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Advancing Dual-Pathway Biologic Candidates:

Addressing Unmet Needs in Autoimmune and Inflammatory Diseases

January 2025

Nasdaq Ticker: ZURA

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Company Summary

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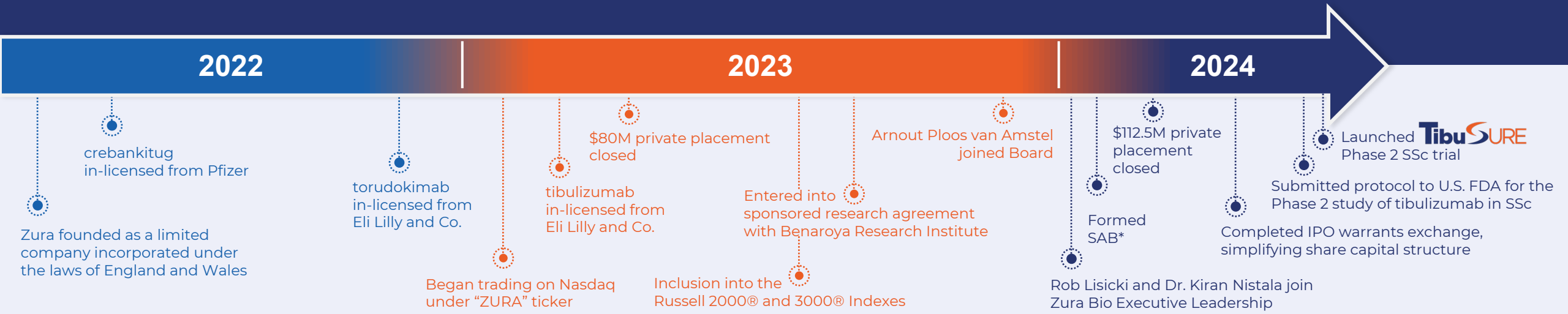
High-Potential Biologics: Three dual-pathway biologics targeting multi-billion-dollar markets, with available Phase 1/1b data and potential to advance to Phase 2 trials

Lead Asset Development: Tibulizumab Phase 2 study for SSc is ongoing, with a subsequent trial for HS anticipated to initiate 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 expected to support planned operations through 2027



(*) A Scientific Advisory Board (SAB) is a panel of experts from relevant scientific fields convened to provide guidance, expertise, and strategic recommendations to organizations, institutions, or projects. SABs typically ensure that decisions and actions are based on sound scientific principles, promote innovation, and maintain credibility in research or development initiatives.

Sources: Zura Bio Press Releases and Filings

Acronyms: FDA, Food and Drug Administration; HS, hidradenitis suppurativa; IPO, Initial Public Offering; Q, quarter; SAB, Scientific Advisory Board; SSc, systemic sclerosis.

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

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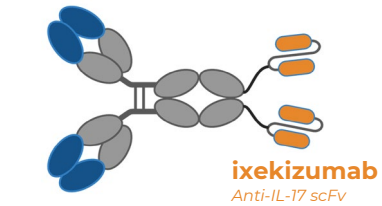
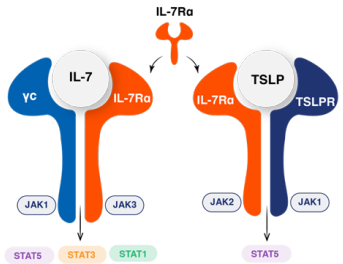
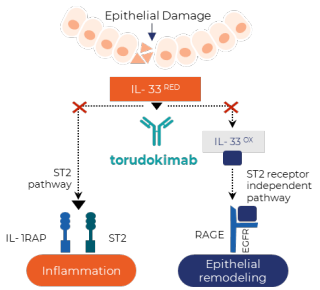
		Phase 1	Phase 2
tibulizumab	 **Only bispecific antibody targeting IL-17A and BAFF**	Tibulizumab has been studied in three Phase 1/1b clinical studies to date * ✓ 78 participants dosed across 3 studies – n=57 single dose – n=21 multiple doses up to 12 weeks	Phase 2 study for SSc is ongoing, with a subsequent trial for HS anticipated to initiate in 2Q 2025
crebankitug	 **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 evaluating potential therapeutic indications to guide our future development efforts.
torudokimab	 **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 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 Sjögren's syndrome, and 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, and 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, and 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.

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)

Systemic Sclerosis is a Multi-Organ Disease with No Comprehensive Treatment*

Systemic sclerosis is a rare & life-threatening disease

~300,000

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

Zero

SSc-specific * drugs approved

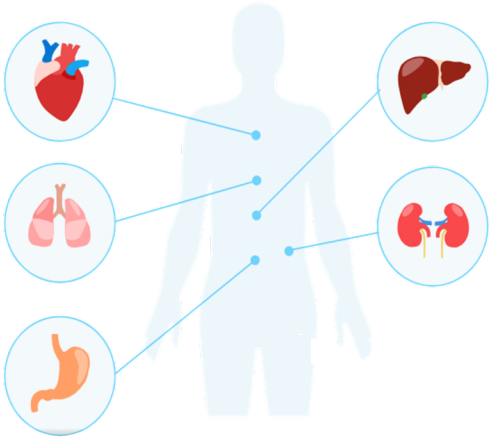
Systemic sclerosis is characterized by *tissue inflammation* and *fibrosis*

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

(*) Two approved therapies are available for SSc-ILD; however, no treatment addresses the disease across organ systems.

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

Acronyms: ILD, interstitial lung disease; SSc, systemic sclerosis.

Tibulizumab milestones* expected through 2026

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MILESTONE LEGEND:

- ❑ Internal Zura
- ▲ External Catalyst

Cash runway expected through 2027

		2H-2024	2025	2026
tibulizumab	TibuSURE Phase 2 SSc study	✓ Select CRO ✓ Submit Protocol to IND for Phase 2 Study in SSc ✓ Initiate a Phase 2 study in SSc	❑ Phase 2 study recruitment	❑ (4Q-2026) SSc Topline Data *Pending data outcomes, Phase 3 → → →*
	HS study	✓ Select CRO	❑ (1Q-2025) Submit Protocol to IND for Phase 2 Study in HS ❑ (2Q-2025) Initiate a Phase 2 study in HS	❑ (3Q-2026) HS Topline Data *Pending data outcomes, Phase 3 → → →*
	External Catalysts		▲ 2025 (NVS): Topline Data for VAY736 (ianalumab) from a Phase 2 Platform Study in HS Patients	

(*) The timing of regulatory and clinical trial milestones is subject to change and may require further interactions with the FDA.
Sources: Internal Zura Planning, clinicaltrials.gov
Acronyms: CRO, contract research organization; HS, hidradenitis suppurativa; IND, Investigational New Drug; Q, quarter; SSc, systemic sclerosis; US FDA, United States Food and Drug Administration.

Rationale for Phase 2 Study in Systemic Sclerosis Patients

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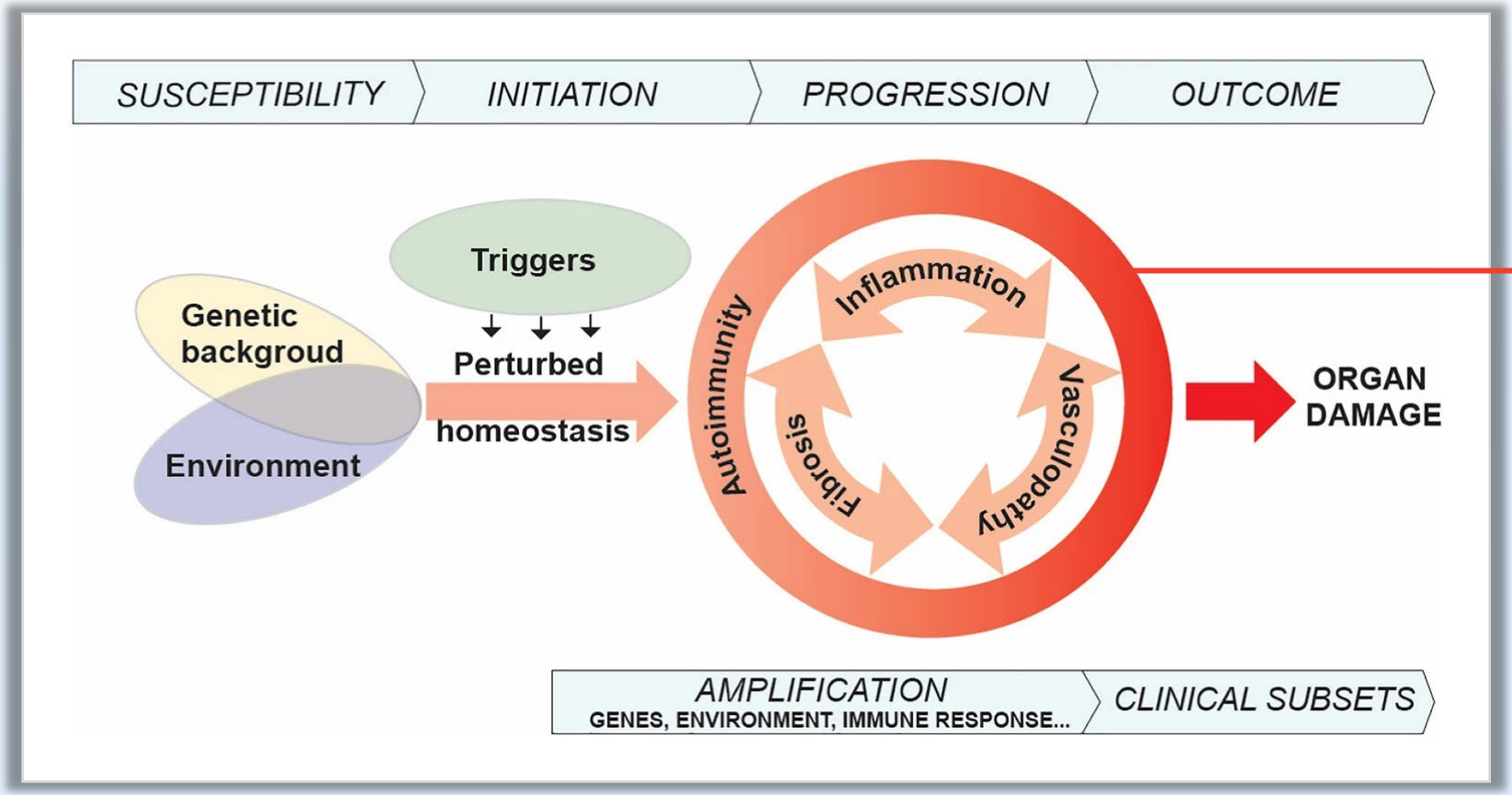
TAM projected to exceed $2B

1. **IL-17A and BAFF both contribute to the pathogenesis and severity of SSc**
2. **Pre-clinical data supports potential for broader or deeper effect with tibulizumab**
   - Demonstrated greater reductions in disease severity compared to IL-17 or BAFF alone in the CIA murine model.
   - Achieved greater reductions in erythema, skin thickness, and scaling compared to IL-17 or BAFF alone in bleomycin murine model.
   - Showed greater reductions in lung fibrosis relative to the standard control.
3. **Clinical evidence of skin and/or lung benefit across multiple direct and indirect mechanistic trials**
   - IL-17 Receptor: (e.g., brodalumab, Phase 1 & 3 trials)
   - CD20 Antagonists: (e.g., rituximab, Phase 2)
   - BAFF Inhibition: (e.g., belimumab, 52-week IIT study)
   - IL-23 Pathway: (e.g., guselkumab, Phase 2 trial)

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

Targeting IL-17 and BAFF to Disrupt SSc Pathophysiology and Potentially Halt Inflammation, Autoimmunity, and Fibrosis



- SSc progresses through interconnected pathways of inflammation, autoimmunity, vasculopathy, and fibrosis, driven by genetic and environmental triggers.

- **IL-17 amplifies inflammation, promotes vascular injury, and perpetuates tissue damage**, making it a pivotal target to disrupt the inflammatory cycle.

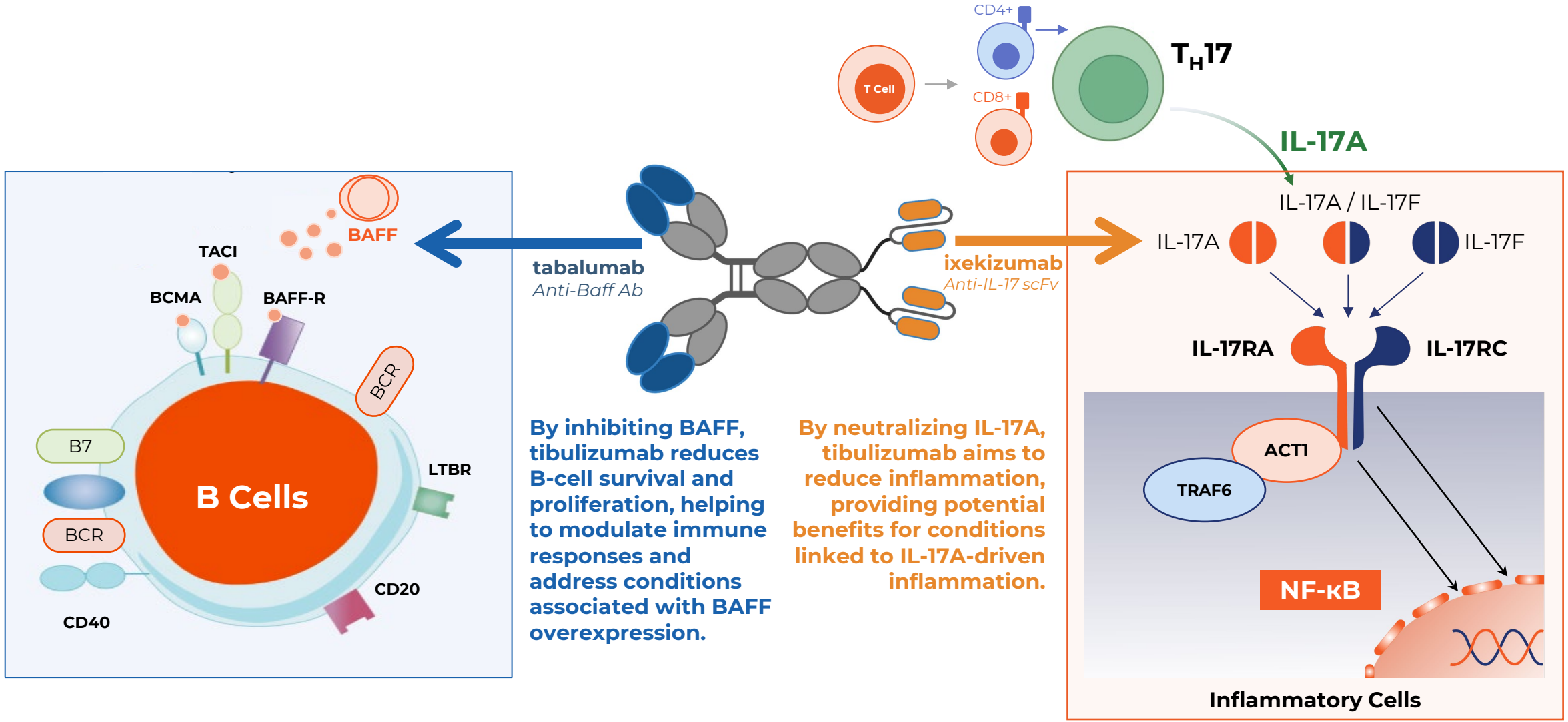
- **BAFF drives B-cell survival and autoantibody production, fueling immune dysregulation and accelerating fibrosis**, presenting a unique opportunity for therapeutic intervention.

Targeting IL-17 and BAFF offers a transformative approach to breaking the amplification loop, mitigating disease progression, and preventing irreversible organ damage in SSc.

Sources: Truchetet, M.E., et al. *Clinical Reviews in Allergy & Immunology*. doi:10.1007/s12016-021-08889-8
Acronyms: BAFF, B cell-activating factor; IL, interleukin; SSc, systemic sclerosis.

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

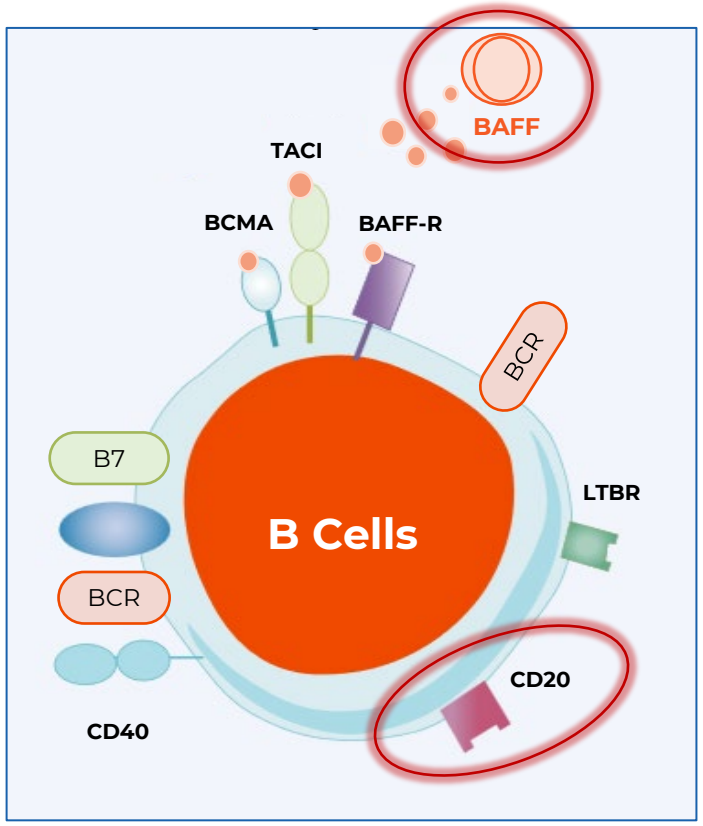
Tibulizumab uniquely targets BAFF for B cell regulation and IL-17A for inflammation control, offering a comprehensive approach to treating autoimmune and inflammatory diseases.



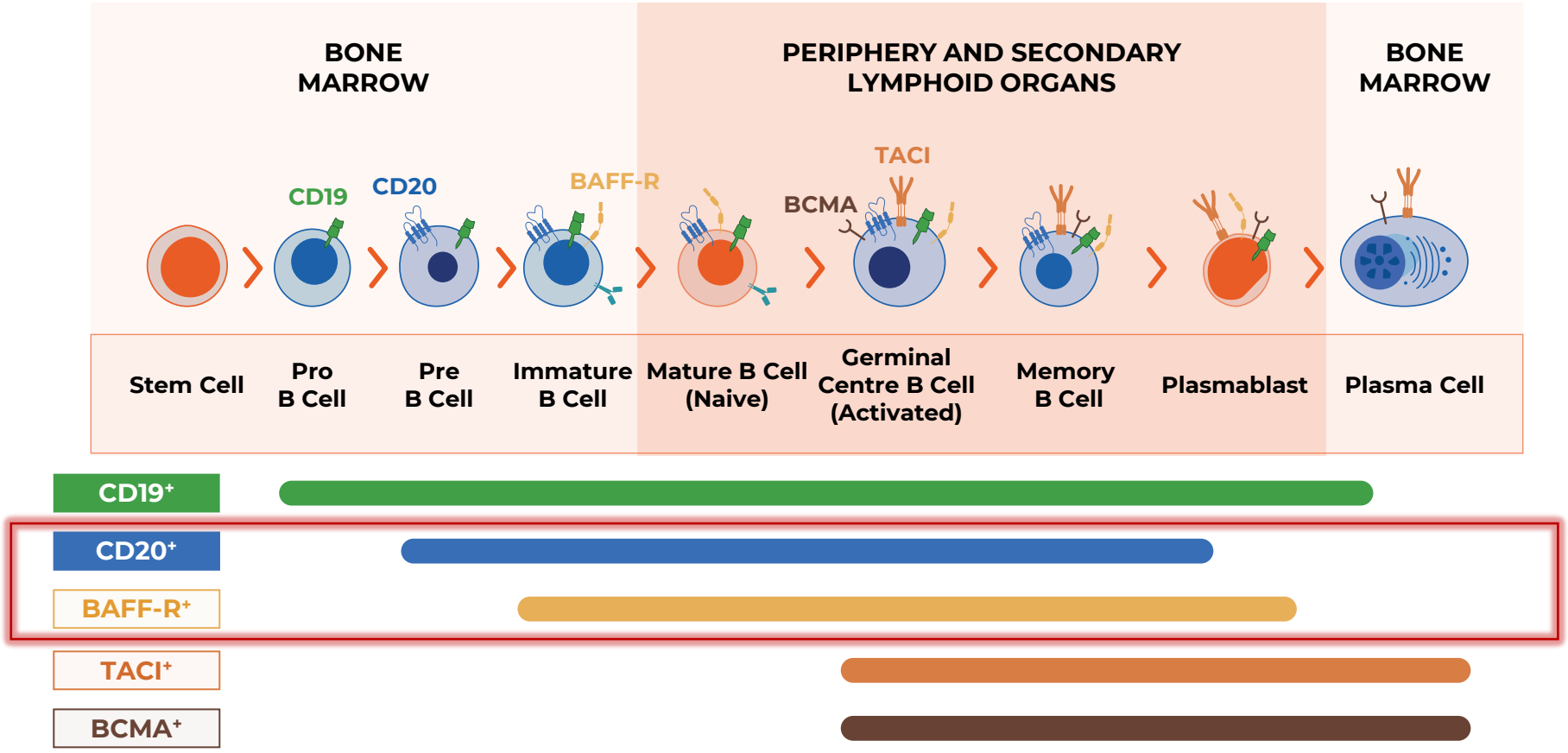
By inhibiting BAFF, tibulizumab reduces B-cell survival and proliferation, helping to modulate immune responses and address conditions associated with BAFF overexpression.

By neutralizing IL-17A, tibulizumab aims to reduce inflammation, providing potential benefits for conditions linked to IL-17A-driven inflammation.

Numerous B Cell Therapies have Overlapping B Cell Targets, BAFF and CD20 Share High Overlap

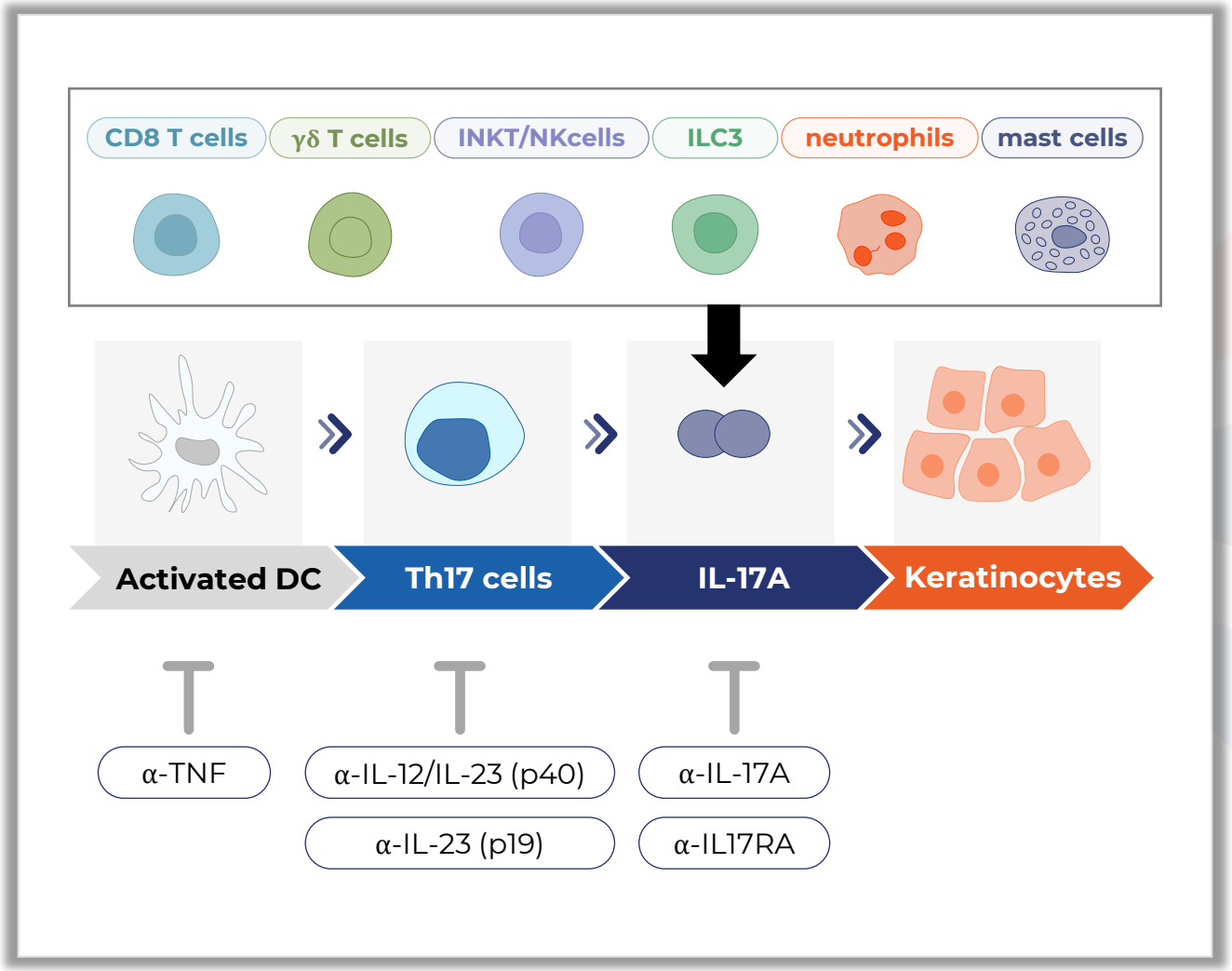


Cell Surface Antigen Expression



Sources: Figured adapted from Baker D, et al., *EBioMedicine*. doi: 10.1016/j.ebiom.2017.01.042
Acronyms: BAFF-R, B-Cell activating factor receptor; BCMA: B-Cell maturation antigen; BCR: B-Cell receptor; CD, cluster of differentiation; LTBR: lymphotoxin beta receptor; TACI: transmembrane activator and CAML interactor

The IL-23 / IL-17 Axis Plays an Important Role in Multiple Inflammatory Diseases, Including SSc



IL-23 plays a pivotal role in stimulating the production of IL-17 by activating the Th17 cells. The IL-23/IL-17 axis is an important pathway for targeted therapy for inflammatory diseases.

IL-17A is associated with the pathogenesis of inflammatory diseases, including systemic sclerosis.

Clinical trials have shown that monoclonal antibodies against IL-23 and IL-17, are effective in the treatment of patients with systemic sclerosis.

Sources: Brembilla, N.C., et al. (2018). The IL-17 Family of Cytokines in Psoriasis: IL-17A and Beyond. Frontiers in Immunology, 9:1682. doi:10.3389/fimmu.2018.01682

Acronyms: α-IL17RA, anti-interleukin-17 receptor A; α-TNF, anti-tumor necrosis factor; CD8, cluster of differentiation 8; DC, dendritic cells; IL-17A, interleukin-17A; ILC3, group 3 innate lymphoid cells; INKT/Nk cells, invariant natural killer T cells/natural killer cells; SSc, systemic sclerosis; Th, T-helper; γδ T cells, gamma-delta T cells.

In Pre-Clinical Model, Simultaneous Blockade of BAFF & IL-17 Produced Greater Reduction in Disease Severity than IL-17 or BAFF Alone

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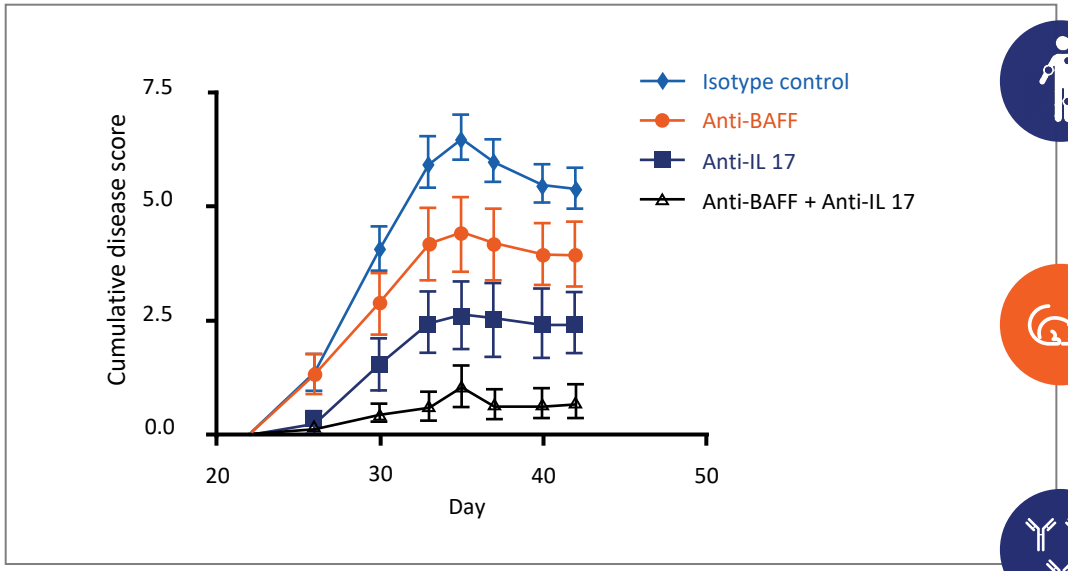

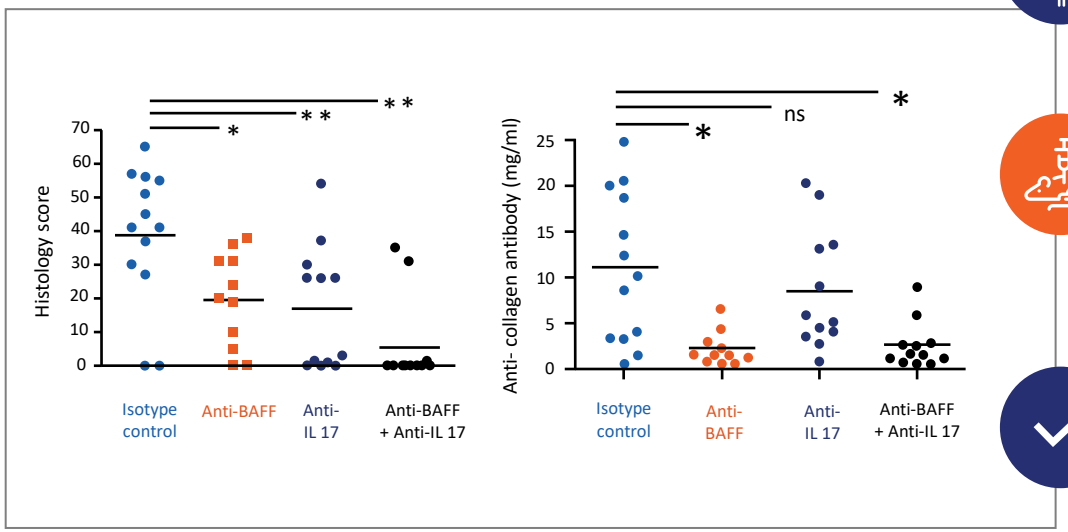

Rheumatoid arthritis is a prototypic autoimmune disease where individually targeting **IL-17A-mediated inflammation or depleting B cells** has been clinically validated

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.

TibuSURE

Tibulizumab Systemic Sclerosis Understanding and Response Evaluation

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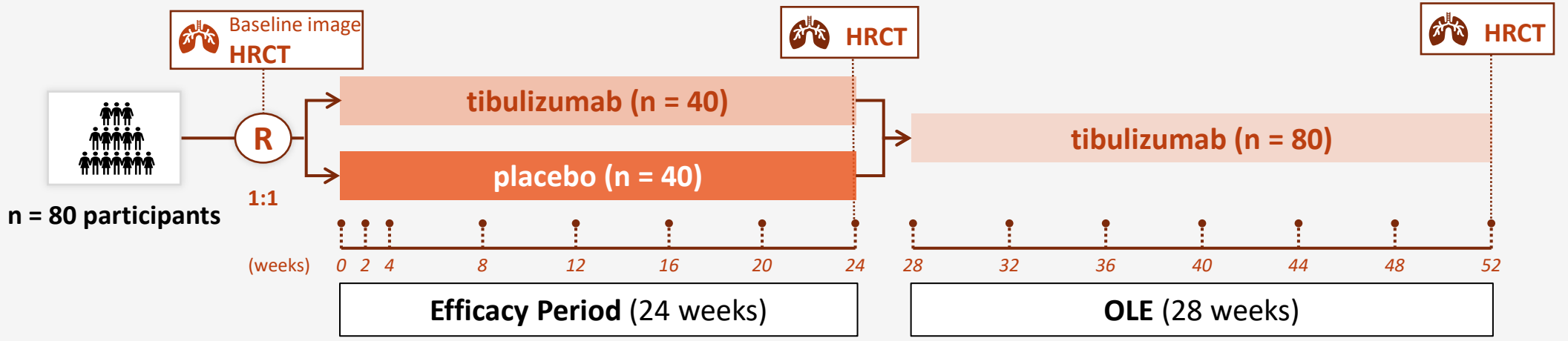
Study is Focused on Demonstrating Benefits for Skin and Lung Outcomes

RANDOMIZED PHASE 2 STUDY* (mRSS and HRCT)



KEY EFFICACY ENDPOINTS

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

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

Sources: Zura Internal Planning

Acronyms: FVC, forced vital capacity; HAQ-DI, health assessment questionnaire – disability index; HRCT, high-resolution computed tomography; ILD, interstitial lung disease; mg, milligram; mRSS, modified Rodnan skin score; OLE, open-label extension; qHRCT, quantitative high-resolution computed tomography; rCRISS, revised Composite Response Index in Systemic Sclerosis; RNA Pol III, RNA polymerase III; SSc, systemic sclerosis.

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

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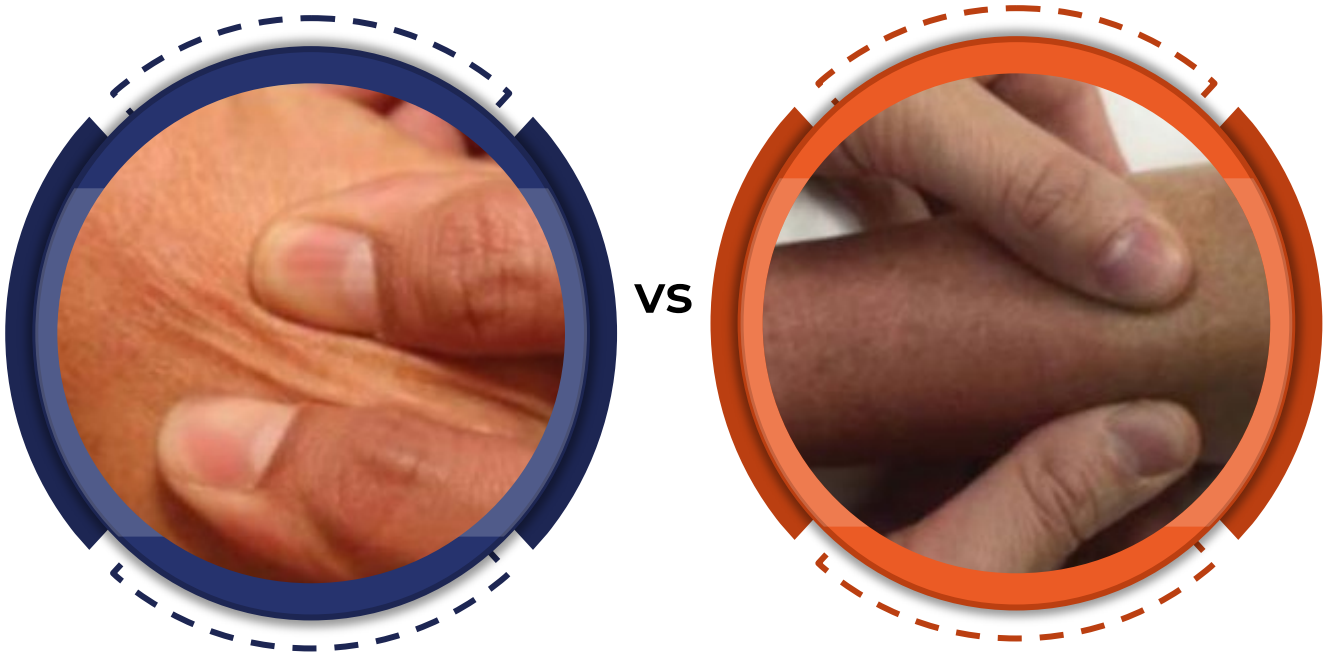


Fine Wrinkles[1] (0/3)

VS

Severe Thickness[1] (3/3)

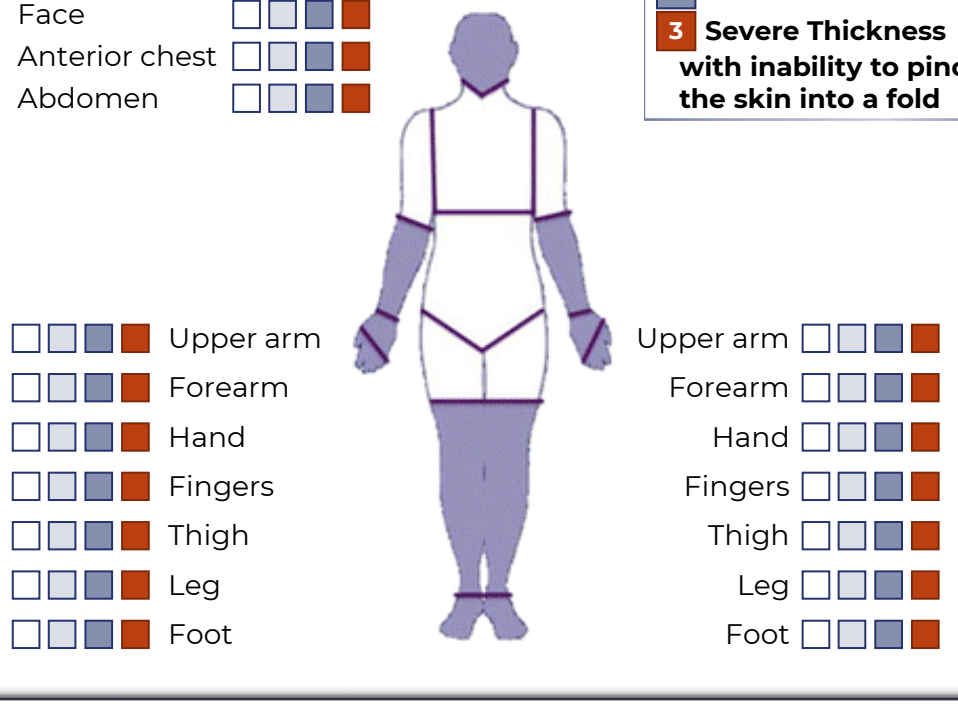
Severe skin thickening and tightening restricts movement and causes painful ulcers on the hands and fingers, significantly impairing daily activities and quality of life.

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**.

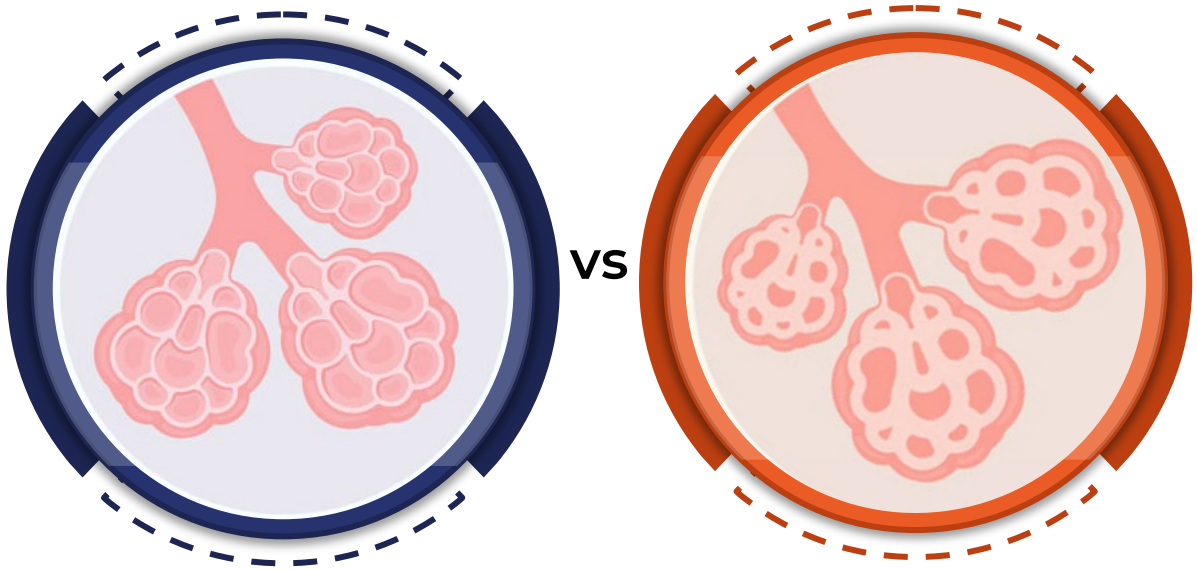
17 Surface Anatomic Areas of the Body

- 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

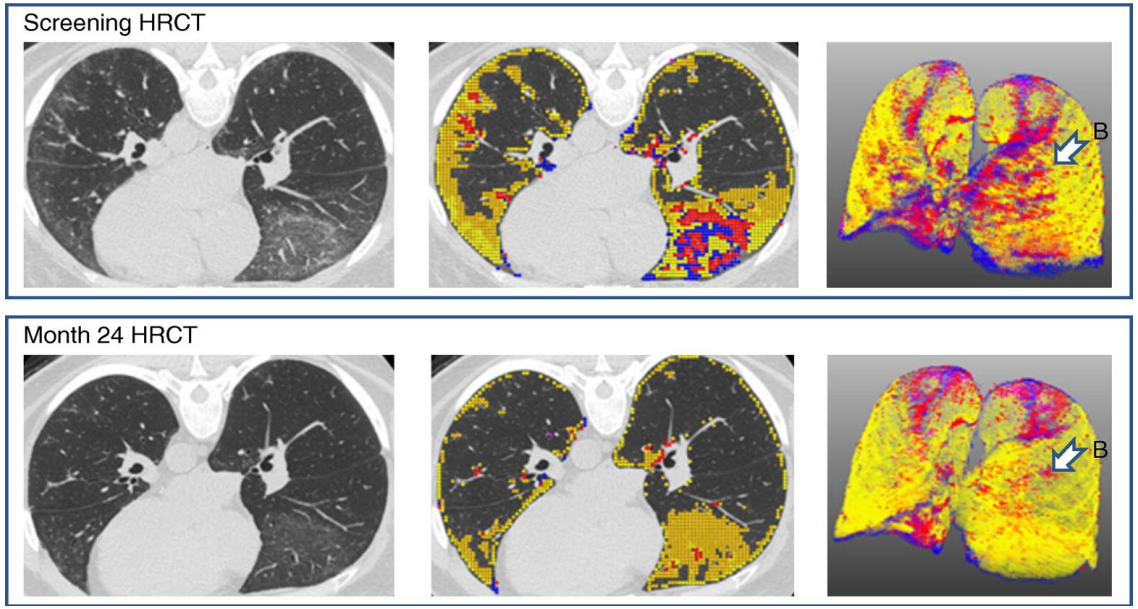
Fibrotic Lung

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.

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

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Historic Drivers of SSc Study Failures:

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

Increasing Probability of Success:

- Larger study sample sizes increase the probability of success for mRSS outcomes
- High-resolution CT shows strong correlation with FVC, potentially improving ILD read-through
- Adequate sample sizes for ILD readouts enable a better understanding of potential Phase 3 effects

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

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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 recognized as significant contributors to the pathology and progression of HS
- Ixekizumab targets IL-17A and IL-17A/F with high affinity
- Dual inhibition of IL-17A and BAFF may improve outcomes by addressing key drivers of HS

Unmet Need and Growth Potential

- > 50% 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 may address
- TAM is projected to reach $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

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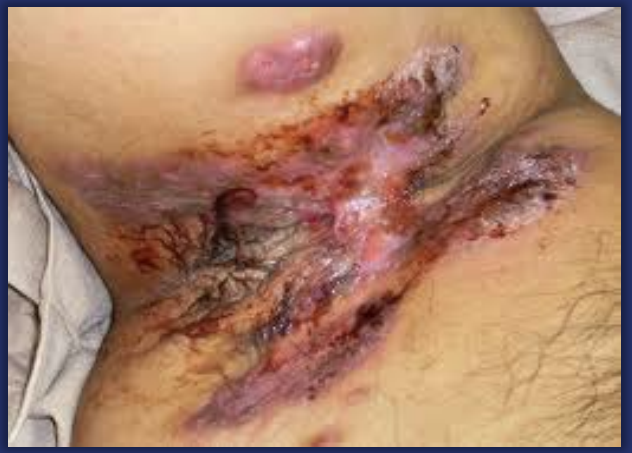
DISEASE OVERVIEW

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.

Acronyms: HS, hidradenitis suppurativa; Th-, T-helper cells; US, United States.

B Cell Signaling Potentiates HS Disease

zurabio

tibulizumab | ZB-106

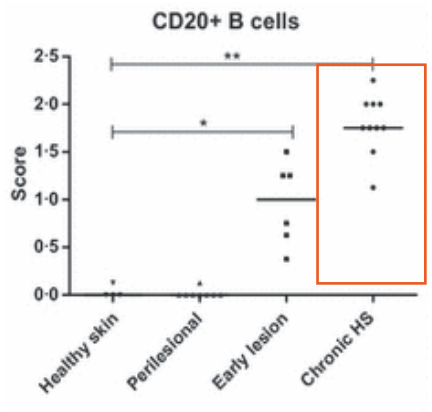
Elevated CD19 in HS **indicates increased B cell activation**, driving inflammation and correlating with higher CD20+ B cells and CD138+ plasma cells.

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

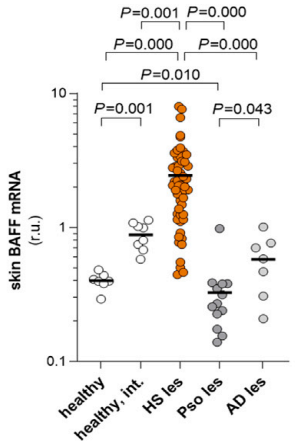
Reducing BAFF in HS lesions decreases B cell and plasma cell gene expression, indicating a potential therapeutic approach. [1,4]

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

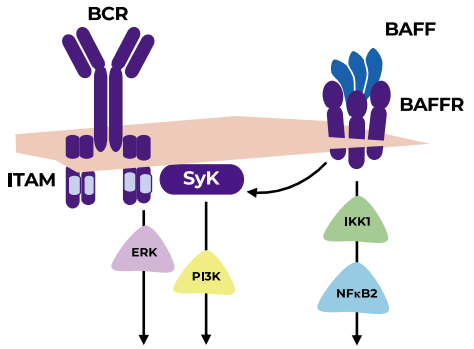
CD20+ B cells in HS Lesions



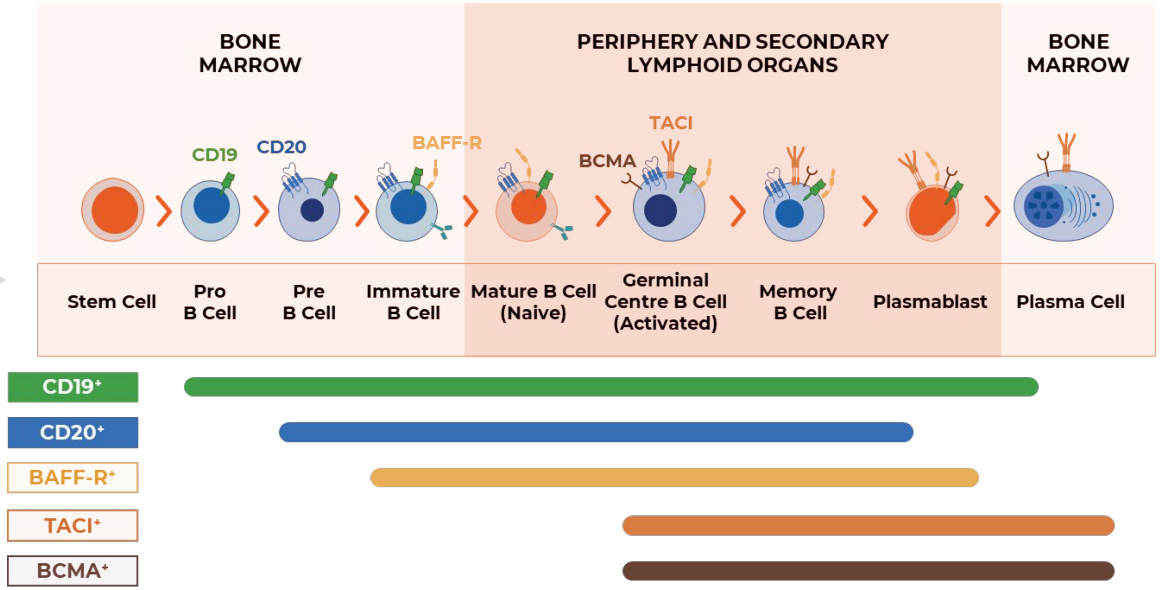
BAFF gene expression in HS



BAFF is essential for B cell activation



Cell Surface Antigen Expression



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

Acronyms: BAFF, B cell-activating factor; BCMA, B cell maturation antigen; CD-, cluster of differentiation; HS, hidradenitis suppurativa; TACI, transmembrane activator and calcium-modulator and cyclophilin ligand interactor.

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

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tibulizumab | ZB-106

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 supports more effective engagement with the target, which may contribute to 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

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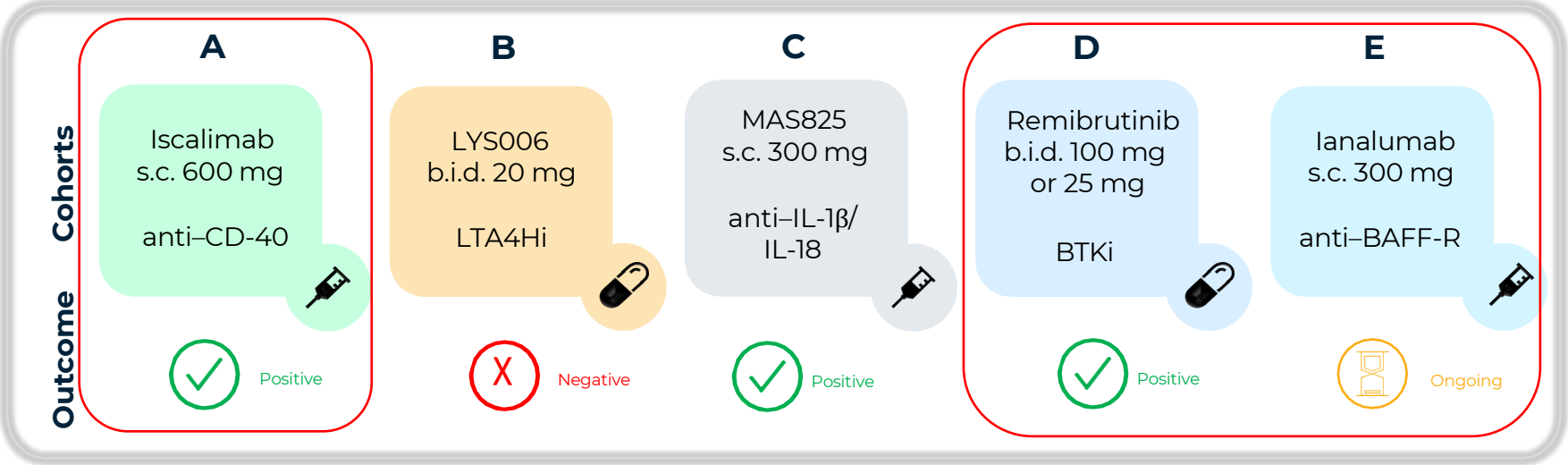
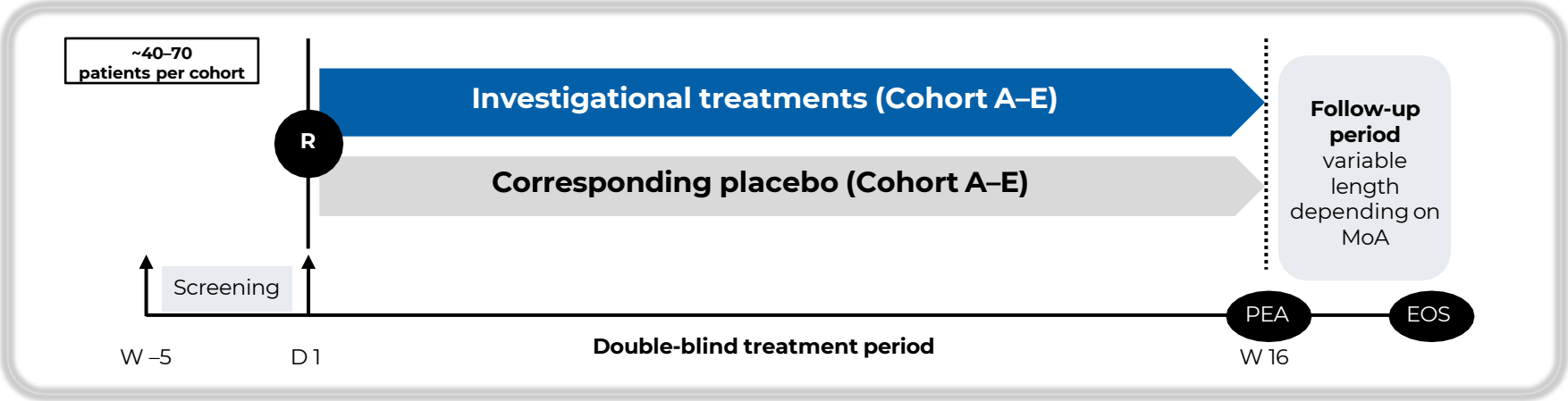
Presented at the American Academy of Dermatology Annual Meeting; March 8–12, 2024; San Diego, CA.





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

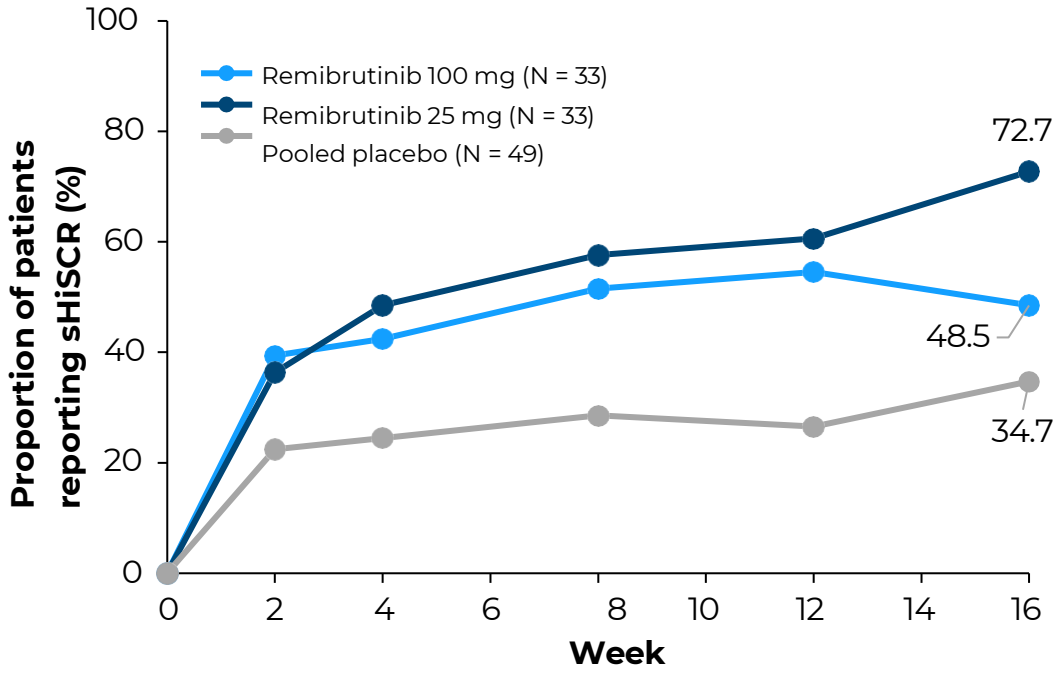
*Study started in February 2019 and is currently ongoing.
BAFF-R, B-cell activating factor of the tumor necrosis alpha family receptor; b.i.d., twice daily; BTKi, Bruton's tyrosine kinase inhibitor; CD, cluster of differentiation; D, day; EOS, end of study; HS, hidradenitis suppurativa; IL, interleukin; 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	

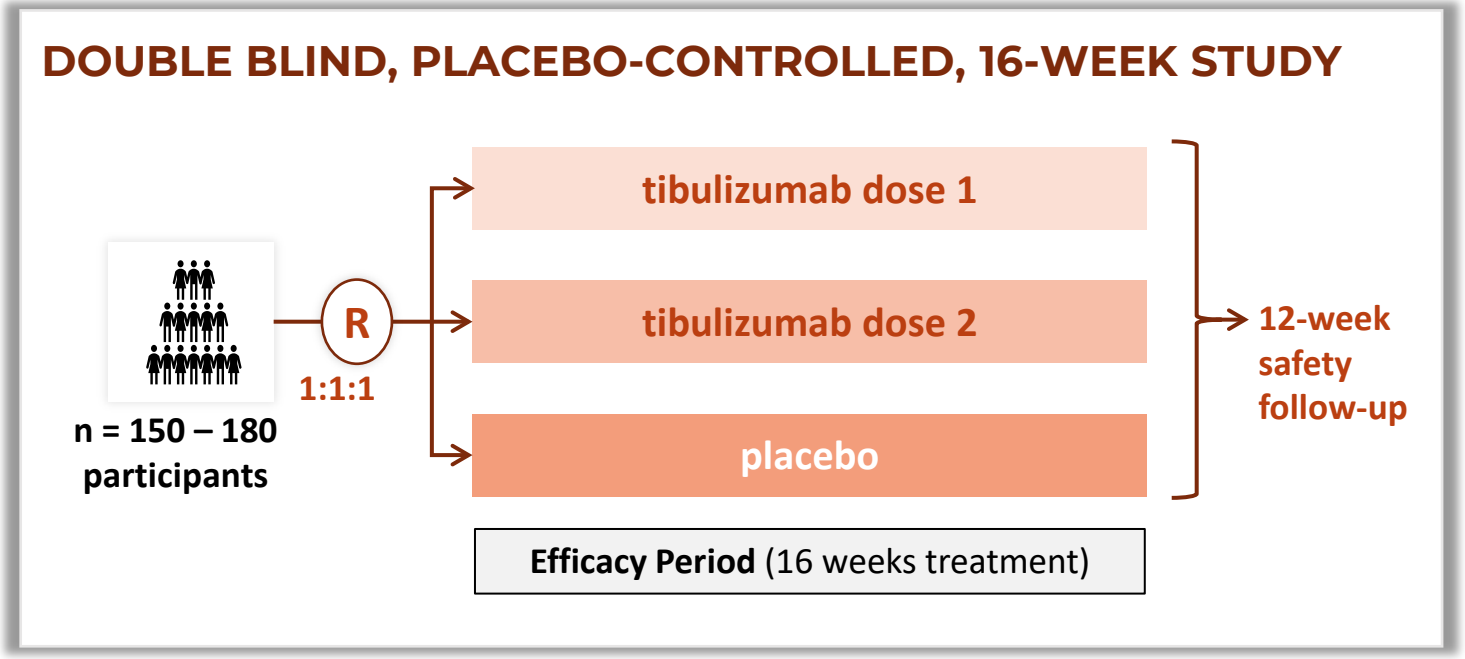


*The sHiSCR is defined as a ≥50% reduction in the abscess and inflammatory nodule count and no increase in draining tunnels compared with baseline. †Difference refers to the difference between remibrutinib (either dose) and pooled placebo at Week 16. ‡Bayesian posterior probability of remibrutinib (either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

- AN count
- HiSCR
- 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

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.

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

zurabio

crebankitug | ZB-168

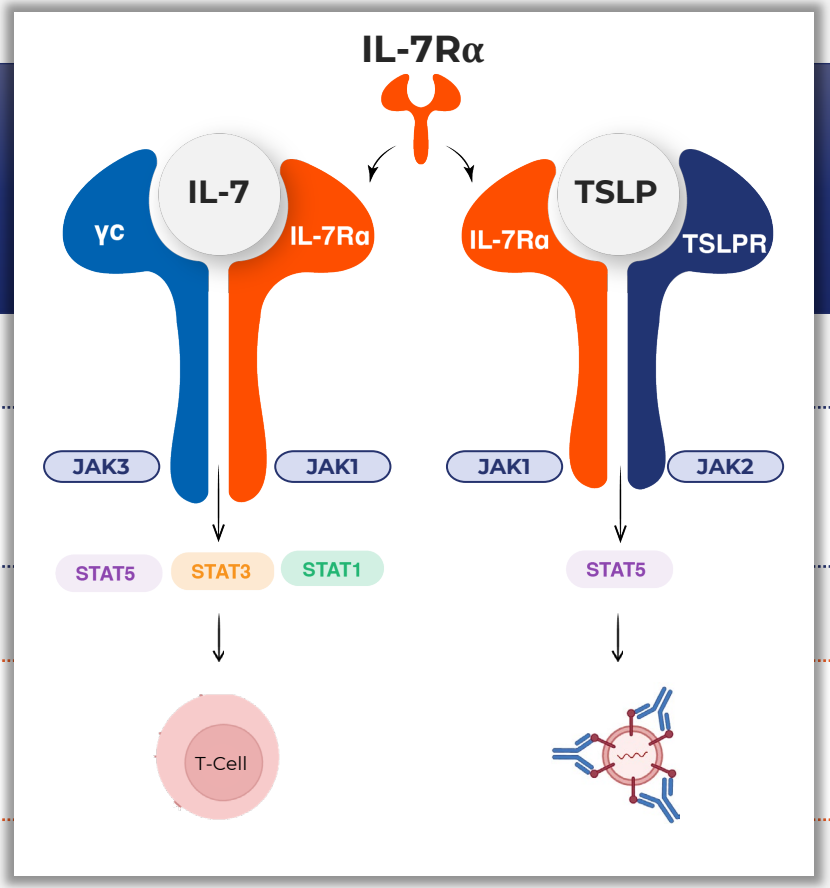
IL-7Rα is a key receptor in immune regulation, central to the signaling of cytokines ***IL-7*** and ***TSLP***

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**

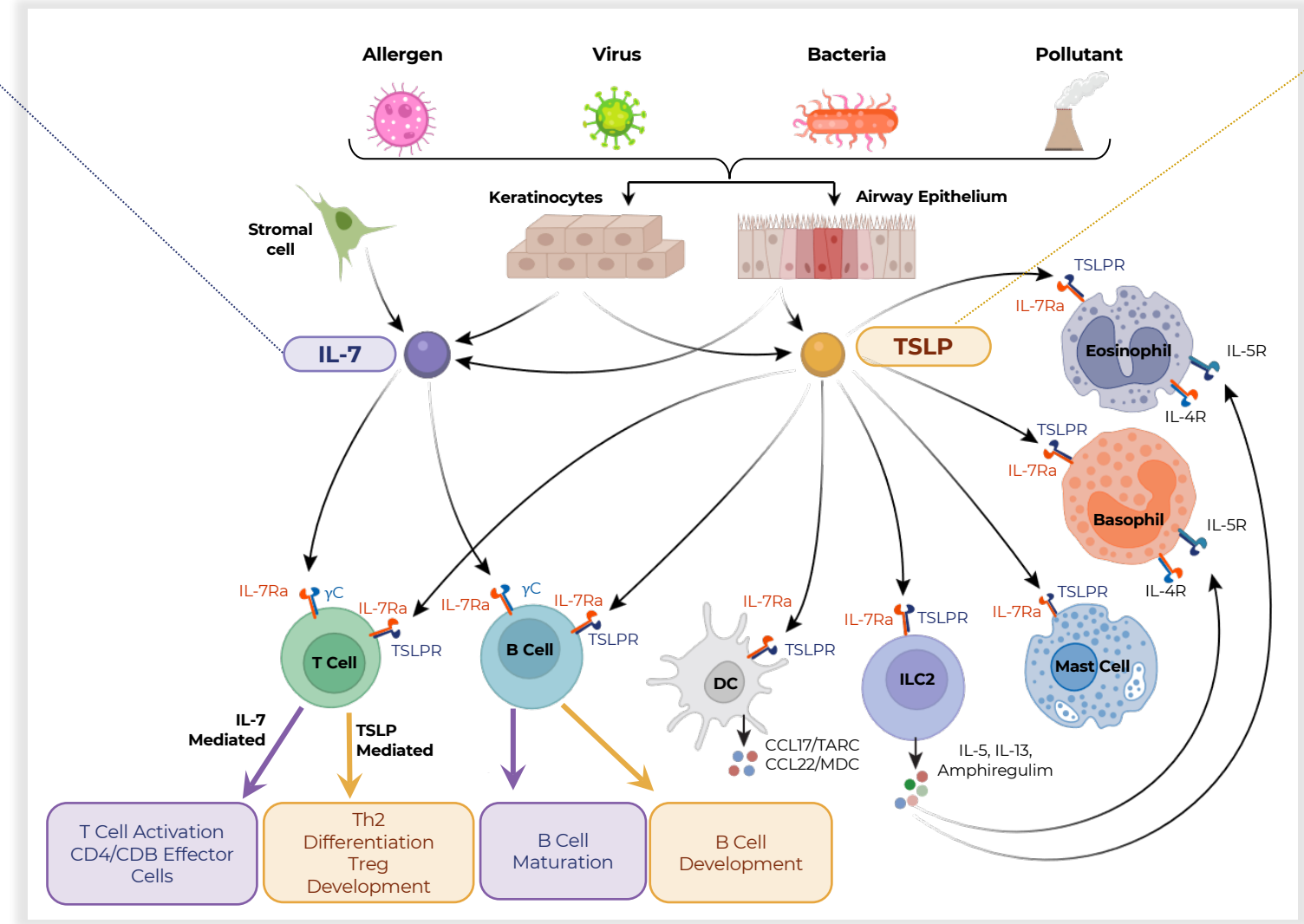
Commonly tied to allergic responses and specific inflammatory scenarios

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

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.

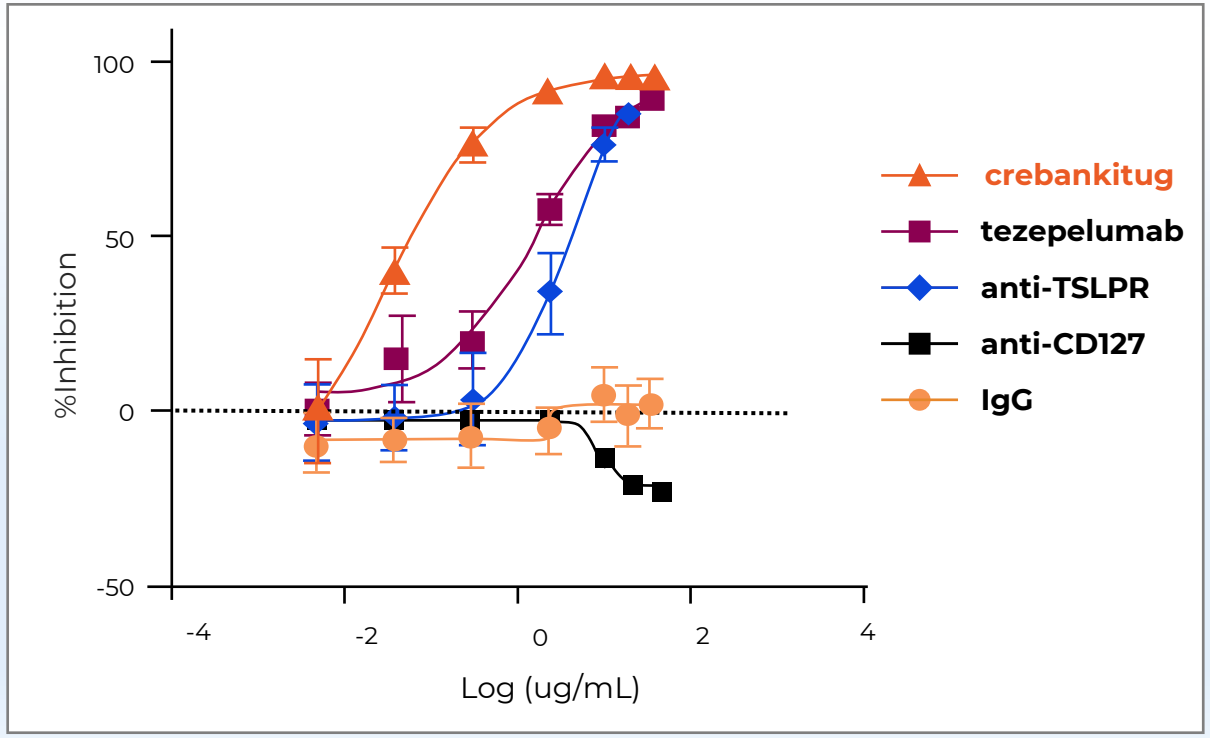
Crebankitug Is the Only mAb to Potently Inhibit Both IL-7R and TSLP

Inhibition of TSLP stimulated CCL17 secretion from human monocytes

	AstraZeneca / AMGEN	32 BIO	zurabio
	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

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

Currently engaged in indication planning

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]

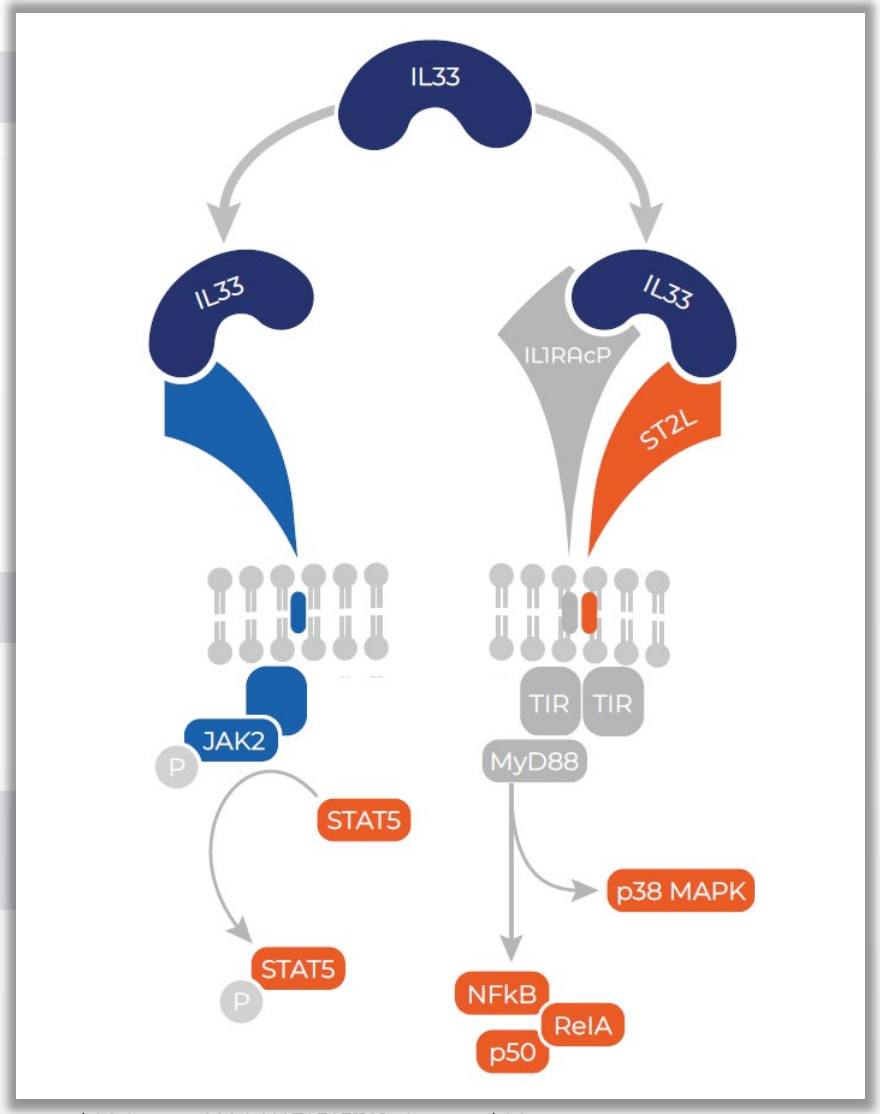
Mechanism of Action

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]

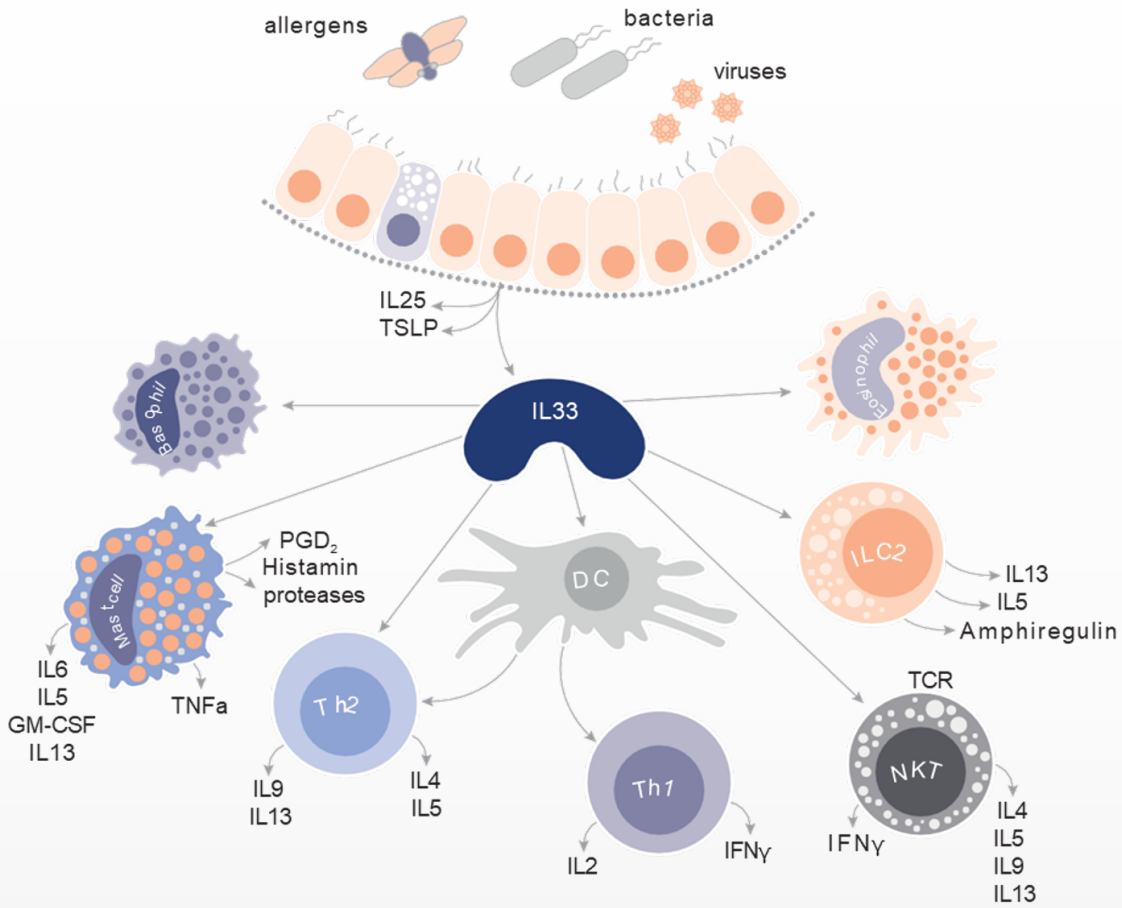


Sources: [1] Cohen, S., et al. *Nature Communications*. doi:10.1038/ncomms9327; [2] ClinicalTrials.gov. NCT03913260. Accessed 26 August 2024; NCT03343587. Accessed 26 August 2024; NCT03831191. Accessed 26 August 2024; Section 6.1, DSUR for the period 23-September-2019 to 22-September-2020; [3] Laquer, 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.

Acronyms: ADA, Anti-Drug Antibodies; CCL17, C-C motif chemokine ligand 17; COPD, chronic obstructive pulmonary disease; IL-33, interleukin-33; PK, pharmacokinetics; RAGE, receptor for advanced glycation end-products; ST2, serum stimulation-2; TARC, thymus and activation-regulated chemokine; Th, T-helper type..

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]

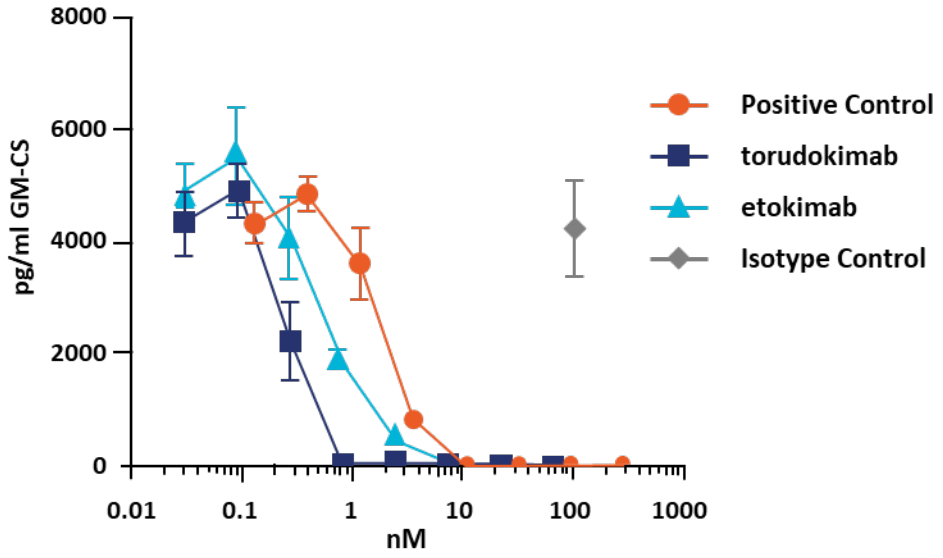


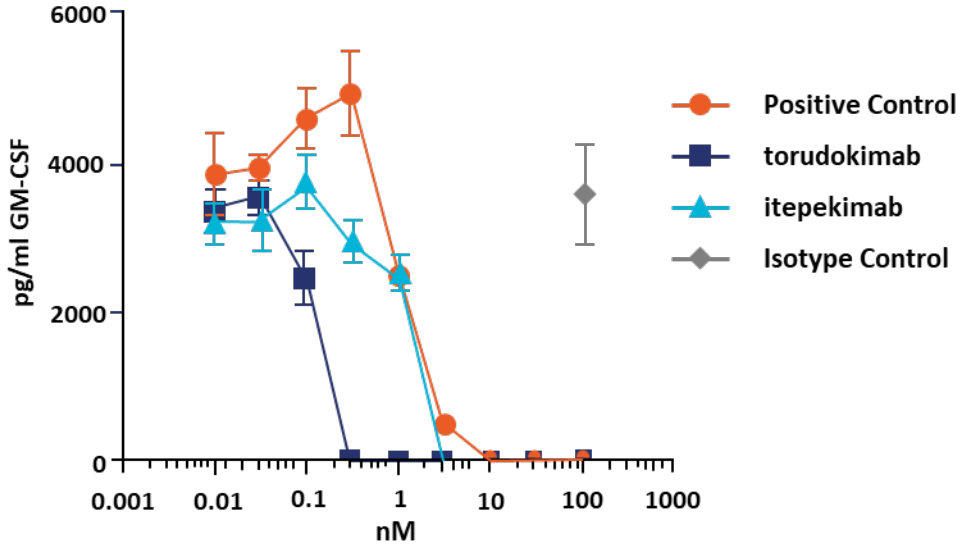
Sources: [1] Chan, B., et al. *Frontiers in Immunology*. doi:10.3389/fimmu.2019.00364; [2] Cayrol, C., and Girard, J.P. *Cytokine*. doi:10.1016/j.cyto.2022.155891; [3] Gudbjartsson, D., et al. *Nature Genetics*. doi:10.1038/ng.323; Ketelaar, M., et al. *Journal of Allergy and Clinical Immunology*. doi:10.1016/j.jaci.2020.04.051;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

Acronyms: COPD, chronic obstructive pulmonary disease; IL-33, interleukin-33; ST2, serum stimulation-2.

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

Acronyms: GM-CSF, granulocyte-macrophage colony-stimulating factor; IL-33, interleukin-33; k_d, dissociation constant; k_{off}, rate of dissociation (s^{-1}); k_{on}, rate of association ($M^{-1}s^{-1}$); nM, nanomolar; pM, picomolar; pg, picogram.

Corporate Update

Team and Cash Runway

Zura's Leadership: Driving Innovation in I&I

zurabio

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	

SCIENTIFIC ADVISORY BOARD

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.

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 September 30, 2024, we have 65,293,530 Class A Ordinary Shares outstanding[2].

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

