

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

October 27, 2026
Date of Report (Date of earliest event reported)

Zura Bio Limited
(Exact name of registrant as specified in its charter)

Cayman Islands (State or other jurisdiction of incorporation)	001-40598 (Commission File Number)	98-1725736 (I.R.S. Employer Identification No.)
1489 W. Warm Springs Rd. #110 Henderson, NV 89014 (Address of principal executive offices, including zip code) (702) 825-9872 (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 2.02. Results of Operations and Financial Condition.

On October 27, 2026, Zura Bio Limited (the “Company”) issued a press release announcing its third quarter 2026 financial results. A copy of the press release is furnished herewith as Exhibit 99.1 and incorporated herein by reference.

The information furnished under this Item 2.02 (including Exhibit 99.1) shall not be deemed “filed” for purposes of Section 18 of the Securities Exchange Act of 1934, as amended (the “Exchange Act”), or subject to the liabilities of that section or Sections 11 and 12(a)(2) of the Securities Act of 1933, as amended (the “Securities Act”). The information in this Item 2.02 (including Exhibit 99.1) shall not be deemed incorporated by reference into any other filing with the Securities and Exchange Commission (the “SEC”) made by the Company, whether made before or after the date hereof, regardless of any general incorporation language in such filing, except as expressly set forth by specific reference in such filing.

Item 7.01. Regulation FD Disclosure.

On October 27, 2026, the Company provided an updated corporate presentation that may be used in connection with upcoming presentations at conferences and investor meetings. The full text of the Company’s corporate presentation is filed as Exhibit 99.2 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 99.2) shall not be deemed “filed” for purposes of Section 18 of the Exchange Act, or subject to the liabilities of that section or Sections 11 and 12(a)(2) of the Securities Act. The information in this Item 7.01 (including Exhibit 99.2) 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, except as expressly set forth by specific reference in such a filing.

Item 9.01. Financial Statements and Exhibits.

(d) Exhibits.

Exhibit No.	Description
99.1	Press Release dated October 27, 2026
99.2	Corporate Presentation ,dated October 27, 2026
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.

Date: October 27, 2026

ZURA BIO LIMITED

By: /s/ Kim Davis

Kim Davis

Chief Operating Officer, Chief Legal Officer and Corporate Secretary



Zura Bio Reports Third Quarter 2026 Financial Results and Recent Corporate Updates

- *TibuSHIELD topline data in hidradenitis suppurativa (HS) remains on track for Q4 2026; enrollment completed with 247 participants randomized, exceeding target*
- *TibuSURE topline data in systemic sclerosis (SSc) remains on track for H1 2027; enrollment completed with 91 participants randomized, exceeding target*
- *NEXUS-PMR, a Phase 2 study evaluating tibulizumab in polymyalgia rheumatica (PMR), remains on track to initiate by year-end 2026*
- *Cash and cash equivalents of \$187.7 million as of September 30, 2026, which is expected to fund planned operations through at least the end of 2028*

HENDERSON, Nev.---(BUSINESS WIRE)---October 27, 2026--- Zura Bio Limited (Nasdaq: ZURA) (“Zura”), a clinical-stage biotechnology company developing novel and differentiated medicines to meaningfully improve the lives of patients with serious and debilitating autoimmune and inflammatory diseases, today announced financial results for the third quarter ended September 30, 2026, and provided recent corporate updates.

“Our team continues to execute efficiently on our development plan for tibulizumab, and we are excited as we enter this data-rich period for the company. We are on track to report topline results from our TibuSHIELD study in HS later this quarter, followed by topline results from our TibuSURE study in SSc in the first half of 2027,” said Sandeep Kulkarni, M.D., co-founder and Chief Executive Officer of Zura. “We continue to believe deeply in tibulizumab’s potential to address complex immune biology and unmet needs across indications, including in PMR, where we will be starting the NEXUS-PMR study by end of year. With cash runway sufficient to deliver results from all three studies, Zura is in a great position to deliver on tibulizumab’s unique potential.”

PROGRAM UPDATES

Tibulizumab (ZB-106)

Both ongoing Phase 2 studies of tibulizumab, a bispecific antibody designed to neutralize interleukin-17 (IL-17) and B-cell activating factor (BAFF), are fully enrolled and progressing toward topline data, and Zura also remains on track to initiate a Phase 2 study in PMR, the third indication for tibulizumab, by year-end 2026.

- **TibuSHIELD (HS):** The Phase 2 clinical study evaluating tibulizumab in adults with hidradenitis suppurativa completed enrollment in June 2026 with 247 participants, exceeding its target. Topline data remains on track for the fourth quarter of 2026 and will include the primary endpoint, percent change from baseline in total abscess and inflammatory nodule count at Week 16, as well as the proportion of participants achieving HiSCR50 or HiSCR75.
- **TibuSURE (SSc):** The Phase 2 clinical study evaluating tibulizumab in adults with early diffuse cutaneous systemic sclerosis completed enrollment in July 2026 with 91 participants, exceeding its target. Topline data remains on track for the first half of 2027 and will include the primary endpoint, change from baseline in modified Rodnan Skin Score (mRSS) at Week 24, as well as measures of lung involvement.
- **NEXUS-PMR (PMR):** The Phase 2 clinical study evaluating tibulizumab in adults with polymyalgia rheumatica, an inflammatory disease in which most patients remain dependent on long-term glucocorticoids, has been cleared by the U.S. Food and Drug Administration (FDA) to proceed under an Investigational New Drug application and remains on track to initiate by year-end 2026.



Tibulizumab is an investigational compound and has not been approved for marketing by the FDA or any other regulatory authority.

Additional Clinical-Stage Product Candidates

In addition to tibulizumab, Zura is continuing its evaluation of potential development approaches for torudokimab (ZB-880) and crebankitug (ZB-168), informed by current clinical and translational evidence and ongoing assessment of the evolving competitive landscape.

THIRD QUARTER 2026 FINANCIAL RESULTS

Cash Position

As of September 30, 2026, Zura had cash and cash equivalents of \$187.7 million. Zura expects that its existing cash and cash equivalents are sufficient to support planned operations through at least the end of 2028.

Research and Development (R&D) Expenses

R&D expenses were \$19.5 million for the third quarter of 2026, compared to \$11.9 million for the same period in 2025. The increase was primarily driven by the continued execution of Zura's Phase 2 tibulizumab clinical programs as well as start-up activities for NEXUS-PMR.

General and Administrative (G&A) Expenses

G&A expenses were \$9.3 million for the third quarter of 2026, compared to \$7.6 million for the same period in 2025. The increase was primarily due to higher professional fees.

Net loss for the third quarter of 2026 was \$25.8 million, or \$0.20 per share, compared to \$18.0 million, or \$0.21 per share, for the same period in 2025.

ABOUT ZURA

Zura is a clinical-stage, multi-asset immunology company developing novel dual-pathway antibodies for autoimmune and inflammatory diseases with unmet need. Zura's pipeline includes product candidates designed to target key mechanisms of immune system imbalance, with the goal of improving efficacy, safety, and dosing convenience for patients.

Zura's lead product candidate, tibulizumab (ZB-106), is being evaluated in two Phase 2 clinical studies in adults: TibuSHIELD, a study in hidradenitis suppurativa (HS), and TibuSURE, a study in systemic sclerosis (SSc). A third Phase 2 clinical study in polymyalgia rheumatica (PMR), NEXUS-PMR, is planned to begin by year-end 2026. Additional product candidates torudokimab (ZB-880) and crebankitug (ZB-168) have completed Phase 1/1b studies and are being evaluated for their potential across a range of autoimmune and inflammatory conditions.

For more information, please visit www.zurabio.com.

FORWARD-LOOKING STATEMENTS

Any statements contained in this press release that do not describe historical facts may constitute "forward-looking statements" as that term is defined in the Private Securities Litigation Reform Act of 1995. These statements may be identified by words and phrases such as "anticipate," "believe," "continue," "could," "designed to," "expect," "goal," "intend," "may," "outlook," "plan," "potential," "should," "will," and similar expressions, and are based on Zura's current beliefs and expectations. These forward-looking statements include, but are not limited to, statements regarding the development and potential therapeutic benefits of Zura's product candidates; the initiation, timing, progress, design and results of Zura's current and future clinical trials, including the anticipated reporting of data therefrom; the planned expansion of Zura's product candidates into additional indications; Zura's evaluation of potential development approaches for its product candidates; the sufficiency of Zura's cash resources and projected cash runway; and other statements that are not historical facts. These statements involve risks and uncertainties that could cause actual results to differ materially from those reflected in such forward-looking statements. Risks and uncertainties that may cause actual results to differ materially



include, but are not limited to: uncertainties inherent in the development of therapeutic product candidates, such as the risk that one or more of Zura's current or future product candidates may not be successfully developed or commercialized; the risk of delay or cessation of any planned clinical trials of Zura's current or future product candidates; the risk that prior results, including signals of safety, activity or durability of effect observed in preclinical studies or earlier clinical trials, may not be replicated or may not continue in ongoing or future studies or clinical trials; the risk that modeling data indicating therapeutic potential, or clinical evidence from other drug candidates, may not be predictive of results in Zura's current or future clinical trials; the risk that Zura's product candidates or procedures in connection with their administration may not have the safety or efficacy profiles anticipated; risks related to the accuracy of Zura's estimates of expenses, capital requirements and needs for additional financing; changes in expected or existing competition; changes in the regulatory environment; uncertainties related to the timing and outcome of the regulatory approval process; unexpected litigation or other disputes; the impact of macroeconomic conditions on Zura's business, clinical trials and financial position; and other risks and uncertainties described in Zura's Annual Report on Form 10-K for the year ended December 31, 2025, and other filings with the Securities and Exchange Commission. Any forward-looking statements speak only as of the date of this press release and are based on information available to Zura as of the date hereof. Zura assumes no obligation to, and does not intend to, update any forward-looking statements, whether as a result of new information, future events or otherwise, except as required by law.

CONTACTS

Investor and media inquiries:
ir@zurabio.com



ZURA BIO LIMITED
CONSOLIDATED BALANCE SHEETS
(In thousands, except share data)

	September 30, 2026	December 31, 2025
	(unaudited)	
Assets		
Current assets		
Cash and cash equivalents	\$ 187,652	\$ 109,407
Prepaid expenses and other current assets	3,940	2,903
Total current assets	191,592	112,310
Property and equipment, net	153	126
Other assets	2,884	1,512
Total assets	\$ 194,629	\$ 113,948
Liabilities and Shareholders' Equity		
Current liabilities		
Accounts payable and accrued expenses	\$ 16,969	\$ 12,410
Total current liabilities	16,969	12,410
Total liabilities	16,969	12,410
Shareholders' Equity		
Class A Ordinary Shares, \$0.0001 par value; 300,000,000 shares authorized as of September 30, 2026 and December 31, 2025; 96,306,411 and 73,680,710 shares issued and outstanding as of September 30, 2026 and December 31, 2025, respectively	10	7
Additional paid-in capital	478,549	326,078
Accumulated deficit	(300,899)	(224,547)
Total shareholders' equity	177,660	101,538
Total liabilities and shareholders' equity	\$ 194,629	\$ 113,948



ZURA BIO LIMITED
CONSOLIDATED STATEMENTS OF OPERATIONS
(Unaudited)
(In thousands, except share and per share data)

	For the Three Months Ended September 30,		For the Nine Months Ended September 30,	
	2026	2025	2026	2025
Operating expenses:				
Research and development	\$ 19,491	\$ 11,948	\$ 54,943	\$ 31,126
General and administrative	9,282	7,571	28,650	25,709
Total operating expenses	28,773	19,519	83,593	56,835
Loss from operations	(28,773)	(19,519)	(83,593)	(56,835)
Other (income), net				
Interest income	(1,759)	(1,526)	(4,998)	(5,060)
Other expense (income), net	32	47	(941)	(300)
Total other (income), net	(1,727)	(1,479)	(5,939)	(5,360)
Loss before income taxes	(27,046)	(18,040)	(77,654)	(51,475)
Income tax benefit	(1,221)	—	(1,302)	—
Net loss	\$ (25,825)	\$ (18,040)	\$ (76,352)	\$ (51,475)
Accretion of redeemable noncontrolling interest to redemption value	—	(2,828)	—	(2,828)
Adjustment of redeemable noncontrolling interest	—	831	—	831
Net loss attributable to Class A Ordinary Shareholders of Zura	\$ (25,825)	\$ (20,037)	\$ (76,352)	\$ (53,472)
Net loss per share attributable to Class A Ordinary Shareholders of Zura, basic and diluted	\$ (0.20)	\$ (0.21)	\$ (0.62)	\$ (0.57)
Weighted-average Class A Ordinary Shares used in computing net loss per share attributable to Class A Ordinary Shareholders of Zura, basic and diluted	127,712,533	94,564,632	122,303,170	93,941,675



zurabio

Corporate Overview

October 2026

DEVELOPING NOVEL MEDICINES FOR SERIOUS IMMUNE SYSTEM DISORDERS



Forward-Looking Statements Disclaimer

This presentation contains “forward-looking statements” within the meaning of the “safe harbor” provisions of the Private Securities Litigation Reform Act of 1995. Words such as “expect,” “estimate,” “project,” “budget,” “forecast,” “anticipate,” “intend,” “plan,” “may,” “will,” “could,” “should,” “believe,” “predict,” “potential,” “continue,” “strategy,” “future,” “opportunity,” “would,” “seem,” “seek,” “outlook,” “goal,” “mission,” and similar expressions are intended to identify such forward-looking statements. Forward-looking statements are predictions, projections and other statements about future events that are based on current expectations, estimates, and assumptions and, as a result, are subject to risks and uncertainties that could cause actual results to differ materially from the expected results. These statements are based on various assumptions, whether or not identified in this presentation. These forward-looking statements may include, without limitation: Zura Bio’s clinical development plans; the design, initiation, conduct, enrollment and timing of its clinical trials; expectations regarding the timing of key milestones and anticipated data readouts; expectations regarding clinical programs, including the safety, efficacy, and therapeutic potential of Zura Bio’s product candidates; expectations regarding the commercial potential of Zura Bio’s product candidates; expectations regarding data readouts from third parties, including regulatory filings and approval status; expectations regarding market opportunities, including estimated total addressable markets, competitive landscape, addressable patient populations, or potential clinical differentiation; Zura Bio’s cash resources and projected cash runway; Zura Bio’s business strategies and objectives; and any other statements that are not historical facts.

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 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 uncertainties, which include, but are not limited to: Zura Bio’s expectations regarding its product candidates and their related benefits, and Zura Bio’s beliefs regarding competing product candidates or approved products, may not be achieved; Zura Bio’s vision and strategy may not be successful; 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; Zura Bio’s expectations regarding the potential general acceptability and maintenance of its 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 expectations with respect to its future operating expenses, capital 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 Bio has incurred significant 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 substantial additional capital to 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 development programs or future commercialization efforts; Zura Bio may be unable to renew existing contracts, or enter into new contracts or may experience disputes or other challenges with respect to our vendors or other third parties; Zura Bio relies on third-party contract development manufacturing organizations for the manufacture of clinical materials; Zura Bio relies on contract research organizations, its clinical trial sites, and other third parties to conduct its preclinical studies and clinical trials; Zura Bio may be unable to 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 to general economic and geopolitical conditions; Zura Bio may be unable to effectively achieve or manage growth; Zura Bio faces competitive pressures from other companies worldwide; Zura Bio may be unable to adequately protect its intellectual property rights; and other factors set forth in documents filed, or to be filed by Zura Bio, with the Securities and Exchange Commission (SEC), including the risks and uncertainties described in the “Risk Factors” section of Zura Bio’s Annual Report on Form 10-K for the year ended December 31, 2025, and other filings with the SEC. Zura Bio cautions that the foregoing list of factors is not exclusive or exhaustive and cautions readers not to place undue reliance upon any forward-looking statements, which speak only as of the date made. Zura Bio does not undertake or accept any obligation to update any forward-looking statements, whether as a result of new information, future developments, or otherwise, except as required by law. Zura Bio gives no assurance that the expectations expressed herein will be achieved or that deviations from such expectations will not be material.

This presentation discusses product candidates that are under clinical investigation and have not been approved for marketing by the U.S. Food and Drug Administration or any other regulatory authority. No representation is made as to the safety, efficacy, or likelihood of regulatory approval of these product candidates for the uses under investigation. Comparisons across clinical trials or product candidates should be interpreted with caution, as differences in study design, inclusion/exclusion criteria, patient populations, endpoints, dosing regimens, and other variables may limit interpretability. Statements in this presentation regarding clinical trials involving product candidates originating from third parties (including Eli Lilly, Pfizer and Novartis) have not been reviewed, verified, or endorsed by such parties.

Advancing tibulizumab: a potential first- and only-in-class bispecific antibody

Tibulizumab, a potential first- and only-in-class bispecific antibody inhibiting IL-17 and BAFF in development

- IL-17 and BAFF among the most clinically validated pathways in immunologic disorders^{1,2}
- Orthogonal nature of IL-17 and BAFF offers potential to modulate non-redundant pathways
- Potent engagement of targets and low immunogenicity observed in Phase 1

Pipeline in a product potential: three biologically de-risked and independent shots on goal

- Hidradenitis suppurativa (HS), systemic sclerosis (SSc) and polymyalgia rheumatica (PMR) span distinct biology
- Yet each disease leverages existing clinical validation of the IL-17 (HS^{3,4} and PMR⁵) or BAFF arm (SSc⁶) with strong scientific support for the orthogonal pathway^{7,8,9}

Zura Bio is financed beyond anticipated key near-term value inflection points

- Topline data expected from HS and SSc Phase 2 studies in Q4 2026 and H1 2027, respectively
- Three-asset pipeline with optionality across multiple immune-mediated indications
- Cash runway through at least year-end 2028



"Biologically de-risked" refers to clinical support for the relevant mechanism(s) of action and is not intended to imply regulatory or clinical success
Sources: 1. Cosentyx®, Taltz®, Bimzelx® Prescribing Information; 2. Benlysta® Prescribing Information; 3. Kimball et al. *Lancet*. 2024; 4. Kimball et al. *Lancet*. 2023; 5. Stone et al. *N Engl J Med*. 2026 (REPLENISH); 6. Gordon et al. *Arthritis Rheumatol*. 2018; 7. Sabat et al. *J Allergy Clin Immunol*. 2023; 8. Lowe et al. *JCI Insight*. 2024; 9. van der Geest et al. *Rheumatology (Oxford)*. 2015; 10. Zura Bio filings.

Addressing the distinct biology of immune-mediated diseases

Immune-mediated diseases are often driven by **multiple, intersecting immune pathways**

Targeting more than one pathway is a rational approach to address **low or inadequate response rates** in complex immune-mediated diseases

Early clinical readouts across a range of immune-mediated indications have already demonstrated **the potential of bispecific antibodies** to break through efficacy ceilings

Untapped market potential across disease areas of interest

TODAY:

Biologic penetration in HS, SSs, and PMR remains below average¹ because current therapies only partially address the multi-pathway biology driving these diseases²⁻⁹

~\$5B
2026

\$15B+

Projected
Mid-2030s

2030s:

Therapies designed to address the multi-pathway biology of each disorder have the potential to break the response-rate ceiling – expanding the eligible patient pool, moving into earlier lines of therapy, and extending duration of use

SIGNIFICANT GROWTH:

Precedent in adjacent immunology indications shows each step-change in efficacy – as in psoriasis and RA – drove a step-change in advanced-therapy utilization and market size¹⁰⁻¹³

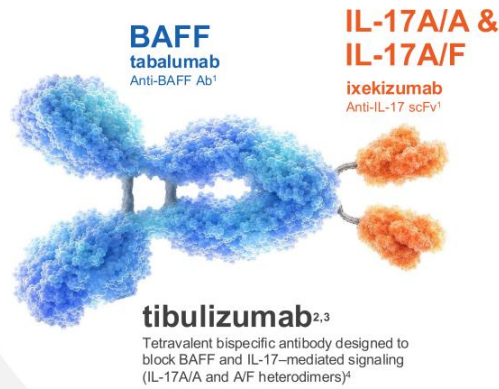
Three shots on goal – biologically de-risked, independent, and fully funded through topline data readouts



Tibulizumab: Potential first- and only-in-class therapy

Tibulizumab is an investigational agent. Its efficacy and safety have not been established or approved by the FDA or any other regulatory agency worldwide.

Tibulizumab is a four-headed hammer designed to inhibit the BAFF and IL-17 pathways



Addresses disease complexity

Aims to target two orthogonal immune pathways implicated in chronic inflammation and autoimmunity

Clinically grounded design

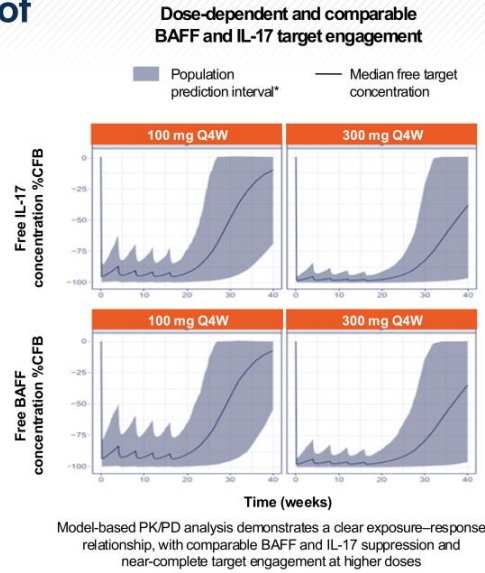
Fusion of a BAFF-binding antibody (tabalumab) with the IL-17-binding scFv from ixekizumab (ixekizumab marketed as Taltz®, ~\$3.6B in global 2025 sales, as reported)^{1,4}

Single-entity advantage

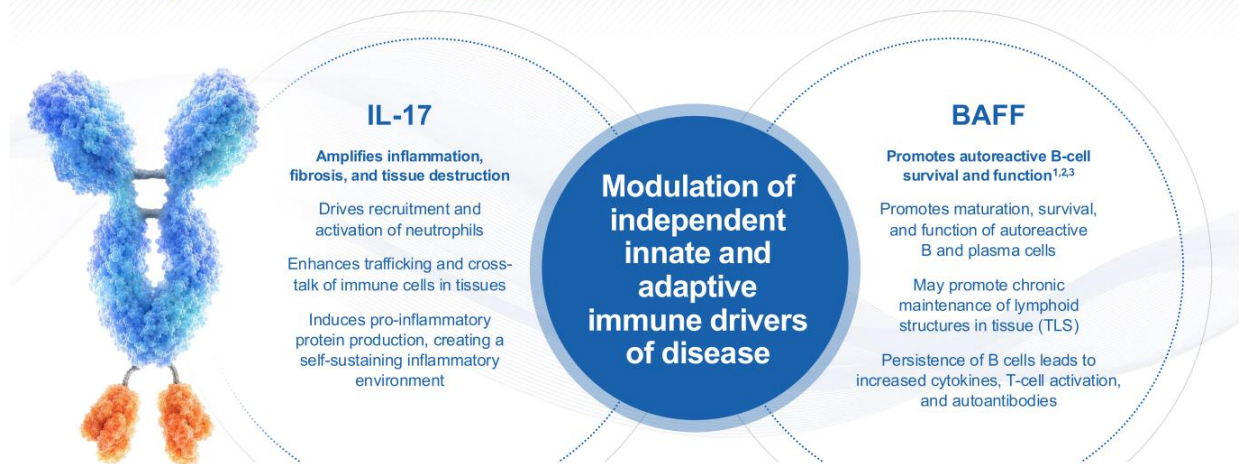
Enables dual-pathway modulation without multi-drug combination complexity

Phase 1/1b data support advancement of tibulizumab into Phase 2 development

- 78 participants dosed across Phase 1/1b studies
- >98% median trough target engagement for both IL-17 and BAFF in peripheral blood at 300 mg Q4W
- Mean terminal half-life ($t_{1/2}$): 27 days, supporting once-monthly dosing
- Pharmacodynamic activity observed, including B-cell and CRP reductions
- Low incidence of treatment-emergent ADAs in multiple-dose cohorts
- Safety profile consistent with published IL-17 and BAFF pathway experience, with no new or unexpected safety signals to date



Broad potential in heterogeneous immune disorders



First and only bispecific antibody in development designed to target the orthogonal IL-17 and B-cell pathways

Ixekizumab contributes a high-efficacy component for IL-17 pathway inhibition

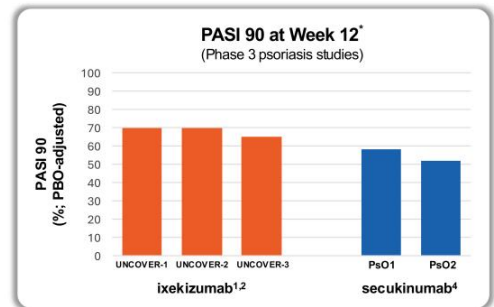
Multiple Phase 3 programs completed

High efficacy demonstrated

Broad IL-17 pathway inhibition

Extensive clinical and real-world experience

- Consistent high efficacy: Demonstrated across multiple inflammatory diseases in large Phase 3 programs*^{1,2}
- Validated IL-17 pathway inhibition: Neutralization of IL-17A/A and IL-17A/F produces robust clinical responses with a well-characterized safety profile^{1,2,3}
- Extensive clinical safety experience: Supported by large global clinical development programs and post-marketing datasets across dermatologic and rheumatologic indications*³
- Low incidence of candidiasis relative to IL-17F/F antagonists



Ixekizumab establishes a well-characterized clinical benchmark for IL-17 pathway inhibition



* PASI 90 values represent placebo-adjusted response rates at Week 12 from independent Phase 3 psoriasis trials. Studies were not designed for head-to-head comparison; trial designs, patient populations, dosing regimens, and placebo response rates differed. Clinical efficacy data shown are limited to psoriasis studies. Efficacy and durability may vary by indication.
Sources: 1. Griffiths et al. *Lancet*. 2015; 2. Gordon et al. *N Engl J Med*. 2015; 3. Eli Lilly and Company and Novartis. Public disclosures and prescribing information; 4. Langley et al. *N Engl J Med*. 2014.

Tibulizumab potentially inhibits IL-17A/A and IL-17A/F: the strongest drivers of IL-17 signaling

Agent	Target description			
		IL-17A/A (K _D)	IL-17A/F (K _D)	IL-17F/F (K _D)
Tibulizumab ¹	Anti-BAFF & IL-17 bispecific mAb	~14 pM	~90 pM	no binding
Secukinumab (Cosentyx®) ²	Anti-IL-17A mAb	~60–90 pM	~2400 pM	no binding
Bimekizumab (Bimzelx®) ³	Anti-IL-17A/F mAb	~3.2 pM	~26 pM	~23 pM

Hidradenitis Suppurativa (HS)

HS is a chronic, progressive inflammatory condition with high symptom burden

Patients experience pain in sensitive, high-friction areas

Painful nodules and abscesses make walking, sitting, and arm movement difficult, limiting daily comfort¹

Patients withdraw from social interactions due to visible symptoms

Embarrassment drives social withdrawal, relationship strain, absenteeism, and mental health consequences²

Patients struggle with persistent drainage and odor from lesions

Managing chronic drainage and odor becomes burdensome, leading to avoidance of exposure or hiding¹

Patients experience prolonged delays in diagnosis and treatment

Delayed diagnosis leads to continued symptom burden and progression to more severe disease³

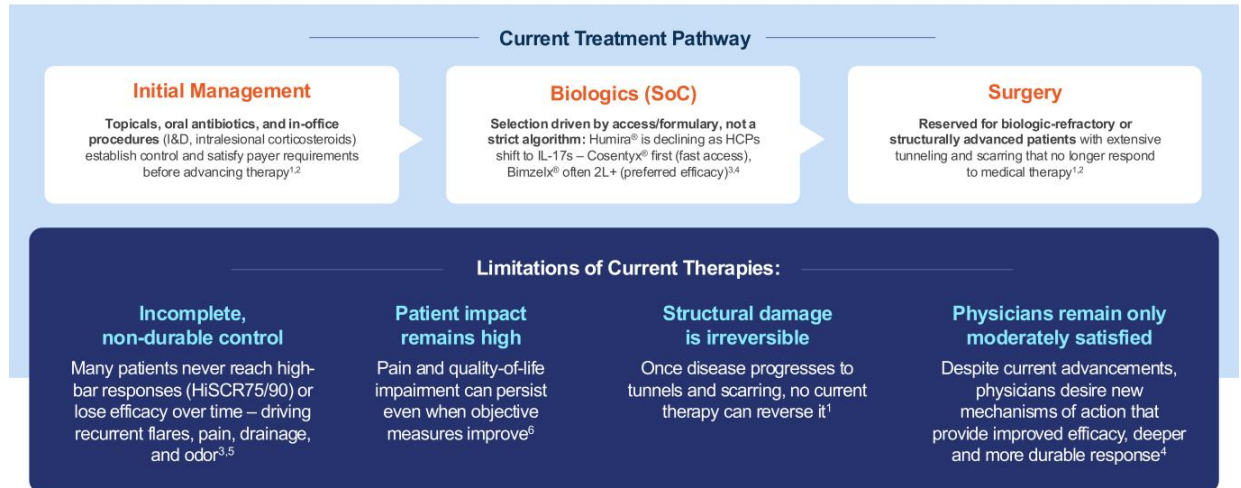
Distinctive for structural skin damage driven by tunneling and scarring¹

Lesions commonly occur in intertriginous areas (skin folds)¹

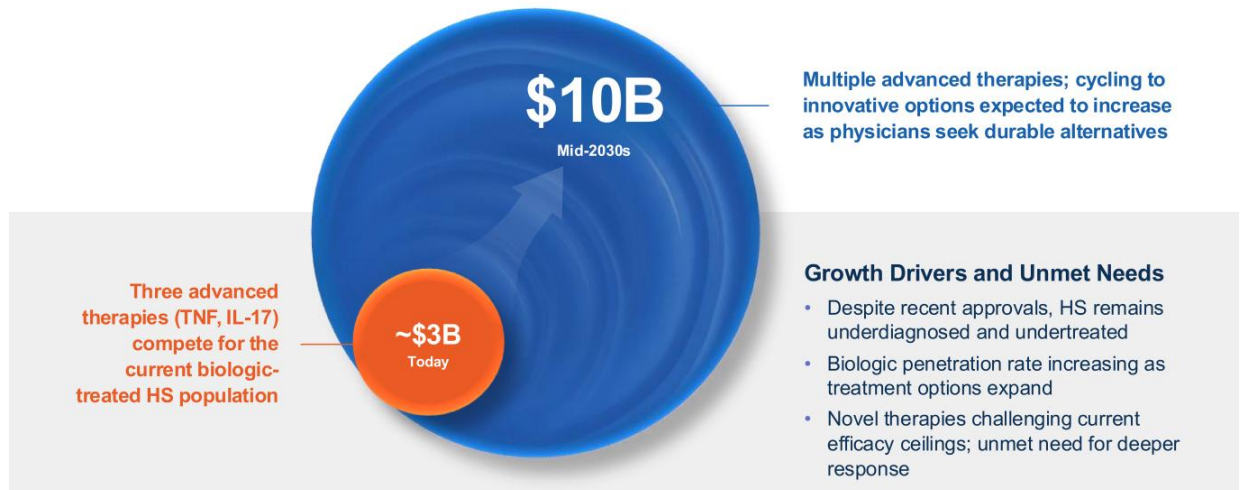


Sources: 1. Sabat et al. *Lancet*. 2025 (Seminar); 2. Kimball et al. *J Am Acad Dermatol*. 2024; 3. Kokolakis et al. *Dermatology*. 2020.

How patients are treated today – and the limitations of existing therapies

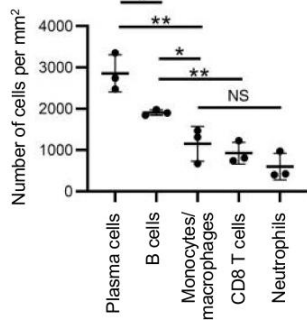


Hidradenitis suppurativa: large, underserved market with high unmet need

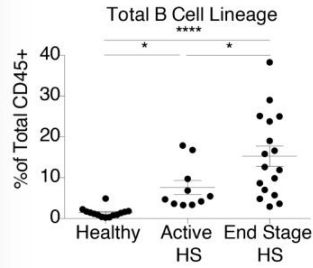


Unlike healthy skin, B cells infiltrate and chronically persist in HS lesions, driving a local amplified and targeted immune response

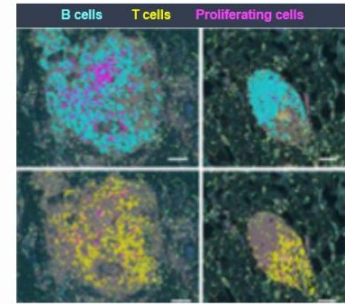
B cells and plasma cells make up the bulk of HS cellular infiltrate¹



B cells expand with disease progression²

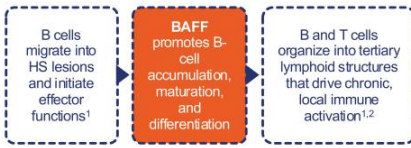


In severe disease, B and T cells organize and persist in tertiary lymphoid structures (TLS)^{3,4}



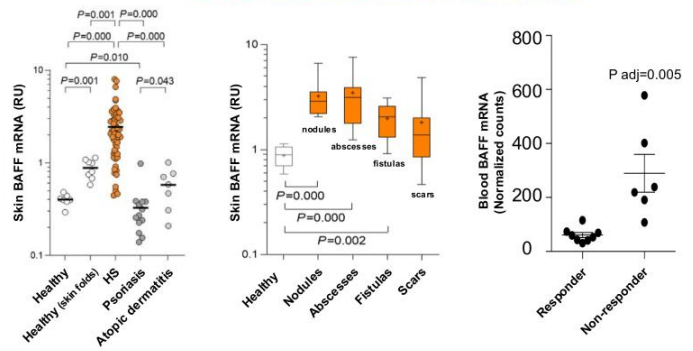
BAFF is elevated across HS lesion types and predicts treatment resistance

Elevated BAFF can retain B cells in the skin



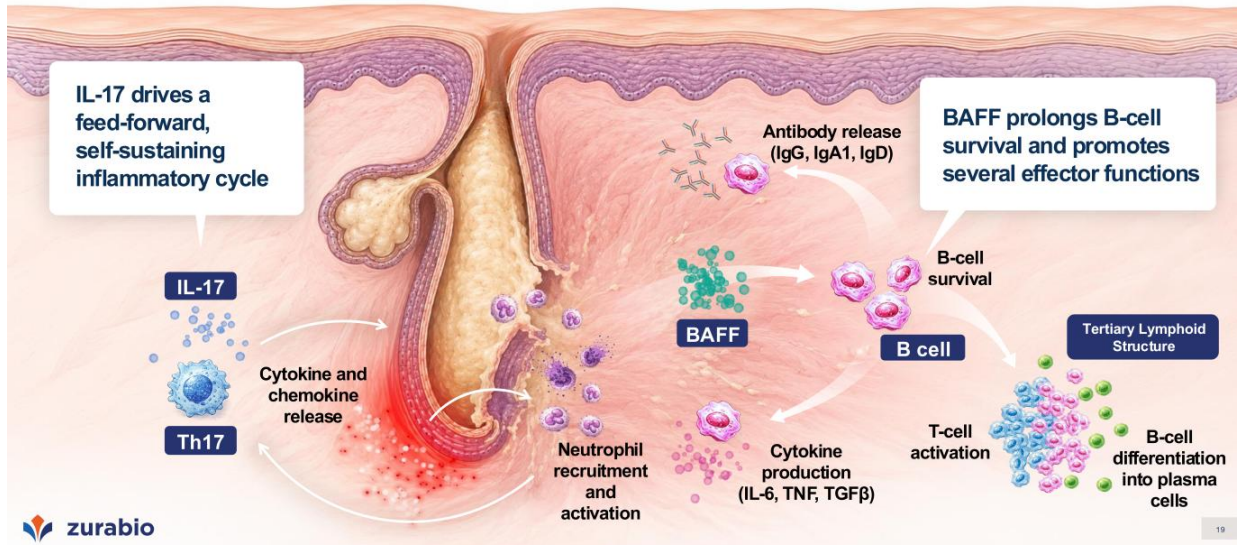
- BAFF is absent in healthy skin and in psoriasis and dermatitis lesions, and B cells transiently migrate in and out of skin^{3,4}
- BAFF is significantly elevated in all HS lesion types, and correlates with increases in B cells^{3,4}
- Patients who have higher baseline levels of BAFF and B-cell counts are less likely to respond to adalimumab^{4,5}

Elevated BAFF is a unique feature of HS lesions, which predicts TNFi non-response

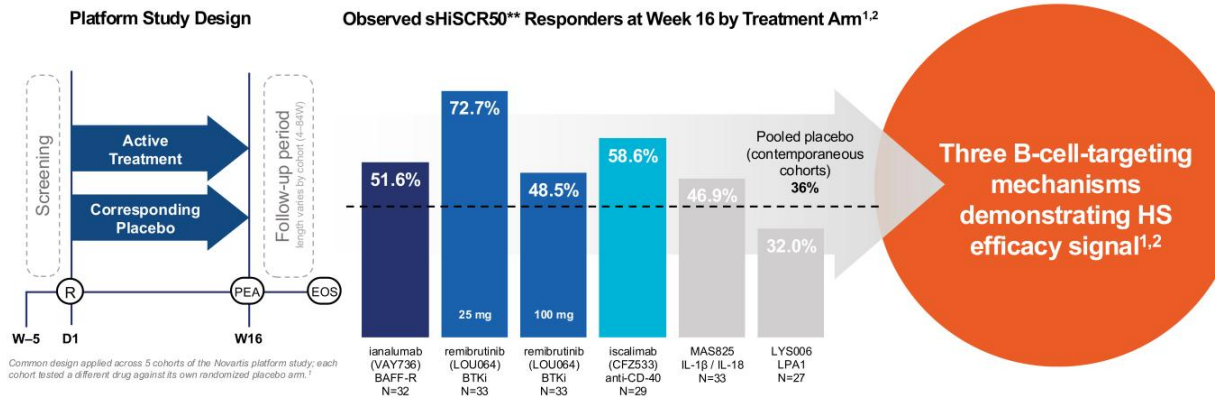


In acute and chronic HS lesions, BAFF expression tracks with B-cell accumulation, supporting BAFF as a key player in B-cell-mediated pathology

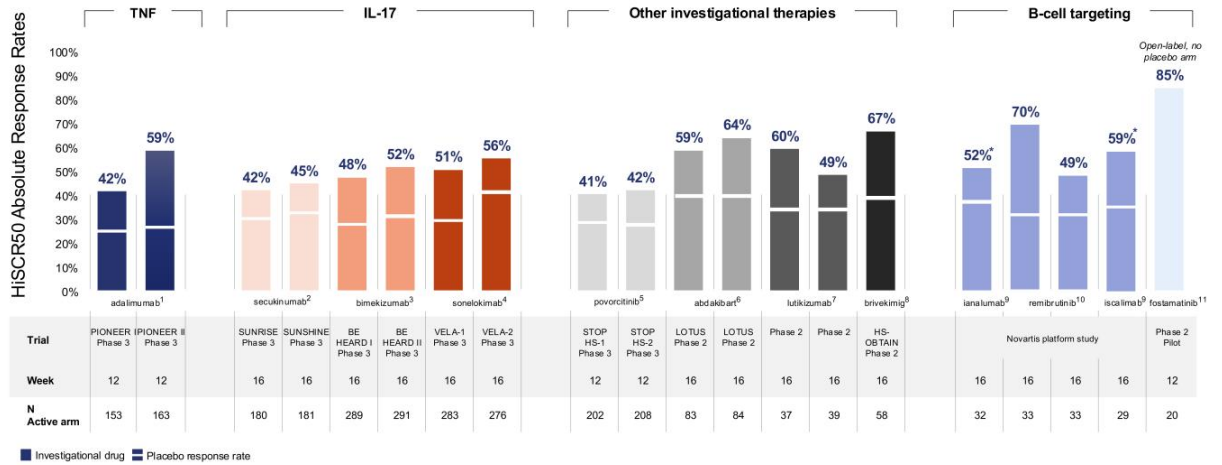
B cells are believed to drive HS pathology via multiple mechanisms



Recent clinical data support the hypothesis that B cells are key drivers of disease rather than bystanders in HS*



B-cell-directed monotherapy has demonstrated absolute response rates in line with other approved and late-stage assets in HS



* shHSCR50: modified HSCR50 omitting the "no increase in abscess count" criterion; For all studies, HSCR50 was the primary endpoint except LOTUS (abdoakibart, HSCR75) and the Novartis platform study (iansalumab, remibrutinib, iscalimab; shHSCR50). Note: Data are derived from separate clinical trials with differences in design and patient populations. No head-to-head clinical trials have been conducted to date; cross-trial comparison limitations exist.



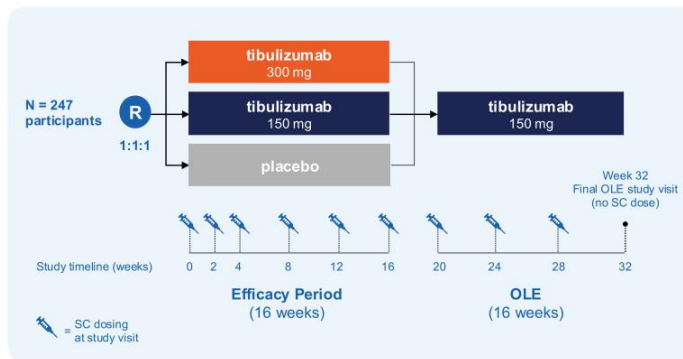
Sources: 1. Kimball et al. *N Engl J Med*. 2016; 2. Kimball et al. *Lancet*. 2023; 3. Kimball et al. *Lancet*. 2024; 4. Porter et al. *SHSA Annual Meeting*. 2025; 5. Porter et al. *Nat Med*. 2026; 6. Avsio Therapeutics. *LOTUS Topline Presentation*. 2026; 7. Kimball et al. *JAMA Dermatol*. 2026; 8. Sanofi. *HS-OBTAIN Phase 2a Readout*. 2025; 9. *ClinicalTrials.gov*. NCT03827798. 2026; 10. Kimball et al. *AAD Annual Meeting*. 2024; 11. Jepsen et al. *J Am Acad Dermatol*. 2023.

Orthogonal nature of IL-17 and BAFF pathways increases confidence in potential benefit over IL-17 inhibition alone



De-risked, biologically relevant approach to potentially break through efficacy ceilings in HS

Phase 2 study ongoing in moderate-to-severe HS



* Includes secondary and exploratory endpoints



Key Inclusion / Exclusion Criteria

- Adults with moderate-to-severe HS, defined as:
 - Hurley Stage II/III (up to 40% Stage III allowed)
 - Total abscess and inflammatory nodule (AN) count ≥ 5
- Up to 30% of participants may have prior TNF- α inhibitor exposure
- Prior IL-17 use excluded
- Stable GLP-1 use allowed (at least 6 months)
- Stable antibiotic use allowed (at least 4 weeks)
- Additional eligibility criteria per protocol

Efficacy Endpoints

Primary endpoint

- Percent change from baseline in AN count at Week 16

Additional endpoints*

- HiSCR50 and HiSCR75
- Draining tunnel count
- IHS4
- DLQI
- Skin pain NRS
- PK/PD assessments

Systemic Sclerosis (SSc)

Systemic sclerosis is a progressive, multisystem autoimmune disease with limited therapeutic options*

approximately

300,000

people are estimated to be living with SSc across major markets (US, EU5, Japan)¹



No therapies are approved that comprehensively address the multisystem pathology of SSc²

Characterized by
Chronic Inflammation and
Fibrosis Across Organs



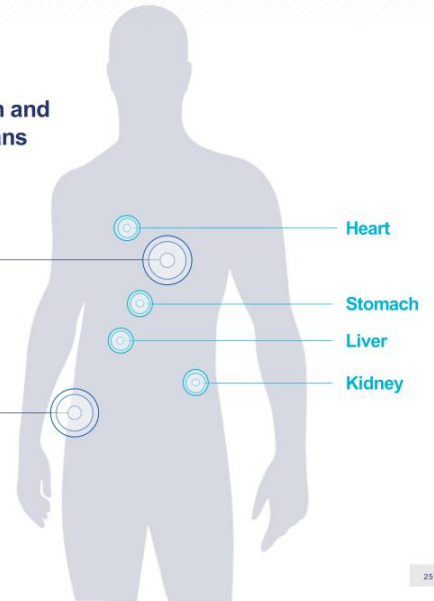
Lungs

SSc-ILD is a major cause of morbidity and mortality in SSc. Two disease-modifying treatments are approved for SSc-ILD²



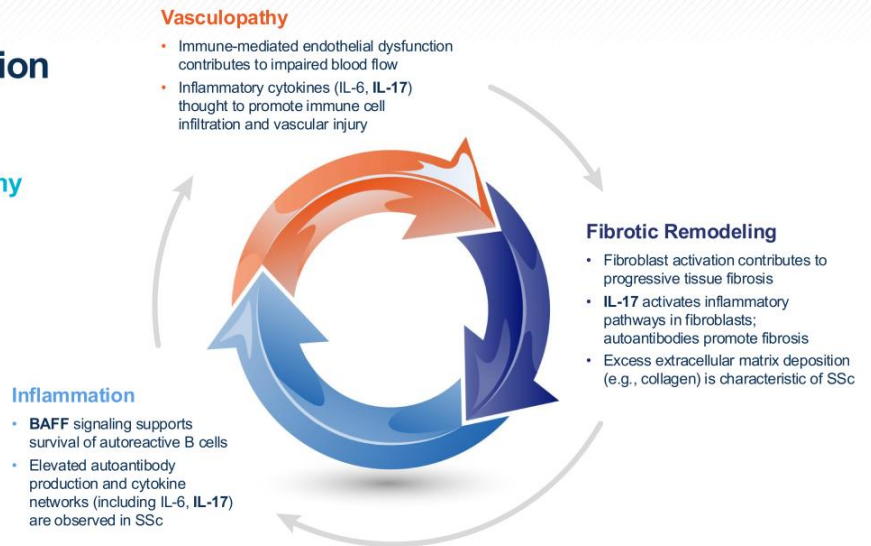
Skin

Skin fibrosis contributes to functional impairment, disability, and reduced quality of life

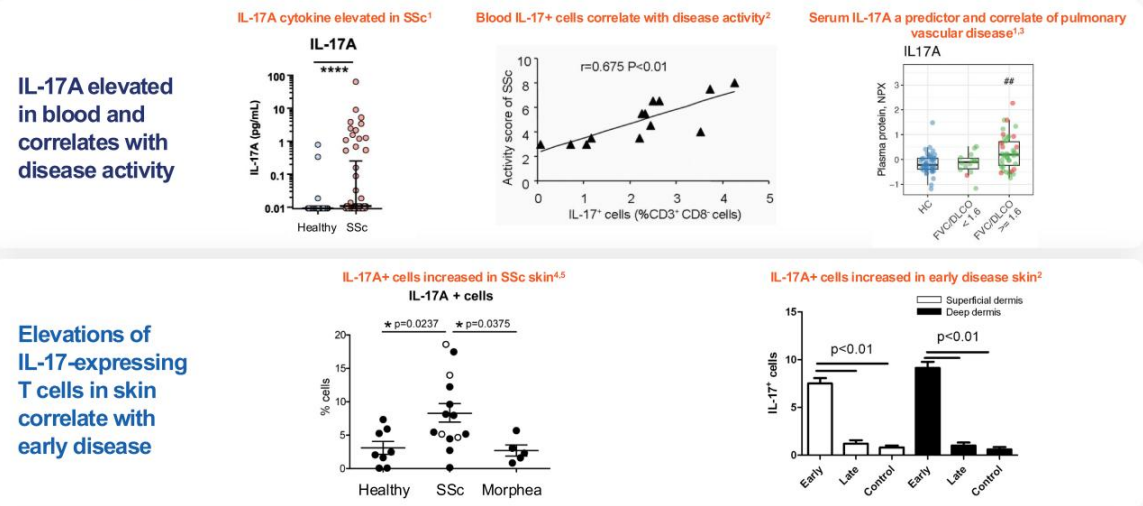


Why SSc requires multi-pathway immune modulation

BAFF- and IL-17-driven inflammation may contribute to vasculopathy and fibrosis, supporting evaluation of multi-pathway therapeutic strategies in SSc

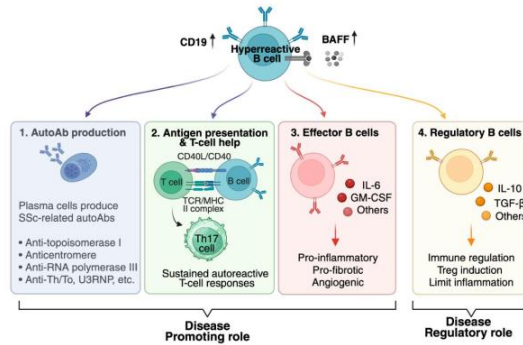


IL-17A is increased in SSc blood and skin and is linked to active and early disease



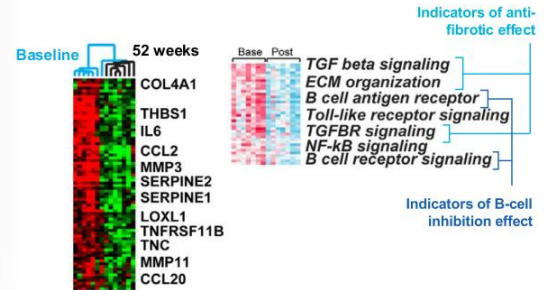
BAFF inhibition reduces profibrotic and B-cell signaling genes in SSc skin^{1,2}

Hypothesized disease-promoting roles of B cells³



Belimumab modulated gene profiles in post-treatment SSc skin¹

RNAseq analysis of skin from patients who demonstrated a 20% reduction in mRSS



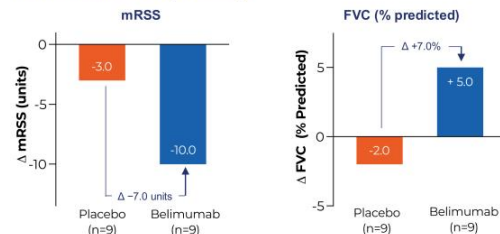
IL-17 and BAFF blockade have each shown improvement in skin and lung outcomes in SSc

BAFF ANTAGONIST

Belimumab¹

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

Phase 2 belimumab IIT (52 weeks)¹

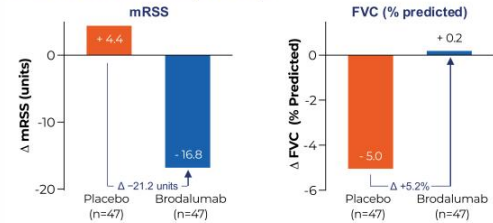


IL-17 RECEPTOR ANTAGONIST

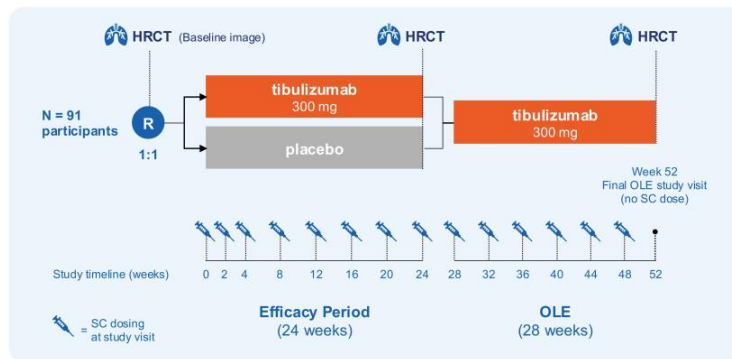
Brodalumab²

- Met the primary endpoint of reduced mRSS at Week 24 for skin involvement, and demonstrated improvement in FVC as a secondary endpoint reflecting lung function
- Also showed therapeutic effects on lung/respiratory functions, digital ulcers, symptoms of gastroesophageal reflux disease, and QOL without noteworthy safety concerns

Phase 3 brodalumab trial (24 weeks)²



Phase 2 study ongoing in SSc; enrollment completed



Key Inclusion Criteria

- Adults (18–75 years) with early diffuse cutaneous SSc, enriched for SSc-ILD
- Stable background immunosuppressive or antifibrotic therapy allowed
- Disease duration ≤ 7 years
- mRSS 15–45 at screening
- Additional eligibility criteria per protocol

Endpoints and Stratification

Primary endpoint

- Change from baseline in mRSS at Week 24

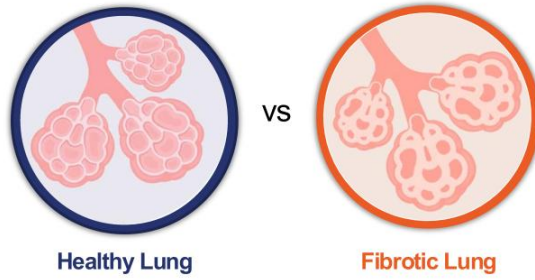
Additional endpoints*

- qHRCT lung imaging
- FVC
- HAQ-DI
- Revised CRIS

Stratification factors

- Presence of ILD (yes/no)
- Duration of disease ($\geq$ or < 2 years)
- Anti-RNA polymerase III (+/-)

In Phase 2, lung involvement using qHRCT is a key secondary endpoint

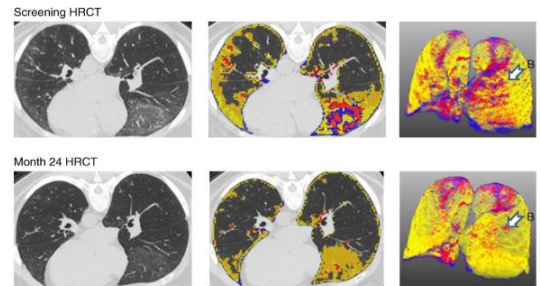


ILD includes various pulmonary disorders marked by inflammation and progressive lung fibrosis, resulting in restrictive lung function and impaired gas exchange.

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

QILD by HRCT provides a sensitive measure of lung involvement, detecting changes as small as 2%.¹

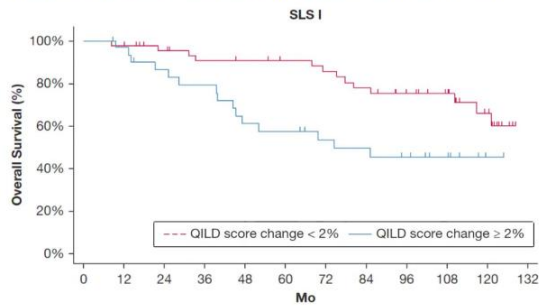
Example of improvement after 24 months of MMF in total lung involvement¹



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

QILD predicts mortality in SSc and closely correlates with FVC – differences of ~2% are clinically meaningful

Two large longitudinal SSc studies (SLS I and SLS II) showed that $\geq 2\%$ increase in QILD associated with increased risk of death¹



- QILD is highly correlated with FVC^{2,3}
- Clinically meaningful FVC changes were associated with 2% QILD change²

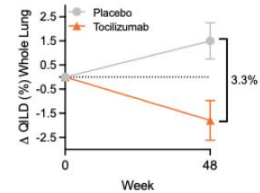
Tocilizumab Phase 3 in SSc-ILD

Approved for SSc-ILD based on FVC (% predicted)

- -6.4% (PBO) vs. 0.1% (tocilizumab) over 48 weeks⁴

Patients were also evaluated by HRCT for QILD:⁵

- Greater QILD was associated with worse FVC at Baseline
- Significant improvement from Baseline (-1.8%) with tocilizumab; treatment difference of 3.3%



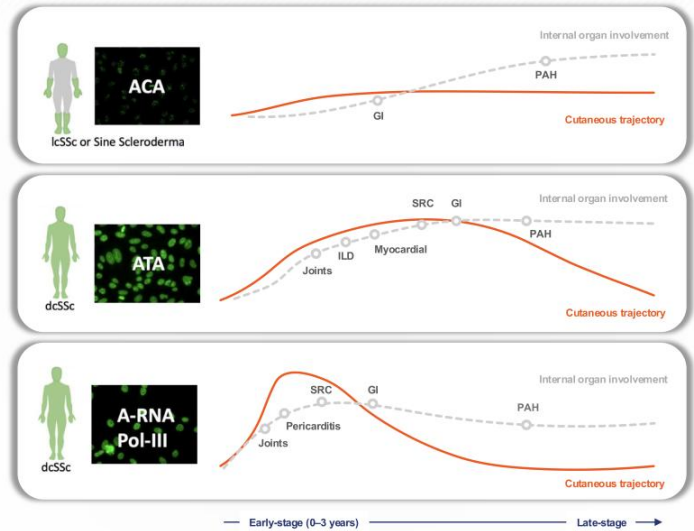
Published SSc disease trajectories validate our Phase 2 enrollment strategy

What the data show

- The type of autoantibody drives disease progression, with cutaneous trajectory peaking early in certain patients
- ATA (anti-topoisomerase antibody) and A-RNA Pol-III (anti-RNA polymerase 3 antibody) endotypes have worst skin disease

Why our design fits

- Stratification factors designed to reduce confounders
- Targets the active window: mRSS 15–45, ≤ 7 years
- Improves probability of success for skin primary endpoint
 - Excludes anti-centromere antibody (ACA) positive participants
 - Allows all anti-topoisomerase participants
 - Includes all participants with anti-RNA Pol-III up to 2 years' duration, after which severe disease is required



Polymyalgia Rheumatica (PMR)

PMR: inflammatory disease that leads to reduced mobility, loss of independence, and significant quality-of-life burden

Chronic inflammatory disease

- Causes bilateral aching and stiffness in the shoulders, hip girdle, neck, and torso^{1,2}
- Systemic inflammation is present with increased erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) levels^{1,2}
- Driven by innate and adaptive immune activation^{3,4}

Signs and symptoms

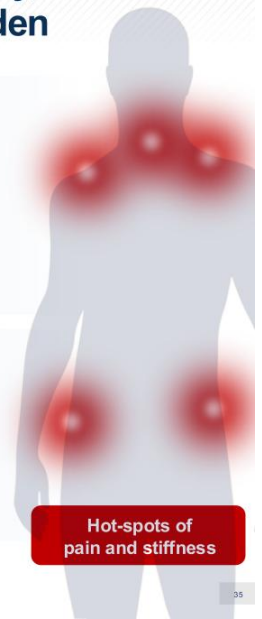
- Morning stiffness/stiffness with inactivity is a hallmark^{1,2}
- Proximal stiffness makes daily activities difficult (e.g., dressing, overhead reaching)²
- Nonspecific systemic signs: malaise, fatigue, low-grade fever, weight loss^{1,2}

Demographics and risk factors

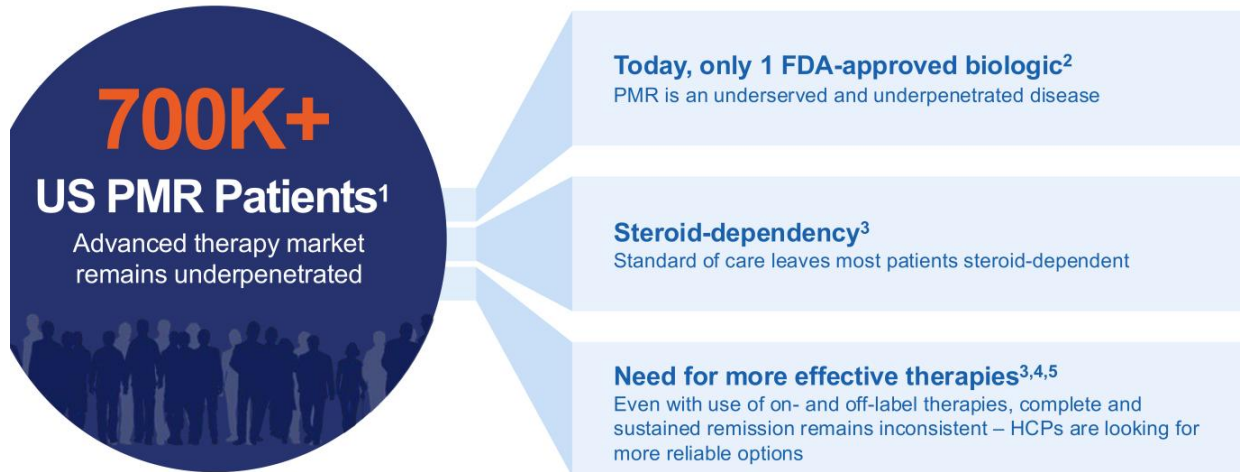
- Almost exclusively patients over 50; peak onset at ages 70–80^{2,5}
- Women are more likely to develop PMR than men⁵
- Highly comorbid population⁶

Steroid dependency, relapse-prone

- Rapid symptom improvement on low-to-moderate corticosteroid doses^{2,7}
- 40–60% of patients relapse during steroid tapering^{4,7}
- Many patients remain on steroids for 5+ years^{4,7}



PMR represents a 700K+ US patient population with significant headroom for advanced therapy adoption



Current reliance on steroids provides short-term relief but leads to long-term dependence, relapse, and toxicity



Steroid-associated toxicities are common (e.g., insomnia, weight gain, hyperglycemia, osteoporosis, and infections) and can be problematic in the PMR age demographic due to high rates of osteoporosis, hypertension, or T2D

Over
50%¹

Relapse on steroid taper

Patients are forced back to higher doses of glucocorticoids with no clear end in sight

Approx.
6 Yrs²

Median time to permanent glucocorticoid discontinuation

Due to high relapse rates during taper, many patients remain on steroids to manage PMR even after 5 years³

“ I’m on prednisone, and it does awful things to you. The side effects are really nasty. If there was anything I could take or do to get rid of those side effects, I would do it in a minute. But at this point, apparently, there isn’t anything that my physician knows about.”

- PMR Patient⁴

“ The plan to reduce was difficult. I started reducing by like half a mg a week. The reduction was difficult because the pain kept returning. I couldn’t get away from the pain.”

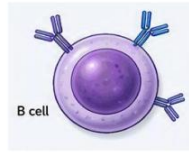
- PMR Patient⁴



Sources: 1. Spiera et al. *N Engl J Med*. 2023; 2. Devauchelle-Pensec et al. *Ann Rheum Dis*. 2018; 3. Camellino et al. *Front Med (Lausanne)*. 2022; 4. Zurabio. Data on file. 2023.

Targeting BAFF and IL-17 may help address both immune dysregulation and inflammation in PMR

B-cell homeostasis and trafficking are dysregulated^{1,2}

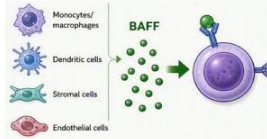


Circulating B cells:

Traffic out of blood during active disease and return in remission^{1,2}

May co-stimulate T cells or produce cytokines (including TNF and IL-6) in sites of inflammation^{1,2}

BAFF levels correlate with disease activity^{1,2}



Serum BAFF is:

Elevated in newly diagnosed PMR^{1,2}

Decreased during remission^{1,2}

Highly correlated with ESR, CRP, and IL-6²

↓ may lead to

↑ B-cell survival

↑ B-cell activation

↑ Cytokine production

↑ Interactions with T cells

IL-17 may amplify local and systemic inflammation^{3,4}



IL-17 and IL-17-producing cells are:

Elevated in circulation^{3,4}

Present in synovial fluid^{3,4}

↓ may lead to

↑ IL-6, IL-8, TNF, and other inflammatory mediators

↑ Number and activation of myeloid cells

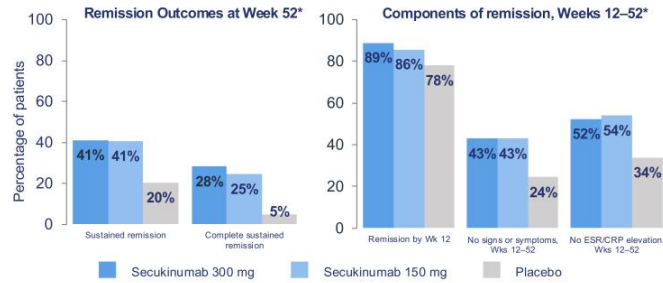
IL-17 and B-cell-targeted therapies have shown positive glucocorticoid-sparing effects in PMR

IL-17A inhibitor – relapsing PMR (Phase 3)

Secukinumab¹

- Met the primary endpoint of sustained remission at Week 52 and all key secondary endpoints
- Demonstrated a meaningful glucocorticoid-sparing effect while maintaining disease control
- Secukinumab has been filed in the US and EU for approval in PMR

Phase 3 REPLENISH trial (52 weeks, N = 381, 1:1:1 ratio)

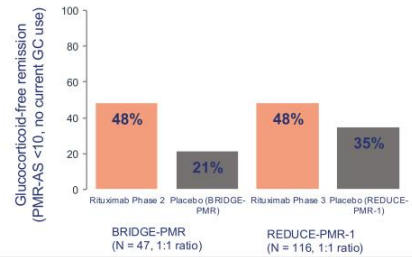


Anti-CD20 – new/relapsing PMR (Phase 2) and new-only PMR (Phase 3)

Rituximab

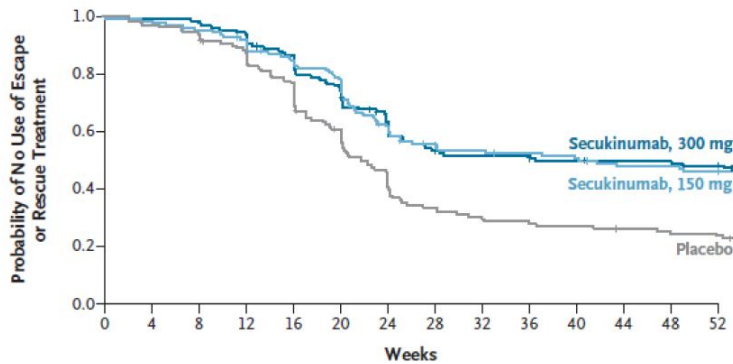
- Anti-CD20 provides clinical evidence that B cells contribute to PMR
- BRIDGE-PMR (Phase 2 IIT) demonstrated that a single rituximab infusion increased glucocorticoid-free remission at 21 weeks and produced durable benefit through one year^{2,3}
- REDUCE-PMR-1 (Phase 3 IIT) showed trend in favor of rituximab⁴

Phase 2 BRIDGE-PMR (21 weeks) and Phase 3 REDUCE-PMR-1 (52 weeks) IITs



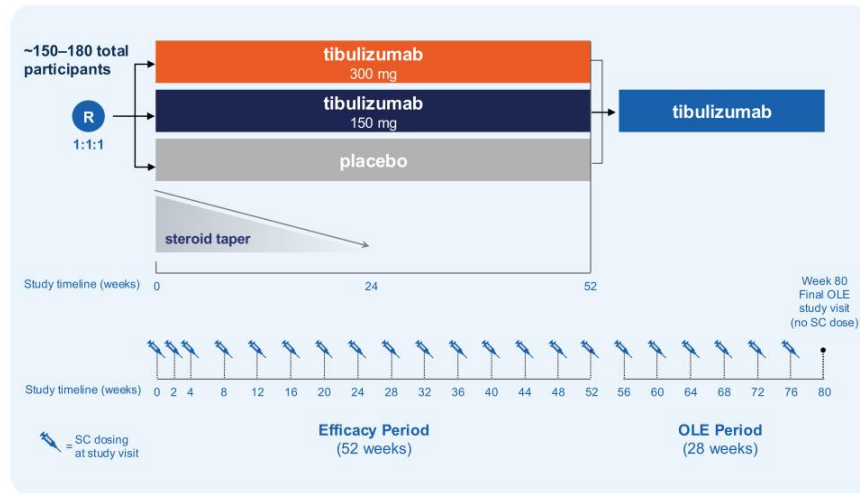
* Sustained remission defined as remission that occurred by Week 12 and was sustained until Week 52, without recurrence of PMR and no new diagnosis of GCA; complete sustained remission defined as sustained remission plus no ESR/CRP elevation at Week 52
Sources: 1. Stone et al. *N Engl J Med*. 2026 (REPLENISH); 2. Marsman et al. *Lancet Rheumatol*. 2021 (BRIDGE-PMR); 3. Bolhuis et al. *Lancet Rheumatol*. 2023 (BRIDGE-PMR 1-year extension); 4. REDUCE-PMR-1. *NCT05533725*.

Despite IL-17 advances, the unmet need for PMR remains significant



- **Glucocorticoids control PMR but cannot be withdrawn:** three in four patients on taper alone needed escape or rescue therapy by Week 52
- **IL-17 inhibition provides meaningful but only partial benefit:** more than half of participants in secukinumab arms do not achieve sustained remission

Proposed PMR Phase 2 study to be initiated by year-end 2026



NEXUS|PMR

Key Inclusion Criteria

- Diagnosis of PMR according to the 2012 provisional ACR/EULAR classification criteria
- History of treatment for ≥ 8 weeks for PMR with prednisone ≥ 10 mg/day
- ≥ 1 episode of PMR relapse at a prednisone dose ≥ 5 mg/day

Key Efficacy Endpoints

Primary endpoint

- Sustained remission at Week 52 defined as:
 - Clinical remission at Week 12 and maintained to Week 52
 - No use of escape or rescue therapy
 - No new diagnosis of giant cell arteritis

Key secondary endpoints

- Complete sustained remission at Week 52
- Time to the first use of escape or rescue treatment up to Week 52
- Cumulative glucocorticoid dose

Summary & Milestones

Executive Team and Board of Directors

Executive Team



Sandeep Kulkarni, MD
Co-Founder, Chief Executive Officer and Director



Kim Davis, JD
Chief Operating Officer, Chief Legal Officer and Corporate Secretary



Muzammil Mustafa
Chief Business Officer



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



Gary Whale, PhD
Chief Technology Officer



- Amit Munshi**
Chairman
- Sandeep Kulkarni, MD**
Co-Founder, CEO & Director
- Dan Becker, MD, PhD**
Director
- Mark Eisner, MD, MPH**
Director
- Jennifer Jarrett**
Director
- Ajay Nirula, MD, PhD**
Director
- Steve Schoch**
Director
- Parvinder Thiara**
Director

Multiple potential near-term clinical milestones and strong pipeline for the future

	TARGET	DISCOVERY	PHASE 1	PHASE 2	PHASE 3
Tibulizumab (ZB-106)	Anti-BAFF and IL-17	Hidradenitis Suppurativa			TibuSHIELD Readout Q4 2026
	Anti-BAFF and IL-17	Systemic Sclerosis			TibuSURE Readout H1 2027
	Anti-BAFF and IL-17	Polymyalgia Rheumatica			Anticipated Study Initiation YE 2026
Crebankitug (ZB-168)	Anti-IL-7R α	Phase 2-ready			
Torudokimab (ZB-880)	Anti-IL-33	Phase 2-ready			

Crebankitug: Phase 2-ready multi-functional antibody with potential in dermatology and beyond

Crebankitug inhibits two distinct cytokine pathways, **IL-7** and **TSLP**, through a single target

IL-7

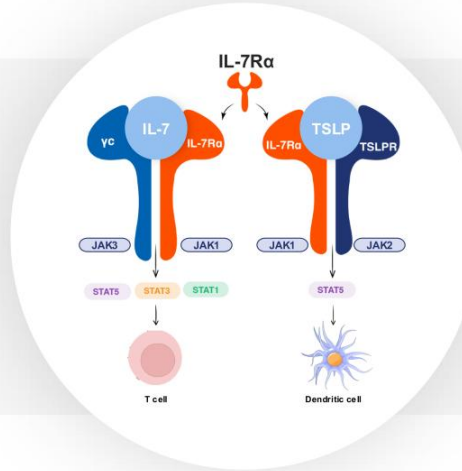
IL-7R α combines with the common gamma chain (γ c) to form the IL-7 receptor complex

- IL-7 signaling is vital for the development, survival, and balance of T cells
- IL-7 is implicated in many autoimmune and inflammatory diseases due to its central role in T-cell development

TSLP

TSLP binds to its specific receptor, TSLPR, initiating a signaling process that is optimized when IL-7R α integrates into the complex

- TSLP activates signaling pathways primarily associated with type 2 immunity
- TSLP pathways are commonly linked to allergic responses and certain inflammatory conditions



Torudokimab: Phase 2-ready IL-33 targeting antibody with potential in respiratory disease and beyond

About IL-33 and torudokimab

IL-33

IL-33 is a damage-associated “alarmin” cytokine released by stressed or injured tissues that activates immune cells through the ST2 receptor to promote inflammation, immune responses, tissue repair, and fibrosis^{1,2}

Torudokimab

Torudokimab is a fully human monoclonal antibody that binds and neutralizes IL-33⁵

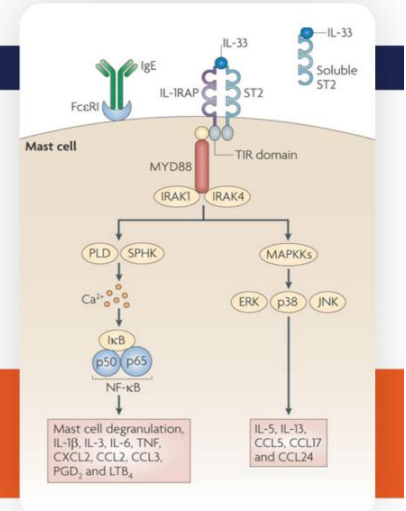
Clinical Results

Torudokimab was well tolerated in Phase 1 and Phase 2 trials conducted by Eli Lilly^{3,4}

- 141 healthy volunteers in Phase 1 study
- 103 participants with moderate-to-severe atopic dermatitis in Phase 2
- Target engagement confirmed with binding to IL-33 and downstream biomarker reductions³
- Treatment-emergent ADA had no apparent impact on PK or target engagement

Growing clinical validation of IL-33-targeting in respiratory:

- Potential for first-in-class opportunities
- Validated pathways in COPD and asthma⁶



Key Takeaways

Clear differentiation

Tibulizumab is a potential first and currently only in-class bispecific antibody in development designed to simultaneously target IL-17-mediated signaling and BAFF, addressing immune complexity beyond single-pathway approaches

Defined, anticipated clinical catalysts

Three independent Phase 2 trials (TibuSHIELD, TibuSURE, and NEXUS-PMR*) evaluating a dual-pathway strategy in diseases with significant unmet need and potential multi-billion-dollar market opportunities

Portfolio optionality

Multi-pathway immune biology provides potential for expansion into additional immune-mediated indications

Appendix

Cash and Shares Outstanding

Cash and cash equivalents balance \$187.7M (as of 9/30/26)	Number of shares (M)
Common stock	96.3
Prefunded warrants	29.3
Total Outstanding	125.6

Glossary

Ab	antibody	HAQ-DI	Health Assessment Questionnaire Disability Index	mRNA	messenger RNA	RU	relative units
ACA	anti-cardiolipin antibodies	HCP	healthcare professional	mRSS	Modified Rodnan Skin Score	SC	subcutaneous
ACR	American College of Rheumatology	HISCR50	Hidradenitis Suppurativa Clinical Response ≥50%	N	number of subjects	sxFv	single-chain variable fragment
ADA	anti-dsDNA antibody	HISCR75	Hidradenitis Suppurativa Clinical Response ≥75%	NETosis	neutrophil extracellular trap formation	SHAO-DI	Scleroderma Health Assessment Questionnaire Disability Index
AN	abscess and inflammatory nodule	HRCT	high-resolution computed tomography	NPX	normalized protein expression	sHISCR50	Simplified Hidradenitis Suppurativa Clinical Response (50%)
A-RNA Pol-III	anti-RNA polymerase 3 antibodies	HS	hidradenitis suppurativa	NRS	Numeric Rating Scale	sHISCR75	Simplified Hidradenitis Suppurativa Clinical Response (75%)
ATA	anti-topoisomerase antibodies	ISD	incision and drainage	NS	not significant	sHISCR90	Simplified Hidradenitis Suppurativa Clinical Response (90%)
BAFF	B-cell activating factor	IgA1, D, G	immunoglobulin A1, D, and G	OLE	open-label extension	SLE	systemic lupus erythematosus
BAFF-R	B-cell activating factor receptor	IS54	International Hidradenitis Suppurativa Severity Score System	P	p-value	SLS	Scleroderma Lung Study
BTk	Bruton's tyrosine kinase	IT	investigator-initiated trial	PAH	pulmonary arterial hypertension	SoC	standard of care
BTkG	Bruton's tyrosine kinase inhibitor	IL-1β	interleukin 1 beta	PASI	Psoarthritis Area and Severity Index	SoC	standard of care
CCL23/5/17/24	C-C motif chemokine ligand 23/5/17/24	IL-3	interleukin 3	PASI 90	Psoarthritis Area and Severity Index 90% improvement	SoC	standard of care
CD38/192/040/45	cluster of differentiation 3/8/19/20/40/45	IL-5	interleukin 5	PBO	placebo	SoC	standard of care
CFB	change from baseline	IL-6	interleukin 6	PD	pharmacodynamics	SSc	systemic sclerosis
COPO	chronic obstructive pulmonary disease	IL-7Rα	interleukin 7 receptor alpha	PEA	primary endpoint assessment	SSc-ILD	systemic sclerosis-associated interstitial lung disease
CRIS	Combined Response Index in Systemic Sclerosis	IL-8	interleukin 8	PGD₁	prostaglandin D2	ST2	suppression of tumorigenicity 2
CRP	C-reactive protein	IL-10	interleukin 10	PK	pharmacokinetics	T2D	type 2 diabetes
CXCL2	C-X-C motif chemokine ligand 2	IL-17	interleukin 17	pM	picomolar	TARC	thymus and activation-regulated chemokine
CyTOF	cytometry by time-of-flight	IL-18	interleukin 18	PMR	polymyalgia rheumatica	TGF-β	transforming growth factor beta
dsDNA	double-stranded DNA	IL-33	interleukin 33	PMR-AS	Polymyalgia Rheumatica Activity Score	TbH7	T helper 17 cells
DLCO	diffusing capacity of the lung for carbon monoxide	ILD	interstitial lung disease	PsO	psoriasis	TLS	tertiary lymphoid structure
DLS	Dermatology Life Quality Index	JAK	Janus kinase	Q2W	once every 2 weeks	TNF	tumor necrosis factor
EOS	end of study	K_d	dissociation constant	Q4W	once every 4 weeks	TNF-α	tumor necrosis factor alpha
ESR	erythrocyte sedimentation rate	lcSSc	limited cutaneous systemic sclerosis	qHRCT	quantitative high-resolution computed tomography	TNFi	tumor necrosis factor inhibitor
EULAR	European Alliance of Associations for Rheumatology	LPA1	lymphoproliferative acid receptor 1	QILD	quantitative interstitial lung disease	TSLP	thymic stromal lymphopoietin
FDA	Food and Drug Administration	LTB₄	leukotriene B4	QLF	quantitative lung fibrosis	TSLPR	thymic stromal lymphopoietin receptor
FVC	forced vital capacity	mAb	monoclonal antibody	QOL	quality of life	VAS	visual analog scale
GC	glucocorticoid	MMF	mycophenolate mofetil	R	randomization		
GCA	giant cell arteritis	MoA	mechanism of action	RA	rheumatoid arthritis		
GI	gastrointestinal			RNA	ribonucleic acid		



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