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Corporate Overview

April 2026

Nasdaq Ticker: **ZURA**

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Zura Bio: advancing a differentiated approach for complex immune disorders

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Our lead program, tibulizumab, is the first and currently only in-class bispecific antibody inhibiting both the IL-17 and BAFF pathways

- Fusion of components from tabalumab, a BAFF-binding antibody, and an IL-17–binding single-chain variable fragment derived from ixekizumab (marketed as Taltz®, ~$3.6B in global 2025 sales, as reported)
- Potent engagement of targets and low immunogenicity observed in completed Phase 1 studies

Tibulizumab has been rationally designed to address complex autoimmune diseases not fully addressed by single-pathway therapies

- Dual-pathway inhibition offers the potential to overcome efficacy ceilings observed with single-pathway inhibition
- Potential to modulate both B-cell– and T-cell–driven pathobiology

Initial clinical focus on hidradenitis suppurativa (HS) and systemic sclerosis (SSc), each characterized by complex immune pathobiology involving both B- and T-cell activation

- HS: potential to be the best-in-class treatment by inhibiting two pathways already independently validated
- SSc: potential to be first-in-disease treatment able to treat both skin and lung manifestations, where both IL-17 and BAFF have been implicated

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

- Topline data expected from HS and SSc phase 2 studies in Q4 2026 and 1H 2027, respectively
- ~$109M in cash (as of December 31, 2025), plus $144M gross proceeds from public offering closed February 26, 2026
- Capital expected to fund planned operations through at least the end of 2028
- As a result of recent February 2026 public offering, ~124M shares outstanding (as-converted)*

(*) Shares outstanding as of March 19, 2026; includes 21.2 million shares issued as part of February 2026 financing and ~29.3 million shares issuable upon conversion of outstanding pre-funded warrants to purchase Class A ordinary shares. This figure does not reflect potential dilution from outstanding stock options or unvested RSUs.

Sources: Zura Bio Ltd., public filings and disclosures; Evaluate Pharma; publicly available information on ixekizumab (Taltz®) and tabalumab.

Acronyms: BAFF, B-cell activating factor; HS, hidradenitis suppurativa; IL-17, interleukin-17; scFv, single-chain variable fragment; SSc, systemic sclerosis.

Executive Team and Board of Directors

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Executive Team

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IMMUNOVANT
ROIVANT SCIENCES
QVT Financial LP

Kim Davis, JD

Chief Operating Officer, Chief Legal Officer and Corporate Secretary

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Director

Two ongoing Phase 2 trials targeting high unmet need in multi-pathway autoimmune diseases

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Anticipated readout: Q4 2026

TibuSHIELD

A Phase 2 trial in adults with HS

Anticipated readout: 1H 2027

TibuSURE

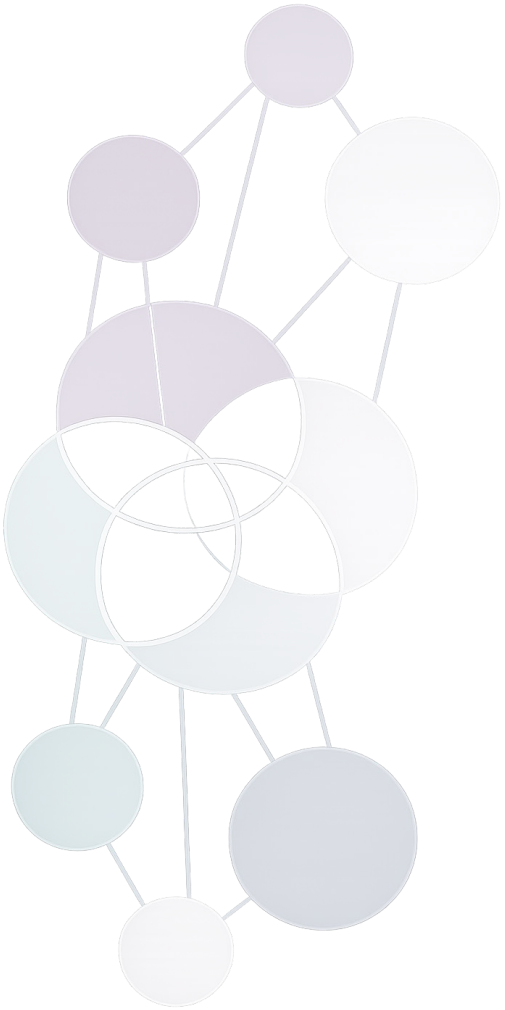
A Phase 2 trial in adults with SSc

Optionality for pipeline expansion into additional autoimmune indications with complex immune pathobiology

Sources: ClinicalTrials.gov ID NCT06993610; ClinicalTrials.gov ID NCT06843239.
Acronyms: HS, hidradenitis suppurativa; SSc, systemic sclerosis.

Limits of monotherapy in multi-pathway autoimmune disease

- Autoimmune diseases, including HS and SSc, are driven by multiple, intersecting immune pathways, resulting in biological heterogeneity across patients
- Single-pathway biologics may deliver low or inadequate response rates in complex autoimmune diseases
- Even among responders, pathway redundancy and compensatory signaling can limit depth and durability of response with monotherapy



Implication: these limitations support therapeutic approaches designed to address more than one disease-driving pathway with a single agent

Acronyms: HS, hidradenitis suppurativa; SSc, systemic sclerosis.

Tibulizumab: a single-molecule dual-pathway approach

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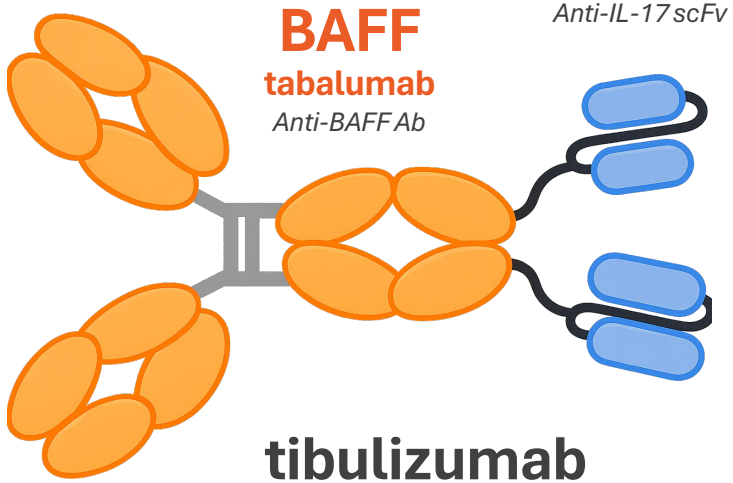
Inhibition of BAFF and IL-17A in one bispecific antibody



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

- **Addresses disease complexity:** aims to target two distinct immune pathways implicated in chronic inflammation and autoimmunity
- **Clinically grounded design:** fusion of a BAFF-binding antibody (tabalumab) with the IL-17A-binding scFv from ixekizumab
- **Single-entity advantage:** enables dual-pathway modulation without multi-drug combination complexity

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

Sources: ClinicalTrials.gov ID NCT06993610; ClinicalTrials.gov ID NCT06843239; Publicly available information on ixekizumab and tabalumab; Benschop et al., *mAbs* (2019), DOI: 10.1080/19420862.2019.1624463.

Acronyms: Ab, antibody; BAFF, B-cell activating factor; HS, hidradenitis suppurativa; IL-17, interleukin-17; IL-17A/F, interleukin-17A/interleukin-17F heterodimer; scFv, single-chain variable fragment; SSc, systemic sclerosis.

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

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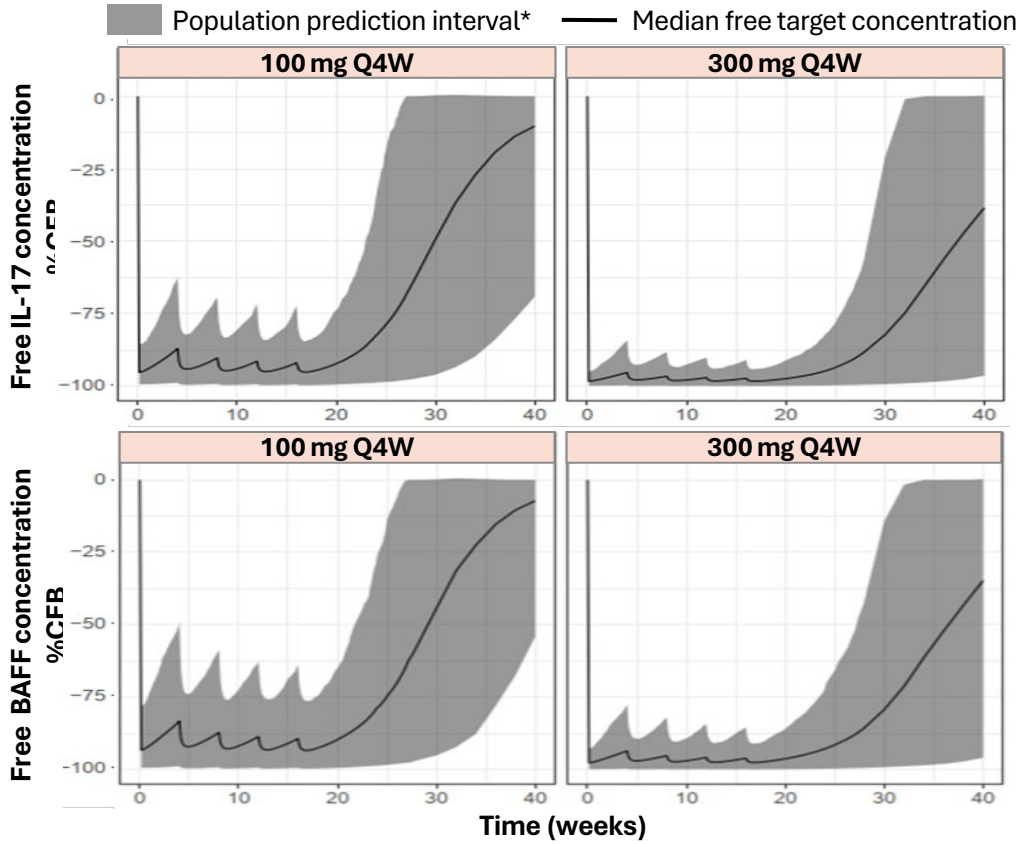
- 78 participants dosed across Phase 1/1b studies
- >98% median trough target engagement for both IL-17 and BAFF in peripheral blood at 300 mg Q4W
- Mean terminal half-life (t½): 26.9 days, supporting once-monthly dosing
- Pharmacodynamic activity observed, including B cell and CRP reductions
- Low incidence 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

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.

Sources: Eli Lilly and Company–conducted Phase 1/1b clinical studies; Zura Bio Ltd. internal clinical study reports (CSRs).

Acronyms: ADA, anti-drug antibody; BAFF, B-cell activating factor; CFB, change from baseline; CRP, C-reactive protein; IL-17, interleukin-17; PD, pharmacodynamics; PK, pharmacokinetics; Q4W, once every 4 weeks.

BAFF: a clinically validated immune pathway central to B cell survival

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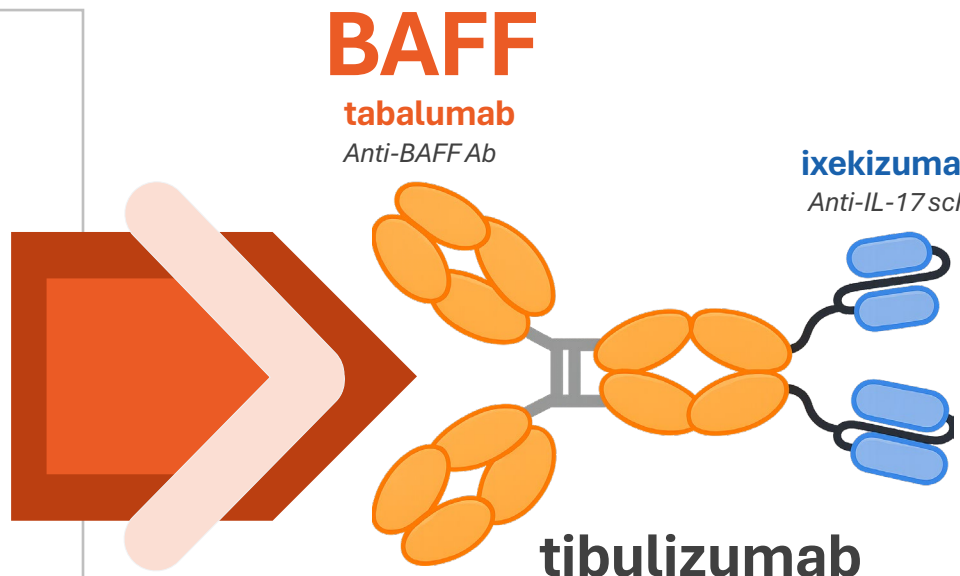
- BAFF is a non-redundant survival factor supporting persistence and differentiation of autoreactive B cells
- Genetic, translational, and clinical data show that disruption of BAFF signaling leads to marked reductions in peripheral B cells
- Elevated BAFF levels and B cell dysregulation are reported across immune-mediated diseases, including HS and SSc
- BAFF inhibition targets a core B cell survival pathway distinct from IL-17–mediated inflammatory signaling



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

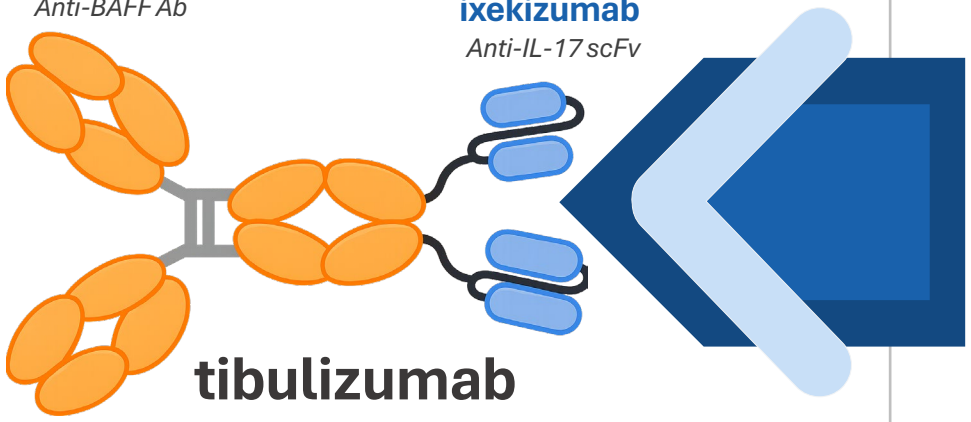
BAFF represents a clinically validated immune pathway addressing a fundamental driver of chronic autoimmune disease biology

Sources: Vincent, F.B. et al. (2013), Cytokine & Growth Factor Reviews, DOI:10.1016/j.cytogfr.2013.04.003; Smulski, C.R. and Eibel, H. (2018), Frontiers in Immunology, DOI:10.3389/fimmu.2018.02285; Matsushita, T. et al. (2005), Arthritis & Rheumatism, DOI:10.1002/art.21526.; Sabat, R. et al. (2023), Journal of Allergy and Clinical Immunology, DOI:10.1016/j.jaci.2022.10.034.

Acronyms: Ab, antibody; BAFF, B-cell activating factor; HS, hidradenitis suppurativa; IL-17, interleukin-17; IL-17A/F, interleukin-17A/interleukin-17F heterodimer; scFv, single-chain variable fragment; SSc, systemic sclerosis.

IL-17A: a clinically validated inflammatory pathway

IL-17A & IL-17A/F



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

- IL-17 is a central amplifier of inflammation across multiple immune-mediated diseases
- IL-17 pathway inhibition has demonstrated efficacy across multiple inflammatory indications, supported by extensive clinical experience
- Ixekizumab represents a well-established clinical benchmark for IL-17 pathway inhibition, blocking IL-17A and IL-17A/F signaling

IL-17A represents a clinically validated inflammatory pathway in autoimmune disease, with approvals in rheumatology and dermatology

Notes: The IL-17A-binding domain of tibulizumab is derived from ixekizumab.

Sources: Gaffen, S.L. et al. (2014), Nature Reviews Immunology, DOI:10.1038/nri3707; Griffiths, C.E. et al. (2015), The Lancet, DOI:10.1016/s0140-6736(15)60125-8; Blauvelt, A. et al. (2021), Journal of the American Academy of Dermatology, DOI:10.1016/j.jaad.2020.11.022; Eli Lilly and Company, ixekizumab (Taltz®) Prescribing Information.

Acronyms: Ab, antibody; BAFF, B-cell activating factor; IL-17, interleukin-17; IL-17A, interleukin-17A; IL-17A/F, interleukin-17A/interleukin-17F heterodimer; scFv, single-chain variable fragment.

Ixekizumab: a high-efficacy clinical benchmark for IL-17 pathway inhibition

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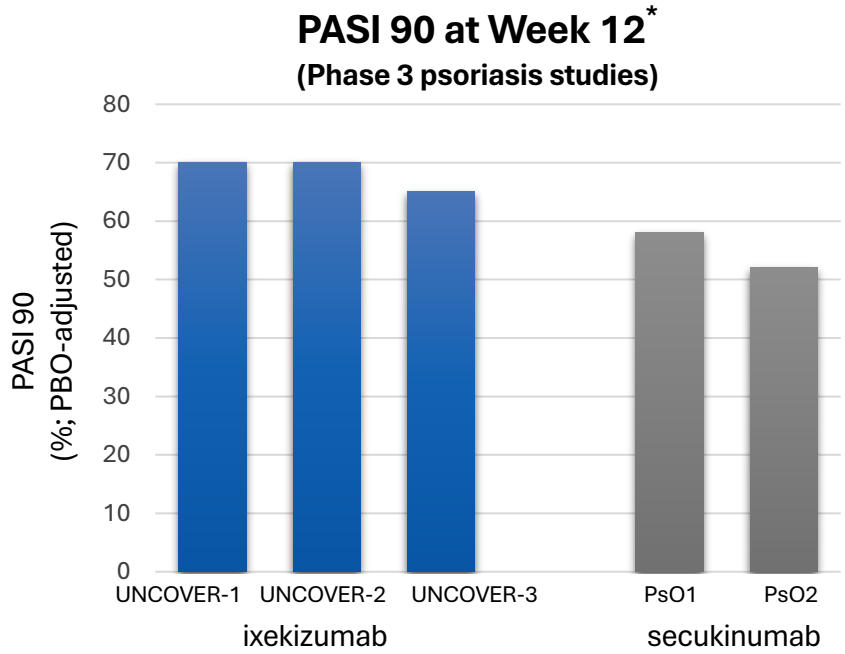
Multiple Phase 3 programs completed	High efficacy demonstrated	Broad IL-17 pathway inhibition	Extensive clinical and real-world experience

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



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: Griffiths, C.E. et al. (2015), The Lancet, DOI:10.1016/s0140-6736(15)60125-8; Langley, R.G. et al. (2014), New England Journal of Medicine, DOI:10.1056/nejmoa1314258; Gordon, K.B. et al. (2016), New England Journal of Medicine, DOI:10.1056/nejmoa1512711; Eli Lilly and Company and Novartis public disclosures and prescribing information.

Acronyms: IL-17, interleukin-17; IL-17A, interleukin-17A; IL-17A/F, interleukin-17A/interleukin-17F heterodimer; PASI 90, Psoriasis Area and Severity Index 90% improvement; PBO, placebo; PsO, psoriasis.

Tibulizumab is the first and only in-class bispecific antibody targeting IL-17 and BAFF

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		IL-17 pathway			B-Cell–driven inflammation
Agent	Target description	IL-17A/A (K_D)	IL-17A/F (K_D)	IL-17F/F* (K_D)	
Tibulizumab	**Anti-BAFF & IL-17 bispecific mAb**	**~14 pM**	**~90 pM**	**no binding**	**BAFF**
Secukinumab (Cosentyx®)	Anti-IL-17A mAb	~60–90 pM	~2400 pM	no binding	no
Bimekizumab (Bimzelx®)	Anti-IL-17A/F mAb	~3.2 pM	~26 pM	~23 pM	no

IL-17–only approaches

- Inhibits IL-17A signaling with varying affinity
- May also inhibit IL-17A/F or IL-17F to further suppress the IL-17 pathway
- Does not address additional disease drivers

Tibulizumab (anti-IL-17 + BAFF)

- Inhibits key IL-17 ligands (IL-17A/A and IL-17A/F)
- Adds BAFF pathway inhibition (B-cell biology)
- Is designed to address multiple disease drivers

(*) Clinical studies have shown higher rates of mucocutaneous candidiasis with broader IL-17 pathway inhibition that includes IL-17F.

Sources: Benschop, R.J. et al. (2019), mAbs, DOI:10.1080/19420862.2019.1624463; Adams, R. et al. (2020), Frontiers in Immunology, DOI:10.3389/fimmu.2020.01894.

Acronyms: BAFF, B-cell activating factor; HS, hidradenitis suppurativa; IL, interleukin; IL-17A, interleukin-17A; IL-17F, interleukin-17F; IL-17A/A, interleukin-17A homodimer; IL-17A/F, interleukin-17A/interleukin-17F heterodimer; IL-17F/F, interleukin-17F homodimer; K_D, dissociation constant; mAb, monoclonal antibody; pM, picomolar.

Tibulizumab targets two potentially high-value autoimmune market opportunities

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HIDRADENITIS SUPPURATIVA

Large and expanding chronic disease

- Growing biologics market supported by increasing diagnosis and utilization
- Chronic inflammatory disease with durable treatment demand and high unmet need
- Heterogeneous disease biology creates opportunity beyond single-pathway approaches

ESTIMATED TAM

~$8B projected by the mid-2030s

SYSTEMIC SCLEROSIS

High-value orphan autoimmune opportunity

- Rare autoimmune disease with significant unmet need and limited effective treatment options
- Specialist-managed market with concentrated prescribing and premium pricing dynamics
- Multisystem inflammatory and fibrotic manifestations contribute to substantial disease burden

ESTIMATED TAM

~$4B projected by mid-2030s

Potential significant multi-billion-dollar market opportunities

Note: Market estimates based on third-party analyses and published literature, including DRG/Clarivate, Evaluate Pharma, GlobalData, peer-reviewed epidemiology studies, and company analyses.
Acronyms: TAM, total addressable market.

tibulizumab

ZB-106

Anti-BAFF + IL-17

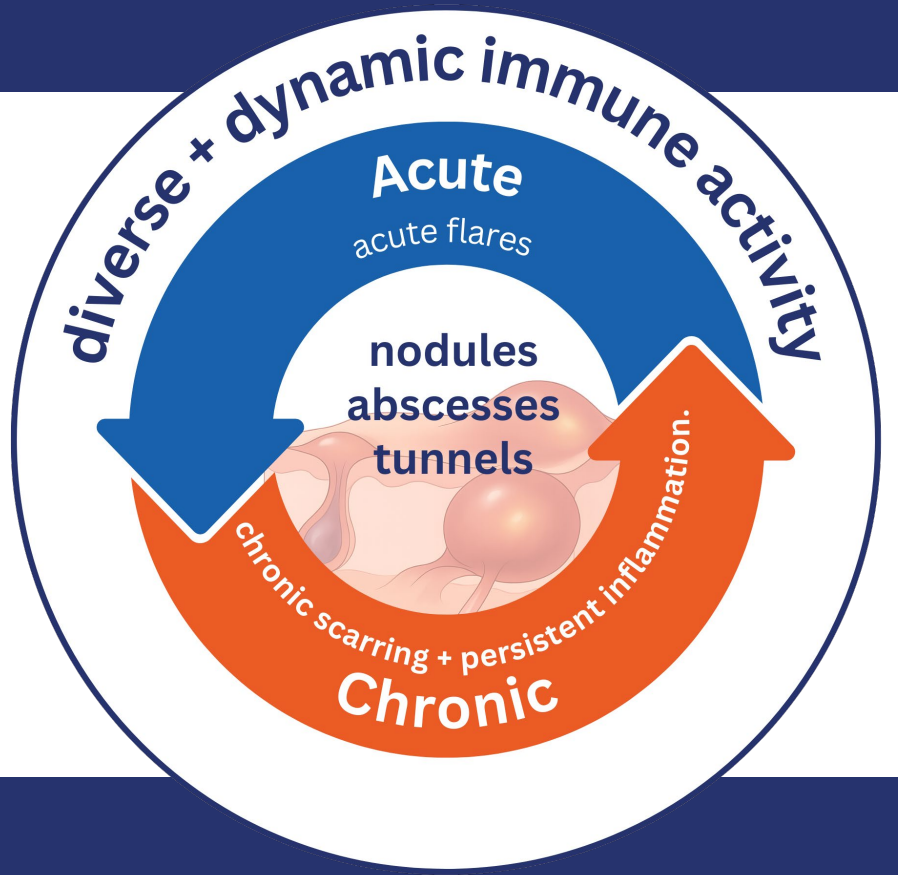
A dual-pathway approach in HS

- *First and currently only in-class bispecific antibody designed to target BAFF and IL-17 signaling, including IL-17A and IL-17A/F*
- *Phase 2 clinical trial (TibuSHIELD) ongoing; topline data anticipated in Q4 2026*

HS is a complex and relapsing disease

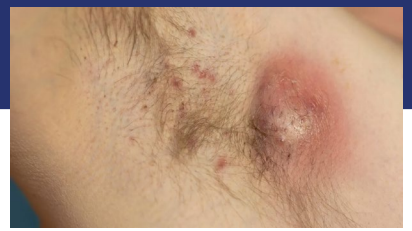
Underlying biology

- HS lesions are driven by a diverse, dynamic immune microenvironment
- Prominent immune signatures: Th1/Th17, neutrophils, B cells
- Chronic immune activity underlies persistent, relapsing disease course



Patient experience

- HS affects ~1% of the population and is widely underdiagnosed
- Painful nodules and abscesses form in sensitive areas
- Lesions may progress to tunnels and scarring
- Acute flares coexist with chronic inflammation
- Delayed diagnosis and variable treatment response



The interplay of acute lesions, chronic inflammation, and complex immune drivers makes HS uniquely challenging, and highlights the opportunity for biology-driven solutions

Sources: Moran, B. et al. (2017), Journal of Investigative Dermatology, DOI:10.1016/j.jid.2017.05.033; Banerjee, A. et al. (2017), Immunological Investigations, doi:10.1080/08820139.2016.1230867; Sabat, R. et al. (2023), Journal of Allergy and Clinical Immunology, DOI:10.1016/j.jaci.2022.10.034; Garg, A. et al. (2017), JAMA Dermatology, DOI:10.1001/jamadermatol.2017.0201; Ingram, J.R. (2020), British Journal of Dermatology, DOI:10.1111/bjd.19435; Midgette, B. et al. (2022), British Journal of Dermatology, DOI:10.1111/bjd.21798; Sabat, R. et al. (2020), Nature Reviews Disease Primers, DOI:10.1038/s41572-020-0149-1; MEDACorp key opinion leader (KOL) discussions.

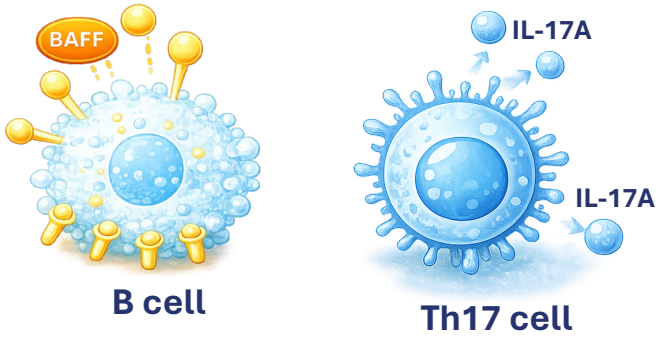
Acronyms: HS, hidradenitis suppurativa; Th1, T helper 1 cell; Th17, T helper 17 cell.

A dual-pathway approach to hidradenitis suppurativa

Targeting immune drivers of chronic inflammation and tissue damage

Disease

- Chronic, inflammatory skin disease with recurrent painful lesions
- Significant impact on quality of life and long-term morbidity



Biology

- Heterogeneous disease driven by activation of multiple immune pathways
- BAFF elevation and associated B cell dysregulation observed in HS lesions
- IL-17A–driven inflammation implicated in dysfunction of neutrophils, macrophages, and keratinocytes

Program

- Tibulizumab (BAFF + IL-17A bispecific antibody)
- Phase 2 TibuSHIELD clinical study in adults with HS; topline data expected Q4 2026

Acronyms: BAFF, B cell activating factor; HS, hidradenitis suppurativa; IL-17A, interleukin-17A; Th17, T helper 17 cells.

Neutrophils and B cells represent orthogonal immune drivers in HS

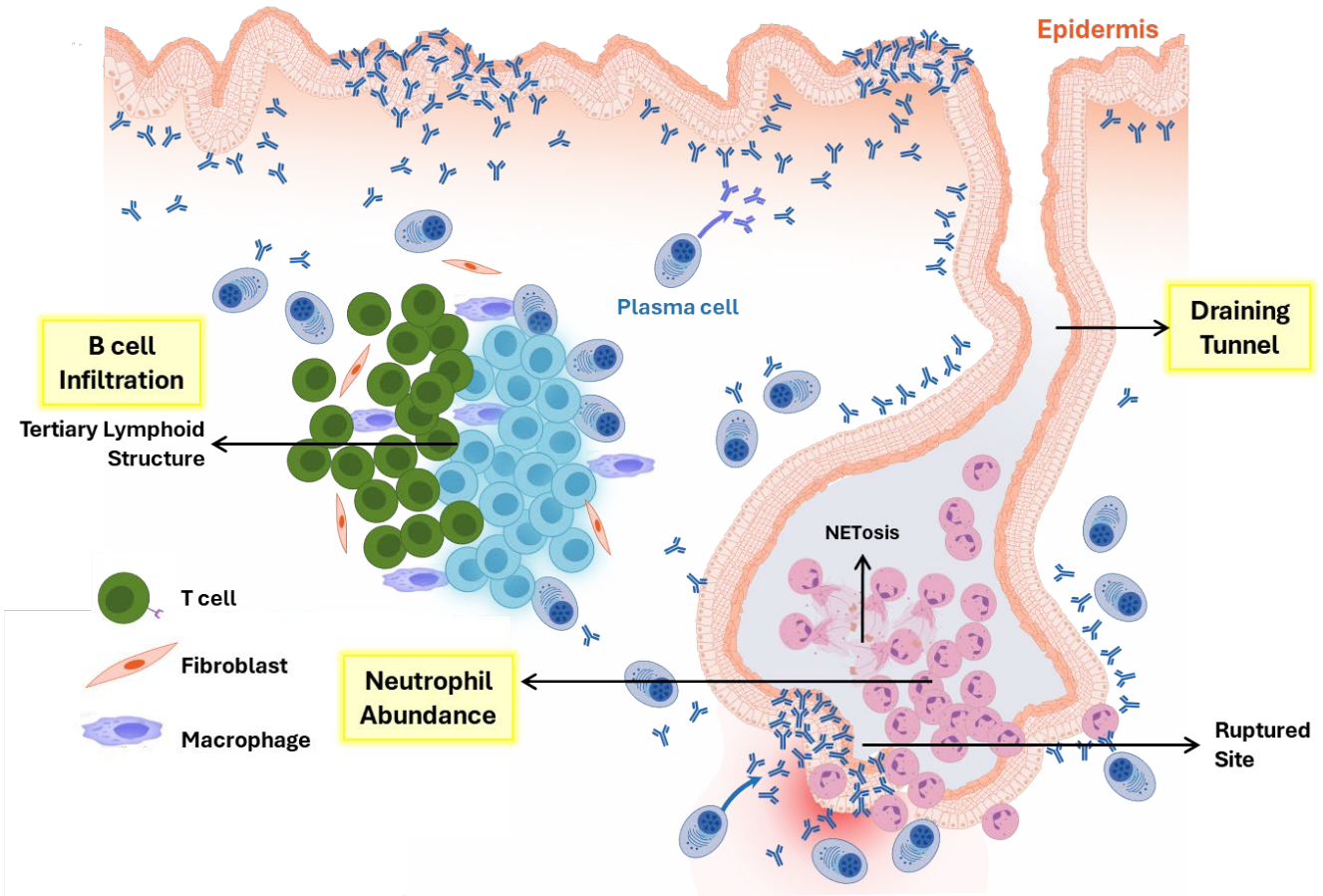
Neutrophils and B cells are rare in healthy skin but infiltrate HS lesions

Neutrophils:

- Abundant in HS lesions and sinus tracts
- Amplify acute inflammatory flares and tissue damage
- Driven in part by IL-17A–mediated recruitment and activation

B cells:

- Activated and persistent in HS lesions
- Contribute to chronic immune activation
- Supported by BAFF-mediated survival and pro-inflammatory functions



IL-17 and BAFF act through distinct pathways that drive neutrophil- and B cell–mediated immune pathology in HS

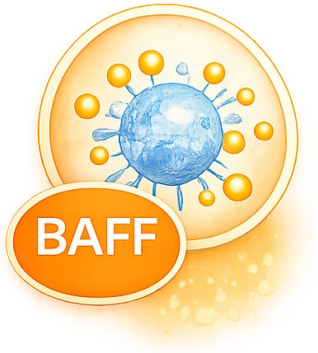
Sources: Rumberger, B.E. et al. (2020), Inflammation Research, DOI:10.1007/s00011-020-01381-7; Sabat, R. et al. (2023), Journal of Allergy and Clinical Immunology, doi:10.1016/j.jaci.2022.10.034; Macchiarella, G. et al. (2023), Journal of Investigative Dermatology, DOI:10.1016/j.jid.2022.08.051; Gudjonsson, J.E. et al. (2020), JCI Insight, doi:10.1172/jci.insight.139930; Rastrick, J. et al. (2024), British Journal of Dermatology, DOI:10.1093/bjd/ljae442.

Acronyms: BAFF, B-cell activating factor; B cell, B lymphocyte; HS, hidradenitis suppurativa; IL-17, interleukin-17; IL-17A, interleukin-17A; NETosis, neutrophil extracellular trap formation; T cell, T lymphocyte; TLS, tertiary lymphoid structure.

IL-17A– and B cell–associated pathways contribute to inflammation in HS

B cell / BAFF-associated biology

Emerging clinical evidence



- B cells and plasma cells infiltrate acute and chronic HS lesions and contribute to persistent immune activation
- BAFF expression aligns with B-cell and plasma-cell signatures in human HS tissue
- Elevated BAFF provides a biological link between B-cell accumulation and chronic immune persistence
- Clinical activity observed with multiple B cell–targeted approaches in HS, including BTK inhibition (remibrutinib) and BAFF-R inhibition (ianalumab)

IL-17A biology

Clinically validated

- Elevated IL-17 pathway activity observed in HS lesions
- Amplifies macrophage and neutrophil-driven inflammation and keratinocyte activation
- Clinical efficacy demonstrated with IL-17 pathway inhibitors

HS is characterized by IL-17A–mediated inflammation, with emerging clinical evidence supporting B cell–pathways, underscoring the approach for multi-pathway strategies

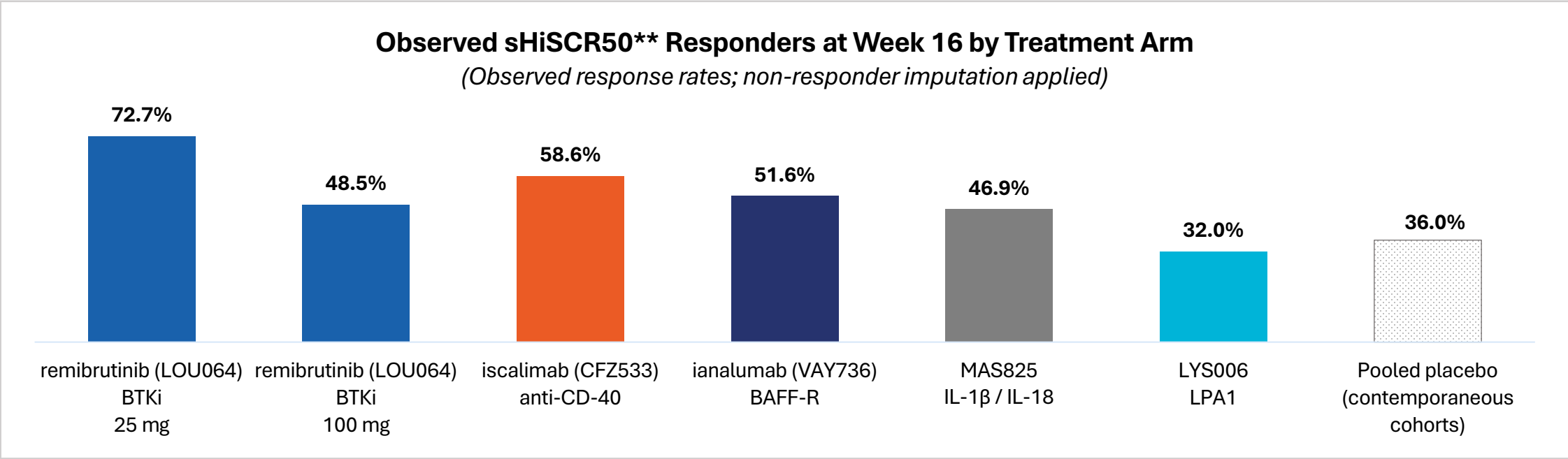
Sources: Novartis, AAD Annual Meeting 2024; ClinicalTrials.gov ID NCT03827798; Sabat, R. et al. (2023), Journal of Allergy and Clinical Immunology, DOI:10.1016/j.jaci.2022.10.034; Macchiarella, G. et al. (2023), Journal of Investigative Dermatology, DOI:10.1016/j.jid.2022.08.051.

Acronyms: BAFF, B cell activating factor; BAFF-R, B cell activating factor receptor; BTK, Bruton's tyrosine kinase; HS, hidradenitis suppurativa; IL-17, interleukin-17; IL-17A, interleukin-17A.

Increasing clinical evidence for B cell–related pathways in hidradenitis suppurativa*

Observations:

- Data show that B-cell–associated pathways (BTK, BAFF-R, CD40) demonstrate numerically higher sHiSCR50 response rates than pooled placebo in this HS platform study.
- BAFF-R inhibition (ianalumab) shows numerically meaningful observed activity, supporting a potential role for B-cell survival pathways in HS.
- IL-1β / IL-18 inhibition (MAS825) shows more modest observed activity with limited separation from pooled placebo.



(*) 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.

Sources: Novartis, AAD Annual Meeting 2024; ClinicalTrials.gov ID: NCT03827798.

Acronyms: BAFF-R, B-cell activating factor receptor; BTK, Bruton's tyrosine kinase; CD40, cluster of differentiation 40; HS, hidradenitis suppurativa; IL-1β, interleukin-1 beta; IL-18, interleukin-18; LPA1, lysophosphatidic acid receptor 1; sHiSCR50, Simplified Hidradenitis Suppurativa Clinical Response (50%).

HS landscape: multiple approved and late-stage therapies highlight ongoing need for differentiation

Company	zurabio	abbvie	NOVARTIS	ucb	MoonLake	NOVARTIS	sanofi	avalo THERAPEUTICS	abbvie	Takeda
Asset	**Tibulizumab (ZB-106)**	Adalimumab (Humira®)	Secukinumab (Cosentyx®)	Bimekizumab (Bimzelx®)	Sonelokimab	Remibrutinib	Brivekimig (SAR444245)	AVTX-009	Lutikizumab (ABT-981)	Zasocitinib (TAK-279)
Mechanism of Action	**Anti-BAFF and IL-17 bispecific mAb**	Anti-TNFα mAb	Anti-IL-17A mAb	Anti-IL-17A /F mAb	Anti-IL-17A/F Nanobody	Covalent BTK inhibitor	TNFα / OX40L bispecific mAb	Anti-IL-1β mAb	IL-1α / IL-1β mAb	TYK2 inhibitor
Stage of Development	**Phase 2**	Approved	Approved	Approved	Phase 3	Phase 3	Phase 2	Phase 2	Phase 3	Phase 2
Route of Administration	**SC**	SC	SC	SC	SC	PO	SC	SC	SC	PO
Dosing Frequency	**Q4W (studied)**	Q2W–Q4W	Q4W	Q2W → Q4W	Q2W / Q4W (studied)	QD	Q4W (studied)	Q2W–Q4W	QW–Q2W	QD

Note: Approved and development status reflect HS or broader inflammatory indications as publicly reported; dosing regimens shown represent studied or labeled schedules and may vary by program. Table is descriptive and not intended to imply direct comparison between products.

Sources: Company public disclosures, prescribing information, conference presentations, and ClinicalTrials.gov.

Acronyms: BAFF, B cell activating factor; BTK, Bruton's tyrosine kinase; HS, hidradenitis suppurativa; IL-1α, interleukin-1 alpha; IL-1β, interleukin-1 beta; IL-17, interleukin-17; IL-17A, interleukin-17A; IL-17A/F, interleukin-17A/interleukin-17F heterodimer; IV, intravenous; mAb, monoclonal antibody; OX40L, OX40 ligand; PO, oral administration; QD, once daily; QW, once weekly; Q2W, once every two weeks; Q4W, once every four weeks; SC, subcutaneous; TNFα, tumor necrosis factor alpha; TYK2, tyrosine kinase 2.

TibuSHIELD

Phase 2 study in moderate-to-severe hidradenitis suppurativa

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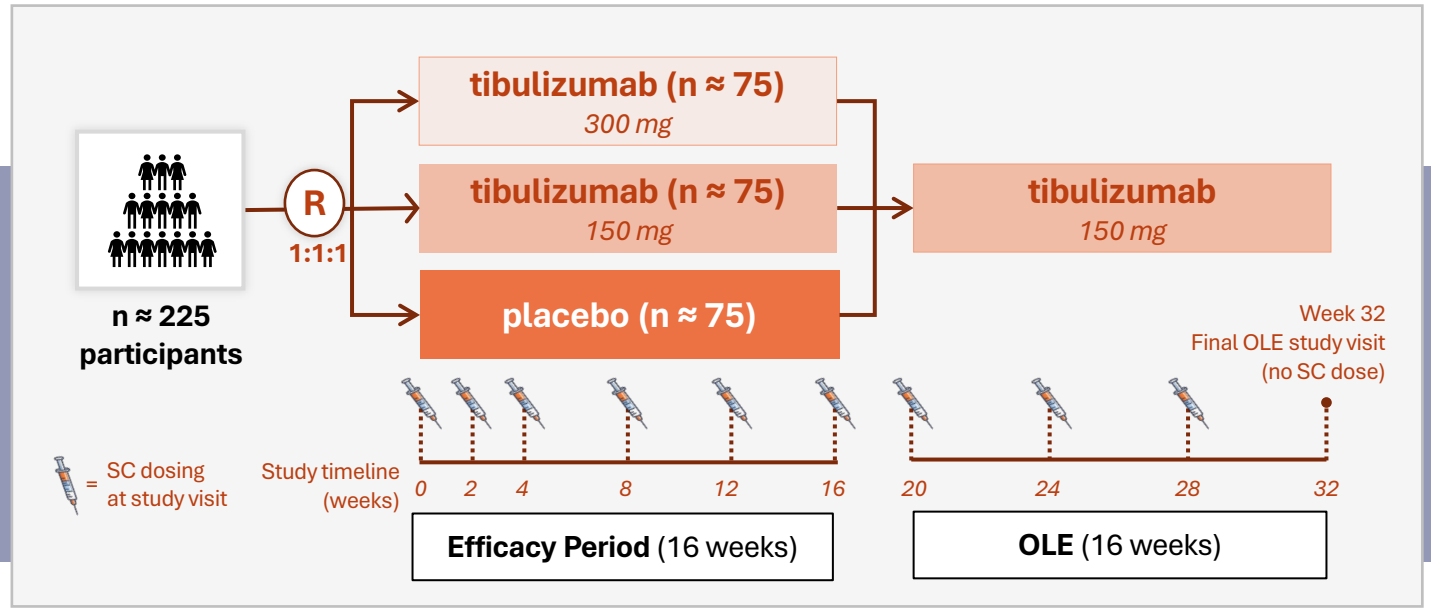


Randomized, double-blind, placebo-controlled, three-arm study
16-week primary efficacy period followed by a 16-week OLE

Tibulizumab dose: 150 mg and 300 mg SC
Dose selection informed by Phase 1 PK/PD and target engagement data

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
- Other symptom and lesion-based measures per protocol

(*) Includes secondary and exploratory endpoints

Acronyms: AN, abscess and inflammatory nodule; DLQI, Dermatology Life Quality Index; HiSCR50, Hidradenitis Suppurativa Clinical Response ≥50%; HiSCR75, Hidradenitis Suppurativa Clinical Response ≥75%; HS, hidradenitis suppurativa; IHS4, International Hidradenitis Suppurativa Severity Score System; NRS, Numeric Rating Scale; OLE, open-label extension; PK, pharmacokinetics; PD, pharmacodynamics; SC, subcutaneous; TNF-α, tumor necrosis factor alpha.

tibulizumab

ZB-106

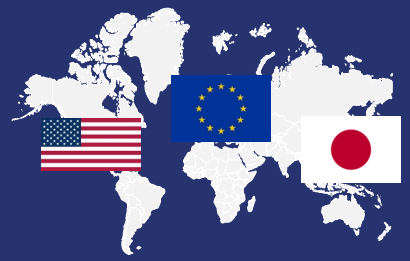
Anti-BAFF + IL-17

A dual-pathway approach in SSc

- *First and currently only in-class bispecific antibody designed to target BAFF and IL-17 signaling, including IL-17A and IL-17A/F*
- *Phase 2 clinical trial (TibuSURE) ongoing; topline data anticipated in 1H 2027*

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

Systemic Sclerosis (SSc) is a Rare, Serious Multisystem Autoimmune Disease

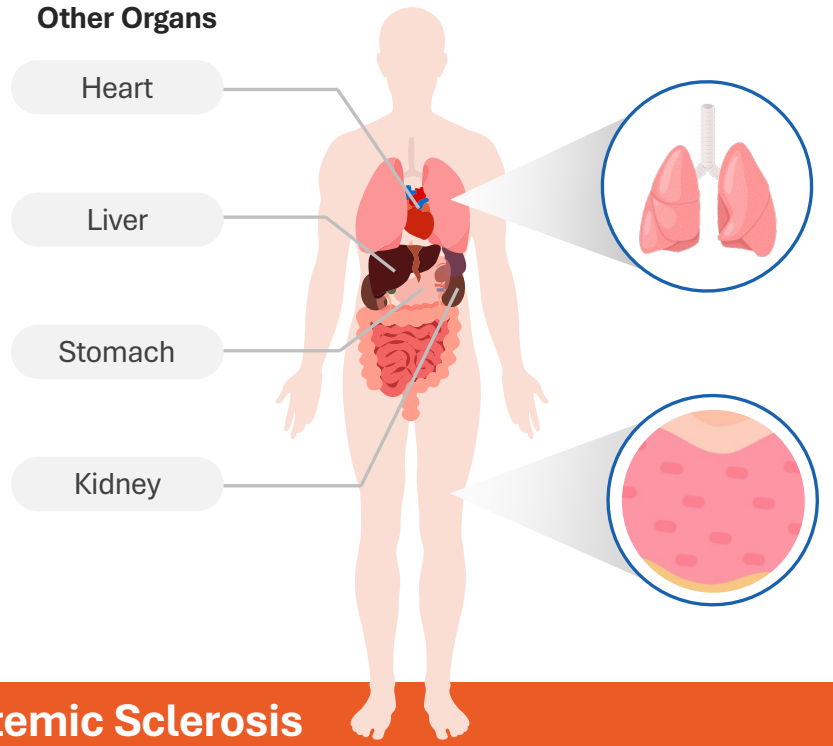


~300,000

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

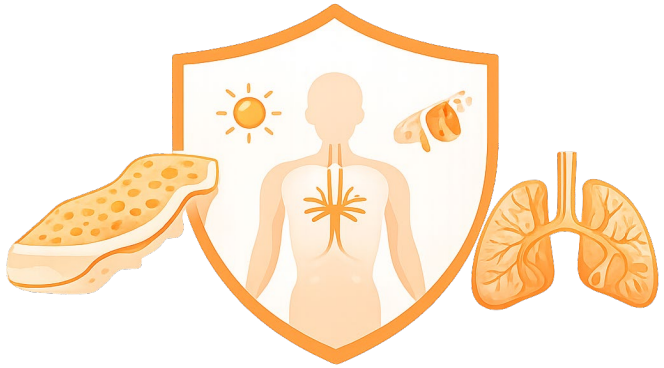
TibuSURE Phase 2 Trial Evaluates Key Domains of Systemic Sclerosis

FVC	mRSS	HAQ-DI	PtGA	CGA
Forced Vital Capacity	modified Rodnan Skin Score	Health Assessment Questionnaire Disability Index	Patient Global Assessment	Clinical Global Assessment
Lung Function	Skin Fibrosis	Functional Impact	Patient-Reported Outcome	Clinician-Reported Outcome

(*) 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.
Acronyms: CGA, Clinical Global Assessment; EU5, five major European markets (France, Germany, Italy, Spain, United Kingdom); FVC, forced vital capacity; HAQ-DI, Health Assessment Questionnaire Disability Index; ILD, interstitial lung disease; mRSS, modified Rodnan Skin Score; PtGA, Patient Global Assessment; SSc, systemic sclerosis; US, United States.

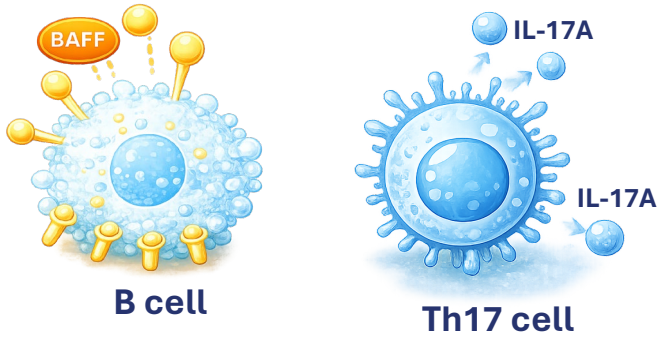
A dual-pathway approach in systemic sclerosis

Targeting immune drivers of autoimmunity, inflammation, and fibrosis



Disease

- Severe, progressive, multisystem autoimmune disease
- Core features: inflammation, vasculopathy, and fibrosis
- No therapies are approved that comprehensively address the multisystem pathology of SSc*



Biology

- Inflammation plays a key role in fibrosis and vasculopathy
- Autoreactive B cells and autoantibodies are key inflammatory signature
- IL-17 pathway elevation observed in SSc patients

Program

- Tibulizumab (BAFF + IL-17A bispecific antibody)
- Phase 2 TibuSURE clinical study; topline data expected 1H 2027

(*) Two therapies are approved for SSc-ILD; however, no treatment approved for SSc addresses multiple organ systems.
Acronyms: BAFF, B cell activating factor; IL-17A, interleukin-17A; SSc, systemic sclerosis; Th17, T helper 17 cells.

IL-17A and BAFF/B cells contribute to inflammation, vasculopathy, and fibrosis in systemic sclerosis

Core Disease Features

- SSc is characterized by chronic inflammation, vasculopathy, and progressive fibrosis
- Infiltration of activated immune cells (macrophages, T cells, B cells) creates a persistent inflammatory tissue environment

BAFF / B Cell Biology

- BAFF is elevated in SSc and supports survival and activation of autoreactive B cells
- Activated B cells produce autoantibodies and inflammatory cytokines (e.g., IL-6) associated with fibrosis and vascular dysfunction

IL-17A Biology

- IL-17A has been reported to be elevated in subsets of patients with SSc
- Preclinical studies suggest IL-17A can activate fibroblasts and endothelial cells, promoting inflammation, immune cell recruitment, and tissue remodeling

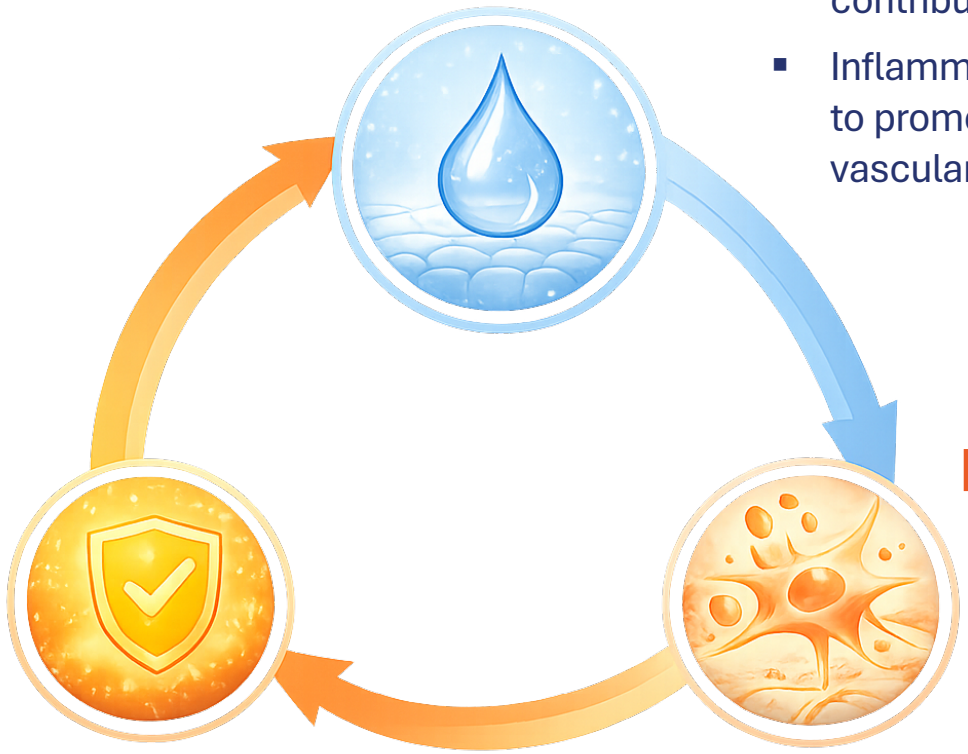
Sources: Seki, N. et al. (2024), Cytokine, DOI:10.1016/j.cyto.2024.156534; Ono, Y. et al. (2024), Scientific Reports, DOI:10.1038/s41598-024-76987-6; Lonati, P.A. et al. (2014), PLoS ONE, DOI:10.1371/journal.pone.0105008; Deng, C.-C. et al. (2025), Frontiers in Immunology, DOI:10.3389/fimmu.2024.1522076; Kurasawa, K. et al. (2000), Arthritis & Rheumatism, 43(11), pp. 2455–2463. doi:10.1002/1529-0131(200011)43:11<2455::aid-anr12>3.0.co;2-k.

Acronyms: BAFF, B cell activating factor; IL-6, interleukin-6; IL-17A, interleukin-17A; SSc, systemic sclerosis.

Targeting interconnected immune pathways in systemic sclerosis

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



INFLAMMATION

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

FIBROTIC REMODELING

- Fibroblast activation contributes to progressive tissue fibrosis
- Excess extracellular matrix deposition (e.g., collagen) is characteristic of SSc

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

Sources: Allanore, Y. et al. (2015), Nature Reviews Disease Primers, DOI:10.1038/nrdp.2015.2; Distler, J.H. et al. (2019), Nature Reviews Rheumatology, DOI:10.1038/s41584-019-0322-7; Asano, Y. and Sato, S. (2015), Seminars in Immunopathology, DOI:10.1007/s00281-015-0505-5; Varga, J. and Abraham, D. (2007), Journal of Clinical Investigation, DOI:10.1172/jci31139.

Acronyms: BAFF, B cell activating factor; IL-6, interleukin-6; IL-17, interleukin-17; SSc, systemic sclerosis.

Separately inhibiting IL-17A or BAFF has shown efficacy in placebo-controlled trials for systemic sclerosis

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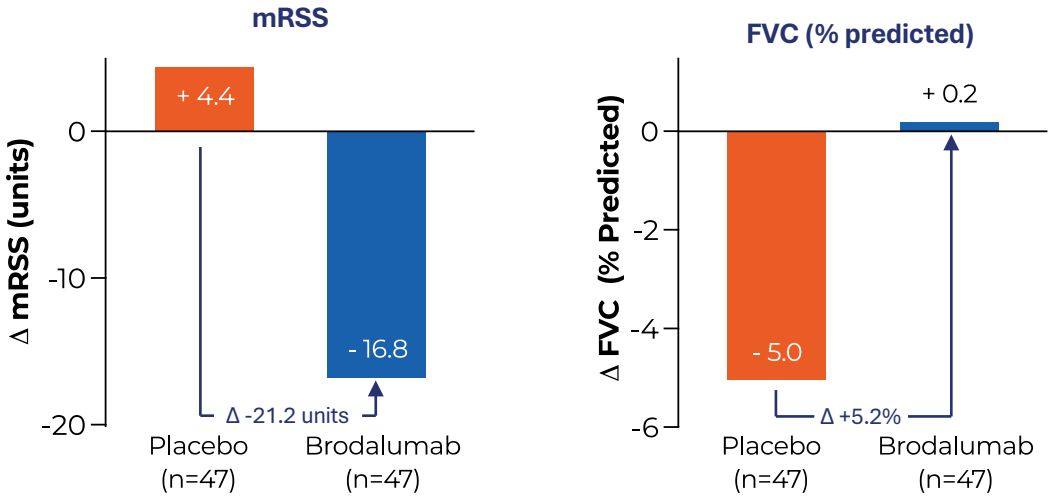
IL-17 RECEPTOR ANTAGONIST – PHASE 3

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

CLINICAL PRECEDENT

Phase 3 brodalumab trial (24 weeks)



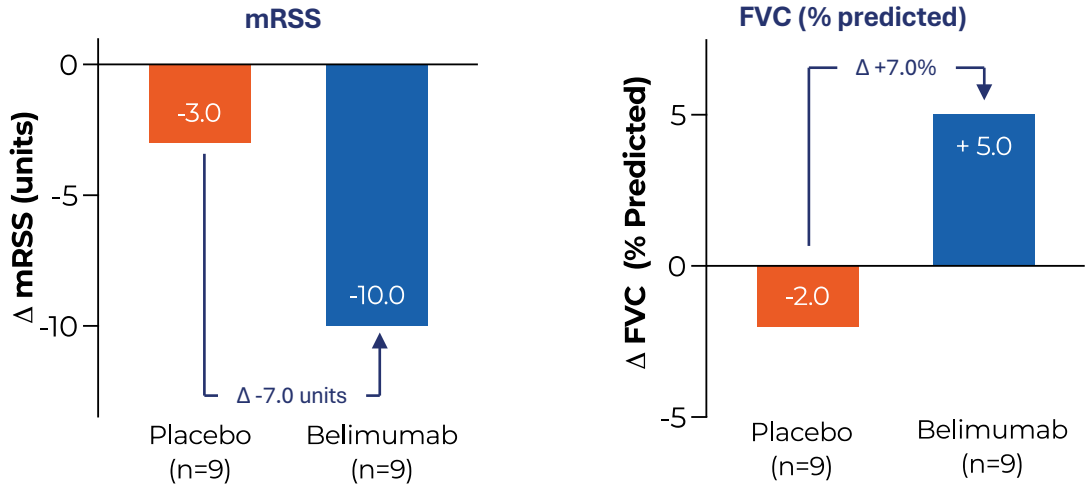
BAFF ANTAGONIST

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

CLINICAL PRECEDENT

Phase 2 belimumab IIT trial (52 weeks)



Sources: Fukasawa, T. et al. (2022), Annals of the Rheumatic Diseases, doi:10.1136/annrheumdis-2022-eular.2519; ClinicalTrials.gov ID NCT03957681; Gordon, J.K. et al. (2018), Arthritis & Rheumatology, DOI:10.1002/art.40358; ClinicalTrials.gov ID NCT01670565.

Acronyms: BAFF, B cell activating factor; dcSSc, diffuse cutaneous systemic sclerosis; FVC, forced vital capacity; IL-17A, interleukin-17A; IIT, investigator-initiated trial; mRSS, modified Rodnan Skin Score; MMF, mycophenolate mofetil; NS, not significant; QOL, quality of life; SHAQ-DI, Scleroderma Health Assessment Questionnaire–Disability Index; SSc, systemic sclerosis; VAS, visual analog scale.

TibuSURE Phase 2 study in diffuse cutaneous systemic sclerosis

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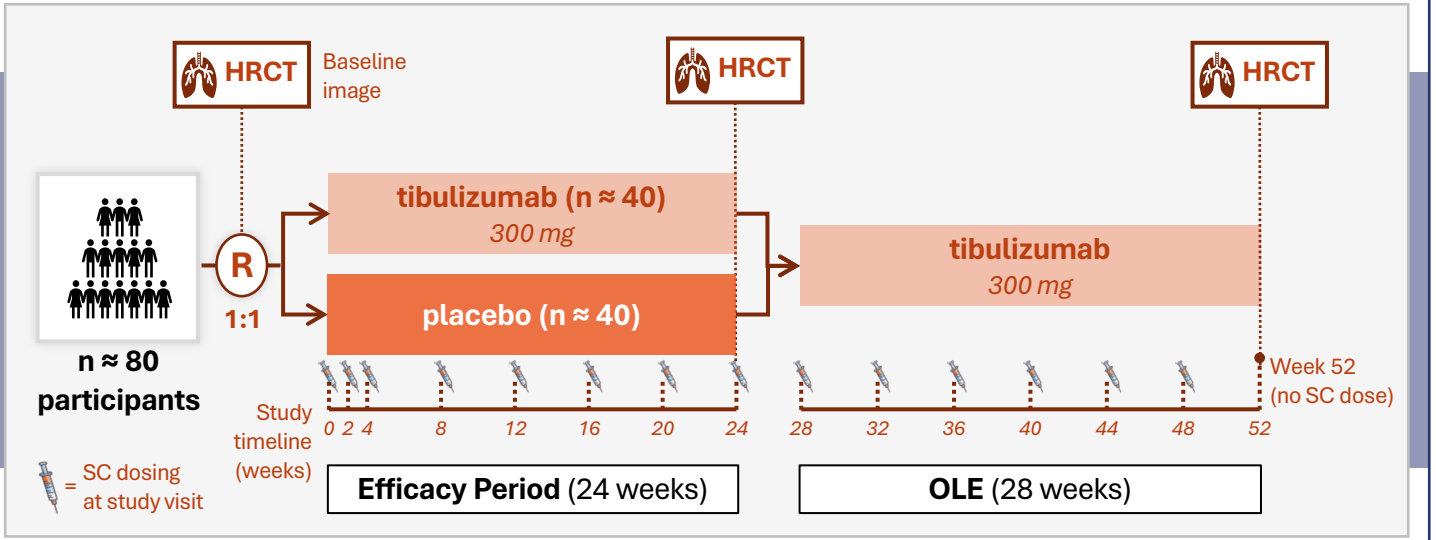


Randomized, double-blind, placebo-controlled design
24-week primary efficacy period, followed by 28-week OLE

Tibulizumab dose: 300 mg SC
Dose selection informed by Phase 1 PK/PD and target engagement data

KEY INCLUSION CRITERIA

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

PLANNED EFFICACY ENDPOINTS

PRIMARY ENDPOINT

- Change from baseline in mRSS at Week 24

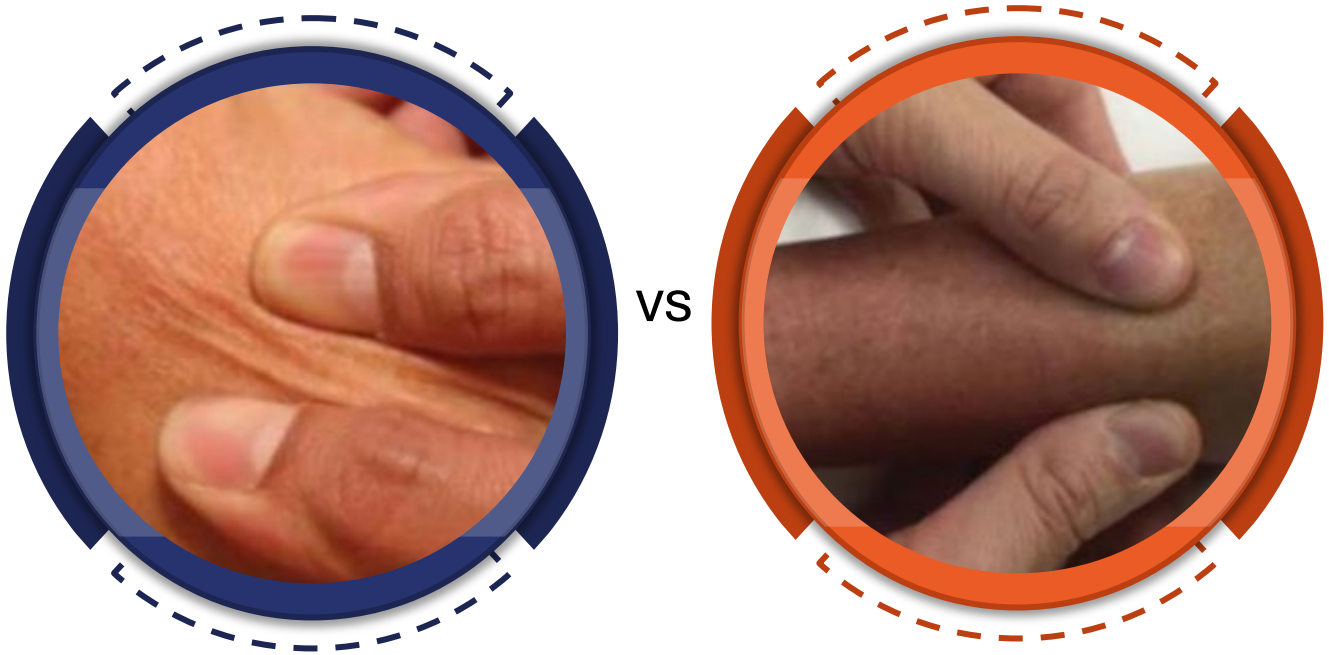
ADDITIONAL ENDPOINTS*

- qHRCT lung imaging
- FVC
- HAQ-DI
- revised CRISS (rCRISS)

(*) Includes secondary and exploratory endpoints

Acronyms: CRISS, Combined Response Index in Systemic Sclerosis; FVC, forced vital capacity; HAQ-DI, Health Assessment Questionnaire Disability Index; HRCT, high-resolution computed tomography; ILD, interstitial lung disease; mRSS, modified Rodnan Skin Score; OLE, open-label extension; PK/PD, pharmacokinetics/pharmacodynamics; qHRCT, quantitative high-resolution computed tomography; R, randomization; SC, subcutaneous; SSc, systemic sclerosis.

modified Rodnan Skin Score (mRSS): Endpoint for assessing skin thickness and fibrosis



Fine Wrinkles (0/3)

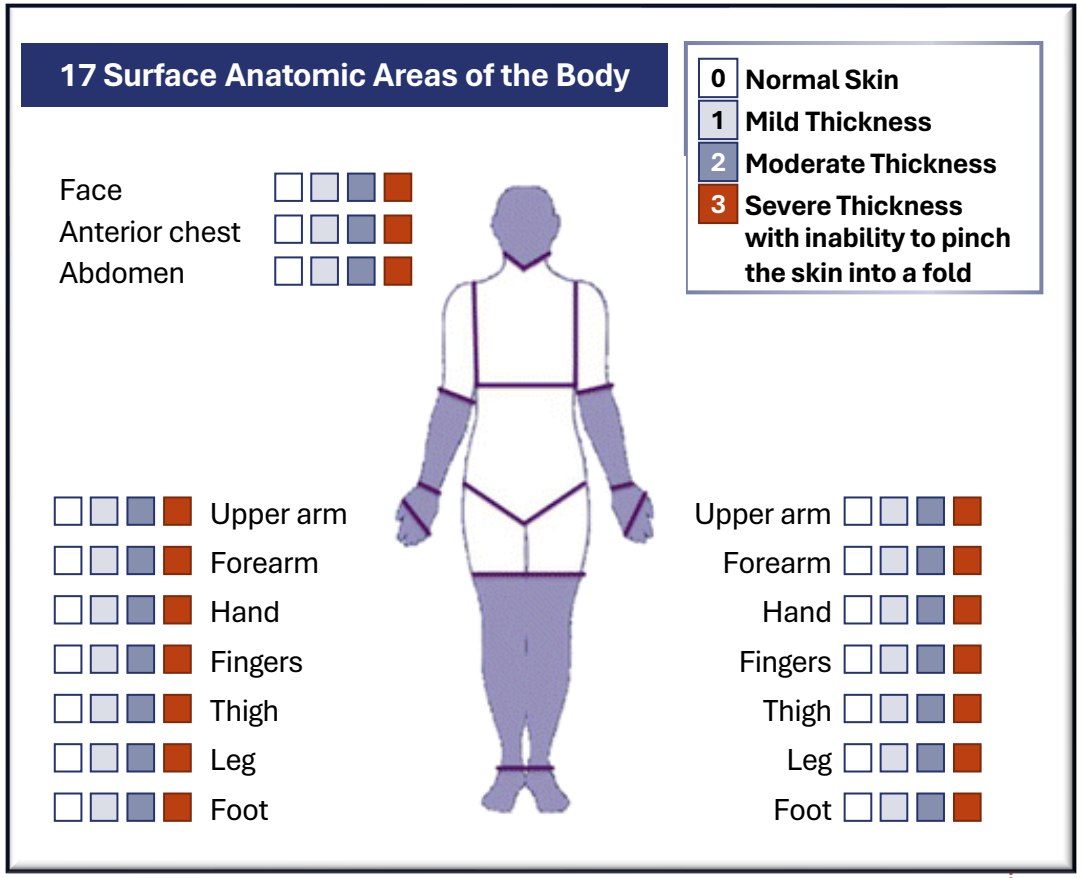
vs

Severe Thickness (3/3)

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

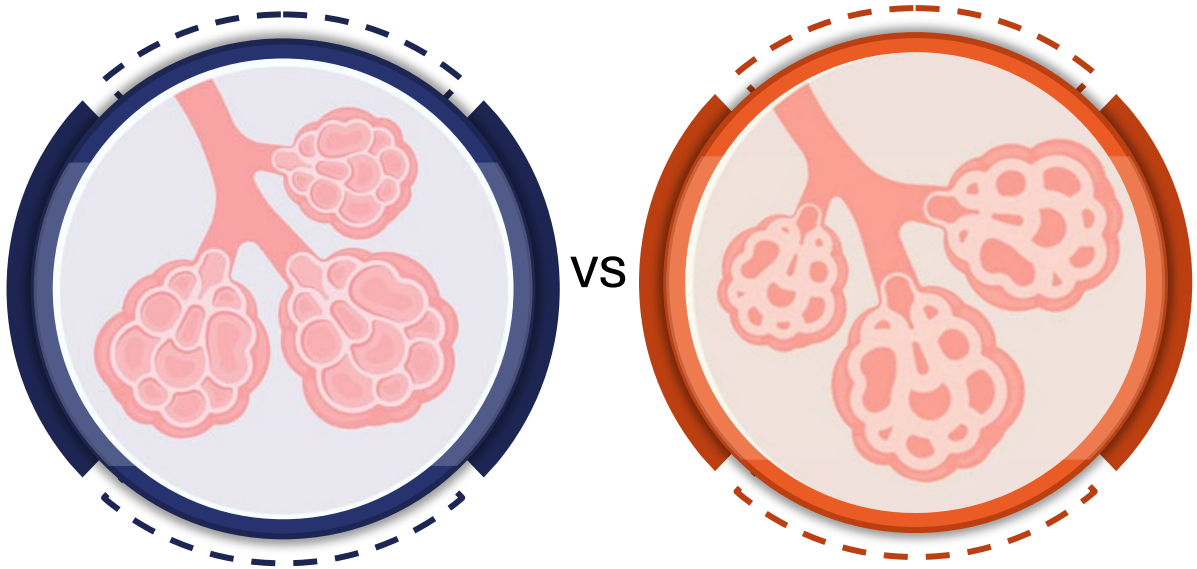
The **mRSS assesses skin thickness** in systemic sclerosis patients by **evaluating 17 body sites** (e.g., face, chest, abdomen, arms, legs). Each site is scored from 0 to 3.

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



Sources: Khanna, D. et al. (2017), Journal of Scleroderma and Related Disorders, doi:10.5301/jsrd.5000231; Ferreli, C. et al. (2018), Clinical Reviews in Allergy & Immunology, doi:10.1007/s12016-017-8625-4.

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



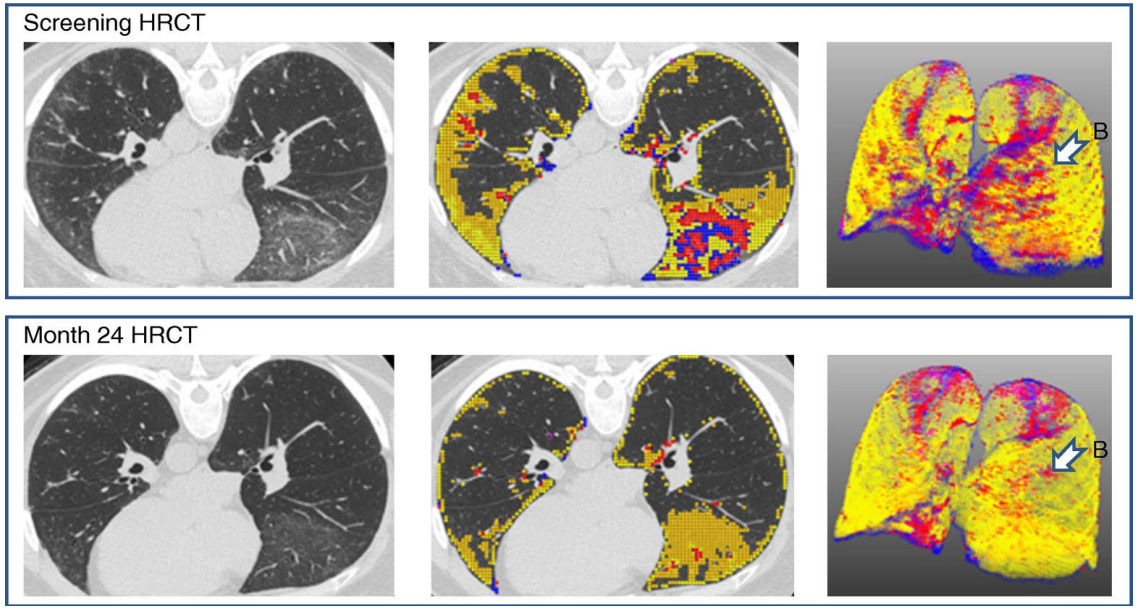
Healthy Lung **Fibrotic Lung**

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



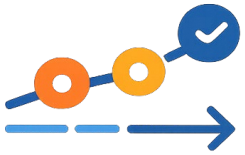
The blue and red areas show QLF, while the yellow area shows quantitative ground glass. The entire colored area represents QILD. After 24 months, QLF areas decreased (*arrow in B*).

Sources: Goldin, J. et al. (2018), Annals of the American Thoracic Society, doi:10.1513/annalsats.201802-079OC; Zura Bio internal planning.
Acronyms: ILD, interstitial lung disease; MMF, mycophenolate mofetil; qHRCT, quantitative high-resolution computed tomography; QILD, quantitative interstitial lung disease; QLF, quantitative lung fibrosis; SSc, systemic sclerosis.

Key takeaways

Clear differentiation

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



Defined, anticipated near-term clinical catalysts

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

Platform optionality

Multi-pathway immune biology provides potential for expansion into additional autoimmune indications, subject to clinical validation

Acronyms: BAFF, B cell activating factor; IL-17, interleukin-17.