

# Company presentation

March 2024

NASDAQ: ASLN



# Legal disclaimer

This presentation contains forward-looking statements. These statements are based on the current beliefs and expectations of the management of ASLAN Pharmaceuticals Limited and/or its affiliates (the "Company"). These forward-looking statements may include, but are not limited to statements regarding: the Company's plans to develop and commercialize *eblasakimab* and *farudodstat*; the potential of *eblasakimab* as a first-in-class treatment for atopic dermatitis and other allergic diseases, and of *farudodstat* as a first-in-class treatment for alopecia areata; the Company's plans and expected timing with respect to clinical trials, clinical trial enrollment and clinical trial results for *eblasakimab* and *farudodstat*; the Company's plans and expected timing with respect to regulatory filings and approvals; the size and growth of the markets for the Company's product candidates; anticipated timelines and milestones; the potential fees, milestone and royalty payments and development activities under the strategic license agreement with Zenyaku; the rising prevalence of atopic dermatitis and significant unmet need to advance the standard of care for atopic dermatitis; forecasts regarding potential demand, size of market and market share, and patient preferences; the Company's competitors, their anticipated developments, and *eblasakimab*'s potential competitive advantages; the safety profile and efficacy of *eblasakimab* and *farudodstat*, including preliminary blinded data and the potential for *eblasakimab* to have an improved safety profile than *dupilumab* and the expectation that *eblasakimab* will be efficacious against a wide range of atopic dermatitis comorbidities; the Company's business strategy and clinical development plans; and the Company's cash runway. The Company's estimates, projections and other forward-looking statements are based on management's current assumptions and expectations of future events and trends, which affect or may affect the Company's business, strategy, operations, or financial performance, and inherently involve significant known and unknown risks and uncertainties, which include, without limitation: unexpected safety or efficacy data observed during preclinical or clinical studies; risks that future clinical trial results may not be consistent with interim, initial or preliminary results or results from prior preclinical studies or clinical trials; risks that trends or characteristics based on preliminary blinded data may not be consistent with unblinded data; clinical site activation rates or clinical trial enrolment rates that are lower than expected; the impact of health epidemics or pandemics; ongoing geopolitical conflicts on the Company's business and the global economy; general market conditions; changes in the competitive landscape; and the Company's ability to obtain sufficient financing to fund its strategic and clinical development plans. Actual results and the timing of events could differ materially from those expressed or implied in such forward-looking statements as a result of these risks and uncertainties, which include, with limitation, the risk factors described in the Company's U.S. Securities and Exchange Commission filings and reports (Commission File No. 001- 38475), including the Company's Annual Report on Form 20-F filed with the U.S. Securities and Exchange Commission on March 24, 2023. This presentation discusses product candidates that are under clinical study, and which have not yet been approved for marketing by the US Food and Drug Administration. No representation is made as to the safety or effectiveness of these product candidates for the use for which such product candidates are being studied. Caution should be exercised when comparing data across trials of different products and product candidates. Differences existing between trial designs and patient populations and characteristics. The results across such trials may not have interpretative value on our existing or future results. All statements other than statements of historical fact are forward-looking statements. The words "anticipate," "estimate," "expect," "forecast," "intend," "plan," "potential," "may," "will," or the negative of those terms, and similar expressions that convey uncertainty of future events or outcomes are intended to identify estimates, projections, and other forward-looking statements. Estimates, projections, and other forward-looking statements speak only as of the date they were made, and, except to the extent required by law, the Company undertakes no obligation to update or review any estimate, projection, or forward-looking statement.



# 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 <sup>1</sup>, **only 2 approved biologics** in US to date
- **Positive phase 2b** study of *eblasakimab* in AD in July 2023, phase 3 preparation underway. First biologic to deliver a **monthly dosing regimen** from initiation with no compromise on efficacy
- Initial positioning as therapy of choice for patients that have **inadequate response to *dupilumab***. Data in *dupilumab*-experienced patients expected end 2024
- 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 mid-2024

## Financials

**Cash position \$40.8M** as of September 30, 2023

- \$12M prepayment to K2 in Dec 2023, further payments deferred to 2025 – reduces total cash burn in 2024
- \$5M registered direct offering closed on March 14, 2024



# Multiple catalysts in upcoming 12 months

Program	Target	Indication	Preclinical	Phase 1	Phase 2	Phase 3	Anticipated milestones in 2024	
<i>Eblasakimab</i>	IL-13R $\alpha$ 1	Atopic dermatitis	Biologic naïve					<ul style="list-style-type: none"> <li>• Selection of partner to advance <i>eblasakimab</i> into Phase 3</li> <li>• Topline readout from <i>dupilumab</i>-experienced trial end 2024</li> </ul>
			<i>Dupilumab</i> experienced					
	COPD						<ul style="list-style-type: none"> <li>• Translational data in COPD to be presented 2Q 2024</li> </ul>	
<i>Farudodstat</i>	DHODH	Alopecia areata					<ul style="list-style-type: none"> <li>• Phase 2a interim topline data mid-2024</li> </ul>	





# *Eblasakimab*

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



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

Rheumatoid Arthritis (RA)



Psoriasis



Atopic Dermatitis



1990

1995

2000

2005

2010

2015

2020

2025



# Recent years have seen many disappointments with new mechanisms

Earlier	2018	2019	2020	2021	2022	2023	2024
<del>IgE</del>	<del>IL-5</del>	<del>IL-22</del>	<del>IL-17</del>	<del>TSLP</del>	<del>IL-1<math>\alpha</math></del>	OX <del>?</del> 40	<del>Siglec-8</del>
<del>CRTH2</del>	<del>NK-1R</del>	<del>IL-17C</del>	<del>IL-33</del>		<del>IL-36</del>	IL- <del>?</del> 31	
<del>NK-1R</del>	<del>CD40</del>	<del>IL-33</del>	<del>H4-R</del>				

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





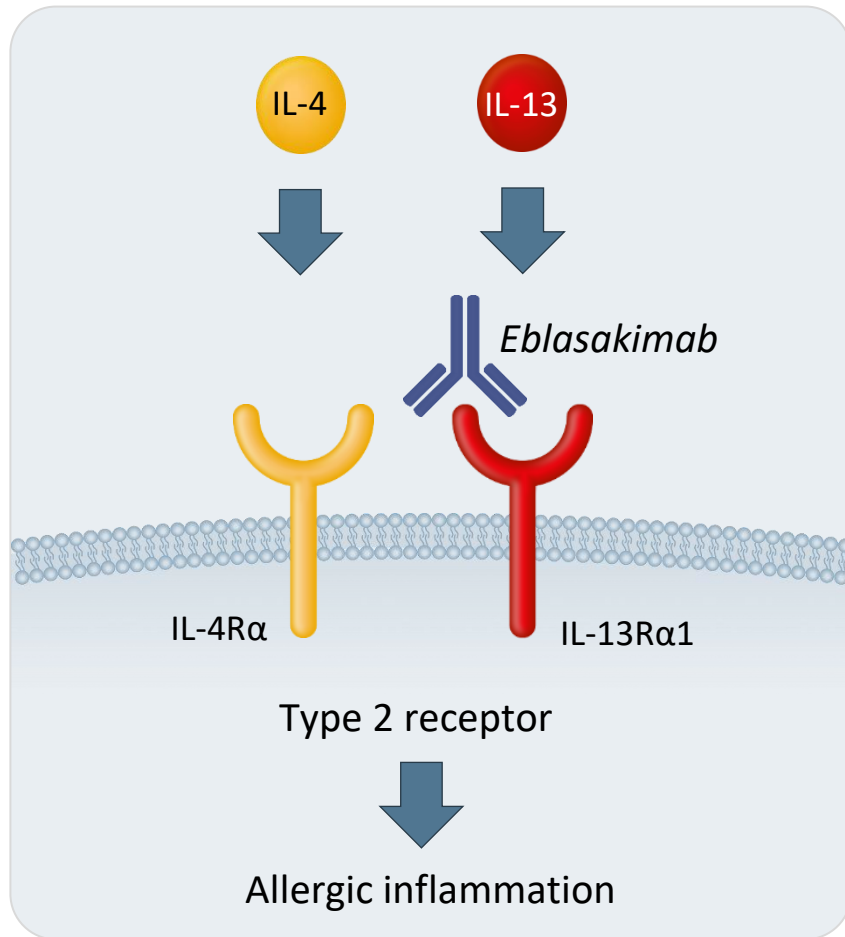
# *Eblasakimab*

A novel mechanism for treatment of AD





# *Eblasakimab* is the only monoclonal antibody in the clinic targeting the IL-13 receptor<sup>1</sup>



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

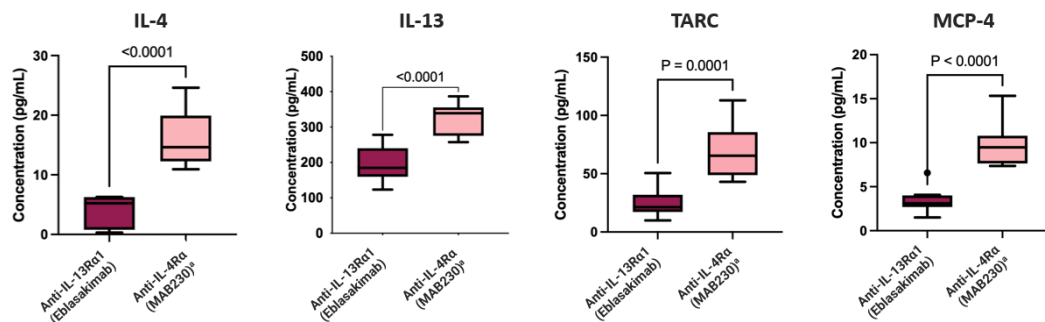
<sup>1</sup> Based on search of Clarivate and BiomedTracker databases



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

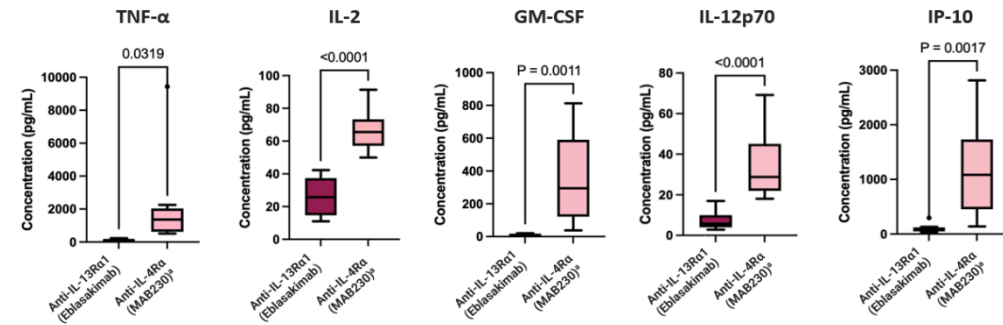
## Th2 cytokines

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:

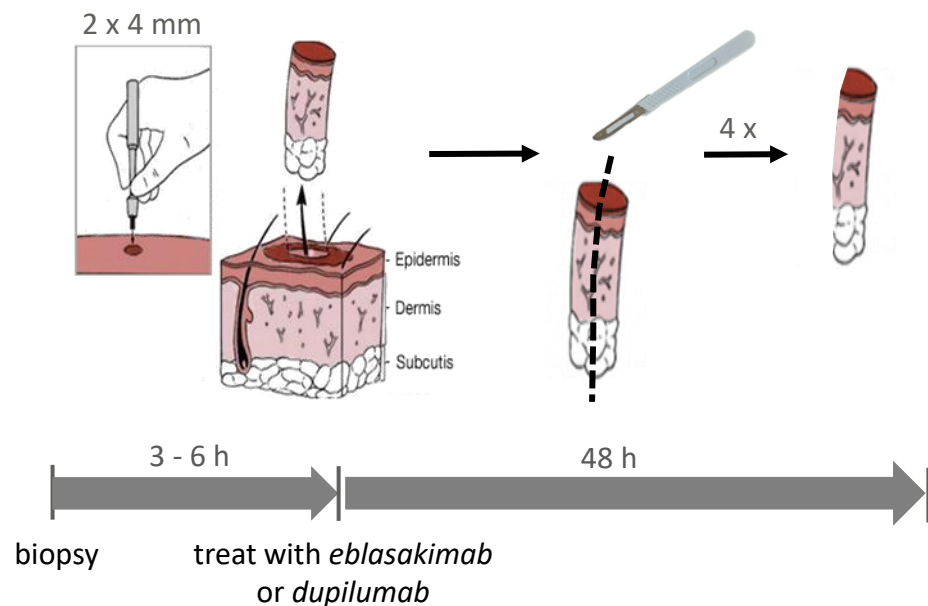
- More efficient reduction of Th2 inflammation
- No increase in Th1 cytokines, compared to IL-4R blockade

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.

Data presented at the 1<sup>st</sup> International Society of Investigative Dermatology Meeting, May 10-14, 2023, in Tokyo, Japan, in late-breaker minisymposium (Cevikbas et al)

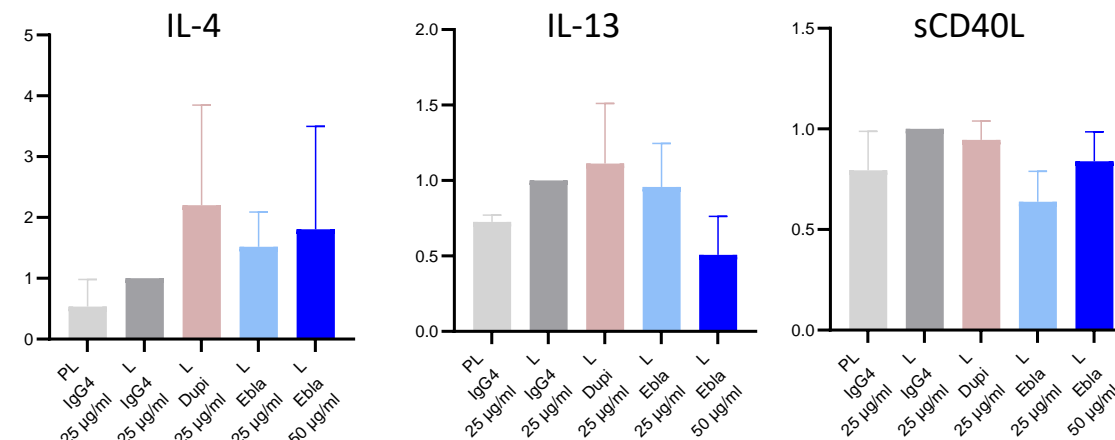


# Head-to-head study between *eblasakimab* and *dupilumab* in skin biopsies confirm differentiated effects of targeting IL-13R vs IL-4R

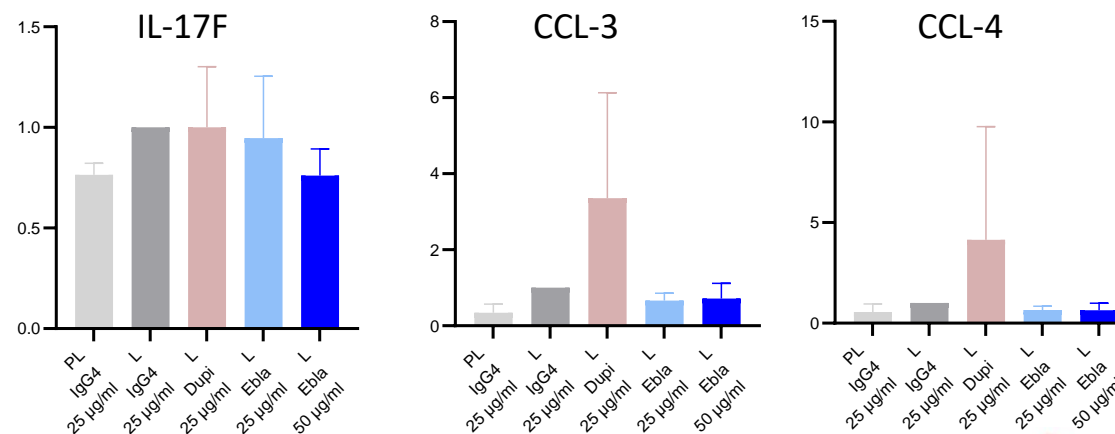


In AD lesional skin biopsies, *eblasakimab* reduced secretion of pro-inflammatory Th2 cytokines as well as other AD relevant mediators more efficiently than *dupilumab*

## Pro-inflammatory Th2 cytokines



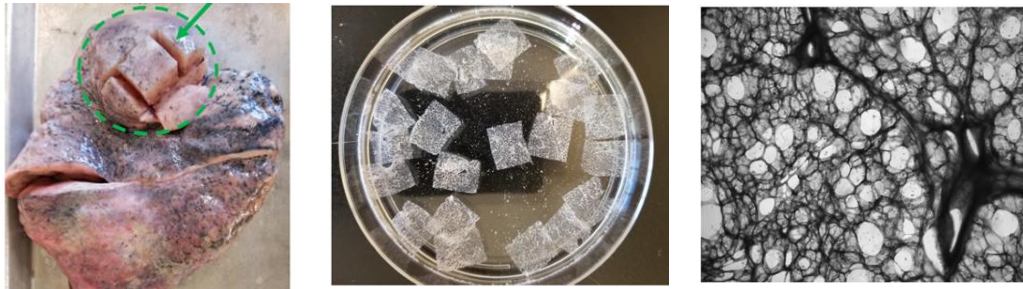
## Other inflammatory markers



# New translational work in COPD reinforces *eblasakimab's* differentiation in treating comorbidities

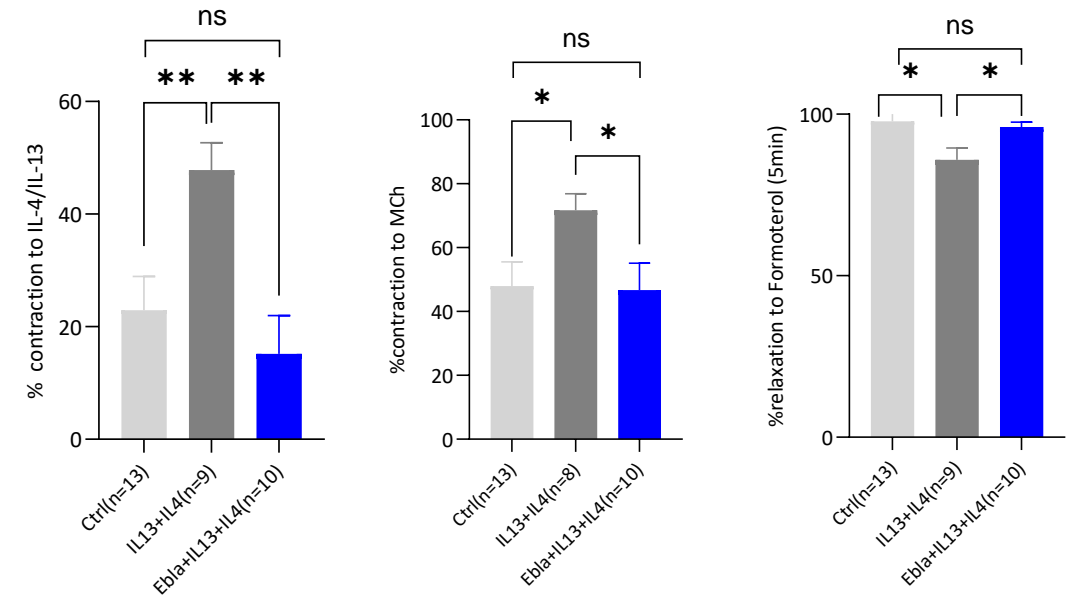
COPD is a heterogeneous lung condition characterized by chronic respiratory symptoms due to abnormalities of the airways and/or alveoli which causes persistent, often progressive, airflow obstruction

## Precision Cut Lung Slices (PCLS): human *ex vivo* model of COPD<sup>1</sup>



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

*Eblasakimab* performed better than *dupilumab* in improving airway function and enhancing bronchodilation in both healthy and COPD lung tissue in a head-to-head study at the same concentrations



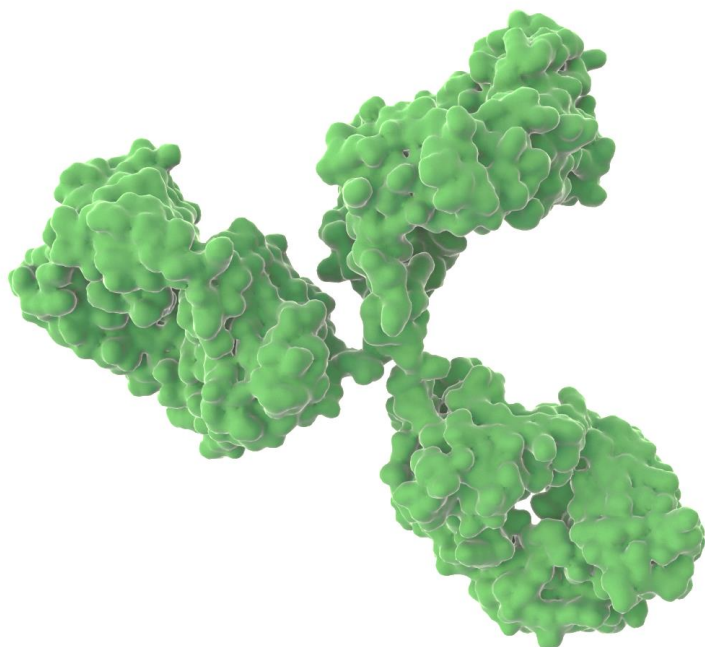
All data from healthy donor tissue

*Eblasakimab* significantly reduced IL-4/IL-13 driven airway constriction and reversed effects of cytokines to limit dilation by formoterol





# *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 1 receptor while sparing the Type 2 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

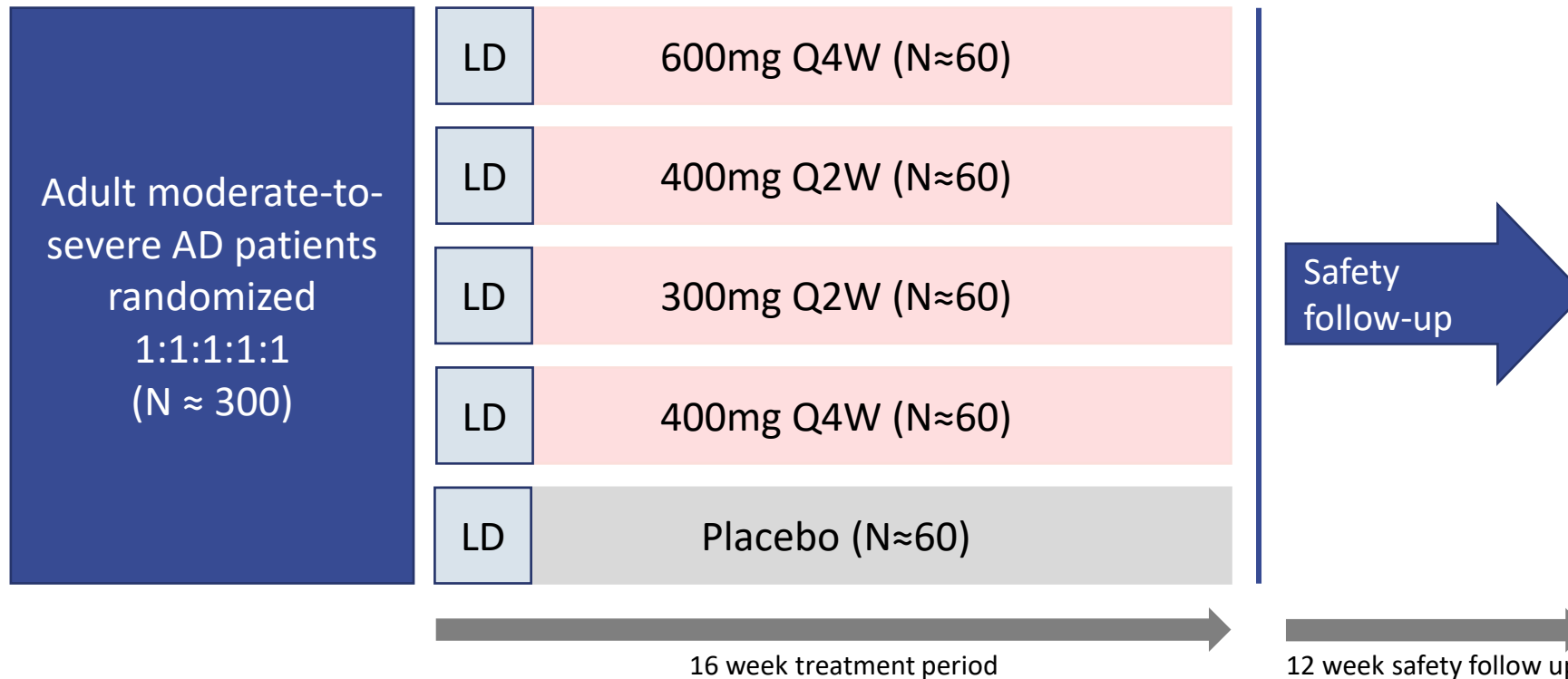
- TREK-AD is a **global dose-ranging study** testing *eblasakimab* conducted across 8 countries with around 300 moderate-to-severe AD patients
- The **study was positive** and demonstrated **potential for monthly dosing**
  - The study met the primary endpoint and key secondary endpoints in the ITT population in the 3 key doses <sup>1</sup>
  - 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

<sup>1</sup> Refers to the 3 highest dose groups out of a total 4 doses that were tested



# TREK-AD: Phase 2b in biologic naïve patients

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



## Key inclusion criteria

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

## Primary endpoint

- % change in EASI from baseline

## Key secondary endpoints

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

- 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

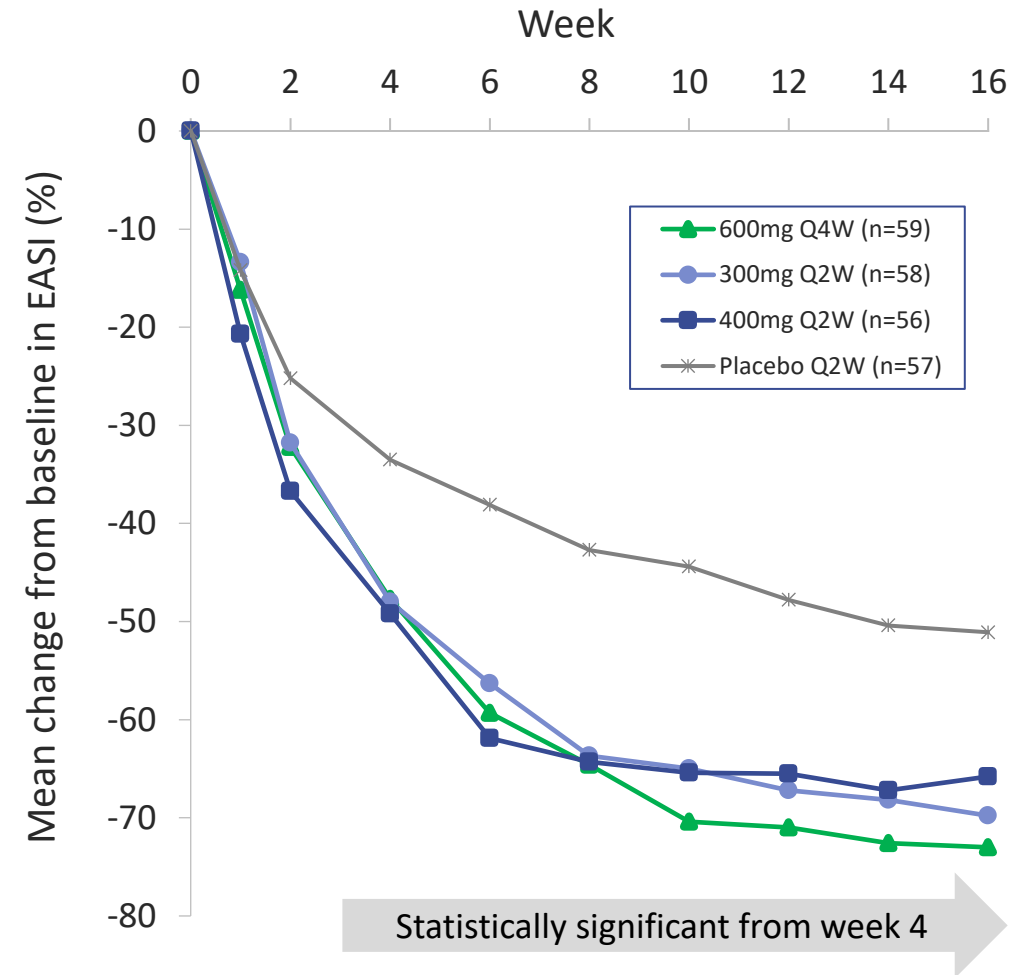




# Monthly dosing with 600mg led to 73% improvement in disease after 16 weeks and was statistically significant from week 4

*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		

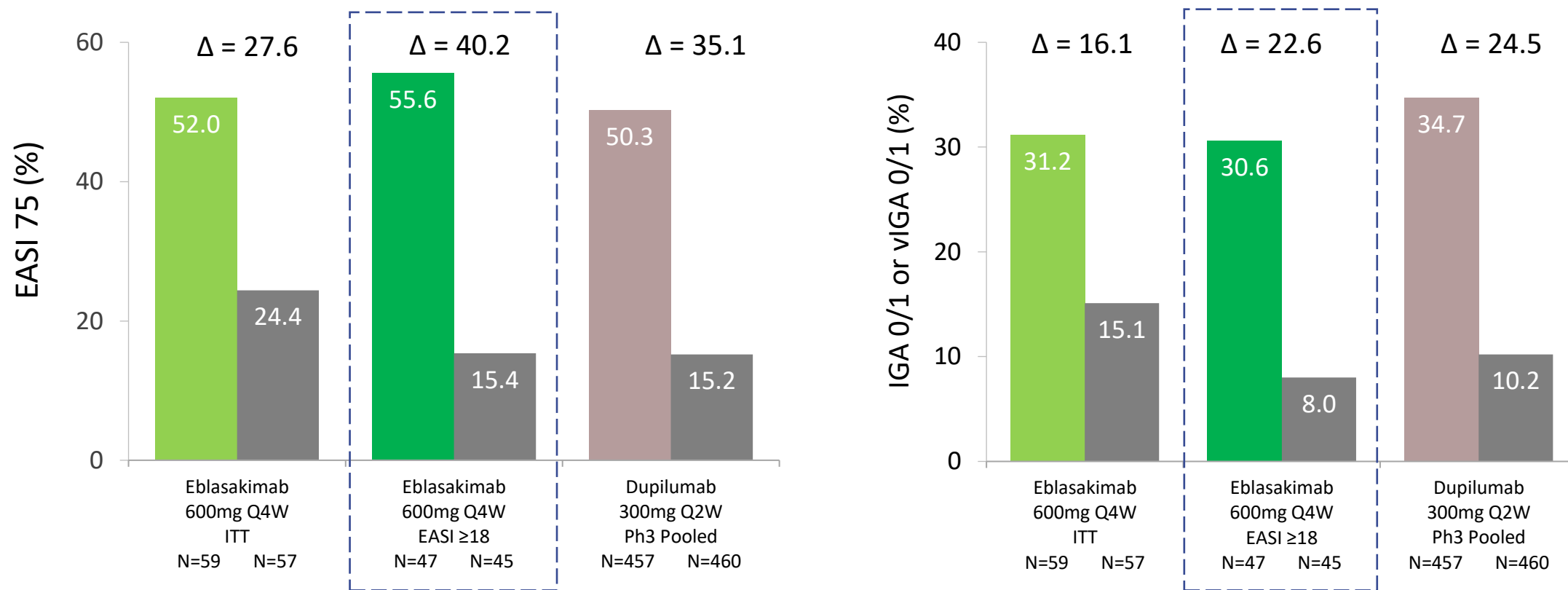


P value is calculated versus placebo for least squares mean values by MMRM method

\* 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



# 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  $\geq 18$ . 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  $\geq 18$  population is comparable to historical dupilumab studies. For more information, refer to: [Changes in clinical trial and treatment landscape for AD](#)







*Eblasakimab*

Positioning in the rapidly evolving AD market



# Eblasakimab has the potential to be a leading therapy in AD

		<i>Eblasakimab</i>	<i>Dupilumab</i>	<i>Tralokinumab</i>	<i>Lebrikizumab</i> <sup>1</sup>
Dosing		Once monthly regimen from start of treatment <sup>2</sup>	✓		
Efficacy attributes		Over half of patients see 75% improvement in disease within 16 weeks <sup>3</sup>	✓	✓	✓
		Significant reduction in EASI score by week 2 <sup>3</sup>	✓		
Safety attributes		Low rates of conjunctivitis and injection site reactions <sup>4</sup>	✓		
Treats comorbidities		Potential to be effective in broad range of allergic diseases <sup>5</sup>	✓	✓	

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<sup>1</sup> *Lebrikizumab* is approved for AD only in EU and Japan, not in US

<sup>2</sup> For approved drugs, based on approved dosing regimens. For candidate drugs, based on regimens tested in phase 3 program at initiation of treatment (after loading doses)

<sup>3</sup> For approved drugs, based on label. For candidate drug, based on published results and potential of establishing a claim in future studies

<sup>4</sup> For all, based on monotherapy phase 3 showing conjunctivitis and injection site reaction rate less than 6%

<sup>5</sup> Allergic diseases relate to Type 2 driven conditions including allergic rhinitis, allergy, asthma, food allergies





# We believe *eblasakimab* can be initially positioned as the therapy of choice for patients that have inadequate response to *dupilumab*

Initially targeting \$10B second line market <sup>1</sup>

***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 2029** <sup>2</sup>
- 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 placebo-controlled trial in 2<sup>nd</sup> line

TREK-DX - Phase 2 study of *eblasakimab* in *dupilumab* experienced patients is currently ongoing  
Only randomized, placebo-controlled study for patients with inadequate response to *dupilumab*

<sup>1</sup> Second line market here refers to a second systemic therapy following inadequate response to *dupilumab*

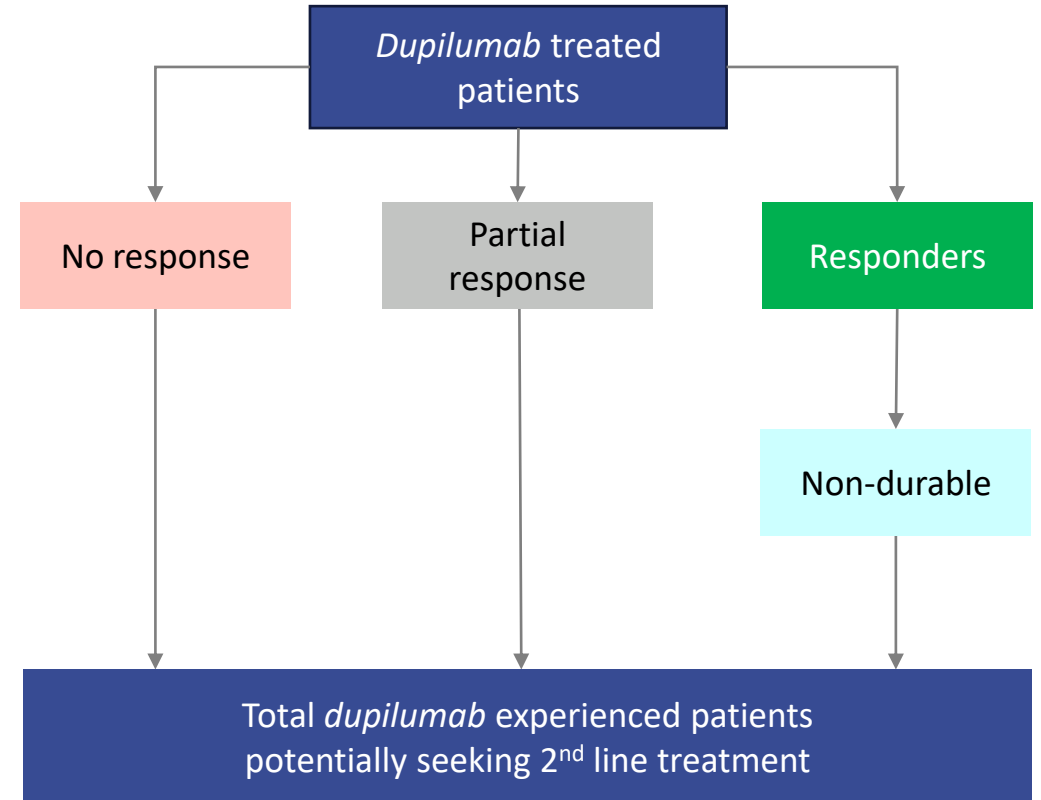
<sup>2</sup> Decision Resources Group, December 2022



# Patients in need of second line 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* <sup>1</sup>
- However, 63% of *dupilumab*-treated patients do not achieve IGA 0/1 <sup>2</sup> within 16 weeks and of those that do, only 54% maintain the response at week 52 <sup>3</sup>
- 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 <sup>4</sup>

Based on market research <sup>4</sup>, we believe around 150,000 patients who are currently using or have used *dupilumab* could switch to an alternative biologic treatment



1. 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  
2. Thaci et al (2019) J Dermatol Sci 94(2):266-275  
3. Worm et al (2020) JAMA Derm 156(2):131-143  
4. 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 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



# What do patients with inadequate response to *dupilumab* look for?

Attribute	<i>Eblasakimab</i> <sup>1</sup>	<i>Lebrikizumab</i> <sup>2</sup>	JAK inhibitors	OX-40 inhibitors <sup>1</sup>	APG777 <sup>1</sup>
<b>Long-term safety</b> <sup>3</sup> <i>AD is a chronic disease</i>	✓	✓	X	?	✓
<b>Efficacy</b> <sup>4</sup> <i>Better than or equal to dupilumab at week 16</i>	✓	✓	✓	X	?
<b>Rapid speed of onset</b> <sup>5</sup> <i>Potential for significant reduction in EASI score by week 2</i>	✓	X	✓	X	?
<b>Potential to treat comorbidities</b> <i>78% of AD patients have allergic comorbidities</i> <sup>6</sup>	✓	X	?	✓	X
<b>Effective in target patient population</b> <i>Data from placebo-controlled study</i> <sup>7</sup>	In progress	X	X	X	?

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1 Currently in clinical development and not approved therapies

2 *Lebrikizumab* is approved for AD only in EU and Japan, not in US

3 Long term safety defined by lack of boxed warnings on FDA label or potential warnings for long term safety based on MoA

4 Efficacy defined by placebo-adjusted EASI-75 scores at week 16 in latest clinical trials in comparison to *dupilumab* Phase 3

5 Defined by significant difference in EASI score reduction between treatment and placebo at week 2. *Eblasakimab* results from EASI ≥18 population in Phase 2b study

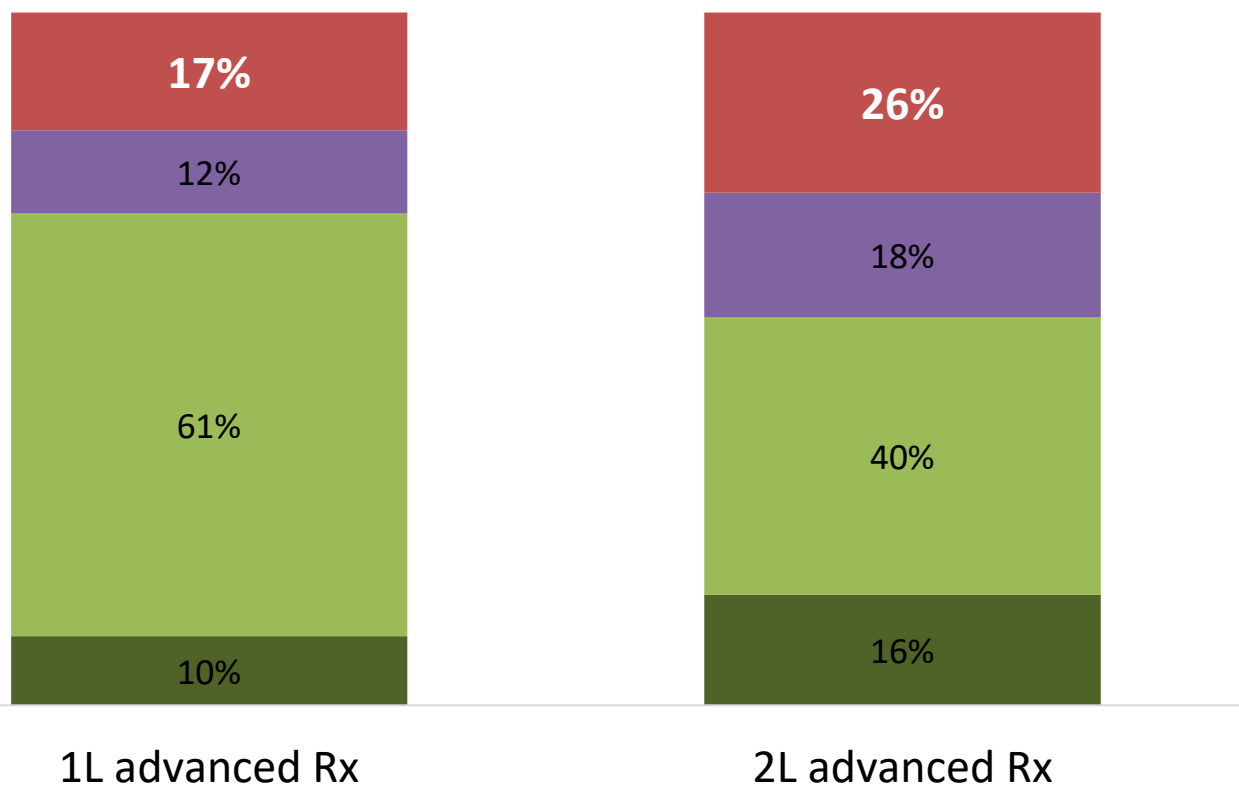
6 Calzavara-Pinton et al (2023) *Adv Ther* 40:5366-5382. Based on MoAs expected to be efficacious in a broad range of co-morbidities

7 Referring to placebo-controlled study designed in *dupilumab* experienced population



# *Eblasakimab* was given the largest market share by health care providers among biologics in 1L and 2L after *dupilumab*

1L / 2L share of patients on biologics from survey <sup>1</sup>



Biologics market in AD expected to be \$19B by 2029 <sup>2</sup>

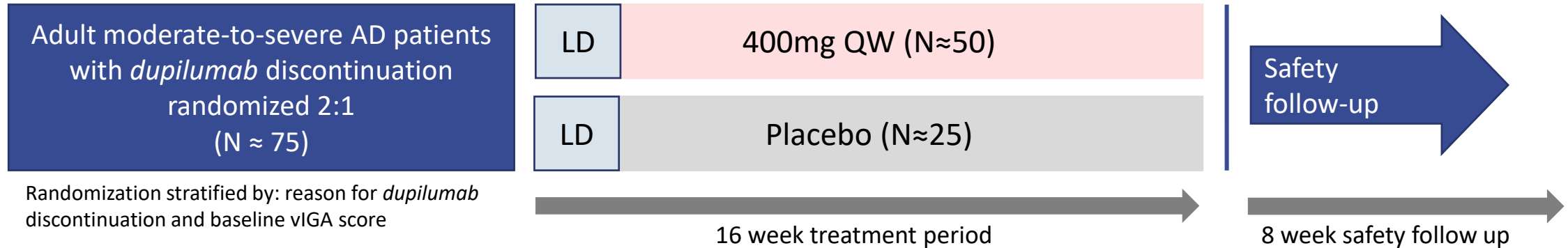
- Eblasakimab*
- Lebrikizumab*
- Dupilumab*
- Tralokinumab*

<sup>1</sup> Market research conducted by ASLAN from August-October 2023 with 93 health care providers for AD in the US (44% dermatologists, 22% allergists and 34% physician assistants and nurse practitioners). 1L: first line of advanced treatment for moderate-to-severe AD patients following topical/ conventional therapies, 2L: second line treatment following an advanced therapy Preference share calculation uses weighted average % of moderate-severe AD patients, includes exposure to *eblasakimab* profile after *lebrikizumab* profile

<sup>2</sup> DRG 2022 Report: AD Disease Landscape and Forecast Report



# TREK-DX: Phase 2 study in *dupilumab* experienced patients ongoing



- Preliminary blinded data review conducted in February 2024 including all 22 patients enrolled to date:
  - 17 patients completed the 16 week treatment period and 5 patients discontinued prior to that
  - 45% of patients saw at least a 90% reduction in their EASI score (EASI-90) and 50% of patients achieved a vIGA score of 0 or 1 (clear or almost clear skin) after 16 weeks
  - Of the 9 patients who previously had an inadequate response to dupilumab, 5 patients (56%) achieved EASI-90 and 5 patients (56%) a vIGA score of 0 or 1
  - Treatments were well-tolerated and no new safety signals were identified. No reports of conjunctivitis or injection site reactions
- Expected readout of topline data end 2024





# *Farudodstat*

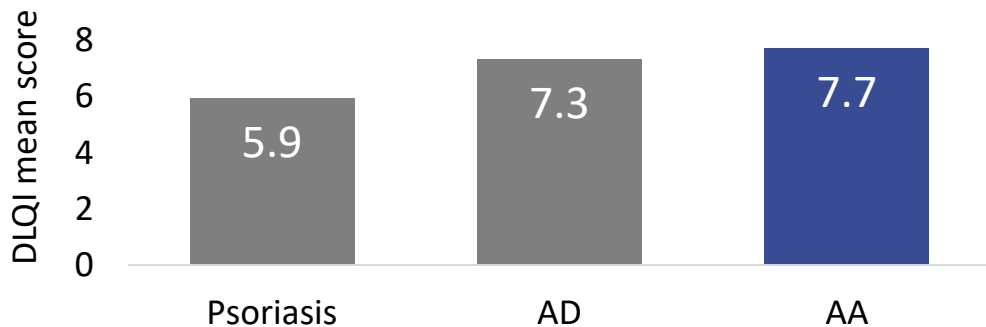


# High burden of disease— around 700,000 patients in the US alone

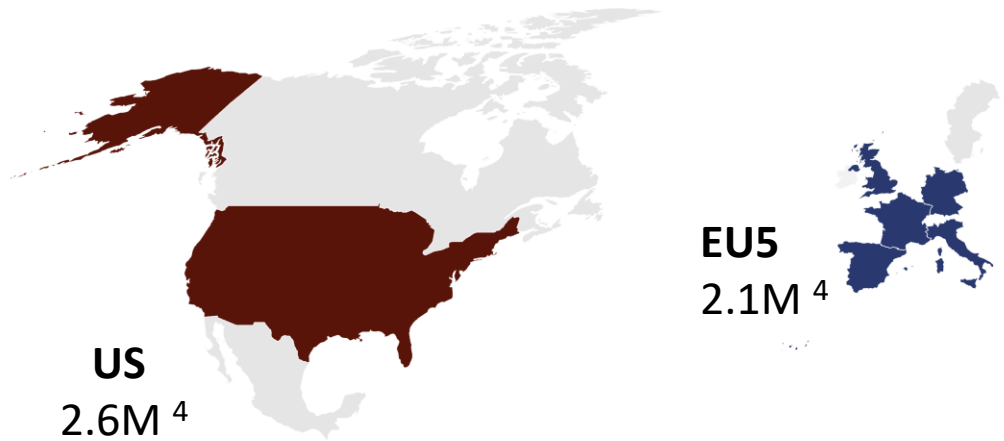
Alopecia areata (AA) is a common autoimmune disease characterised by complete or partial hair loss <sup>1</sup>



AA has profound negative impact on quality-of-life scores, similar or worse than other dermatologic diseases <sup>2,3</sup>



Total diagnosed lifetime prevalence AA cases



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

1. Zhou et al (2021) Clin Rev All Imm 61:403-423  
2. Liu et al (2018) JAAD 79(3):556-558

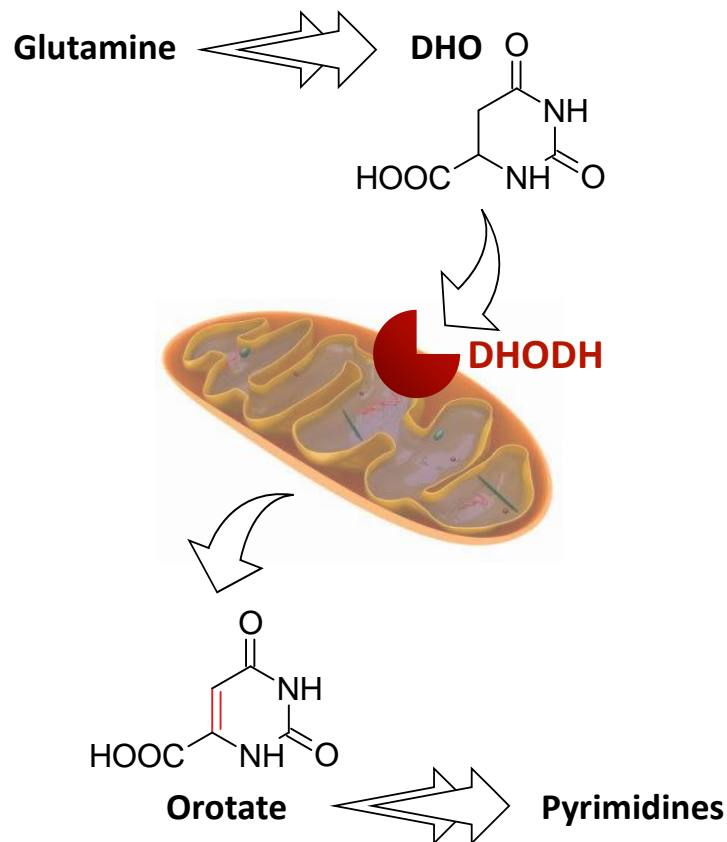
3. Lundberg et al (2000) Acta Derm Venereol 80(6):430-434  
4. DRG Alopecia Areata Disease Landscape and Forecast report 2023

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 159(4):411-418



# DHODH is a validated target for autoimmune disease

## The *de novo* pathway

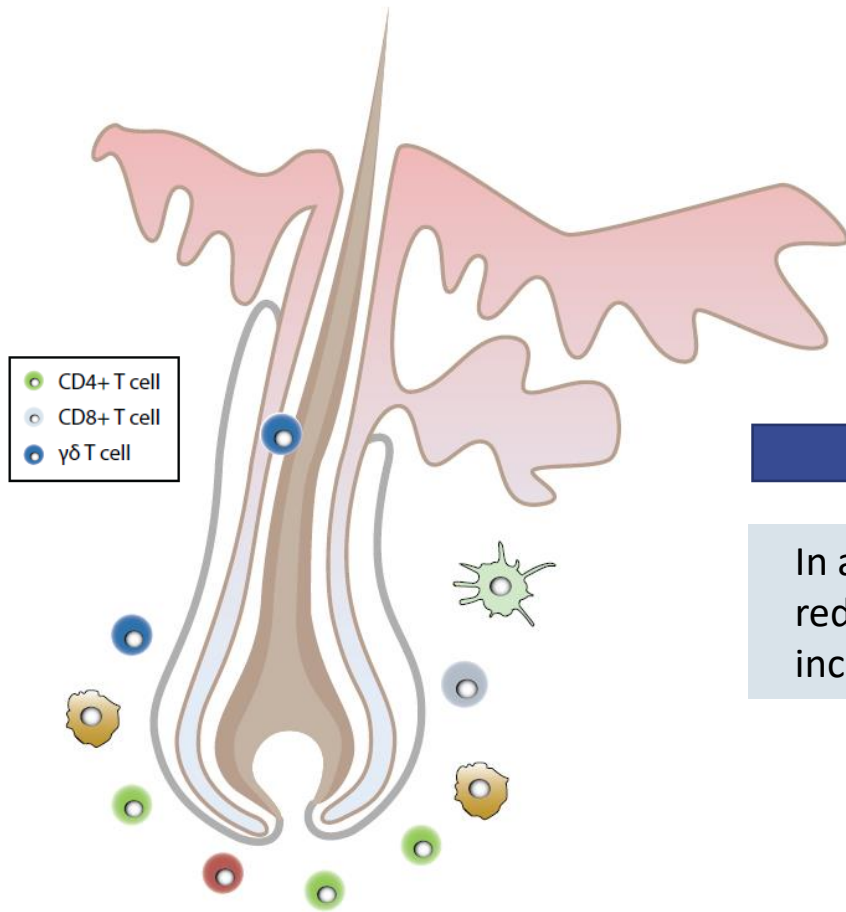


- DHODH inhibition will block *de novo* pathway of pyrimidines, impacting rapidly dividing cells eg T cells during autoimmune triggers
- Other cells can use salvage pathways to make pyrimidines
- DHODH inhibitors are approved in multiple sclerosis and rheumatoid arthritis
- However, first-generation 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

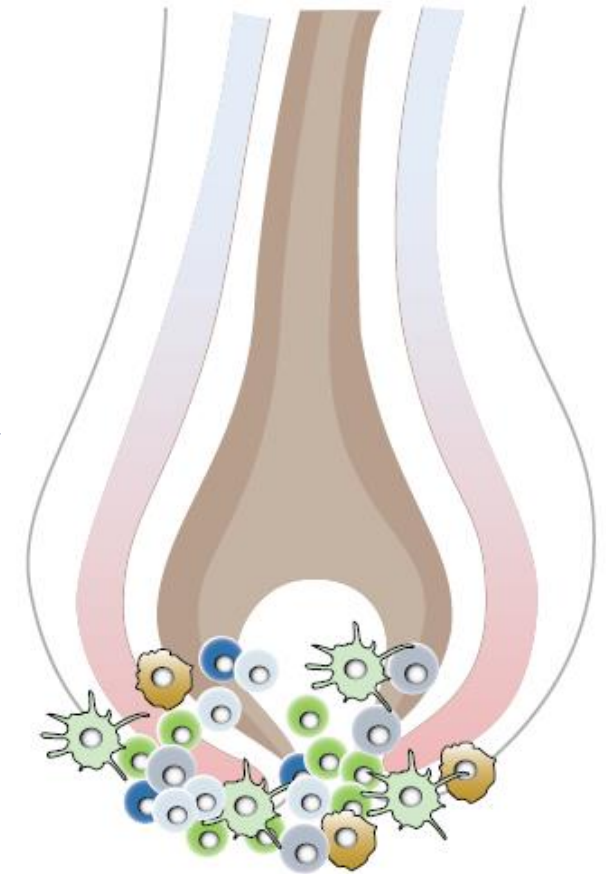


# Farudodstat's mechanism of action inhibits key processes in AA

Healthy hair follicle



Alopecia areata affected hair follicle



*farudodstat*














T cell activation and cytokine IFN $\gamma$  production

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		Olumiant ( <i>baricitinib</i> )	Approved
		Litfulo ( <i>ritlecitinib</i> )	Approved
		<i>Deuruxolitinib</i>	Phase 3
		<i>Deucravacitinib</i>	Phase 2
		SHR0302	Phase 2
S1P Inhibitor		<i>Etrasimod</i>	Phase 2
IL2/9/15 inhibitor		EQ101 (exBNZ-1)	Phase 2 open label
Anti-ILT7		<i>Daxdilimab</i>	Phase 2 open label
IL-2		Rezpegaldesleukin	Phase 2a
IL-7R $\alpha$ antagonist		ADX-914	Phase 2a PoC
OX40/CD134		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.

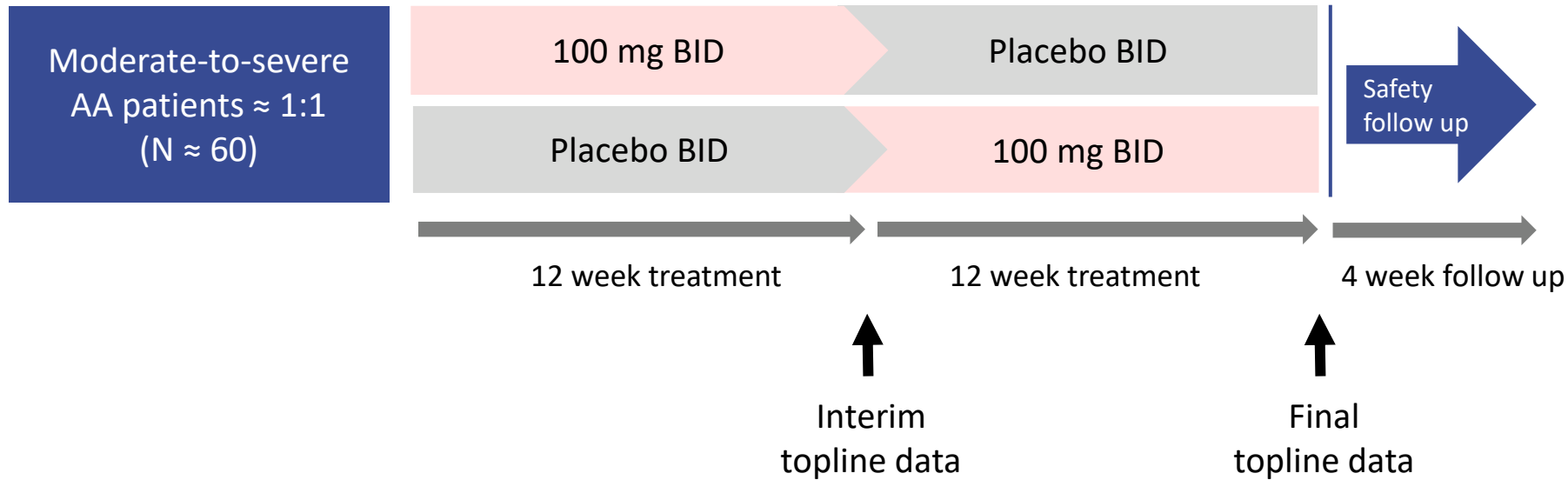
- Increased risk of serious bacterial, fungal, viral and opportunistic infections leading to hospitalization or death, including tuberculosis (TB). Interrupt treatment with OLUMIANT if serious infection occurs until the infection is controlled. OLUMIANT should not be given to patients with active tuberculosis. 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 mid-2024



Primary efficacy endpoint: % change from baseline in SALT score

Select inclusion criteria:

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



# Upcoming milestones



# Multiple upcoming catalysts over the next 12 months

Ticker	NASDAQ: ASLN
Net operating cash used	\$ 13.7M (3Q 2023)
Cash balance	\$ 40.8M as of September 30, 2023 <sup>1</sup> \$ 5.0M registered direct offering closed March 14, 2024
Upcoming milestones expected in 2024	<ul style="list-style-type: none"><li>• <i>Eblasakimab</i> topline readout from TREK-DX trial end 2024</li><li>• Partnership selection to advance <i>eblasakimab</i> into Phase 3</li><li>• <i>Farudodstat</i> Phase 2a interim topline data mid-2024</li><li>• Publication and presentation of further data from the TREK-AD study of <i>eblasakimab</i> and on <i>farudodstat</i> at major conferences</li></ul>

<sup>1</sup> \$12M prepayment to K2 in December 2023, further payments deferred to 2025 – reduces total cash burn in 2024

