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Three Unique Dual-Pathway Biologics, Clinically Validated for Therapeutic Areas with Unmet Needs

October 2024

Nasdaq Ticker: ZURA

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Company Summary

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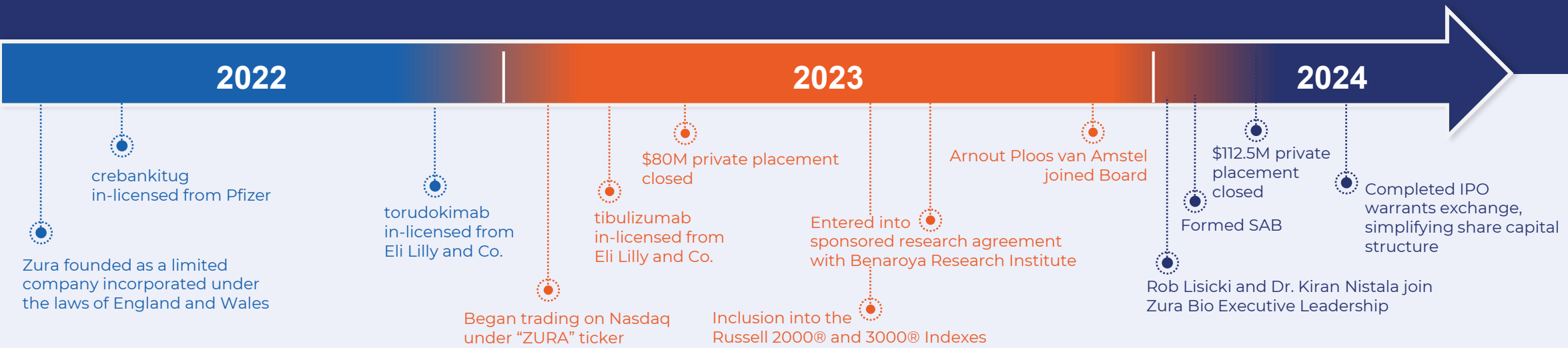
High-Potential Biologics: Three novel, clinically validated* dual-pathway biologics, each targeting multi-billion-dollar markets, advancing towards Phase 2 trials

Lead Asset Development: Tibulizumab Phase 2 study for SSc expected to commence in 4Q 2024, with a subsequent trial for HS anticipated in 2Q 2025

Strategic Milestones: Anticipating 2 key internal catalysts and up to 11 external readouts over the next 36 months, with potential to significantly drive value creation

Proven Leadership: An experienced team with a demonstrated history of driving over $8 billion in mergers and acquisitions within the last three years, showcasing their ability to execute strategic growth and value creation

Financial Strength: Cash runway to support operations as currently planned through 2027



(*) Studies conducted by Pfizer and Eli Lilly & Co.
Sources: Zura Bio Press Releases and Filings
Acronyms: HS, hidradenitis suppurativa; Q, quarter; SAB, scientific advisory board; SSc, systemic sclerosis

Pipeline of Novel Dual-Pathway Biology Designed to Meaningfully Advance Outcomes in I&I Diseases

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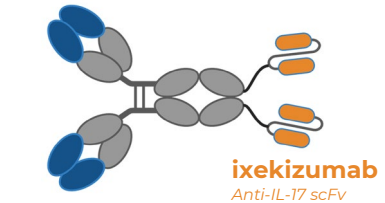
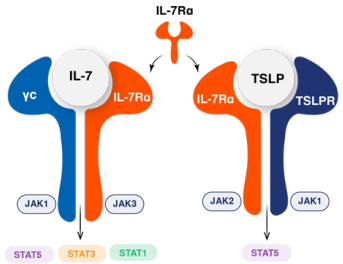
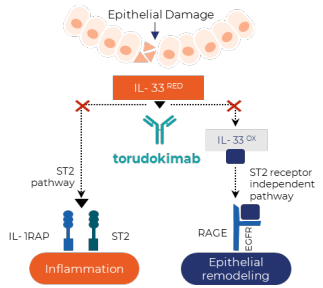
		Phase 1	Phase 2
tibulizumab ***Pipeline-in a-product***	 **Only bispecific antibody targeting IL-17A and BAFF**	Tibulizumab has been studied in three Phase 1/1b clinical studies to date * ✓ 78 participants dosed across 3 studies – n=57 single dose – n=21 multiple doses up to 12 weeks	Planning to initiate Phase 2 studies of tibulizumab for SSc in 4Q 2024 and for HS in 2Q 2025.
crebankitug ***Potential best-in-class***	 **IL-7R and TSLP Inhibition with potential best in class TSLP inhibition**	Crebankitug has been studied in three Phase 1/1b clinical studies to date ** ✓ 93 participants dosed – n=60 single dose – n=33 multiple doses up to 12 weeks	Actively assessing the competitive landscape and evaluating potential therapeutic indications to guide our future development efforts.
torudokimab ***Potential best-in-class***	 **Best in class in inhibiting GM-CSF production by human mast cells**	Torudokimab has been studied in three Phase 1/2 clinical studies to date *** ✓ 244 participants dosed – n= 81 single dose – n=163 multiple doses up to 52 weeks	Actively assessing the competitive landscape and evaluating potential therapeutic indications to guide our future development efforts.

(*) Phase 1/1b studies conducted by Eli Lilly & Co. (Phase 1 SAD in participants with rheumatoid arthritis, Phase 1b MAD in participants with Sjogren's syndrome, Phase 1 SAD in healthy Japanese and Caucasian participants)
(**) Phase 1/1b studies conducted by Pfizer. (Phase 1a SAD in healthy adult volunteers, Phase 1b MAD in participants with type-1 diabetes, Phase 1b MAD in participants with multiple sclerosis)
(***) Phase 1/2 studies conducted by Eli Lilly & Co. (Phase 1 SAD, MAD and Safety / PK in in healthy participants, Phase 2 in participants with atopic dermatitis)
Sources: Clinical Study Reports
Acronyms: BAFF, B cell-activating factor; GM-CSF, granulocyte-macrophage colony-stimulating factor; HS, hidradenitis suppurativa; I&I, inflammation and immunology; IL, interleukin; Q, quarter; SSc, systemic sclerosis; TSLP, thymic stromal lymphopoietin

Dual-Biology Pathways Present Opportunities for High-Impact Therapeutic Targets with Attractive Market Potential

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Prevalence[1] U.S., EU5* and Japan	Potential Therapeutic Area	TAM ($USD) U.S., EU5* and Japan
270 k	**SSc** *systemic sclerosis*	$2,500 M
700 k	**HS** *hidradenitis suppurativa*	$3,200 M
7 M	**AA** *alopecia areata*	$5,200 M
32 M	**COPD** *chronic obstructive pulmonary disease*	$23,692 M
25 M	**AD** *atopic dermatitis*	$31,000 M
47 M	**Asthma**	$25,247 M
~112 M		**$90,839 M**

(*) Germany, France, Italy, Spain, and United Kingdom

Sources: [1] Clarivate/DRG. Accessed 19 August 2024. Projected Prevalence and TAM 2032; "Alopecia Areata - National Alopecia Areata Foundation." NAAF, www.naaf.org/alopecia-areata; Internal Analysis; Evaluate Pharma

Acronyms: k, kilo-thousand; M, million; TAM, total addressable market

Zura is Led by a Strong Leadership Team with a Proven Track Record in Drug and Business Development

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ROBERT LISICKI
Chief Executive Officer
and Director

VERENDER BADIAL
Chief Financial Officer

KIRAN NISTALA MBBS, PhD
Chief Medical Officer and
Head of Development

GARY WHALE PhD
Chief Technology Officer

KIM DAVIS JD
Chief Legal Officer and
Corporate Secretary

Key Anticipated Events Through 2026

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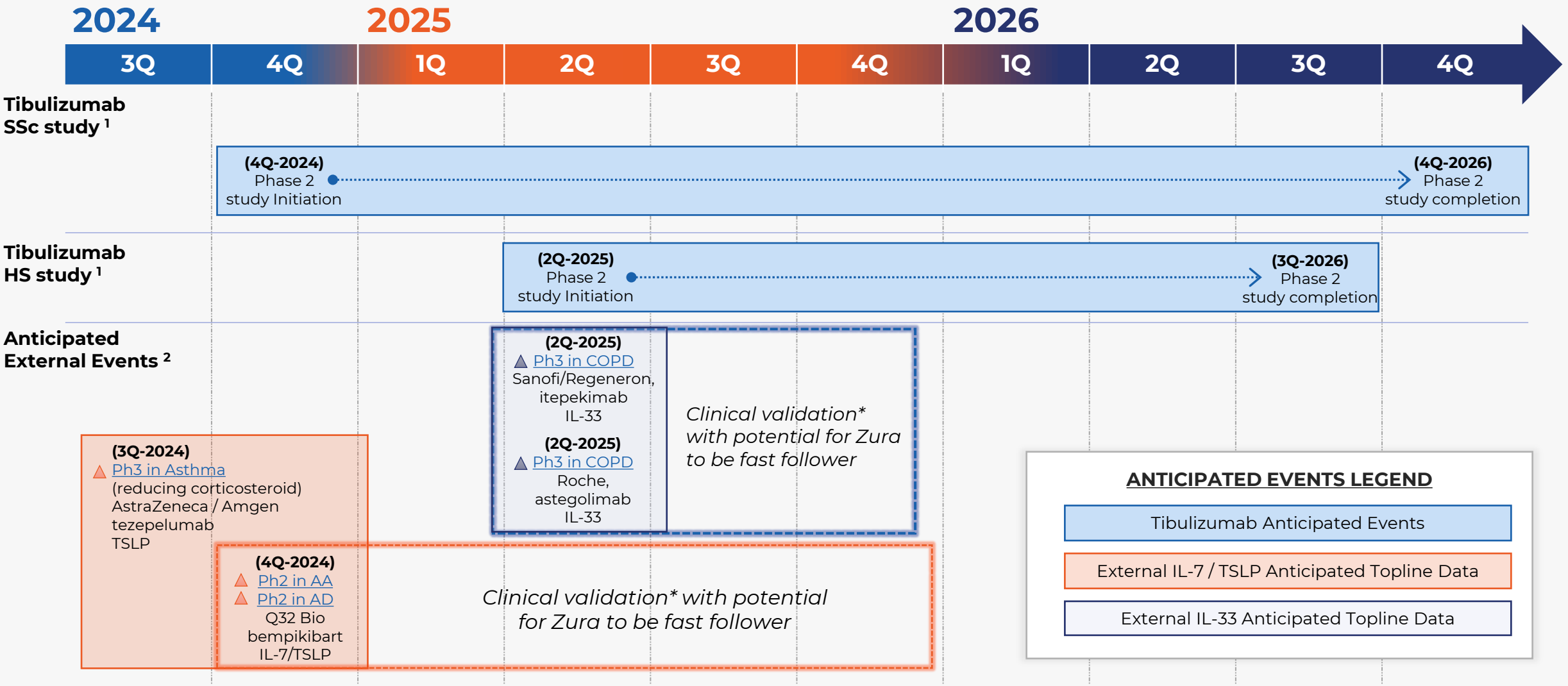

(*) Other IL-7Rα clinical data sources that may be available include a Phase 2 trial of lusvertikimab in ulcerative colitis.

Sources: [1] Zura Planning Assumptions; [2] clinicaltrials.gov; Company Presentations
Acronyms: AA, alopecia areata; AD, atopic dermatitis; COPD, chronic obstructive pulmonary disease; HS, hidradenitis suppurativa; IL, interleukin; Q, quarter; SSc, systemic sclerosis; TSLP, thymic stromal lymphopoietin

tibulizumab

ZB-106

Anti-BAFF + IL-17

Tibulizumab, is a humanized bispecific dual antagonist antibody, and has been engineered to bind to and neutralize both BAFF and IL-17A. Our approach with tibulizumab is to inhibit both pathways with a single agent, potentially providing clinical benefits to a broader range of patients, as well as a greater level of effect.

systemic sclerosis (SSc)

Rationale for Phase 2 Study in Systemic Sclerosis Patients

TAM projected to exceed $2B

Increased Probability of Success Through Scientific and Clinical Validation

- IL-17 and BAFF are both validated as key contributors to SSc progression
- Multiple clinical studies demonstrate that inhibiting IL-17 and targeting B cells benefits SSc treatment
- Simultaneously inhibiting these validated targets may improve outcomes

High Unmet Need and Significant Value

- Rare and life-threatening condition with a 40%-60% mortality rate within 10 years
- Limited treatment options available, with no advanced-line agents approved specifically for SSc
- 96% of rheumatologists identify SSc as the highest area of patient need [1]

Sources: [1] Spherix Global Insights: Market Dynamix: Systemic Sclerosis (US) 2024
Acronyms: B, billion; BAFF, B cell-activating factor; IL, interleukin; SSc, systemic sclerosis; TAM, total addressable market

Systemic Sclerosis is a Multi-Organ Disease with No Effective Treatments

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Systemic sclerosis is a rare & life-threatening disease

~300,000

people with SSc in US, EU and Japan [1]

Zero

SSc-specific * drugs approved

(*) no effective treatment exists that combats the disease across organ systems

Sources: [1] Clarivate/DRG. Accessed 19 August 2024. Projected Prevalence 2032.

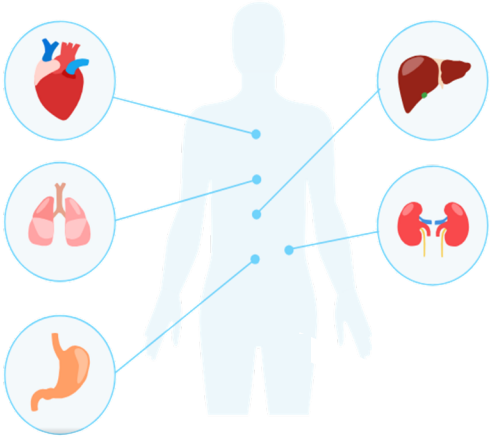
Systemic sclerosis is characterized by *tissue inflammation* and *fibrosis*

Lung

*Two disease-modifying drugs are approved for severe lung complications of the disease (SSc-ILD)

Skin

Other Organs



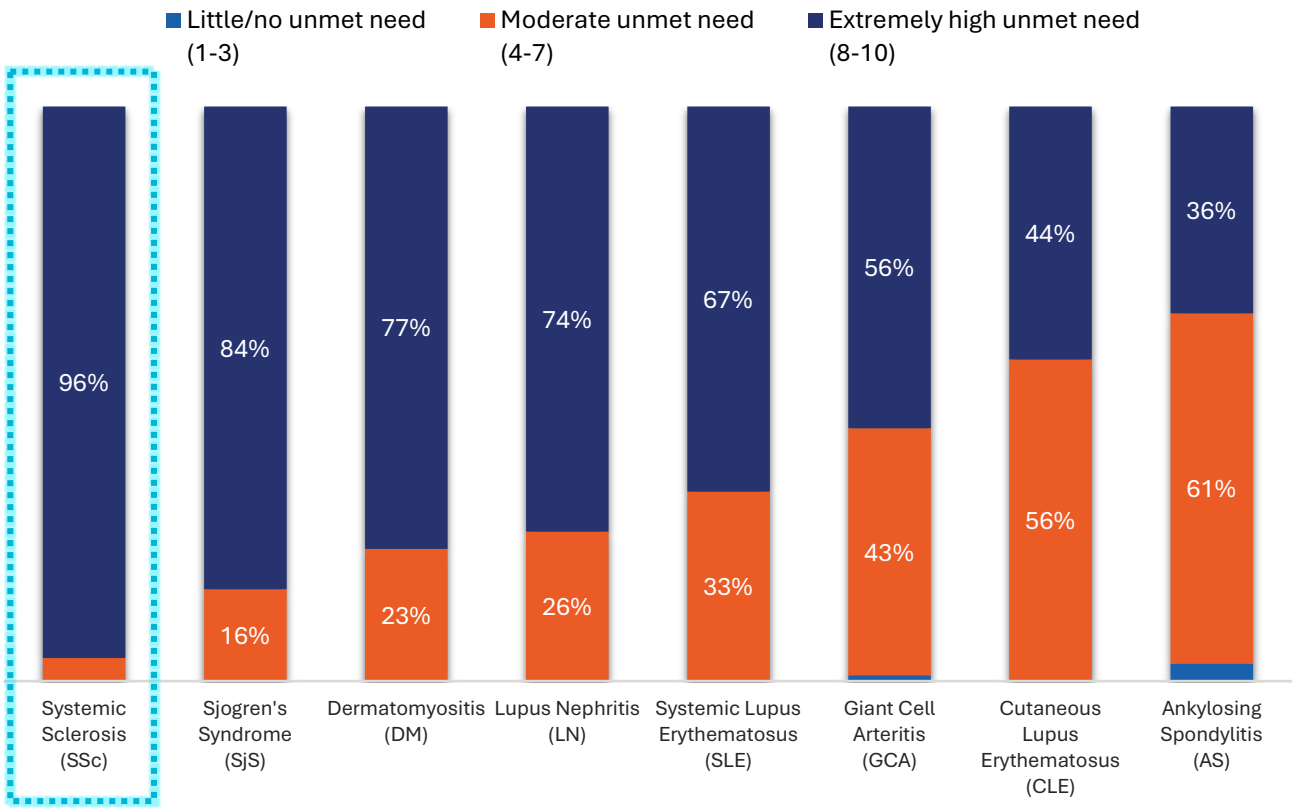
Multiple areas for evaluation and improvement in SSc

FVC	mRSS	HAQ-DI	PtGA	CGA
Forced Vital Capacity	modified Rodnan Skin Score	Health Assessment Questionnaire Disability Index	Patient Global Assessment	Clinical Global Assessment

Rheumatologists Rank SSc as the Highest Area of Need, with Significant Opportunity for Greater and Broader Clinical Benefit

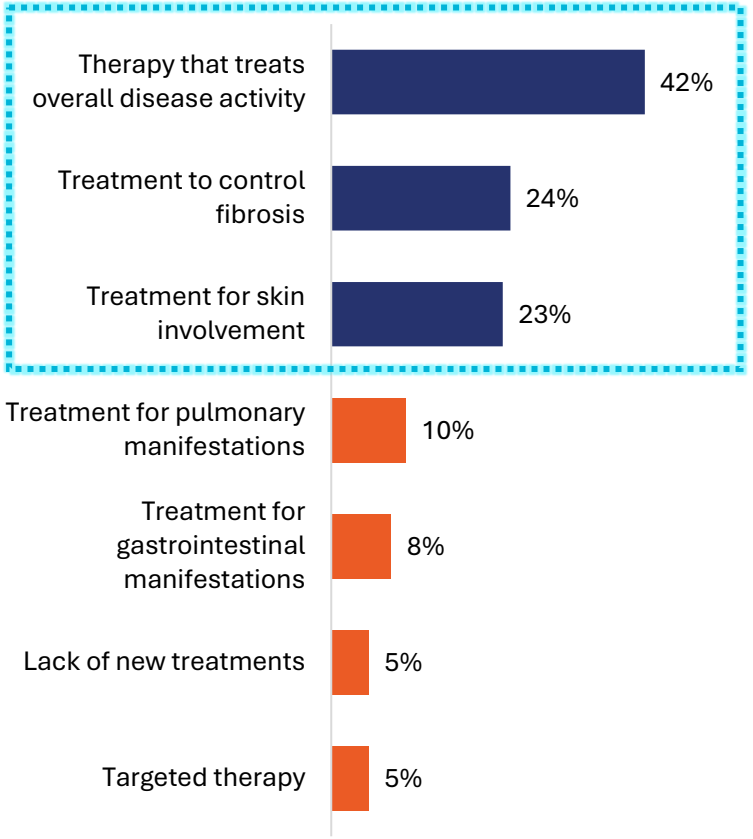
Unmet Need for New Pharmacological Treatment Options

% of respondents



Unmet Need for the Treatment of SSc

Coded, open-end responses



96% of respondents highlight a pressing need for new pharmacological treatments in SSc

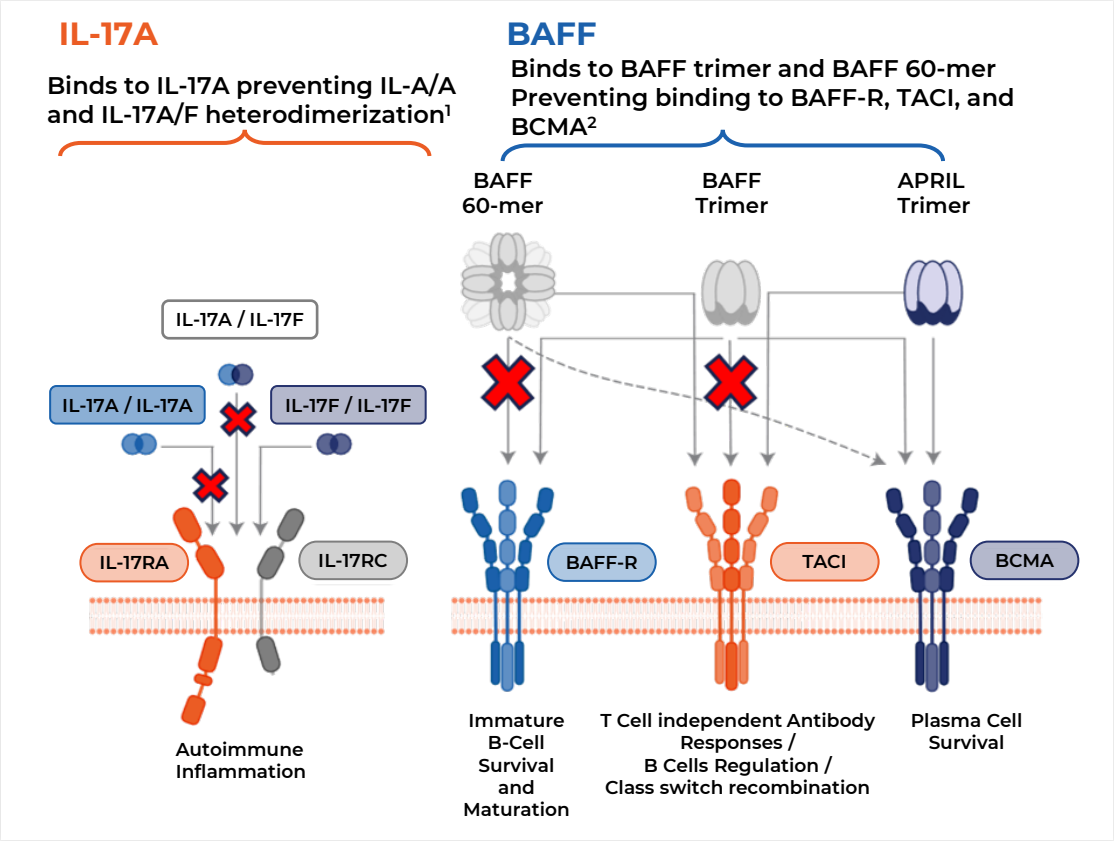
Source: Spherix Global Insights: Market Dynamix: Systemic Sclerosis (US) 2024

Both IL-17A and BAFF-Mediated Inflammation Contribute to SSc Progression

Selective antibody therapy may be insufficient to address the heterogeneity of SSc

IL-17A is a pro-inflammatory cytokine that has been identified as a key contributor to SSc progression.

- IL-17A is increased in skin lesions and peripheral blood[1,2]
- Neutralization of IL-17A protected against bleomycin induced fibrosis[3]



B cell activating factor (BAFF) is a potent B-cell activator and promotes the survival and differentiation of B-cells.

- BAFF is increased in peripheral blood and correlates with skin fibrosis and incidence of pulmonary fibrosis[4,5]
- In pre-clinical models BAFF blockade prevents skin fibrosis & autoantibody production[6,7]

Inhibiting both IL-17A and BAFF may lead to better clinical outcomes in SSc

Sources: [1] Zhou, Q., et al. The Journal of Immunology, doi:10.4049/jimmunol.1500956; [2] Yang, X., et al. Arthritis Research & Therapy, doi:10.1186/ar4430; [3] Cipolla, E., et al. The FASEB Journal, doi:10.1096/fj.201700289r; [4] Matsushita, T. Arthritis & Rheumatism, doi:10.1002/art.21526; [5] Matsushita et al. J Rheum 2007; [6] Matsushita et al. J Invest Dermatol 2007; [7] François, A., et al. Journal of Autoimmunity, doi:10.1016/j.jaut.2014.08.003.

Tibulizumab Was 'designed and engineered' to Enable Engagement with IL-17A, BAFF, or *Both Simultaneously*

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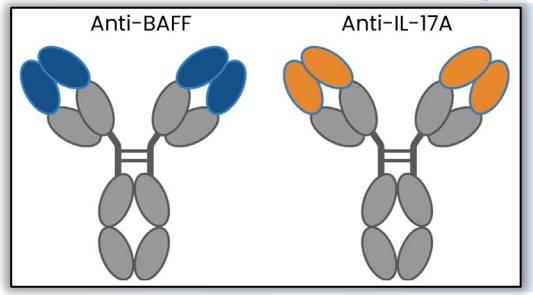
Monoclonal Antibody

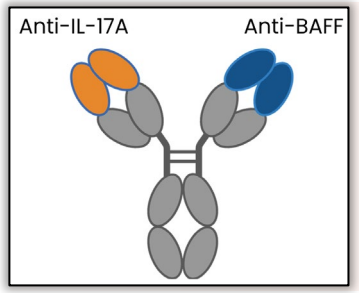


Targets a single, selective molecule in the body

May not address disease heterogeneity

1st Generation

Bispecific Antibody

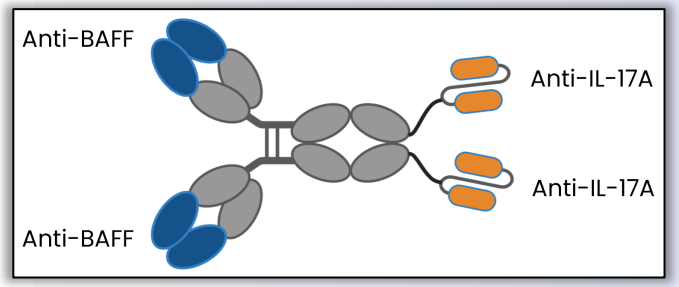


Binds two different targets simultaneously

Activation may require dual engagement

2nd Generation

Dual Antagonist Bispecific Antibody



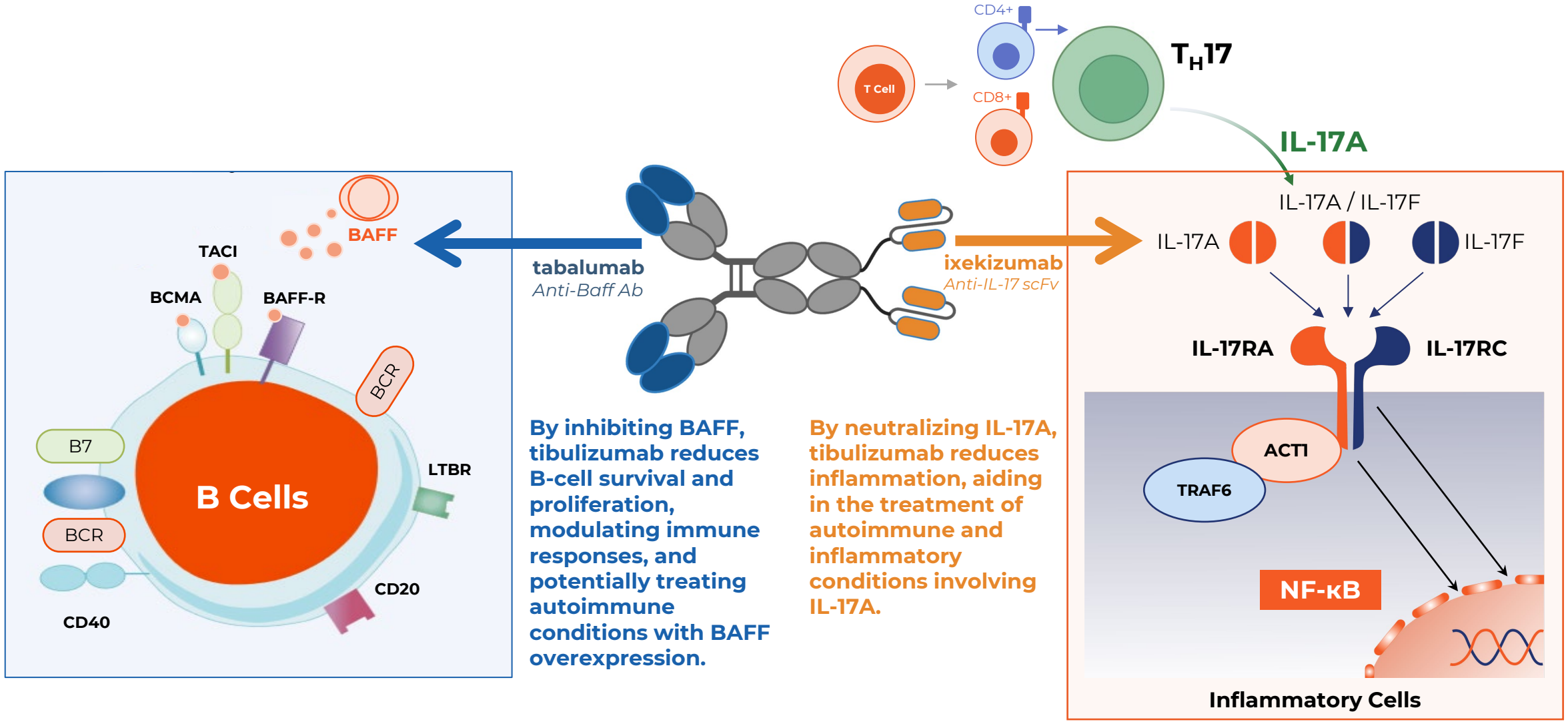
Functions as a single selective agent or two independent agents

Blocks two distinct ligands, potentially enhancing disease suppression

NEXT GENERATION

Tibulizumab Targets IL-17A, BAFF, or Both Simultaneously

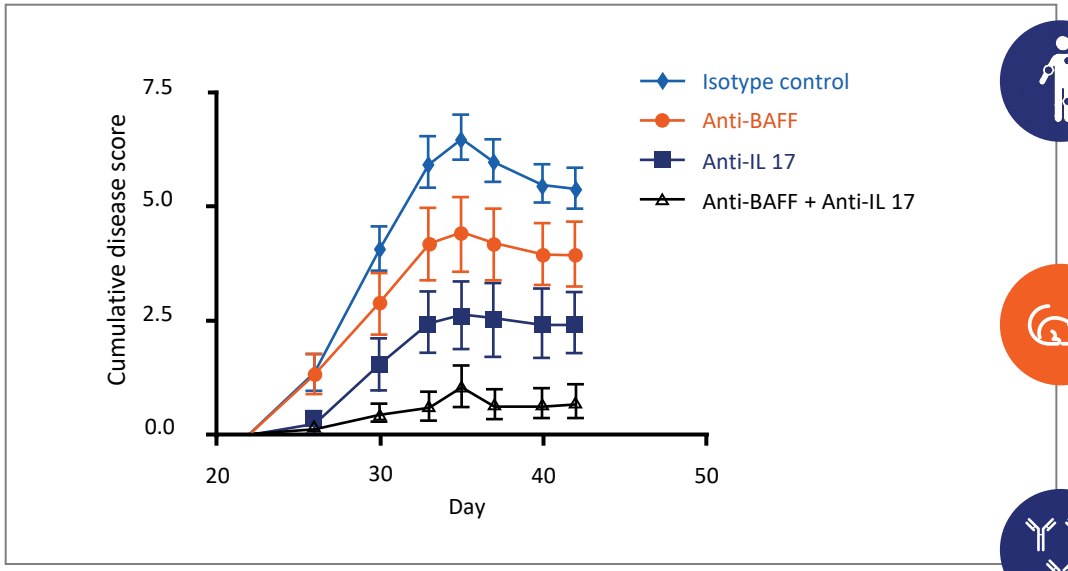
Tibulizumab uniquely targets BAFF for B cell regulation and IL-17A for inflammation control, offering a comprehensive approach to treating autoimmune and inflammatory diseases.

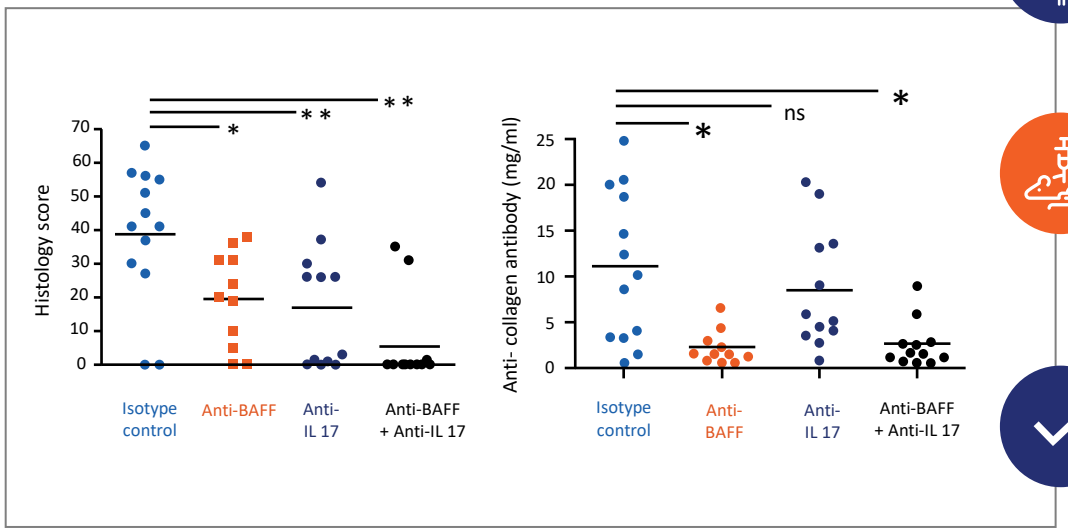


By inhibiting BAFF, tibulizumab reduces B-cell survival and proliferation, modulating immune responses, and potentially treating autoimmune conditions with BAFF overexpression.

By neutralizing IL-17A, tibulizumab reduces inflammation, aiding in the treatment of autoimmune and inflammatory conditions involving IL-17A.

Pre-Clinical Evidence of Additive Benefit from Inhibiting IL-17A and Neutralizing BAFF





Rheumatoid arthritis is a prototypic autoimmune disease where individually targeting **IL-17A-mediated inflammation or depleting B cells** has been clinically validated

The collagen-induced arthritis (CIA) murine model is similarly characterized by **increased IL-17A production and B cells** that drive disease pathogenesis

Surrogate murine antibodies were used to evaluate whether **neutralization of IL-17A and BAFF** was superior to targeting individual pathways

Mice were injected with **anti-IL-17A and/or anti-BAFF** on days 22, 29, and 36

Blockade of both IL-17A and BAFF was associated with reduced:

- **Disease severity**
- **Anti-collagen antibodies**
- **Inflammation in the hind paw (histology score)**

Sources: Zura Internal Data, Investigational New Drug (IND) Briefing
Acronyms: BAFF, B cell-activating factor; IL, interleukin

Separately Inhibiting IL-17A or BAFF Has Demonstrated Efficacy in SSc Placebo-Controlled Studies

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IL-17 receptor antagonist – Phase 3

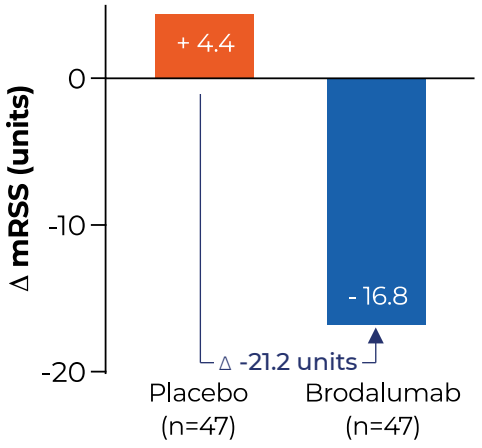
Brodalumab

- Achieved primary and endpoints for skin (reduced mRSS) and lung (improved FVC), respectively. [1]
- Demonstrated therapeutic effects on lung/respiratory functions, digital ulcers, symptoms of gastroesophageal reflux disease, and QOL without noteworthy safety concerns

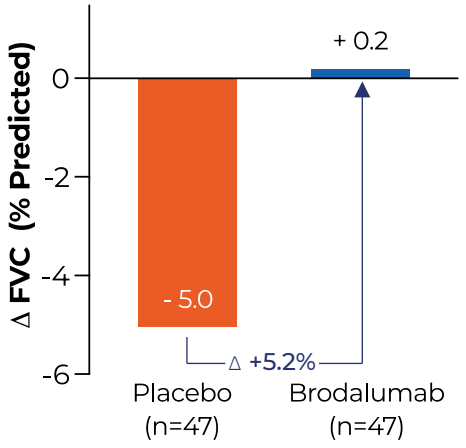
CLINICAL PRECEDENT

Phase 3 brodalumab study (24 weeks)

mRSS



FVC (% predicted)



BAFF antagonist – IIT

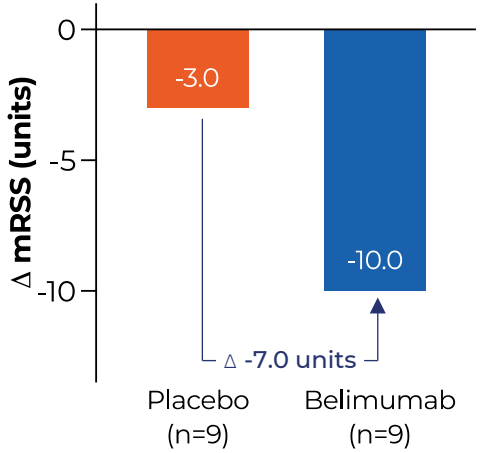
Belimumab

- A 52-week, investigator-initiated, single-center, double-blind, placebo-controlled pilot study in 20 participants with dcSSc on MMF [2]
- Both treatment groups experienced improvements in mRSS, favoring belimumab (-10 vs. -3; p = NS)
- Secondary endpoints were met with statistical significance in two endpoints: SHAQ-DI and VAS Raynaud's phenomenon

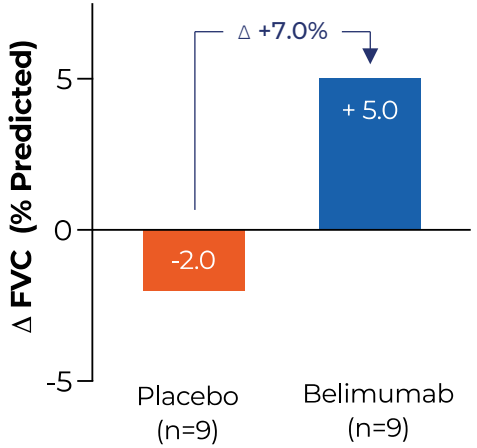
CLINICAL PRECEDENT

Phase 2 belimumab IIT study (52 weeks)

mRSS



FVC (% predicted)



Sources: [1] Fukasawa, T., et al. Annals of the Rheumatic Diseases, doi:10.1136/annrheumdis-2022-eular.2519. [2] Gordon, Jessica K., et al. Arthritis Rheumatology, doi:10.1002/art.40358.

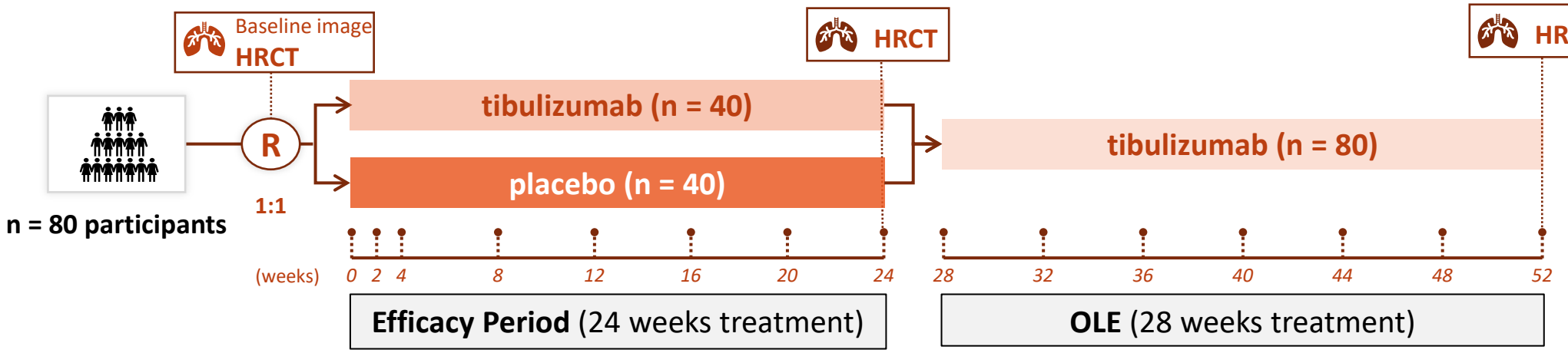
Acronyms: BAFF, B cell-activating factor; dcSSc, diffuse cutaneous systemic sclerosis; FVC, forced vital capacity; IIT, investigator-initiated trial; MMF, mycophenolate mofetil; mRSS, modified Rodnan Skin Score; QOL, quality of life; SHAQ-DI, scleroderma health assessment questionnaire – disability index; SSc, systemic sclerosis; VAS, visual analogue scale

Planned Phase 2 SSc Study* is Focused on Demonstrating Benefit in Skin and Lung Endpoints

KEY INCLUSION CRITERIA

- Early diffuse cutaneous SSc, enriched for SSc-ILD
- Stable background therapy
- Anti-centromere antibody negative
- Disease duration ≤2 years: mRSS 15-45
- Disease duration 2-5 years:
  - mRSS 20-45
  - RNA Pol III negative, or evidence of recent progression

RANDOMIZED PHASE 2 STUDY* (mRSS and HRCT)



KEY EFFICACY ENDPOINTS

mRSS (*primary*)	qHRCT / FVC	HAQ-DI (Function)	revised CRISS (rCRISS)

(*) Study design is subject to change.

Sources: Zura Internal Planning
Acronyms: CRISS, Composite Response Index in Systemic Sclerosis; FVC, forced vital capacity; HAQ-DI, health assessment questionnaire – disability index; ILD, interstitial lung disease; mRSS, modified Rodnan skin score; OLE, open-label extension; qHRCT, quantitative high-resolution computed tomography; SSc, systemic sclerosis

modified Rodnan Skin Score (mRSS): Endpoint for Assessing Skin Thickness and Fibrosis

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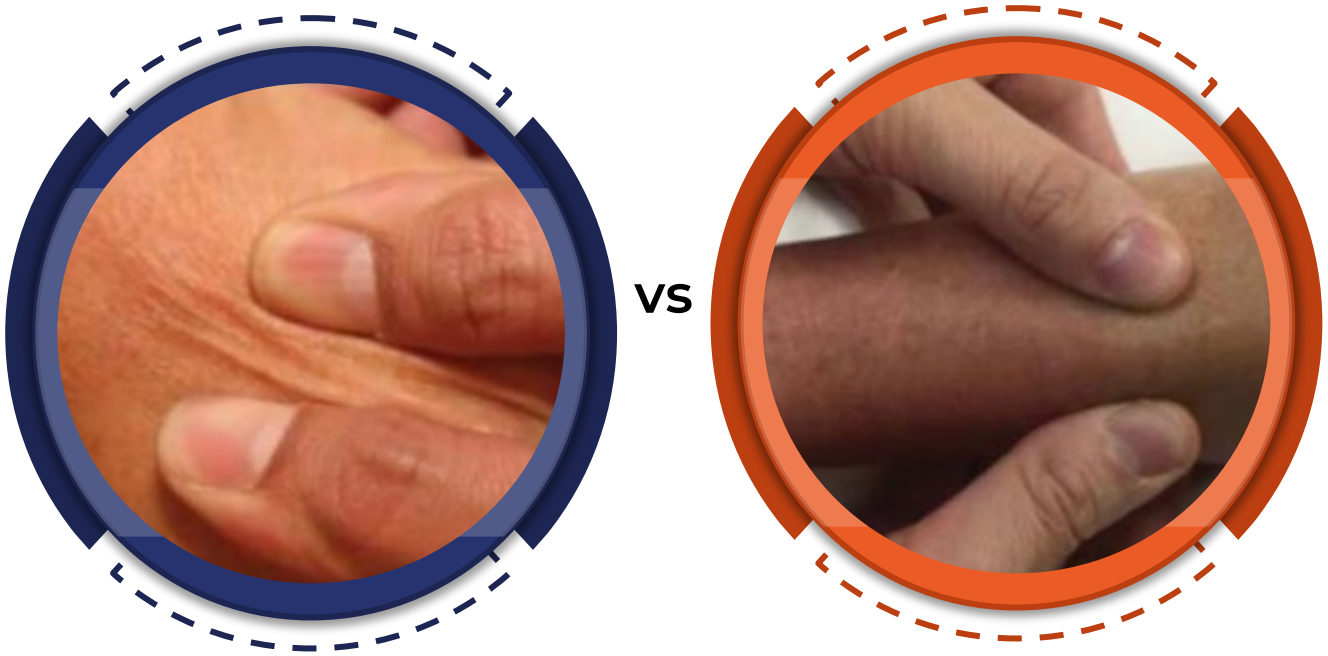


Fine Wrinkles[1] (0/3)

VS

Severe Thickness[1] (3/3)

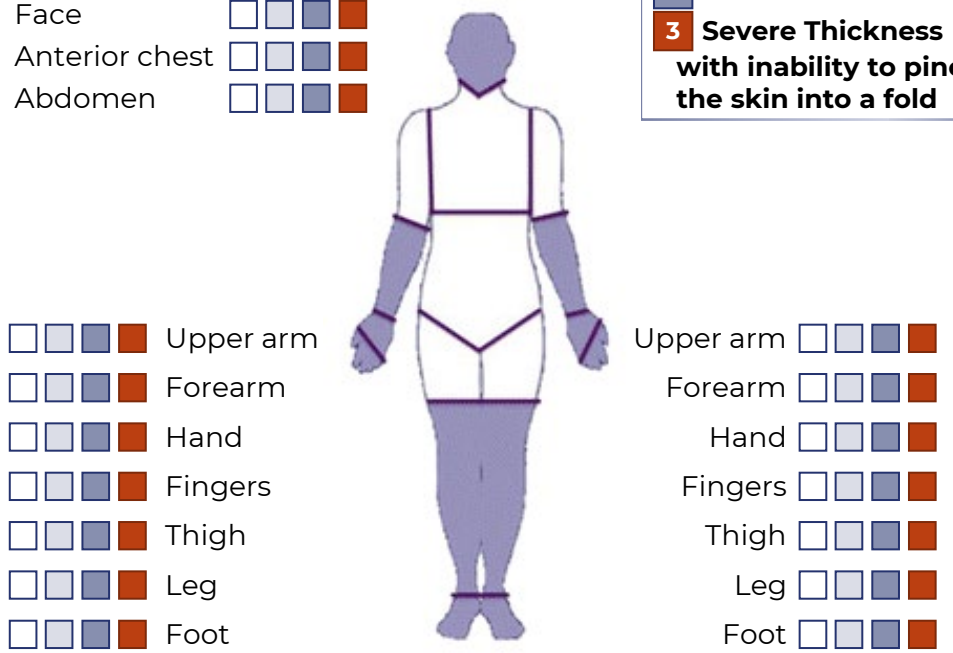
Severe skin thickening and tightening restricts movement and causes painful ulcers on the hands and fingers, significantly impairing daily activities and quality of life.

The **mRSS assesses skin thickness** in systemic sclerosis patients by **evaluating 17 body sites** (e.g., face, chest, abdomen, arms, legs). Each site is scored from 0 to 3.

The total score ranges from 0 to 51, with **higher scores indicating greater skin involvement**.

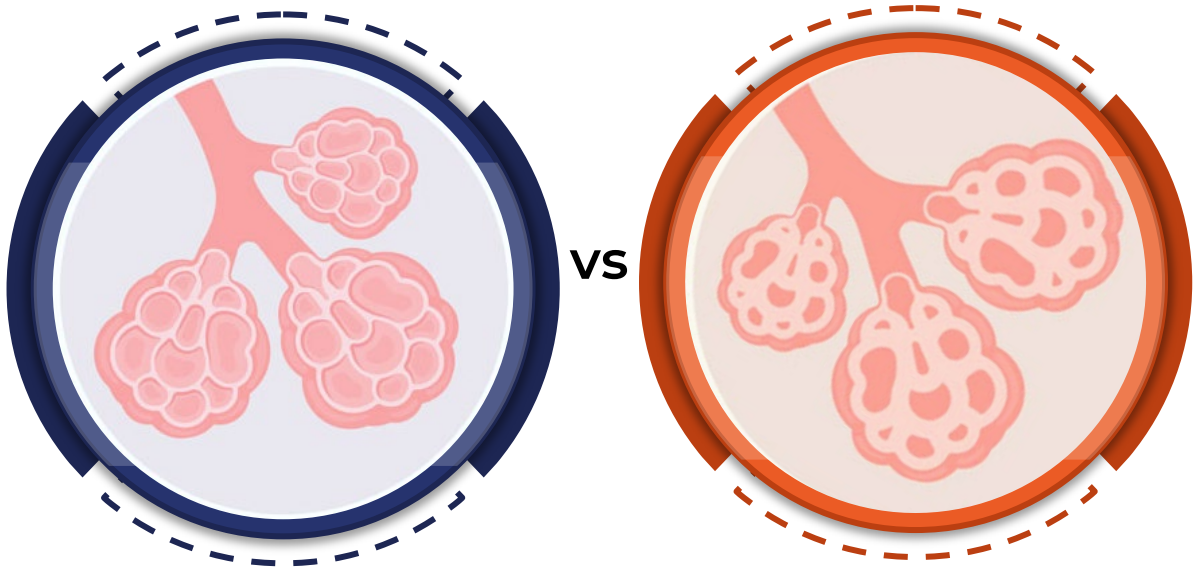
17 Surface Anatomic Areas of the Body

- 0 Normal Skin
- 1 Mild Thickness
- 2 Moderate Thickness
- 3 Severe Thickness with inability to pinch the skin into a fold



Sources: [1] Khanna, D., et al. Journal of Scleroderma and Related Disorders, doi:10.5301/jsrd.5000231; [2] Ferreli, C., et al. Clinical Reviews in Allergy & Immunology, doi:10.1007/s12016-017-8625-4.

In Phase 2, Lung Involvement using qHRCT will be a Key Secondary Endpoint



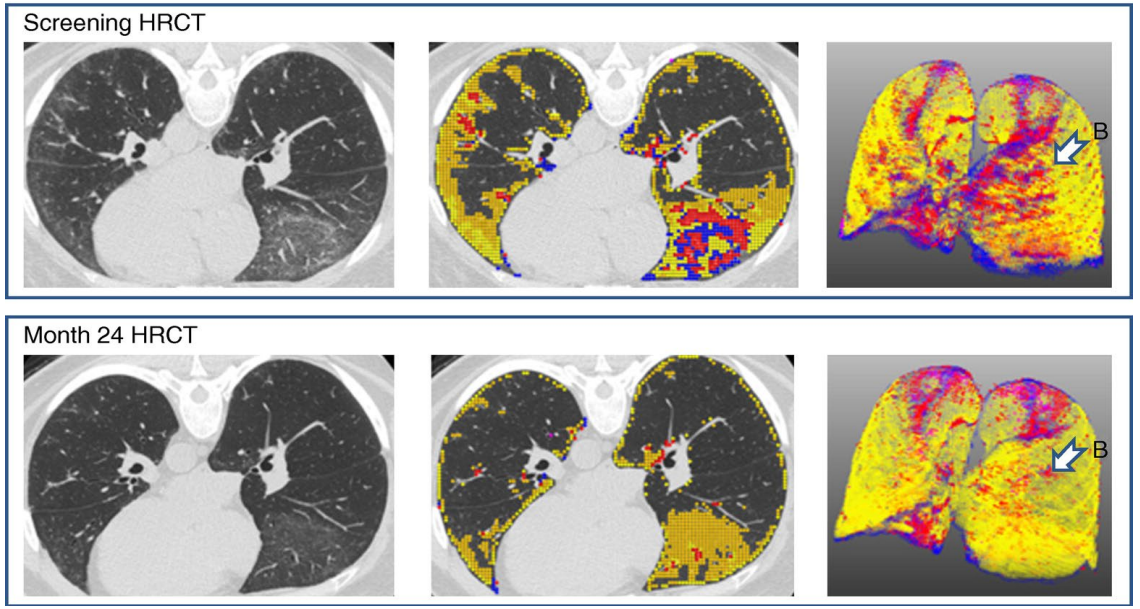
Healthy Lung **Fibrotic Lung**

ILD includes various pulmonary disorders marked by inflammation and progressive lung fibrosis, resulting in restrictive lung function and impaired gas exchange.

SSc often leads to ILD due to immune system dysregulation and subsequent lung interstitial fibrosis.

QILD by HRCT provides a sensitive measure of lung involvement, detecting changes as small as 2%.

Example of improvement after 24 months of MMF in total lung involvement



The blue and red areas show QLF, while the yellow area shows quantitative ground glass. The entire colored area represents QILD. After 24 months, QLF areas decreased (*arrow in B*).

Sources: Goldin, J.., et al. Annals of the American Thoracic Society, doi:10.1513/annalsats.201802-079oc; Zura Bio internal planning
Acronyms: ILD, interstitial lung disease; MMF, mycophenolate mofetil; qHRCT, quantitative high-resolution computed tomography; QILD, quantitative interstitial lung disease; QLF, Quantitative Lung Fibrosis; SSc, systemic sclerosis

External Development Programs*:

Key Studies and Marketed SSc-ILD Specific Products for Systemic Sclerosis

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	Phase 1	Phase 2	Phase 3	Marketed
SSc**	***RO7303509*** *(unknown)* *Genentech Roche*	***Privigen and Hizentra*** *(IgG)* *20% and 10% human IgG* *CSL Behring* ***Orencia*** *(CD80/CD86)* *abatacept* *Bristol Meyers Squibb* ***SAR100842*** *(LPA1)* *Sanofi* ***GLPG1690*** *(autotaxin)* *ziritaxestat* *Galapagos* ***IVA337*** *(PPAR)* *lanifibranor* *Inventiva* ***Adempas*** *(sGC)* *riociguat* *Bayer* ***SAR156597*** *(IL-4/IL-13)* *romilkimab* *Sanofi* ***FT-011*** *(GPCR-68)* *Certa Therapeutics* ***MT-7117*** *(MC1R)* *dersimelagon* *Mitsubishi Tanabe Pharma* ***Vasculan*** *(sGC)* *ifetroban* *Cumberland Pharmaceuticals* ***ZB-106*** *(IL-17/BAFF)* *tibulizumab* *Zura*	***Lenabasum*** *(CB_2)* *anabusum* *Corbus Pharmaceuticals* ***Saphnelo*** *(IFNAR1)* *Anifrolimab* *AstraZeneca* ***HZN-825*** *(LPAR1 antagonist)* *fipaxalparant* *Amgen/Horizon*	
SSc-ILD***		***BI 685509*** *(sGC)* *avenciguat* *Boehringer Ingelheim* ***ATYR1923*** *(neuropilin-2)* *Efzofitimod* *aTyr* ***RO7622888*** *(OSMRβ)* *vixarelimab* *Kiniksa/Genentech* ***PRA023/MK-7240*** *(TL1A)* *tulisokibart* *Prometheus Biosciences/Merck*	***Benlysta*** *(anti-BLyS)* *belimumab* *GSK* ***CONQUEST*** *(OX40 / PDE4B)* *amlitelimab / nerandomilast* *Sanofi / BI*	***Ofev/Vargatef*** *(TKI)* *nintedanib* *Boehringer Ingelheim* ***Actemra*** *(IL-6)* *tocilizumab* *Genentech Roche*

Route of Administration Legend

ORAL IV SUB-Q

RED ***discontinued study***

PURPLE ***ongoing study***

(*) As of September 2024. Does not include trials only conducted in Japan

(**) Studies on clinicaltrials.gov where the primary condition listed is systemic sclerosis (diffuse or limited cutaneous)

(***) Studies on clinicaltrials.gov with the condition listed is systemic sclerosis with interstitial lung disease

Revised CRISS Endpoint in SSc Assessment

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STEP 1: Assess for significant SSc-related events:

- New scleroderma renal crisis
- New decline in percent predicted FVC≥15% in established ILD or new percent predicted FVC below 80% predicted
- New onset of left ventricular failure requiring treatment
- New onset of pulmonary arterial hypertension requiring treatment
- Gastrointestinal dysmotility requiring enteral or parenteral nutrition
- Digital ischemia with gangrene, amputation, or hospitalization requiring treatment

If no significant SSc-related event, *proceed to Step 2*

STEP 2: Assess each core measure for improvement / worsening:

	Typical Threshold
FVC	**5%**
mRSS	**25%**
HAQ-DI	
PtGA	
CGA	

RESPONDER: Improvement in ≥2 core measures with worsening in ≤1 core measure

Sources: Zura Internal Planning

Acronyms: CGA, clinical global assessment; CRISS: composite response index in systemic sclerosis; FVC, forced vital capacity; HAQ-DI, health assessment questionnaire – disability index; ILD, interstitial lung disease; mRSS, modified Rodnan skin score; PtGA, patient global assessment

Phase 2 SSc Development Aims to Reduce Historical Risks in Therapeutic Area Development

Historic Drivers of SSc Study Failures:

- Novel and unvalidated mechanisms
- Inclusion/exclusion criteria oversights
- Challenges in balancing sample sizes for mRSS and ILD participants

Increasing Probability of Success:

- Larger study sample size increases the probability of success (mRSS)
- High-resolution CT highly correlates with FVC, improving ILD read-through
- Sufficient sample size for ILD readouts to understand potential Phase 3 effects

Acronyms: CT, computed tomography; FVC, forced vital capacity; IL, interleukin; ILD, interstitial lung disease; mRSS, modified Rodnan Skin Score

tibulizumab

ZB-106

Anti-BAFF + IL-17

Tibulizumab, is a humanized bispecific dual antagonist antibody, and has been engineered to bind to and neutralize both BAFF and IL-17A. Our approach with tibulizumab is to inhibit both pathways with a single agent, potentially providing clinical benefits to a broader range of patients, as well as a greater level of effect.

hidradenitis suppurativa (HS)

Rationale for Phase 2 Study in Hidradenitis Suppurativa Patients

Increased Probability of Success Through Scientific and Clinical Validation

- IL-17A and BAFF are clinically validated as key contributors to HS pathology
- Inhibiting IL-17A or disrupting B cells individually shows strong clinical support
- Ixekizumab shows high affinity for IL-17A and IL-17A/F
- Dual inhibition of these targets could improve patient outcomes

Unmet Need and Growth Potential

- 50% to 70% of HS patients do not achieve HiSCR75 with current treatments
- In the chronic phase, BAFF may play a more significant role, highlighting an unmet need that tibulizumab can address
- The TAM is projected to grow to $3.5B - $4B by 2030

Acronyms: BAFF, B cell-activating factor; HiSCR, Hidradenitis Suppurativa Clinical Response; HS, hidradenitis suppurativa; IL, interleukin; TAM, total addressable market

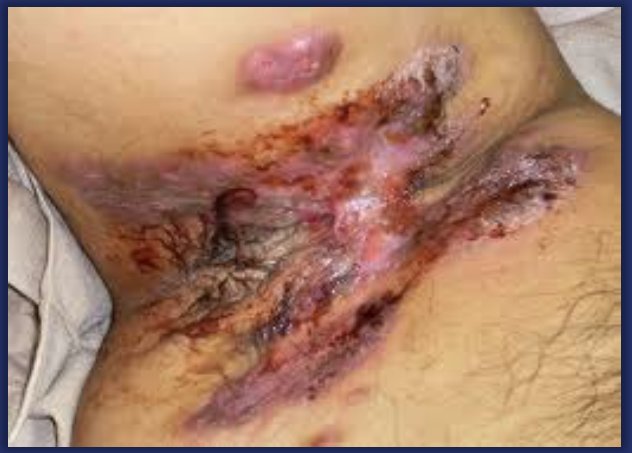
An Overview of Hidradenitis Suppurativa

DISEASE OVERVIEW

Hidradenitis suppurativa is an inflammatory follicular skin disease

Skin lesions form in the armpits, groin, and under the breasts due to inflammation and infection of the sweat glands

- ✓ Recurrent nodules and abscesses resulting in pus-like discharge
- ✓ Difficult-to-heal open wounds and scarring
- ✓ Increased Th1/Th17 and B cell mediated inflammation [1-3]
- ✓ Disproportionately affects women between adolescent age to 55 years of age [4, 5]



CLINICAL OPPORTUNITY [6]

~300K people living with HS in the U.S.

Average time to diagnosis is **7 years**

~>50% patients still left **inadequately treated**

Sources: [1] Moran, Barry, et al. Journal of Investigative Dermatology, doi:10.1016/j.jid.2017.05.033. [2] Banerjee, Anirban, et al. Immunological Investigations, doi:10.1080/08820139.2016.1230867. [3] Sabat, Robert, et al. Journal of Allergy and Clinical Immunology, doi:10.1016/j.jaci.2022.10.034. [4] Garg, Amit, et al. JAMA Dermatology, doi:10.1001/jamadermatol.2017.0201. [5] Ingram, John R. British Journal of Dermatology, doi:10.1111/bjd.19435. [6] Medical Literature, MEDACorp KOL Discussions

B Cell Signaling Potentiates HS Disease

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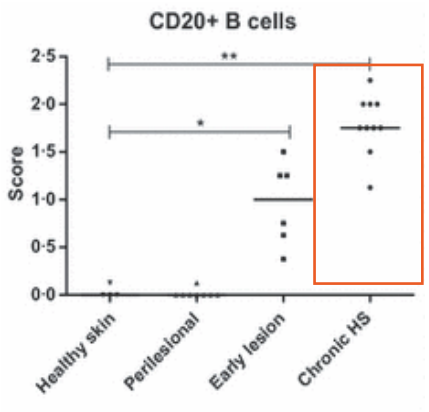
Elevated **CD20+** B cells and **CD138+** plasma cells in chronic HS lesions sustain inflammation. [2,3]

Increased BAFF in HS lesions promotes B cell activation and inflammation. [2,3]

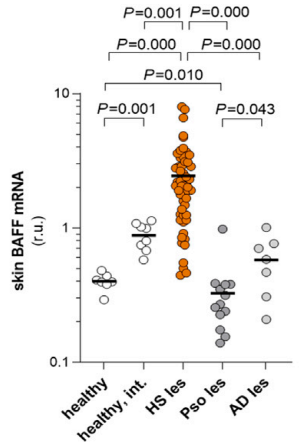
Reducing BAFF in HS lesions decreases B cell and plasma cell gene expression, indicating a potential therapeutic approach. [1,4]

Overlapping expression of **CD20+** and **BAFF**. [1,4]

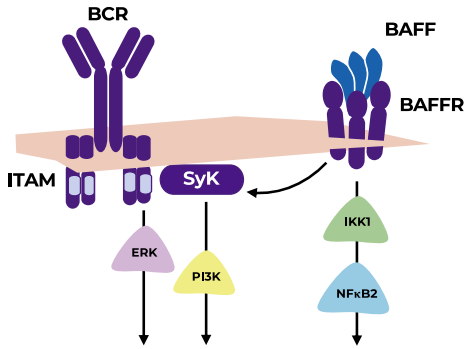
CD20+ B cells in HS Lesions



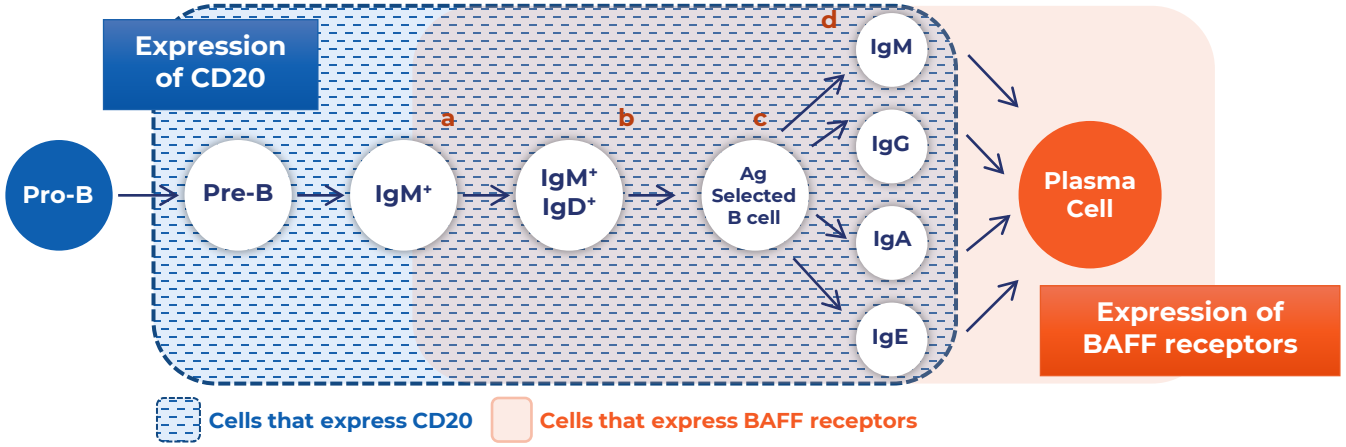
BAFF gene expression in HS



BAFF is essential for B cell activation



Overlapping expression of CD20+ and BAFF



[a] immature B cells; [b] mature B cells; [c] activated B cells; [d] memory B cells

Sources: [1] Van der Zee, H.H., et al. British Journal of Dermatology, doi:10.1111/j.1365-2133.2011.10698.x. [2] Rumberger, Beth E., et al. Inflammation Research, doi:10.1007/s00011-020-01381-7. [3] Sabat, Robert, et al. Journal of Allergy and Clinical Immunology, doi:10.1016/j.jaci.2022.10.034. [4] Gudjonsson, Johann E., et al. JCI Insight, doi:10.1172/jci.insight.139930

Role of IL-17A and B Cells Is Clinically Validated; However, Clinical Effect Remains Modest with Single-Pathway Inhibition

company *and drug INN* >>		NOVARTIS ***secukinumab***	NOVARTIS ***remibrutinib****	UCB ***bimekizumab***	MoonLake ***sonelokimab***
Mechanism		IL-17 A	BTKi	IL-17 A/F	IL-17 A/F
Administration		SC/IV	PO	SC	SC
Phase		Phase 3	Phase 2b	Phase 2	Phase 2
Dosing		30mg Q2W for 16W	100 mg or 25 mg BID	320mg Q2W for 12W	120mg Q2W for 12W
Total Patients		n = 360	n = 77	n = 88	n = 234
Efficacy (HiSCR50)	Non-Placebo Adjusted	42% - 45%	48.5% - 72.7%	63%	66%
	Placebo Adjusted	11% +	38%	35%	38%
Efficacy (HiSCR75)	Non-Placebo Adjusted	N/A	27.3% - 42.4%	50%	43%
	Placebo Adjusted	N/A	24%	29%	29%
Safety	Candidiasis	0% - 3%[1]	0	9%	10.5%

(*) There have been no head-to-head clinical trials between the product candidates listed above. Study designs and protocols for each product candidate were different, and as a result, results may not be comparable between product candidates.

Sources: Company Presentations, Publications and Research.

[1] Represents data from psoriasis trial. [2] Represents safety data from psoriatic arthritis trial remibrutinib, 2024 AAD S026.

Acronyms: BID, twice a day; BTKi, Bruton tyrosine kinase inhibitors; HiSCR, Hidradenitis Suppurativa Clinical Response; HS, hidradenitis suppurativa; IL, interleukin; INN, international non-proprietary name; IV, intravenous; PO, per os or by mouth; Q2W, every two weeks; Q4W, every four weeks; SC, subcutaneous

Tibulizumab* IL-17A Component Has Class-Leading Binding Affinity Against IL-17A and IL-17A/F

Ixekizumab is a humanized IgG4 monoclonal antibody

	IL-17A Binding Affinity	IL-17A/F Binding Affinity	IL-17F Binding Affinity
Taltz® (ixekizumab)	K_D 1.8 pM	K_D <3 pM	N/A
Bimzelx® (bimekizumab)	K_D 3.2 pM	K_D 26 pM	K_D 23 pM
Cosentyx® (secukinumab)	K_D 60 pM – 90 pM	K_D 2400 pM	N/A

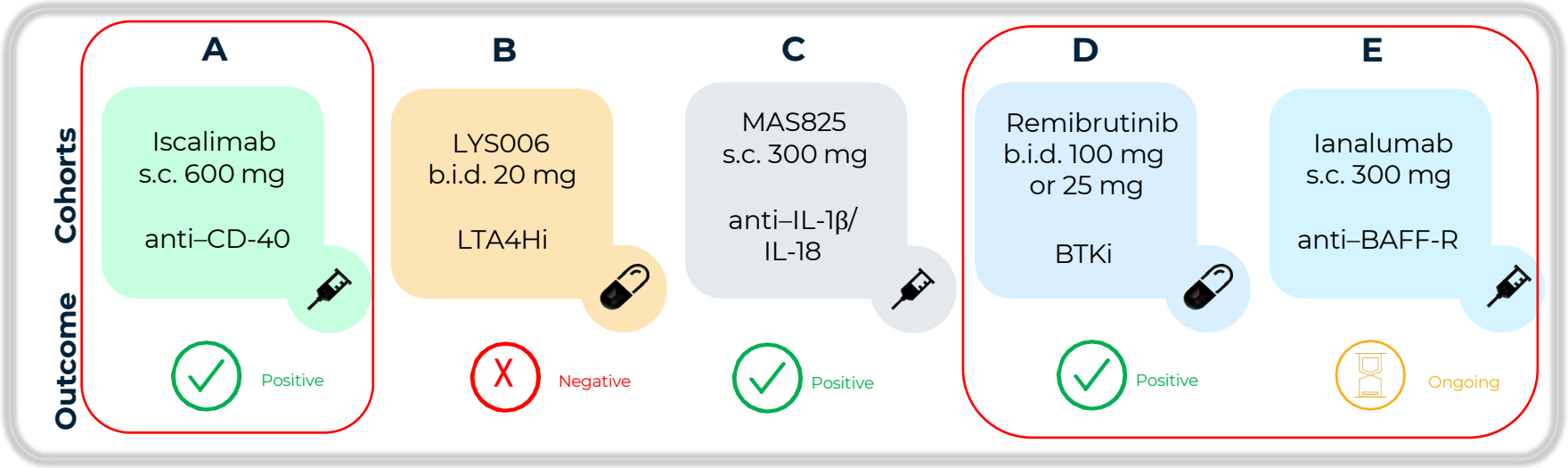
- Binding affinity, quantified by the dissociation constant (K_D), reflects the strength of interaction between a drug and its target, with a lower K_D indicating stronger binding.
- Stronger binding affinity ensures effective neutralization of the target, leading to the potential for improved clinical outcomes.

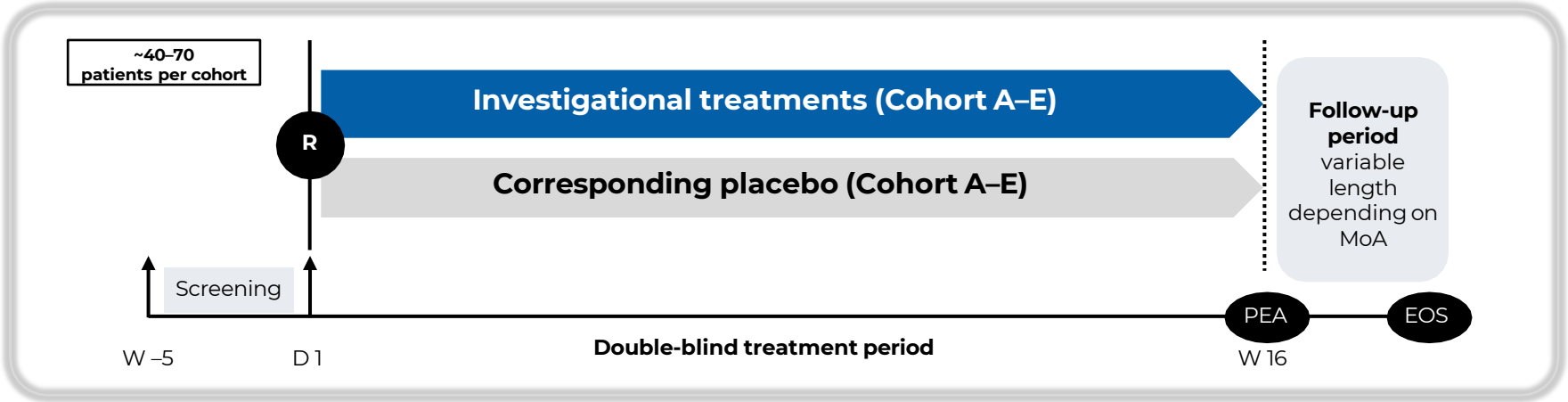
(*) Tibulizumab was engineered from Taltz® (ixekizumab)

Sources: [1]Taltz®. Prescribing Information. Lilly USA, LLC, https://taltz.lilly.com/hcp/moa-il17a-igg4 .
Acronyms: IgG4, immunoglobulin G4; K_D, dissociation constant; pM, picomolar

Ongoing Novartis Phase 2b Multicenter Platform Study Offers Additional Clinical Evidence of B Cell Targeting Benefit in HS

Presented at the American Academy of Dermatology Annual Meeting; March 8–12, 2024; San Diego, CA.





Patients

- Adult patients aged 18–65 years
- Moderate to severe HS for ≥12 months in ≥2 anatomical areas with ≤15 tunnels
- ***Cohorts A, C, and E:*** ≥5 inflammatory lesions
- ***Cohorts B and D:*** ≥3 inflammatory lesions

*Study started in February 2019 and is currently ongoing.
BAFF-R, B-cell activating factor of the tumor necrosis alpha family receptor; b.i.d., twice daily; BTKi, Bruton's tyrosine kinase inhibitor; CD, cluster of differentiation; D, day; EOS, end of study; HS, hidradenitis suppurativa; IL, interleukin; LTA4H, leukotriene A4 hydrolase; MoA, mechanism of action; PEA, primary endpoint analysis; R, randomization; s.c., subcutaneous; W, week. Clinicaltrials.gov NCT03827798. Available at: https://classic.clinicaltrials.gov/ct2/show/NCT03827798 (Accessed 6 Mar 2024).

Novartis' Interim Results Presented at '24 AAD: BTKi PBO-Adjusted Delta Aligns with Approved and In-Development Agents

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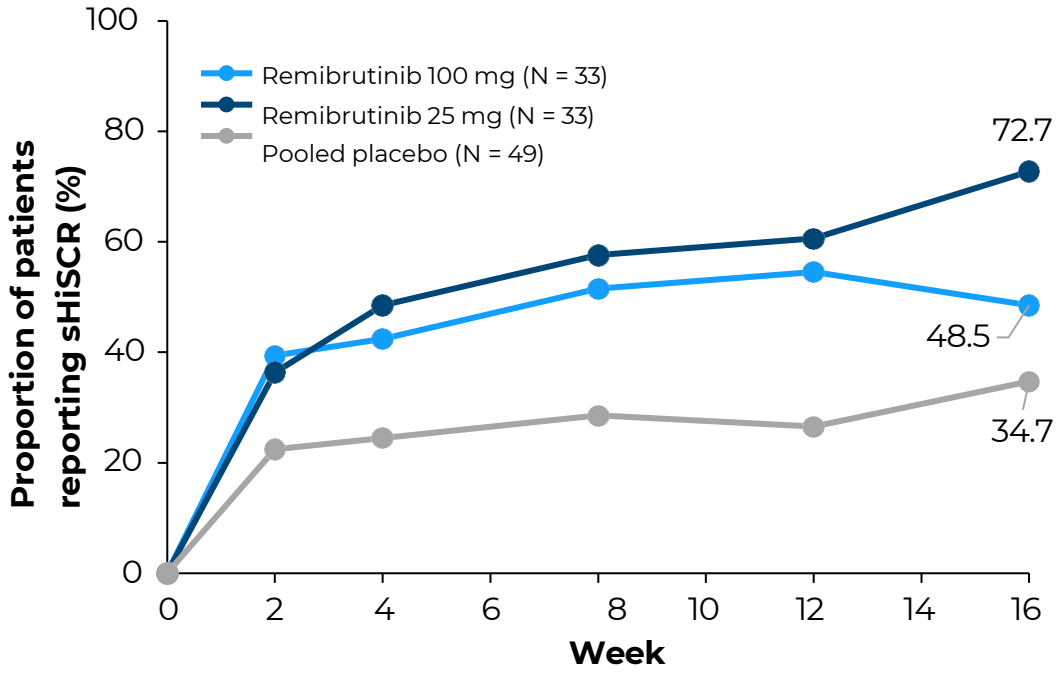
Presented at the American Academy of Dermatology Annual Meeting; March 8–12, 2024; San Diego, CA.

- The primary endpoint of this study was met for both doses of remibrutinib; patients treated with remibrutinib reported a greater rate of sHiSCR* at Week 16 compared with placebo

	Cohort D		Cohort A–D
	Remibrutinib 25 mg (N = 33)	**Remibrutinib 100 mg (N = 33)**	**Pooled Placebo (N = 49)**
Proportion of patients with sHiSCR*:			
Observed with NRI (%)	**72.7**	**48.5**	**34.7**
Difference† (%) (95% CI)	38.0 (21.1 to 55.0)	13.8 (–4.4 to 32.0)	
Bayesian estimated (%)	**72.3**	**48.5**	**34.9**
Difference† (%) (95% CI)	37.2 (19.7 to 53.0)	13.9 (–4.2 to 31.9)	
Probability of difference‡	99.9	89.6	

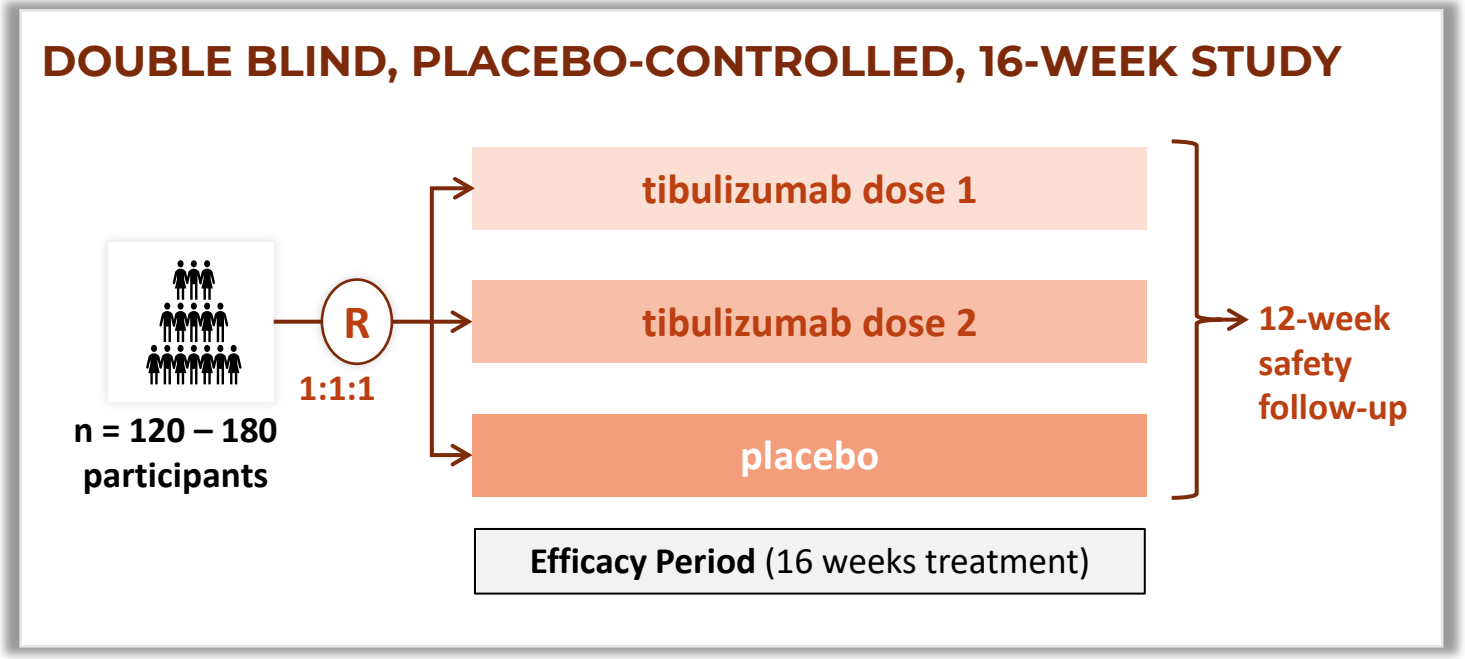


*The sHiSCR is defined as a ≥50% reduction in the abscess and inflammatory nodule count and no increase in draining tunnels compared with baseline. †Difference refers to the difference between remibrutinib (either dose) and pooled placebo at Week 16. ‡Bayesian posterior probability of remibrutinib (either dose) being better than pooled placebo.
CI, confidence interval; HiSCR, hidradenitis suppurativa clinical response; n, total number of patients with response; N, total number of patients in each treatment arm; NRI, non-responder imputation; sHiSCR, simplified hidradenitis suppurativa clinical response.

Planned Phase 2 HS Study Design*

KEY INCLUSION CRITERIA

- Moderate to Severe HS
- Hurley Stage II/III
- Total abscess and inflammatory nodule count (AN) ≥ 5
- Up to 30% TNF inadequate responders



KEY EFFICACY ENDPOINTS

- HiSCR
- AN count
- IHS4
- PGA
- DLQI
- PK / PD assessments

KEY SAFETY ENDPOINTS

- General Safety and Tolerability
- Severe infection

(*) Study design is subject to change.

Sources: Zura Internal Planning

Acronyms: DLQI, dermatology life quality index; HiSCR, Hidradenitis Suppurativa Clinical Response; HS, hidradenitis suppurativa; IHS4, international hidradenitis suppurativa severity score system; PD, pharmacodynamic; PGA, physician's global assessment; PK, pharmacokinetic; R, randomization

Tibulizumab Summary

The Phase 2 development programs for systemic sclerosis and hidradenitis suppurativa are progressing

The strategy follows validated pathways with the potential for improved outcomes, supported by related mechanism-of-action data from IL-17 and BAFF/B-cell depletion therapies

We believe tibulizumab is positioned as a potential first-in-class dual-pathway biologic

The next 6 to 12 months offer significant growth opportunities, with multiple key milestones anticipated

crebankitug

ZB-168

Anti-IL-7Rα + TSLP

Crebankitug is a high-affinity, fully human monoclonal antibody that neutralizes the IL-7 receptor alpha (IL-7Rα) chain, potentially blocking the immune pathways of IL-7 and thymic stromal lymphopoietin (TSLP).

Crebankitug

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Crebankitug, a fully human IL-7Rα antibody

- Originally developed by Pfizer
- IL-7Rα inhibition has potential to inhibit two critical immune pathways (IL-7 and TSLP)
- Potential applicability in broad range of T-cell mediated diseases and atopic diseases.

Well tolerated in Phase 1 and Phase 1b studies

- >90 participants dosed with crebankitug
- Adverse events generally mild and not treatment-related.

Phase 1b data demonstrate clear evidence of impact on key T-cell compartments

- Only anti-IL7R program that has reported safety, PK, and PD data in participants with an auto immune disease (not just healthy volunteers)
- Potentially clinically relevant changes observed in memory T-cell counts and T_{reg} : T_{memory} ratios.

Actively assessing Phase 2 strategy

- Ongoing internal planning for indications in areas with unmet needs.
- Will be evaluating Phase 2 IL-7Rα and TSLP competitor data readouts.

creban- *creating balance* **ki-** *cytokine or cytokine receptor* **tug-** unmodified immunoglobulin

Crebankitug: A Multi-Functional Antibody That Blocks Signaling via IL-7 and TSLP Pathways

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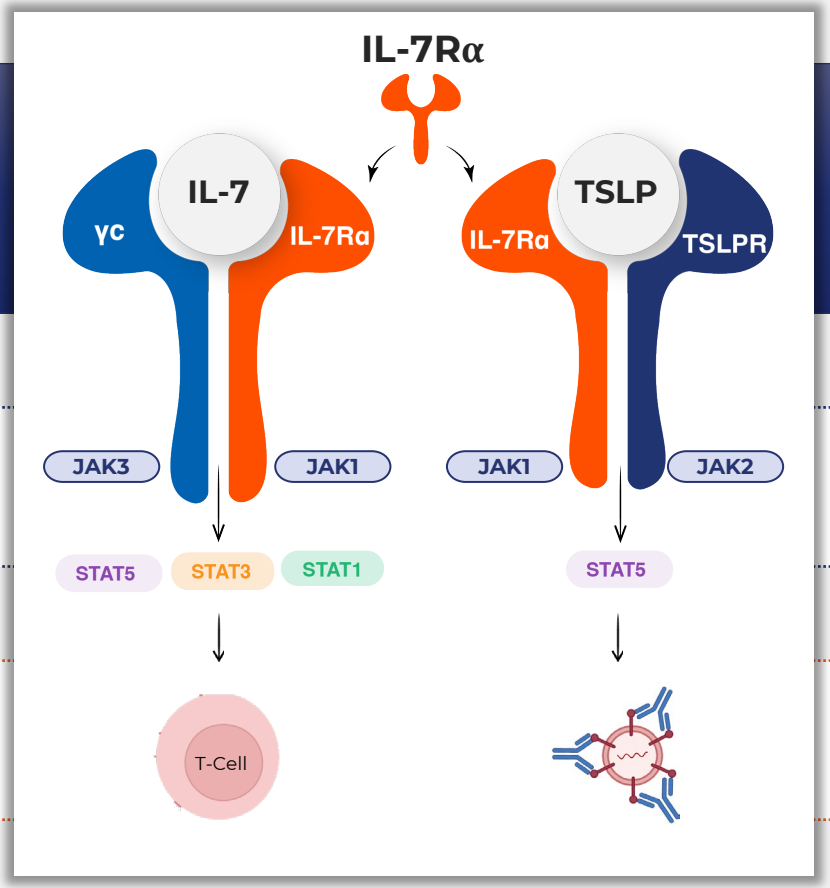
IL-7Rα is a key receptor in immune regulation, central to the signaling of cytokines ***IL-7*** and ***TSLP***

IL-7

IL-7Rα collaborates with the common gamma chain (γc) to establish the IL-7 receptor complex

Triggers a sequence of cellular events, notably **JAKs & STATs**

Vital for the growth, sustenance, and balance of T-cells



TSLP

TSLP binds to its dedicated receptor, TSLPR. For optimal signaling, IL-7Rα joins the mix, creating a composite complex with TSLPR and TSLP

This assembled complex **initiates pathways** primarily linked to **type 2 immunity**

Commonly tied to allergic responses and specific inflammatory scenarios

Positioning crebankitug for potential applications in diverse immune-related and autoimmune conditions

Both TSLP and IL-7 Have a Role in Activating Th1, Th2, and Th17-Driven Inflammation

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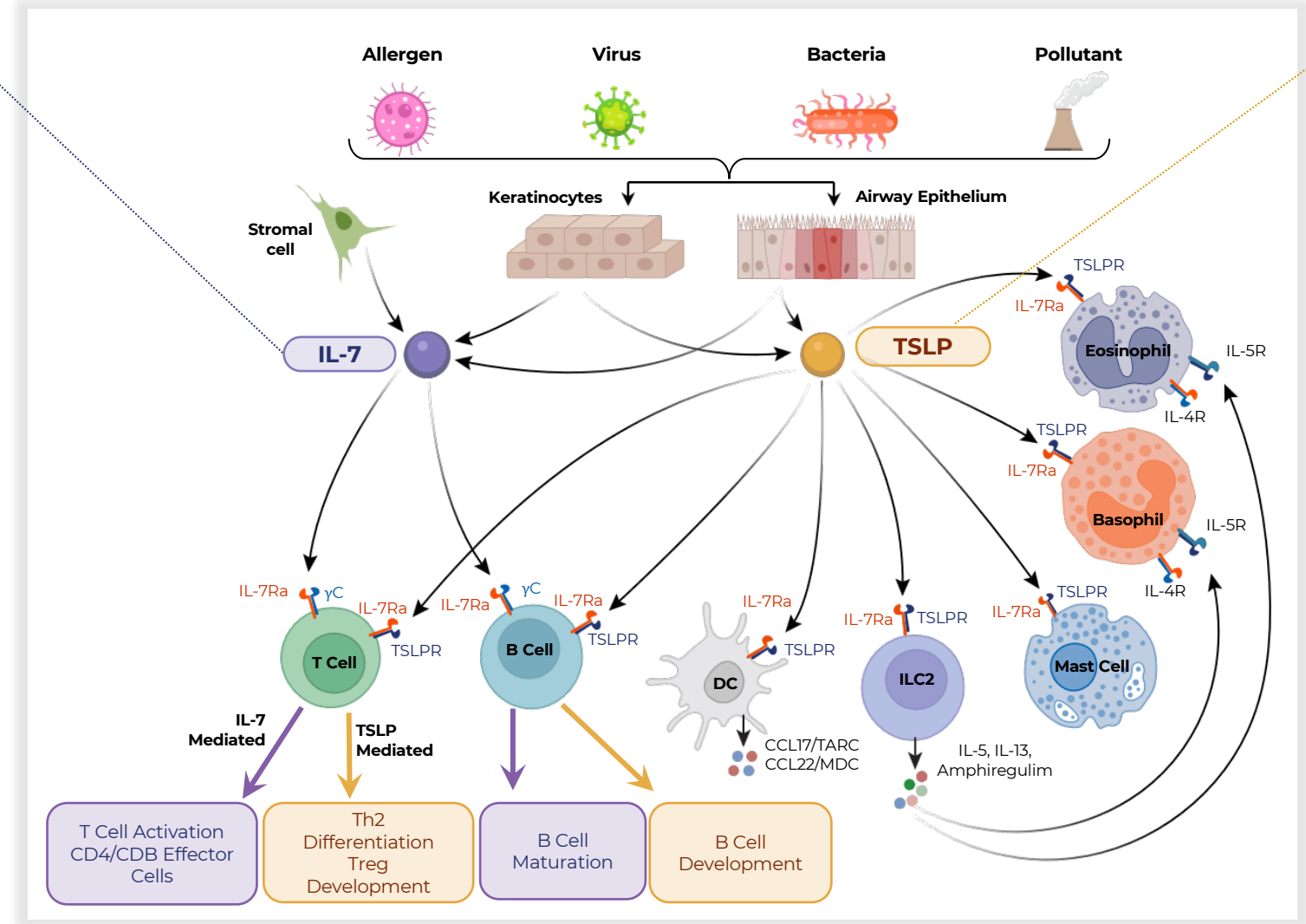
IL-7 PATHWAY

- IL-7 is a growth factor that binds to the heterodimeric receptor of IL-7R:γC and is critical for the survival, development and homeostasis of central and effector memory T cells [4]
- Due to the high expression of IL-7R on T_{eff} compared to T_{reg}, inhibition results in a 20-fold greater activity in reducing T_{eff}, leading to an increase in T_{reg}:T_{eff} ratio [5, 6]



TSLP PATHWAY

- Thymic stromal lymphopoietin (TSLP) is an epithelial-derived cytokine primarily expressed in the lungs, skin and gastrointestinal tract [1]
- TSLP is released from the epithelium by disease amplifying Th2 immune response, including the production of IL-4, -5, -9 and -13. [1]
- TSLP inhibition is clinically validated in severe asthma and has shown positive therapeutic benefit in additional Th2 driven diseases [2,3]

Sources: [1] Ebina-Shibuya, R. and Warren L.eonard. Nature Reviews Immunology, doi:10.1038/s41577-022-00735-y; [2] Marone, G., et al. Expert Opinion on Investigational Drugs, doi:10.1080/13543784.2019.1672657; [3] Menzies-Gow, A., et al. Respiratory Research, doi:10.1186/s12931-020-01505-x; [4] Chen, 2021. Frontiers Immunol, 5. Herold, K., et al. JCI Insight, doi:10.1172/jci.insight.126054; graphic created in BioRender; [5] Martin, M.and Badovinac P. Frontiers in Immunology, doi:10.3389/fimmu.2018.02692; [6] Waickman, A., et al. iScience, doi:10.1016/j.isci.2020.101421; [7] Marković, I. and Savvides S. Frontiers in Immunology, doi:10.3389/fimmu.2020.01557.

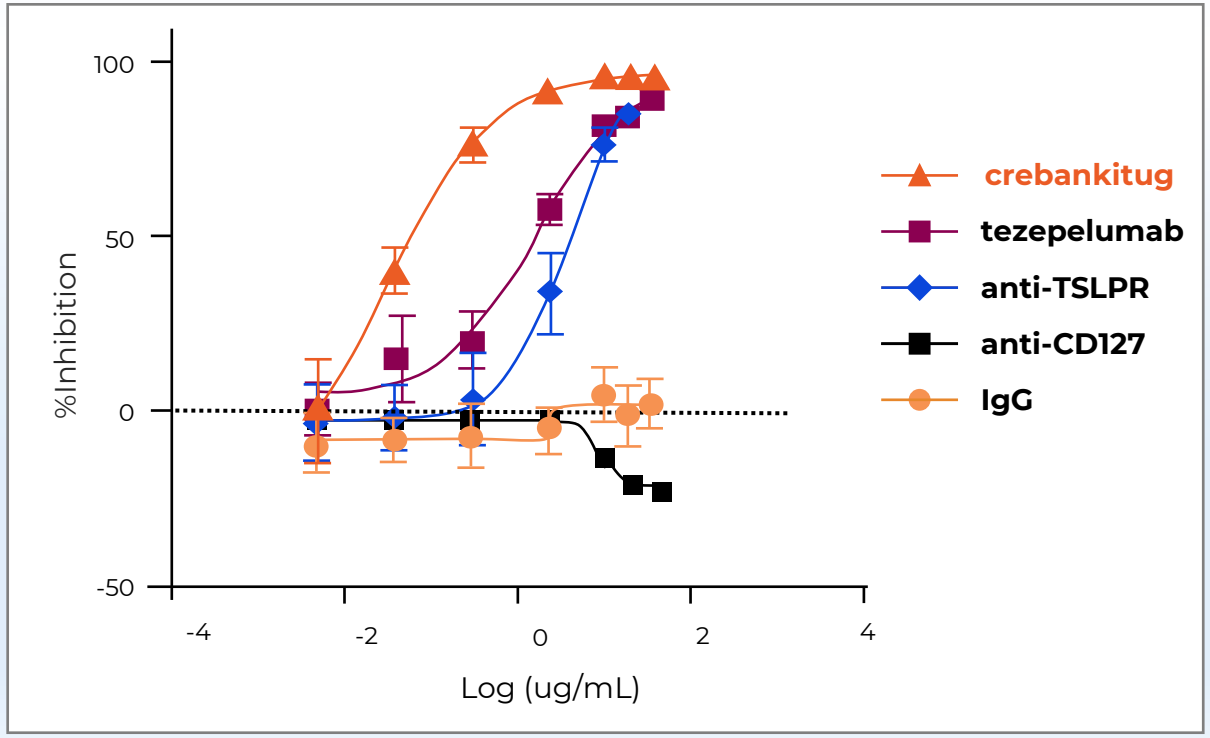
Crebankitug Is the Only mAb to Potently Inhibit Both IL-7R and TSLP

Inhibition of TSLP stimulated CCL17 secretion from human monocytes

	tezepelumab (TSLP)	bempikibart (IL-7Rα)	crebankitug (IL-7Rα)
	TSLP mAb	IL-7Rα mAb	IL-7Rα mAb
TSLP-Induced Signals	67 ng / ml / 0.44nM (CCL17)[3]	24 nM (CCL2)[4]	7.5 ng / ml / 0.05nM (CCL17)[1] 11 ng / ml / 0.07nM (CCL22)[1] **0.08 nM (CCL2)[4]**
IL-7-Induced Signals	*Neg*	0.6 nM (IL-7 at 0.25ng/ml)[4] 2.1 nM (IL-7 at 2.5ng/ml)[4]	0.46nM (pSTAT5)[2]

World Allergy Congress Poster, Dec 2023

% inhibition of TSLP stimulated CCL17 secretion from human monocytes



Source: [1] Zura Internal Data; [2] Herold, Kevan C., et al. JCI Insight, doi:10.1172/jci.insight.126054; [3] Numazaki, Mako, et al. Journal of Pharmacology and Experimental Therapeutics, doi:10.1124/jpet.121.000686; [4] Yamniuk, Aaron P., et al. Antibodies against Il-7r Alpha Subunit and Uses Thereof. 18 May 2021.

Acronyms: CCL17, C-C motif chemokine ligand 17; IL, interleukin; mAb, monoclonal antibody; TSLP, thymic stromal lymphopoietin

Dual Inhibition of Th1 and Th2 May Offer Broader or Deeper Levels of Response in Atopic Dermatitis

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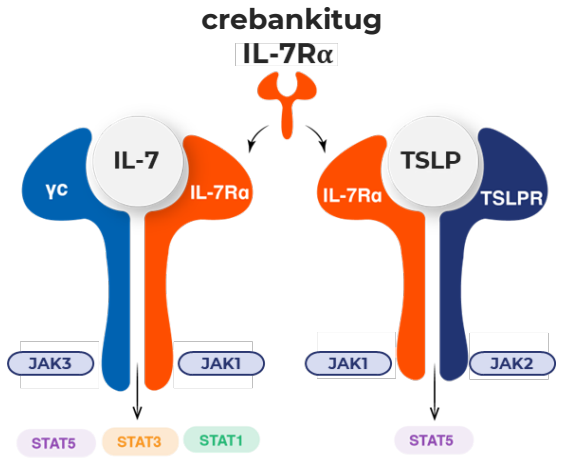
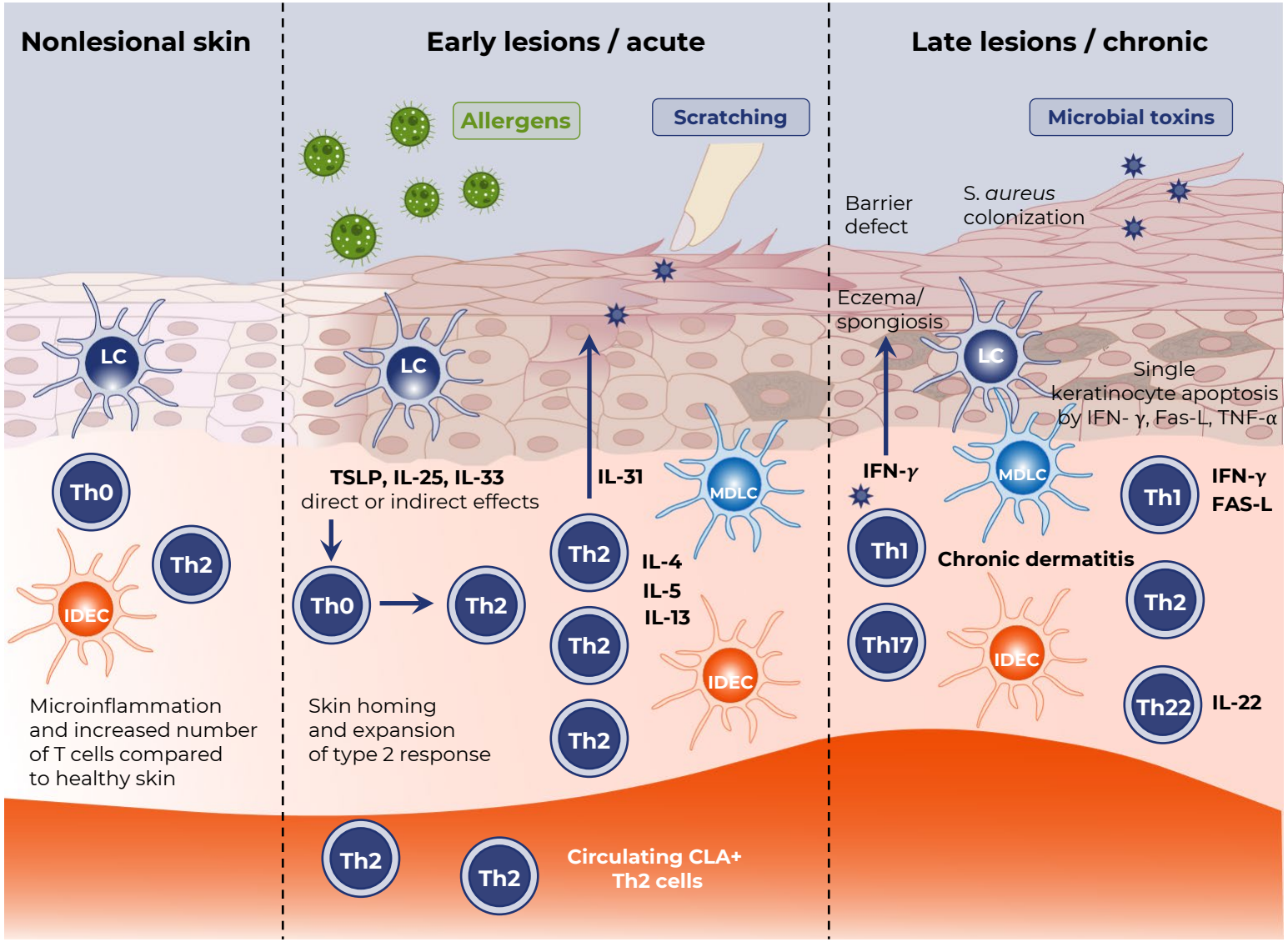
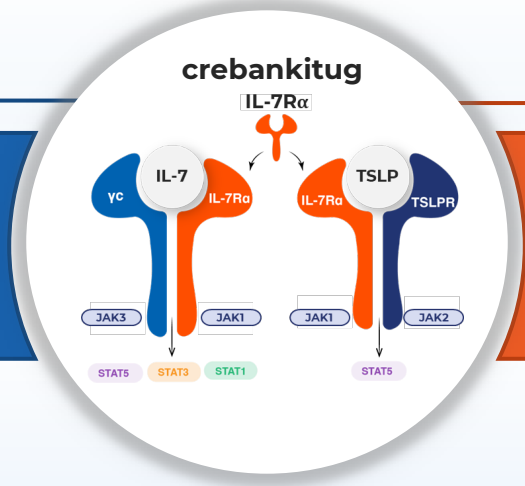
- TSLP and IL-25 activate Th2 cells, which are crucial in the early stages of atopic dermatitis.
- As the condition progresses, the influence of Th2 decreases, while Th1 and Th17 responses become more prominent.
- Targeting both Th2 and Th1 pathways may offer broader and more effective treatment options.





Crebankitug Provides Optionality for Clinically Validated Development or Novel Indications

External Phase 2 PoC 2024



Internal Evaluation 2024

Explore the potential indication space	Solid biologic rationale for IL-7 / TSLP	Expected Clinical Readout[1]
atopic dermatitis & alopecia areata	✓	✓
Ulcerative colitis (and other GI)	✓	✓
Asthma (and other Respiratory)	✓	✓

Zura Internal Evaluation of New or Orphan Indications[2]

Common autoimmune disease

Orphan autoimmune disease

Sources: [1] ClinicalTrials.gov database, Company Presentations; [2] Zura Internal Planning
Acronyms: GI, gastrointestinal; IL, interleukin; PoC, proof-of-concept; TSLP, thymic stromal lymphopoietin

Crebankitug Summary

Crebankitug targets immune-related and autoimmune conditions linked to IL-7R or TSLP signaling

In three Phase 1 and 1b studies involving 93 participants, crebankitug was well-tolerated, with most adverse events being mild and unrelated to the treatment

Only anti-IL-7Rα program with clinical data showing impact on key T-cell subpopulations, indicating potential for broad T-cell mediated and atopic diseases

Advancing indication planning and monitoring IL-7R and TSLP therapies to support Phase 2 strategy

torudokimab

ZB-880

Anti-IL-33

Torudokimab is a fully human, high affinity monoclonal antibody that neutralizes IL-33, preventing ST2-dependent and ST2-independent (e.g., RAGE) inflammation.

Torudokimab Asset Overview

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torudokimab | ZB-880

About torudokimab

01 IL-33 implicated in driving Th2 biology through ST2 and Th1/Th17 through alternative signaling [1]

02 Well tolerated in Phase 1 and Phase 2 trials conducted by Eli Lilly [2]

- 141 healthy volunteers in Phase 1 study
- 103 participants with moderate to severe atopic dermatitis in Phase 2

03 The target engagement evaluation suggested binding to IL-33 and treatment emergent ADA had no apparent impact on PK or target engagement

- Analyses confirmed key biomarker reductions (IL-13, periostin and CCL17/TARC) and no ADA impact [3]
- Potential utility in diseases driven by epithelial inflammation [1]

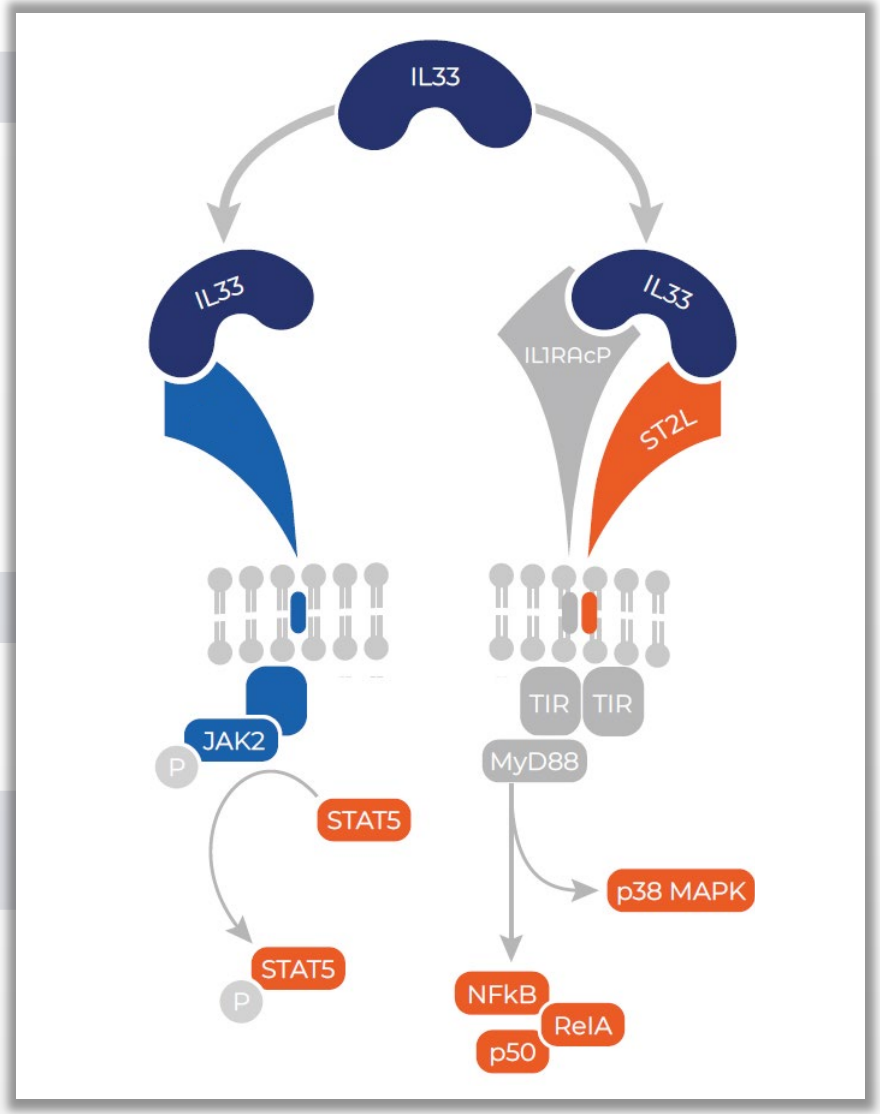
Mechanism of Action

01 Inhibition of IL-33 blocks both ST2 and RAGE signaling [4]

Initial Focus on Respiratory, Dermatologic, Gastrointestinal and Orphan Autoimmune Indications

01 Potential for 1st-in-class opportunities

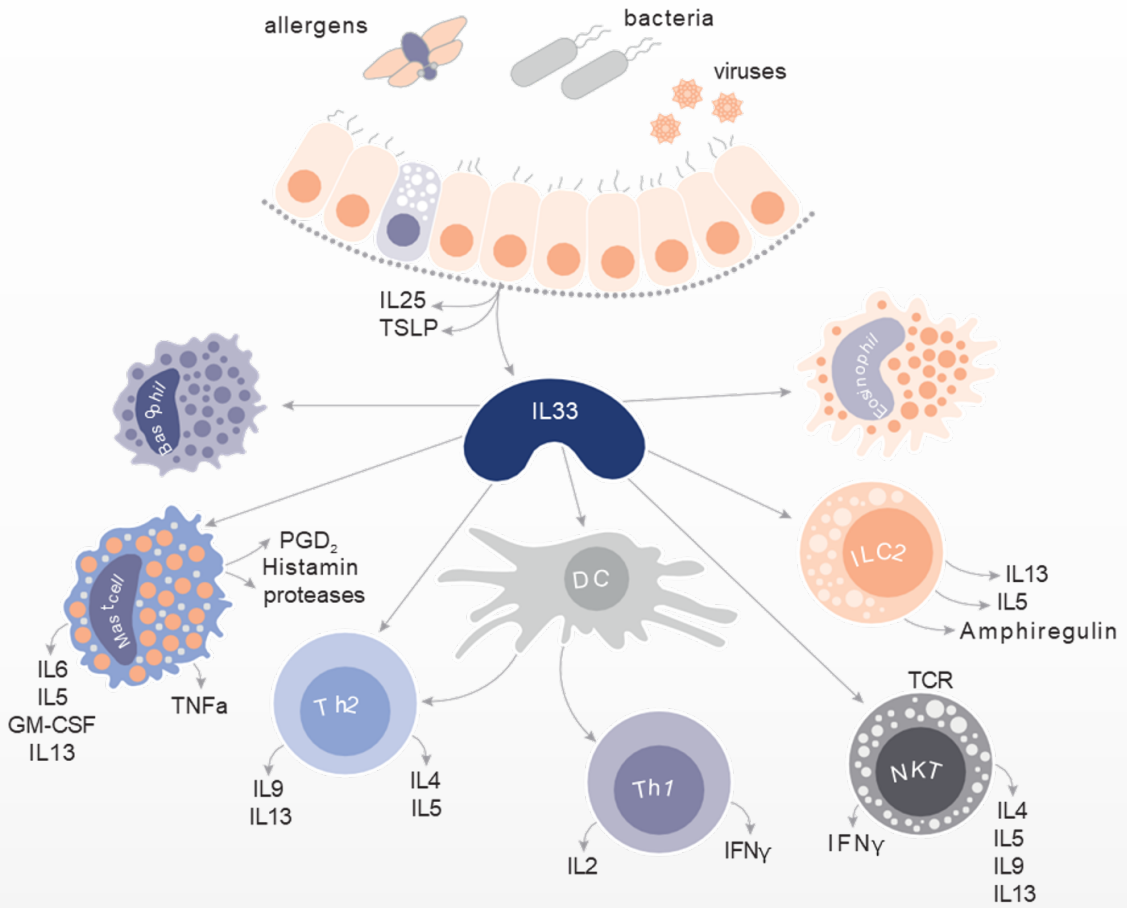
02 Validated pathways in COPD [4] and asthma [5]



Sources: [1] Cohen, S., et al. Nature Communications, doi:10.1038/ncomms9327; [2] Clinicaltrials.Gov, clinicaltrials.gov/ct2/show/NCT03913260. Accessed 26 Aug. 2024; Clinicaltrials.Gov, clinicaltrials.gov/ct2/show/NCT03343587. Accessed 26 Aug. 2024; Clinicaltrials.Gov, clinicaltrials.gov/ct2/show/NCT03831191. Accessed 26 Aug. 2024; Section 6.1, DSUR for period 23-Sep-2019 to 22-Sep-2020; [3] Laquer, V., et al. British Journal of Dermatology, doi:10.1111/bjd.21631. [4] Okragly, A., et al. Journal of Inflammation Research, doi:10.2147/jir.s320287. [5] Wechsler, M., et al. New England Journal of Medicine, doi:10.1056/nejmoa2024257.

Torudokimab IL-33 Pathway

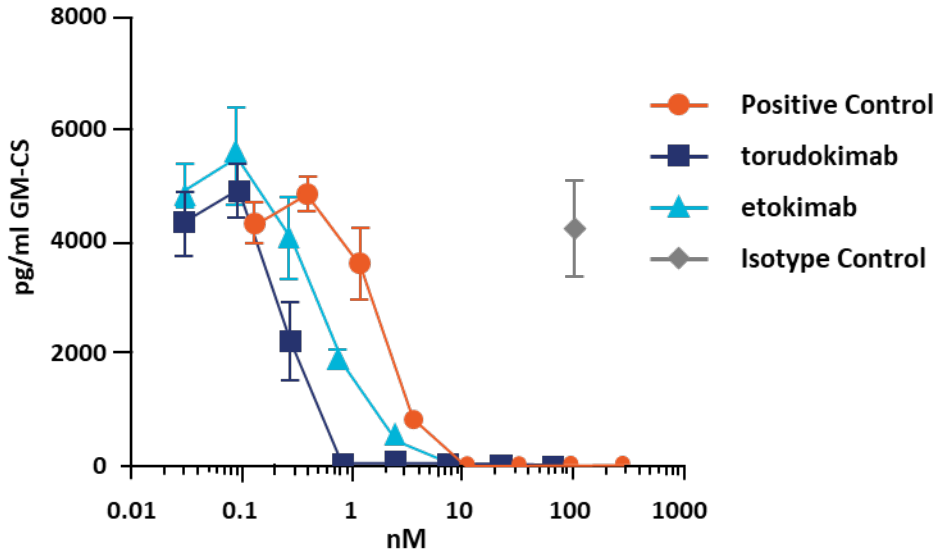
IL-33 is a member of the IL-1 cytokine family that is constitutively expressed in structural and lining cells including fibroblasts, endothelial, and epithelial cells of skin, gastrointestinal tract, and lungs [1]	IL-33 is released from the epithelium by disease exacerbating environmental factors and is an important amplifier of innate inflammation during exacerbations [2]
Polymorphisms in IL-33 and ST2 are associated with increased eosinophil production as well as respiratory diseases such as COPD and severe asthma	IL-33 inhibition clinically validated in severe asthma, COPD [3], and subsets of other epithelial disorders [4]
Pre-clinical data demonstrates superior activity of torudokimab compared with etokimab and itepekimab suggesting the potential for best-in-class activity [5]	Emerging biology suggests expanding opportunity for IL-33 in fibrotic and autoimmune conditions [6]

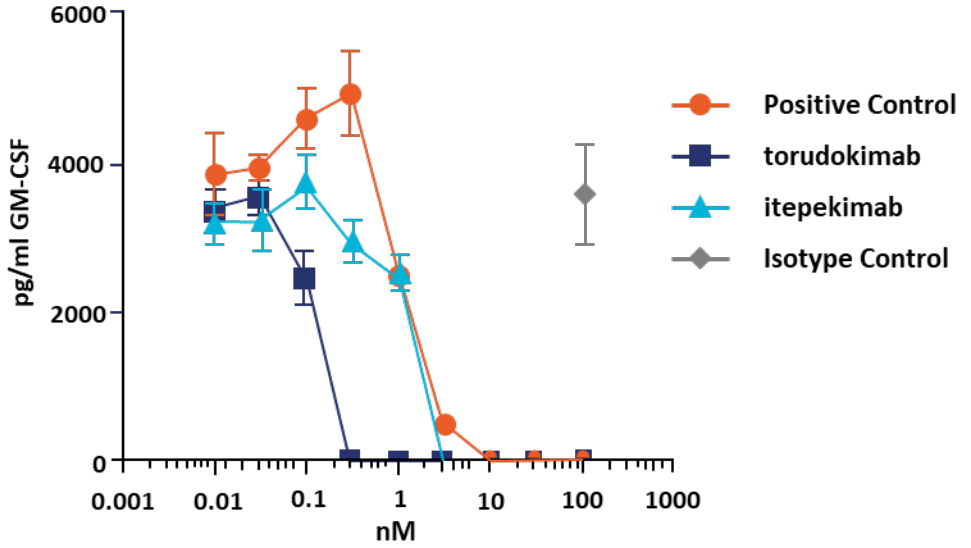


Sources: [1] Chan, B., et al. Frontiers in Immunology, doi:10.3389/fimmu.2019.00364; [2] Cayrol, C. and Girard, J.P. Cytokine, doi:10.1016/j.cyto.2022.155891. [3] Gudbjartsson, D., et al. Nature Genetics, doi:10.1038/ng.323; Ketelaar, M., et al. Journal of Allergy and Clinical Immunology, doi:10.1016/j.jaci.2020.04.051; [4] Singh, D. The Lancet Respiratory Medicine, doi:10.1016/s2213-2600(22)00005-4; Wechsler, M., et al. New England Journal of Medicine, doi:10.1056/nejmoa2024257; Chen, Yi-Ling, et al. Science Translational Medicine, doi:10.1126/scitranslmed.aax2945; [5] Zura Internal data; [6] Pei, C., et al. Immunology, doi:10.1111/imm.12174; Kurimoto, M., et al. Frontiers in Physiology, doi:10.3389/fphys.2021.781012; Dong, Y., et al. Frontiers in Medicine, doi:10.3389/fmed.2021.739489.

Torudokimab Has Potential for "Best-in-Class" Activity

Torudokimab was 2.9 and 5.5-fold more potent than etokimab and itepekimab, respectively, inhibiting IL-33-induced GM-CSF production by human mast cells





Antibody	k_{on} ($M^{-1}s^{-1}$)	k_{off} (s^{-1})	k_d (pM)	Torudokimab Potency
torudokimab (LY3375880)	1.7×10^6	6.7×10^{-5}	39	
etokimab (AnaptysBio)	9.4×10^5	1.2×10^{-4}	112	**2.9x**
itepekimab (Regeneron)	7.6×10^5	1.6×10^{-4}	215	**5.5x**

Sources: Zura Internal Data

Corporate Update

Team and Cash Runway

Zura's Leadership: Driving Innovation in I&I

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Nasdaq: ZURA

EXECUTIVE TEAM

Robert Lisicki
Chief Executive Officer and Director

Verender Badial
Chief Financial Officer

Kiran Nistala, MBBS, PhD
Chief Medical Officer and Head of Development

Gary Whale PhD
Chief Technology Officer

Kim Davis JD
Chief Legal Officer and Corporate Secretary

BOARD OF DIRECTORS

Amit Munshi
Chairman

Arnout Ploos van Amstel
Director

Jennifer Jarrett
Director

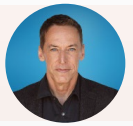
Neil Graham, MBBS, MD, MPH
Director

Parvinder Thiara
Director

Robert Lisicki
Chief Executive Officer and Director

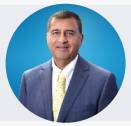
Sandeep C. Kulkarni, MD
Director

Someit Sidhu, MD
Founder and Director



Steve Schoch
Director

SAB



Ajay Nirula, MD, PhD

Dinesh Khanna, MD, MSc

Johann Gudjonsson, MD, PhD

Michael Weinblatt, MD

Steven Ziegler, PhD

Acronyms: I&I, inflammation and immunology; SAB, scientific advisory board

Key milestones* expected through 2026

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Cash runway expected through 2027

MILESTONE LEGEND:
- ❑ Internal Zura
- ▲ External Catalyst

		2H-2024	2025	2026
tibulizumab	**SSc study**	☑ Select CRO ❑ Obtain IND from US FDA ❑ Initiate a Phase 2 study in SSc	❑ Phase 2 study recruitment	❑ SSc Topline Data *Pending data outcomes, Phase 3 → → →*
	HS study	❑ Select CRO	❑ Obtain IND from US FDA ❑ Initiate a Phase 2 study in HS	❑ HS Topline Data *Pending data outcomes, Phase 3 → → →*
crebankitug	**Indication Planning On-going**	❑ Complete internal indication planning ▲ 4Q-2024: Phase 2a Topline Data bempikibart in AA, Q32 Bio ▲ 4Q-2024: Phase 2 Topline Data bempikibart in AD, Q32 Bio ▲ 3Q-2024: Phase 3 Topline Data tezepelumab in Asthma (reducing corticosteroid), Amgen	❑ Select indication for future development* *Indication selection to guide future crebankitug milestones → → →*	
torudokimab	**Indication Evaluation On-going**		▲ 2Q-2025: Phase 3 Study Complete itepekimab in COPD, Sanofi ▲ 2Q-2025: Phase 3 Topline Data astegolimab in COPD, Roche ❑ Complete internal indication planning *Indication planning to guide future torudokimab milestones → → →*	

(*) The timing of regulatory and clinical trial milestones are subject to change and additional discussion with the FDA
Sources: Internal Zura Planning
Acronyms: AA, alopecia areata; AD, atopic dermatitis; COPD, chronic obstructive pulmonary disease; CRO, contract research organization; HS, hidradenitis suppurativa; SSc, systemic sclerosis; US FDA, United States Food & Drug Administration

Nasdaq ticker: ZURA

April 2024
Photo courtesy of ©Nasdaq, Inc.

- **Our mission:** Driving scientific breakthroughs by turning drug discoveries into transformative, life-saving treatments.
- **Tibulizumab shows best-in-class potential:** Introducing a tetravalent antibody therapy designed to target and potentially treat autoimmune diseases.
- **Promising pipeline for value creation:** Integrating validated biological pathways into multifunctional antibody assets to potentially improve therapeutic outcomes.
- **Upcoming external catalysts:** Anticipating near-term developments that could further expand the pipeline's potential.
- **Proven leadership:** Experienced team with a track record of contributing to over $8 billion in mergers and acquisitions in the past three years.
- **Strong financial position:** With approximately $188 million[1] in cash, cash equivalents, and investments, we are funded to support our planned operations through 2027. The 3Q 2024 IPO warrant exchange has streamlined our share structure, and additional financing through ATM options remains available for future needs. As of August 29, 2024, we have 63,774,183 Class A Ordinary Shares outstanding[2].

Source: [1] Cash includes cash and cash equivalents as of 30-June-2024; [2] S-3 dated 03-Sept-2024
Acronyms: ATM, at-the-market offering; IPO, initial public offering

