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

FORM 8-K

CURRENT REPORT
PURSUANT TO SECTION13 OR 15(D)
OF THE SECURITIES EXCHANGE ACT OF1934

August 11, 2026
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) 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 August 11, 2026, Zura Bio Limited (the "Company") issued a press release announcing its second 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 August 11, 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 August 11, 2026
99.2	Corporate Presentation, dated August 11, 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.

ZURA BIO LIMITED

Date: August 11, 2026

By: /s/ Kim Davis

Kim Davis

Chief Operating Officer, Chief Legal Officer and Corporate Secretary

Exhibit 99.1

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

- *TibuSHIELD enrollment complete, exceeding initial enrollment target with 247 participants randomized in HS; topline data expected Q4 2026*
- *TibuSURE enrollment complete, exceeding initial enrollment target with 91 participants randomized in SSc; topline data expected H1 2027*
- *Zura plans to initiate a Phase 2 study for tibulizumab in polymyalgia rheumatica (PMR) by year-end 2026*
- *Cash and cash equivalents of $205.1 million as of June 30, 2026, which is expected to fund planned operations through at least the end of 2028*

HENDERSON, Nev.--(BUSINESS WIRE)--August 11, 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 second quarter ended June 30, 2026, and provided recent corporate updates.

"The second quarter was marked by strong execution across our tibulizumab programs, with enrollment now complete in both TibuSHIELD and TibuSURE—each exceeding its initial enrollment target—underscoring growing conviction in dual IL-17 and BAFF inhibition," said Sandeep Kulkarni, M.D., Chief Executive Officer of Zura. "We are excited to nominate polymyalgia rheumatica as the third indication for tibulizumab, a debilitating disorder with high unmet need and important evidence supporting the roles of both IL-17 and BAFF. With cash expected to fund planned operations through at least the end of 2028, we believe we are well positioned—operationally, financially, and scientifically—as we approach our first topline readout in the fourth quarter of this year."

PROGRAM UPDATES

Tibulizumab (ZB-106)

Zura has completed enrollment in both Phase 2 studies of tibulizumab, a bispecific antibody designed to neutralize interleukin-17 (IL-17) and B-cell activating factor (BAFF), key mediators of inflammation and fibrosis, and announced plans to initiate a Phase 2 study in a third indication for tibulizumab by year end 2026.

- **TibuSHIELD (HS):** The Phase 2 clinical study evaluating tibulizumab in adults with hidradenitis suppurativa (HS) has completed and exceeded enrollment, with 247 participants. Topline data are anticipated in the fourth quarter of 2026.
- **TibuSURE (SSc):** The Phase 2 clinical study evaluating tibulizumab in adults with SSc has completed and exceeded enrollment, with 91 participants. Topline data are anticipated in the first half of 2027.
- **NEXUS-PMR (PMR) — new indication:** Zura has selected PMR as the third indication for tibulizumab. PMR is a common inflammatory rheumatic disease of older adults characterized by disabling bilateral shoulder and hip-girdle pain and stiffness, for which treatment options beyond long-term glucocorticoids remain limited. Over 700,000 people live with PMR today in the United States and the standard of care leaves most patients steroid-dependent, with a median time to permanent discontinuation of glucocorticoids of approximately six years. Zura believes tibulizumab's combined inhibition of IL-17 and BAFF may provide greater and more durable therapeutic benefit relative to other agents that only block a single pathway. Zura has engaged with the U.S. Food and Drug Administration (FDA) and received feedback that was constructive and supportive of the Company's proposed development strategy. Incorporating this feedback, Zura plans to initiate a Phase 2 study, NEXUS-PMR, evaluating tibulizumab in adult participants with PMR 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.

SECOND QUARTER 2026 FINANCIAL RESULTS

Cash Position

As of June 30, 2026, Zura had cash and cash equivalents of $205.1 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 $20.7 million for the second quarter of 2026, compared to $8.7 million for the same period in 2025. The increase was primarily driven by the advancement of Zura’s Phase 2 tibulizumab clinical programs.

General and Administrative (G&A) Expenses

G&A expenses were $8.6 million for the second quarter of 2026, compared to $9.4 million for the same period in 2025. The decrease was primarily due to lower expense in professional fees.

Net loss for the second quarter of 2026 was $26.3 million, or $0.21 per share, compared to $16.0 million, or $0.17 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). 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 potential to expand Zura’s product candidates into additional indications; Zura's interactions and engagement with the FDA, and the outcome of any related feedback on Zura's development strategy; 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*)

	June 30, 2026	December 31, 2025
	(unaudited)	
Assets		
Current assets		
Cash and cash equivalents	$ 205,134	$ 109,407
Prepaid expenses and other current assets	5,366	2,903
Total current assets	210,500	112,310
Property and equipment, net	149	126
Other assets	2,754	1,512
Total assets	$ 213,403	$ 113,948
Liabilities and Shareholders' Equity		
Current liabilities		
Accounts payable and accrued expenses	$ 16,318	$ 12,410
Total current liabilities	16,318	12,410
Total liabilities	16,318	12,410
Commitments and contingencies		
Shareholders' Equity		
Class A Ordinary Shares, $0.0001 par value; 300,000,000 shares authorized as of June 30, 2026 and December 31, 2025; 95,422,452 and 73,680,710 shares issued and outstanding as of June 30, 2026 and December 31, 2025, respectively	10	7
Additional paid-in capital	472,149	326,078
Accumulated deficit	(275,074)	(224,547)
Total shareholders' equity	197,085	101,538
Total liabilities and shareholders' equity	$ 213,403	$ 113,948

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

	For the Three Months Ended June 30,		For the Six Months Ended June 30,	
	2026	2025	2026	2025
Operating expenses:				
Research and development	$ 20,706	$ 8,704	$ 35,452	$ 19,178
General and administrative	8,615	9,358	19,368	18,138
Total operating expenses	29,321	18,062	54,820	37,316
Loss from operations	(29,321)	(18,062)	(54,820)	(37,316)
Other (income), net				
Interest income	(1,920)	(1,717)	(3,239)	(3,534)
Other income, net	(1,016)	(352)	(973)	(347)
Total other (income), net	(2,936)	(2,069)	(4,212)	(3,881)
Loss before income taxes	(26,385)	(15,993)	(50,608)	(33,435)
Income tax benefit	(81)	—	(81)	—
Net loss	$ (26,304)	$ (15,993)	$ (50,527)	$ (33,435)
Net loss per share, basic and diluted	$ (0.21)	$ (0.17)	$ (0.42)	$ (0.36)
Weighted-average shares outstanding, basic and diluted	126,803,970	94,289,954	119,553,659	93,630,719


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Corporate Overview
August 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 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 gives no assurance can be given 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 PMR[7]) or BAFF arm (SSc[9]) with strong scientific support for the orthogonal pathway[5,6,8]
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 1H 2027, respectively • Three-asset pipeline with optionality across multiple immune-mediated indications • Cash runway through at least year end 2028

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"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® PI; 2. Benlysta® PI; 3. Kimball et al., Lancet 2024;403:2504-2519; 4. Kimball et al., Lancet 2023;401:747-761; 5. Sabat et al., J Allergy Clin Immunol 2023;151:1015-1026; 6. Lowe et al., JCI Insight 2024;9:e169870; 7. Stone et al., NEJM 2026 (REPLENISH); 8. van der Geest et al., Rheumatology 2015;54:1397-1402; 9. Gordon et al., Arthritis Rheumatol 2018;70:308-316; 10. Zura Bio Ltd. filings; Evaluate Pharma

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

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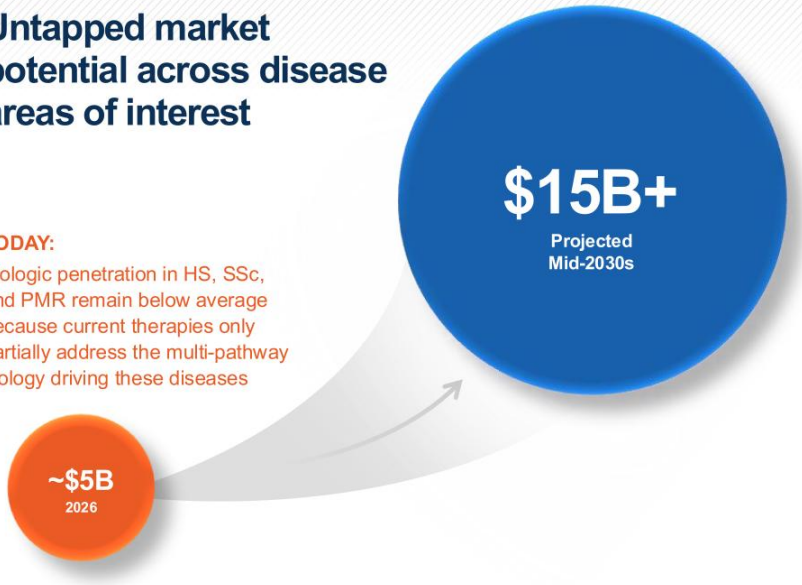
Untapped market potential across disease areas of interest



TODAY:

Biologic penetration in HS, SSc, and PMR remain below average because current therapies only partially address the multi-pathway biology driving these diseases

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 size

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Both circles represent combined HS, SSc, & PMR global markets

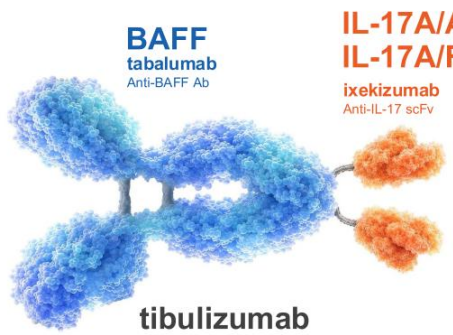
Sources: Gordon Lancet 2021 – BE READY, Gordon Lancet 2018 – UltIMMa 1/2, McInnes Lancet 2023 – BE OPTIMAL / BE COMPLETE, Mease NEJM 2015 – FUTURE 1, Kimball Lancet 2024 – BE HEARD I/II, Kimball Lancet 2023 – SUNSHINE/SUNRISE, Kimball NEJM 2016 – PIONEER I/II, Stone NEJM 2026 – REPLENISH, Spiera NEJM 2023 – SAPHYR, Khanna Lancet Resp Med 2020 – focuSSced, Distler NEJM 2019 – SENSCIS, Fukasawa JAAD 2023, Market estimates based on third-party analyses, earnings reports of currently marketed therapies, and published literature, including DRG/Clarivate, Evaluate Pharma, GlobalData, peer-reviewed epidemiology studies, and company analyses

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



tibulizumab

Tetravalent bispecific antibody designed to block BAFF and IL-17–mediated signaling (IL-17A/A and A/F heterodimers)

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)

Single-entity advantage

Enables dual-pathway modulation without multi-drug combination complexity

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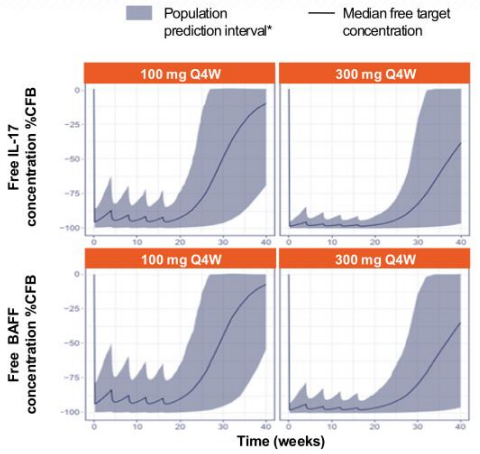
Tibulizumab is an investigational agent. Its efficacy and safety have not been established or approved by the FDA or any regulatory agency worldwide.
Sources: 1. ClinicalTrials.gov NCT06993610; 2. ClinicalTrials.gov NCT06843239; 3. Publicly available information on ixekizumab and tabalumab; 4. Benschop et al., mAbs 2019, doi:10.1080/19420862.2019.1624463.

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 (t½): 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

Dose-dependent and comparable BAFF and IL-17 target engagement



Model-based PK/PD analysis demonstrates a clear exposure–response relationship, with comparable BAFF and IL-17 suppression and near-complete target engagement at higher doses

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Tibulizumab is an investigational agent. Its efficacy and safety have not been established or approved by the FDA or any regulatory agency worldwide.
* Model-predicted median maximum percent change from baseline of free unbound BAFF and IL-17 following the last dose. Values shown reflect steady-state trough suppression.
Eli Lilly and Company–conducted Phase 1/1b clinical studies; Zura Bio Ltd. internal clinical study reports (CSRs).

Broad potential in heterogeneous immune disorders

IL-17

Amplifies inflammation, fibrosis, and tissue destruction

Drives recruitment and activation of neutrophils

Enhances trafficking and cross-talk of immune cells in tissues

Induces pro-inflammatory protein production, creating a self-sustaining inflammatory environment

Modulation of independent innate & adaptive immune drivers of disease

BAFF

Promotes autoreactive B-cell survival and function

Promotes maturation, survival, and function of autoreactive B and plasma cells

May promote chronic maintenance of lymphoid structures in tissue (TLS)

Persistence of B cells leads to increased cytokines, T cell activation, and autoantibodies

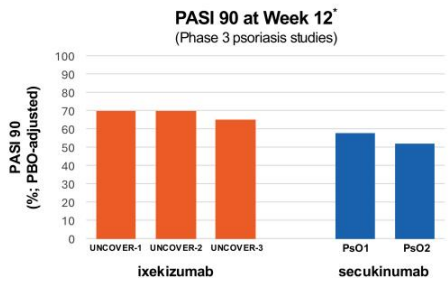
First and only bispecific antibody in development designed to target the orthogonal IL-17 and B cell pathways

Schematic representation of mechanism rationale. Not a clinical efficacy claim. References for B-cell axis in HS: Sabat JACI 2022; Lowe JCI Insight 2023; Gudjonsson JCI Insight 2020

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*
- Validated IL-17 pathway inhibition: Neutralization of IL-17A/A and IL-17A/F produces robust clinical responses with a well-characterized safety profile
- Extensive clinical safety experience: Supported by large global clinical development programs and post-marketing datasets across dermatologic and rheumatologic indications*
- Low incidence of candidiasis relative to IL-17F/F antagonists



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

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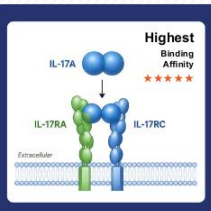
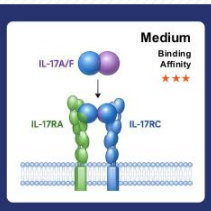
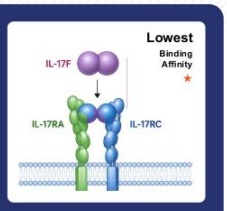
* PASI 90 values represent placebo-adjusted response rates at Week 12 from independent Phase 3 psoriasis trials. Studies were not designed for head-to-head comparison; trial designs, patient populations, dosing regimens, and placebo response rates differed. Clinical efficacy data shown are limited to psoriasis studies. Efficacy and durability may vary by indication.

Sources: 1. Griffiths CE, et al. Lancet 2015, doi:10.1016/s0140-6736(15)60125-8; 2. Langley RG, et al. N Engl J Med 2014, doi:10.1056/nejmoa1314258; 3. Gordon KB, et al. N Engl J Med 2016, doi:10.1056/nejmoa1512711; 4. Eli Lilly and Company and Novartis public disclosures and prescribing information

Tibulizumab potently 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

Sources: 1. Goedken ER, et al. J Pharmacol Exp Ther 2025;392(4):103525; 2. Br J Dermatol 2025;192:660-671; 3. JID Innovations 2024;4:100297, doi:10.1016/j.xjidi.2024.100297

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 comfort
Patients withdraw from social interactions due to visible symptoms	Embarrassment drives social withdrawal, relationship strain, absenteeism, mental health consequences
Patients struggle with persistent drainage and odor from lesions	Managing chronic drainage and odor becomes burdensome, leading to avoidance of exposure or hiding
Patients experience prolonged delays in diagnosis and treatment	Delayed diagnosis leads to continued symptom burden and progression to more severe disease

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Sources: Kimball, JAAD 2024; Kokolakis, Dermatology 2020; Lancet 2025 (Seminar)

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

Current Treatment Pathway

Initial Management

Topicals, oral antibiotics, and in-office procedures (I&D, intralesional corticosteroids) establish control and satisfy payer requirements before advancing therapy

Biologics (SoC)

Selection driven by access/formulary, not a strict algorithm: Humira® is declining as HCPs shift to IL-17s — Cosentyx® first (fast access), Bimzelx® often 2L+ (preferred efficacy)

Surgery

Reserved for biologic-refractory or structurally advanced patients with extensive tunneling and scarring that no longer respond to medical therapy

Limitations of Current Therapies:

Incomplete, non-durable control

Many patients never reach high-bar responses (HiSCR75/90) or lose efficacy over time - driving recurrent flares, pain, drainage, and odor

Patient impact remains high

Pain and quality of life impairment can persist even when objective measures improve

Structural damage is irreversible

Once disease progresses to tunnels and scarring, no current therapy can reverse it

Physicians remain only moderately satisfied

Despite current advancements, physicians desire new mechanisms of action that provide improved efficacy, deeper and more durable response

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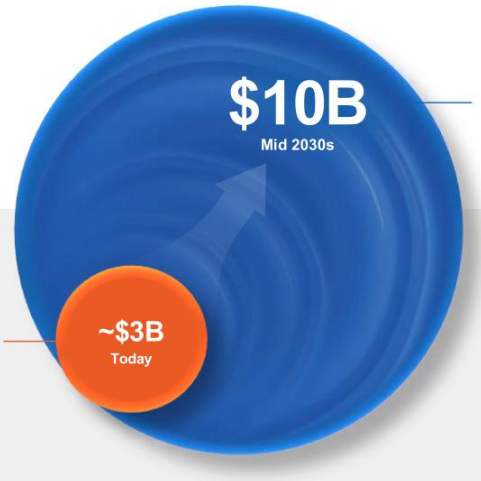
Sources: 1. Zouboulis et al., Lancet 2025; 2. Alikhan et al., JAAD 2019;81:91-101; 3. Kimball et al., Lancet 2024;403:2504-2519 (BE HEARD I&II); 4. Garbayo-Salmons et al., JEADV 2025;39:221-229; 5. Ingram et al., Pain Ther 2025; 6. Zura Bio, data on file, 2026

Hidradenitis suppurativa: large, underserved market with high unmet need



Multiple advanced therapies; increased cycling expected to innovative therapies, physicians seeking durable alternatives for chronic, treatment-experienced HS patients

Three advanced therapies (TNF, IL-17) compete for the current biologic-treated HS population

Growth Drivers & Unmet Needs

- Despite recent approvals, HS remains underdiagnosed and undertreated
- Biologic penetration rate increasing as treatment options expand
- Novel therapies challenging current efficacy ceilings; unmet need for deeper response

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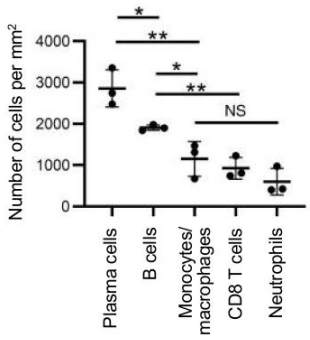
Sources: Real-world US claims analysis of HS treatment exposures, Dermatol Ther (Heidelb) 2022. Biologic penetration in moderate-to-severe HS and market projections ($3B 2026 to ~$10B mid-2030s) reflect internal market research and company analysis. All figures approximate.

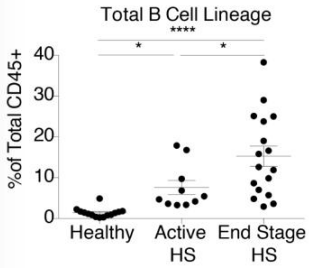
Unlike healthy skin, B cells infiltrate and chronically persist in HS lesions, driving a local amplified and targeted immune response

B cells and plasma cells make up the bulk of HS cellular infiltrate [1]



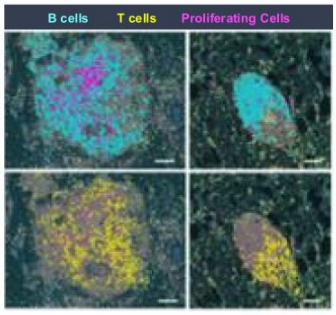
Imaging CyTOF analysis of HS lesions

B cells expand with disease progression [2]



Flow cytometry analysis of healthy skin biopsies, active HS biopsies (inflammatory nodules), or end stage HS tissue (tunnel from surgical excision)

In severe disease, B and T cells organize and persist in tertiary lymphoid structures (TLS) [3,4]



Immunofluorescence analysis of HS chronic lesion with tunnel

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Sources: 1. Gudjonsson et al., JCI Insight 2020; 2. Lowe et al., JCI Insight 2020; 3. Lowe et al., JCI Insight 2024;9:e169870; 4. Yu et al., Immunity 2024

BAFF is elevated across HS lesion types and predicts treatment resistance

Elevated BAFF can retain B cells in the skin

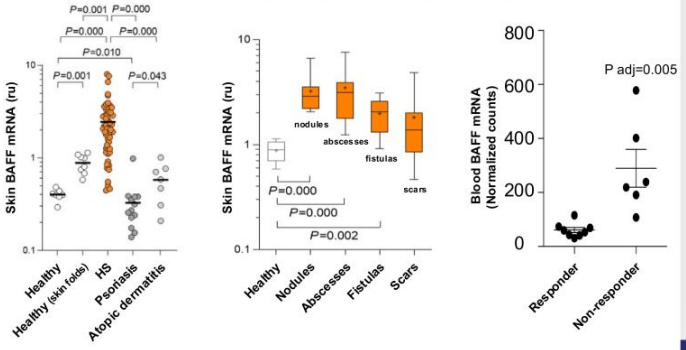
B cells migrate into HS lesions and initiate effector functions[3]

BAFF promotes B-cell accumulation, maturation, and differentiation

B and T cells organize into tertiary lymphoid structures that drive chronic, local immune activation[3,4]

- BAFF is absent in healthy skin and psoriasis and dermatitis lesions, and B cells transiently migrate in and out of skin[1,2]
- BAFF is significantly elevated in all HS lesion types, and correlates with increases in B cells[1,2]
- Patients who have higher baseline levels of BAFF and B-cell counts are less likely to respond to adalimumab[2,5]

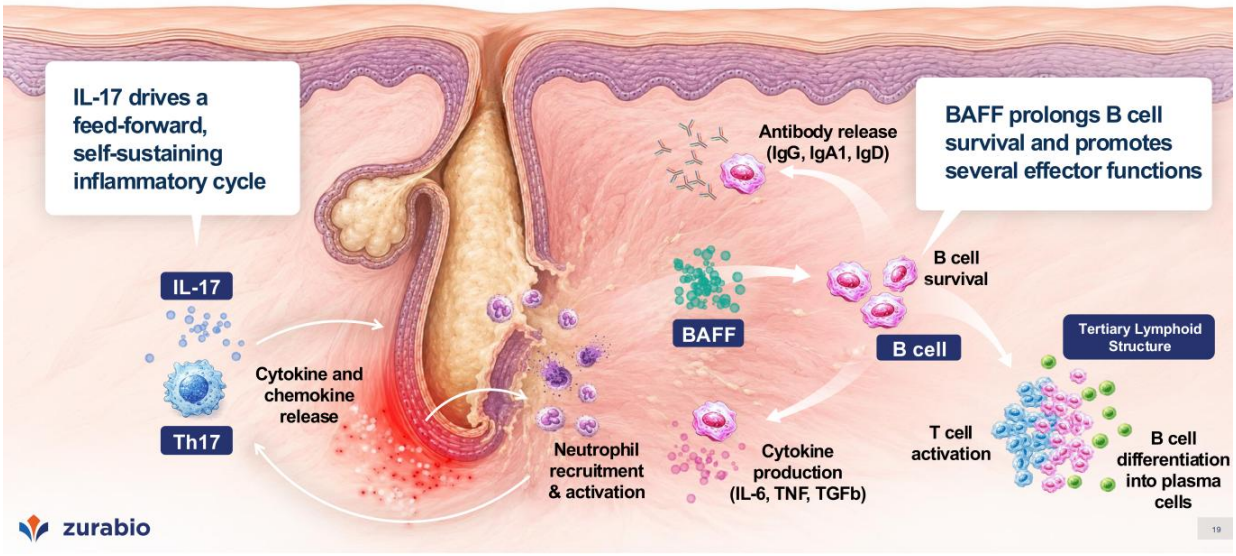
Elevated BAFF is unique feature of HS lesions, which predicts TNFi non-response



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

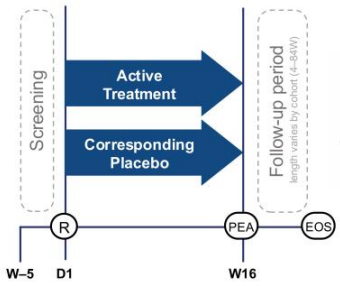
Sources: 1. Sabat et al., J Allergy Clin Immunol 2023;151:1015-1026; 2. Lowe et al., JCI Insight 2020; 3. Yu et al., Immunity 2024; 4. Gudjonsson et al., JCI Insight 2020; 5. Hambly et al., BJD 2022

B cells are believed to drive HS pathology via multiple mechanisms



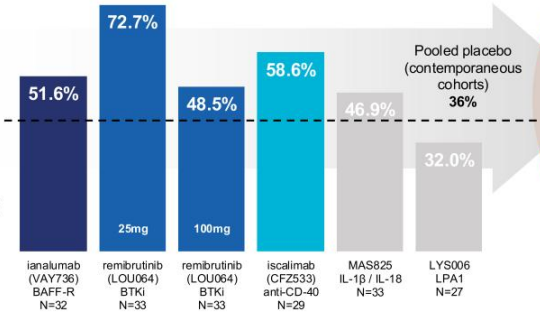
Recent clinical data supports the hypothesis that B cells are key drivers of disease rather than bystanders in HS*

Platform Study Design



Common design applied across 5 cohorts of the Novartis platform study; each cohort tested a different drug against its own randomized placebo arm.

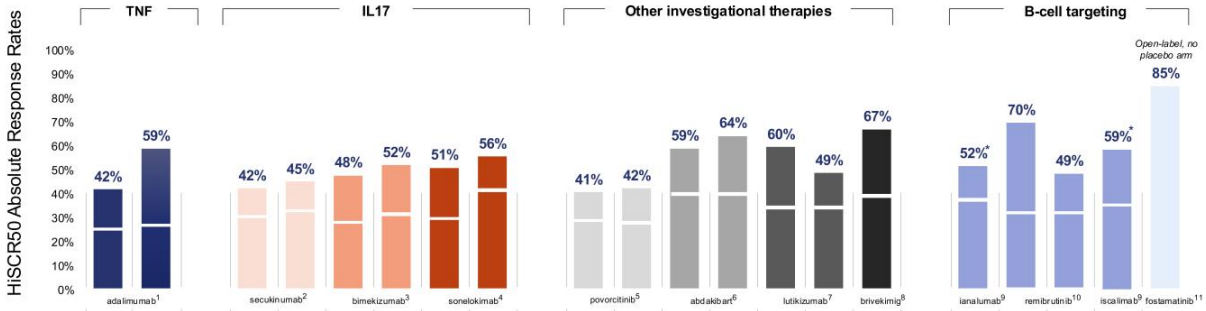
Observed sHiSCR50 Responders at Week 16 by Treatment Arm**



Three B cell-targeting mechanisms demonstrating HS efficacy signal

(*) Data derived from a randomized, placebo-controlled, multi-arm hidradenitis suppurativa platform study (ClinicalTrials.gov Identifier: NCT03827798). Reported results are based on information posted on ClinicalTrials.gov (as of 09-Jan-2026) and are subject to sponsor quality review and potential update. Results are descriptive and not powered for formal cross-arm comparisons.
(**) sHiSCR50: ≥50% reduction in abscess and inflammatory nodule count, with no increase in abscesses or draining fistulas, assessed using a simplified lesion-count methodology. Observed response rates; non-responder imputation applied
(***) Mean results from 25 mg and 100 mg arm shown
Sources: Novartis, AAD Annual Meeting 2024; ClinicalTrials.gov ID: NCT03827798.

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



Trial	PIONEER I Phase 3	PIONEER II Phase 3	SUNRISE Phase 3	SUNSHINE Phase 3	BE HEARD I Phase 3	BE HEARD 2 Phase 3	VELA-1 Phase 3	VELA-2 Phase 3	STOP HS-1 Phase 3	STOP HS-2 Phase 3	LOTUS Phase 2	LOTUS Phase 2	Phase 2	Phase 2	HS-OBTAIN Phase 2	Novartis platform study				Phase 2 Pilot
Week	12	12	16	16	16	16	16	16	12	12	16	16	16	16	16	16	16	16	16	12
N Active arm	153	163	180	181	289	291	283	276	202	208	83	84	37	39	58	32	33	33	29	20

Investigational drug Placebo response rate

* sHiSCR50: modified HiSCR50 omitting the "no increase in abscess count" criterion; For all studies, HiSCR50 was the primary endpoint except LOTUS (abdakibart; HiSCR75) and the Novartis platform study (ianalumab, remibrutinib, iscalimab; sHiSCR50). 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.

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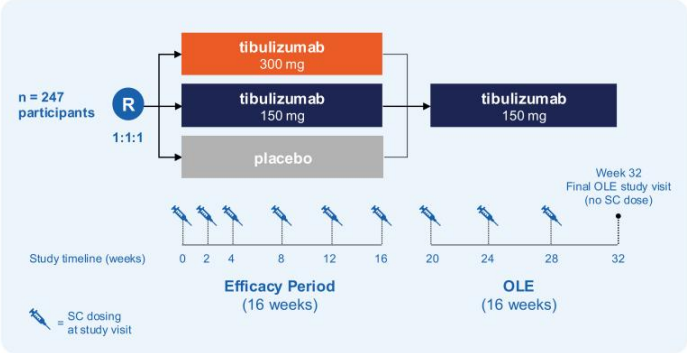
Sources: 1. Kimball AB, et al. N Engl J Med. 2016;375(5):422-434; 2. Kimball AB, et al. Lancet. 2023;401(10378):747-761; 3. Kimball AB, et al. Lancet. 2024;403(10443):2504-2519; 4. Porter ML, Kimball AB, et al. SHSA 2025, Oct 31–Nov 2, 2025. Presentation 3000674; 5. Porter ML, et al. Nature Medicine. Published online July 23, 2026; 6. Avalo Therapeutics. LOTUS Topline Presentation, May 2026; 7. Kimball AB, et al. JAMA Dermatol. 2026;162(5):469-477; 8. Sanofi. HS-OBTAIN Phase 2a Readout, EADV Congress, September 17, 2025; 9. ClinicalTrials.gov. NCT03827798. Results posted February 6, 2026; 10. Kimball AB, et al. AAD Annual Meeting, March 8–12, 2024; 11. Jepsen R, et al. JAAD. 2023;89(4):694-702

Orthogonal nature of IL-17 & 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



TibuSHIELD

Key Inclusion 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
- Additional eligibility criteria per protocol

Planned 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

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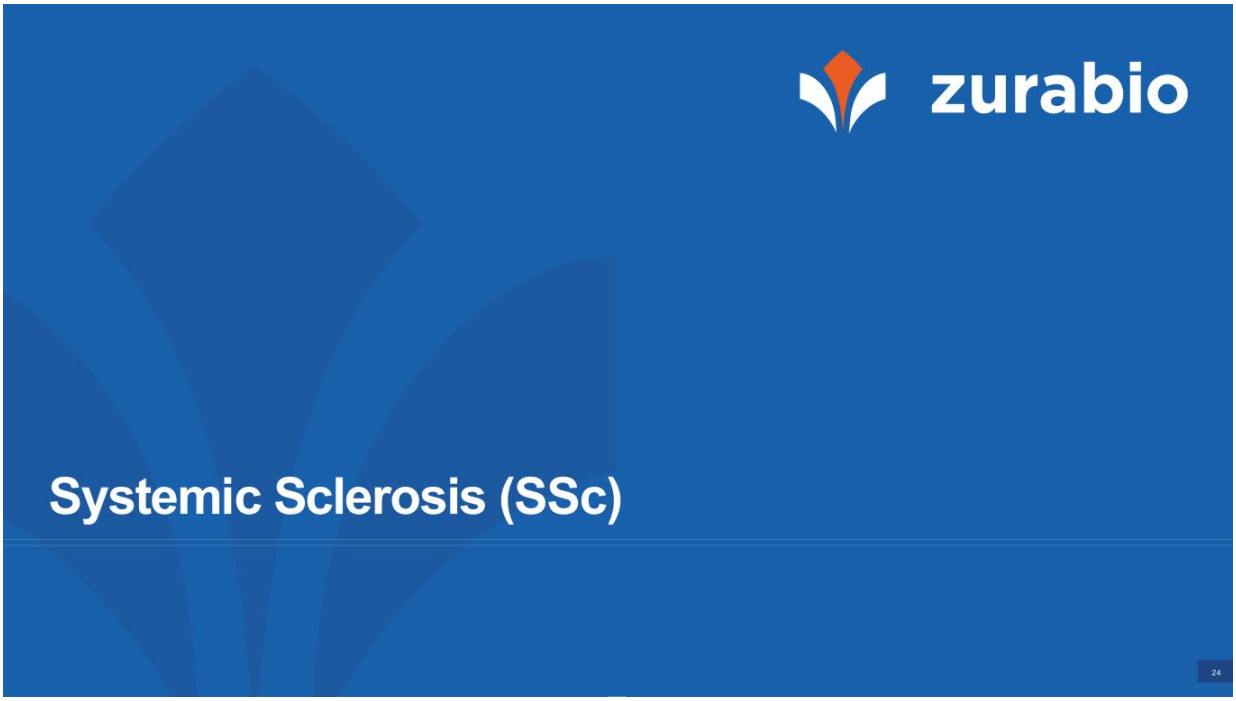
* Includes secondary and exploratory endpoints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Systemic Sclerosis (SSc)
24

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)

No therapies are approved that comprehensively address the multisystem pathology of SSc*

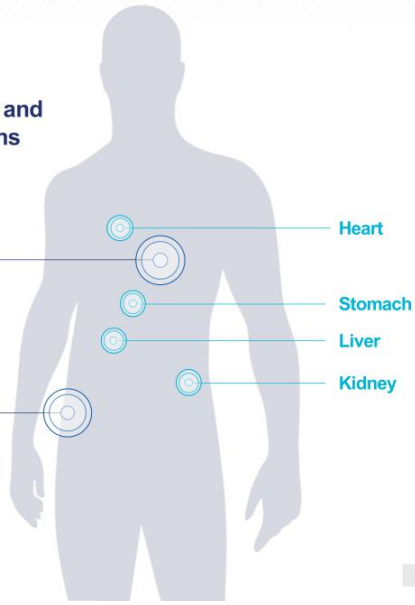
Characterized by Chronic Inflammation and Fibrosis Across Organs

Lungs

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

Skin

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



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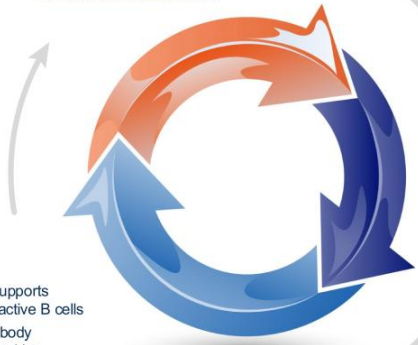
* Two therapies are approved for SSc-ILD; however, no treatment approved for SSc addresses multiple organ systems.
Sources: Clarivate/DRG (accessed 19 August 2024); public regulatory disclosures and prescribing information.

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

Vasculopathy

- Immune-mediated endothelial dysfunction contributes to impaired blood flow
- Inflammatory cytokines (IL-6, **IL-17**) thought to promote immune cell infiltration and vascular injury



Fibrotic Remodeling

- Fibroblast activation contributes to progressive tissue fibrosis
- **IL-17** activates inflammatory pathways in fibroblasts; autoantibodies promote fibrosis
- Excess extracellular matrix deposition (e.g., collagen) is characteristic of SSc

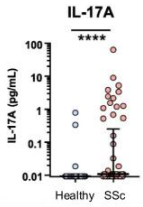
Inflammation

- **BAFF** signaling supports survival of autoreactive B cells
- Elevated autoantibody production and cytokine networks (including IL-6, **IL-17**) are observed in SSc

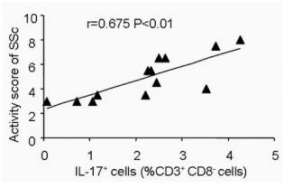
IL-17A is increased in SSc blood and skin and is linked to active and early disease

IL-17A elevated in blood and correlates with disease activity

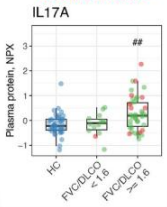
IL-17A cytokine elevated in SSc[1]



Blood IL-17+ cells correlate with disease activity[2]

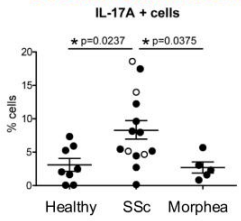


Serum IL-17A a predictor and correlate of pulmonary vascular disease[1,3]

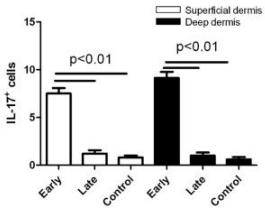


Elevations of IL-17-expressing T cells in skin correlate with early disease

IL-17A+ cells increased in SSc skin[4,5]



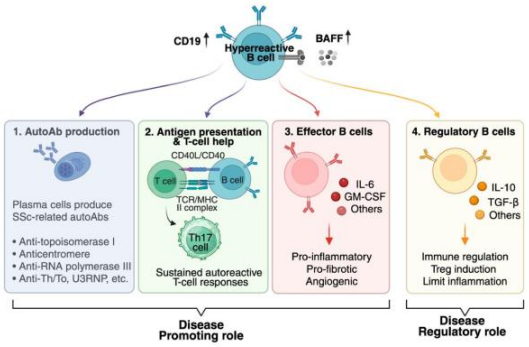
IL-17A+ cells increased in early disease skin[2]



Sources: 1. Seki et al., Cytokine 2024; 2. Yang et al., Arthritis Research & Therapy 2014; 3. Ono et al., Scientific Reports 2024; 4. Lonati et al., PLoS ONE 2014; 5. Deng et al., Frontiers in Immunology 2025

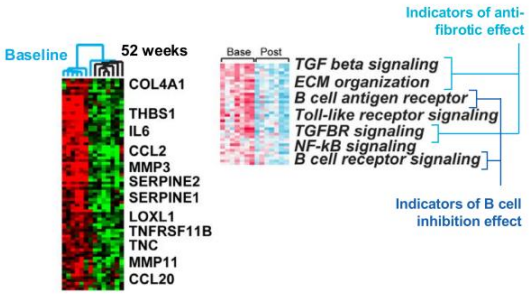
BAFF inhibition reduces profibrotic and B cell signaling genes in SSc skin[2]

Hypothesized disease-promoting roles of B cells[1]



Belimumab modulated gene profiles in post-treatment SSc skin[2]

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



Sources: 1. Matsushita et al, Sclerosis 2026; 2. Gordon et al., Arthritis and Rheumatology 2018

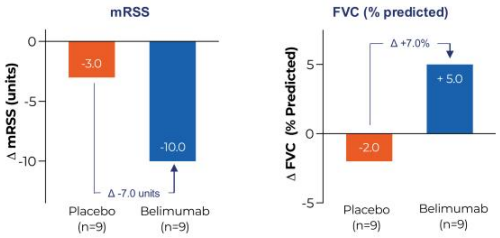
IL-17 and BAFF blockade have each improved skin and lung outcomes in SSc

BAFF ANTAGONIST

Belimumab

- In 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 trial (52 weeks)

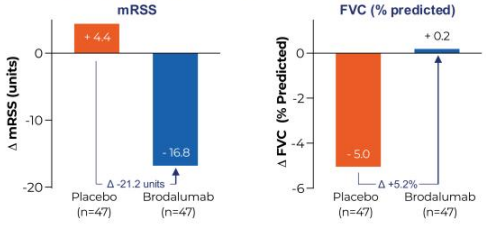


IL-17 RECEPTOR ANTAGONIST

Brodalumab

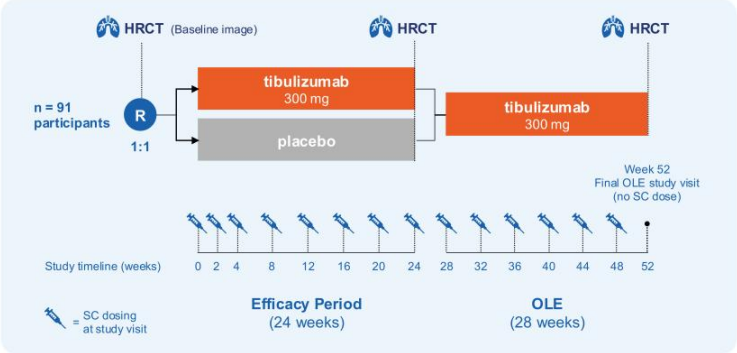
- Met the primary endpoint of reduced mRSS at Week 24 for skin involvement, and demonstrated improvement in FVC as a secondary endpoint reflecting lung function
- Also showed therapeutic effects on lung/respiratory functions, digital ulcers, symptoms of gastroesophageal reflux disease, and QOL without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

Phase 3 brodalumab trial (24 weeks)



Sources: 1. Fukasawa T, et al. Ann Rheum Dis 2022, doi:10.1136/annrheumdis-2022-eular.2519 (NCT03957681); 2. Gordon JK, et al. Arthritis Rheumatol 2018;70:308-316 (NCT01670565)

Phase 2 study ongoing in SSc; enrollment completed



TibuSURE

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

Endpoints and Stratification

Primary Endpoint

- Change from baseline in mRSS at Week 24

Additional Endpoints*

- qHRCT lung imaging
- FVC
- HAQ-DI
- revised CRISS

Stratification factors

- Presence of ILD (yes/no)
- Duration of disease (≥ or <2 years)
- Anti-RNA polymerase 3 (+/-)

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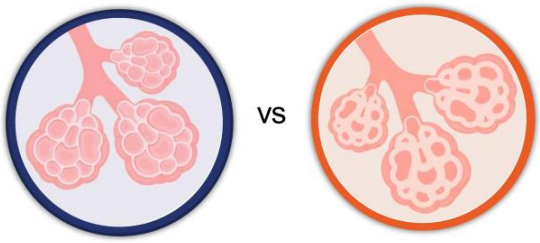
* Includes secondary and exploratory endpoints

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



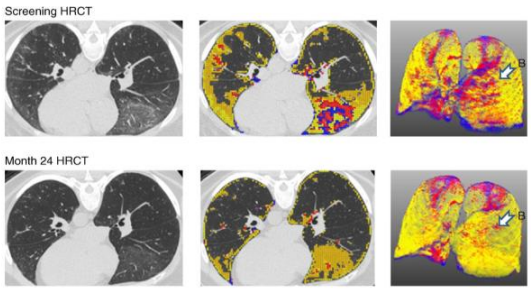
Healthy Lung | Fibrotic Lung

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

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

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

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

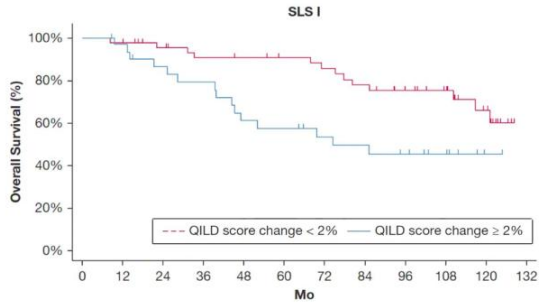


The blue and red areas show QLF, while the yellow area shows quantitative ground glass. The entire colored area represents QILD. After 24 months, QLF areas decreased (*arrow in B*).

Sources: 1. Goldin J, et al. Ann Am Thorac Soc 2018, doi:10.1513/annalsats.201802-079OC; 2. Zura Bio, data on file, 2026

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

2 large longitudinal SSc studies (SLS I and SLS 2) showed that ≥2% increase in QILD associated with increased risk of death [1]



- QILD is highly correlated with FVC [2,3]
- Clinically meaningful FVC changes were associated with 2% QILD change [2]

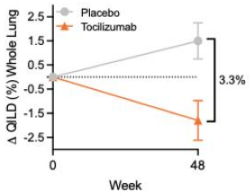
Tocilizumab Phase 3 in SSc-ILD

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

- -6.4% (PBO) vs. 0.1% (tocilizumab) over 48 weeks[4]

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%



Sources: 1. Volkmann E, et al. CHEST 2022; 2. Kafaja S, et al. Am J Respir Crit Care Med 2018; 3. Goldin J, et al. Ann Am Thorac Soc 2018; 4. Actemra USPI; 5. Roofeh D, et al. Arthritis Rheumatol 2021;73(7)

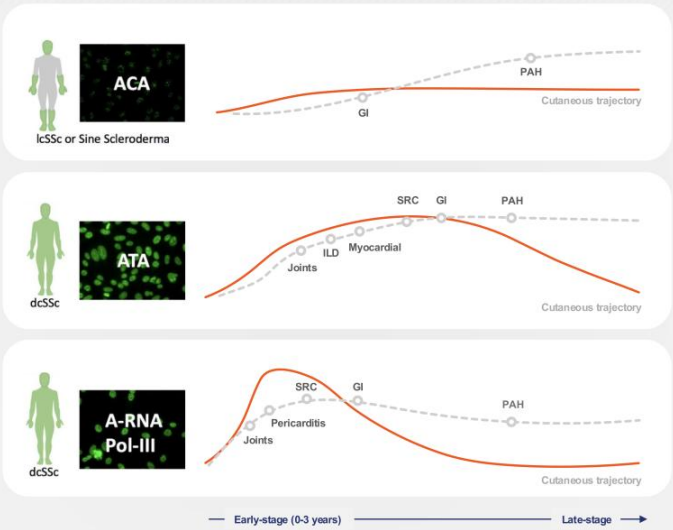
Published SSc disease trajectories validate our Phase 2 enrollment strategy

What the data show

- Autoantibody drives disease progression
- ATA and RNA Pol III drive the fastest fibrosis
- mRSS peaks early, then regresses on its own

Why our design fits

- TibuSURE stratification factors designed to reduce confounders
- Targets the active window: mRSS 15–45, ≤7 years
- Maximizes the probability of success for skin primary endpoint
  - Exclude Anti-centromere
  - Include all participants with anti-RNA pol-III up to 2 years duration, after which require severe disease



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Sources: 1. Lescoat et al., Seminars in Immunopathology 2025;47:39

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
- Systemic inflammation is present with increased erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) levels
- Driven by innate and adaptive immune activation

Demographics and risk factors

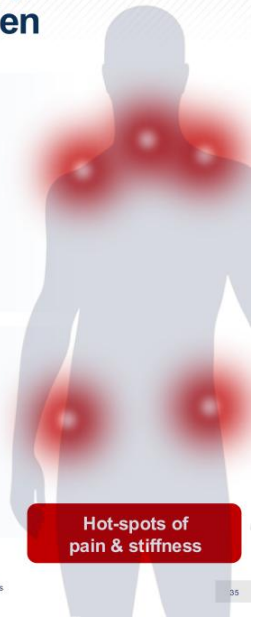
- Almost exclusively patients over 50; peak onset at ages 70–80
- Women are more likely to develop PMR than men
- Highly comorbid population

Signs and symptoms

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

Steroid dependency, relapse-prone

- Rapid symptom improvement on low-to-moderate corticosteroid doses
- 40–60% of patients relapse during steroid tapering
- Many patients remain on steroids for 5+ years



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Sources: Dasgupta et al., Ann Rheum Dis2012;71:484–92; Weyand & Goronzy, NEJM2014;371:50–57; Dejaco et al., Ann Rheum Dis2015;74:1799–807; Dejaco et al., Lancet 2017; Raheel et al., Arthritis Care Res 2017;69:1282–85; Floris et al., Clin Rheumatol 2022;41:19–31; Partington et al., Semin Arthritis Rheum 2020

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

Today, only 1 FDA-approved biologic[2]
PMR is an underserved and underpenetrated disease

Steroid-dependency[4]
Standard of care leaves most patients steroid-dependent

Need for more effective therapies[3,4,5]
Even with use of on and off-label therapies, complete and sustained remission remains inconsistent – HCPs are looking for more reliable options

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Sources: 1. Lawrence et al., Arthritis Rheum 2008;58:26-35; 2. KEVZARA (sarilumab) Prescribing Information, Sanofi/Regeneron; 3. Spiera et al., N Engl J Med 2023;389:1263-1272; 4. Floris et al., Clin Rheumatol 2022;41:19-31; 5. Zura Bio, data on file, 2026

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

Steroids-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%[3]

Relapse on steroid taper

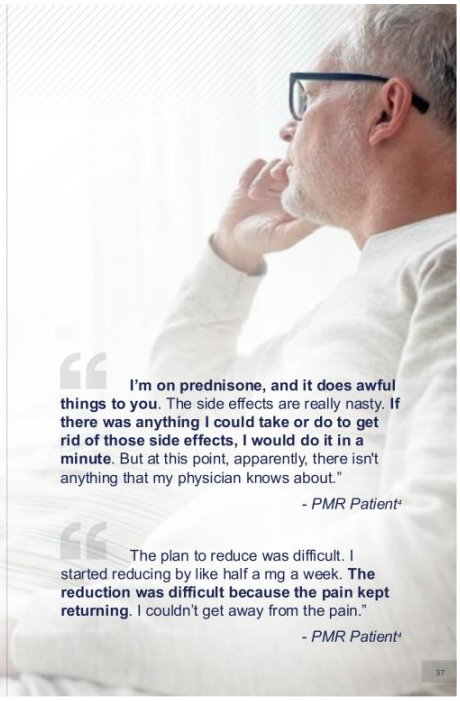
Patients are forced back to higher doses of glucocorticoids with no clear end in sight

Approx.

6 Yrs[1]

Median time to permanent glucocorticoid discontinuation

Due to high relapse rates during taper, many patients remain on steroids to manage PMR even after 5 years[2]



"**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 Patient[4]

"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 Patient[4]

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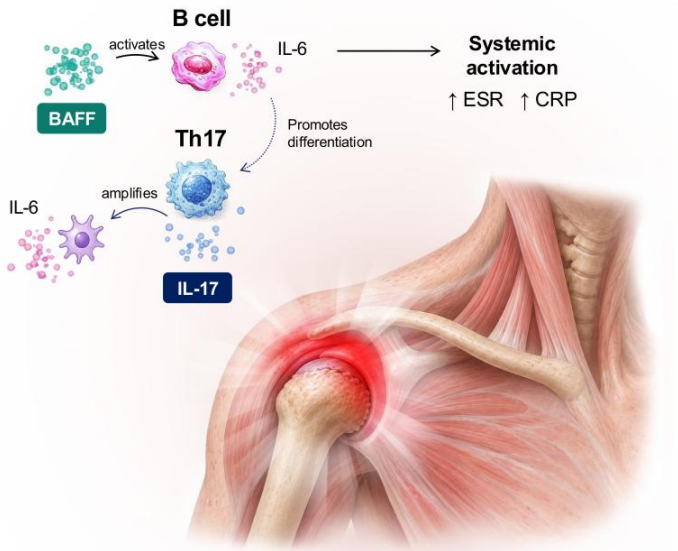
Sources: 1. Devauchelle-Pensec et al., Ann Rheum Dis, 2018, 2. Camellino et al., Front Med, 2022, 3. Spiera et al., N Engl J Med 2023;389:1263-1272, 4. Zura Bio, data on file, 2026

BAFF and IL-17 believed to contribute to PMR pathology

Elevated BAFF strongly correlates with disease activity and key PMR biomarkers: CRP, ESR, & IL-6[1,2]

IL-17 is elevated in blood & tissue around PMR joints and correlates with disease activity[3,4]



Sources: 1. van der Geest et al., Rheumatology 2015;54:1397-1402, 2. van der Geest et al., Arthritis Rheumatol 2014, 3. Samson et al., Arthritis Rheum 2012;64:3788-3798, 4. Ann Rheum Dis 2025 (PMR immunopathophysiology review)

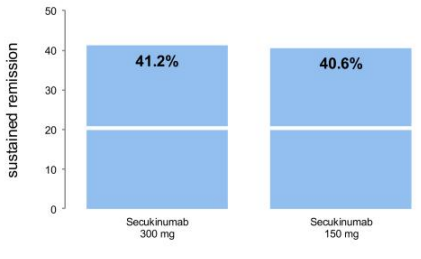
IL-17 and B-cell targeted therapies have shown positive glucocorticoid-sparing effects in PMR

IL-17A inhibitor – Phase 3 in relapsing PMR

Secukinumab[1]

- 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 & EU for approval in PMR

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

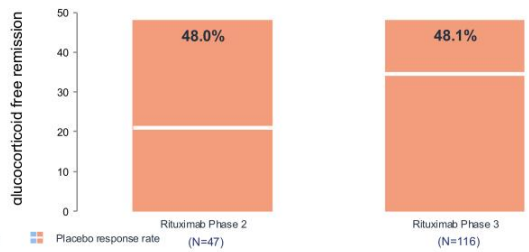


Anti-CD20 – Phase 2 and 3 in newly diagnosed PMR

Rituximab

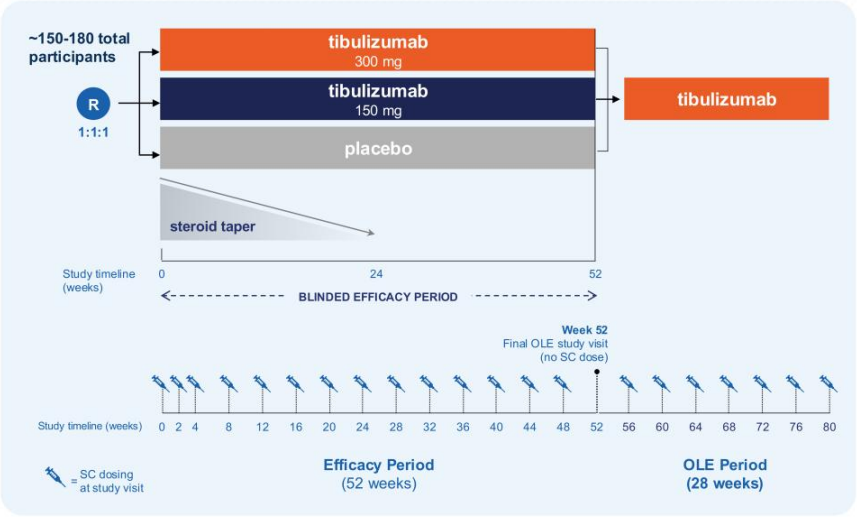
- BRIDGE-PMR (Phase 2 IIT) demonstrated that a single rituximab infusion increased glucocorticoid-free remission at 21 weeks and produced durable benefit through one year[2,3]
- REDUCE-PMR-1 (Phase 3 IIT) study in newly diagnosed PMR patients showed trend in favor of rituximab[4]
- REDUCE-PMR-2 (Phase 3 IIT) results expected in 2027 in relapsing PMR[5]

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



Sources: 1. Stone et al., N Engl J Med 2026, DOI:10.1056/NEJMoa2602567 (REPLENISH; NCT05767034); 2. Marsman et al., Lancet Rheumatol 2021;3:e758-e766 (BRIDGE-PMR); 3. Bolhuis et al., Lancet Rheumatol 2023;5:e208-e214 (BRIDGE-PMR 1-year extension); 4. REDUCE-PMR-1, NCT05533125; 5. REDUCE-PMR-2, NCT05533164

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

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

TOURMALINE
IMMUNOVANT
ROIVANT SCIENCES
QVT Financial LP
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Kim Davis, JD
Chief Operating Officer, Chief Legal Officer and Corporate Secretary

ARENA PHARMACEUTICALS
kaléo
Impax
AMGEN

Muzammil Mustufa
Chief Business Officer

Amicus Therapeutics
gsk
D E Shaw & Co

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

AstraZeneca
wellcome
gsk

Gary Whale, PhD
Chief Technology Officer

emergent BioSolutions
VHsquared
EUSA Pharma

Board of Directors

Amit Munshi
Chairman

Sandeep Kulkarni, MD
Co-Founder, CEO & Director

Dan Becker, MD, PhD
Director

Mark Eisner, MD, MPH
Director

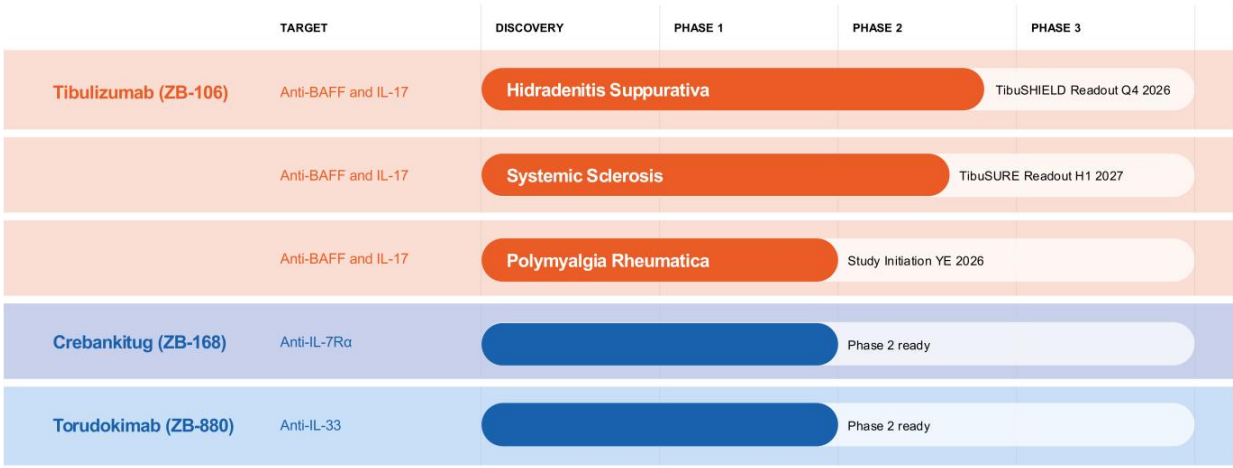
Jennifer Jarrett
Director

Ajay Nirula, MD, PhD
Director

Steve Schoch
Director

Parvinder Thiara
Director

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



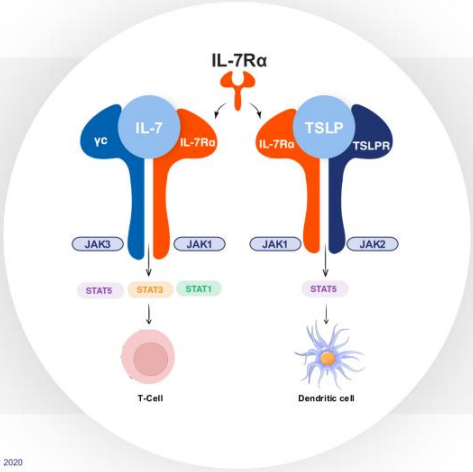
Crebankitug: Phase 2 ready multi-functional antibody with potential in dermatology & beyond

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

IL-7

IL-7Rα combines with the common gamma chain (γc) 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 central role in T cell development



TSLP

TSLP binds to its specific receptor, TSLPR, initiating a signaling process that is optimized when IL-7Rα 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

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Sources: 1. Markovic et al., Frontiers in Immunology 2020

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]

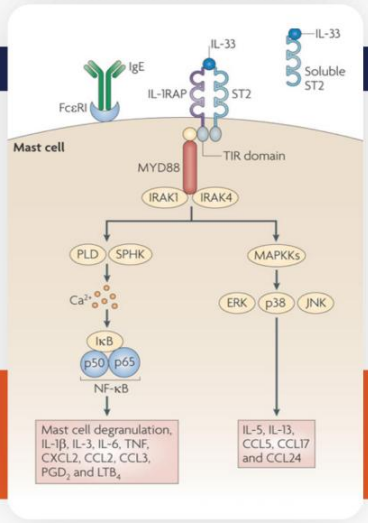
Torudokimab

Torudokimab is a fully human monoclonal antibody that binds and neutralizes IL-33[5]

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 reductions[3]
- 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 asthma[6]

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Sources: 1. Cohen ES, et al. Nat Commun 2015;6:8327; 2. Liew et al., Nature Reviews Immunology 2010; 3. Zura Bio, data on file, 2026; 4. Laquer et al., British Journal of Dermatology 2022; 5. Okragly et al., Journal of Inflammation Research 2021; 6. Wechsler et al., New England Journal of Medicine 2021

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

* NEXUS-PMR Phase 2 trial planned to be initiated by year end 2026

Appendix

Cash and Shares Outstanding

Cash and cash equivalents balance $205.1M (as of 6/30/26)	Number of shares (M)
Common stock	95.4
Prefunded warrants	29.3
Total Outstanding	124.7

Glossary

Ab	antibody
ACA	anti-centromere antibodies
ACR	American College of Rheumatology
ADA	anti-drug antibody
AN	abscess and inflammatory nodule
A-RNA-Pol-III	anti-RNA polymerase 3 antibodies
ATA	anti-topoisomerase antibodies
BAFF	B cell activating factor
BAFF-R	B-cell activating factor receptor
BTK	Bruton's tyrosine kinase
BTKi	Bruton's tyrosine kinase inhibitors
CCL17	C-C motif chemokine ligand 17
CD3/8/19/20/40/45	cluster of differentiation 3/8/19/20/40/45
CFB	change from baseline
COPD	chronic obstructive pulmonary disease
CRISS	Combined Response Index in Systemic Sclerosis
CRP	C-reactive protein
CyTOF	cytometry by time-of-flight
DLCO	diffusing capacity of the lung for carbon monoxide
DLQI	Dermatology Life Quality Index
dcSSc	diffuse cutaneous systemic sclerosis
ESR	erythrocyte sedimentation rate
EULAR	European Alliance of Associations for Rheumatology
FDA	Food and Drug Administration
FVC	forced vital capacity
GC	glucocorticoid
GI	gastrointestinal
HAQ-DI	Health Assessment Questionnaire Disability Index
HCP	healthcare professional
HiSCR50	Hidradenitis Suppurativa Clinical Response ≥50%
HiSCR75	Hidradenitis Suppurativa Clinical Response ≥75%
HRCT	high-resolution computed tomography
HS	hidradenitis suppurativa
I&D	incision and drainage
lcSSC	limited cutaneous systemic sclerosis
IgA1, D, G	Immunoglobulin A1, D, and G
IHS4	International Hidradenitis Suppurativa Severity Score System
IIT	investigator-initiated trial
IL-6	interleukin 6
IL-10	interleukin 10
IL-17	interleukin 17
IL-18	interleukin 18
IL-33	interleukin 33
IL-1β	interleukin 1 beta
ILD	interstitial lung disease
K_D	dissociation constant
LPA1	lysophosphatidic acid receptor 1
mAb	monoclonal antibody
MMF	mycophenolate mofetil
mRNA	messenger RNA
MoA	mechanism of action
mRSS	Modified Rodnan Skin Score
N	number of subjects
NETosis	neutrophil extracellular trap formation
NPX	normalized protein expression
NRS	Numeric Rating Scale
NS	not significant
OLE	open-label extension
P	p-value
PAH	pulmonary arterial hypertension
PASI	Psoriasis Area Severity Index
PASI 90	Psoriasis Area and Severity Index 90% improvement
PBO	placebo
PD	pharmacodynamics
PK	pharmacokinetics
pM	picomolar
PMR	polymyalgia rheumatica
PsO	psoriasis
Q4W	once every 4 weeks
qHRCT	quantitative high-resolution computed tomography
QILD	quantitative interstitial lung disease
QLF	quantitative lung fibrosis
QOL	quality of life
R	randomization
RA	rheumatoid arthritis
RNA	ribonucleic acid
ru	relative units
SC	subcutaneous
scFv	single-chain variable fragment
SHAQ-DI	Scleroderma Health Assessment Questionnaire–Disability Index
sHiSCR50	Simplified Hidradenitis Suppurativa Clinical Response (50%)
sHiSCR75	Simplified Hidradenitis Suppurativa Clinical Response (75%)
sHiSCR90	Simplified Hidradenitis Suppurativa Clinical Response (90%)
SoC	standard of care
SSc	systemic sclerosis
SSc-ILD	systemic sclerosis-associated interstitial lung disease
SRC	scleroderma renal crisis
ST2	serum stimulation-2
T2D	type 2 diabetes
TARC	thymus and activation-regulated chemokine
TGFb	transforming growth factor beta
Th17	T helper 17 cells
TLS	tertiary lymphoid structure
TNF	tumor necrosis factor
TNFi	tumor necrosis factor inhibitor
TNF-α	tumor necrosis factor alpha
VAS	visual analog scale


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