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Corporate Overview

August 2026

DEVELOPING NOVEL MEDICINES FOR SERIOUS IMMUNE SYSTEM DISORDERS

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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[5]) or BAFF arm (SSc[6]) with strong scientific support for the orthogonal pathway[7,8,9]

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

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

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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® *Prescribing Information.*; 2. Benlysta® *Prescribing Information.*; 3. Kimball et al. *Lancet.* 2024; 4. Kimball et al. *Lancet.* 2023; 5. Stone et al. *N Engl J Med.* 2026 (REPLENISH); 6. Gordon et al. *Arthritis Rheumatol.* 2018; 7. Sabat et al. *J Allergy Clin Immunol.* 2023; 8. Lowe et al. *JCI Insight.* 2024; 9. van der Geest et al. *Rheumatology (Oxford).* 2015;10. Zura Bio filings.

Addressing the distinct biology of immune-mediated diseases

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

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

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

Untapped market potential across disease areas of interest

TODAY:

Biologic penetration in HS, SSc, and PMR remains below average[1] because current therapies only partially address the multi-pathway biology driving these diseases[2–9]

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[10–13]

Both circles represent combined HS, SSc, and PMR global markets[1]

Sources: 1. 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; 2. Kimball et al. *Lancet*. 2024 (BE HEARD I/II); 3. Kimball et al. *Lancet*. 2023 (SUNSHINE/SUNRISE); 4. Kimball et al. *N Engl J Med*. 2016 (PIONEER I/II); 5. Stone et al. *N Engl J Med*. 2026 (REPLENISH); 6. Spiera et al. *N Engl J Med*. 2023 (SAPHYR); 7. Khanna et al. *Lancet Respir Med*. 2020 (focuSSced); 8. Distler et al. *N Engl J Med*. 2019 (SENSCIS); 9. Fukasawa et al. *J Am Acad Dermatol*. 2023; 10. Gordon et al. *Lancet*. 2021 (BE READY); 11. Gordon et al. *Lancet*. 2018 (UltIMMa 1/2); 12. McInnes et al. *Lancet*. 2023 (BE OPTIMAL/BE COMPLETE); 13. Mease et al. *N Engl J Med*. 2015 (FUTURE 1).

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

A Phase 2 trial in adults with HS

TibuSHIELD HS

Anticipated readout:

Q4 2026

Enrollment complete; overenrolled

A Phase 2 trial in adults with SSc

TibuSURE SSc

Anticipated readout:

H1 2027

Enrollment complete; overenrolled

A Phase 2 trial in adults with PMR

NEXUS|PMR

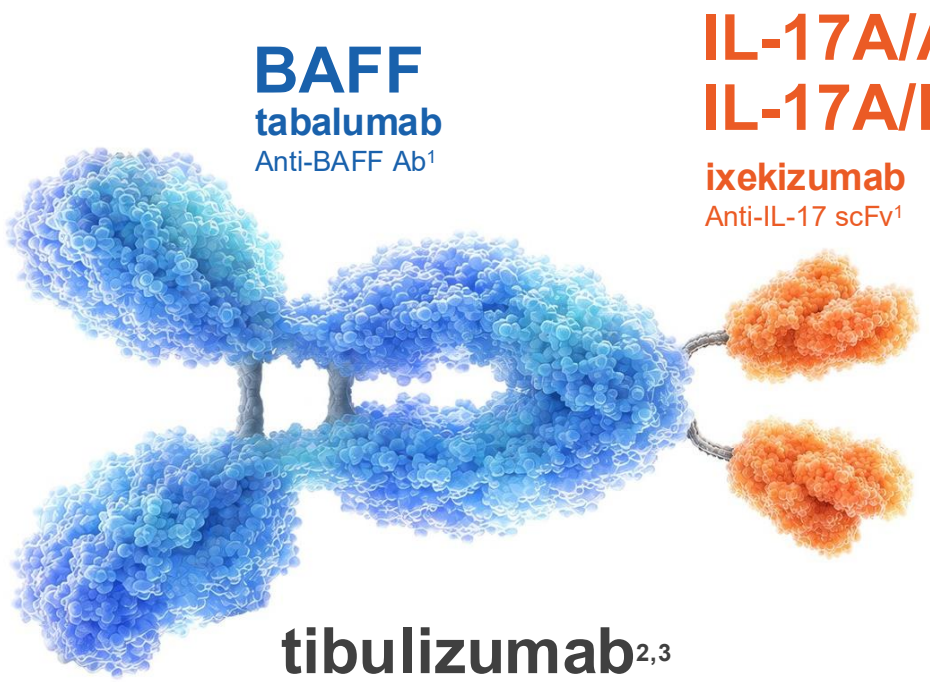
Anticipated initiation:

YE 2026

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[2,3]

Tetravalent bispecific antibody designed to block BAFF and IL-17–mediated signaling (IL-17A/A and A/F heterodimers)[4]

Addresses disease complexity

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

Clinically grounded design

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

Single-entity advantage

Enables dual-pathway modulation without multi-drug combination complexity

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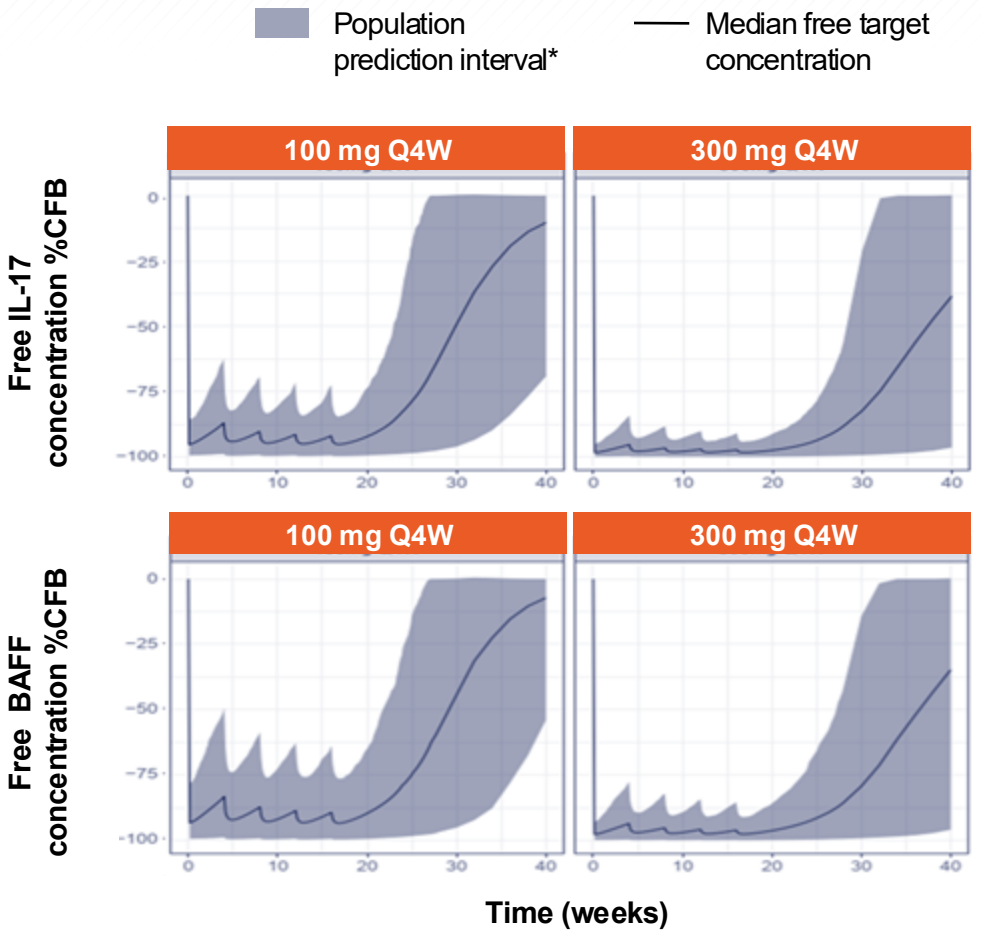
Tibulizumab is an investigational agent. Its efficacy and safety have not been established or approved by the FDA or any other regulatory agency worldwide.

Sources: 1. Publicly available information on ixekizumab and tabalumab; 2. ClinicalTrials.gov. *NCT06993610.*; 3. ClinicalTrials.gov. *NCT06843239.*; 4. Benschop et al. *mAbs*. 2019.

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

Tibulizumab is an investigational agent. Its efficacy and safety have not been established or approved by the FDA or any other 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 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 and adaptive immune drivers of disease

BAFF

Promotes autoreactive B cell survival and function[1,2,3]

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

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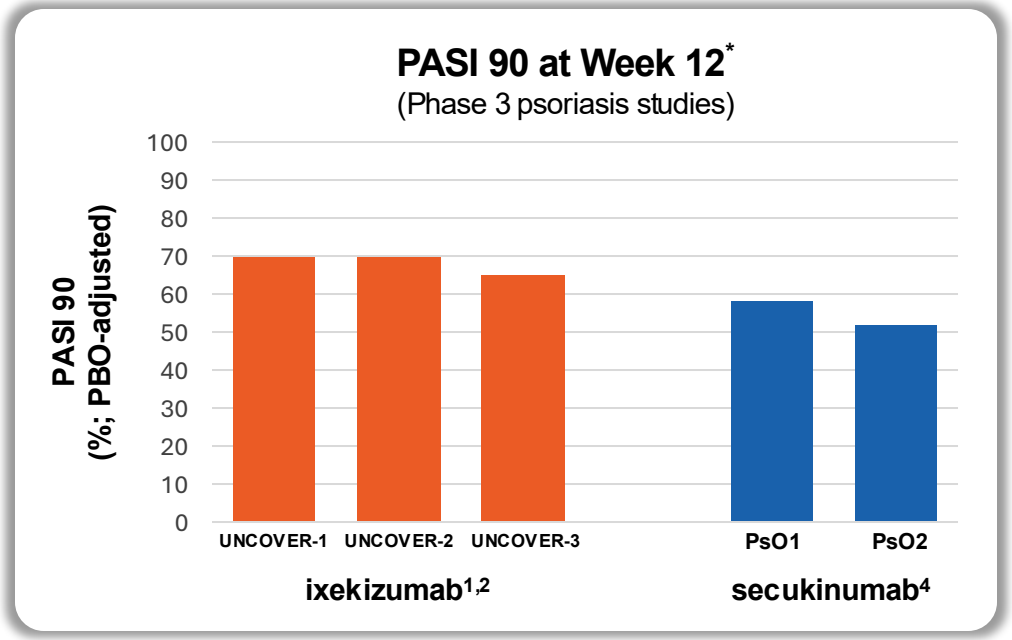
Schematic representation of mechanism rationale. Not a clinical efficacy claim. References for B cell axis in HS: 1. Sabat et al. *J Allergy Clin Immunol.* 2022; 2. Lowe et al. *JCI Insight.* 2023; 3. Gudjonsson et al. *JCI Insight.* 2020.

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*[1,2]
- Validated IL-17 pathway inhibition: Neutralization of IL-17A/A and IL-17A/F produces robust clinical responses with a well-characterized safety profile[1,2,3]
- Extensive clinical safety experience: Supported by large global clinical development programs and post-marketing datasets across dermatologic and rheumatologic indications*[3]
- Low incidence of candidiasis relative to IL-17F/F antagonists



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

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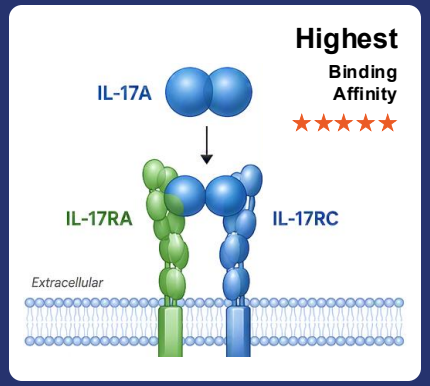
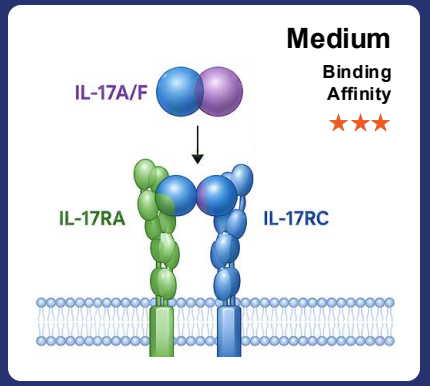
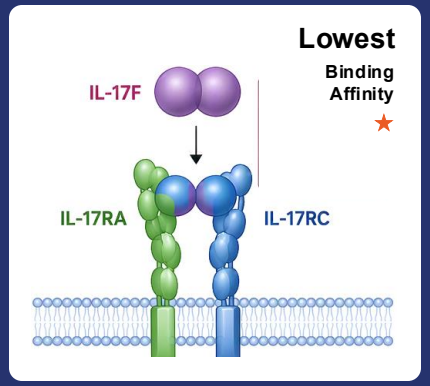
* PASI 90 values represent placebo-adjusted response rates at Week 12 from independent Phase 3 psoriasis trials. Studies were not designed for head-to-head comparison; trial designs, patient populations, dosing regimens, and placebo response rates differed. Clinical efficacy data shown are limited to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

Sources: 1. Griffiths CE et al. *Lancet*. 2015; 2. Gordon KB et al. *N Engl J Med*. 2016; 3. Eli Lilly and Company and Novartis. Public disclosures and prescribing information.; 4. Langley RG et al. *N Engl J Med*. 2014

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

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Sources: 1. Goedken ER et al. *J Pharmacol Exp Ther.* 2025; 2. Patel DD et al. *Ann Rheum Dis.* 2013; 3. Adams R et al. *Front Immunol.* 2020.

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[1]

Patients withdraw from social interactions due to visible symptoms

Embarrassment drives social withdrawal, relationship strain, absenteeism, mental health consequences[2]

Patients struggle with persistent drainage and odor from lesions

Managing chronic drainage and odor becomes burdensome, leading to avoidance of exposure or hiding[1]

Patients experience prolonged delays in diagnosis and treatment

Delayed diagnosis leads to continued symptom burden and progression to more severe disease[3]

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

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

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[1,2]

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)[3,4]

Surgery

Reserved for biologic-refractory or structurally advanced patients with extensive tunneling and scarring that no longer respond to medical therapy[1,2]

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[3,5]

Patient impact remains high

Pain and quality of life impairment can persist even when objective measures improve[6]

Structural damage is irreversible

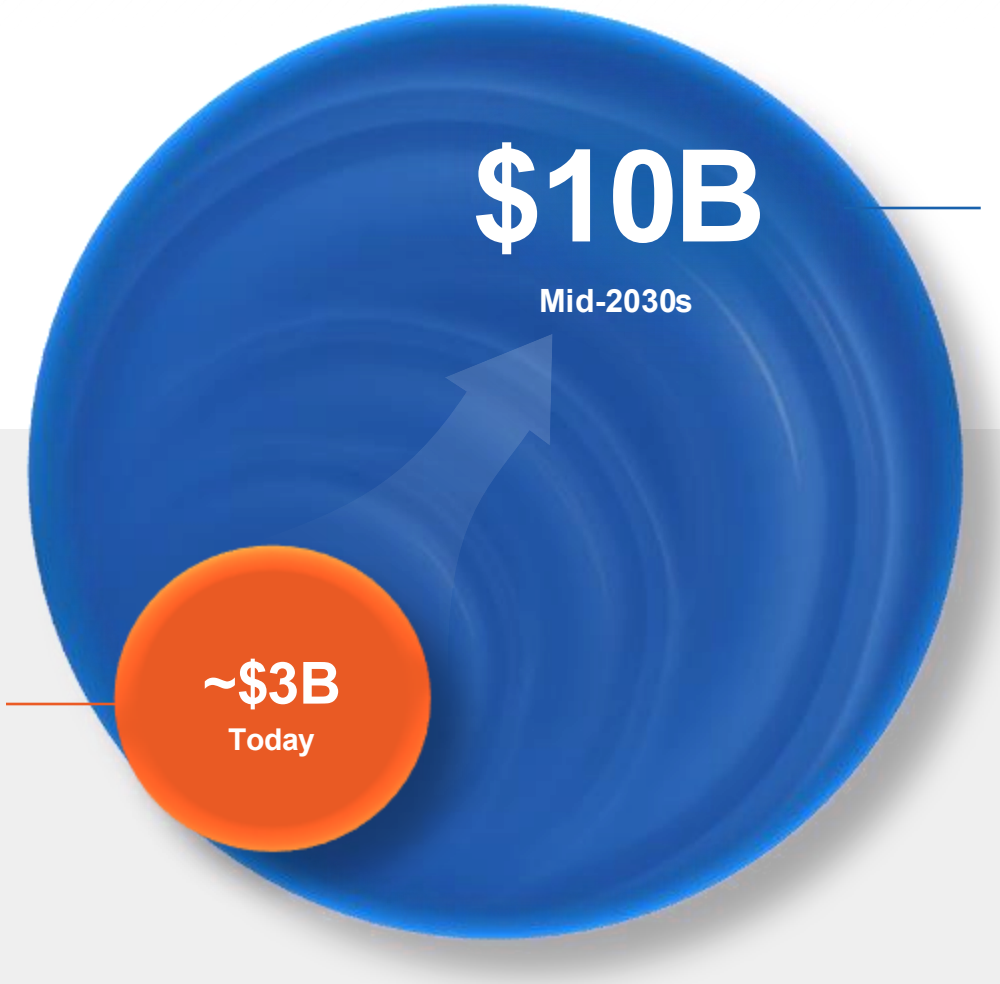
Once disease progresses to tunnels and scarring, no current therapy can reverse it[1]

Physicians remain only moderately satisfied

Despite current advancements, physicians desire new mechanisms of action that provide improved efficacy, deeper and more durable response[4]

Sources: 1. Zouboulis et al. *Lancet*. 2025; 2. Alikhan et al. *J Am Acad Dermatol*. 2019; 3. Kimball et al. *Lancet*. 2024 (BE HEARD I&II); 4. Zura Bio. *Data on file*. 2026; 5. Garbayo-Salmons et al. *J Eur Acad Dermatol Venereol*. 2025; 6. Ingram et al. *Pain Ther*. 2025.

Hidradenitis suppurativa: large, underserved market with high unmet need



Multiple advanced therapies; cycling to innovative options expected to increase as physicians seek durable alternatives

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

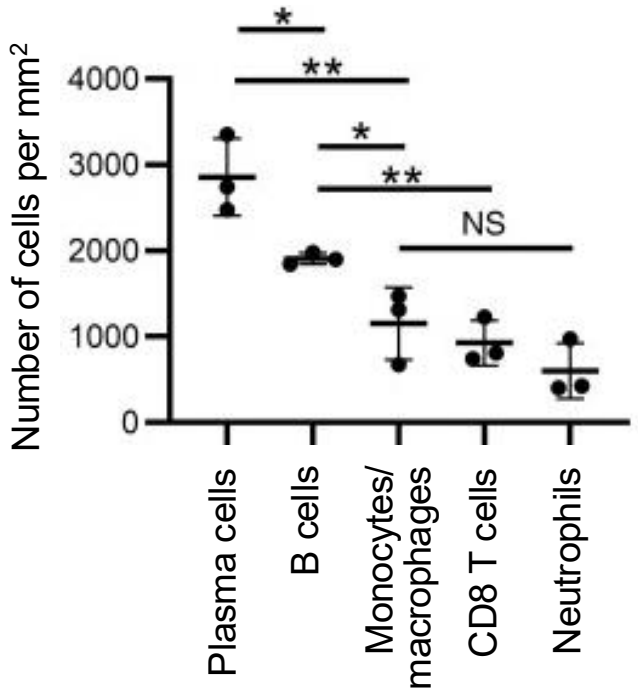
Growth Drivers and Unmet Needs

- 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

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 in 2026 to ~$10B by the mid-2030s) are based on internal market research and company analyses. All figures are approximate.

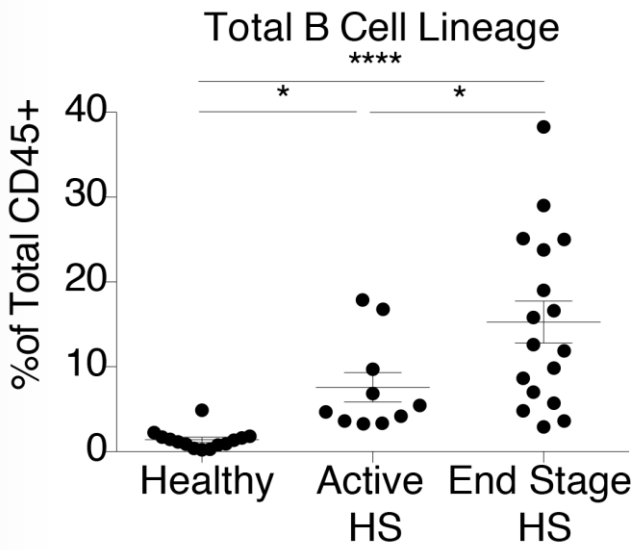
Unlike healthy skin, B cells infiltrate and chronically persist in HS lesions, driving a local amplified and targeted immune response

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



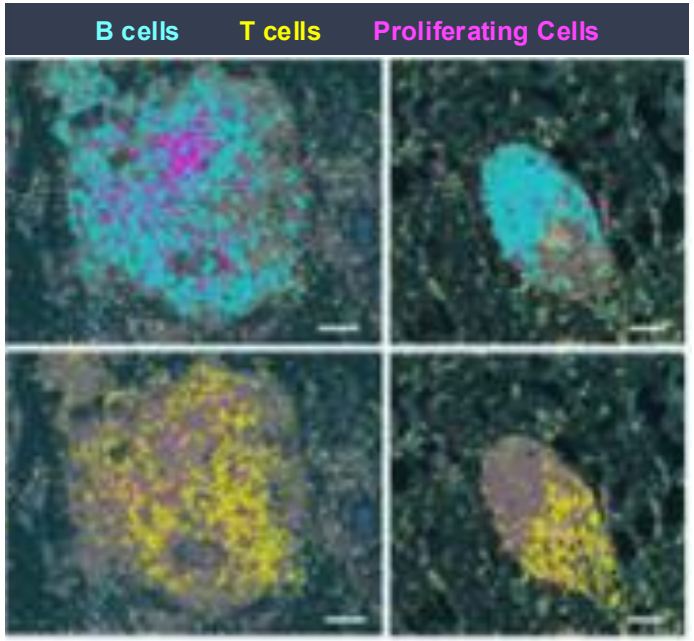
Imaging CyTOF analysis of HS lesions

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

Sources: 1. Gudjonsson et al. *JCI Insight.* 2020; 2. Lowe et al. *JCI Insight.* 2020; 3. Lowe et al. *JCI Insight.* 2024; 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

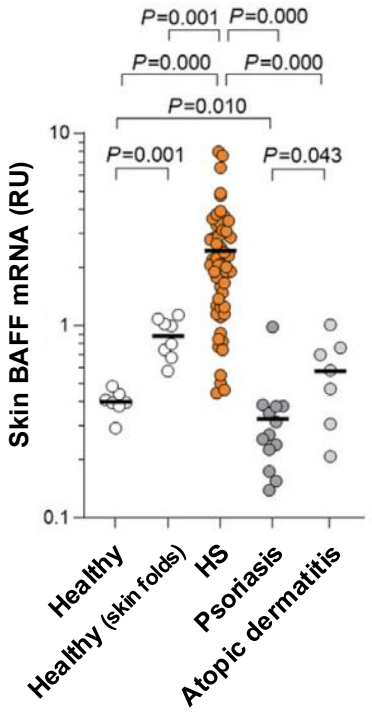
B cells migrate into HS lesions and initiate effector functions[1]

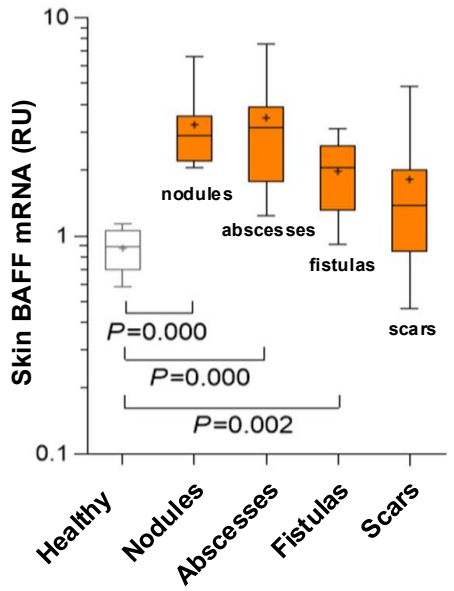
BAFF promotes B cell accumulation, maturation, and differentiation

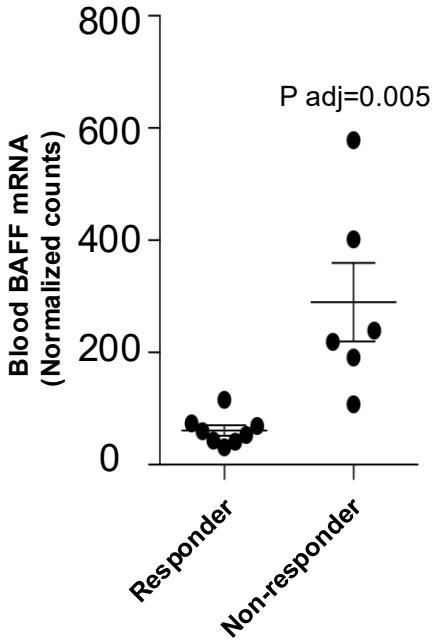
B and T cells organize into tertiary lymphoid structures that drive chronic, local immune activation[1,2]

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

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



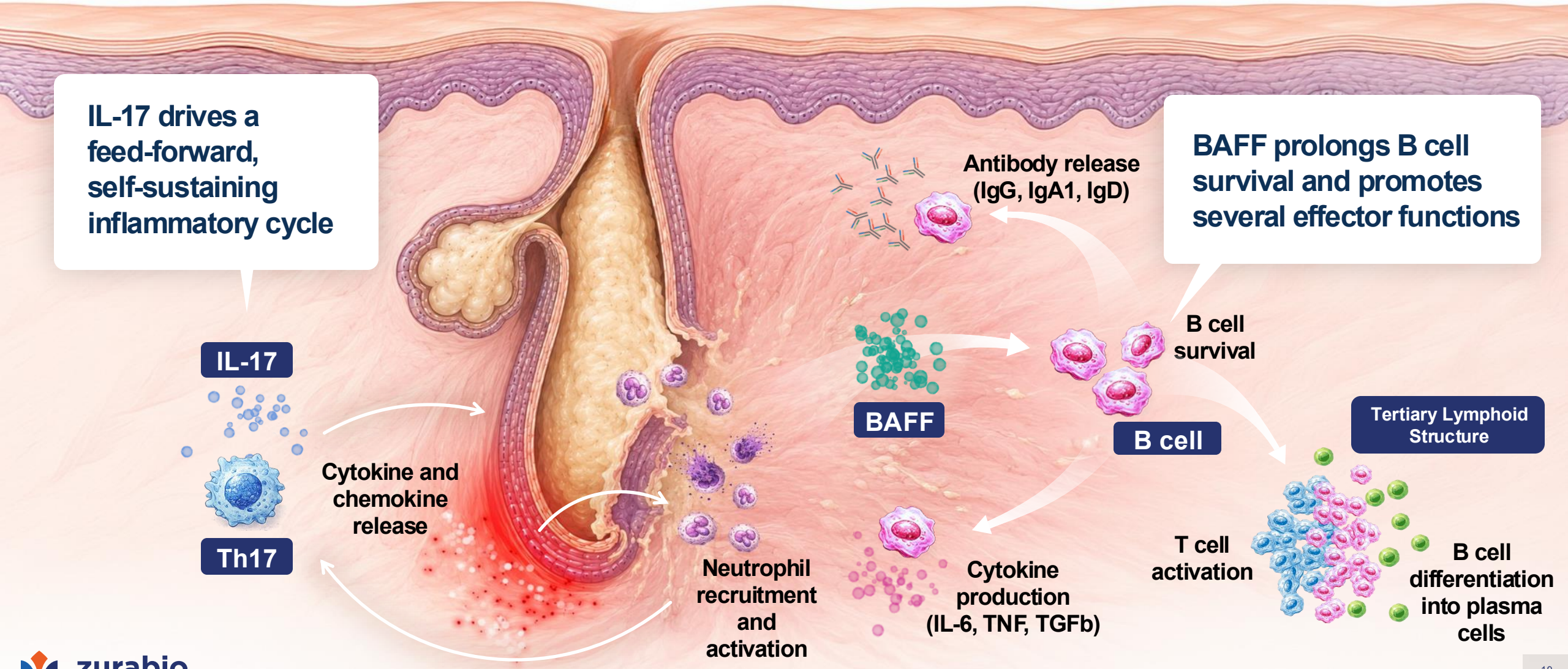




In acute and chronic HS lesions, BAFF expression tracks with B cell accumulation, supporting BAFF as a key player in B cell mediated pathology

Sources: 1. Yu et al. *Immunity*. 2024 2. Gudjonsson et al. *JCI Insight*. 2020; 3. Sabat et al. *J Allergy Clin Immunol*. 2023; 4. Lowe et al. *JCI Insight*. 2020; 5. Hambly et al. *Br J Dermatol*. 2022.

B cells are believed to drive HS pathology via multiple mechanisms



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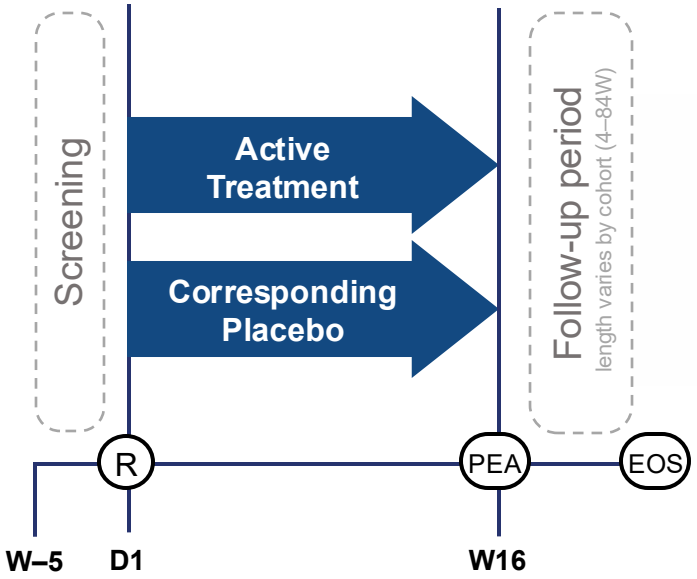
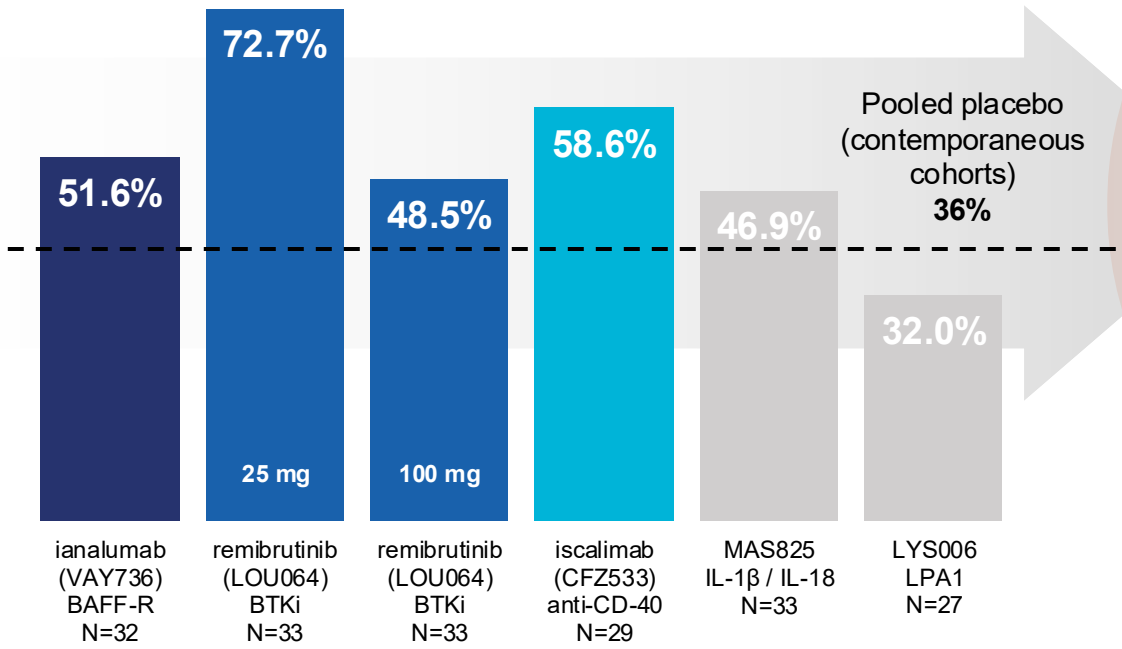
Recent clinical data supports the hypothesis that B cells are key drivers of disease rather than bystanders in HS*

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.[1]

Observed sHiSCR50** Responders at Week 16 by Treatment Arm[1,2]



Three B cell-targeting mechanisms demonstrating HS efficacy signal[1,2]

(*) 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: 1. ClinicalTrials.gov. *NCT03827798*. 2026; 2. Novartis. AAD Annual Meeting. 2024.

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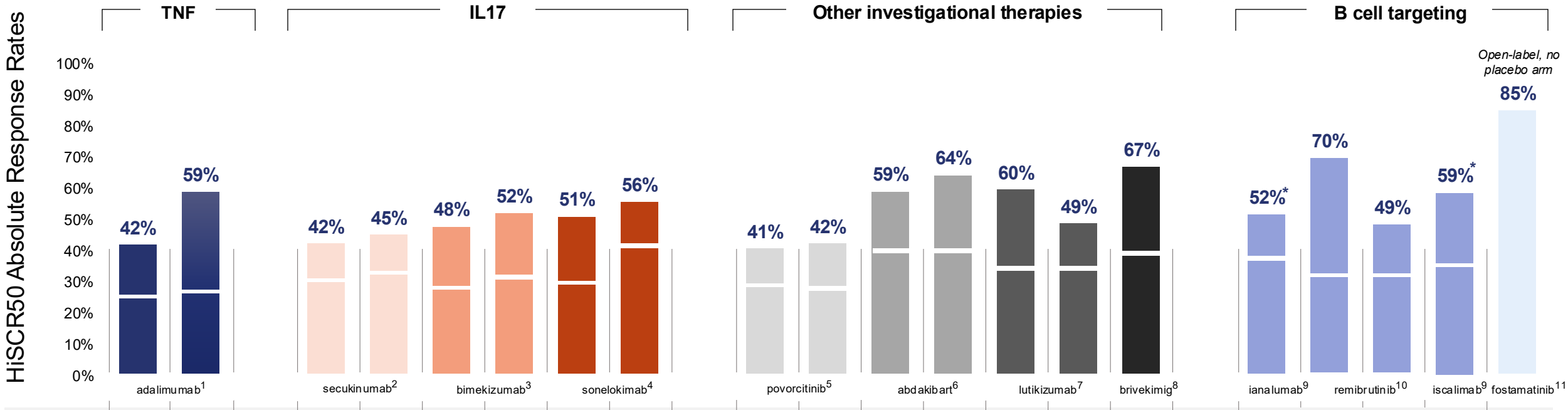
B cell directed monotherapy has demonstrated absolute response rates in line with other approved and late-stage assets in HS



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

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

Targets distinct biology of HS

Tibulizumab is designed to target distinct orthogonal pathways, increasing probability of additive effect

+

Targets IL-17, the most validated MoA in HS

IL-17-binding scFv from ixekizumab (a potent IL-17 pathway inhibitor)

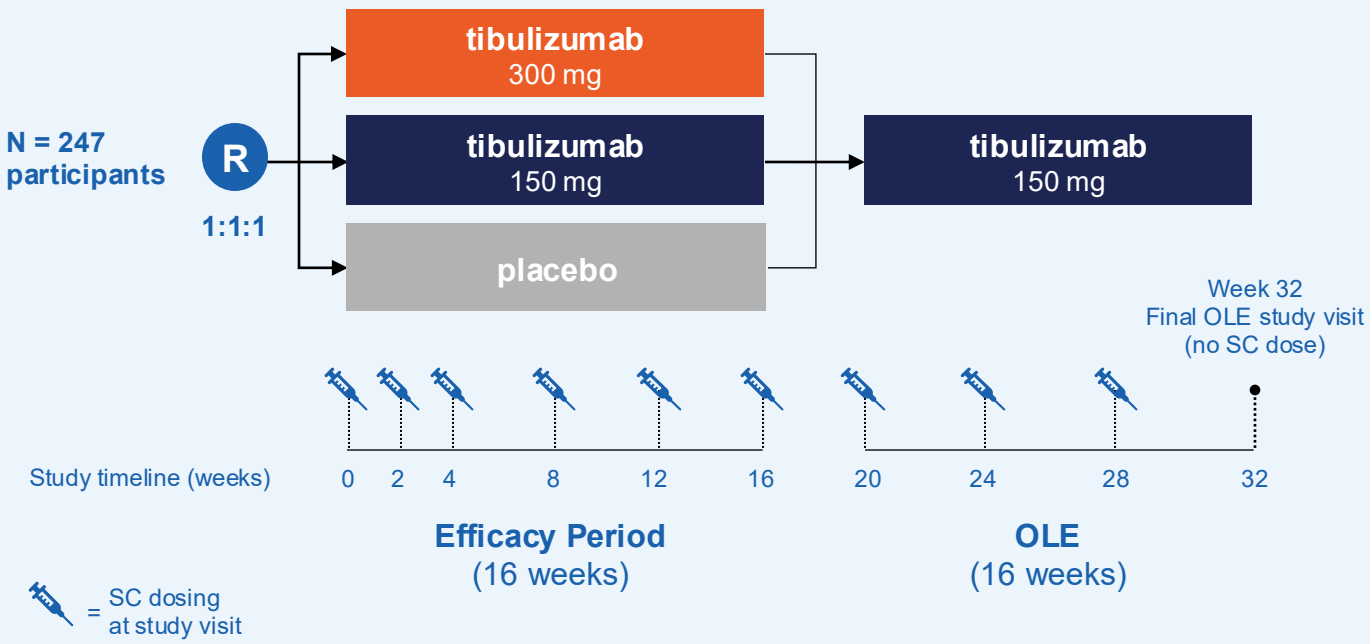
+

BAFF

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

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

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

Systemic Sclerosis (SSc)

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

approximately

300,000

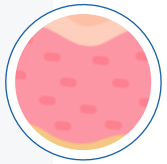
people are estimated to be living with SSc across major markets (US, EU5, Japan)[1]

No therapies are approved that comprehensively address the multisystem pathology of SSc*[2]

Characterized by Chronic Inflammation and Fibrosis Across Organs

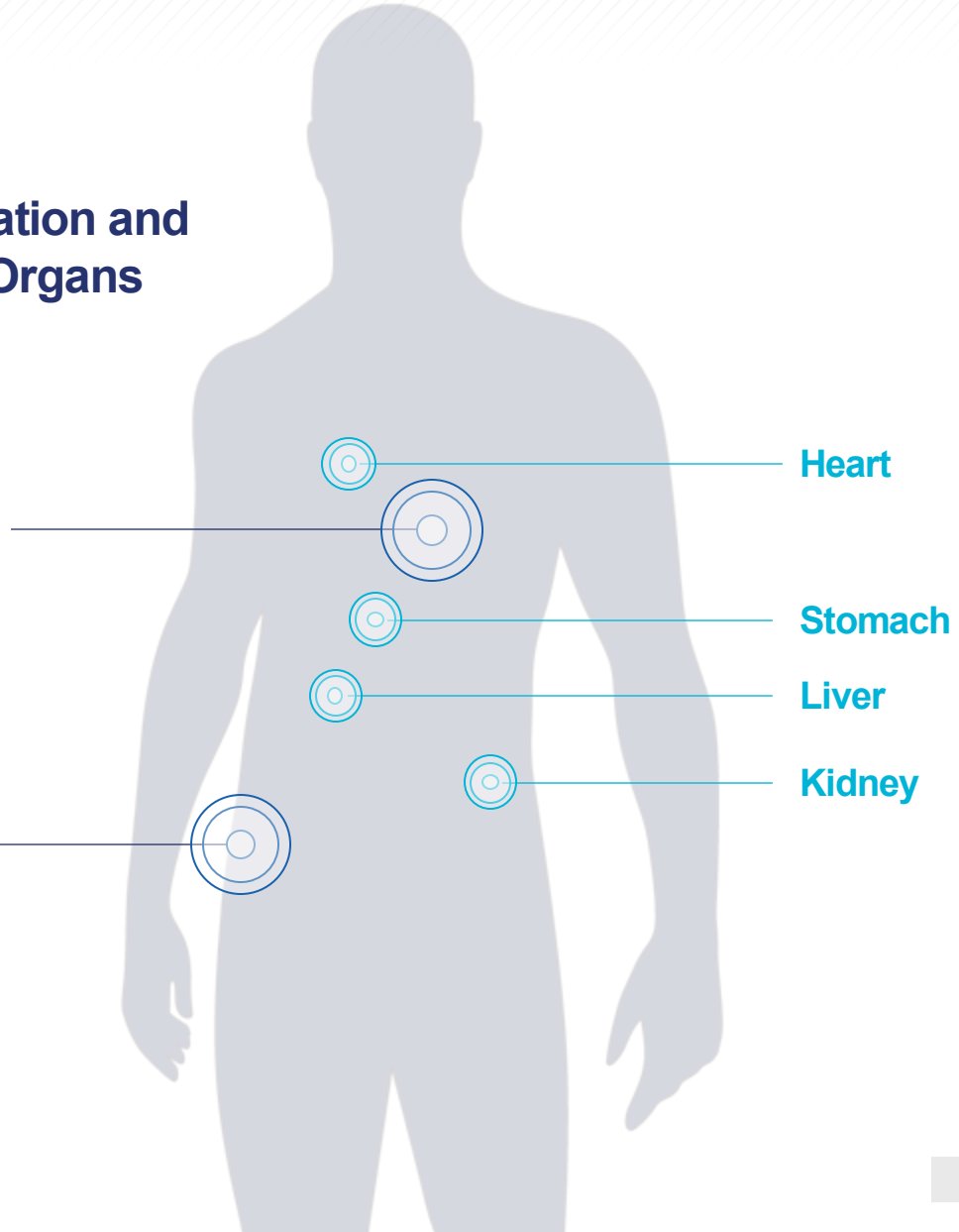
Lungs

SSc-ILD is a major cause of morbidity and mortality in SSc. Two disease-modifying treatments are approved for SSc-ILD[2]



Skin

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



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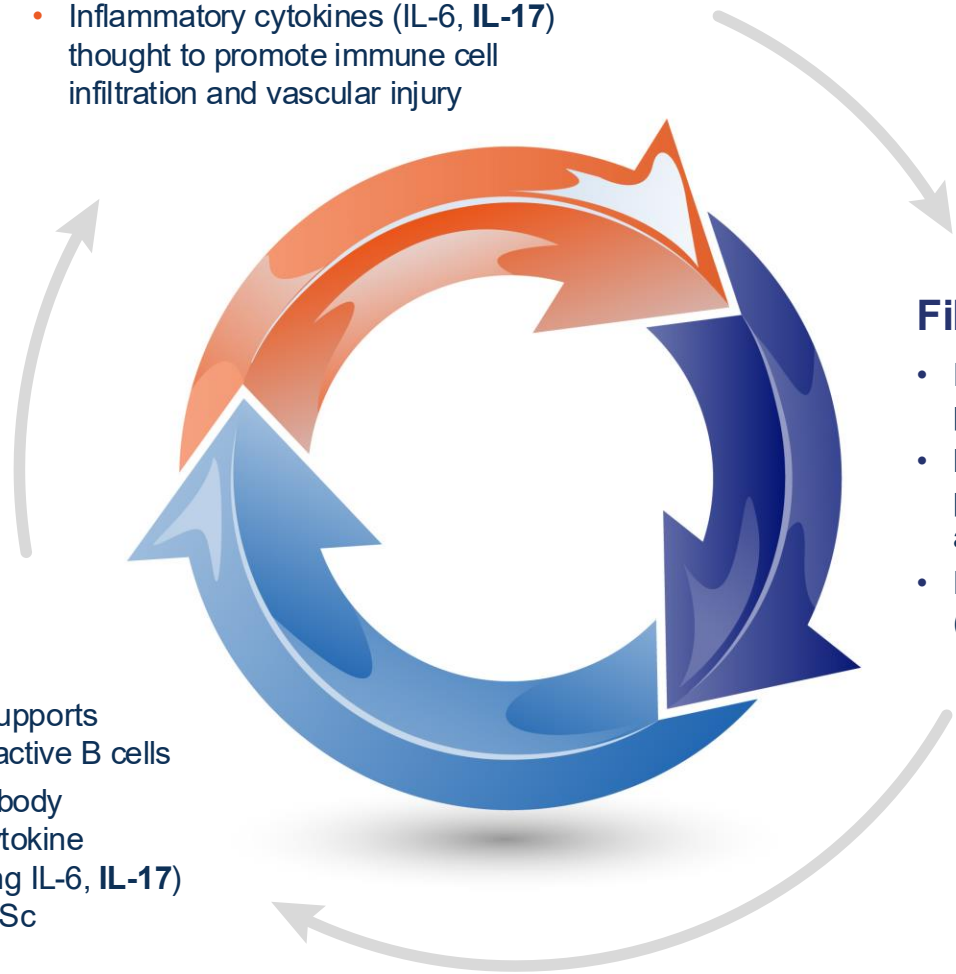
* Two therapies are approved for SSc-ILD; however, no treatment approved for SSc addresses multiple organ systems.
Sources: 1. Clarivate/DRG (accessed 19-Aug-2024); 2. Public regulatory disclosures and prescribing information.

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

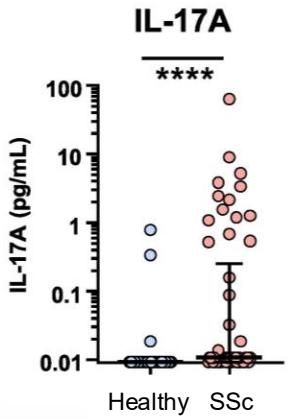
Inflammation

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

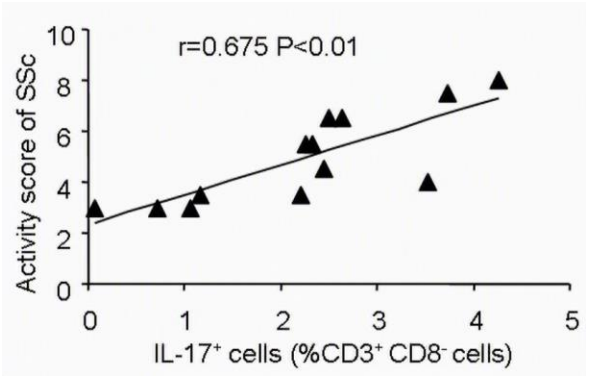
IL-17A is increased in SSc blood and skin and is linked to active and early disease

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

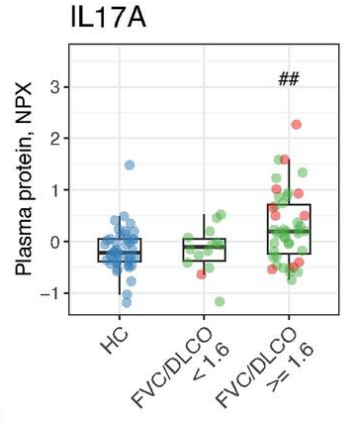
IL-17A cytokine elevated in SSc[1]



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

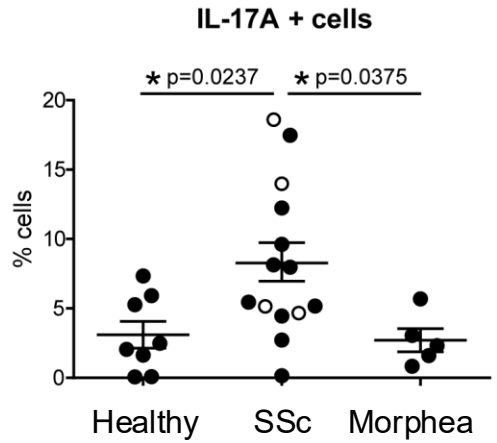


Serum IL-17A a predictor and correlate of pulmonary vascular disease[1,3]

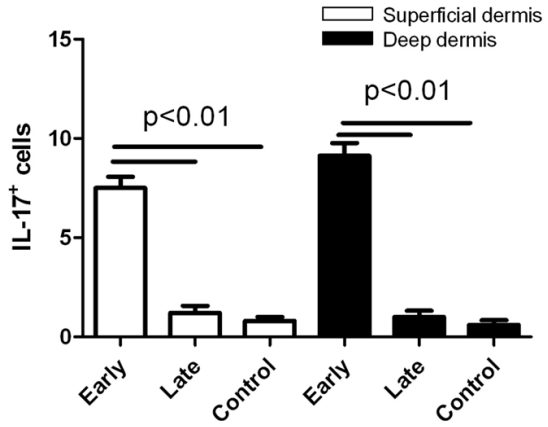


Elevations of IL-17-expressing T cells in skin correlate with early disease

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



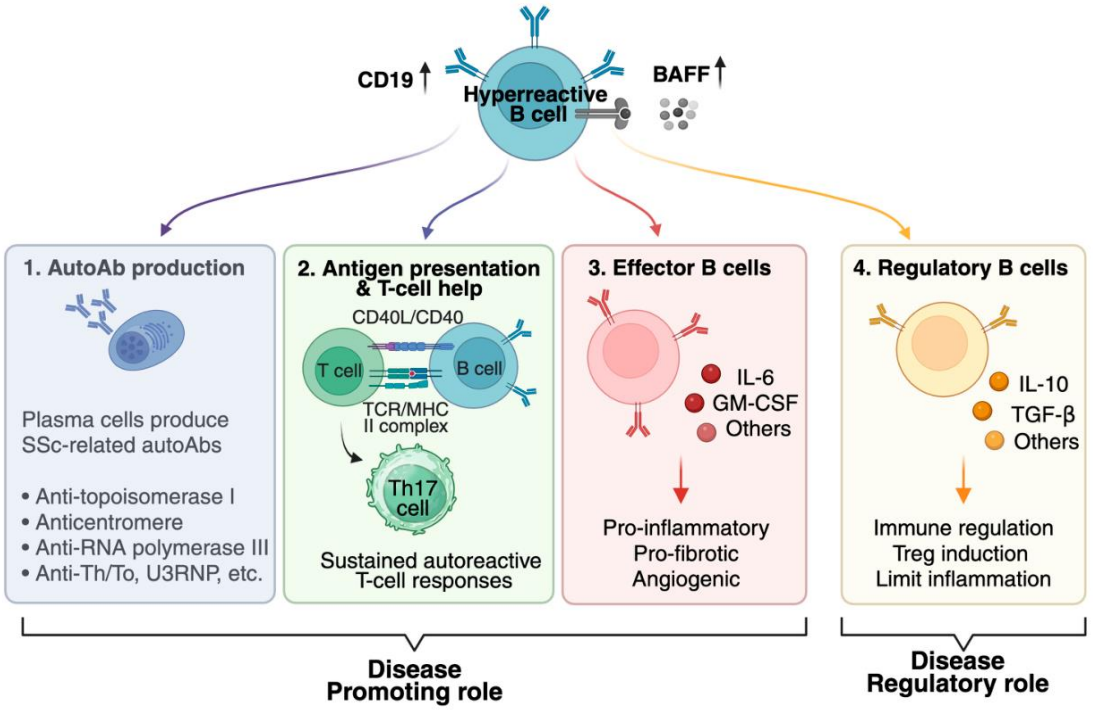
IL-17A+ cells increased in early disease skin[2]



Sources: 1. Seki et al. *Cytokine*. 2024; 2. Yang et al. *Arthritis Res Ther*. 2014; 3. Ono et al. *Sci Rep*. 2024; 4. Lonati et al. *PLoS One*. 2014; 5. Deng et al. *Front Immunol*. 2025.

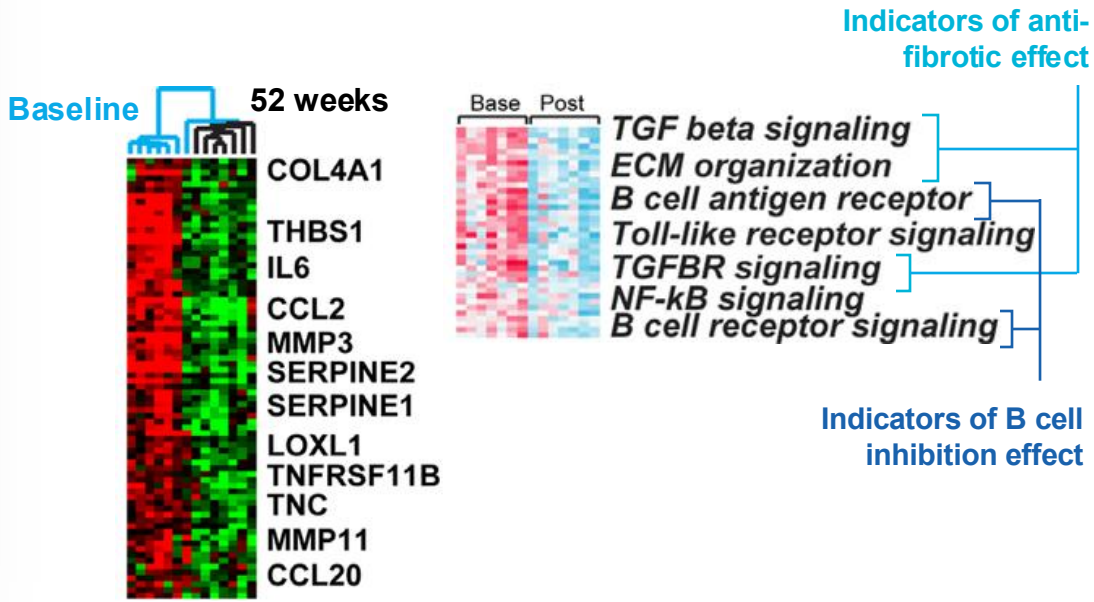
BAFF inhibition reduces profibrotic and B cell signaling genes in SSc skin[1,2]

Hypothesized disease-promoting roles of B cells[3]



Belimumab modulated gene profiles in post-treatment SSc skin[1]

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



Sources: 1. Gordon et al. *Arthritis Rheumatol.* 2018; 2. Matsushita et al. *Arthritis Rheum.* 2006; 3. Matsushita et al. *Sci Adv.* 2018.

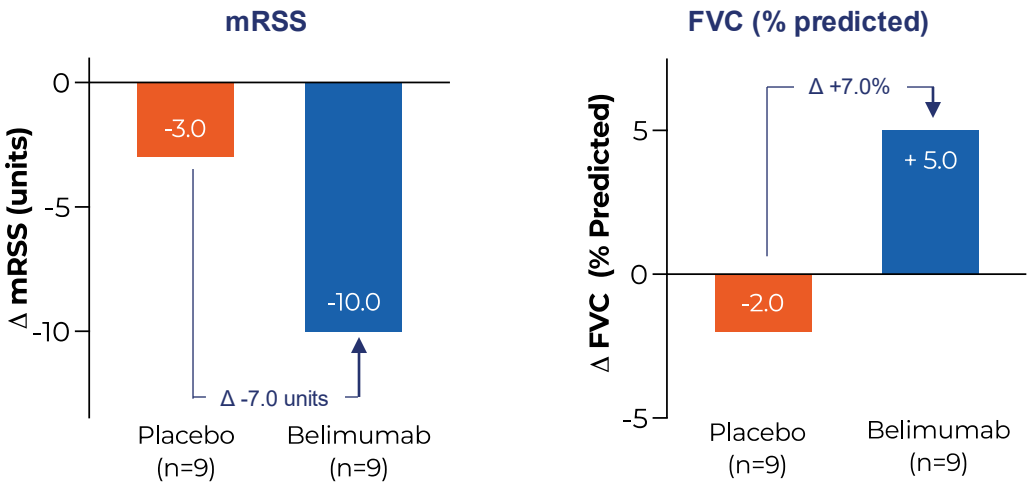
IL-17 and BAFF blockade have each shown improvement in skin and lung outcomes in SSc

BAFF ANTAGONIST

Belimumab[1]

- 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)[1]

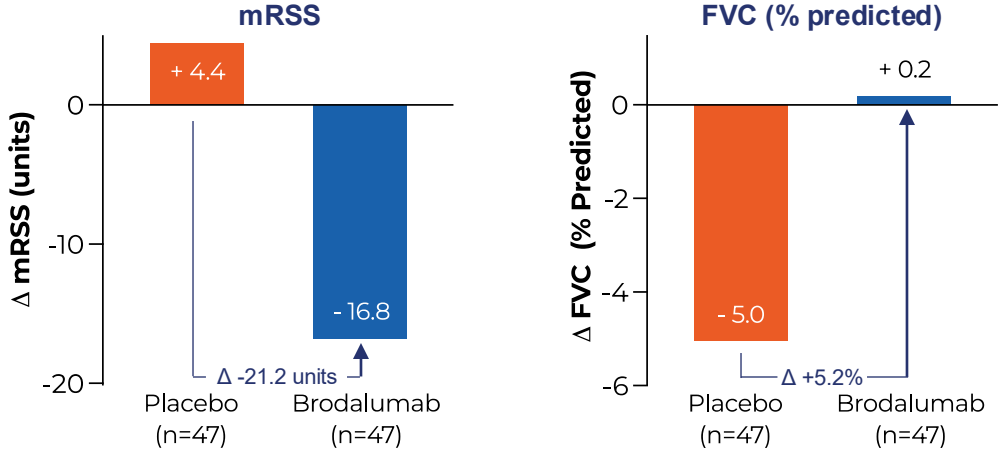


IL-17 RECEPTOR ANTAGONIST

Brodalumab[2]

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

Phase 3 brodalumab trial (24 weeks)[2]



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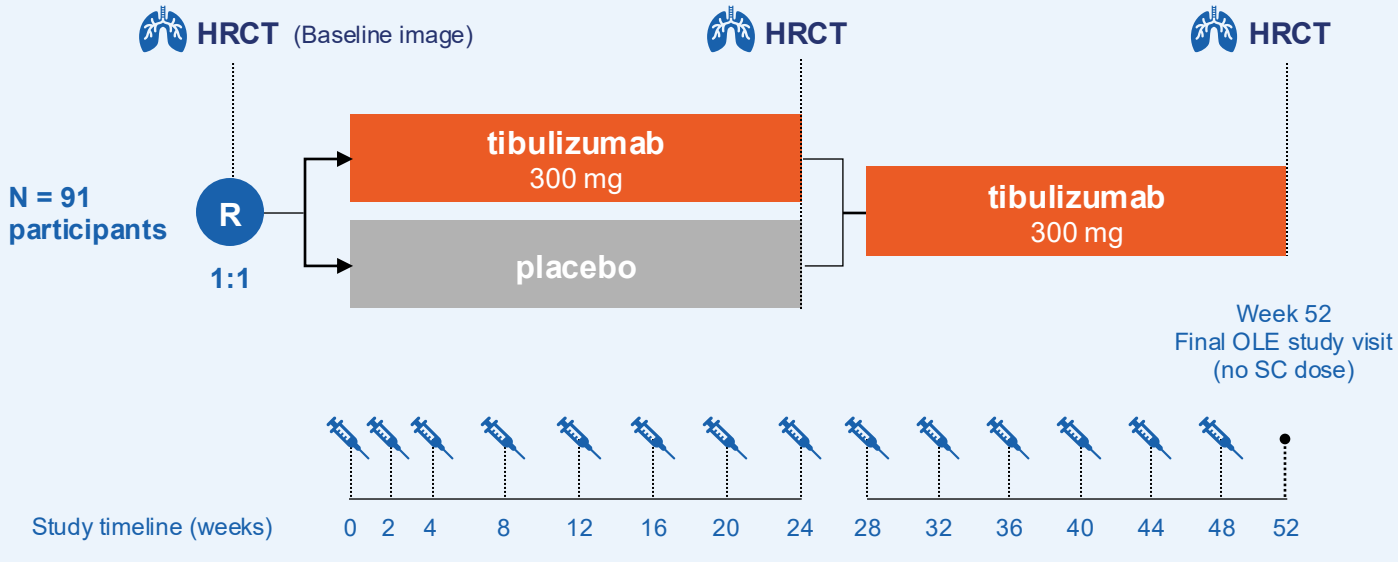
Sources: 1. Gordon JK et al. *Arthritis Rheumatol.* 2018; 2. Fukasawa T et al. *Ann Rheum Dis.* 2022.

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 III (+/-)

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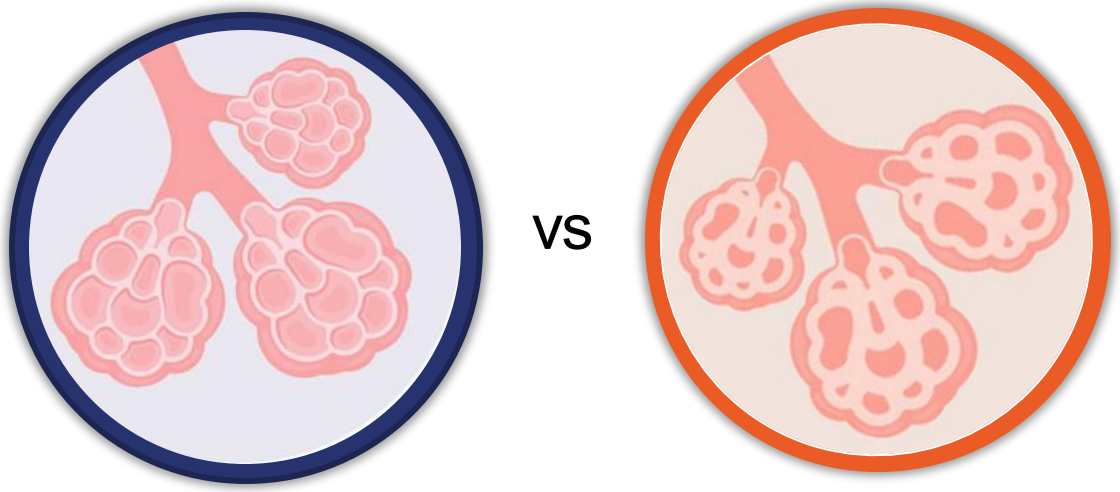
* Includes secondary and exploratory endpoints

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



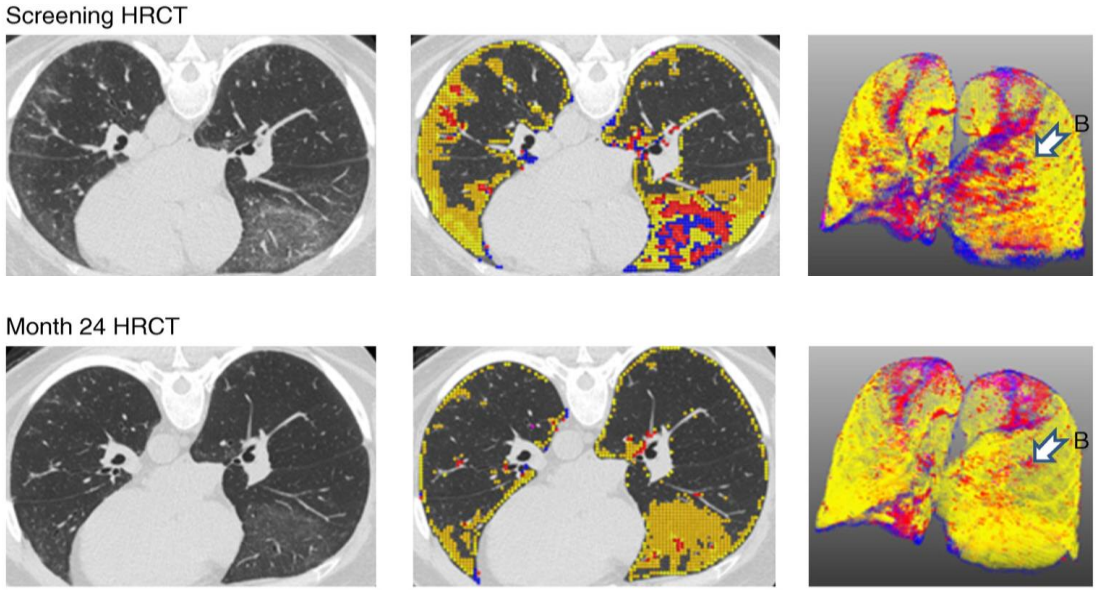
Healthy Lung | **Fibrotic Lung**

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%.[1]

Example of improvement after 24 months of MMF in total lung involvement[1]

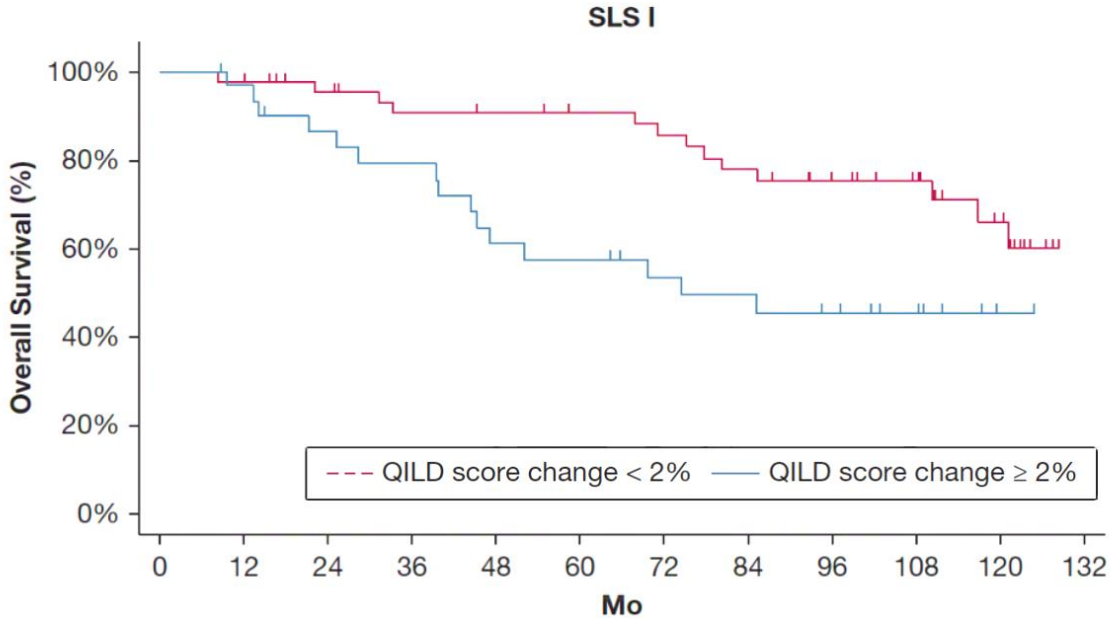


The blue and red areas show QLF, while the yellow area shows quantitative ground glass. The entire colored area represents QILD. After 24 months, QLF areas decreased (*arrow in B*).

Sources: 1. Goldin J et al. *Ann Am Thorac Soc.* 2018; 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]

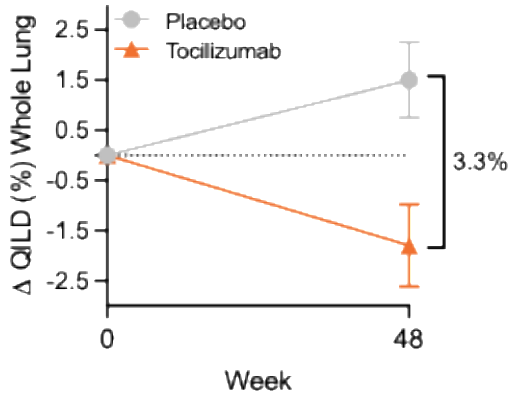
Tocilizumab Phase 3 in SSc-ILD

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%



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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® (tocilizumab) *Prescribing Information.*; 5. Roofeh D et al. *Arthritis Rheumatol.* 2021.

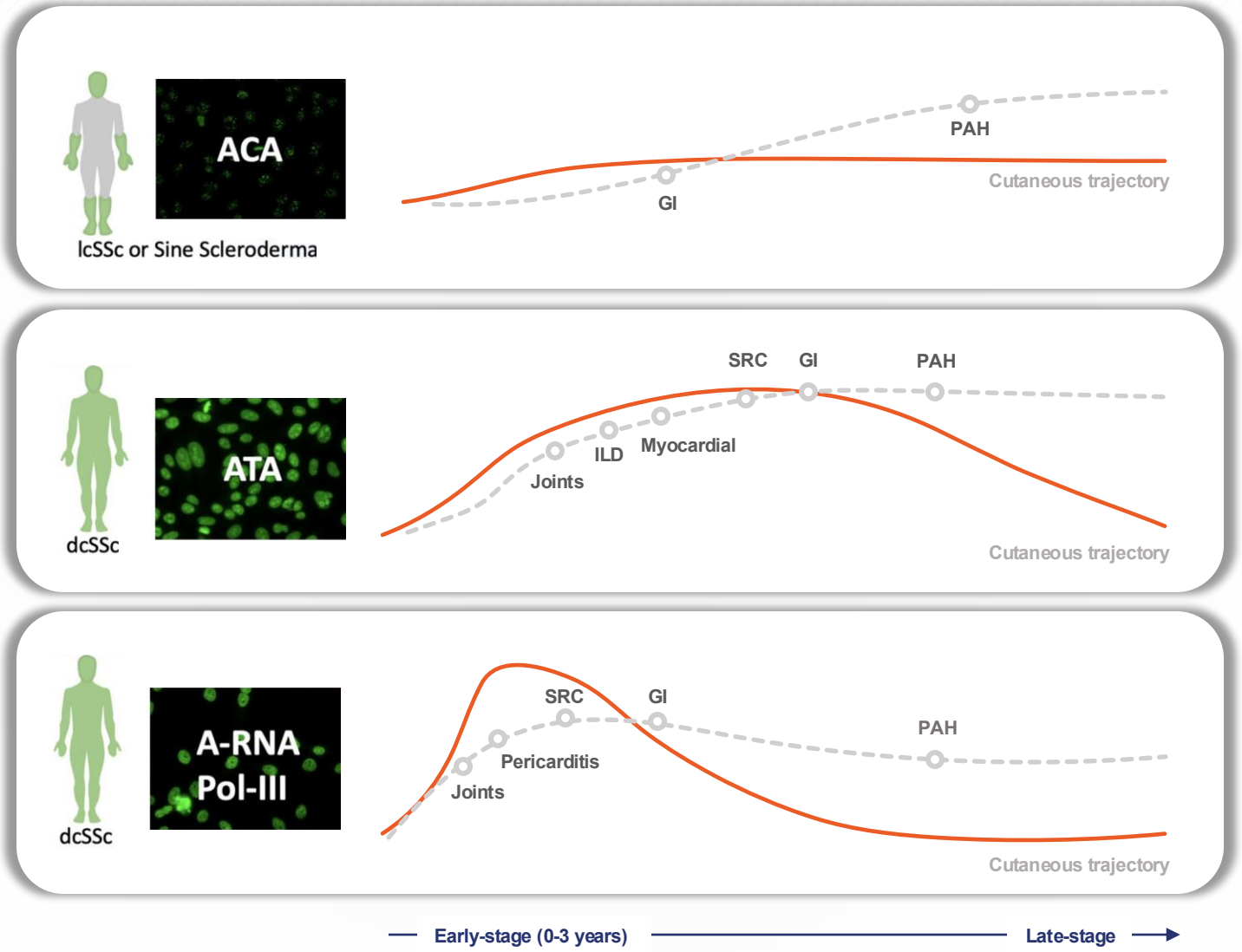
Published SSc disease trajectories validate our Phase 2 enrollment strategy

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

- 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



Source: Lescoat et al. *Semin Immunopathol.* 2025.

Polymyalgia Rheumatica (PMR)

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

Chronic inflammatory disease

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

Demographics and risk factors

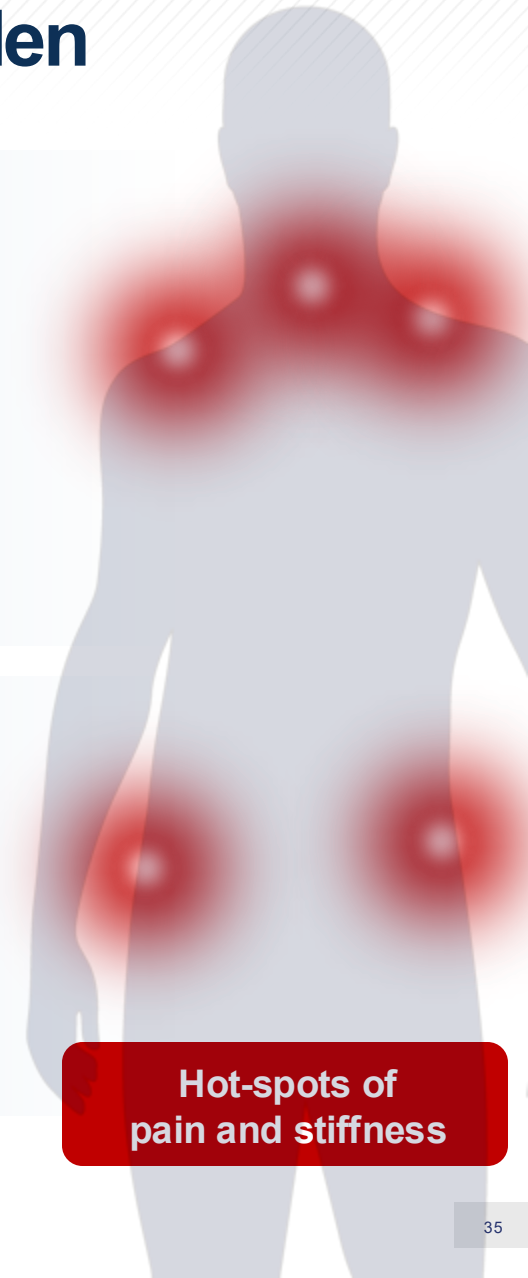
- Almost exclusively patients over 50; peak onset at ages 70-80[2,5]
- Women are more likely to develop PMR than men[5]
- Highly comorbid population[6]

Signs and symptoms

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

Steroid dependency, relapse-prone

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



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Sources: 1. Dasgupta et al. *Ann Rheum Dis.* 2012; 2. Dejaco et al. *Lancet*. 2017; 3. Weyand & Goronzy. *N Engl J Med.* 2014; 4. Floris et al. *Clin Rheumatol.* 2022; 5. Raheel et al. *Arthritis Care Res (Hoboken).* 2017; 6. Partington et al. *Semin Arthritis Rheum.* 2020; 7. Dejaco et al. *Ann Rheum Dis.* 2015.

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

700K+

US PMR Patients[1]

Advanced therapy market remains underpenetrated

Today, only 1 FDA-approved biologic[2]

PMR is an underserved and underpenetrated disease

Steroid-dependency[3]

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

Sources: 1. Lawrence et al. *Arthritis Rheum.* 2008; 2. KEVZARA® (sarilumab) *Prescribing Information.* Sanofi/Regeneron; 3. Floris et al. *Clin Rheumatol.* 2022; 4. Spiera et al. *N Engl J Med.* 2023; 5. Zura Bio. *Data on file.* 2026.

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

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

Over
50%[1]

Relapse on steroid taper

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

Approx.
6 Yrs[2]

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[3]

> **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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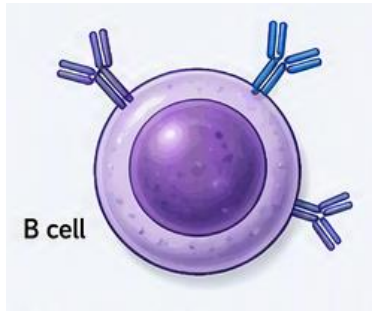
Sources: 1. Spiera et al. *N Engl J Med.* 2023; 2. Devauchelle-Pensec et al. *Ann Rheum Dis.* 2018; 3. Camellino et al. *Front Med (Lausanne).* 2022; 4. Zura Bio. *Data on file.* 2026.

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

B cell homeostasis and trafficking is dysregulated[1,2]

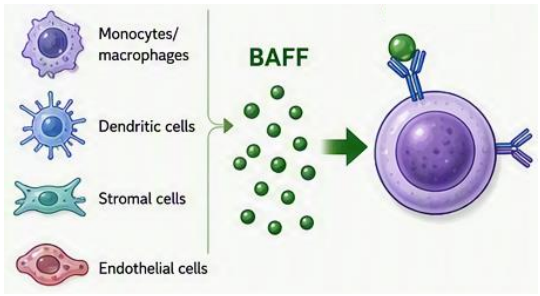


Circulating B cells:

Traffic out of blood during active disease and return in remission[1,2]

May be acting as antigen presenting cells and cytokine producers (including TNF and IL-6) in sites of inflammation[1,2]

BAFF correlates with disease activity[1,2]



Serum BAFF is:

Elevated in newly diagnosed PMR[1,2]

Decreased during remission[1,2]

Correlated with ESR, CRP, and IL-6[2]

↓ may lead to

↑ B cell survival
↑ B cell activation
↑ Cytokine production
↑ Interactions with T cells

IL-17 may amplify local and systemic inflammation[3,4]

IL-17 and IL-17-producing cells (e.g. Th17, MAIT) are:

Elevated in circulation[3,4]

Present in synovial fluid[3,4]

↓ may lead to

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

↑ number and activation of myeloid cells

Sources: 1. van der Geest et al. *Arthritis Rheumatol.* 2014; 2. van der Geest et al. *Rheumatology (Oxford).* 2015; 3. Reitsema et al. *Front Immunol.* 2022; 4. Ramiro et al. *Rheumatol Ther.* 2025.

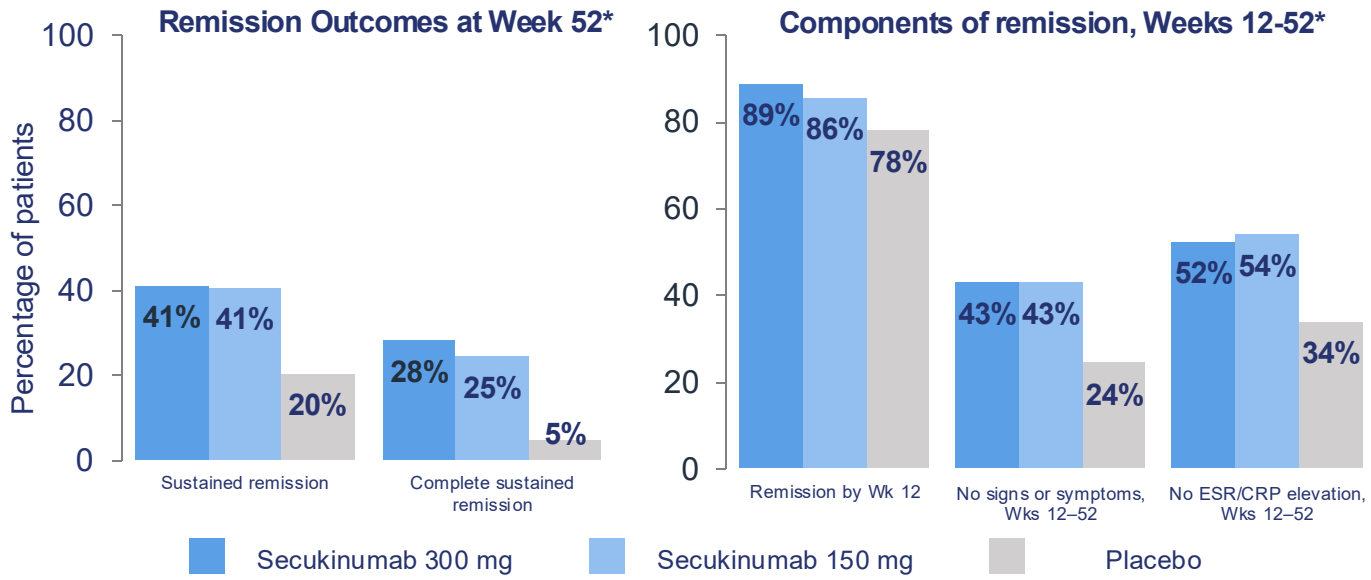
IL-17 and B cell targeted therapies have shown positive glucocorticoid-sparing effects in PMR

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

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 and EU for approval in PMR

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

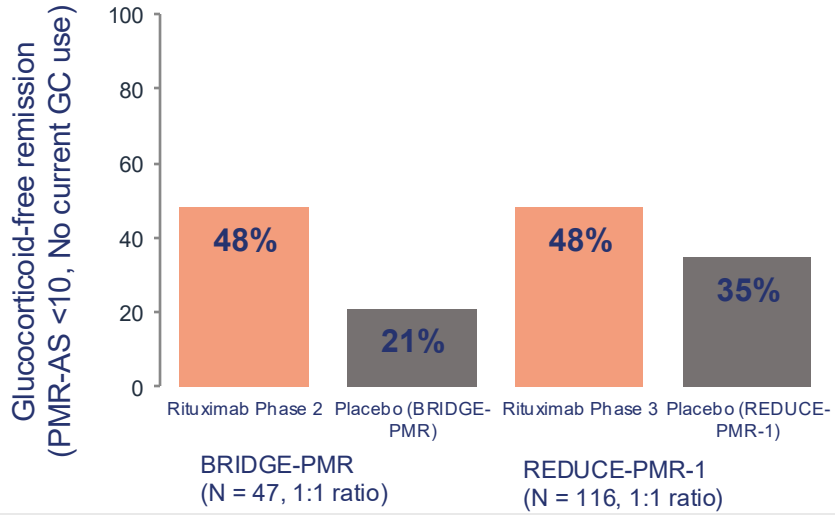


Anti-CD20 – newly diagnosed PMR (Phase 2 & 3)

Rituximab

- Anti-CD20 clinical evidence that B cells drive PMR
- BRIDGE-PMR (Phase 2 IIT) demonstrated that a single rituximab infusion increased glucocorticoid-free remission at 21 weeks and produced durable benefit through one year[2,3]
- REDUCE-PMR-1 (Phase 3 IIT) showed trend in favor of rituximab[4]

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



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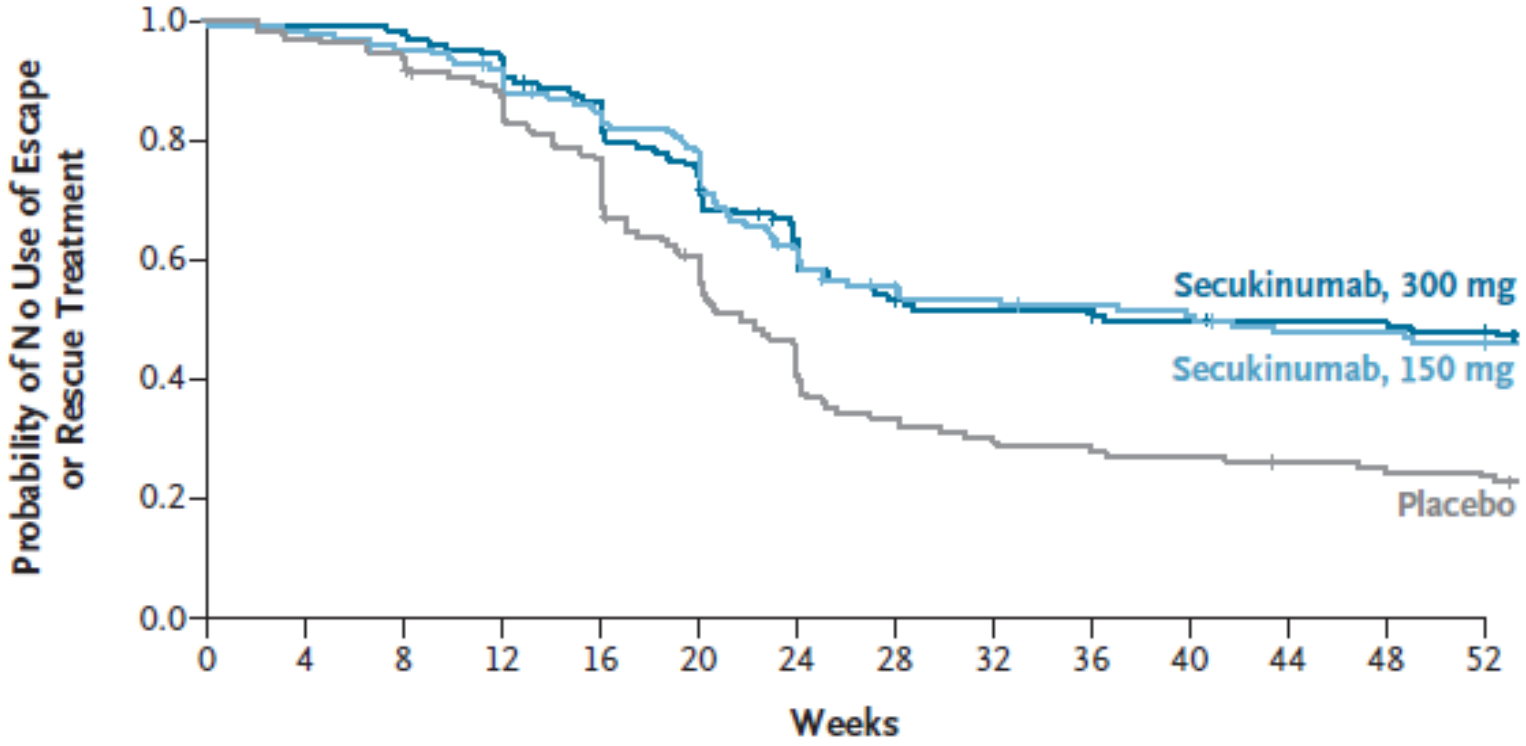
* Sustained remission defined as remission that occurred by week 12 and was sustained until week 52, without recurrence of PMR and no new diagnosis of GCA; complete sustained remission defined as sustained remission plus no ESR/CRP elevation at Week 52
** 52 week results shown from extension phase of BRIDGE-PMR

Sources: 1. Stone et al. *N Engl J Med.* 2026 (REPLENISH); 2. Marsman et al. *Lancet Rheumatol.* 2021 (BRIDGE-PMR); 3. Bolhuis et al. *Lancet Rheumatol.* 2023 (BRIDGE-PMR 1-year extension); 4. REDUCE-PMR-1. *NCT05533125.*

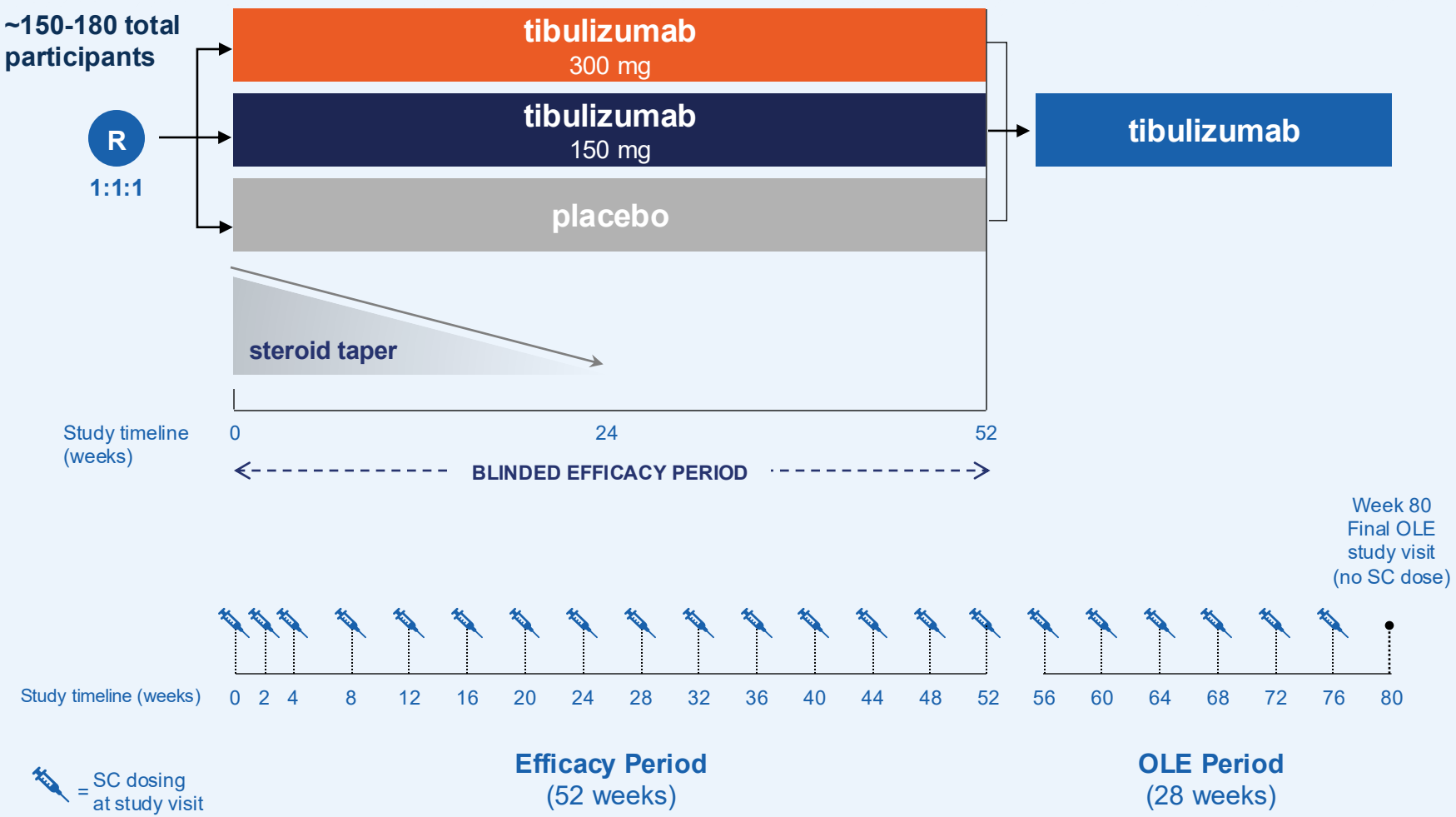
Despite IL-17 advances, the unmet need for PMR remains significant



- **Glucocorticoids control PMR but cannot be withdrawn:** three in four patients on taper alone needed escape or rescue therapy by Week 52
- **IL-17 provides real but partial benefit:** More than half of participants in secunikumab arms do not reach sustained remission

Source: Stone et al. *N Engl J Med.* 2026 (REPLENISH).

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



NEXUS|PMR

Key Inclusion Criteria

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

Key Efficacy Endpoints

Primary endpoint:

- Sustained remission at Week 52 defined as:
  - Clinical remission at Week 12 maintained to Week 52
  - No use of escape or rescue therapy
  - No new diagnosis of Giant Cell Arteritis

Key secondary endpoints:

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

Summary & Milestones

Executive Team and Board of Directors

Executive Team

Sandeep Kulkarni, MD

Co-Founder, Chief Executive Officer and Director

Kim Davis, JD

Chief Operating Officer, Chief Legal Officer and Corporate Secretary

Muzammil Mustufa

Chief Business Officer

Kiran Nistala, MBBS, PhD

Chief Medical Officer and Head of Development

Gary Whale, PhD

Chief Technology Officer

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

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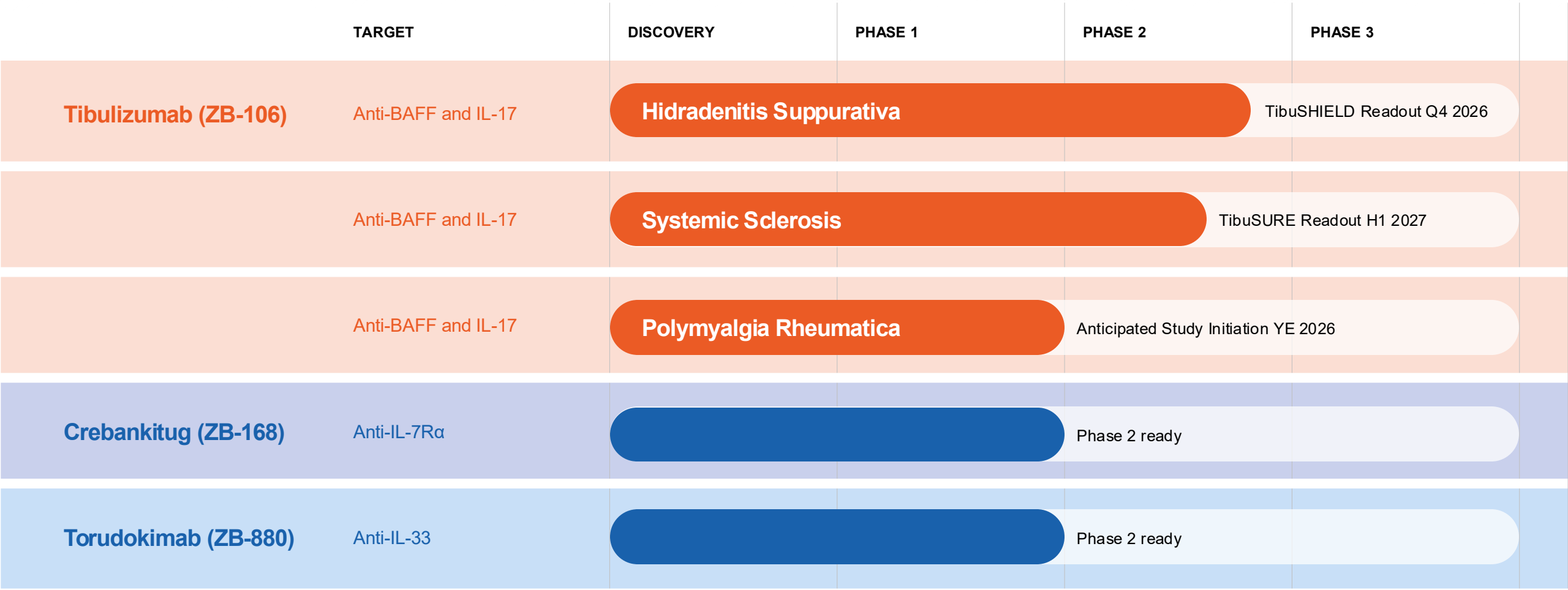
Multiple potential near-term clinical milestones and strong pipeline for the future



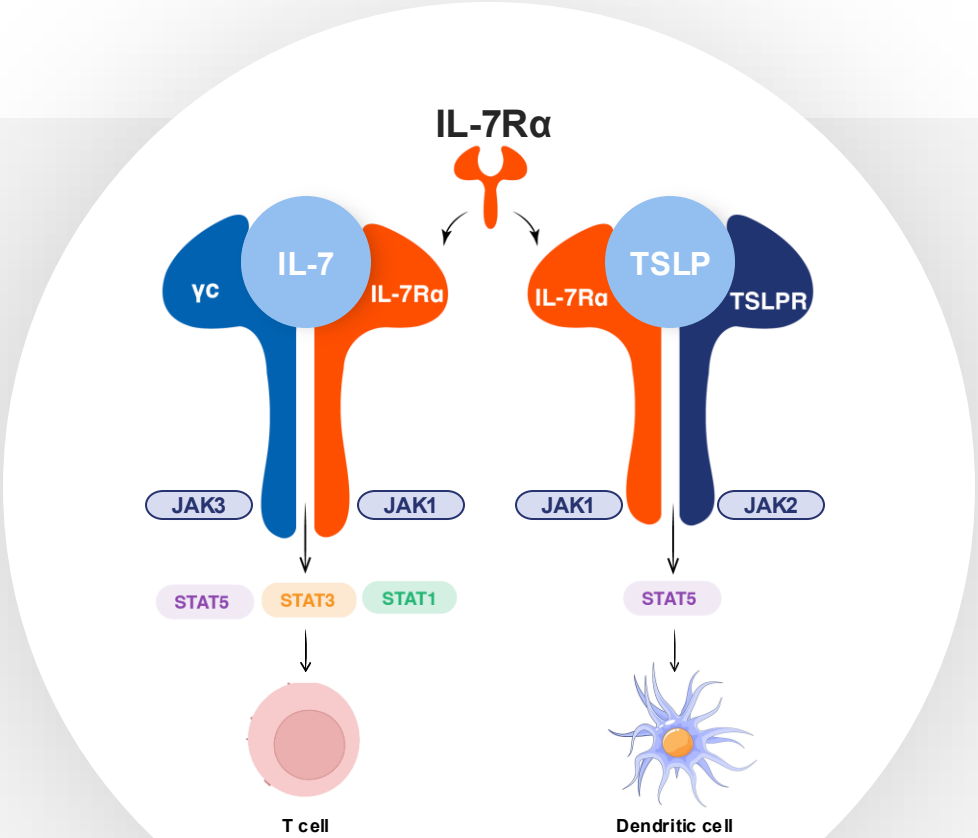
Crebankitug: Phase 2 ready multi-functional antibody with potential in dermatology and beyond

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

IL-7

IL-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 its 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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Source: Markovic et al. *Front Immunol.* 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]

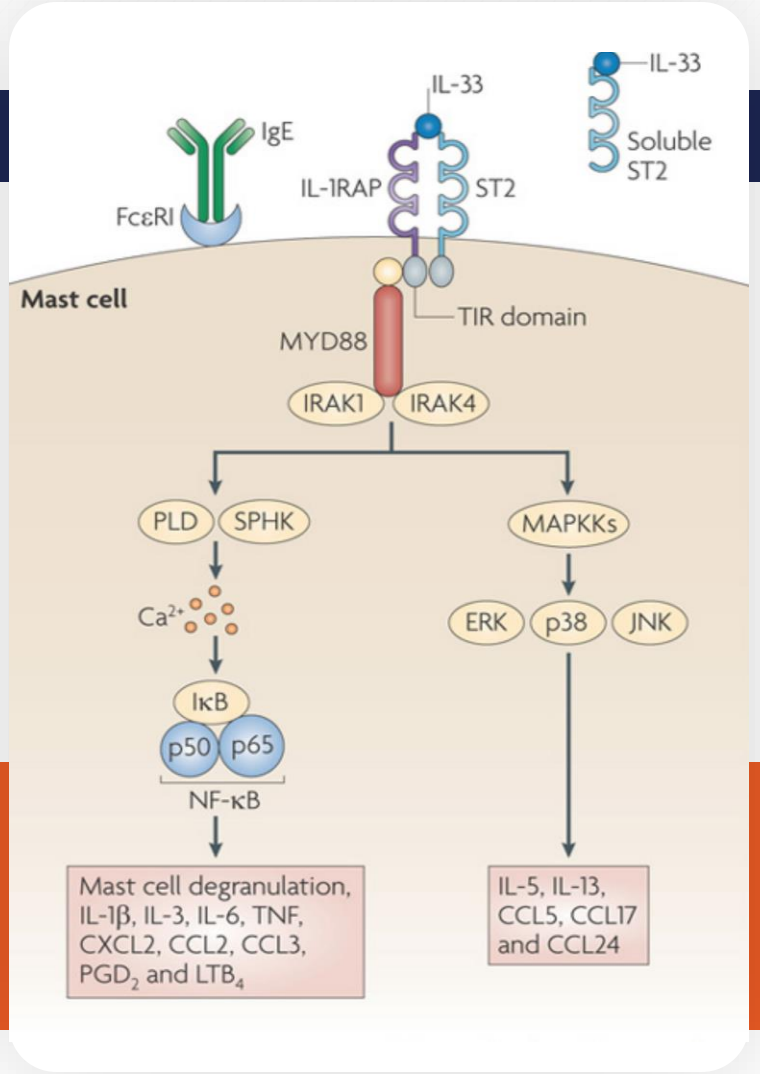
Torudokimab

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; 2. Liew et al. *Nat Rev Immunol*. 2010; 3. Zura Bio. *Data on file*. 2026; 4. Laquer et al. *Br J Dermatol*. 2022; 5. Okragly et al. *J Inflamm Res*. 2021; 6. Wechsler et al. *NEngl J Med*. 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
dcSSc	diffuse cutaneous systemic sclerosis
DLCO	diffusing capacity of the lung for carbon monoxide
DLQI	Dermatology Life Quality Index
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-1β	interleukin 1 beta
IL-6	interleukin 6
IL-7Rα	interleukin 7 receptor alpha
IL-8	interleukin 8
IL-10	interleukin 10
IL-17	interleukin 17
IL-18	interleukin 18
IL-33	interleukin 33
ILD	interstitial lung disease
JAK	Janus kinase
K_D	dissociation constant
LPA1	lysophosphatidic acid receptor 1
mAb	monoclonal antibody
MAIT	Mucosal-associated invariant T cells
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 and Severity Index
PASI 90	Psoriasis Area and Severity Index 90% improvement
PBO	placebo
PD	pharmacodynamics
PK	pharmacokinetics
pM	picomolar
PMR	polymyalgia rheumatica
PsO	psoriasis
Q2W	once every 2 weeks
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%)
SLE	systemic lupus erythematosus
SLS	Scleroderma Lung Study
SoC	standard of care
SRC	scleroderma renal crisis
SSc	systemic sclerosis
SSc-ILD	systemic sclerosis-associated interstitial lung disease
ST2	suppression of tumorigenicity 2
T2D	type 2 diabetes
TARC	thymus and activation-regulated chemokine
TGFβ	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
TSLP	thymic stromal lymphopoietin
VAS	visual analog scale

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