**UNITED STATES**
**SECURITIES AND EXCHANGE COMMISSION**
**Washington, D.C. 20549**

# FORM 8-K

**CURRENT REPORT**
**PURSUANT TO SECTION 13 OR 15(D)**
**OF THE SECURITIES EXCHANGE ACT OF 1934**

**January 13, 2025**
**Date of Report (Date of earliest event reported)**

## Zura Bio Limited

(Exact name of registrant as specified in its charter)

| Cayman Islands | 001-40598 | 98-1725736 |
|---|---|---|
| **(State or other jurisdiction of incorporation)** | **(Commission File Number)** | **(I.R.S. Employer Identification No.)** |

**1489 W. Warm Springs Rd. #110**
**Henderson, NV 89014**
**(Address of principal executive offices, including zip code)**

**(702) 757-6133**
**(Registrant's telephone number, including area code)**

**(Former name or former address, if changed since last report)**

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions:

☐ Written communication pursuant to Rule 425 under the Securities Act (17 CFR 230.425)

☐ Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)

☐ Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))

☐ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Securities registered pursuant to Section 12(b) of the Act:

| Title of each class | Trading Symbol(s) | Name of each exchange on which registered |
|---|---|---|
| **Class A Ordinary Shares, par value $0.0001 per share** | **ZURA** | **The Nasdaq Stock Market** |

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (§230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (§240.12b-2 of this chapter).

Emerging growth company ☒

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act. ☐

**Item 7.01. Regulation FD Disclosure.**

On January 13, 2025, Zura Bio Limited ("***ZURA***", the "***Company***") provided an updated corporate presentation that may be used in connection with presentations at conferences and investor meetings. The full text of the Company's corporate presentation is filed as Exhibit 99.1 hereto, and incorporated herein by reference, and may also be accessed through the "News & Events" section of the Company's website at investors.zurabio.com.

The information furnished under this Item 7.01, including Exhibit 99.1, shall not be deemed "filed" for purposes of Section 18 of the Securities Exchange Act of 1934, or subject to the liabilities of that section or Sections 11 and 12(a)(2) of the Securities Act of 1933, or the Securities Act. The information in this Item 7.01, including Exhibit 99.1, shall not be deemed incorporated by reference into any other filing with the SEC, made by the Company, whether made before or after the date hereof, regardless of any general incorporation language in such filing.

**Item 9.01. Financial Statements and Exhibits.**

**(d) Exhibits.**

| Exhibit No. | Description |
|---|---|
| 99.1 | Corporate Presentation, dated January 13, 2025 |
| 104 | Cover Page Interactive Data File (embedded within the Inline XBRL document). |

**SIGNATURES**

Pursuant to the requirements of the Securities Exchange Act of 1934, the registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

**ZURA BIO LIMITED**

Date: January 13, 2025

By: /s/ Kim Davis
Kim Davis
Chief Legal Officer

Exhibit 99.1


zurabio
Advancing Dual-Pathway Biologic Candidate
Addressing Unmet Needs in
Autoimmune and Inflammatory Diseases
January 2025
Nasdaq Ticker: ZURA

# Forward Looking Statements Disclaimer

This communication includes "forward-looking statements" within the meaning of the "safe harbor" provisions of the Private Securities Litigation Reform Act of 1995. Words such as "budget," "forecast," "anticipate," "intend," "plan," "may," "will," "could," "should," "believe," "predict," "potential," "continue," "strategy," "future," "opportunity," "would," "seem," "seek," such terms and similar expressions are intended to identify such forward-looking statements. Forward-looking statements are predictions, projections and other statements about f current expectations and assumptions and, as a result, are subject to risks and uncertainties that could cause the actual results to differ materially from the expected results. These fo include, but are not limited to: expectations with respect to development and regulatory plans, trial designs, and the costs, timing and results thereof, expectations with respect including the timing of study initiation and completion; expectations with respect to Zura Bio's development program, including clinical trials and the timing thereof, and expectation programs, data readouts and product candidates of other parties; Zura Bio's cash resources and projected cash runway; the potential to raise additional capital to support the compar pipeline assets to offer broader and improved clinical responses; expectations with respect to addressable markets, projected CAGRs and patient populations; and expectations with from any financing transactions. These statements are based on various assumptions, whether or not identified in this communication. These forward-looking statements are provid and are not intended to serve as, and must not be relied on by an investor as, a guarantee, an assurance, a prediction or a definitive statement of fact or probability.

Actual events are difficult or impossible to predict and could differ materially from those expressed or implied in such forward-looking statements, as a result of these risks and uncer not limited to: the potential of Zura Bio's product candidates and their related benefits, competing product candidates and products both in development and approved; Zura Bio's of key events and initiation of Zura Bio's studies and release of clinical data may take longer than anticipated or may not be achieved at all; the potential general acceptability a product candidates by regulatory authorities, payors, physicians, and patients may not be achieved; Zura Bio's ability to attract and retain key personnel; Zura Bio's future operating and needs for additional financing may not be achieved; Zura Bio has not completed any clinical trials, and has no products approved for commercial sale; Zura Bio has incurred sig and expects to incur significant losses for the foreseeable future and may not be able to achieve or sustain profitability in the future; Zura Bio requires substantial additional capital to is unable to raise such capital when needed or on acceptable terms, Zura Bio may be forced to delay, reduce, and/or eliminate one or more of its development programs or future Bio may be unable to renew existing contracts or enter into new contracts; Zura Bio relies on third-party contract development manufacturing organizations for the manufacture of on contract research organizations, clinical trial sites, and other third parties to conduct of its preclinical studies and clinical trials; Zura Bio may be unable to obtain regulatory appr and there may be related restrictions or limitations of any approved products; Zura Bio may be unable to successfully respond to general economic and geopolitical condition effectively manage growth; Zura Bio faces competitive pressures from other companies worldwide; Zura Bio may be unable to adequately protect its intellectual property rights documents filed, or to be filed by Zura Bio, with the SEC, including the risks and uncertainties described in the "Risk Factors" section of Zura Bio's Annual Report on Form 10-K for the and other filings with the SEC. These risks and uncertainties may be amplified by health epidemics or other unanticipated global disruption events, which may continue to cause e cautions that the foregoing list of factors is not exclusive or exhaustive and not to place undue reliance upon any forward-looking statements, including projections, which speak only gives no assurance that we will achieve our expectations.

Zura Bio does not undertake or accept any obligation to publicly provide revisions or updates to any forward-looking statements, whether as a result of new information, future should circumstances change, except as otherwise required by securities and other applicable laws.

This presentation discusses product candidates that are under clinical study, and which have not yet been approved for marketing by the US Food and Drug Administration. No re safety or effectiveness of these product candidates for the use for which such product candidates are being studied. Caution should be exercised when comparing data across product candidates. Differences existing between trial designs and patient populations and characteristics. The results across such trials may not have interpretative value on Zura Statements included herein concerning clinical trials for the product candidates have not been reviewed or endorsed by Eli Lilly ("Lilly") or Pfizer.

This presentation shall not constitute an offer to sell or the solicitation of an offer to buy securities, nor shall there be any sale of securities in any state or jurisdiction in which such off unlawful prior to registration or qualification under the securities laws of any such state or jurisdiction.

# Company Summary

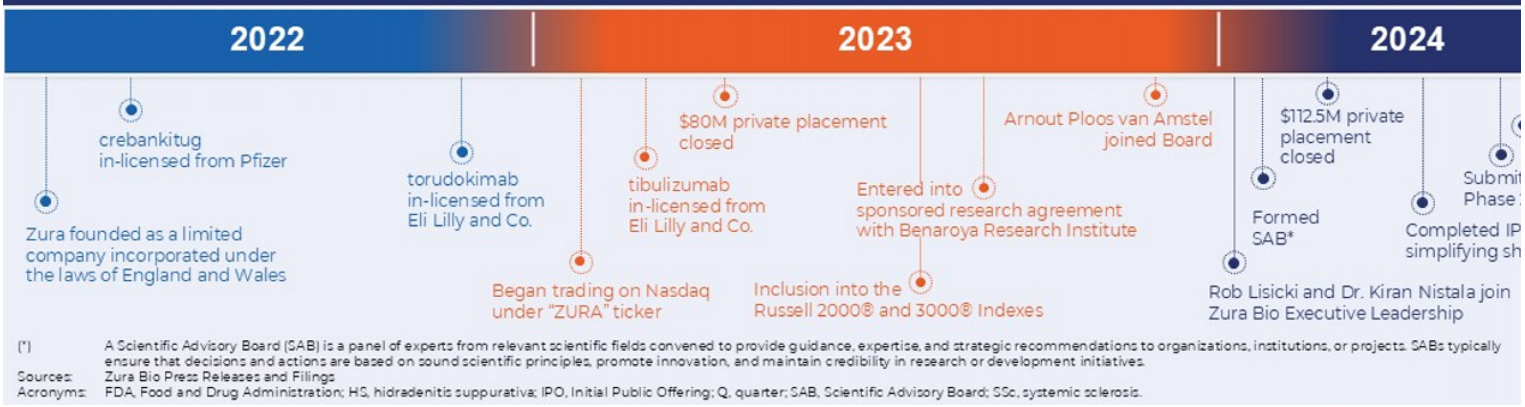
**High-Potential Biologics:** Three dual-pathway biologics targeting multi-billion-dollar markets, with ava data and potential to advance to Phase 2 trials

**Lead Asset Development:** Tibulizumab Phase 2 study for SSc is ongoing, with a subsequent trial for HS a initiate in 2Q 2025

**Strategic Milestones:** Anticipating 2 key internal catalysts and up to 11 external readouts over the ne with potential to significantly drive value creation

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

**Financial Strength:** Cash runway expected to support planned operations through 2027

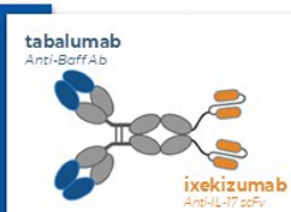
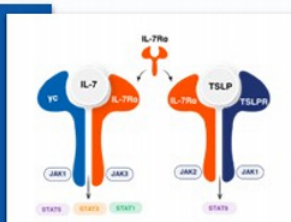
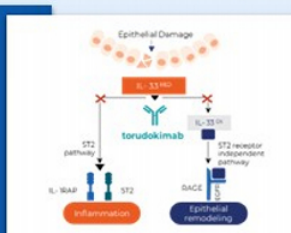



(*) A Scientific Advisory Board (SAB) is a panel of experts from relevant scientific fields convened to provide guidance, expertise, and strategic recommendations to organizations, institutions, or projects. SABs typically ensure that decisions and actions are based on sound scientific principles, promote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

| | | 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 *<br>✓ 78 participants dosed across 3 studies<br>– n=57 single dose<br>– n=21 multiple doses up to 12 weeks | Phase 2 study for subsequent trial f 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 **<br>✓ 93 participants dosed<br>– n=60 single dose<br>– n=33 multiple doses up to 12 weeks | Actively evaluatin indications to gui development effo |
| **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 ***<br>✓ 244 participants dosed<br>– n= 81 single dose<br>– n=163 multiple doses up to 52 weeks | Actively evaluatin indications to gui development effo |

(*) 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, thy stromal lymphopoietin.


tibulizumab
ZB-106
Anti-BAFF + IL-17
Tibulizumab, is a humanized bispecific du
antibody, and has been engineered to bin
neutralize both BAFF and IL-17A. Our appr
tibulizumab is to inhibit both pathways wit
agent, potentially providing clinical benefit
range of patients, as well as a greater level
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

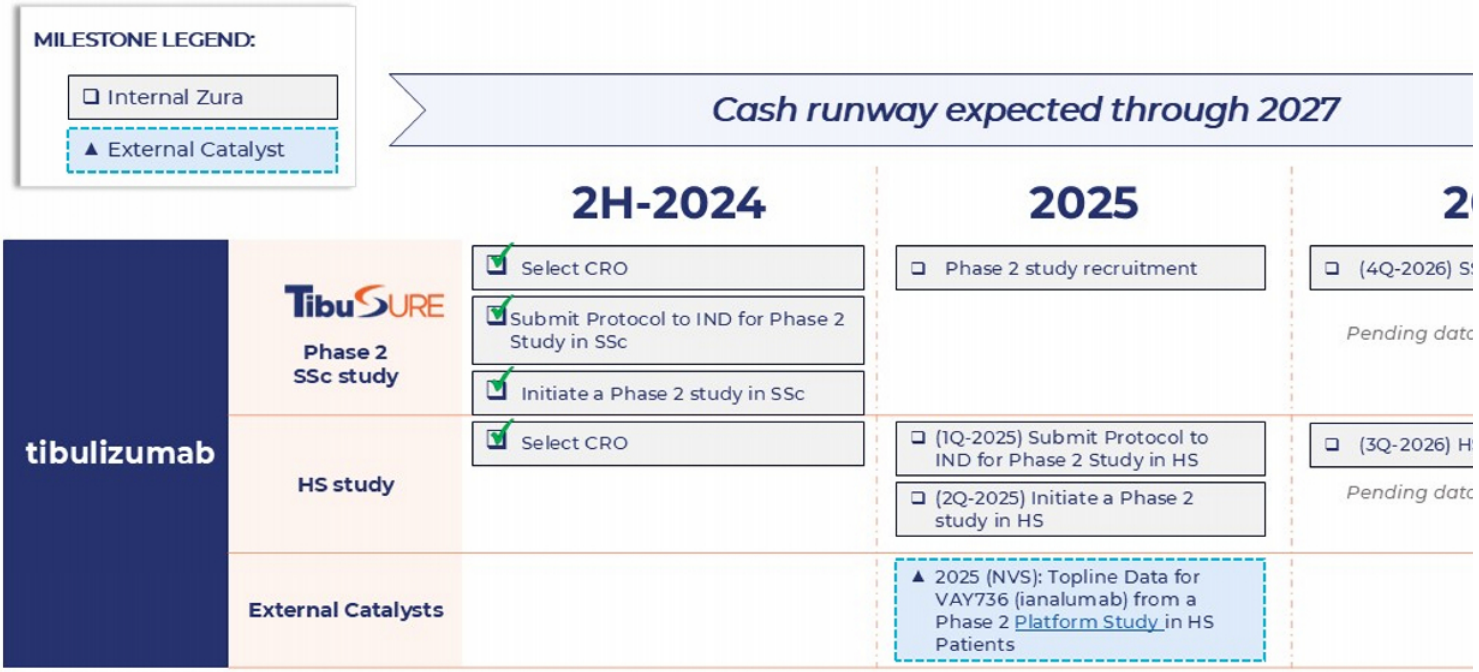
(*) Two approved therapies are available for SSc-ILD; however, no treatment addresses the disease across organ systems.
Sources: [1]Clarivate/DRG. Accessed 19 August 2024. Projected Prevalence 2032.
Acronyms: ILD, interstitial lung disease; SSc, systemic sclerosis.

## Systemic sclerosis is characterized by *tissue inflamma*


## Multiple areas for evaluation and improveme

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

# Tibulizumab milestones* expected through 2026



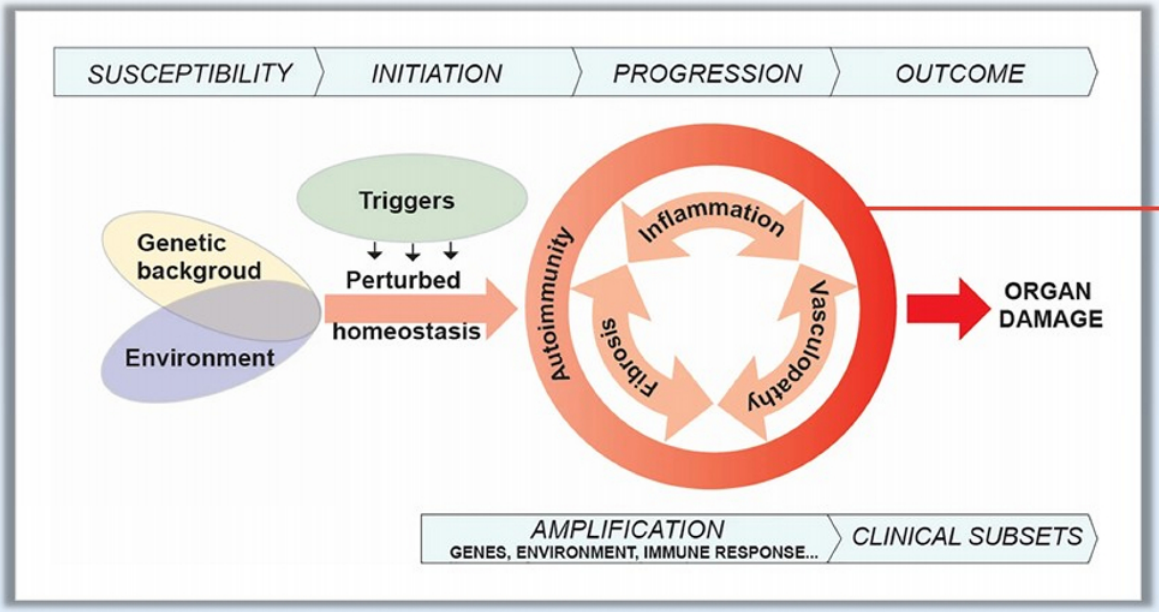

(*) The timing of regulatory and clinical trial milestones is subject to change and may require further interactions with the FDA.
Sources: Internal Zura Planning, clinicaltrials.gov
Acronyms: CRO, contract research organization; HS, hidradenitis suppurativa; IND, Investigational New Drug; Q, quarter;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

# Rationale for Phase 2 Study in Systemic Sclerosis Patients

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 throu
  pathways of inflamm
  autoimmunity, vascu
  fibrosis, driven by ge
  environmental trigg

→ **IL-17 amplifies infla**
**vascular injury, and**
**damage**, making it a
disrupt the inflamm

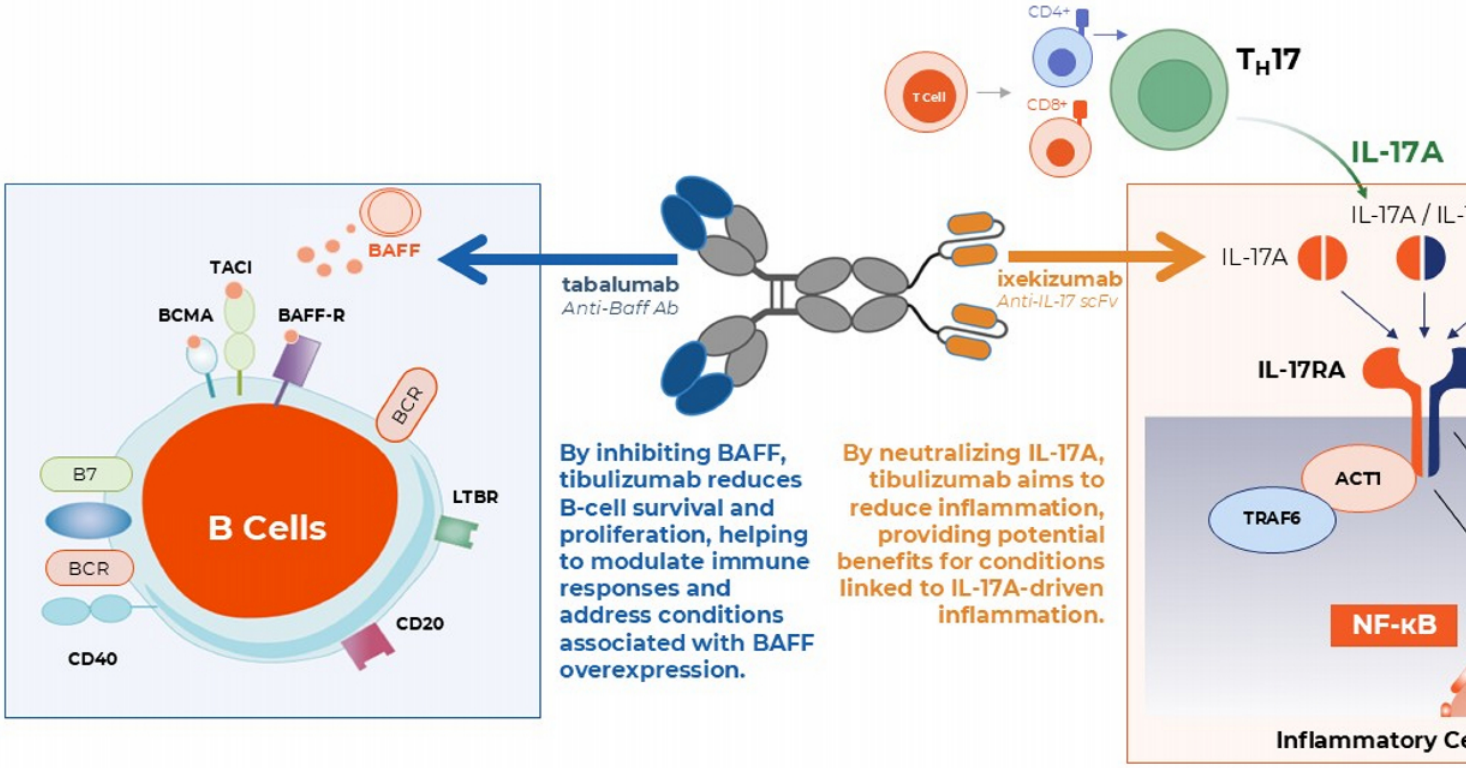
→ **BAFF drives B-cell s**
**autoantibody produ**
**immune dysregulat**
**accelerating fibrosi**
unique opportunity
intervention.

**Targeting IL-17 and**
**transformative appr**
**the amplification l**
**disease progression**
**irreversible organ**

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

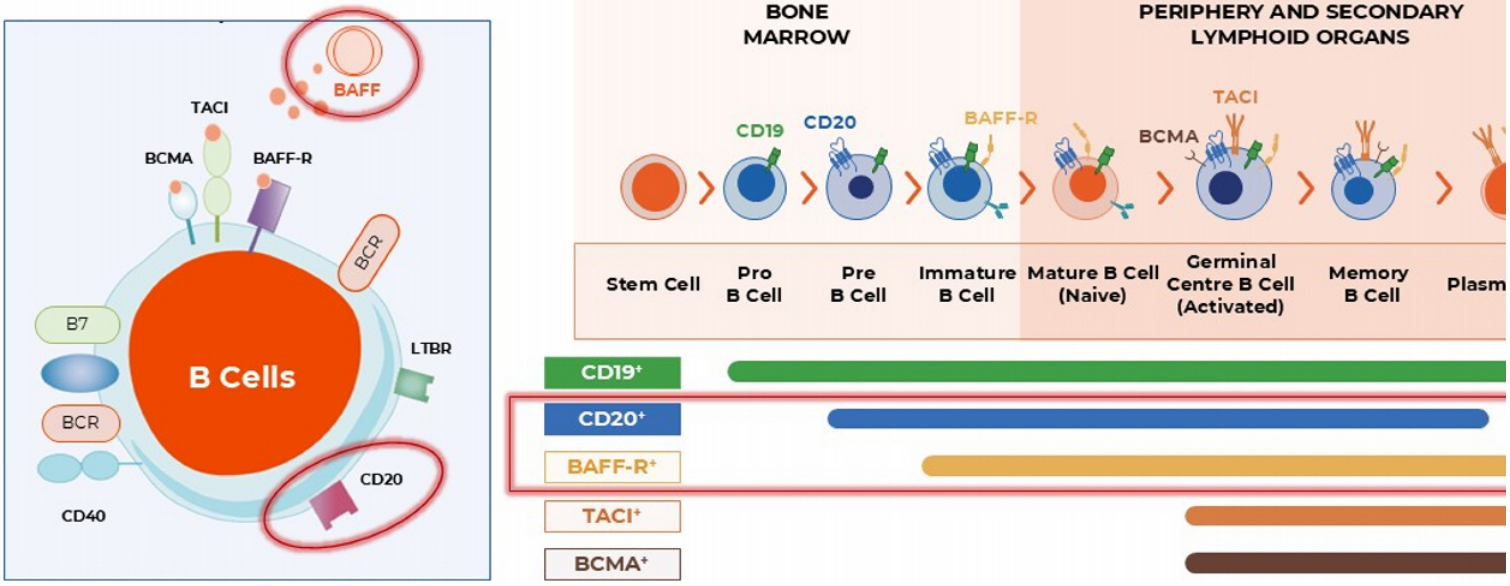
**Tibulizumab uniquely targets BAFF for B cell regulation and IL-17A for inflammation contro
comprehensive approach to treating autoimmune and inflammatory diseases.**




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

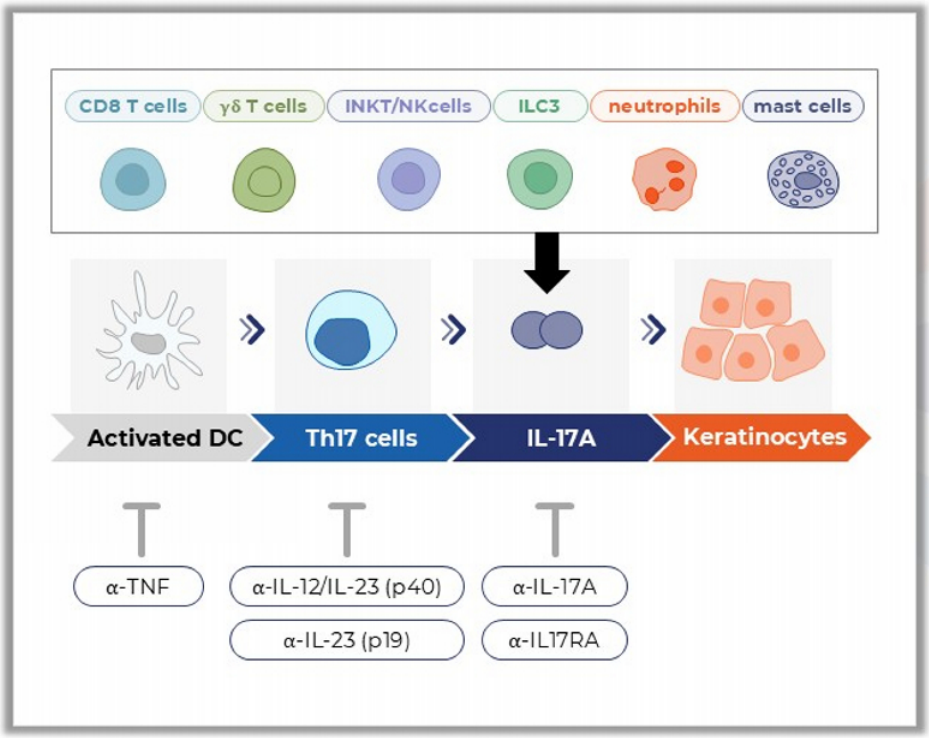
**By neutralizing IL-17A, tibulizumab aims to reduce inflammation, providing potential benefits for conditions linked to IL-17A-driven inflammation.**

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




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 st
production of IL-17 by activat
cells. The IL-23/IL-17 axis is ar
pathway for targeted therap
inflammatory diseases.

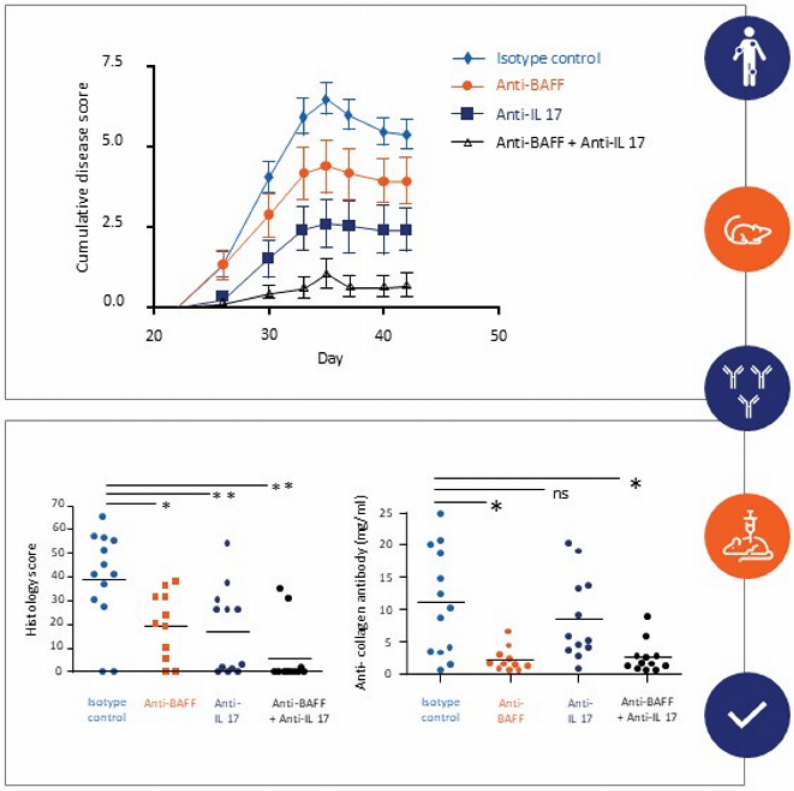
IL-17A is associated with the
of inflammatory diseases, inc
systemic sclerosis.

Clinical trials have shown tha
antibodies against IL-23 and
effective in the treatment of
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




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

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

Surrogate murine antibodies were used to eva whether **neutralization of IL-17A and BAFF** w to targeting individual pathways

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

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

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

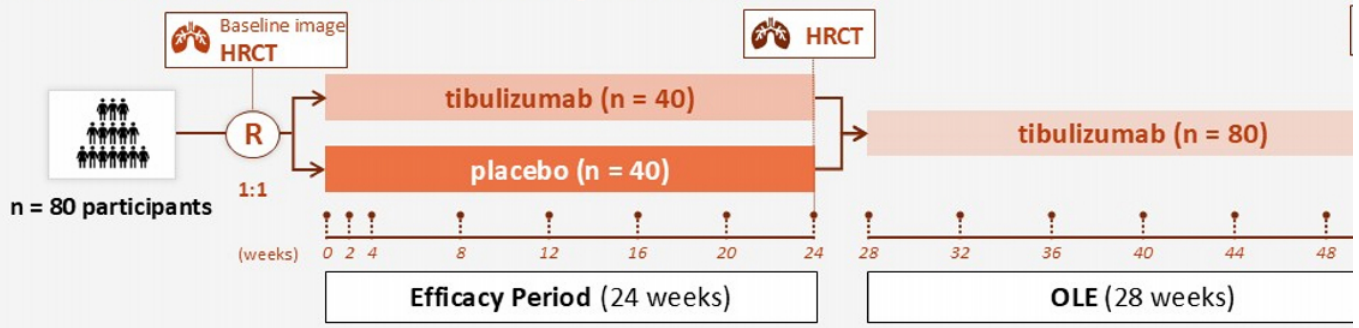
Sources: Zura Internal Data, Investigational New Drug (IND) Briefing
Acronyms: BAFF, B cell-activating factor; IL, interleukin.

# TibuSURE

Tibulizumab Systemic Sclerosis Understanding and Response Evaluation

**Study is Focused on Der**
**Benefits for Skin and Lu**

## RANDOMIZED PHASE 2 STUDY* (mRSS and HRCT)




## KEY EFFICACY ENDPOINTS

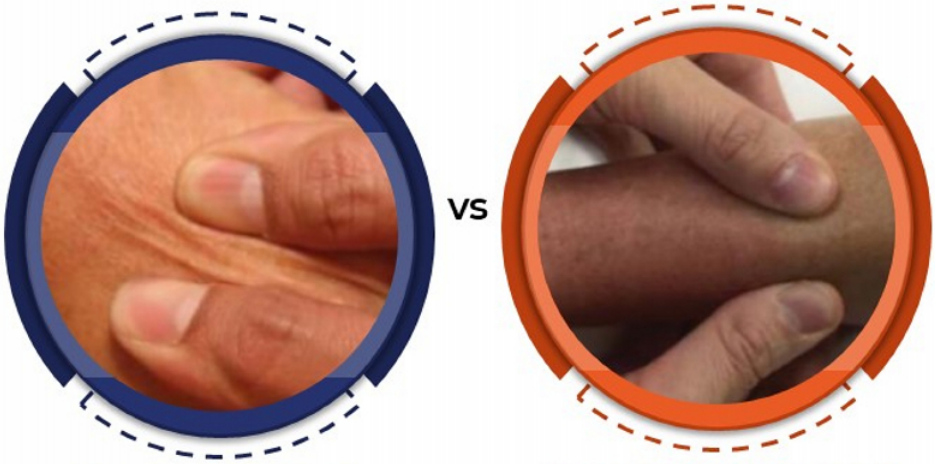
- mRSS (primary)
- qHRCT / FVC
- HAQ-DI (Function)
- revised CRISS (rCRISS)

## 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
  - mRSS 20-45
  - RNA Pol III negati
    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 Sclerosi RNA Pol III, RNA polymerase III; SSc, systemic sclerosis.

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

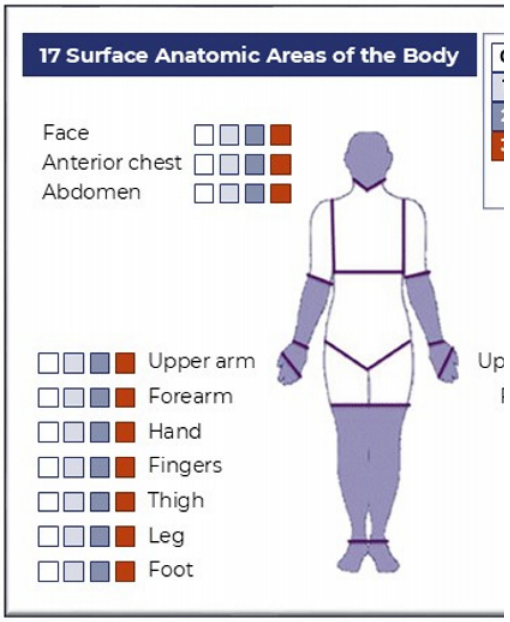


**Fine Wrinkles[1] (0/3)** VS **Severe Thickness[1] (3/3)**

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

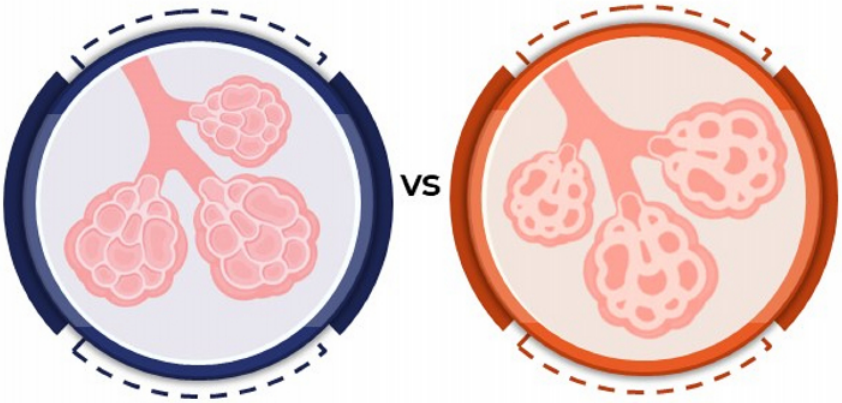
The **mRSS assesses skin thickness** in syst patients by **evaluating 17 body sites** (e.g., abdomen, arms, legs). Each site is scored f

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




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



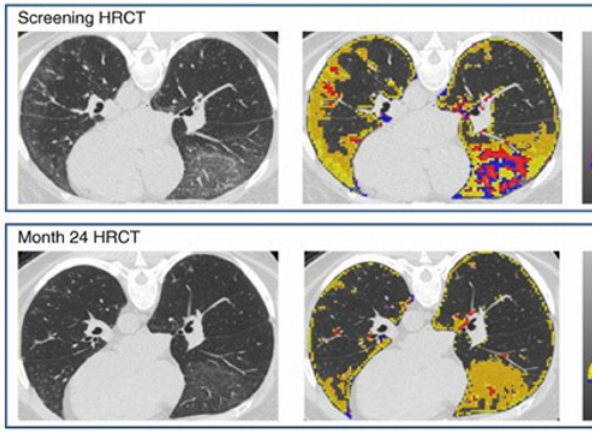

**Healthy Lung** **Fibrotic Lung**

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 measur involvement**, detecting changes as small as

**Example of improvement after 24 months of MMF in to**




The blue and red areas show QLF, while the yell quantitative ground glass. The entire colored area represents 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

## 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.


tibulizumab
ZB-106
Anti-BAFF + IL-17
Tibulizumab, is a humanized bispecific du
antibody, and has been engineered to bin
neutralize both BAFF and IL-17A. Our appr
tibulizumab is to inhibit both pathways wit
agent, potentially providing clinical benefit
range of patients, as well as a greater level
hidradenitis suppurativa

# 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.

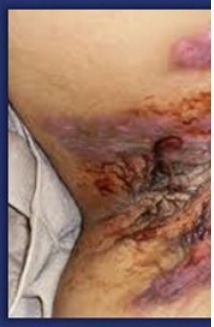
# An Overview of Hidradenitis Suppurativa

## 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%** patien **inadequatel**

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.

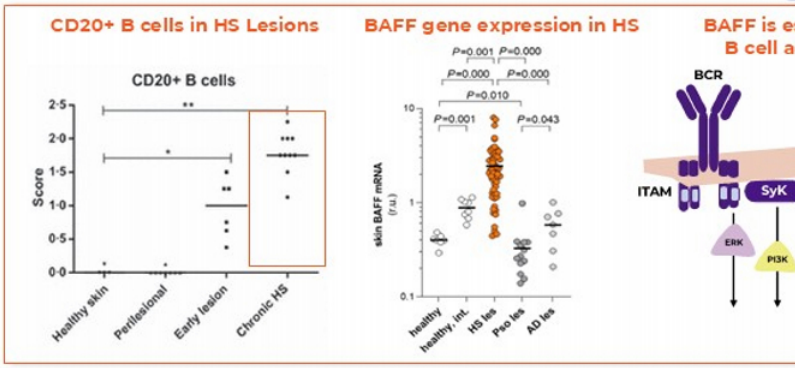
# B Cell Signaling Potentiates HS Disease

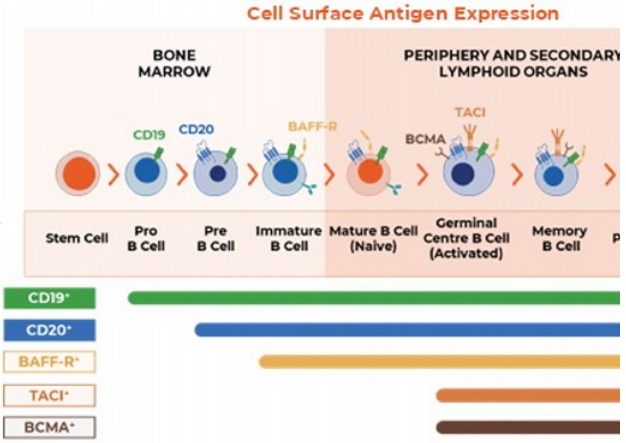
**Elevated CD19** in HS **indicates increased B cell activation**, driving inflammation and correlating with higher CD20+ B cells and CD138+ plasma cells.

**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]




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

*Ixekizumab is a humanized IgG4 monoclonal antibody*

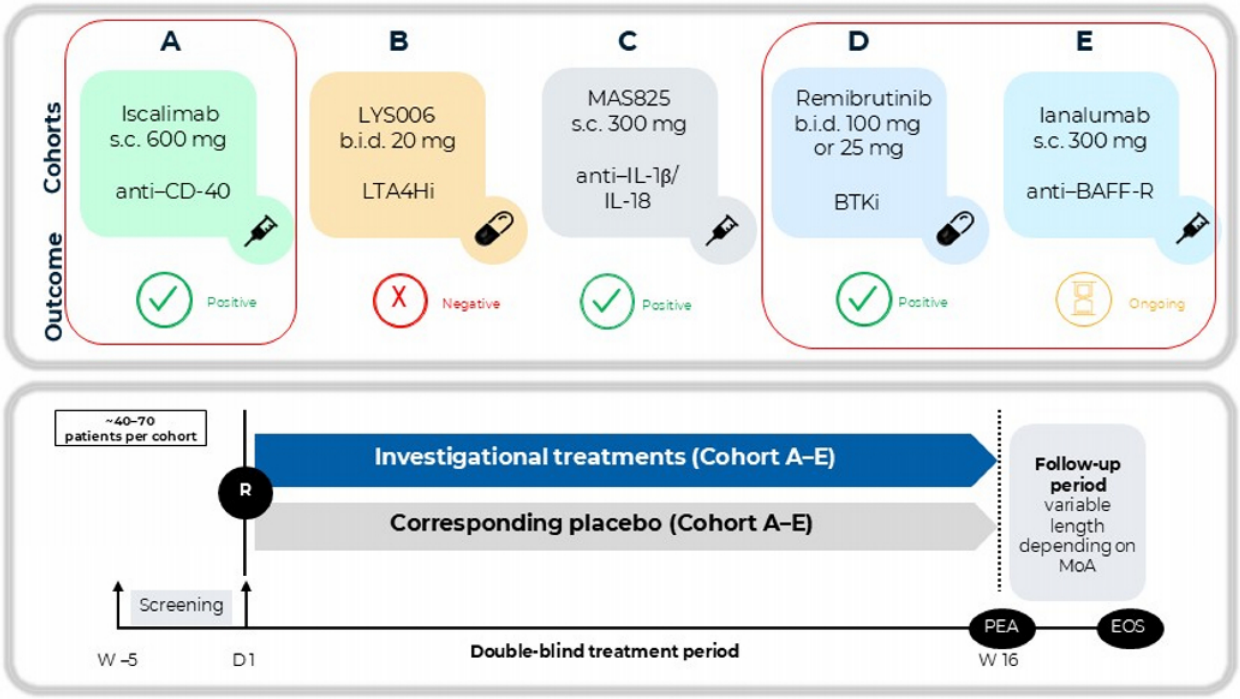
| | IL-17A Binding Affinity | IL-17A/F Binding Affinity | IL-17F Binding Affinity |
|---|---|---|---|
| **Taltz®** (ixekizumab) | $K_d$ 1.8 pM | $K_d$ <3 pM | N/A |
| **Bimzelx®** (bimekizumab) | $K_d$ 3.2 pM | $K_d$ 26 pM | $K_d$ 23 pM |
| **Cosentyx®** (secukinumab) | $K_d$ 60 pM – 90 pM | $K_d$ 2400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

- Binding affinity, quant
  dissociation constant
  the strength of interac
  between a drug and it
  with a lower $K_d$ indica
  stronger binding.
- Stronger binding affin
  more effective engage
  the target, which may
  to improved clinical ou

(*) Tibulizumab was engineered from Taltz® (ixekizumab)
Sources: [1] Taltz®. Prescribing Information. Lilly USA, LLC, https://taltz.lilly.com/hcp/moa-il17a-igg4.
Acronyms: IgG4, immunoglobulin G4; $K_d$, dissociation constant; pM, picomolar

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

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




## Patients

- Adult pat
  aged 18–6
- Moderate
  for ≥12 mo
  anatomic
  ≤15 tunne
- ***Cohorts A***
  ≥5 inflam
- ***Cohorts B***
  inflamma

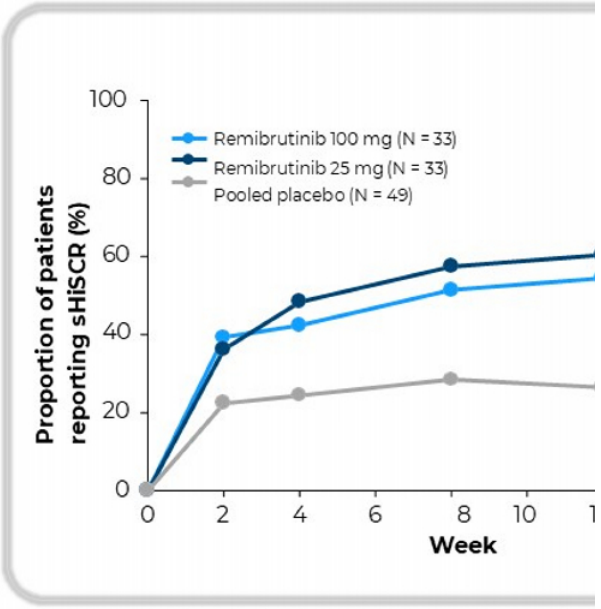
*Study started in February 2019 and is currently ongoing.
BAFF-R, B-cell activating factor of the tumor necrosis alpha family receptor; b.i.d., twice daily; BTKi, Bruton's tyrosine kinase inhibitor; CD, cluster of differentiation; D, day; EOS, end of study; HS, hidradenitis suppurativa; IL, interleukin;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

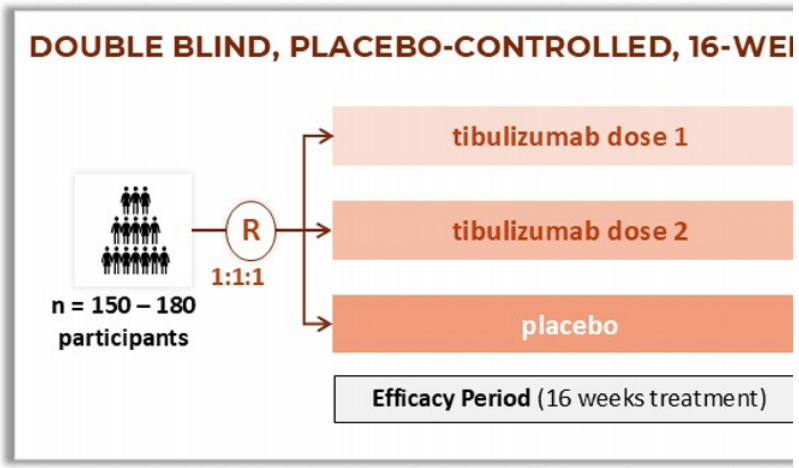



*The sHiSCR is defined as a ≥50% reduction in the abscess and inflammatory nodule count and no increase in draining tunnels compared with baseline. †Difference refers to the difference between remibrutinib (either dose) and pooled placebo at Week 16. ‡Bayesian posterior probability of remibrutinib (either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

**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
antibody that neutralizes the IL-7 recepto
7Rα) chain, potentially blocking the immu
of IL-7 and thymic stromal lymphopoietin

# 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.

## Active Phas

- Ongoin planni in area needs.

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

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

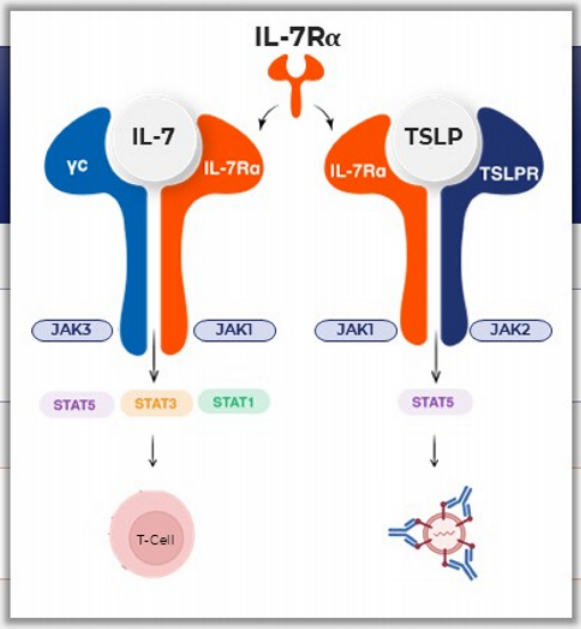
**IL-7Rα** is a key receptor in immune regulation, central to the signaling of cytokines ***IL-7*** an

## 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 dec
TSLPR. For optimal s
joins the mix, creat
complex with

This assembled c
**pathways** primarily

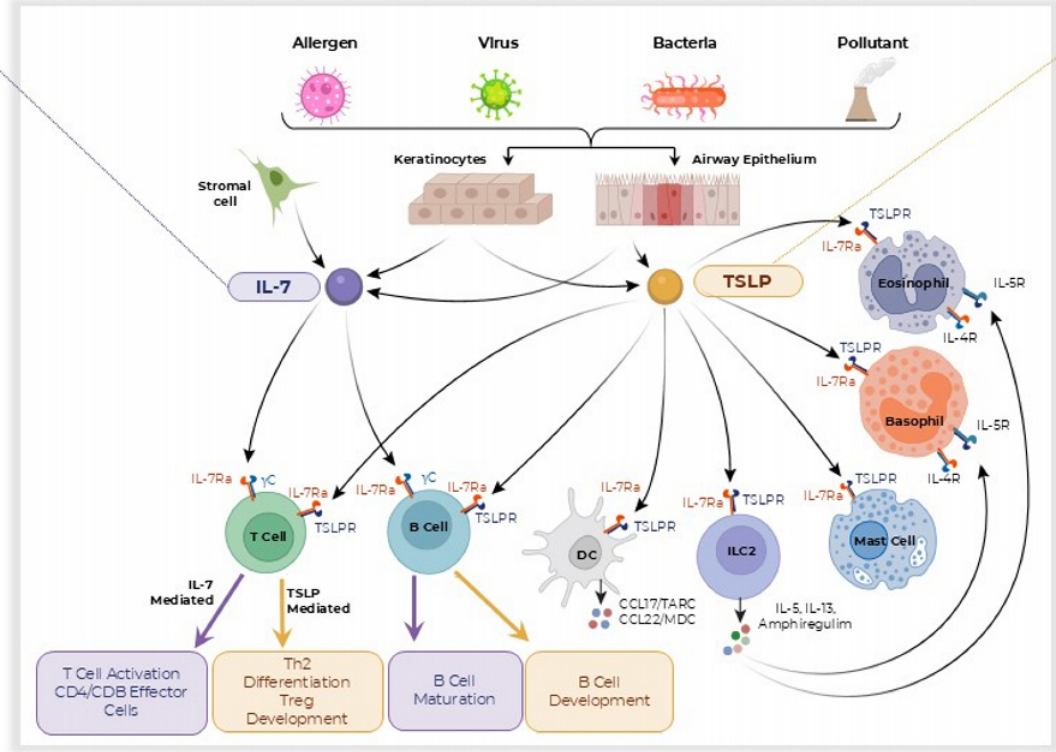
Commonly tied to al
and specific inflamm

Positioning crebankitug for potential applications in diverse immune-related and autoimmun

# 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]




TSL

- Thy lym an e cyto exp skin trac
- TSL epit amp resp proc and
- TSL clini seve sho ther add dise


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 Savvides S. Frontiers in Immunology, doi:10.3389/fimmu.2020.01557.

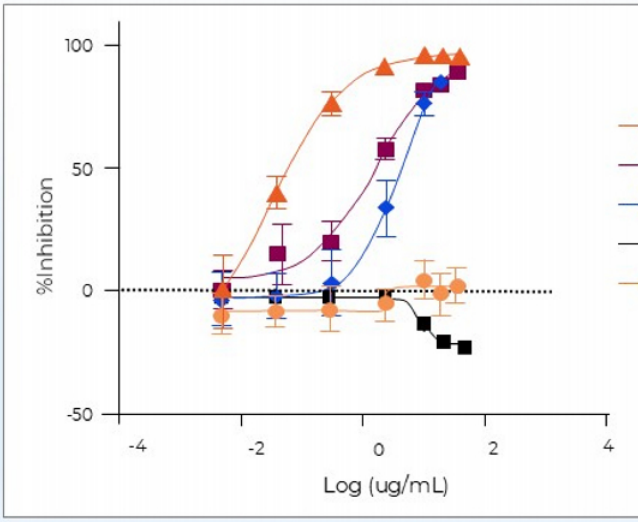
# Crebankitug Is the Only mAb to Potently Inhibit Both IL-7R and TSLP

**Inhibition of TSLP stimulated CCL17 secretion from human monocytes**

| | AstraZeneca / AMGEN | 32 BIO | zurabio |
|---|---|---|---|
| | **tezepelumab (TSLP)** TSLP mAb | **bempikibart (IL-7Rα)** IL-7Rα mAb | **crebankitug (IL-7Rα)** 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]<br>11 ng / ml / 0.07nM (CCL22)[1]<br>**0.08 nM (CCL2)[4]** |
| **IL-7-Induced Signals** | *Neg* | 0.6 nM (IL-7 at 0.25ng/ml)[4]<br>2.1 nM (IL-7 at 2.5ng/ml)[4] | 0.46nM (pSTAT5)[2] |

**World Allergy Congress Poster, Dec**

**% inhibition of TSLP stimulated CCL17 secretion from hun**




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


torudokima
ZB-880
Anti-IL-33
Torudokimab is a fully human, high affinity m
antibody that neutralizes IL-33, preventing S
and ST2-independent (e.g., RAGE) inflammat

# Torudokimab Asset Overview

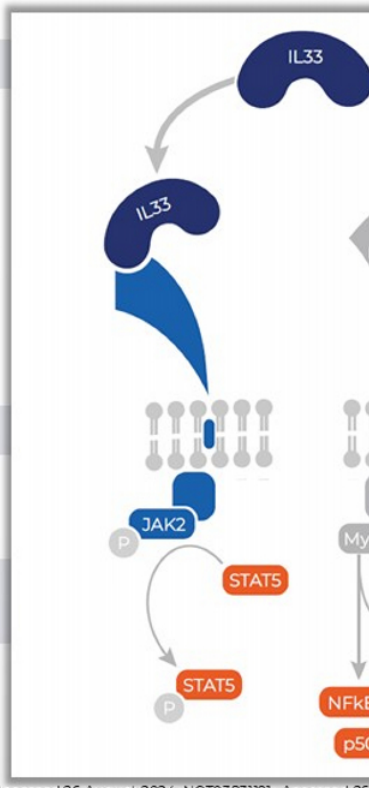
## About torudokimab

**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
- 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]

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




## 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

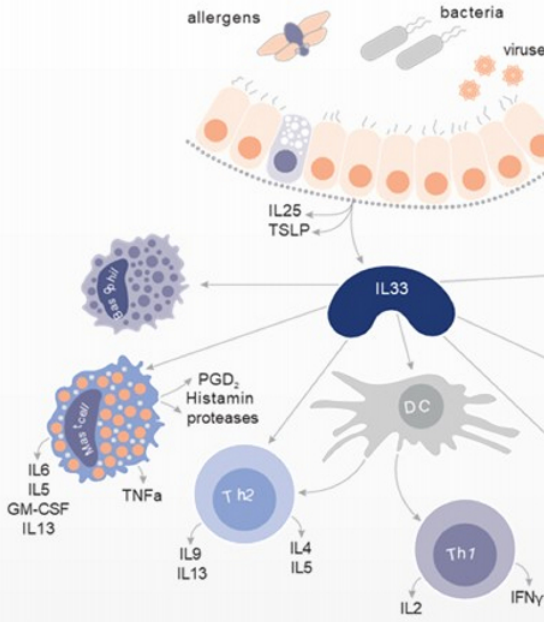
**02** Validated pathways in COPD [4] and asthma [5]

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] |

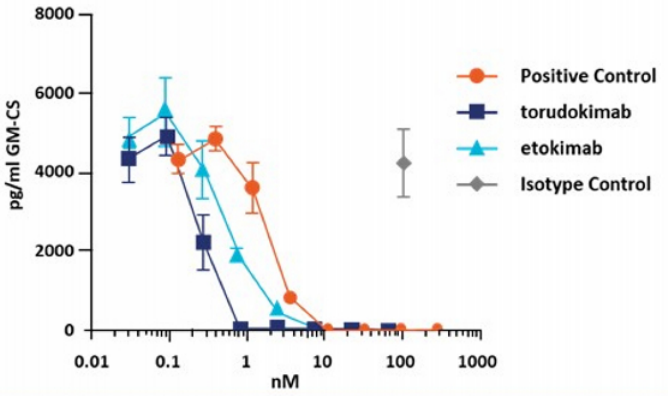


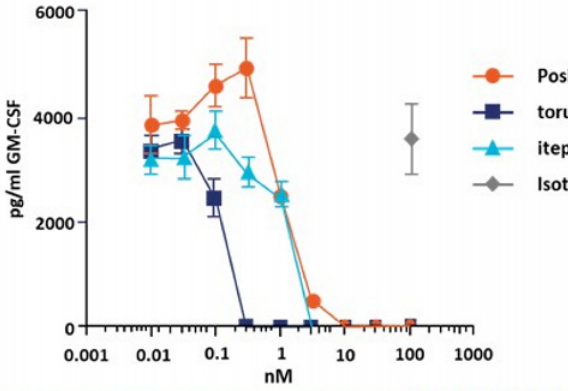

Sources: [1] Chan, B., et al. *Frontiers in Immunology*. doi:10.3389/fimmu.2019.00364; [2] Cayrol, C., and Girard, J.P. *Cytokine*. doi:10.1016/j.cyto.2022.155891; [3] Gudbjartsson, D., et al. *Nature Genetics*. doi:10.1038/ng.323; Ketelaar, M., et al. *Journal of Allergy and Clinical Immunology*. doi:10.1016/j.jaci.2020.04.051;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

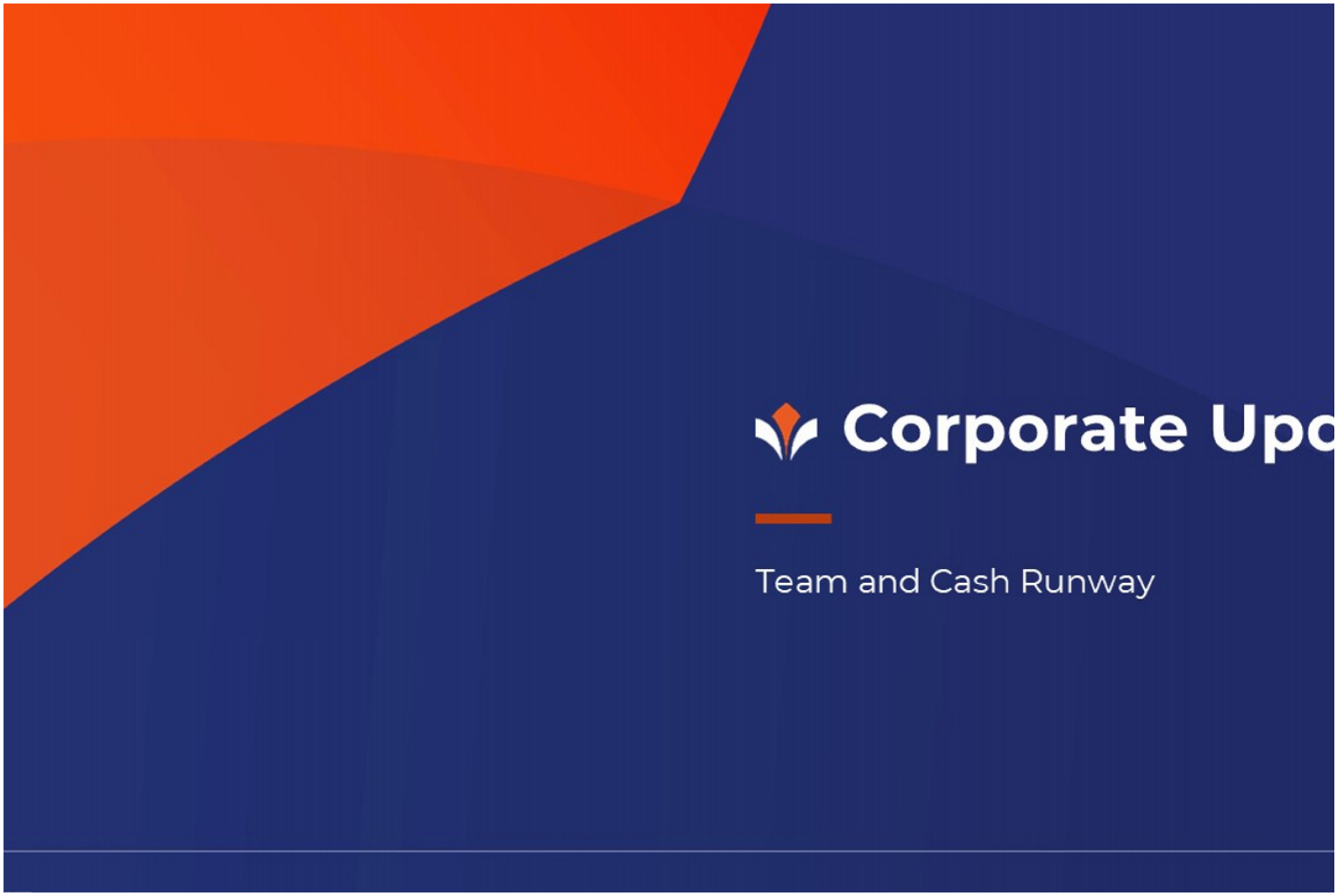
***Torudokimab was 2.9 and 5.5-fold more potent than etokimab and itepekimab***, respe
inhibiting IL-33-induced GM-CSF production by human mast cells




| Antibody | $k_{on}$ ($M^{-1}s^{-1}$) | $k_{off}$ ($s^{-1}$) | $k_d$ (pM) | Torudokim |
|---|---|---|---|---|
| torudokimab (LY3375880) | $1.7 \times 10^{6}$ | $6.7 \times 10^{-5}$ | 39 | |
| etokimab (AnaptysBio) | $9.4 \times 10^{5}$ | $1.2 \times 10^{-4}$ | 112 | 2 |
| itepekimab (Regeneron) | $7.6 \times 10^{5}$ | $1.6 \times 10^{-4}$ | 215 | 5 |

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 Upd
Team and Cash Runway

# Zura's Leadership: Driving Innovation in I&I

## EXECUTIVE TEAM

| | | | | |
|---|---|---|---|---|
| **Robert Lisicki**<br>Chief Executive Officer and Director | **Verender Badial**<br>Chief Financial Officer | **Kiran Nistala, MBBS, PhD**<br>Chief Medical Officer and Head of Development | **Gary Whale, PhD**<br>Chief Technology Officer | C and |

## BOARD OF DIRECTORS

| | | | | |
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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
transformative, life-saving treatments.

**Tibulizumab shows best-in-class potential:** Introducing a tetr
therapy designed to target and potentially treat autoimmune c

**Promising pipeline for value creation:** Integrating validated bi
pathways into multifunctional antibody assets to potentially im
therapeutic outcomes.

**Upcoming external catalysts:** Anticipating near-term develop
further expand the pipeline's potential.

**Proven leadership:** Experienced team with a track record of co
over $8 billion in mergers and acquisitions in the past three yea

**Strong financial position:** With approximately $188 million[1] in c
equivalents, and investments, we are funded to support our pla
through 2027. The 3Q 2024 IPO warrant exchange has streamli
structure, and additional financing through ATM options remai
future needs. As of September 30, 2024, we have 65,293,530 Cla
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.