



zurabio

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

September 2024

Nasdaq Ticker: ZURA

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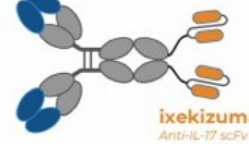
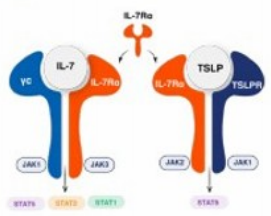
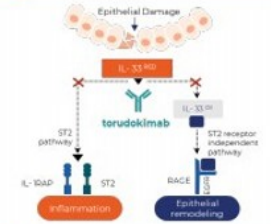
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Company Summary

- 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



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

		Phase 1	Phase 2
<p>tibulizumab <i>Pipeline-in a-product</i></p>	<p>tabalumab <i>Anti-Baff Ab</i></p>  <p>ixekizumab <i>Anti-IL-17 scFv</i></p> <p>Only bispecific antibody targeting IL-17A and BAFF</p>	<p>Tibulizumab has been studied in three Phase 1/1b clinical studies to date *</p> <ul style="list-style-type: none"> ✓ 78 participants dosed across 3 studies <ul style="list-style-type: none"> - n=57 single dose - n=21 multiple doses up to 12 weeks 	<p>Planning to initiate Phase 2 studies of tibulizumab for SSc in 4Q 2024 and for HS in 2Q 2025.</p>
<p>crebankitug <i>Potential best-in-class</i></p>	 <p>IL-7R and TSLP Inhibition with potential best in class TSLP inhibition</p>	<p>Crebankitug has been studied in three Phase 1/1b clinical studies to date **</p> <ul style="list-style-type: none"> ✓ 93 participants dosed <ul style="list-style-type: none"> - n=60 single dose - n=33 multiple doses up to 12 weeks 	<p>Actively assessing the competitive landscape and evaluating potential therapeutic indications to guide our future development efforts.</p>
<p>torudokimab <i>Potential best-in-class</i></p>	 <p>Best in class in inhibiting GM-CSF production by human mast cells</p>	<p>Torudokimab has been studied in three Phase 1/2 clinical studies to date ***</p> <ul style="list-style-type: none"> ✓ 244 participants dosed <ul style="list-style-type: none"> - n= 81 single dose - n=163 multiple doses up to 52 weeks 	<p>Actively assessing the competitive landscape and evaluating potential therapeutic indications to guide our future development efforts.</p>

(*) 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 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



Nasdaq: ZURA

Prevalence¹ U.S., EU5* and Japan	Potential Therapeutic Area	TAM (\$USD) U.S., EU5* and Japan
270 k	SSc <i>systemic sclerosis</i>	\$2,500 M
700 k	HS <i>hidradenitis suppurativa</i>	\$3,200 M
7 M	AA <i>alopecia areata</i>	\$5,200 M
32 M	COPD <i>chronic obstructive pulmonary disease</i>	\$23,692 M
25 M	AD <i>atopic dermatitis</i>	\$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



Nasdaq: ZURA



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



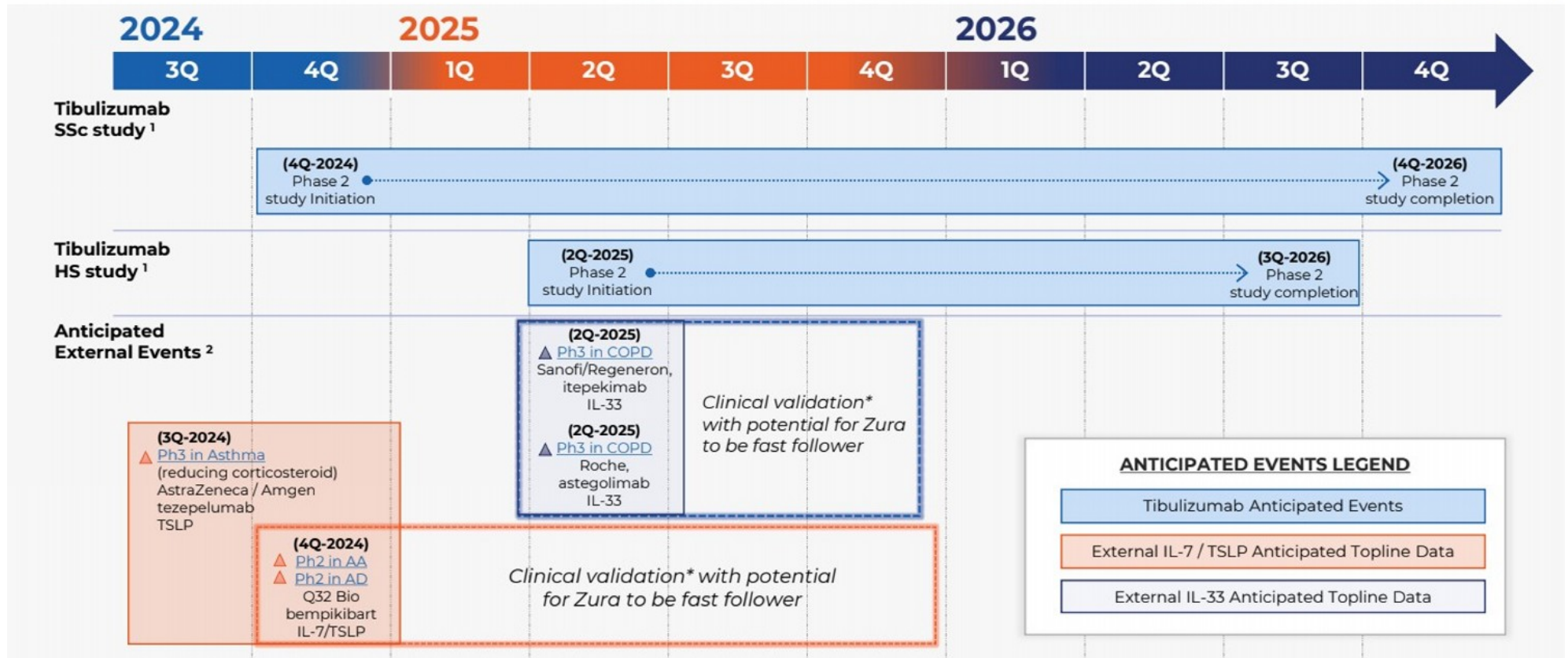
KIM DAVIS JD
Chief Legal Officer and
Corporate Secretary



MICHAEL HOWELL PhD
Chief Scientific Officer and
Head of Translational Medicine



Key Anticipated Events Through 2026



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

Sources: ¹Zura Planning Assumptions; ²clinicaltrials.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

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

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

Systemic sclerosis is characterized by *tissue inflammation and fibrosis*



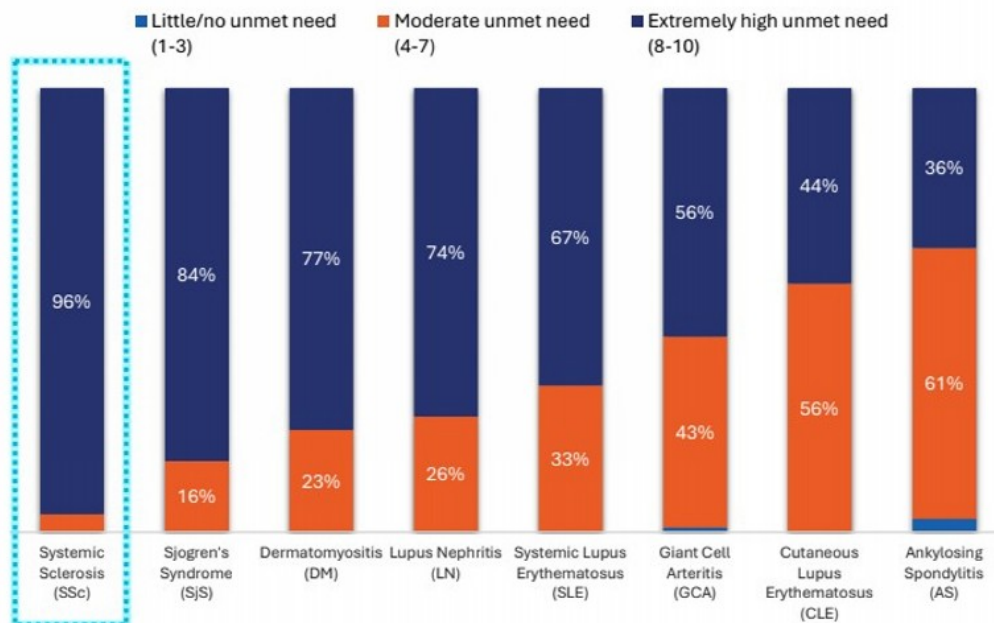
Multiple areas for evaluation and improvement in SSc



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

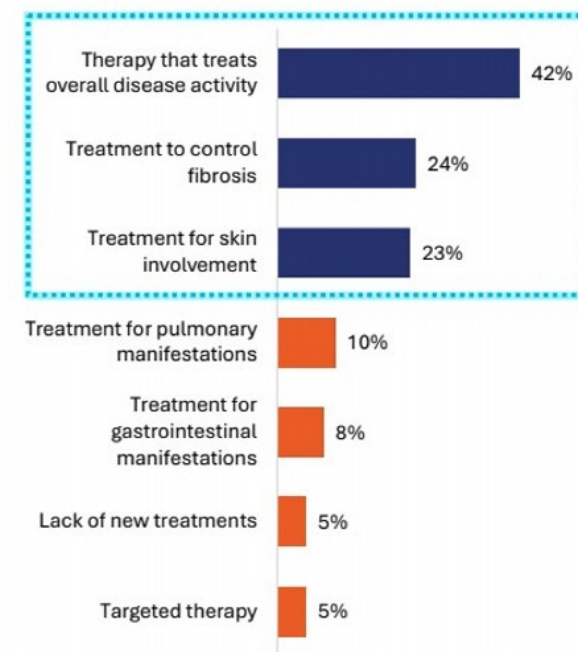
Unmet Need for New Pharmacological Treatment Options

% of respondents



Unmet Need for the Treatment of SSc

Coded, open-end responses



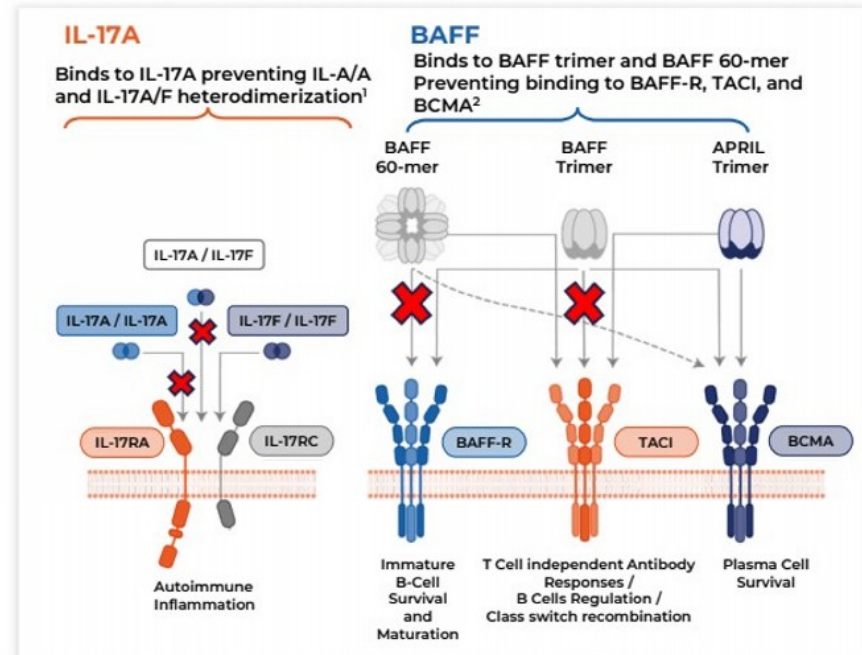
96% of respondents highlight a pressing need for new pharmacological treatments in SSc

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 fibrosis³



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

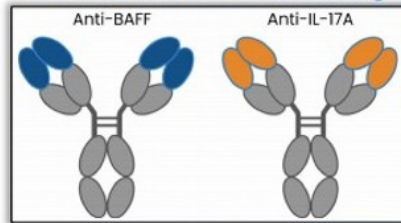
- BAFF is increased in peripheral blood and correlates with skin fibrosis and incidence of pulmonary fibrosis^{4,5}
- In pre-clinical models BAFF blockade prevents skin fibrosis & autoantibody production^{6,7}

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.201700289; 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*

Monoclonal Antibody

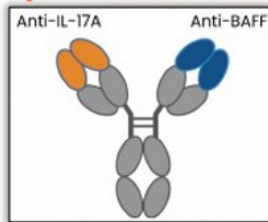


Targets a single, selective molecule in the body

May not address disease heterogeneity

1st Generation

Bispecific Antibody

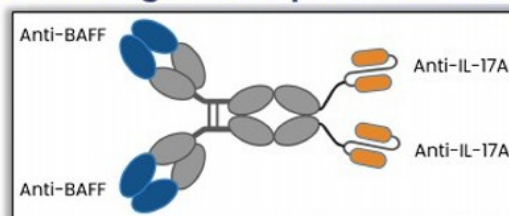


Binds two different targets simultaneously

Activation may require dual engagement

2nd Generation

Dual Antagonist Bispecific Antibody



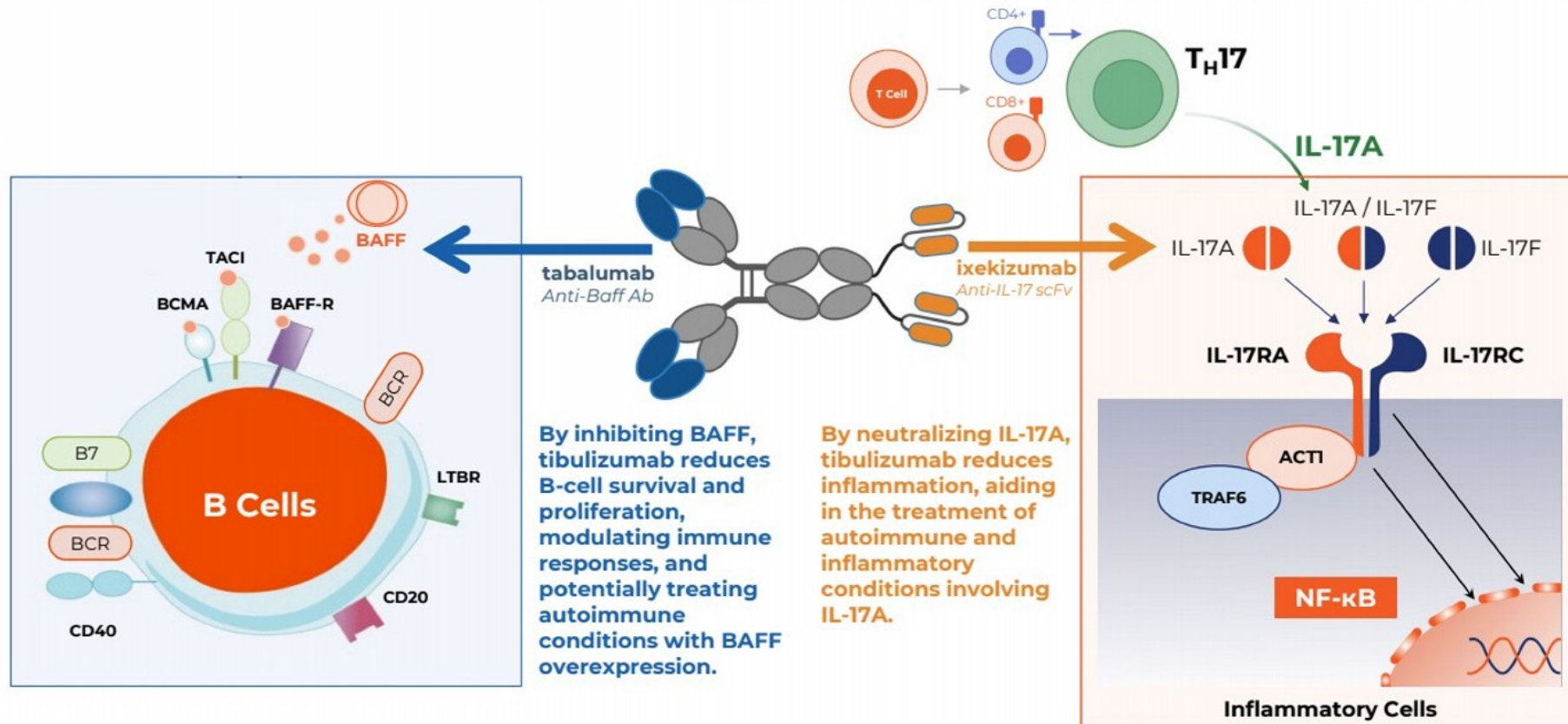
Functions as a single selective agent or two independent agents

Blocks two distinct ligands, potentially enhancing disease suppression

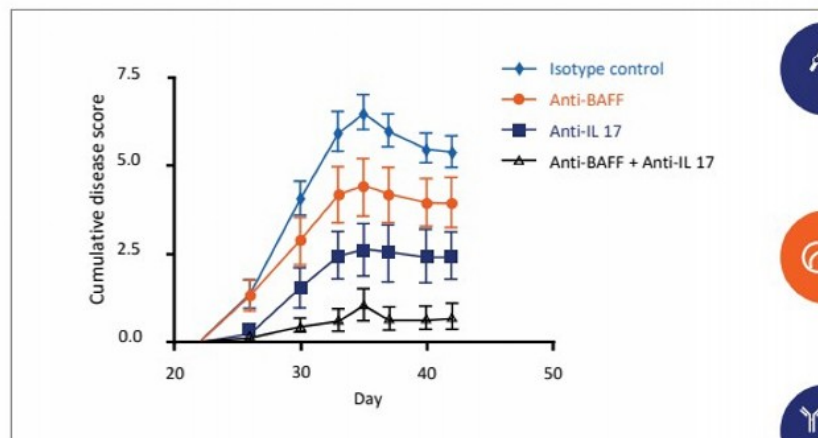
NEXT GENERATION

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.



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

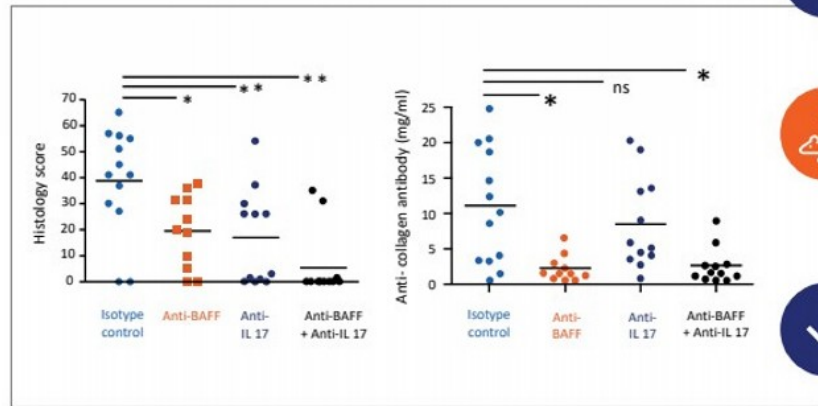


Mice were injected with **anti-IL-17A and/or anti-BAFF** on days 22, 29, and 36



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

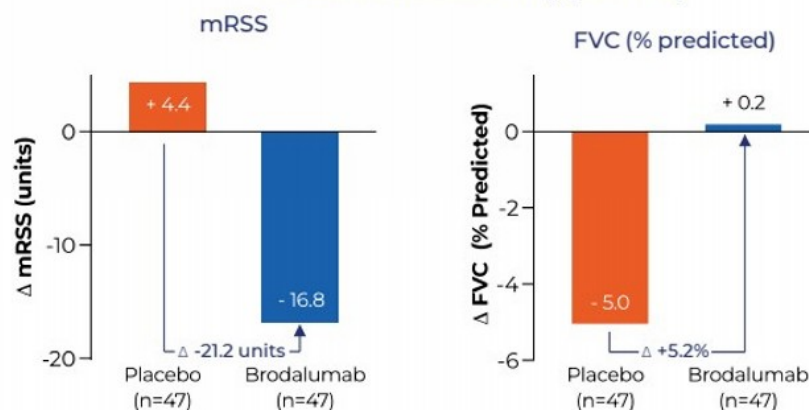
IL-17 receptor antagonist – Phase 3

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)



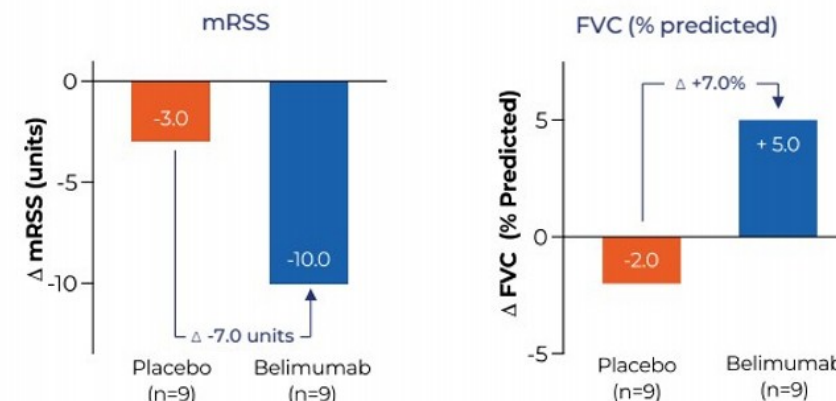
BAFF antagonist – IIT

Belimumab

- A 52-week, investigator-initiated, single-center, double-blind, placebo-controlled pilot study in 20 participants with dcSSc on MMF²
- 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)



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;

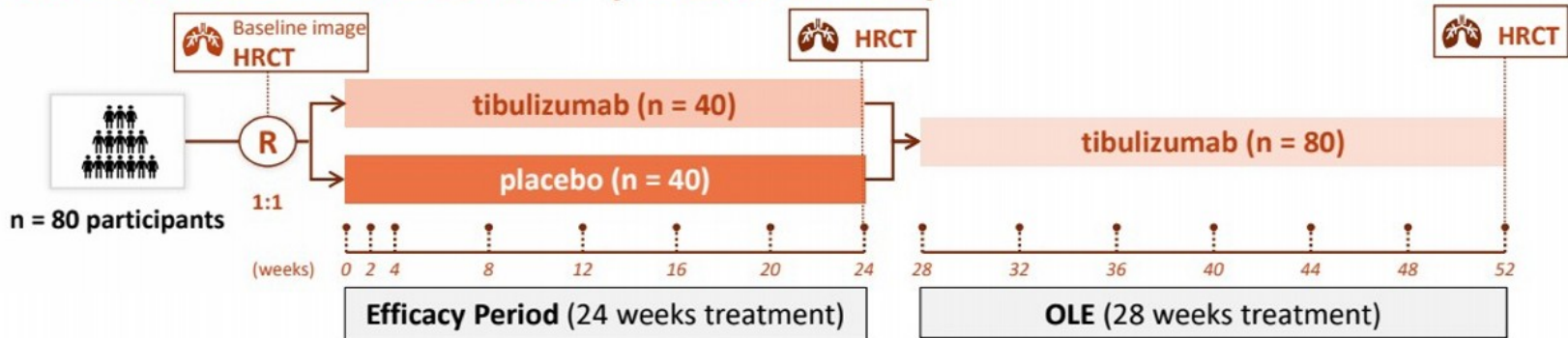
mRSS, modified Rodnan Skin Score; QOL, quality of life; SHAQ-DI, scleroderma health assessment questionnaire – disability index; SSc, systemic sclerosis; VAS, visual analogue scale

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 CRIS (rCRIS)

(*) Study design is subject to change.

Sources: Zura Internal Planning

Acronyms: CRIS, 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

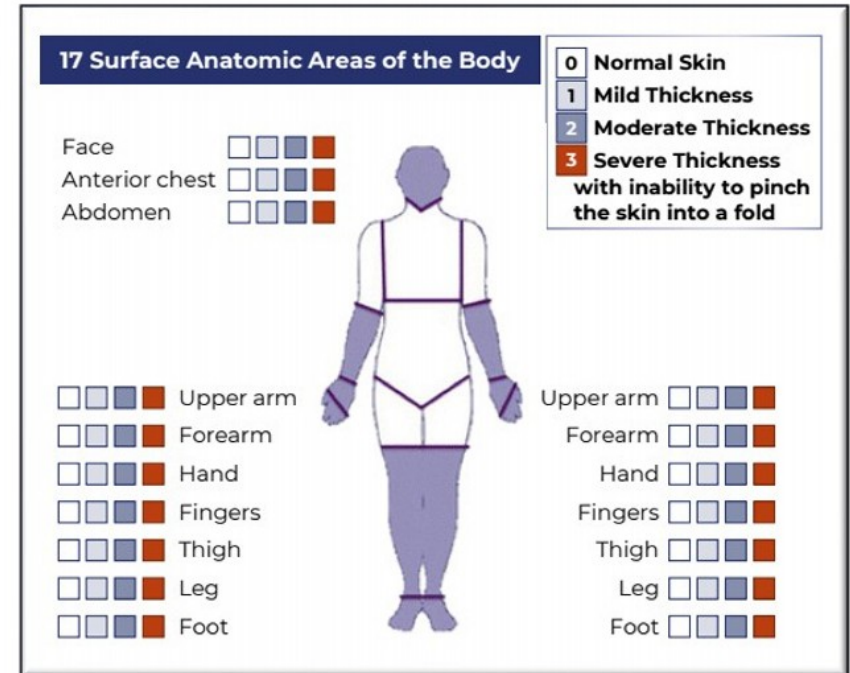
modified Rodnan Skin Score (mRSS): Endpoint for Assessing Skin Thickness and Fibrosis



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

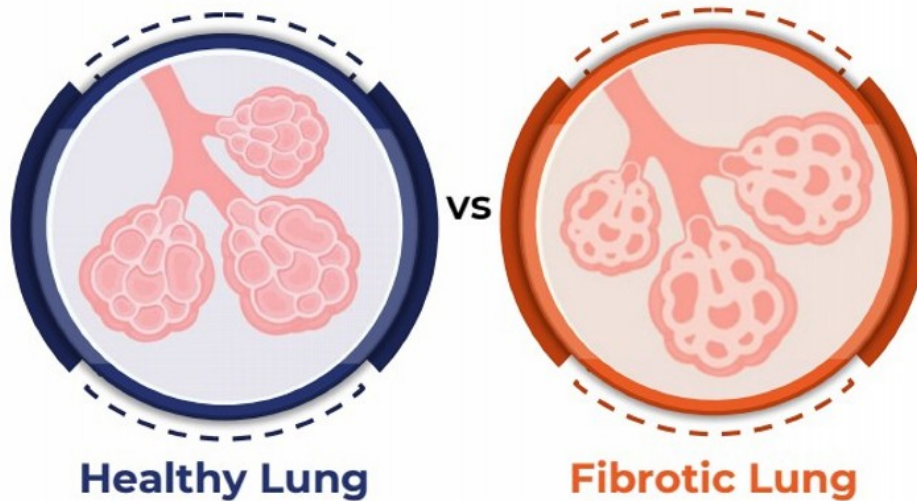
The **mRSS assesses skin thickness** in systemic sclerosis patients by **evaluating 17 body sites** (e.g., face, chest, abdomen, arms, legs). Each site is scored from 0 to 3.

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



Sources: ¹ Khanna, D., et al. Journal of Scleroderma and Related Disorders, doi:10.5301/jsrd.5000231; ² Ferrelli, 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

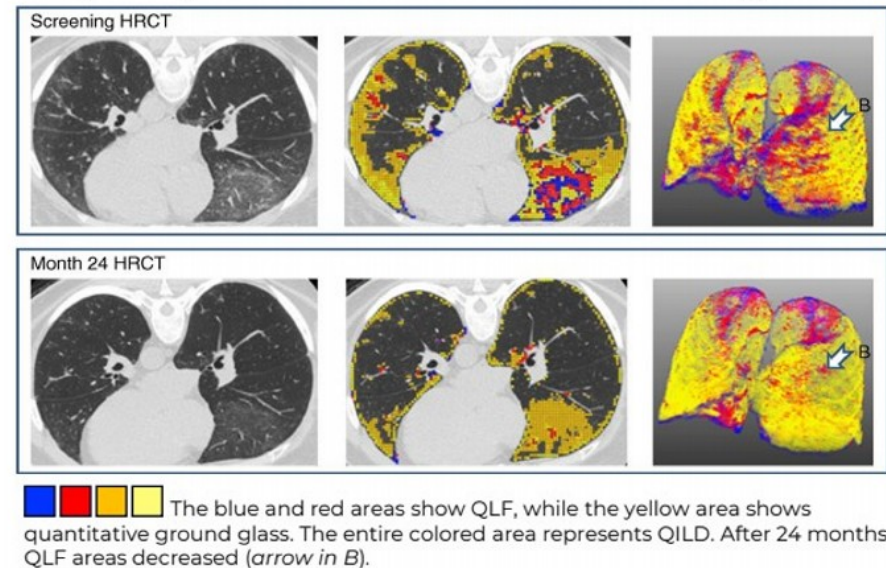


ILD includes various pulmonary disorders marked by inflammation and progressive lung fibrosis, resulting in restrictive lung function and impaired gas exchange.

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



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

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

STEP 1: Assess for significant SSc-related events:

- New scleroderma renal crisis
- New decline in percent predicted FVC $\geq 15\%$ 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



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

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 inflammation¹⁻³
- ✓ Disproportionately affects women between adolescent age to 55 years of age^{4,5}



CLINICAL OPPORTUNITY⁶

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

Average time to diagnosis is **7 years**

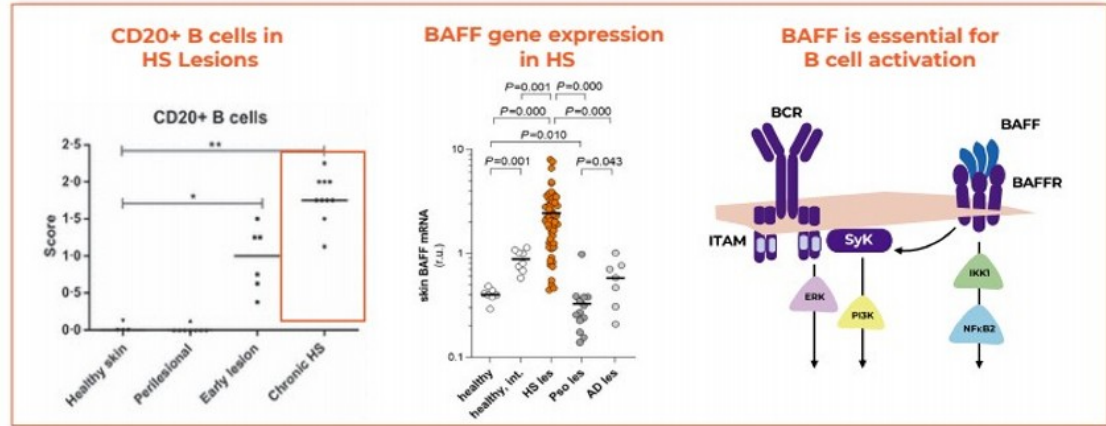
~>50% patients still left inadequately treated

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

B Cell Signaling Potentiates HS Disease

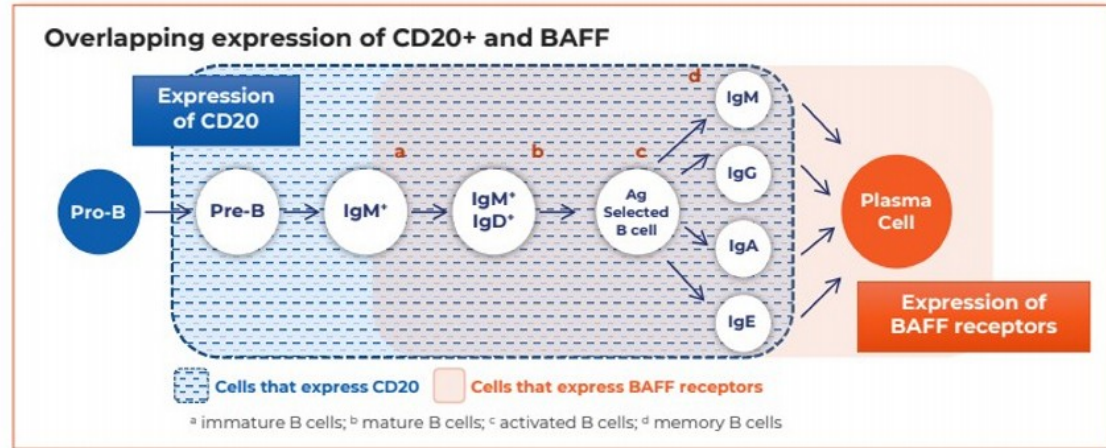
Elevated **CD20+** B cells and **CD138+** plasma cells in chronic HS lesions sustain inflammation.^{2,3}

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}



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



tibulizumab | ZB-106

company and drug INN >>					
		<i>secukinumab</i>	<i>remibrutinib*</i>	<i>bimekizumab</i>	<i>sonelokimab</i>
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

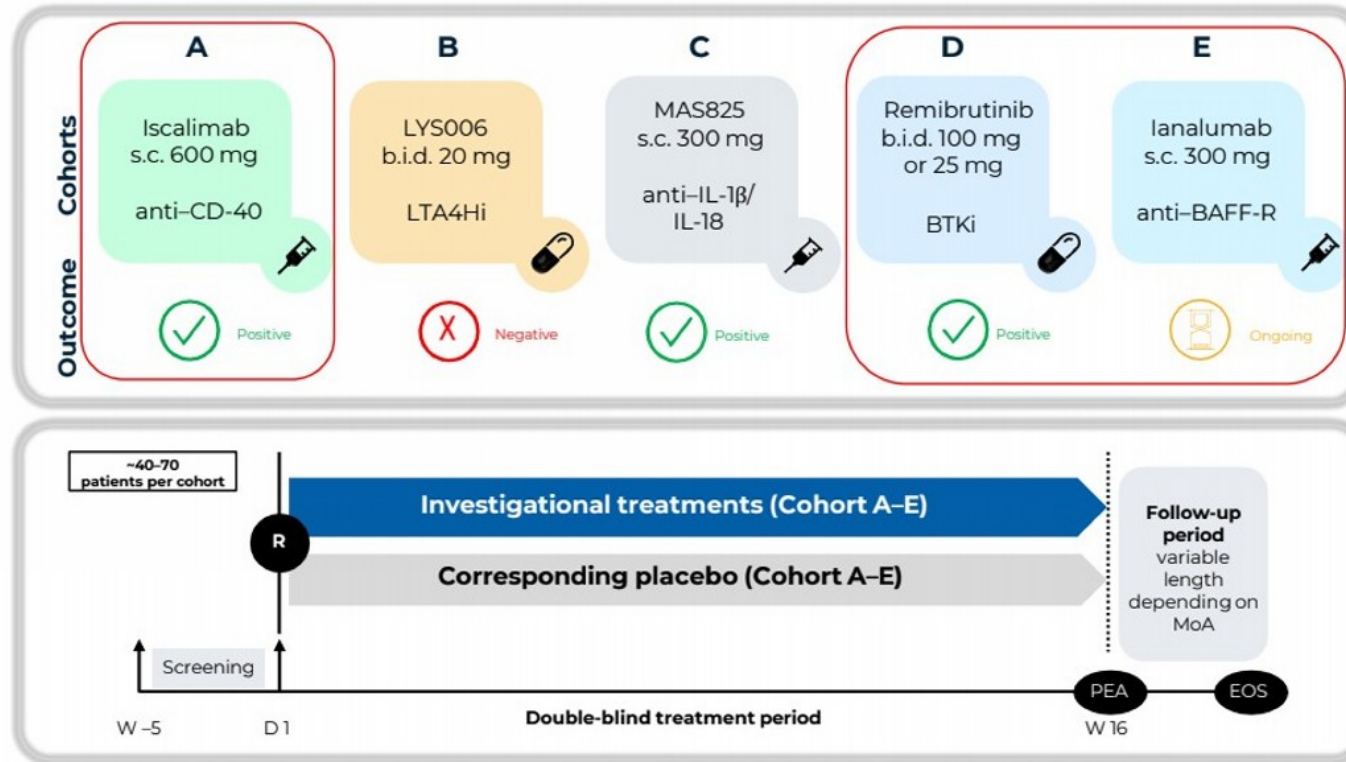
- Binding affinity, quantified by the dissociation constant (K_D), reflects the strength of interaction between a drug and its target, with a lower K_D indicating stronger binding.
- Stronger binding affinity ensures effective neutralization of the target, leading to the potential for improved clinical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

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

Sources: ¹Taltz®. 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.

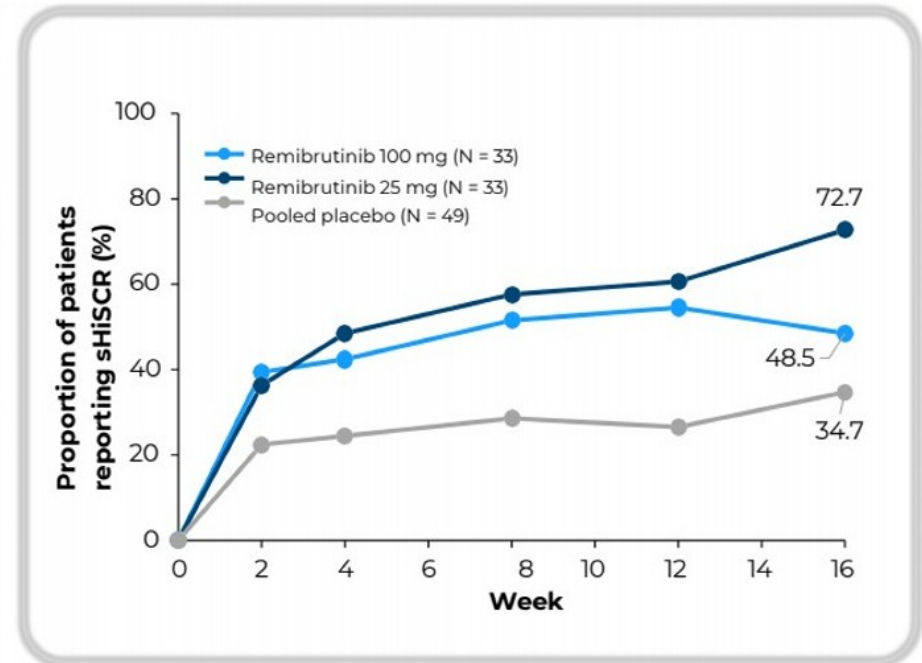
BAFF-R, B-cell activating factor of the tumor necrosis alpha family receptor; b.i.d., twice daily; BTKi, Bruton's tyrosine kinase inhibitor; CD, cluster of differentiation; D, day; EOS, end of study; HS, hidradenitis suppurativa; IL, interleukin; LTA4H, leukotriene A4 hydrolase; MoA, mechanism of action; 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

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	



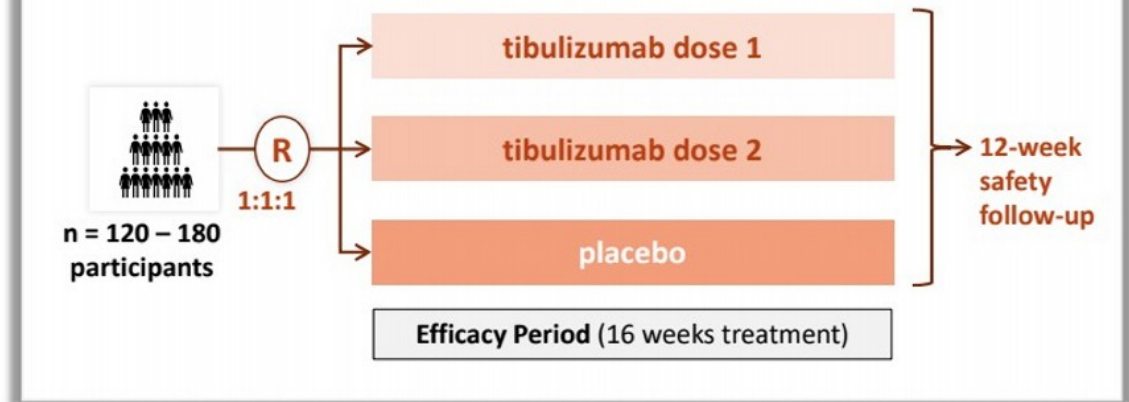
*The sHiSCR is defined as a $\geq 50\%$ reduction in the abscess and inflammatory nodule count and no increase in draining tunnels compared with baseline. †Difference refers to the difference between remibrutinib (either dose) and pooled placebo at Week 16. ‡Bayesian posterior probability of remibrutinib (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

DOUBLE BLIND, PLACEBO-CONTROLLED, 16-WEEK STUDY



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-7R α + TSLP

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

Crebankitug, a fully human IL-7R α antibody	Well tolerated in Phase 1 and Phase 1b studies	Phase 1b data demonstrate clear evidence of impact on key T-cell compartments	Actively assessing Phase 2 strategy
<ul style="list-style-type: none">Originally developed by PfizerIL-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.	<ul style="list-style-type: none">>90 participants dosed with crebankitugAdverse events generally mild and not treatment-related.	<ul style="list-style-type: none">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.	<ul style="list-style-type: none">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

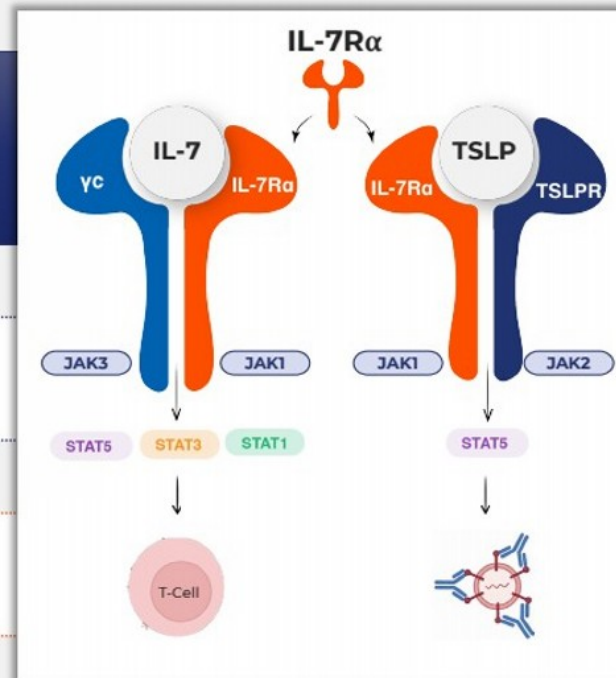
IL-7R α is a key receptor in immune regulation, central to the signaling of cytokines **IL-7** and **TSLP**

IL-7

IL-7R α collaborates with the common gamma chain (γ c) 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-7R α joins the mix, creating a composite complex with TSLPR and TSLP

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

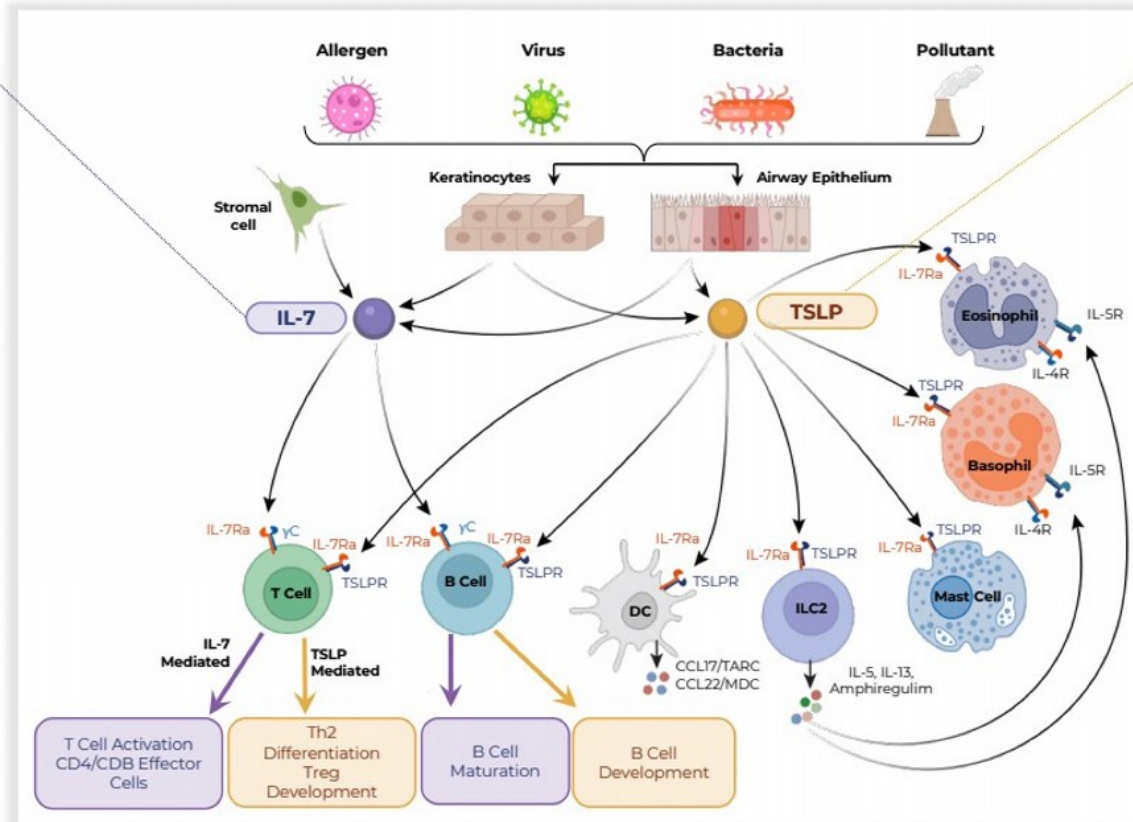
Commonly tied to allergic responses and specific inflammatory scenarios

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

IL-7 PATHWAY

- IL-7 is a growth factor that binds to the heterodimeric receptor of IL-7R α : γ C 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

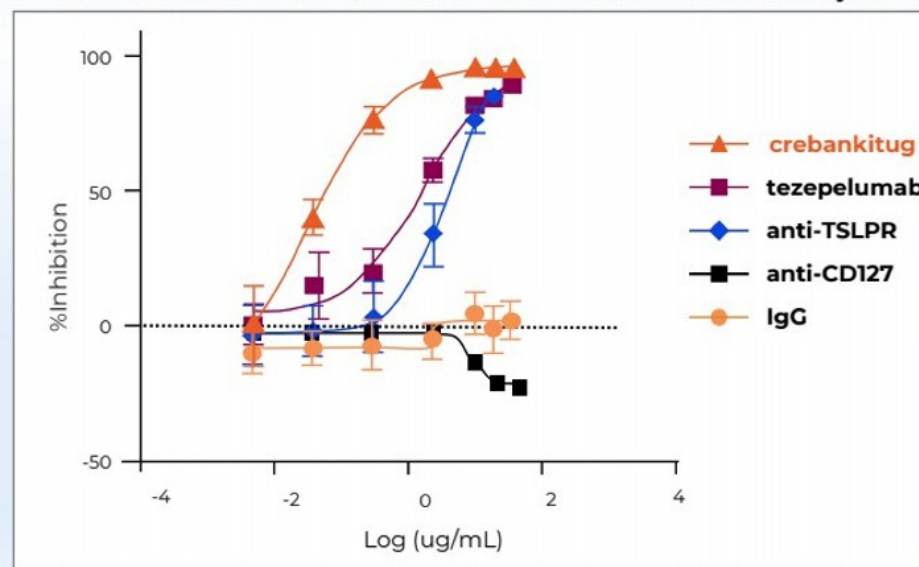
- Thymic stromal lymphopoietin (TSLP) is an epithelial-derived cytokine primarily expressed in the lungs, skin and gastrointestinal tract ¹
- TSLP is released from the epithelium by disease amplifying Th2 immune response, including the production of IL-4, -5, -9 and -13. ¹
- TSLP inhibition is clinically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

Sources: ¹ Ebina-Shibuya, R. and Warren Leonard. 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 Sawides S. Frontiers in Immunology, doi:10.3389/fimmu.2020.01557.

Inhibition of TSLP stimulated CCL17 secretion from human monocytes

	  tezepelumab (TSLP) TSLP mAb	 bempikibart (IL-7Rα) IL-7R α mAb	 crebankitug (IL-7Rα) IL-7R α 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⁾

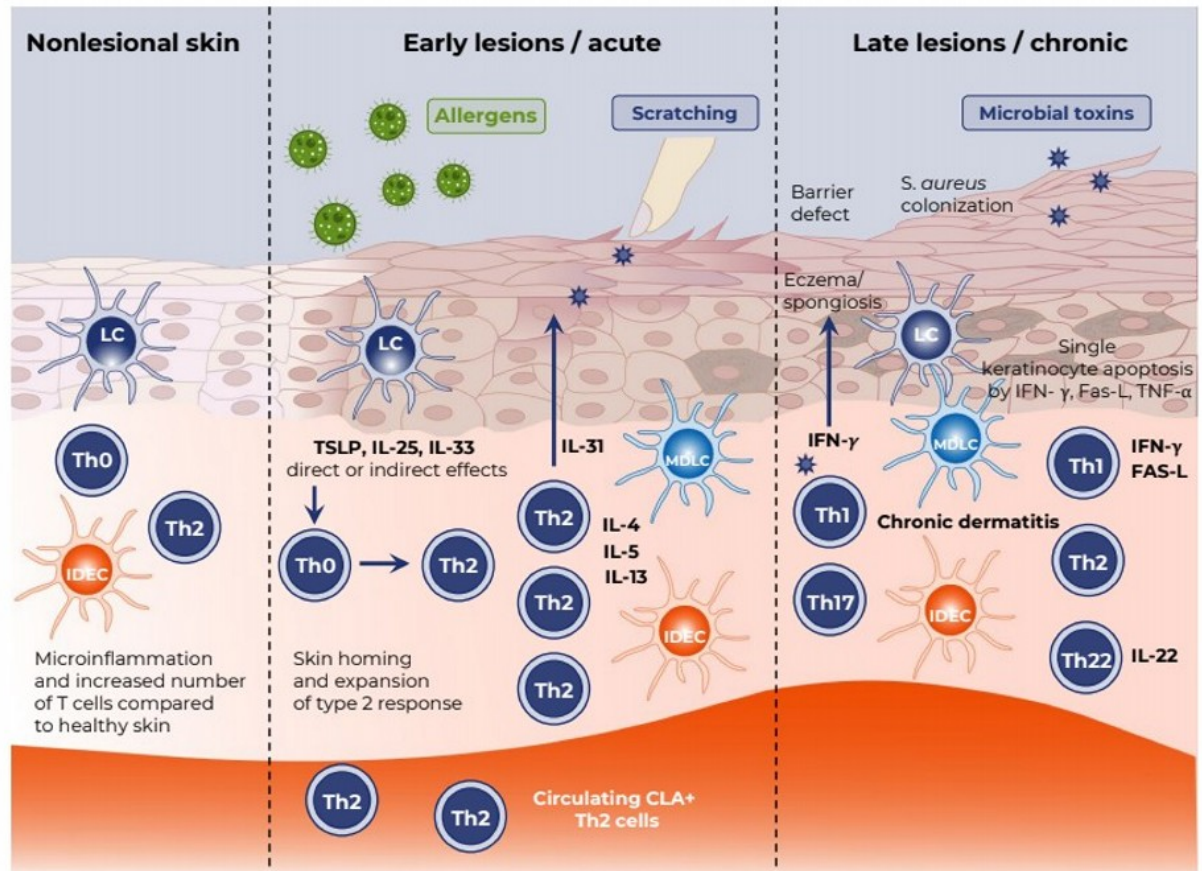
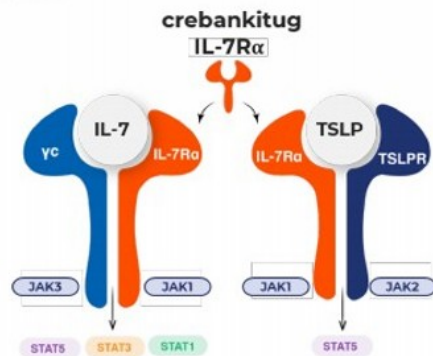
World Allergy Congress Poster, Dec 2023 % inhibition of TSLP stimulated CCL17 secretion from human monocytes



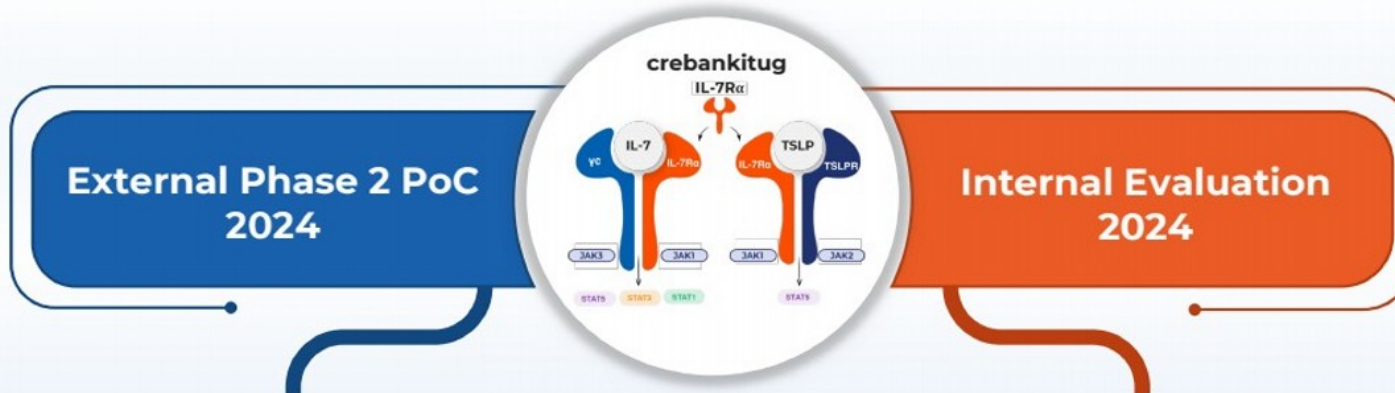
Source: ¹ Zura Internal Data; ² Herold, Kevan C., et al. JCI Insight, doi:10.1172/jci.insight.126054; ³ Numazaki, Mako, et al. Journal of Pharmacology and Experimental Therapeutics, doi:10.1124/jpet.121.006886; ⁴ Yamniuk, Aaron P., et al. Antibodies against IL-7r Alpha Subunit and Uses Thereof. 18 May 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

- 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



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 Indications²

- Common autoimmune disease
- Orphan autoimmune disease

Sources: ¹ClinicalTrials.gov database, Company Presentations; ²Zura 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-7R α 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.

About torudokimab

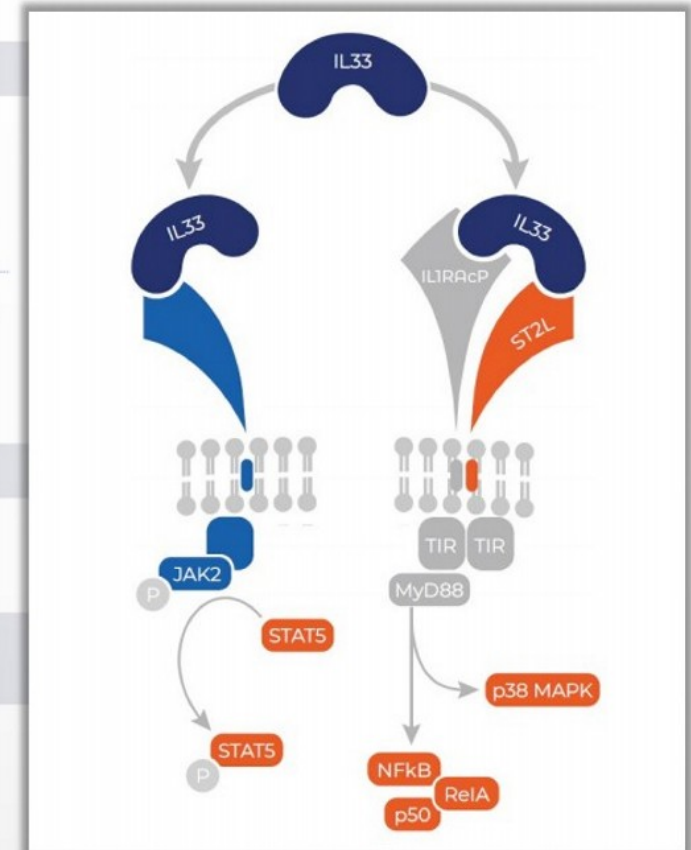
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| <p>01 IL-33 implicated in driving Th2 biology through ST2 and Th1/Th17 through alternative signaling ¹</p> <p>03 The target engagement evaluation suggested binding to IL-33 and treatment emergent ADA had no apparent impact on PK or target engagement</p> | <p>02 Well tolerated in Phase 1 and Phase 2 trials conducted by Eli Lilly ²</p> <p>141 healthy volunteers in Phase 1 study</p> <p>Analyses confirmed key biomarker reductions (IL-13, periostin and CCL17/TARC) and no ADA impact ³</p> | <p>103 participants with moderate to severe atopic dermatitis in Phase 2</p> <p>Potential utility in diseases driven by epithelial inflammation ¹</p> |
|--|--|--|

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

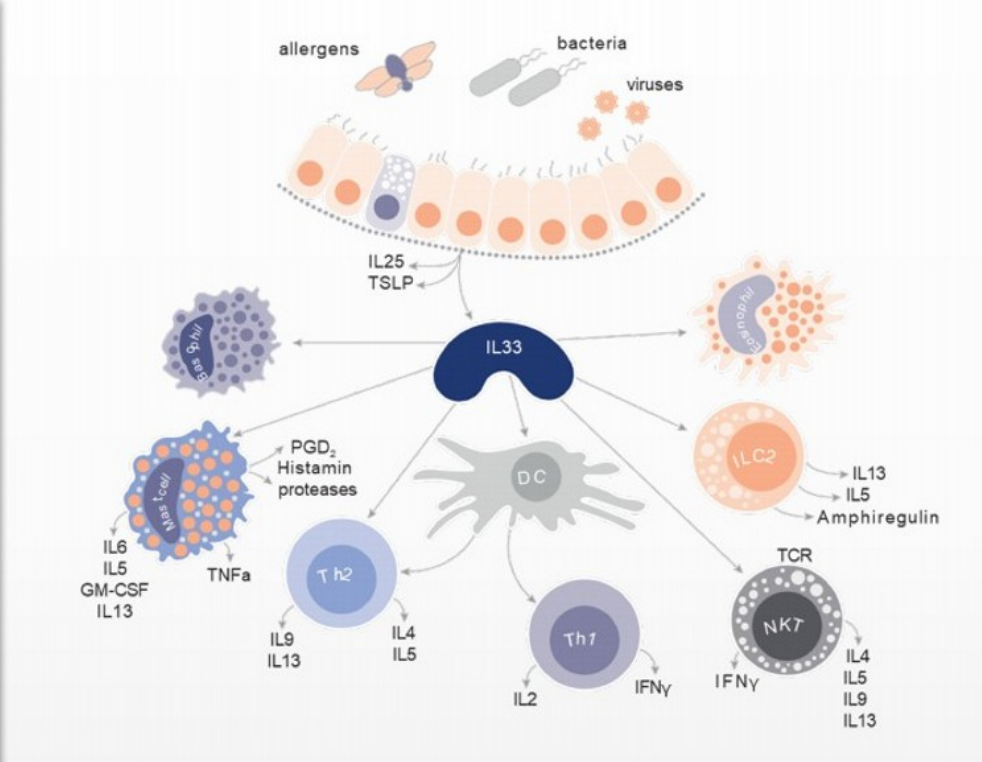
- 01** Potential for 1st-in-class opportunities **02** Validated pathways in COPD ⁴ and asthma ⁵



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

<p>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</p>	<p>IL-33 is released from the epithelium by disease exacerbating environmental factors and is an important amplifier of innate inflammation during exacerbations ²</p>
<p>Polymorphisms in IL-33 and ST2 are associated with increased eosinophil production as well as respiratory diseases such as COPD and severe asthma</p>	<p>IL-33 inhibition clinically validated in severe asthma, COPD ³, and subsets of other epithelial disorders ⁴</p>
<p>Pre-clinical data demonstrates superior activity of torudokimab compared with etokimab and itepekimab suggesting the potential for best-in-class activity ⁵</p>	<p>Emerging biology suggests expanding opportunity for IL-33 in fibrotic and autoimmune conditions ⁶</p>



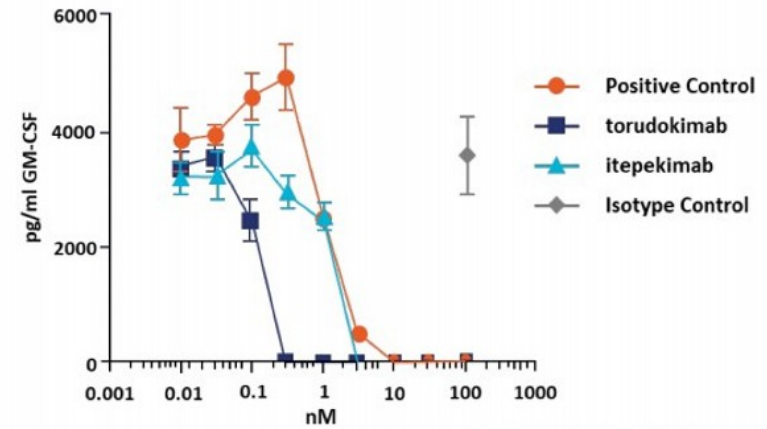
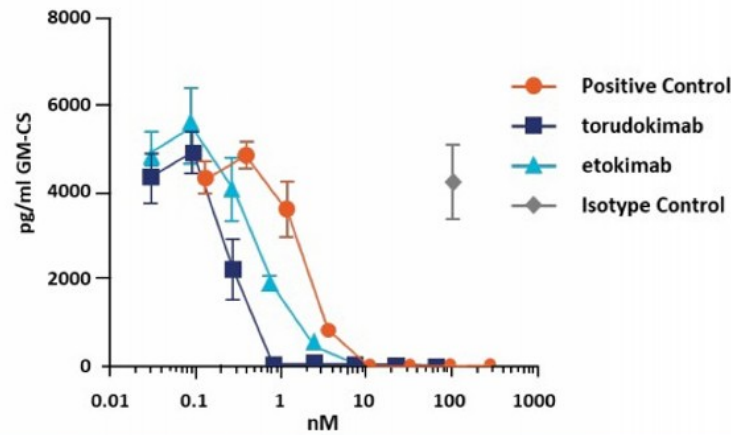
Sources: ¹ Chan, B., et al. *Frontiers in Immunology*, doi:10.3389/fimmu.2019.00364; ² Cayrol, C. and Girard, J.P. *Cytokine*, doi:10.1016/j.cyto.2022.155891. ³ Gudbjartsson, D., et al. *Nature Genetics*, doi:10.1038/ng.323; Ketelaar, M., et al. *Journal of Allergy and Clinical Immunology*, doi:10.1016/j.jaci.2020.04.051; 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 | ZB-880

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



Corporate Update








Team and Cash Runway

Zura's Leadership: Driving Innovation in I&I






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 <p>Michael Weinblatt, MD</p>	 <p>Steven Ziegler, PhD</p>	

Key milestones* expected through 2026

Cash runway expected through 2027

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

(*) 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.

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Our mission: Driving scientific breakthroughs by turning drug discoveries into transformative, life-saving treatments.
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Tibulizumab shows best-in-class potential: Introducing a tetravalent antibody therapy designed to target and potentially treat autoimmune diseases.
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Promising pipeline for value creation: Integrating validated biological pathways into multifunctional antibody assets to potentially improve therapeutic outcomes.
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Upcoming external catalysts: Anticipating near-term developments that could further expand the pipeline's potential.
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Proven leadership: Experienced team with a track record of contributing to over \$8 billion in mergers and acquisitions in the past three years.
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Strong financial position: With approximately \$188 million¹ 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 outstanding².

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