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

August 14, 2024
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**
Warrants, each whole warrant exercisable for one Class A Ordinary Share at an exercise price of $11.50 per share	**ZURAW**	**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 August 14, 2024, 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 August 14, 2024
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: August 14, 2024

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

zurabio

Three unique dual-pathway biologics, clinically validated for therapeutic areas with unmet needs

August 2024

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 su “project,” “budget,” “forecast,” “anticipate,” “intend,” “plan,” “may,” “will,” “could,” “should,” “believe,” “predict,” “potential,” “continue,” “strategy,” “future,” “opportunity,” “would,” “seem negatives of such terms and similar expressions are intended to identify such forward-looking statements. Forward-looking statements are predictions, projections and other stater that are based on 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 expec looking statements may include, but are not limited to: expectations with respect to development and regulatory plans, trial designs, and the costs, timing and results thereof; ex milestones and key events, including the timing of study initiation and completion; expectations with respect to Zura Bio's development program, including clinical trials an expectations with respect to development programs, data readouts and product candidates of other parties; Zura Bio's cash resources and projected cash runway; the potential to support the company's operations; the potential of pipeline assets to offer broader and improved clinical responses; expectations with respect to addressable markets, proj populations; and expectations with respect to the use of proceeds from any financing transactions. These statements are based on various assumptions, whether or not identifie These forward-looking statements are provided for illustrative purposes only and are not intended to serve as, and must not be relied on by an investor as, a guarantee, an as 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 un but are 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; Zur the timing 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 accepta Zura Bio's 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 c requirements 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 B losses since inception, 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 subst finance its operations, and if it 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 future commercialization efforts; Zura Bio may be unable to renew existing contracts or enter into new contracts; Zura Bio relies on third-party contract development manufactu manufacture of clinical materials; Zura Bio relies on contract research organizations, clinical trial sites, and other third parties to conduct of its preclinical studies and clinical trials; obtain regulatory approval for its product candidates, and there may be related restrictions or limitations of any approved products; Zura Bio may be unable to successfully respond geopolitical conditions; Zura Bio may be unable to effectively manage growth; Zura Bio faces competitive pressures from other companies worldwide; Zura Bio may be unable intellectual property rights; and other factors set forth in documents filed, or to be filed by Zura Bio, with the SEC, including the risks and uncertainties described in the “Risk Fac Annual Report on Form 10-K for the year ended December 31, 2023 and other filings with the SEC. These risks and uncertainties may be amplified by health epidemics or other unan events, which may continue to cause economic uncertainty. Zura Bio cautions that the foregoing list of factors is not exclusive or exhaustive and not to place undue reliance statements, including projections, which speak only as of the date made. Zura Bio 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 deve 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 the safety or effectiveness of these product candidates for the use for which such product candidates are being studied. Caution should be exercised when comparing data across and product candidates. Differences existing between trial designs and patient populations and characteristics. The results across such trials may not have interpretative value on ou 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 suc would be unlawful prior to registration or qualification under the securities laws of any such state or jurisdiction.

Company Summary

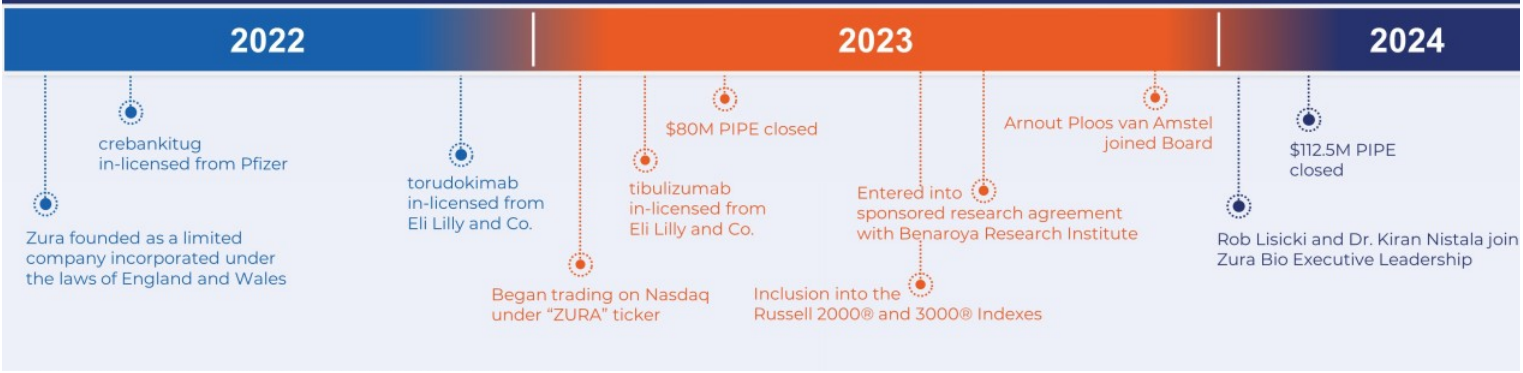
High-Potential Biologics: Three novel, clinically validated dual-pathway biologics, e with multi-billion-dollar potential, ready for Phase 2.

Lead Asset Development: Phase 2 study for tibulizumab targeting SSc starts in 4Q 2024, followed by HS in 2Q 2025.

Strategic Milestones: Expecting 2 internal catalysts and up to 11 external readc over the next 36 months, driving value creation.

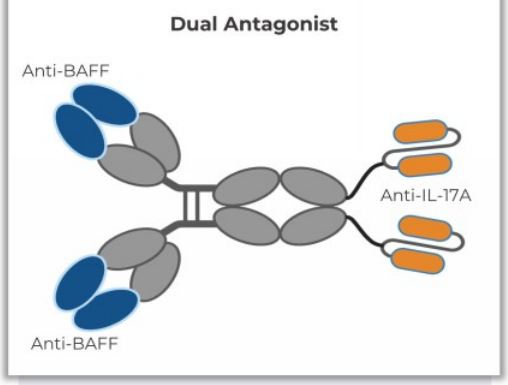
Proven Leadership: Experienced team with a strong track record in autoimn drug development and commercialization.

Financial Strength: Cash runway through 2027.



Pipeline of novel dual-pathway biology clinical stage assets potentially offers broader and improved clinical responses

tibulizumab
ZB-106

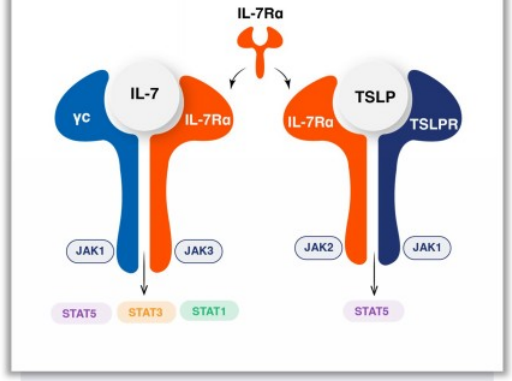


78 Participants Dosed Across Three Ph 1/1b studies

57 participants with single dose

21 participants with multiple doses up to 12 weeks

crebankitug
ZB-168

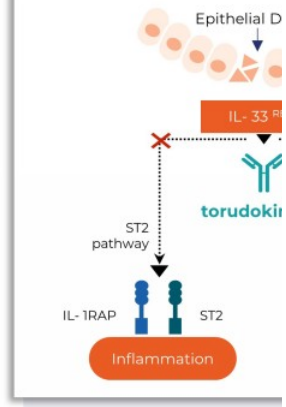


93 Participants Dosed

60 participants with single dose

33 participants with multiple doses up to 12 weeks

torudok
ZB-88



244 Participa

81 participants wit

163 participants wi
doses up to 52 we

(*) includes data from trials run by Pfizer and Eli Lilly
Sources: Zura CSRs and Internal Data
Acronyms: BAFF, B cell-activating factor; EGFR, epidermal growth factor receptor; JAK, janus tyrosine kinase; IL, interleukin; RAGE, receptor for advanced glycation end products; ST2, growth STimulation expressed gene 2; TSLP, thymic stromal lymphopoietin

Zura is led by a strong leadership team with a successful track record in drug and business development

ROBERT LISICKI
Chief Executive Officer
and Director

VERENDER BADIAL
Chief Financial Officer

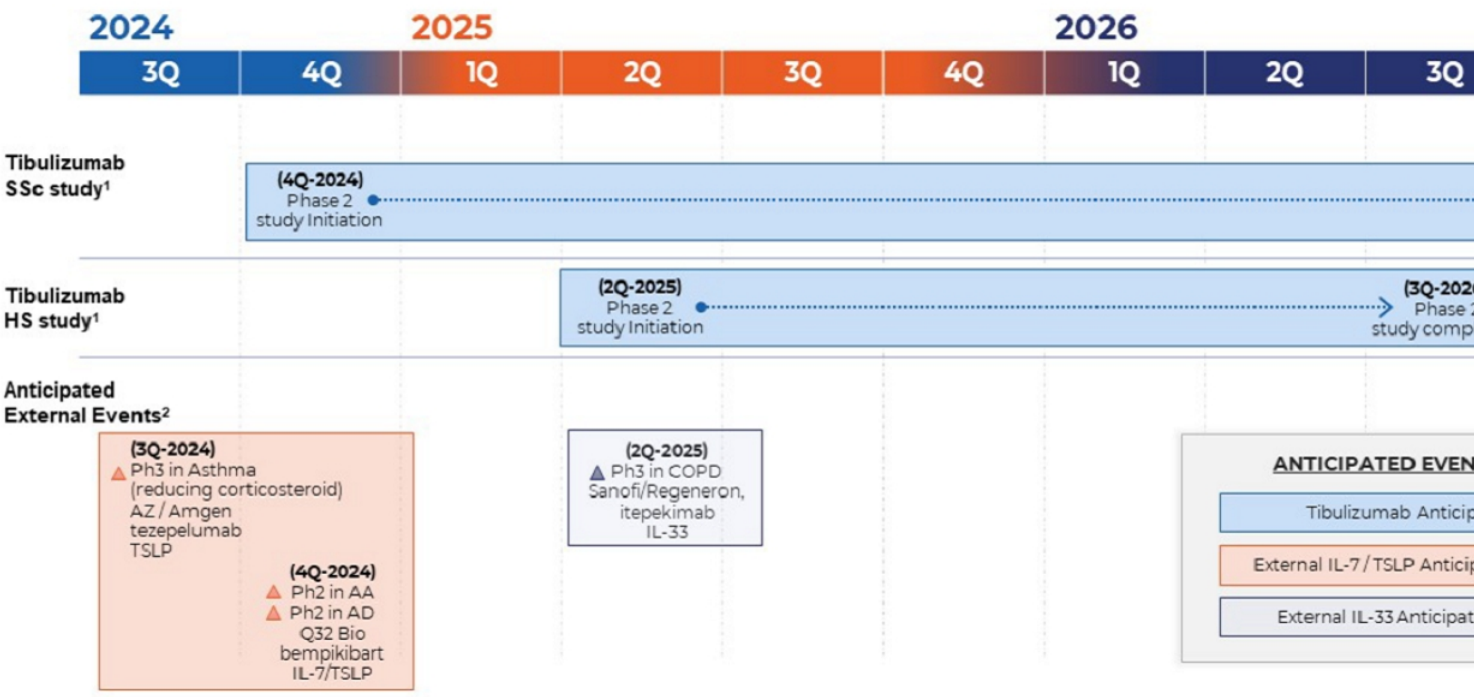
KIRAN NISTALA M.B.B.S., Ph.D.
Chief Medical Officer and
Head of Development

GARY WHALE Ph.D.
Chief Technology Officer

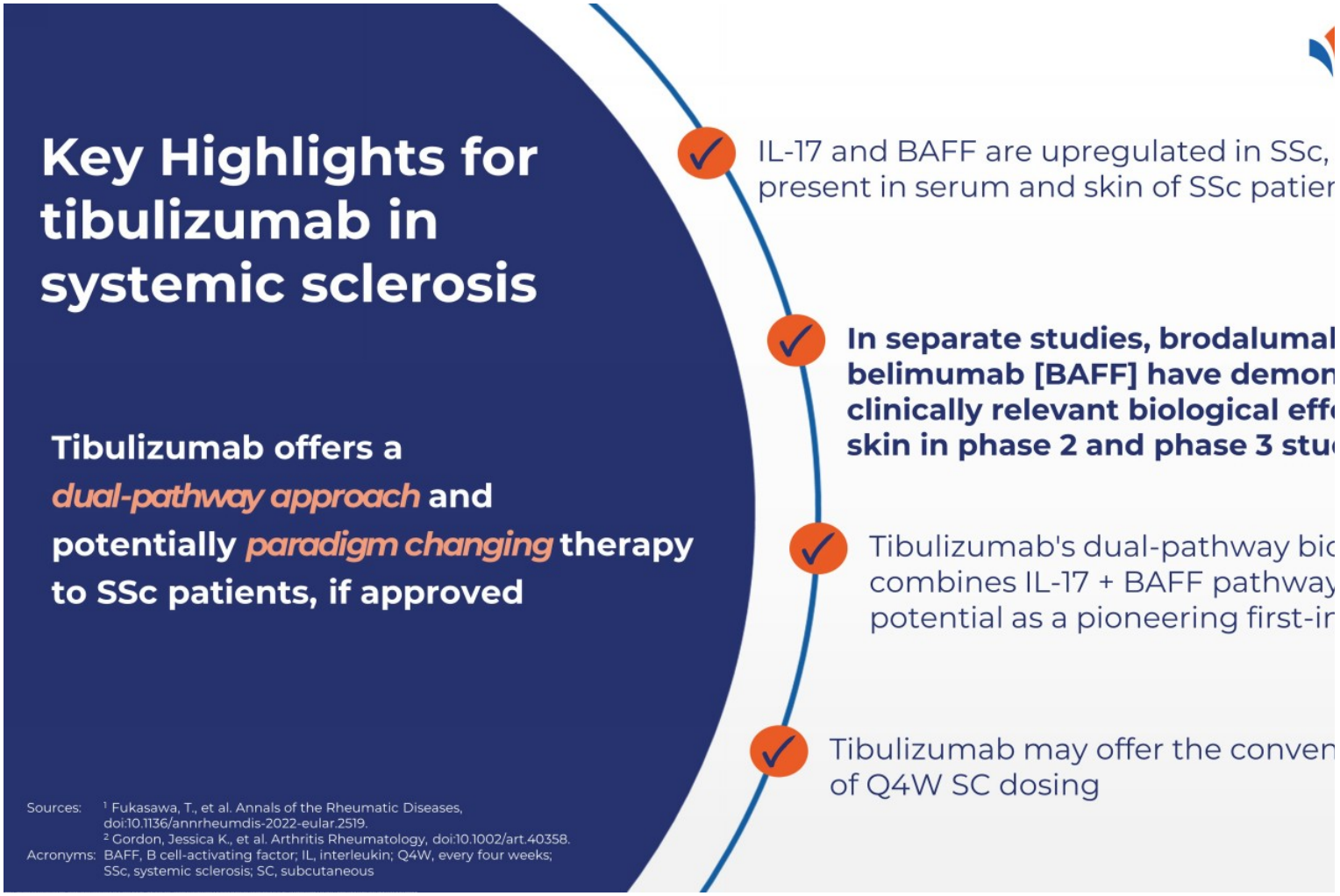
KIM DAVIS J.D.
Chief Legal Officer

MICHAEL HOWELL Ph.D.
Chief Scientific Officer and
Head of Translational Medicine

Key Anticipated Events through 2026



Sources: [1] Zura Planning Assumptions, [2] clinicaltrials.gov, Company Presentations
Acronyms: AA, alopecia areata; AD, atopic dermatitis; COPD, chronic obstructive pulmonary disease; CRO, contract research organization; FDA, Food and Drug Administration; HS, hidradenitis suppurativa; IL, interleukin; SSc, systemic sclerosis; TLD, topline data; TSLP, thymic stromal lymphopoietin; UC, ulcerative colitis


Key Highlights for tibulizumab in systemic sclerosis
Tibulizumab offers a dual-pathway approach and potentially paradigm changing therapy to SSc patients, if approved
IL-17 and BAFF are upregulated in SSc, present in serum and skin of SSc patien
In separate studies, brodalumal belimumab [BAFF] have demon clinically relevant biological eff skin in phase 2 and phase 3 stu
Tibulizumab's dual-pathway bio combines IL-17 + BAFF pathway potential as a pioneering first-in
Tibulizumab may offer the conven of Q4W SC dosing
Sources: [1] Fukasawa, T., et al. Annals of the Rheumatic Diseases, doi:10.1136/annrheumdis-2022-eular.2519.
[2] Gordon, Jessica K., et al. Arthritis Rheumatology, doi:10.1002/art.40358.
Acronyms: BAFF, B cell-activating factor; IL, interleukin; Q4W, every four weeks; SSc, systemic sclerosis; SC, subcutaneous

Tibulizumab is designed to target the combination of two clinically validated pathways for SSc

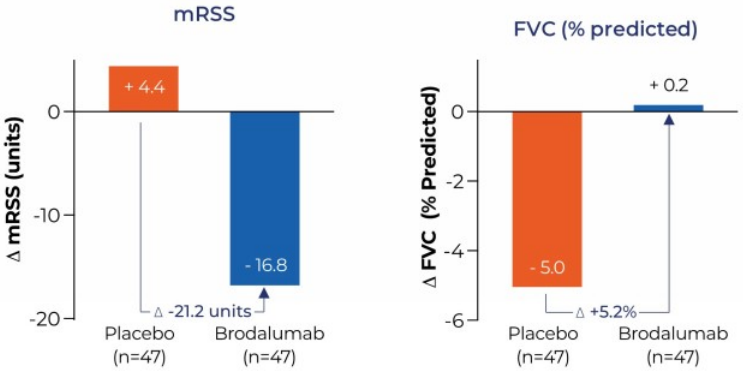
Brodalumab

IL-17 receptor antagonist

- Achieved 1° endpoint of treatment difference of least square mean: (−21.2 [95% CI -3.9, -18.5]; P<0.001), in mRSS and 2° endpoint of improved FVC, both at 24 weeks [1]
- Demonstrated therapeutic effects on lung/respiratory functions, digital ulcers, the symptoms of gastroesophageal reflux disease, and QOL without noteworthy safety concerns

CLINICAL PRECEDENT

Phase 3 brodalumab study (24 weeks)



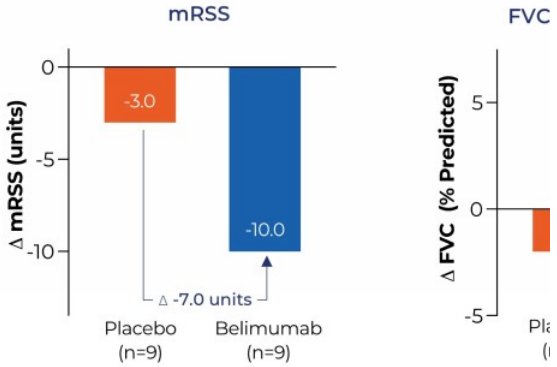
Belimumab

BAFF antagonist

- 52-week, investigator initiated, single center, doub placebo-controlled pilot study in 20 participants w on MMF [2]
- Both treatment groups experienced improvement favoring belimumab (-10 vs -3; p=NS)
- Secondary endpoints were met with statistical sig two endpoints: SHAQ-DI and VAS Raynaud's phen

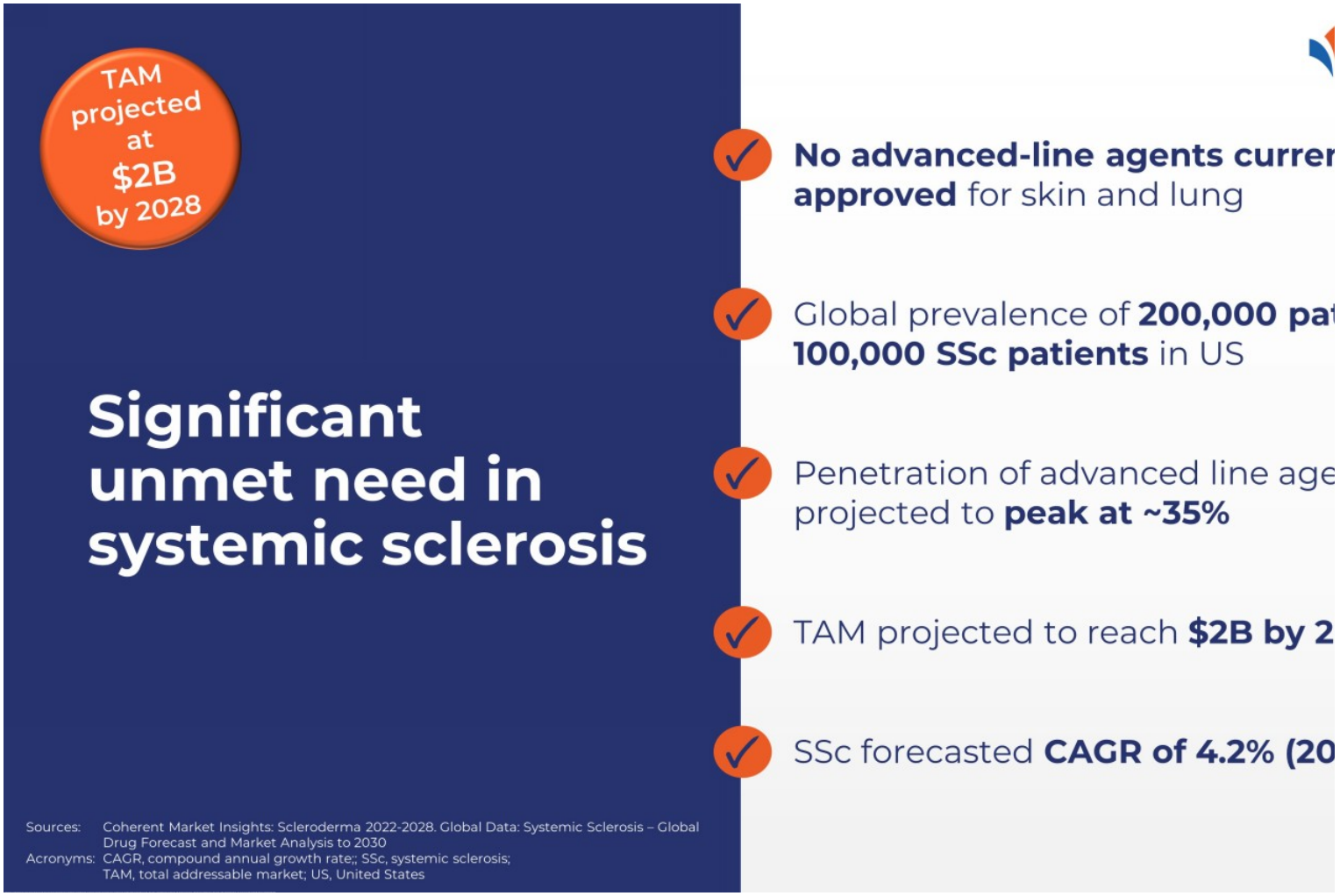
CLINICAL PRECEDENT

Phase 2 belimumab IIT study (52 v

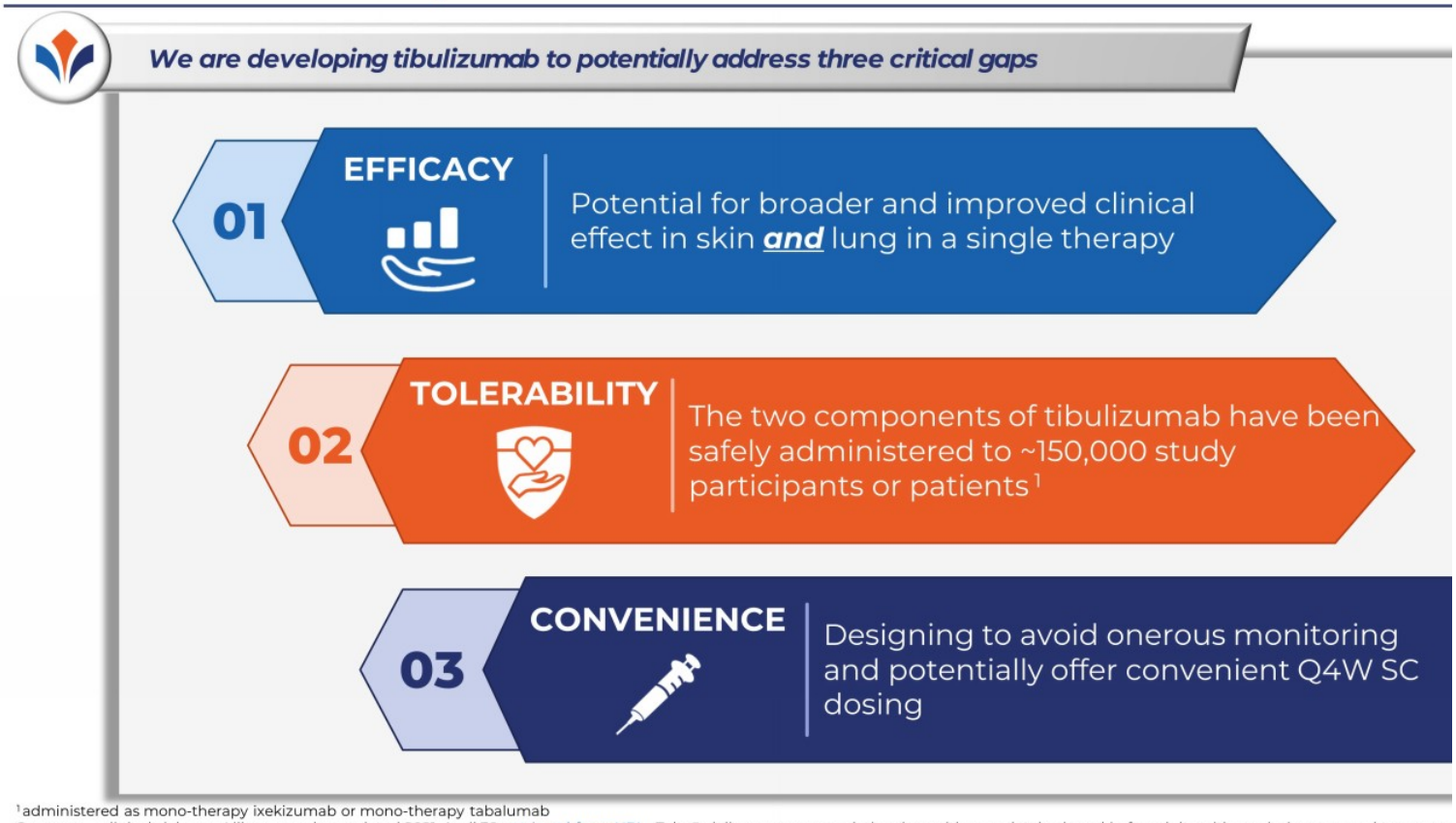


Sources: [1] Fukasawa, T., et al. Annals of the Rheumatic Diseases, doi:10.1136/annrheumdis-2022-eular.2519. [2] Gordon, Jessica K., et al. Arthritis Rheumatology, doi:10.1002/art.40358.

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


TAM projected at $2B by 2028
Significant unmet need in systemic sclerosis
No advanced-line agents currer approved for skin and lung
Global prevalence of 200,000 pat 100,000 SSc patients in US
Penetration of advanced line age projected to peak at ~35%
TAM projected to reach $2B by 2
SSc forecasted CAGR of 4.2% (20
Sources: Coherent Market Insights: Scleroderma 2022-2028. Global Data: Systemic Sclerosis – Global Drug Forecast and Market Analysis to 2030
Acronyms: CAGR, compound annual growth rate;; SSc, systemic sclerosis; TAM, total addressable market; US, United States

We are developing Tibulizumab as a differentiated treatment for SSc patients



[1] administered as mono-therapy ixekizumab or mono-therapy tabalumab

Sources: clinicaltrials.gov, Lilly press release, dated 2021, April 30, retrieved from URL., Taltz® delivers more cumulative days with completely clear skin for adults with psoriasis compared to seven c biologics in novel network meta-analysis

Acronyms: HCP, healthcare provider; Q4W, every four weeks; SC, subcutaneous; SSc, systemic sclerosis

Key Highlights for tibulizumab in hidradenitis suppurativa

Tibulizumab combines *two validated HS mechanisms* into one single therapy

- Scientific validation of the role of IL-17 and B hidradenitis suppurativa
- Multiple positive phase 2 and phase 3 stu the industry with IL-17 inhibitors or B cell therapies [1]
- Despite new options unmet need rem adjusted HiSCR75 deltas are in the 20
- Dual-pathway biology combines two validated therapeutic targets into a si
- Developing to potentially offer convenient dosing

Sources: [1] Company Presentations, Publications and Research.
Acronyms: HiSCR, Hidradenitis Suppurativa Clinical Response; HS, hidradenitis suppurativa; IL, interleukin; PBO, placebo, Q4W, every four weeks; SC, subcutaneous

Role of IL-17 and B cells is clinically validated, however clinical effect remains modest with single-pathway inhibition

Company / Asset*		NOVARTIS COSENTYX®	NOVARTIS remibrutinib*	UCB BIMZELX®	MoonLake sonelokimab	MoonLake sonelokimab	ACELYRIN izokibep	ACELYRIN izokib
Mechanism		IL-17 A	BTKi	IL-17 A/F	IL-17 A/F	IL-17 A/F	IL-17 A/A	IL-17 A
Administration		SC/IV	PO	SC	SC	SC	SC	SC
Phase		Phase 3	Phase 2b	Phase 2	Phase 2	Phase 2	Phase 2b	Phase
Dosing		30mg Q2W for 16W	100 mg or 25 mg BID	320mg Q2W for 12W	120mg Q2W for 12W	120mg Q2W for 24W	160mg QW for 12W	160 mg Q QW for
Total Patients		n = 360	N = 77	n = 88	n = 234	n = 234	n = 30	n = 1
Efficacy (HiSCR50)	Non-Placebo Adjusted	42% - 45%	48.5% - 72.7%	63%	66%	76%	71%	42% - 4
	Placebo Adjusted	11% +	38%	35%	38%	48%	N/A	1% - 5
Efficacy (HiSCR75)	Non-Placebo Adjusted	N/A	27.3% - 42.4%	50%	43%	57%	57%	34% - 3
	Placebo Adjusted	N/A	24%	29%	29%	N/A	N/A	5% - 1
Safety	Candidiasis	0% - 3%[1]	0	9%	10.5%	>10%	0%[2]	TBD

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

Sources: Company Presentations, Publications and Research.

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

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

TAM projected at $2.2B By 2030

Significant opportunity and clinical need in hidradenitis suppurativa

- US estimates of **300,000 to 400,**
  patients
- **High market need**, 60% of HS pa
  biologic eligible
- Tibulizumab may offer **convenie**
  dosing regimen

Assumes COSENTYX® becomes first line biologic for HS following FDA approval for HS on 31-Oct-2023.
Sources: Medical Literature, MEDACorp KOLs, Company websites, IQVIA, US Department of Veteran's Affairs, Zura Bio Management
Acronyms: HS, hidradenitis suppurativa; Q4W, every four weeks; SC, subcutaneous

We are developing tibulizumab as a differentiated treatment for HS patients

We are developing tibulizumab to potentially address three critical gaps

01 EFFICACY

Combined IL-17 + BAFF inhibition potentially results in broader and improved clinical responses for HS patients

02 TOLERABILITY

The two components of tibulizumab have been safely administered to ~150,000 study participants or patients[1]

03 CONVENIENCE

Developing to avoid onerous monitoring; potentially offer convenient Q4W SC dosing

[1] administered as mono-therapy ixekizumab or mono-therapy tabalumab

Sources: clinicaltrials.gov, Lilly press release, dated 2021, April 30, retrieved from URL., Taltz® delivers more cumulative days with completely clear skin for adults with psoriasis compared to seven other biologics in novel network meta-analysis

Acronyms: BAFF, B cell-activating factor; HS, hidradenitis suppurativa; IL, interleukin; Q4W, every four weeks; SC, subcutaneous


tibulizumab
ZB-106
Anti-BAFF x 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)

Initial area of development in orphan disease, systemic sclerosis

Systemic sclerosis is a rare & life-threatening disease with no approved therapy

~200,000
people with SSc in US, EU and Japan [1]

40-60%
mortality in 10 years [2]

Zero
SSc-specific * drugs approved

$2B+
annual potential market opportunity

Sources: Medscape, BMJ best practice [1] Health Advanced, LLC; Lenabasum Commercial Market Assessment. [2] Tyndall et al, 2010 [3] Bergamasco, A. et al., Clin Epidemiol. 2019 Apr 18;11:257-273 [4] Zura Bio internal analysis and benchmarking, [5] Internal assumption based on demand research and rare disease analogues

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

No effective treatment exists that combats the disease acr

Systemic sclerosis is characterized by tissue inflammat

Lung

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

Skin

O

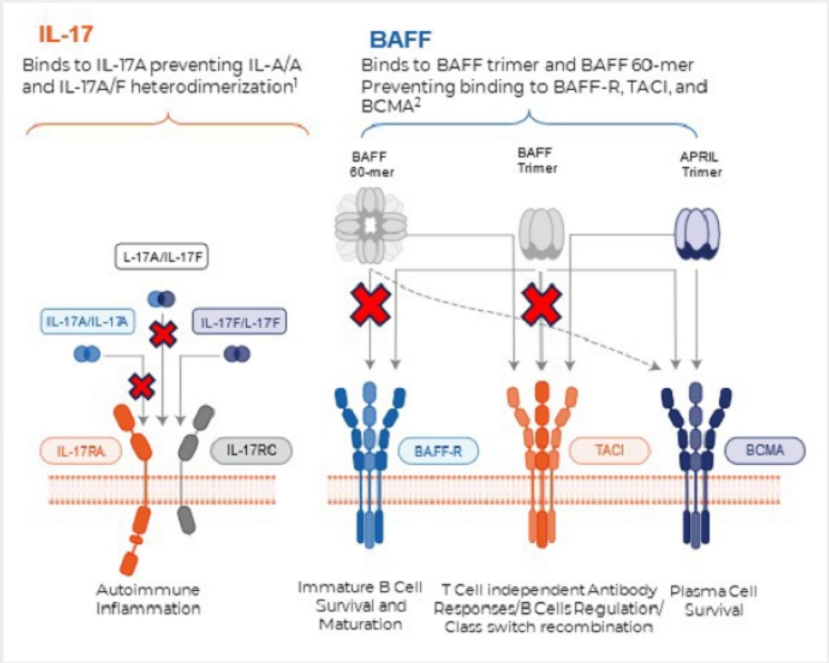
Tibulizumab has the potential to provide bro working in more patients not just certai

IL-17 and BAFF-Mediated Inflammation both contribute to SSc progression

SSc includes the presence of autoantibodies, and aberrant activation of B-cells, T-cells,

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

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



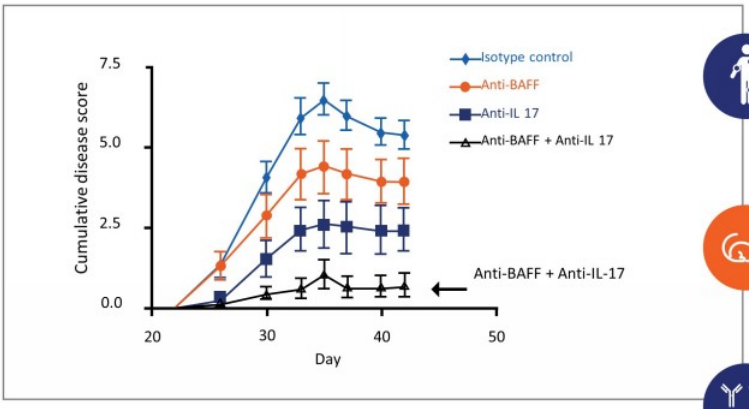
B cell activati
a potent B-ce
promotes the
differentiatio

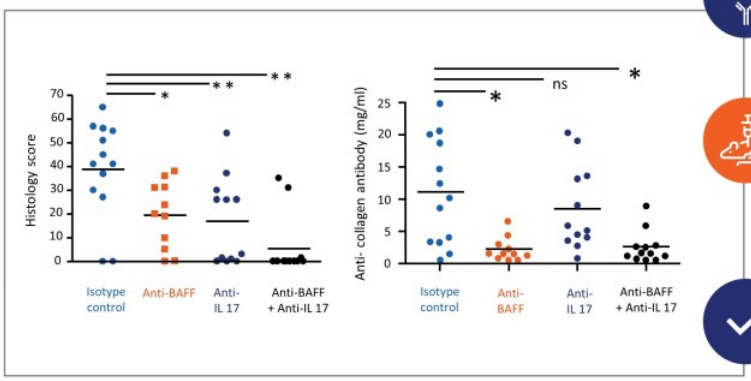
- BAFF is in
  blood and
  fibrosis an
  pulmonar
- In pre-clin
  blockade
  autoantib

Combined approaches to address T-cell and B-cell drivers of autoimmunity have the potential to increas

Sources: [1] Zhou et al. Human Immunology 2015; [2] Yang et al. Arthritis Res Ther 2014; [3] Cipolla et al. FASEB 2017; [4] Matsushita et al. Arthritis Rheum 2006; [5] Matsushita et al. J Rheum 2007; [6] Matsushita et al. J Invest Dermatol 200
[7] François et al. J Autoimmun 2015

Synergistic benefit of IL-17 and BAFF Neutralization has been demonstrated in classic Collagen Induced Arthritis (CIA) model





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

The CIA murine model is similarly characterize **increased IL-17 production** and B cells that d pathogenesis

Surrogate antibodies were used to evaluate w **neutralization of IL-17 and BAFF** was superio 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, IND Briefing

Tibulizumab is Clinically De-Risked Through Phase 1b

78 Participants Dosed Across Three Phase 1/1b studies

57 participants with single dose; 21 participants with multiple dose up to 12 weeks

PHARMACOKINETICS

- $t_{1/2}$ is 26.9 days
- Bioavailability after SC doses was 62.9%
- At doses tested there is evidence of maximum target engagement with clinical safety supporting 6-fold "window" between max target engagement and max human dose tested

Established dosing regimen

PHARMACODYNAMICS

- In Phase 1b studies in both RA and Sjögren's there were multiple impacts on PD markers:
  - Decrease in CD20+ B-cells with higher doses generally associated with larger changes from baseline
  - Decrease in hs-CRP AUC was associated with higher ZB-106 AUCs

Demonstrated PD in participants in Ph1b

SAFETY and

- SAD Studies: No deaths
- MAD study: No deaths, related SAE of neutrope resolution
- Most frequent TEAE: He transient neutropenia, diarrhea
- No TEAE of infection at doses
- In the MAD study, one participant had TE-ADA detected at a low titer

Safety / ADA pr in line with TA

Tibulizumab is a highly validated molecule that enables the opportunity to deliver on the promise of both IL-17 and BAFF inhibition in autoimmune disease

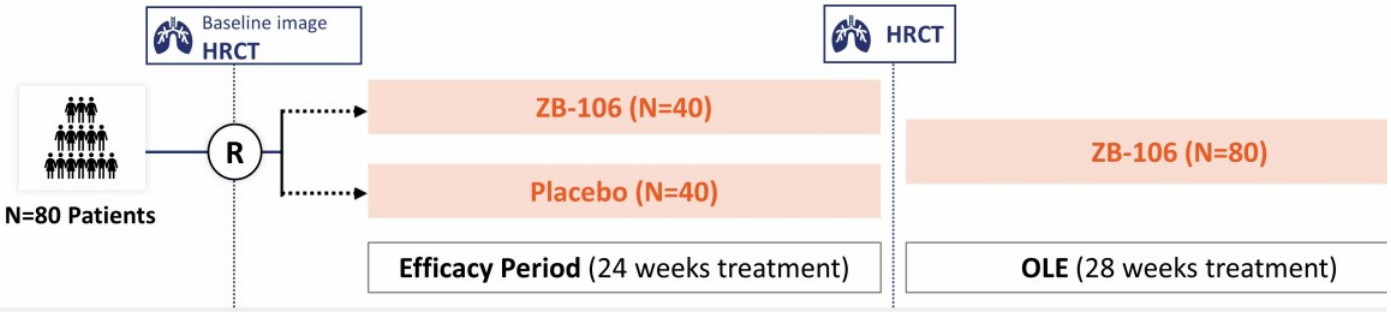
Abbreviations: MAD, multiple ascending dose; SAD, single ascending dose

Phase 2 SSc study focused on skin/lung endpoints

Key Inclusion Criteria

- Early diffuse cutaneous SSc, enriched for SSc-ILD
- mRSS 15-45
- Disease duration < 5years
- Stable background therapy, including MMF for 6 r
- Anti-centromere antibody negative

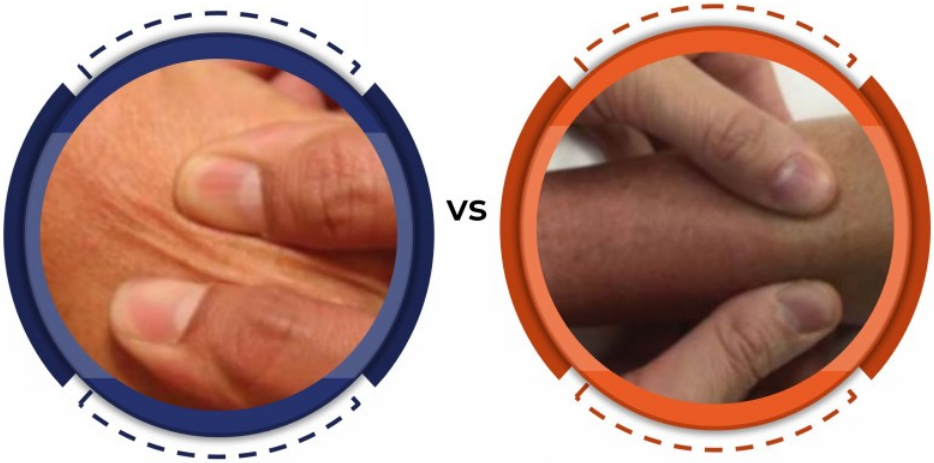
Randomized Trial (mRSS and HRCT)



Key Efficacy Endpoints

mRSS (Primary) | qHRCT / FVC | HAQ-DI (Function) | Clinician / Pat

Assessing Skin Thickness and Fibrosis with modified Rodnan skin score (mRSS)

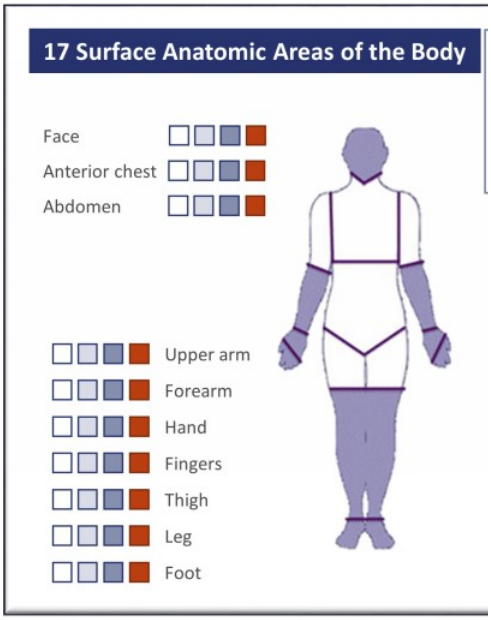


Fine Wrinkles (0/3) VS **Severe Thickness (3/3)**

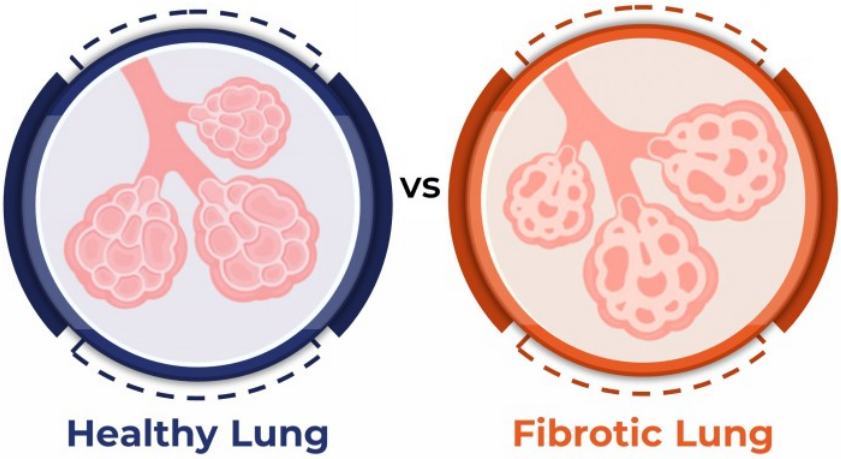
Severe skin thickening and tightening restricts movement and causes painful ulcers on the hands and fingers, significantly impairing daily activities and quality of life.

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

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

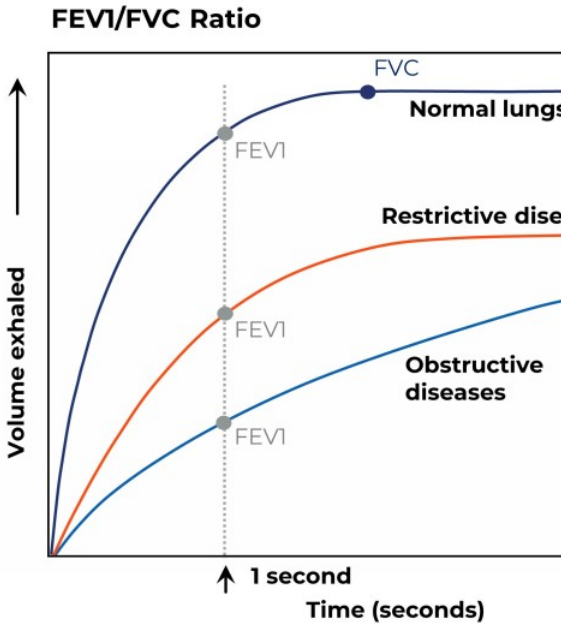


Assessing Interstitial Lung Disease (ILD) progression in SSc



ILD encompasses a diverse group of pulmonary disorders characterized by inflammation and progressive fibrosis of the lung interstitium, leading to restrictive lung physiology and impaired gas exchange.

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



Phase 2 SSc development aims to reduce historical risks associated with therapeutic area development

Historic drivers of SSc study failures

1. Novel, and unvalidated mechanisms
2. Inclusion/exclusion criteria misses
3. Balancing sample size for mRSS and ILD participants

Increase probability of success

1. Larger study sample size increases probability of success (mRSS)
2. Sufficient sample size for ILD to understand potential Phase 3 effect
3. High Resolution CT highly correlates with FVC > ILD read-through

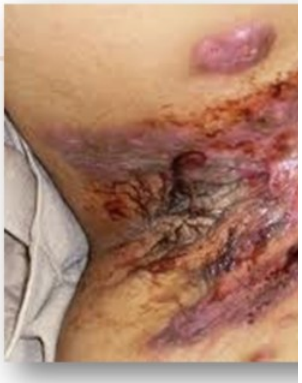
Acronyms: CT, computed tomography; FVC, forced vital capacity; IL, interleukin; ILD, interstitial lung disease; mRSS, modified Rodnan skin score


tibulizumab
ZB-106
Anti-BAFF x 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

TAM projected at $3.5 -$4B By 2030

Overview of hidradenitis suppurativa (HS)

DISEASE OVERVIEW



- Hidradenitis suppurativa is an inflammatory follicular skin disease
- Skin lesions develop in axillae, in the groin, and under the breasts, are formed as a result of inflammation & infection of sweat glands and are characterized by:
  - Recurrent boil-like nodules and abscesses that culminate in pus-like discharge
  - Difficult-to-heal open wounds (sinuses) 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]

Estimated

~300K people

living with Hidradenitis suppurativa in the U.S.

(1-2% global prevalence)

Average of

7 years

to diagnose globally

High unmet n

>50% patients inadequately

According to HiSCR

CURRENT APPROVED TREATMENTS ONLY AIM TO MANAGE SYMPTOMS AND INCLUDE ST IMMUNOSUPPRESSANTS TO MANAGE SYSTEMIC SYMPTOMS

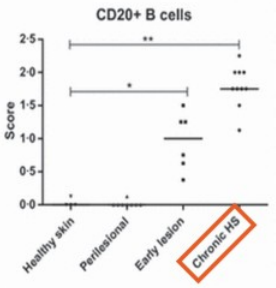
Sources: [1] Moran, Barry, et al. Journal of Investigative Dermatology, doi:10.1016/j.jid.2017.05.033. [2] Banerjee, Anirban, et al. Immunological Investigations, doi:10.1080/08820139.2016.1230867. [3] Sabat, Robert, et al. Journal of Allergy an Clinical Immunology, doi:10.1016/j.jaci.2022.10.034. [4]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

B Cell Signaling Potentiates HS Disease

Pathogenic Role for B Cells and Plasma Cells

- CD20+ B and CD138+ Plasma Cells are increased in chronic HS lesions [1]

CD20+ B cells in HS Lesions

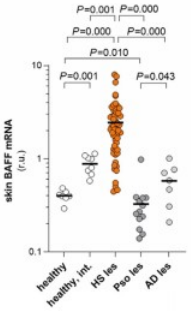


- B cell depletion with rituximab provided therapeutic benefit with 4 out of 5 cases reporting complete remission of HS lesions [5]

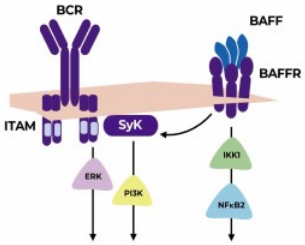
BAFF Drives B Cell Activation and Inflammation

- Increased BAFF expression in HS lesions and tunnels [2-4]
- Neutralization of BAFF in HS lesional explants reduced the expression of B & plasma cell gene signatures [2]

BAFF gene expression in HS



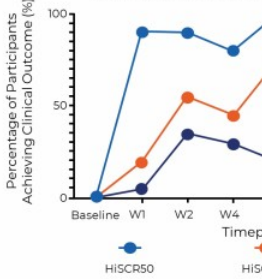
BAFF is essential for B cell activation



Clinical Benefit o B Cell

- Modulating B cell functi fostamatinib (SYK inhib therapeutic benefit in H
- B cell depletion with ritu therapeutic benefit [5]
- 4/5 cases report comple lesions [5]

B cell disruption is

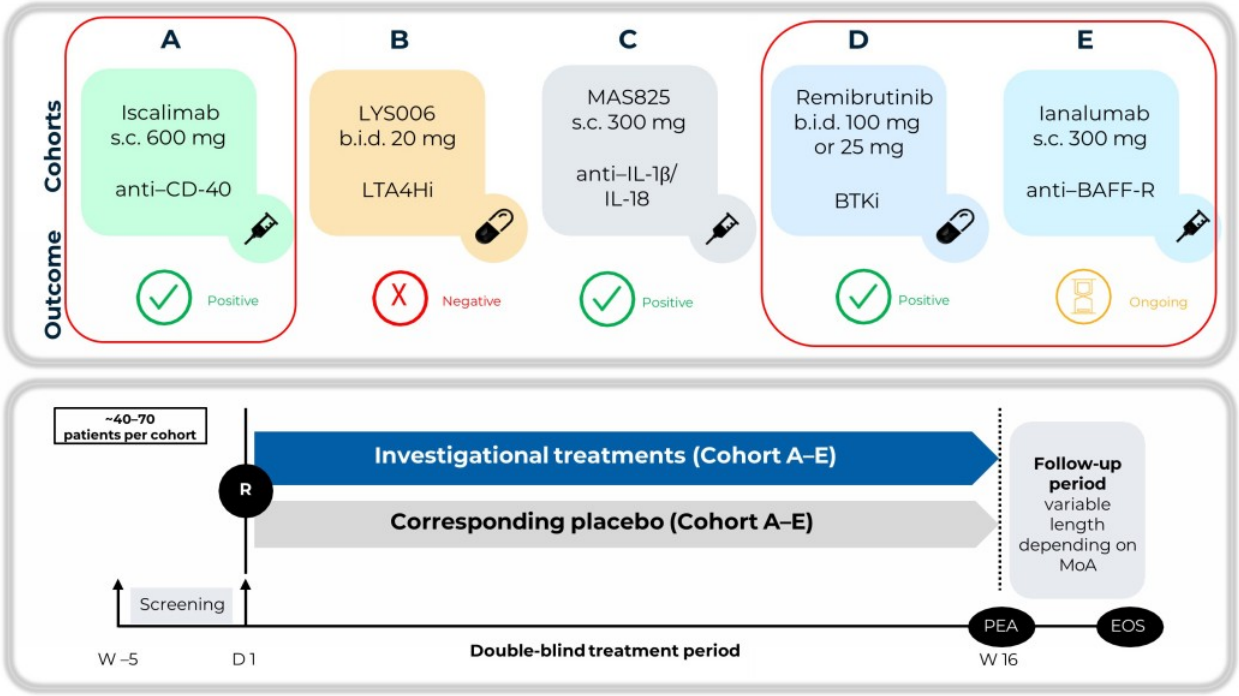


Week 12	%
Fostamatinib (SYK inhibition)[6]	

Sources: [1] Van der Zee, H.H., et al. British Journal of Dermatology, doi:10.1111/j.1365-2133.2011.10698.x. [2] Rumberger, Beth E., et al. Inflammation Research, doi:10.1007/s00011-020-01381-7. [3] Sabat, Robert, et al. Journal of Allergy and Clinical Immunology, doi:10.1016/j.jaci.2022.10.034. [4] Gudjonsson, Johann E., et al. JCI Insight, doi:10.1172/jci.insight.139930. [6] Jepsen, Rebecca, et al. Journal of the American Academy of Dermatology, doi:10.1016/j.jaad.2023.05.076.

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

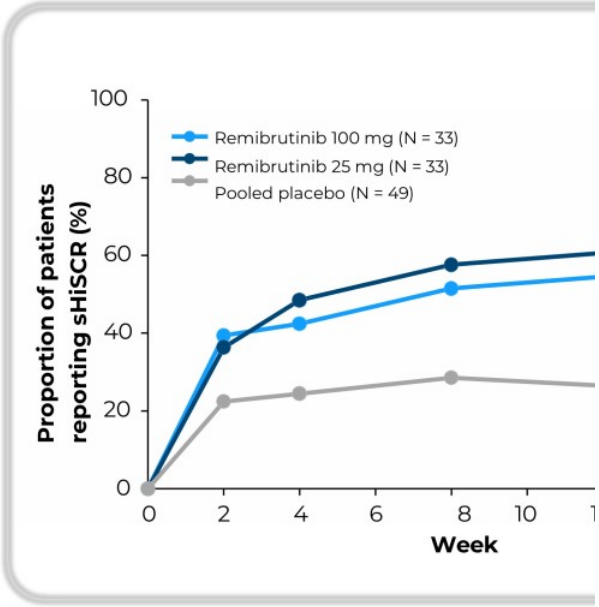
*Study started in February 2019 and is currently ongoing.
BAFF-R, B-cell activating factor of the tumor necrosis alpha family receptor; b.i.d., twice daily; BTKi, Bruton's tyrosine kinase inhibitor; CD, cluster of differentiation; D, day; EOS, end of study; HS, hidradenitis suppurativa; IL, interleukin;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

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

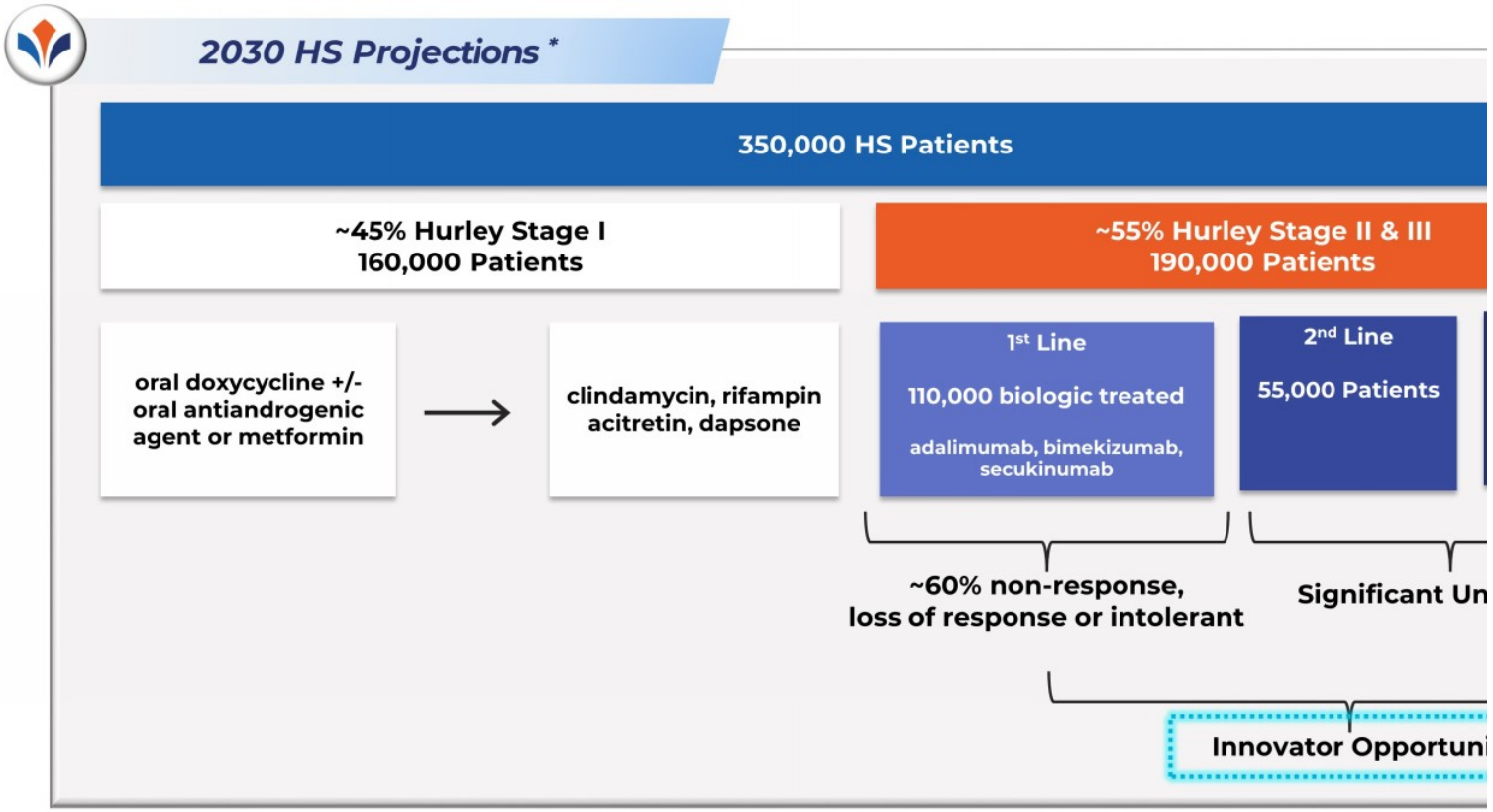
- The primary endpoint of this study was met for both doses of remibrutinib; patients treated remibrutinib reported a greater rate of sHiSCR* at Week 16 compared with placebo

	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	



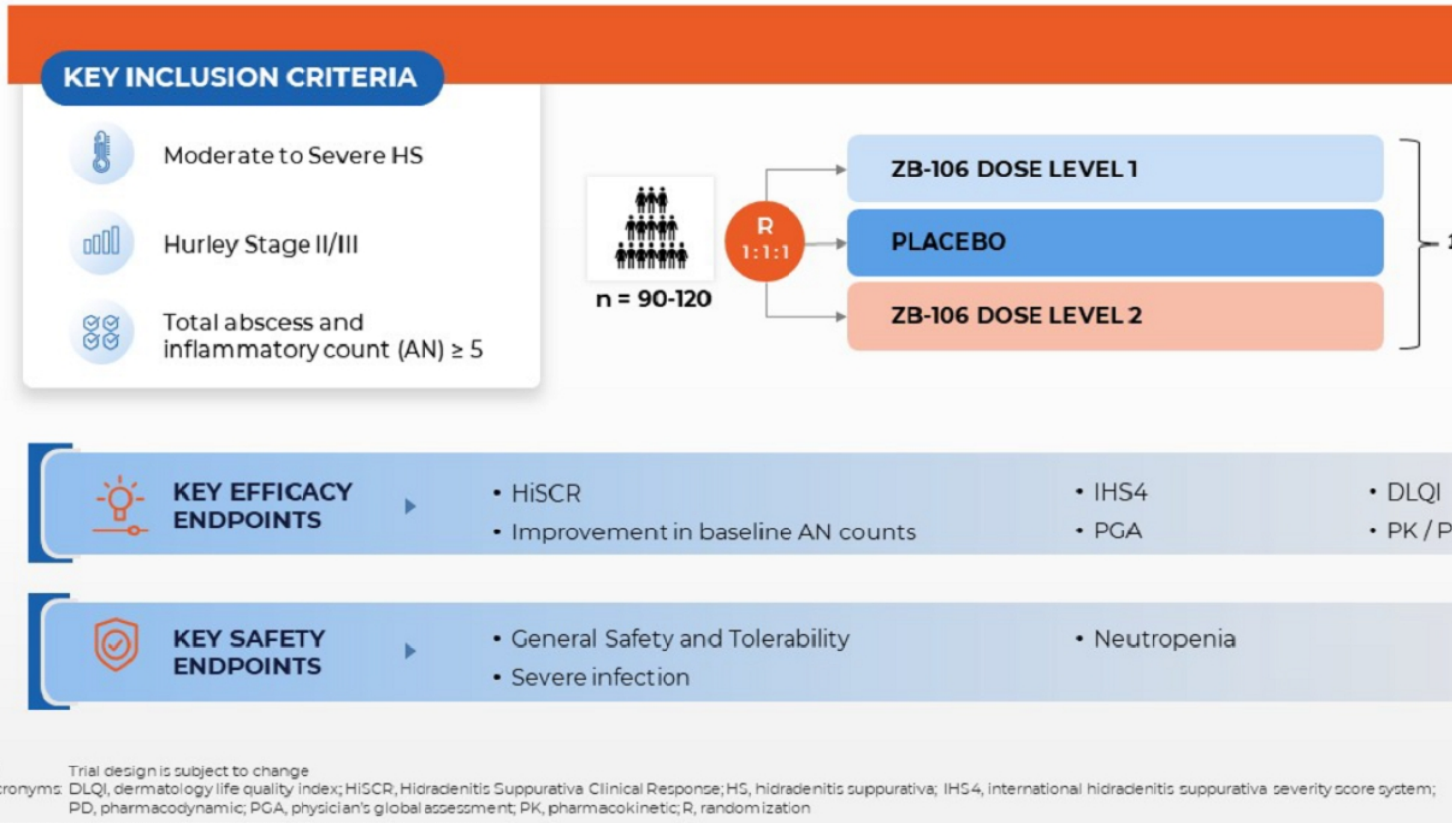
*The sHiSCR is defined as a ≥50% reduction in the abscess and inflammatory nodule count and no increase in draining tunnels compared with baseline. †Difference refers to the difference between remibrutinib (either dose) and pooled placebo at Week 16. ‡Bayesian posterior probability of remibrutinib (either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

HS innovator expected to be uniquely positioned to capture opportunities across 1st, 2nd, and 3rd-line HS patients



(*) Assumes Cosentyx® becomes first line biologic for HS following FDA approval for HS on 31-Oct-2023.
Sources: Medical Literature, MEDACorp KOLs, Company websites, IQVIA, US Department of Veteran's Affairs, Zura Bio Management
Acronyms: HS, hidradenitis suppurativa

Planned Phase 2 HS Trial Design*



(*) Trial design is subject to change

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


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, a multi-functional antibody with cytokine signaling via IL-7R and TSLP pathways

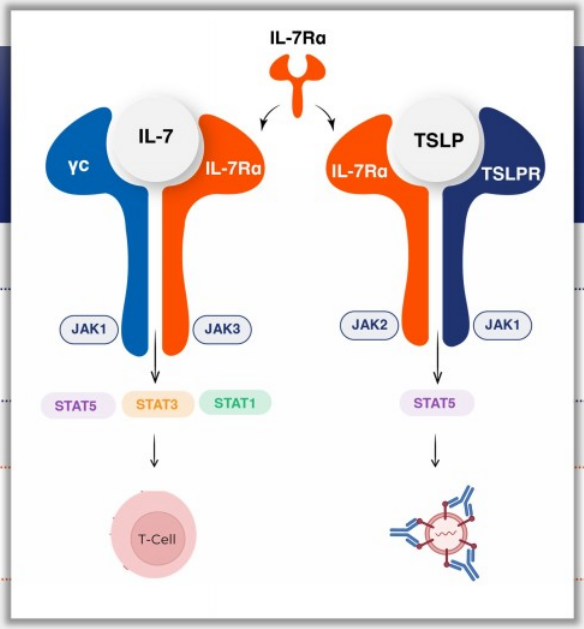
IL-7Rα is a key receptor in immune regulation, central to the signaling of cytokines ***IL-7*** ar

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

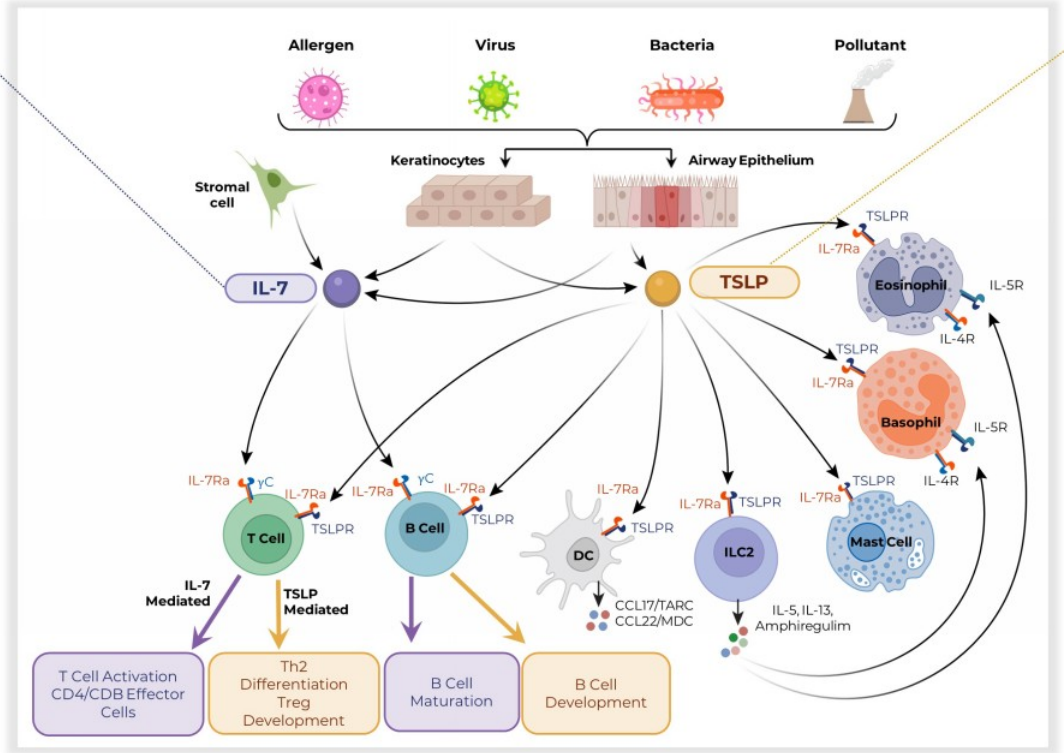
Commonly tied to al
and specific inflamn

Positioning crebankitug for diverse immune-related and autoimmune conditions

Both TSLP and IL-7 have a role in activating Th1, Th2 and Th7 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 clin seve sho ther add dise

Sources: 1. Ebina-Shibuy, 2022. Nat Rev Immunol, 2. Marone, 2019. Expert Opin Investig Drugs, 3. Menzies-Gow, 2020. Respir Res, 4. Chen, 2021. Frontiers Immunol, 5. Herold, 2019 JCI Insight, Graphic created in BioRender; 5. doi.org/10.3389/fimmu.2018.02692, 6. doi.org/10.1016/j.isci.2020.101421, 7. https://www.frontiersin.org/articles/10.3389/fimmu.2020.01557/full


torudokima
ZB-880
Anti-IL-33
Torudokimab is a fully human, high affinit
antibody that neutralizes IL-33, preventin
and ST2-independent (e.g., RAGE) inflamm

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 Ph1 and Ph2 trials conducted by Eli Lilly[2]

- 141 healthy volunteers in Ph1 study
- 103 participants with moderate to severe atopic dermatitis in Ph2
- 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

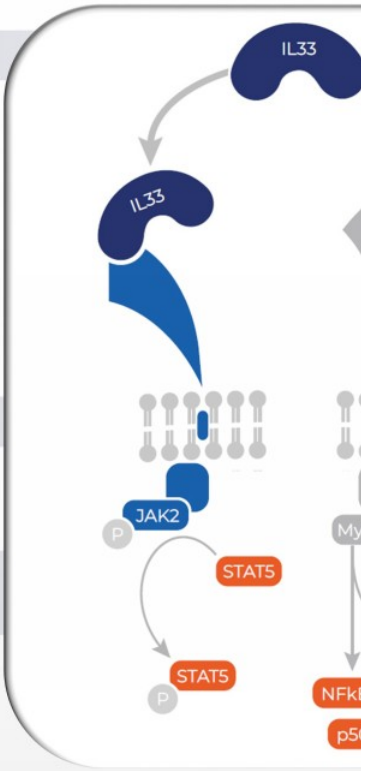
Mechanism of Action

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 COPD4 and asthma[5]



Sources: 1. Cohen et al. 2015 Nature, 2. https://clinicaltrials.gov/ct2/show/NCT03913260; https://clinicaltrials.gov/ct2/show/NCT03343587; https://clinicaltrials.gov/ct2/show/NCT03831191, Section 6.1, DSUR for period 23-Sep-2019 to 2
Sep-2020, 3. doi.org/10.1111/bjd.21631 4. Okragly et al Journal of Inflammation Research 2021:14 3823–3835, 5.. doi:10.1056/NEJMoa2024257

Torudokimab IL-33 Pathway

IL-33 is a member of the IL-1 cytokine family that is constitutively expressed in structural and lining cells including fibroblasts, endothelial, and epithelial cells of skin, gastrointestinal tract, and lungs[1]

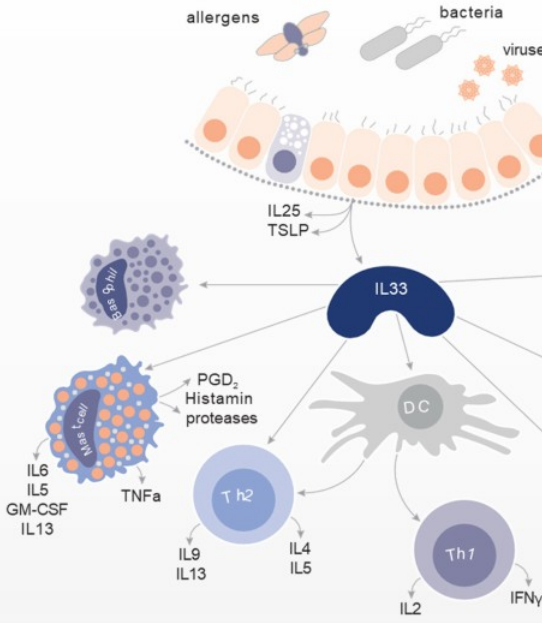
IL-33 is released from the epithelium by disease exacerbating environmental factors and is an important amplifier of innate inflammation during exacerbations[2]

Polymorphisms in IL-33 and ST2 are associated with increased eosinophil production as well as respiratory diseases such as COPD and severe asthma

IL-33 inhibition clinically validated in severe asthma, COPD3, and subsets of other epithelial disorders[4]

Pre-clinical data demonstrates superior activity of torudokimab compared with etokimab suggesting the potential for best-in-class activity[5]

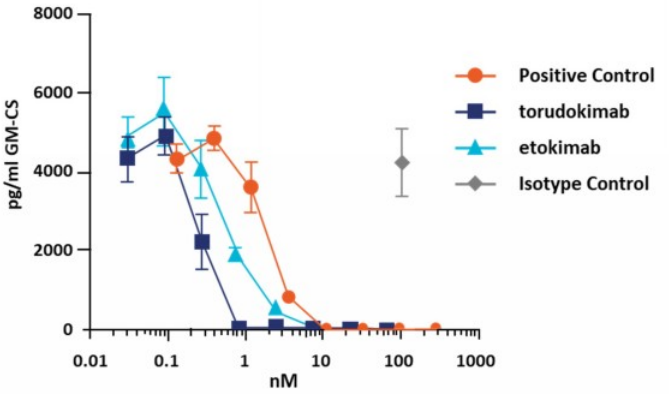
Emerging biology suggests expanding opportunity for IL-33 in fibrotic and autoimmune conditions[6]

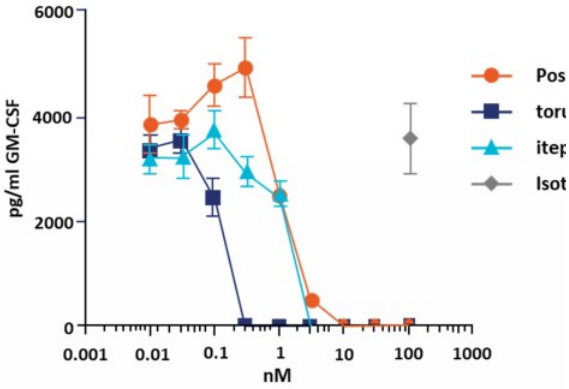


Sources: 1. Chan, 2019. Frontiers Immunol, 2. doi.org/10.1016/j.cyto.2022.155891, 3. https://doi.org/10.1038/ng.323 and doi:10.1016/j.jaci.2020.04.051 , 4. .:https://doi.org/10.1016/S2213-2600(22)00005-4; doi:10.1056/NEJMoa2024257 and doi:10.1126/scitranslmed.aax2945 , 5. Sci Trans Med., Zura Bio Internal data, 6. doi: 10.1111/imm.12174; https://doi.org/10.3389/fphys.2021.781012 and https://doi.org/10.3389/fmed.2021.739489

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

Torudokimab was 2.9 and 5.5-fold more potent than etokimab and itepekimab, 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)	Torudokin
torudokimab (LY3375880)	1.7×10^{6}	6.7×10^{-5}	39	
etokimab (AnaptysBio)	9.4×10^{5}	1.2×10^{-4}	112	2
itepekimab (Regeneron)	7.6×10^{5}	1.6×10^{-4}	215	5

Sources: Zura Bio Internal data