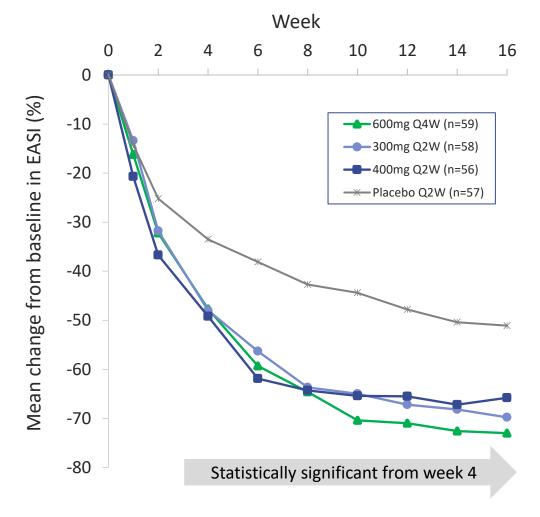
- EASI-75
- EASI-90
- vIGA-AD 0/1
- PROs
- BSA
- SCORAD



Monthly dosing with 600mg led to 73% improvement in disease after 16 weeks and was statistically significant from week 4

Eblasakimab met the primary endpoint in three dose groups*

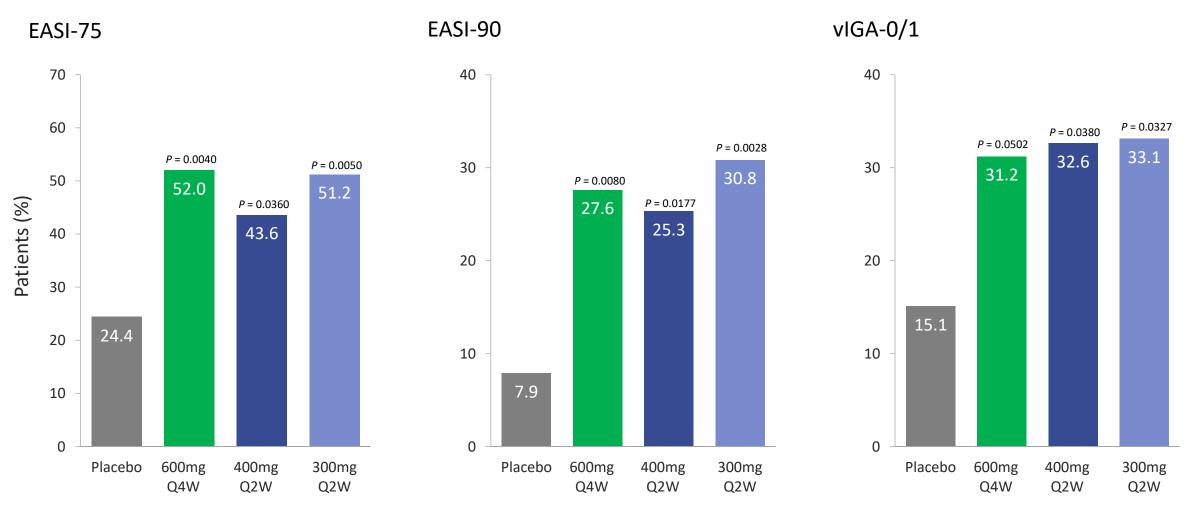
Dose	LS Mean (%)	P value	Statistically significant
600mg Q4W	-73.0	0.0010	✓
400mg Q2W	-65.8	0.0294	✓
300mg Q2W	-69.8	0.0050	✓
Placebo	-51.1		





^{*} The lowest dose group (400mg Q4W) had a LS mean change of 62% improvement in disease after 16 weeks and did not reach statistical significance

Robust efficacy across key secondary endpoints



Eblasakimab was generally well-tolerated, consistent with previous studies

Treatment Emergent Adverse Event (TEAE) ¹ by category - n (%)	Placebo (n=57)	All Ebla (n=232)	600mg Q4W (n=59)	400mg Q2W (n=56)	300mg Q2W (n=58)	400mg Q4W (n=59)
Any	33 (57.9)	164 (70.7)	41 (69.5)	43 (76.8)	32 (55.2)	48 (81.4)
Serious Adverse Event (SAE) ²	1 (1.8)	3 (1.3)	0	1 (1.8)	1 (1.7)	1 (1.7)
AEs with frequency of 5% or more across treatment arms: ³						
 Nasopharyngitis 	5 (8.8)	31 (13.4)	8 (13.6)	8 (14.3)	5 (8.6)	10 (16.9)
Atopic dermatitis	4 (7.0)	20 (8.6)	3 (5.1)	5 (8.9)	4 (6.9)	8 (13.6)
Headache	4 (7.0)	16 (6.9)	8 (13.6)	1 (1.8)	1 (1.7)	6 (10.2)
Upper respiratory tract infection	3 (5.3)	15 (6.5)	3 (5.1)	2 (3.6)	6 (10.3)	4 (6.8)
AEs of interest:						
Injection site reactions	1 (1.8)	11 (4.7)	4 (6.8)	3 (5.4)	0	4 (6.8)
• Conjunctivitis ⁴	1 (1.8)	12 (5.2)	4 (6.8)	5 (8.9)	1 (1.7)	2 (3.4)
Herpes infections	2 (3.5)	7 (3.0)	3 (5.1)	0	1 (1.7)	3 (5.1)
- Herpes simplex infection ⁵	2 (3.5)	6 (2.6)	3 (5.1)	0	0	3 (5.1)
- Herpes zoster infection	0	1 (0.4)	0	0	1 (1.7)	0

¹ This includes all adverse events recorded through to week 16 or last dose for completed patients

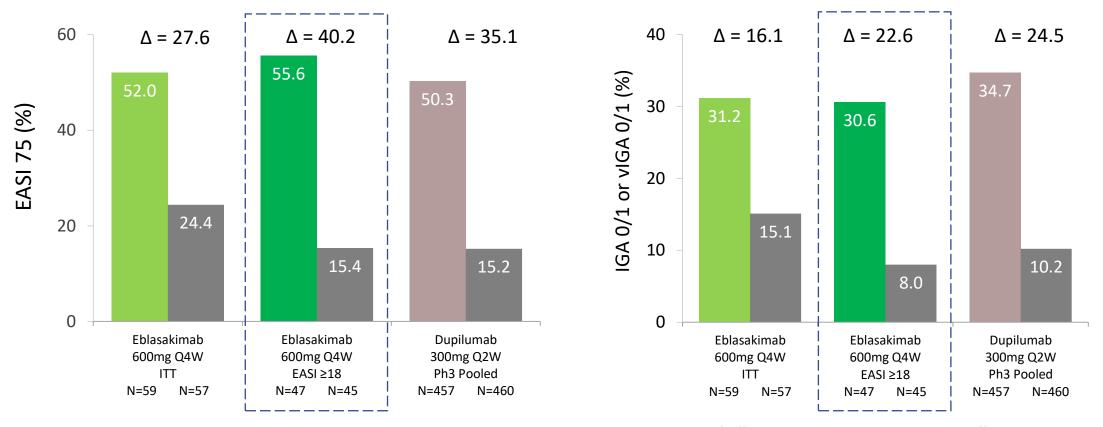
² None were deemed as being drug related, all three across active arms were related to worsening of AD

Applies to AEs that map to the Medical Dictionary for Regulatory Activities dictionary term

⁴ Includes conjunctivitis, noninfectious conjunctivitis and conjunctivitis allergic

Includes oral herpes, herpes simplex infection, herpes virus infection, nasal herpes and herpes ophthalmic

Eblasakimab delivers competitive placebo-adjusted deltas in key secondary endpoints



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.

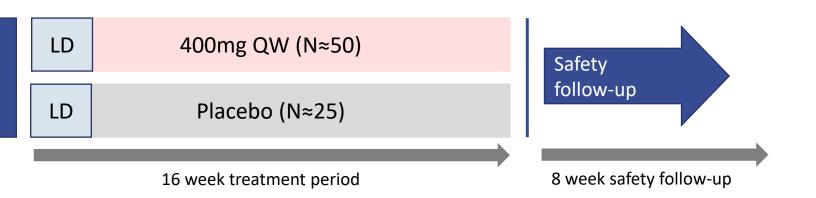
Post-hoc analyses were conducted in patients with baseline EASI score ≥ 18. In keeping with several other recent studies, the placebo response was higher in TREK-AD than *dupilumab* studies conducted a decade ago. A high proportion of patients with milder disease in the US contributed to the high placebo response (over a third of patients in the US had an EASI score between 16 and 18). Average baseline disease severity of EASI ≥ 18 population is comparable to historical dupilumab studies. For more information, refer to: Changes in clinical trial and treatment landscape for AD

Eblasakimab Positive data in dupi experienced patients

TREK-DX: Phase 2 study in *dupilumab* experienced patients testing higher dose regimen ongoing

Adult moderate-to-severe AD patients with *dupilumab* discontinuation $(N \approx 75)$

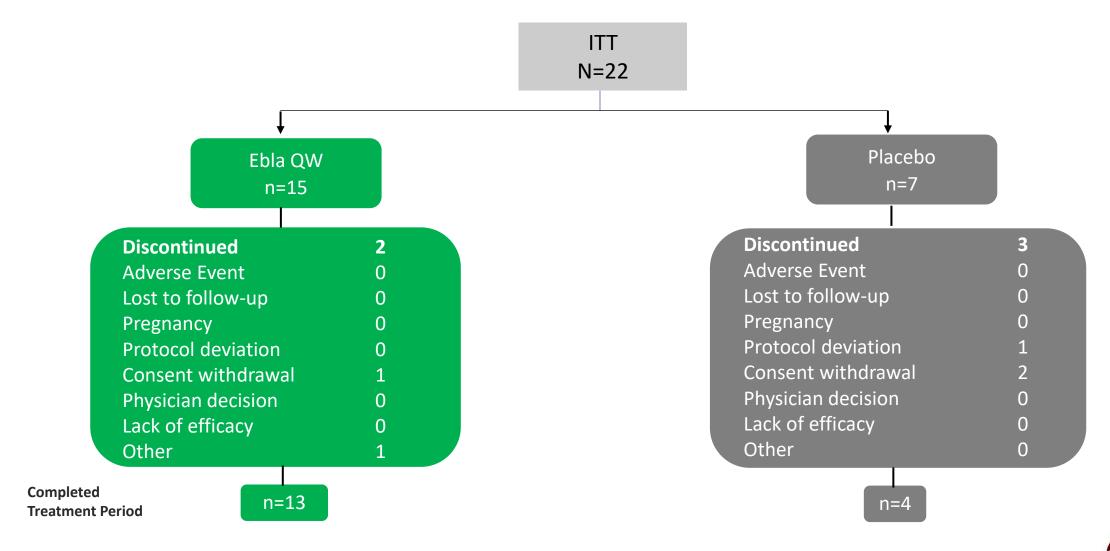
Randomization stratified by: reason for *dupilumab* discontinuation and baseline vIGA score



Key Inclusion/ Exclusion Criteria	EASI ≥16, BSA ≥10% of AD involvement, vIGA 3 or 4 at screening and baseline				
Endpoints	Primary: % change from baseline in EASI at 16 weeks Secondary: EASI-75, EASI-90, vIGA-0/1, PP-NRS (itch)				
Dosing	400mg QW				
Interim analysis populations ¹	ITT : 22 patients EASI ≥18 population : 15 patients				

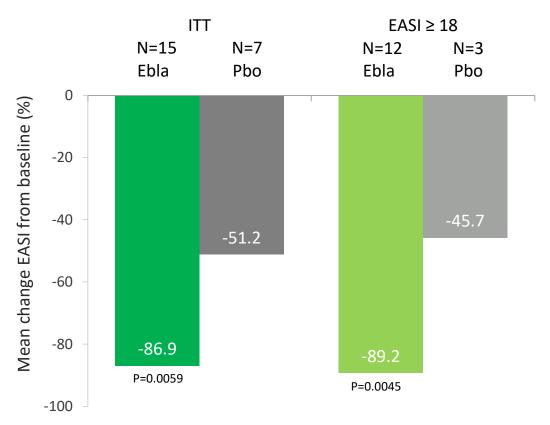


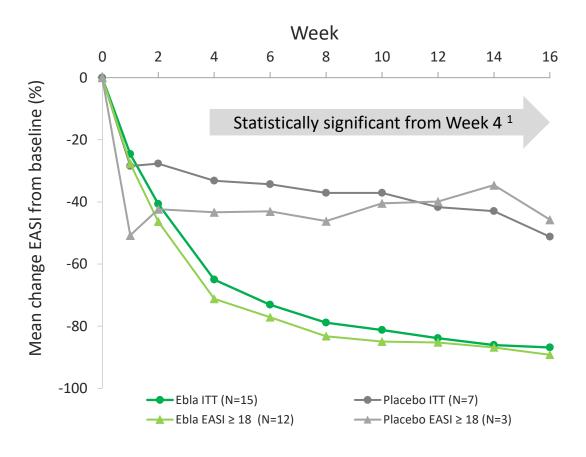
Patient flow



Eblasakimab achieved rapid and significant reduction of EASI scores in dupilumab experienced AD patients in the interim analysis

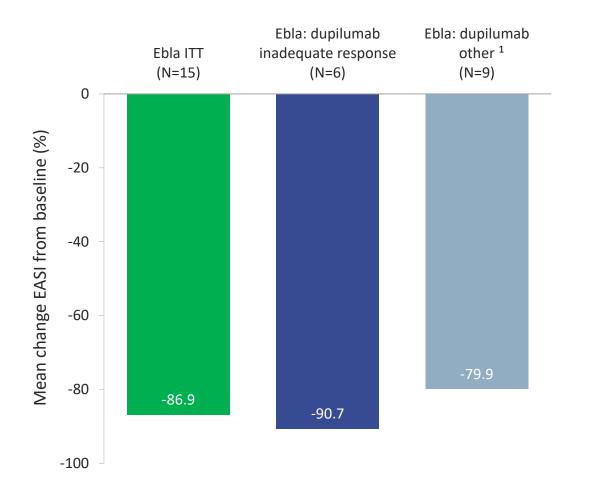
At Week 16 (primary endpoint)

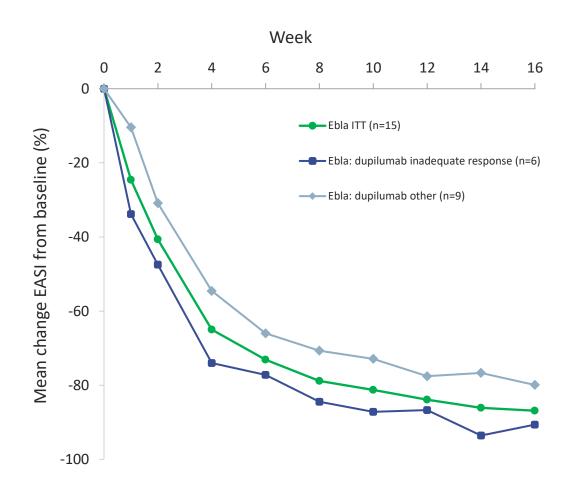




¹ Significant from week 4 for ITT population and from week 6 for EASI ≥ 18 population Least squares mean values using LOCF for missing data. Pbo: Placebo Interim analysis readout April 2024

Patients with prior inadequate response to dupilumab also showed rapid reductions in EASI scores with eblasakimab treatment

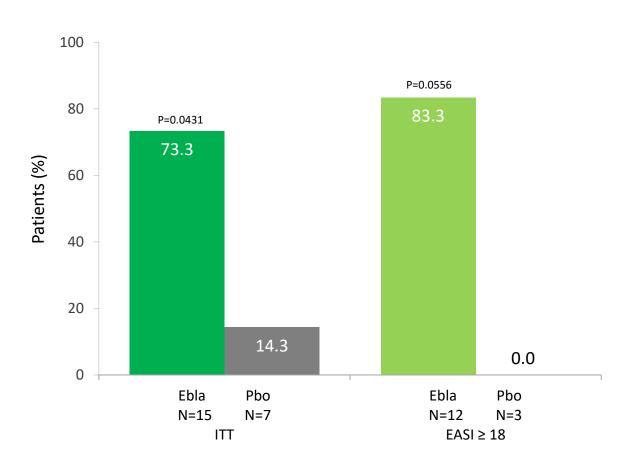


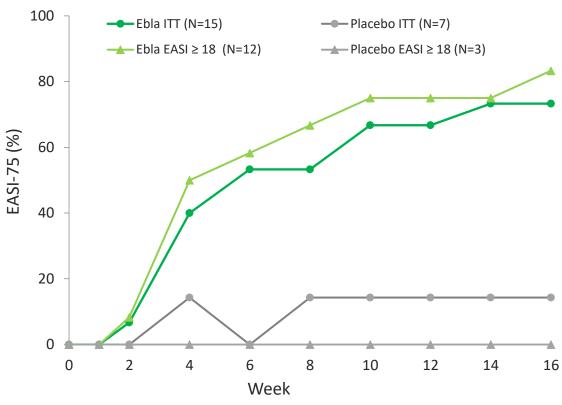




¹ Patients that discontinued *dupilumab* due to reasons other than inadequate response Least squares mean values using LOCF for missing data. Interim analysis readout April 2024

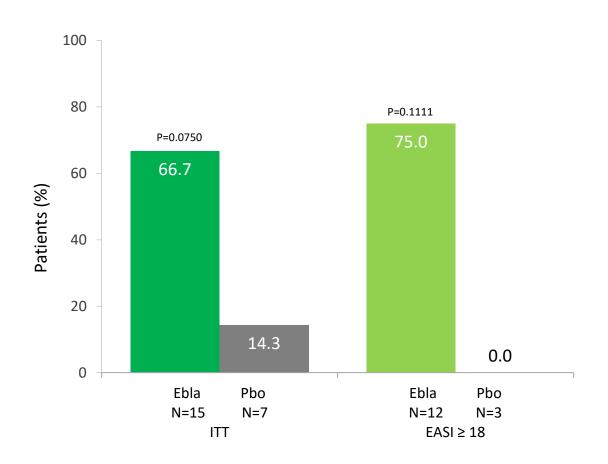
Over half patients achieve EASI-75 by week 6, 73% patients achieve EASI-75 by week 16

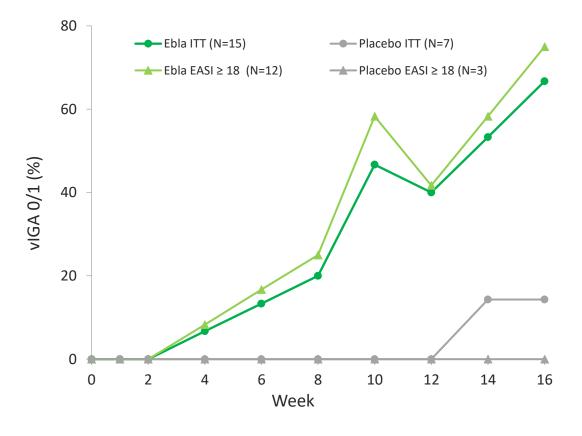






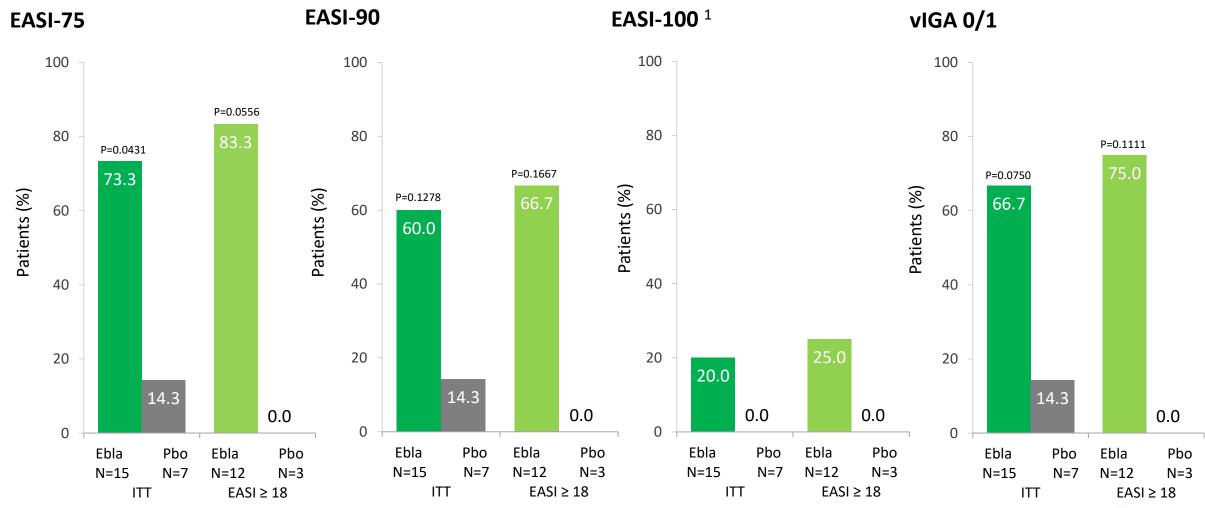
Most patients treated with eblasakimab achieved vIGA 0/1





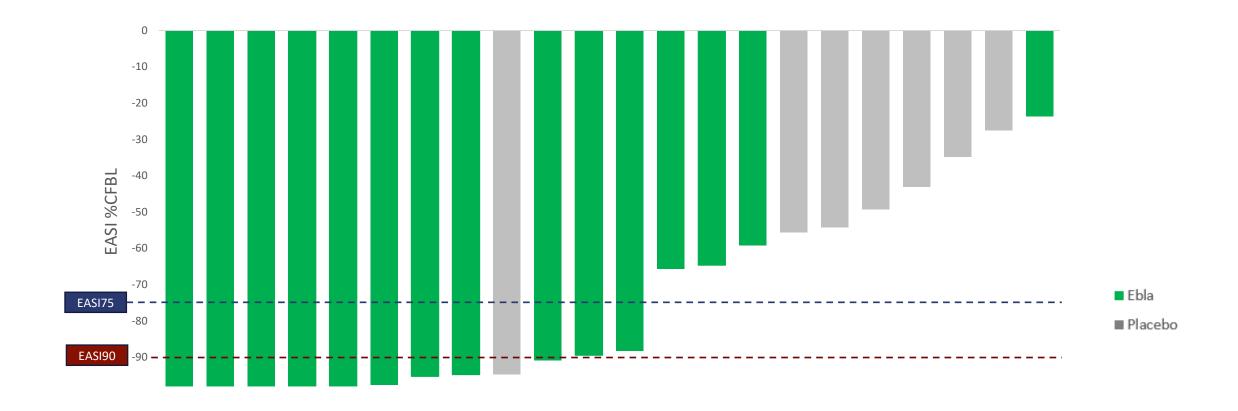


Over 60% of *eblasakimab* treated patients achieved EASI-90 and vIGA 0/1 – unprecedented in prior AD studies with biologics

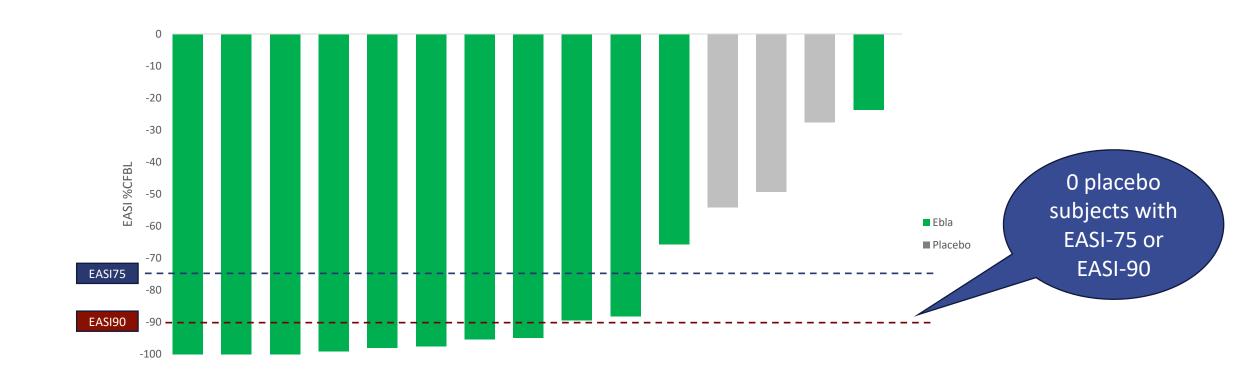


¹ EASI-100 was not a prespecified endpoint and statistical tests were not performed Binary endpoints analyzed using NRI/LOCF Interim analysis readout April 2024

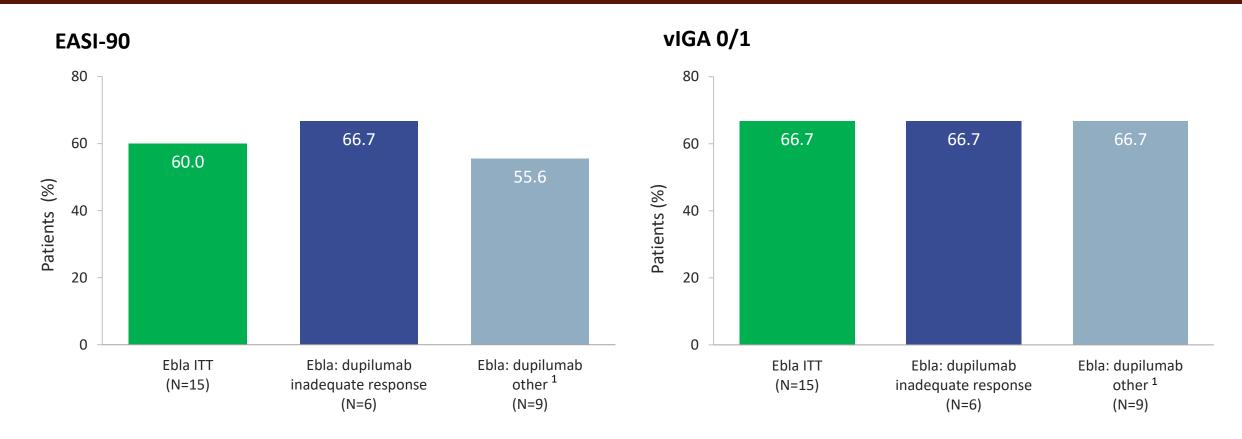
Individual data for ITT population



Individual data for EASI ≥ 18 population



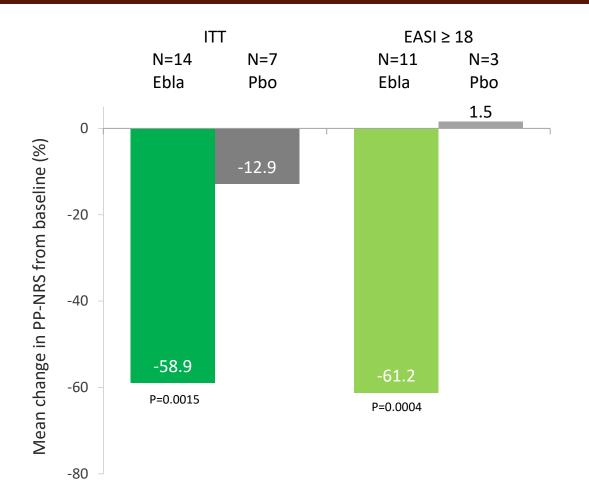
Eblasakimab was equally effective in patients who have an inadequate response to dupilumab

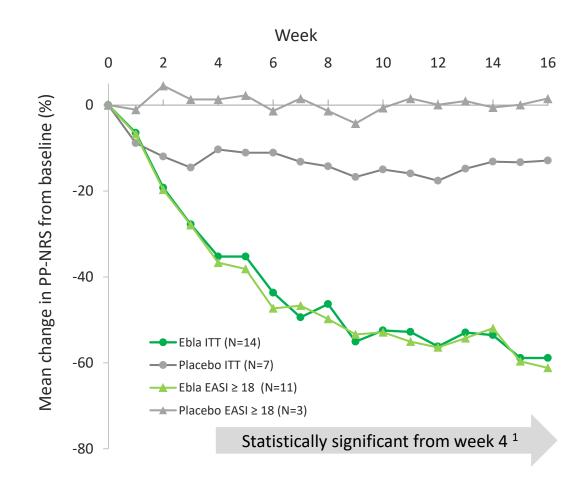


Two thirds of patients treated with *eblasakimab* achieved EASI-90 and vIGA 0/1 even after they previously had an inadequate response to *dupilumab*



Eblasakimab produced rapid and clinically meaningful relief in itch – one of the most burdensome symptoms of AD



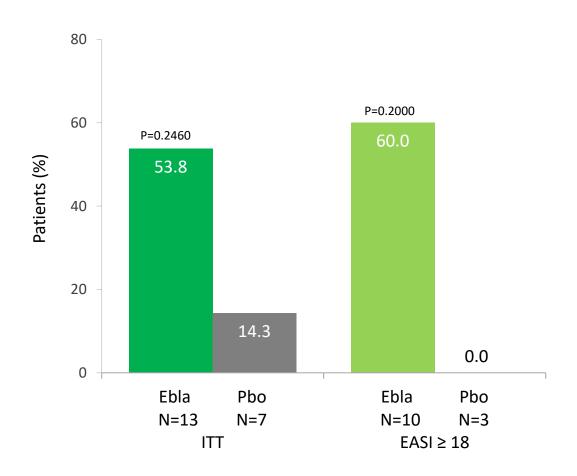


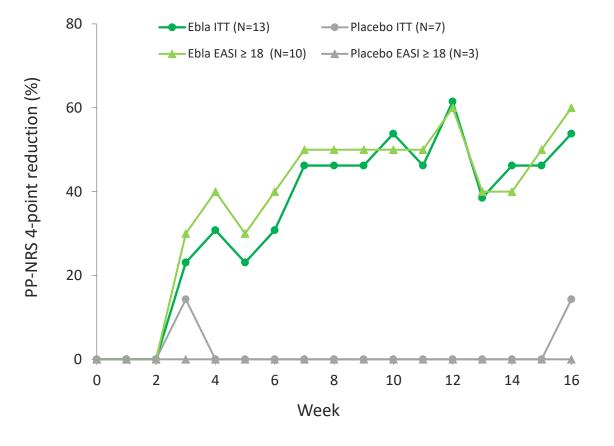


¹ statistically significant from week 4 for EASI ≥ 18 population and from week 6 for ITT population Least squares mean values using LOCF for missing data.

Interim analysis readout April 2024

Proportion with a 4-point reduction in PP-NRS







Safety

- No conjunctivitis reported
- No ISRs
- One SAE (diverticulitis) reported not considered related to randomized treatment
- No new safety signals identified

Case study:

- 74-year-old female on tralokinumab for 4 months, switched to dupilumab
- Developed conjunctivitis after first dose and discontinued dupilumab
- Enrolled into TREK-DX and achieved EASI-100
- No reported conjunctivitis during 24-week treatment and safety follow up

We believe *eblasakimab* can be initially positioned as the therapy of choice for patients with inadequate response or intolerance to *dupilumab*

Initially targeting \$10B second line market ¹

Eblasakimab is the first antibody to target the IL-13 receptor with potential to become a **leading** therapy in treating atopic dermatitis (AD) and other allergic disease

- Potential to be leading second line biologic therapy for patients with inadequate response to dupilumab
- Second line market is substantial with potential to be \$10B by 2029²
- Prescriber experience in second line could enhance use in first line treatment

Translational data supports positioning

Eblasakimab has a unique mechanism of action compared to dupilumab

- Translational data in AD skin biopsies demonstrates *eblasakimab* is more effective at downregulating inflammatory markers than *dupilumab*
- Eblasakimab's MoA has potential to be effective in dupilumab refractory patients

Only placebocontrolled trial in 2nd line

TREK-DX - Phase 2 study of *eblasakimab* in *dupilumab* experienced patients is currently ongoing

First and only double-blind randomized study for patients with inadequate response to *dupilumab*

2 Decision Resources Group, December 2022

¹ Second line market here refers to a second systemic therapy following inadequate response to dupilumab

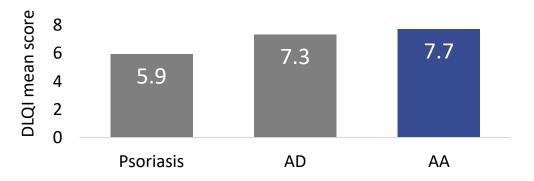


High burden of disease— around 700,000 patients in the US alone

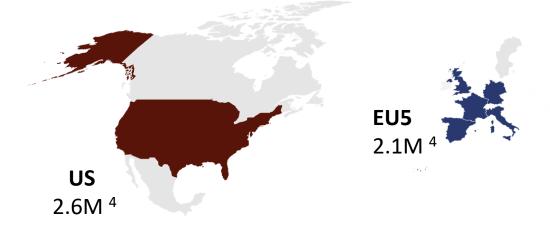
Alopecia areata (AA) is a common autoimmune disease characterised by complete or partial hair loss ¹



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 lifetime⁵
- **700k** patients in the US in 2020 ^{6,7}
- 25% of patients have severe disease ⁶
- 62% of AA patients receive drug treatment 4

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

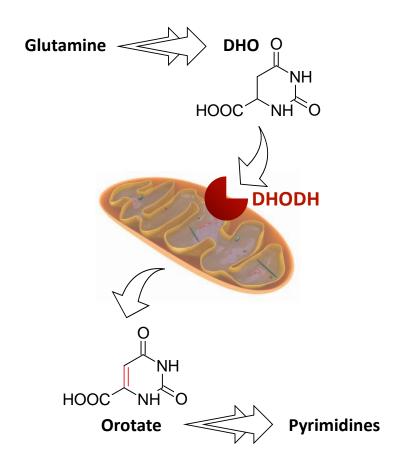
^{5.} Mirzoyev et al (2014) J Inv Derm 134(4):1141-1142

^{6.} Benigno et al (2020) Clin, Cos & Invest Derm 13:259-266

^{7.} Mostaghimi et al (2023) JAMA Derm 159(4):411-418

DHODH is a validated target for autoimmune disease

The *de novo* pathway



- DHODH inhibition will block *de novo* pathway of pyrimidines, impacting rapidly dividing cells eg T cells during autoimmune triggers
- Other cells can use salvage pathways to make pyrimidines
- DHODH inhibitors are approved in multiple sclerosis and rheumatoid arthritis
- However, first-generation DHODH inhibitors have limited potency and significant safety liabilities
- Farudodstat was designed to be more potent and to address the toxicities associated with first-generation inhibitors
- New Composition of Matter Patent for farudodstat received positive opinion from European Patent Office in February 2024, could provide commercial exclusivity until 2043

Farudodstat's mechanism of action inhibits key processes in AA

Healthy hair follicle Alopecia areata affected hair follicle farudodstat CD4+Tcell CD8+Tcell T cell activation and cytokine IFNy production γδ T cell In an ex vivo human model of AA, farudodstat reduced key drivers of AA disease pathology, including T cell expansion

AA pipeline is dominated by JAKi, novel mechanisms are needed

MoA	Company	Product/Product Candidate	Stage
JAK inhibitors	Lilly	Olumiant <i>(baricitinib)</i>	Approved
	P fizer	Litfulo (ritlecitinib)	Approved
	CoNCERT Pharmaceuticals Inc.*	Deuruxolitinib	Phase 3
	ulli Bristol Myers Squibb	Deucravacitinib	Phase 2
	Reistone	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
IL-2	NEKTAR [°]	Rezpegaldesleukin	Phase 2a
IL-7Rα antagonist	@32 BIO	ADX-914	Phase 2a PoC
OX40/CD134	INMA ENE BORDERLESS INNOVATION	IMG-007	Phase 1b/2a, open label

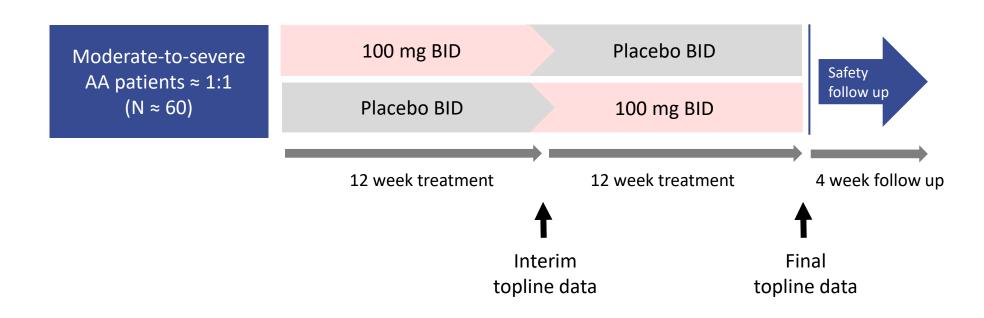
WARNING: SERIOUS INFECTIONS, MORTALITY, MALIGNANCY, MAJOR ADVERSE CARDIOVASCULAR EVENTS (MACE), and THROMBOSIS

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

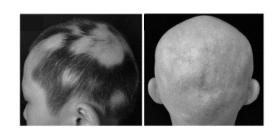
Phase 2a: Proof-of-concept trial in AA, topline expected Q3 2024



Primary efficacy endpoint: % change from baseline in SALT score Select inclusion criteria:

- Adults with 30% or greater scalp hair loss (SALT score ≥ 30)
- Current episode of hair loss duration between 6 months to 7 years

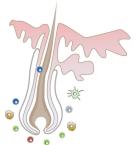
Farudodstat has the potential to be an effective, novel approach in the treatment of AA



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



Farudodstat is approximately 30-fold more potent at inhibiting DHODH, a validated target, than first-generation inhibitors



Farudodstat potentially inhibits the key drivers of AA pathophysiology



Phase 2a proof-of-concept study in AA initiated, interim **topline readout expected Q3 2024**

Upcoming milestones

Multiple upcoming catalysts over the next 12 months

Ticker

Net operating cash used

Cash balance

Upcoming milestones expected in 2024

NASDAQ: ASLN

\$ 7.4M (1Q 2024)

\$18.4M as of March 31, 2024

- Eblasakimab topline readout from TREK-DX trial end 2024
- Partnership selection to advance *eblasakimab* into Phase 3
- Farudodstat Phase 2a interim topline data Q3 2024
- Publication and presentation of further data from the TREK-AD and TREK-DX studies of *eblasakimab* and on *farudodstat* at major conferences