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Corporate Overview

October 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 PMR⁵) or BAFF arm (SSc⁶) with strong scientific support for the orthogonal pathway^{7,8,9}

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

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

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 average¹ because current therapies only partially address the multi-pathway biology driving these diseases²⁻⁹

~\$5B
2026

\$15B+

Projected
Mid-2030s

2030s:

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

SIGNIFICANT GROWTH:

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

Both circles represent combined HS, SSc, and PMR global markets¹

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 (UITIMMA 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

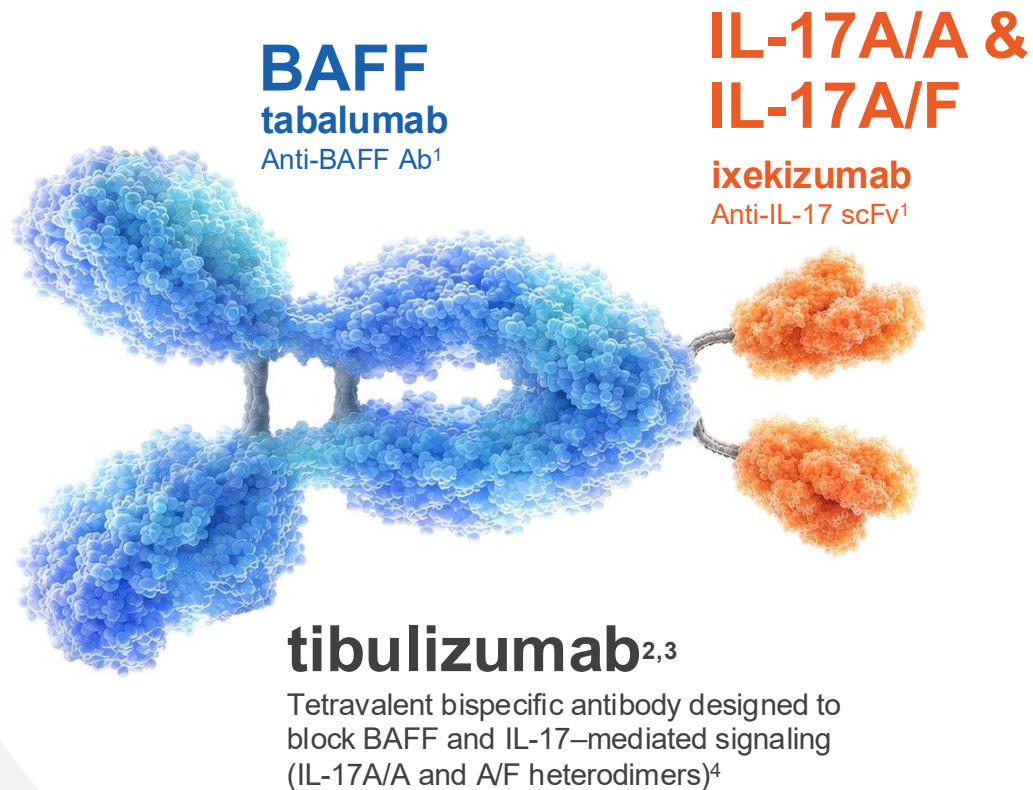
Anticipated initiation:
YE 2026



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

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



Addresses disease complexity

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

Clinically grounded design

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

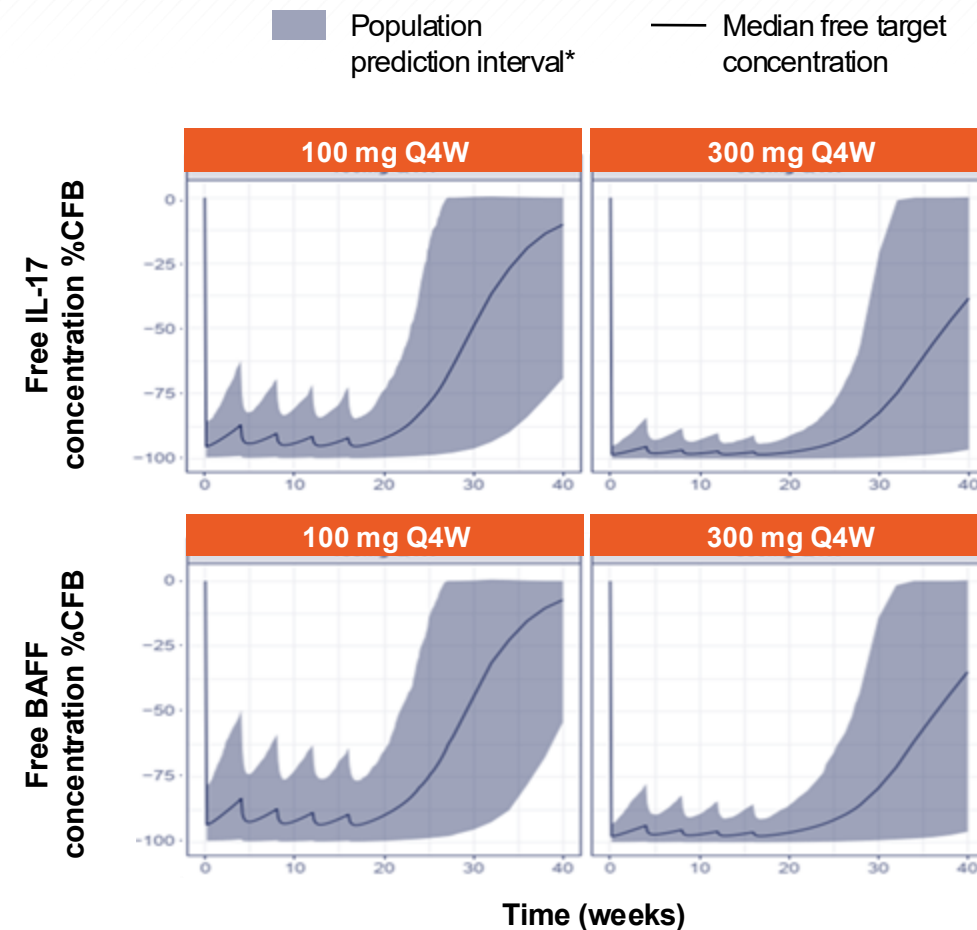
Single-entity advantage

Enables dual-pathway modulation without multi-drug combination complexity

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

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

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

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

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

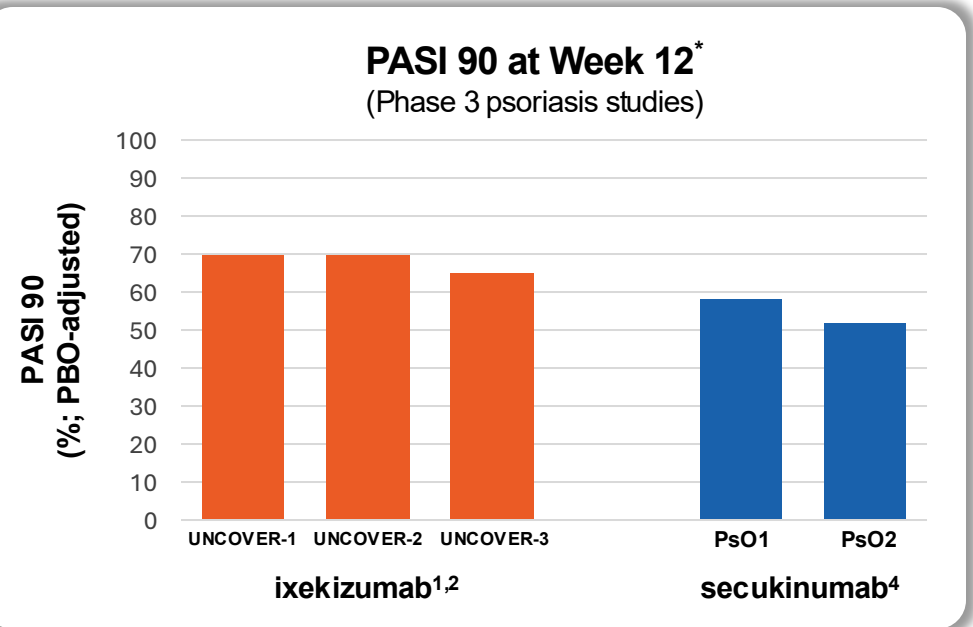
Multiple Phase 3 programs completed

High efficacy demonstrated

Broad IL-17 pathway inhibition

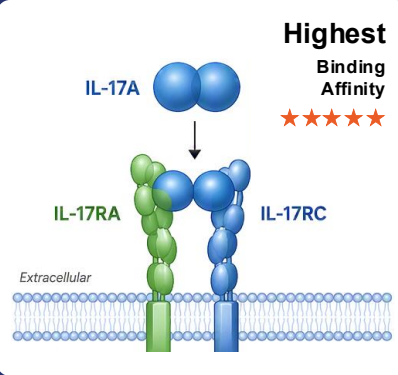
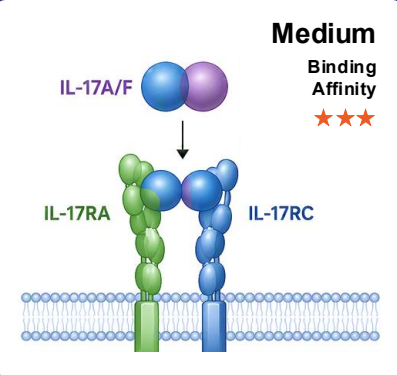
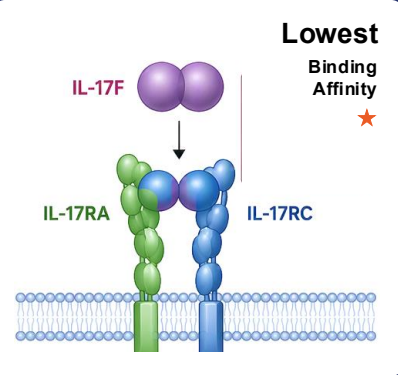
Extensive clinical and real-world experience

- 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

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

Agent	Target description	 Highest Binding Affinity ★★★★★ IL-17A/A (K_D)	 Medium Binding Affinity ★★★ IL-17A/F (K_D)	 Lowest Binding Affinity ★ 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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Hidradenitis Suppurativa (HS)

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

Patients experience pain in sensitive, high-friction areas

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

Patients withdraw from social interactions due to visible symptoms

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

Patients struggle with persistent drainage and odor from lesions

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

Patients experience prolonged delays in diagnosis and treatment

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

Distinctive for structural skin damage driven by tunneling and scarring¹

Lesions commonly occur in intertriginous areas (skin folds)¹

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 improve⁶

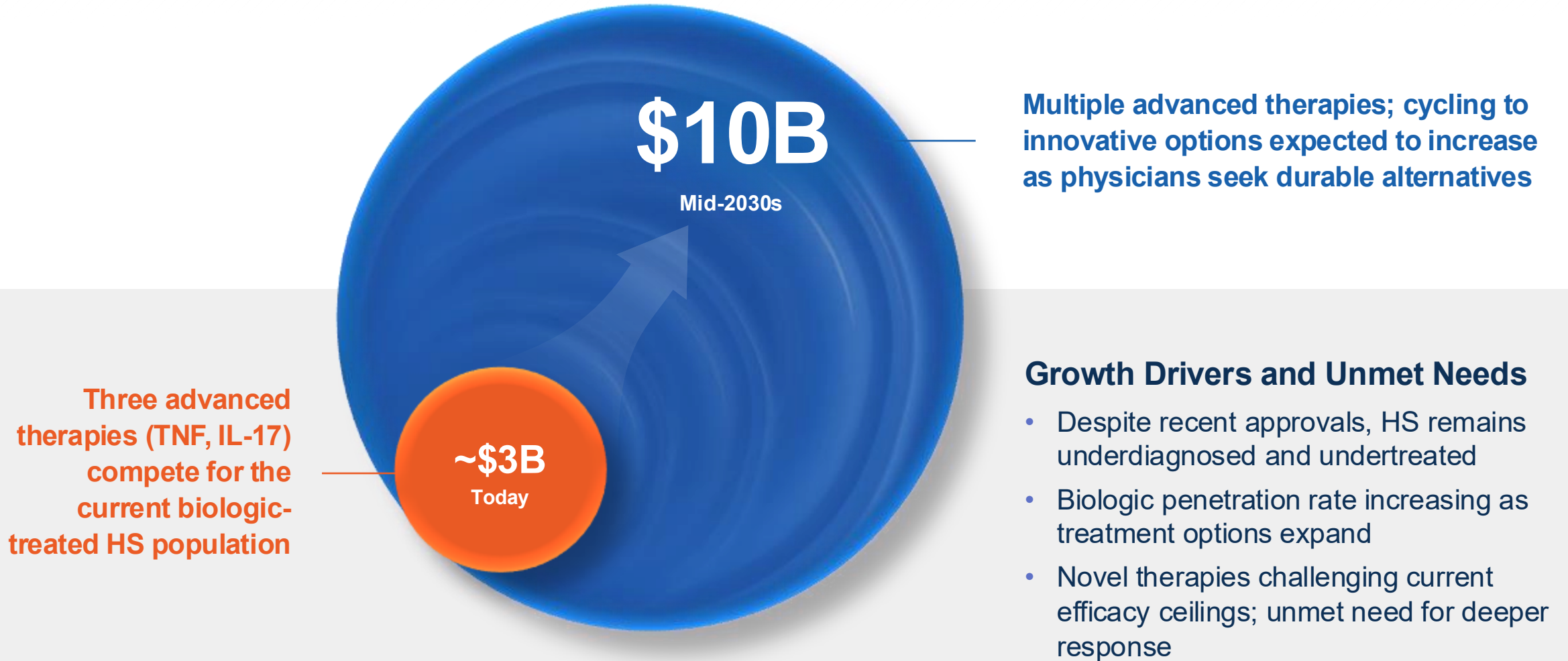
Structural damage is irreversible

Once disease progresses to tunnels and scarring, no current therapy can reverse it¹

Physicians remain only moderately satisfied

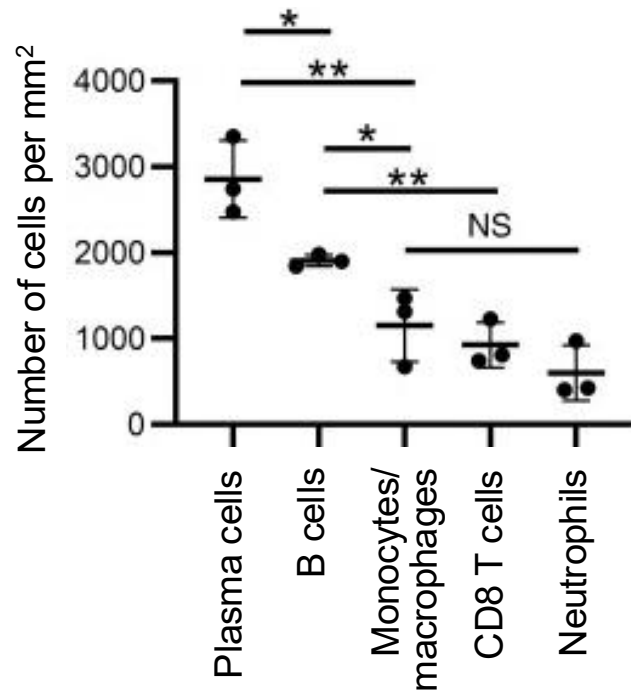
Despite current advancements, physicians desire new mechanisms of action that provide improved efficacy, deeper and more durable response⁴

Hidradenitis suppurativa: large, underserved market with high unmet need



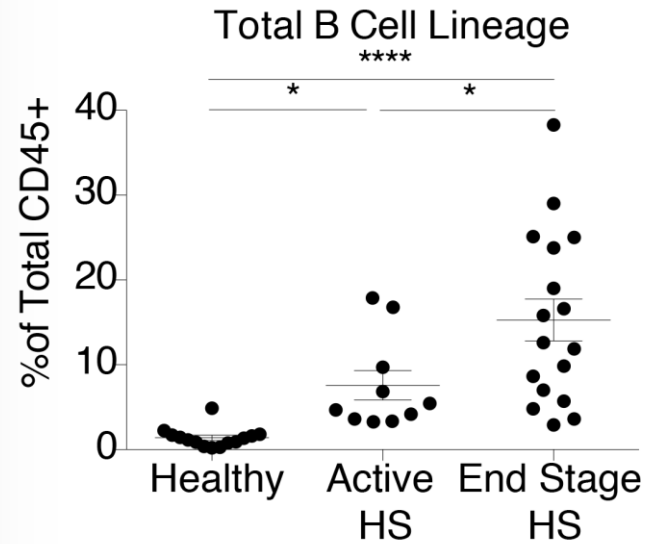
Unlike healthy skin, B cells infiltrate and chronically persist in HS lesions, driving a local amplified and targeted immune response

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



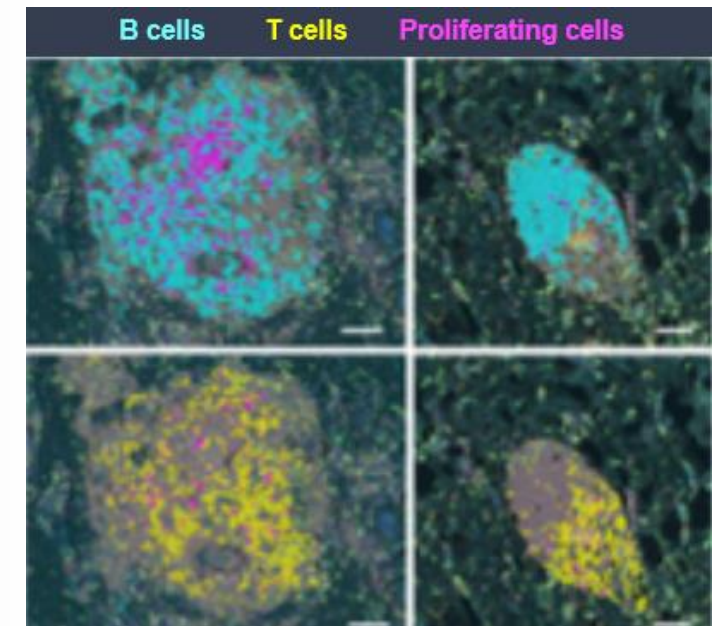
Imaging CyTOF analysis of HS lesions

B cells expand with disease progression²



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

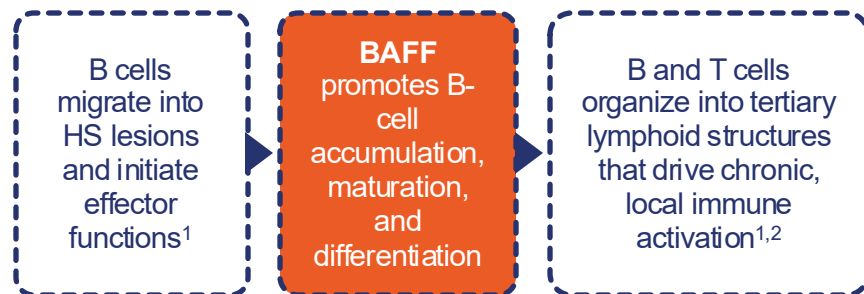
In severe disease, B and T cells organize and persist in tertiary lymphoid structures (TLS)^{3,4}



Immunofluorescence analysis of HS chronic lesion with tunnel

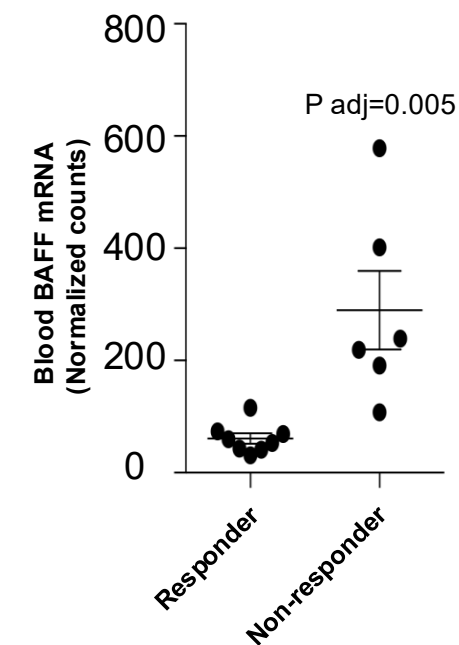
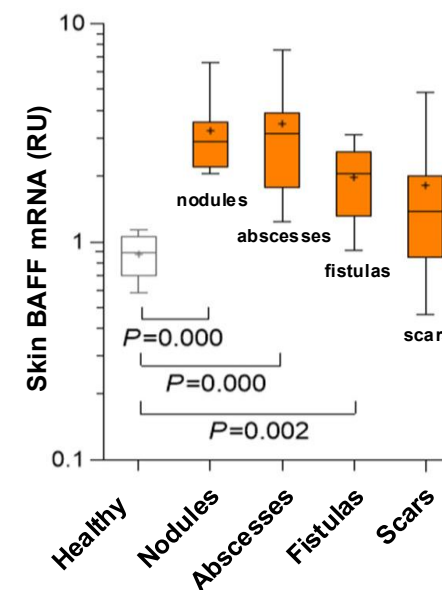
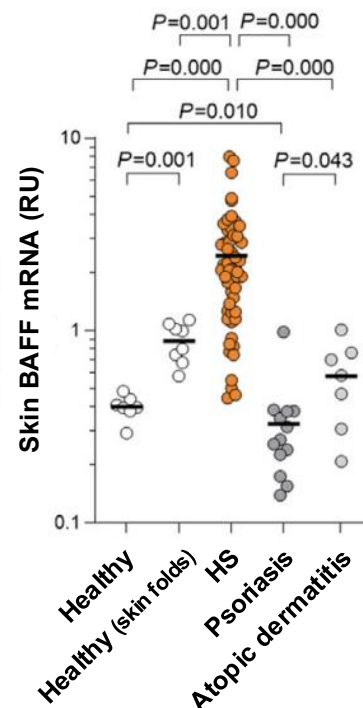
BAFF is elevated across HS lesion types and predicts treatment resistance

Elevated BAFF can retain B cells in the skin



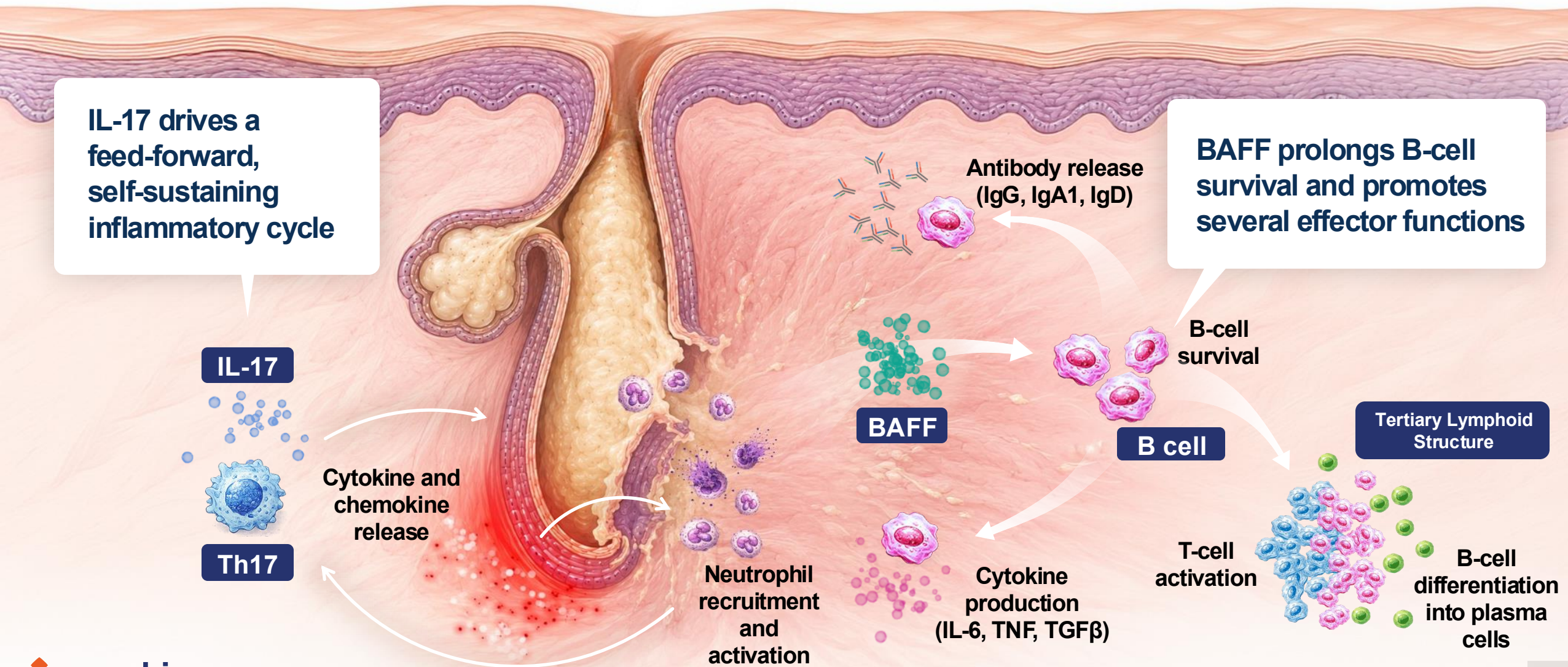
- BAFF is absent in healthy skin and in psoriasis and dermatitis lesions, and B cells transiently migrate in and out of skin^{3,4}
- BAFF is significantly elevated in all HS lesion types, and correlates with increases in B cells^{3,4}
- Patients who have higher baseline levels of BAFF and B-cell counts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

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



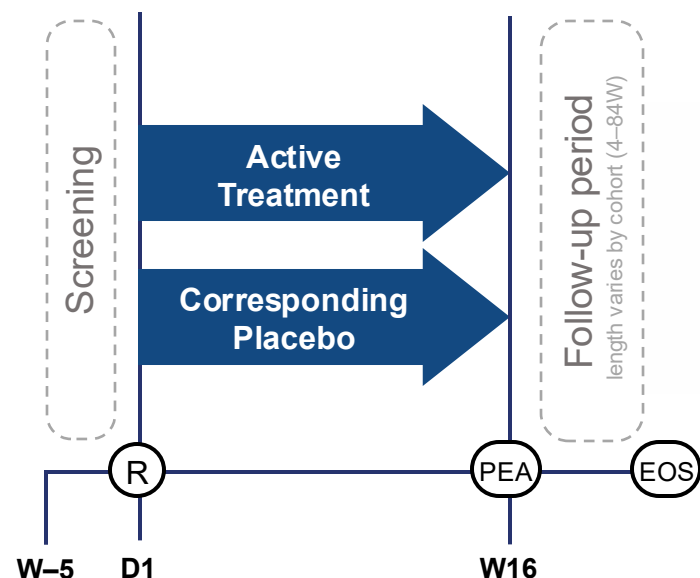
In acute and chronic HS lesions, BAFF expression tracks with B-cell accumulation, supporting BAFF as a key player in B-cell-mediated pathology

B cells are believed to drive HS pathology via multiple mechanisms



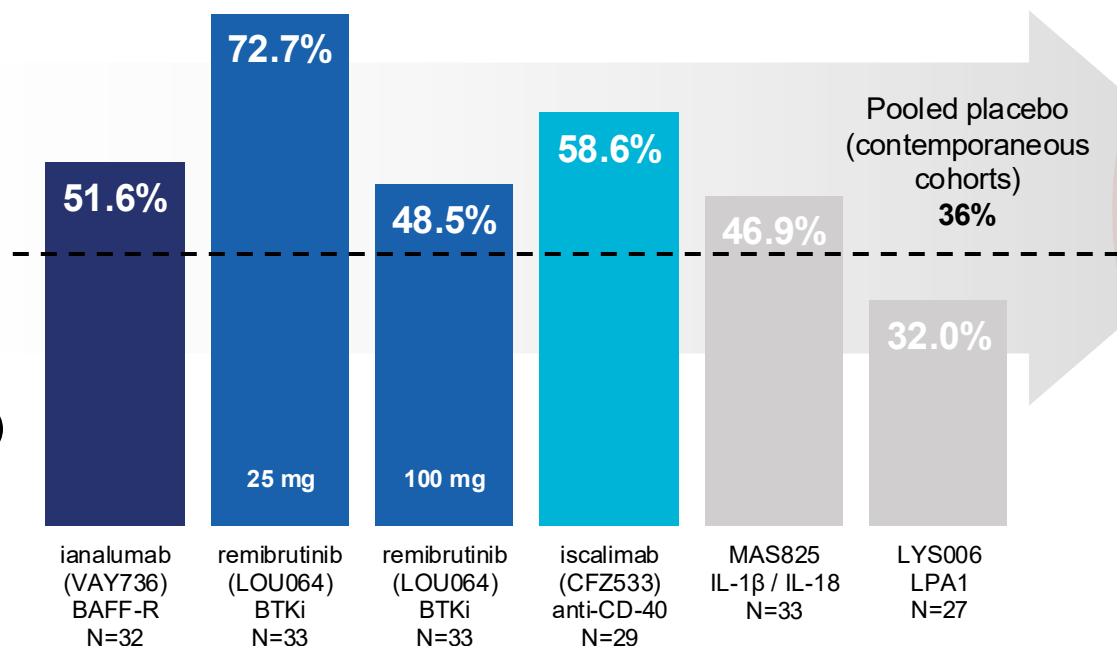
Recent clinical data support the hypothesis that B cells are key drivers of disease rather than bystanders in HS*

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}

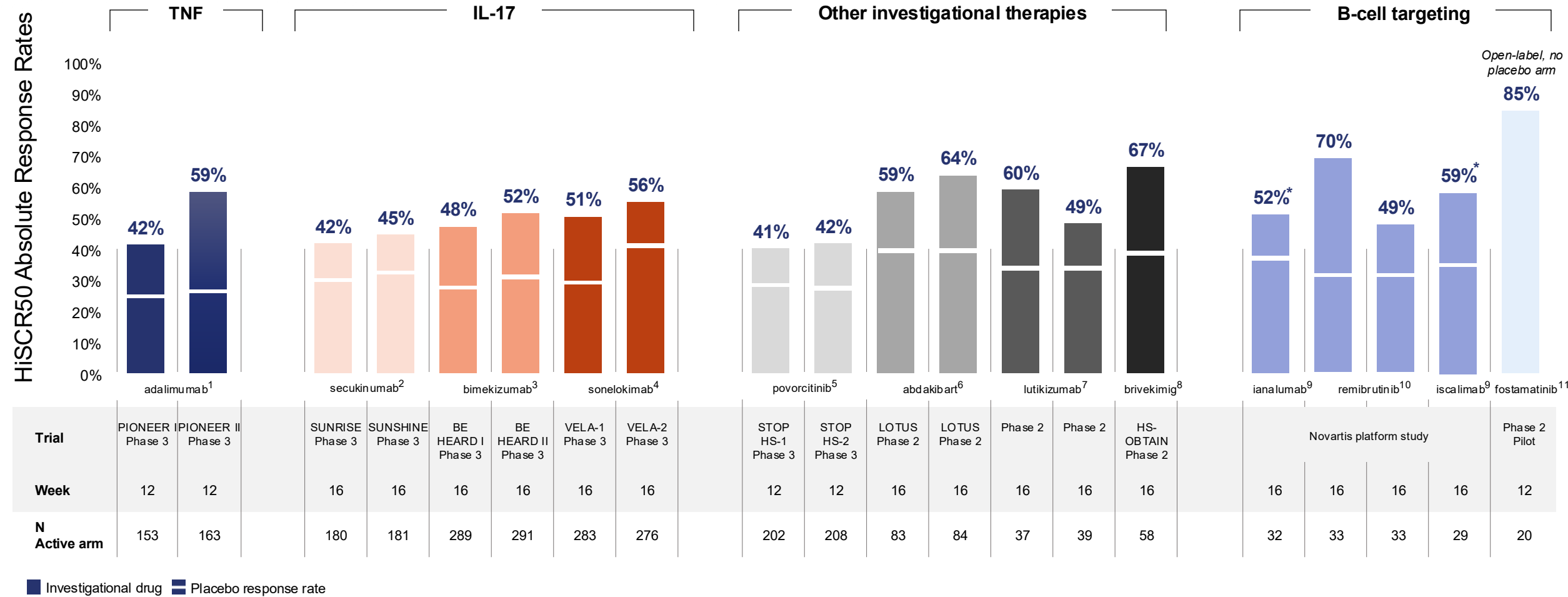
(*) Data derived from a randomized, placebo-controlled, multi-arm hidradenitis suppurativa platform study (ClinicalTrials.gov Identifier: NCT03827798). Reported results are based on information posted on ClinicalTrials.gov (as of 09-Jan-2026) and are subject to sponsor quality review and potential update. 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 arms shown

Sources: 1. ClinicalTrials.gov. NCT03827798. 2026; 2. Novartis. AAD Annual Meeting. 2024.

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



* 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.

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)

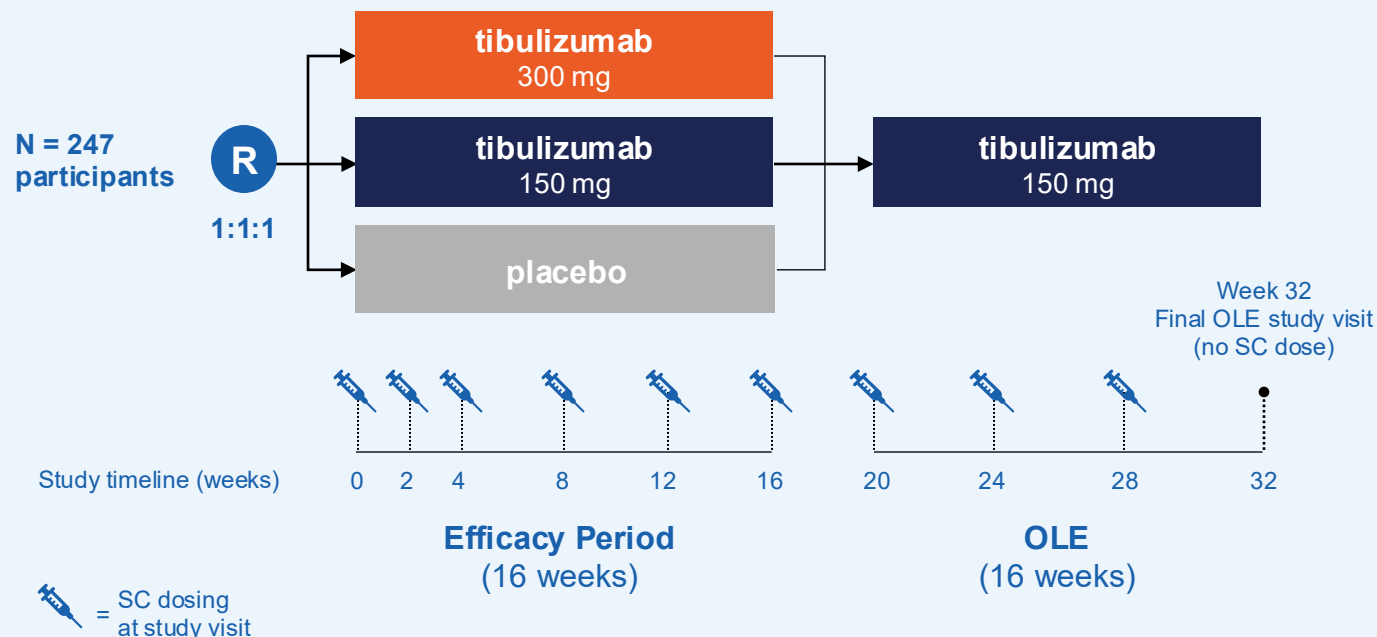


BAFF

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



Key Inclusion / Exclusion Criteria

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

Efficacy Endpoints

Primary endpoint

- Percent change from baseline in AN count at Week 16

Additional endpoints*

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

Systemic Sclerosis (SSc)

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

approximately

300,000

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



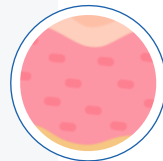
No therapies are approved that comprehensively address the multisystem pathology of SSc^{*2}

Characterized by Chronic Inflammation and Fibrosis Across Organs



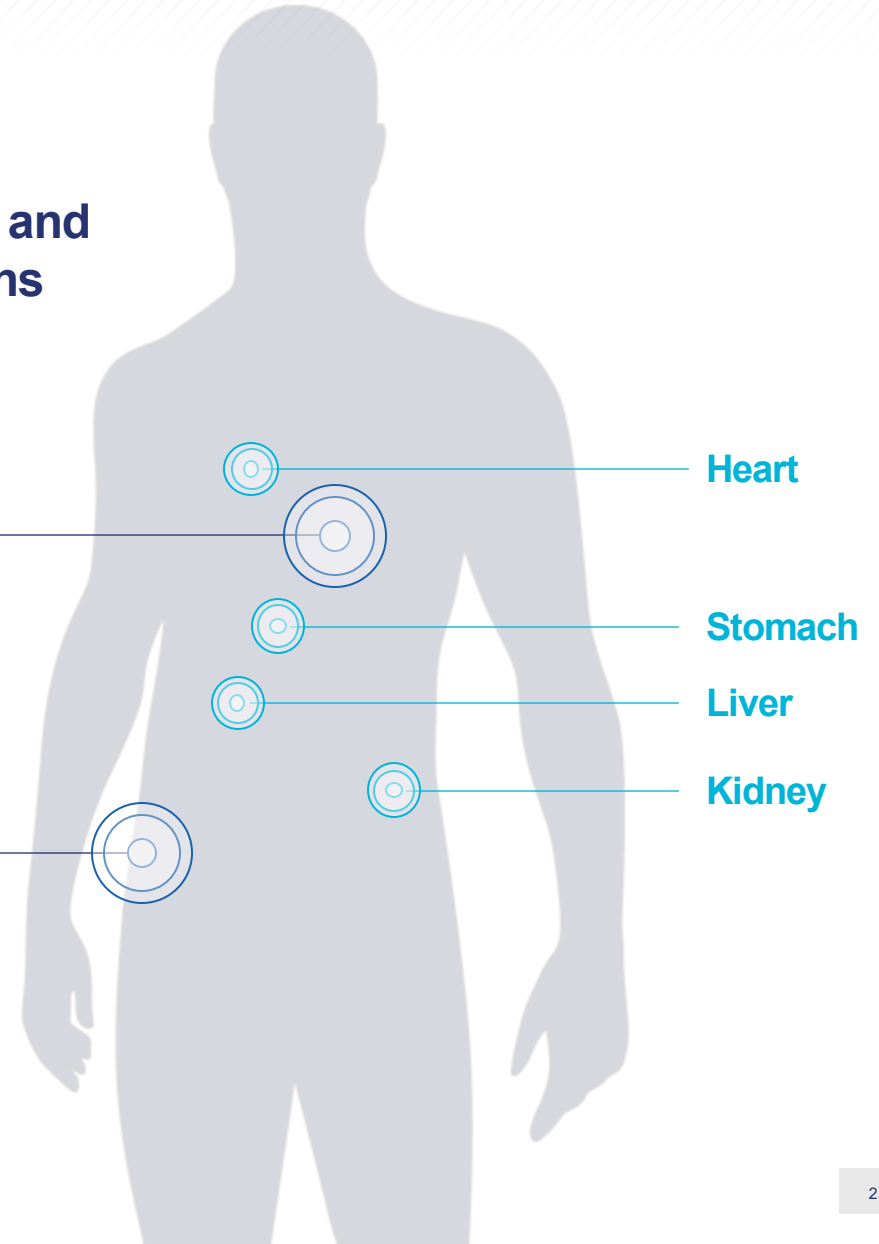
Lungs

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



Skin

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



Heart

Stomach

Liver

Kidney

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

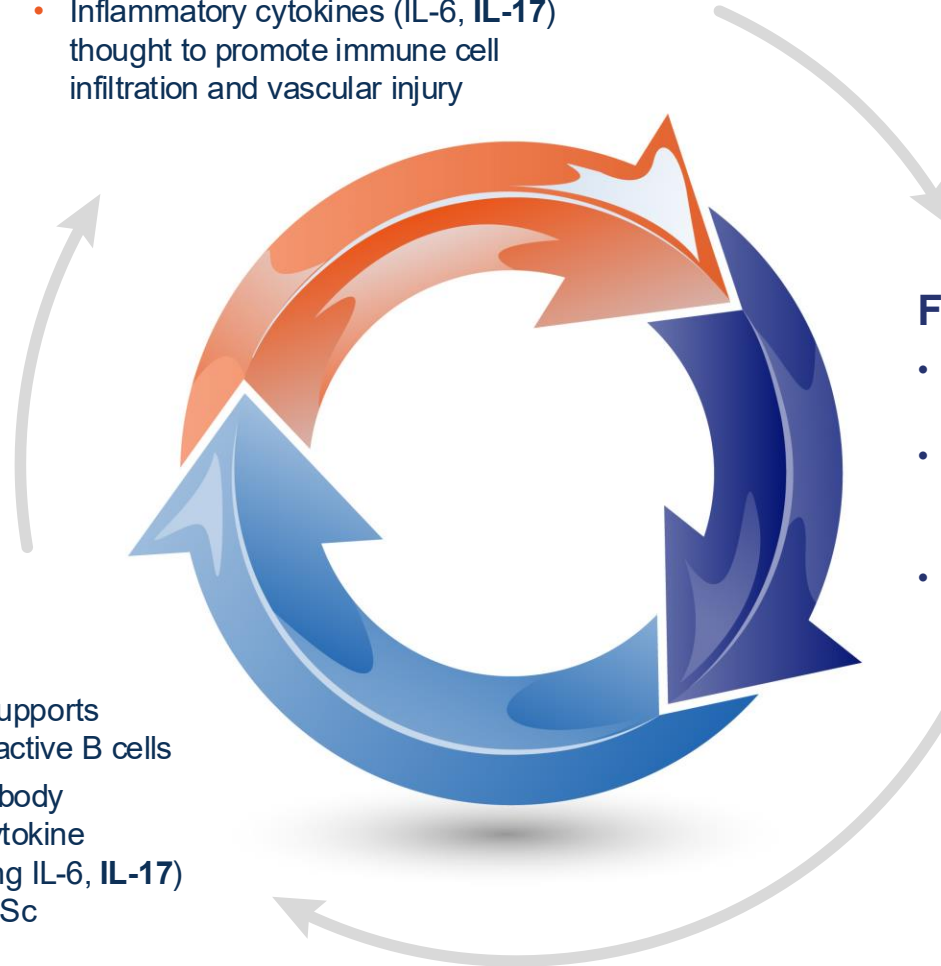
- Immune-mediated endothelial dysfunction contributes to impaired blood flow
- Inflammatory cytokines (IL-6, **IL-17**) thought to promote immune cell infiltration and vascular injury

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

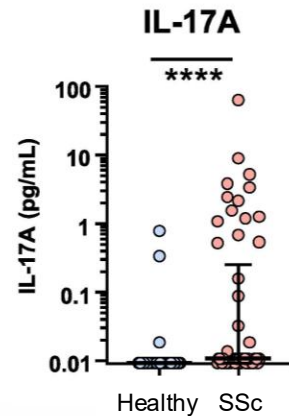
- **BAFF** signaling supports survival of autoreactive B cells
- Elevated autoantibody production and cytokine networks (including IL-6, **IL-17**) are observed in SSc



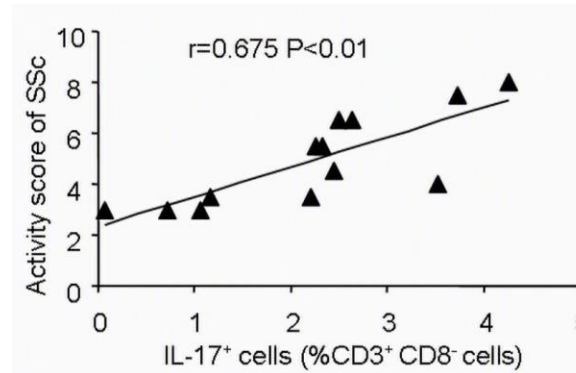
IL-17A is increased in SSc blood and skin and is linked to active and early disease

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

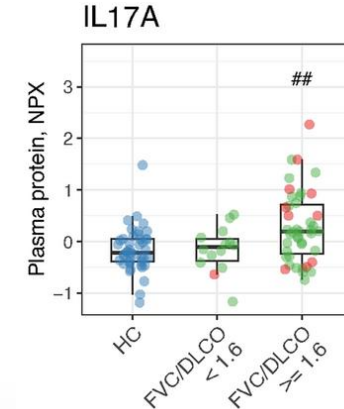
IL-17A cytokine elevated in SSc¹



Blood IL-17+ cells correlate with disease activity²

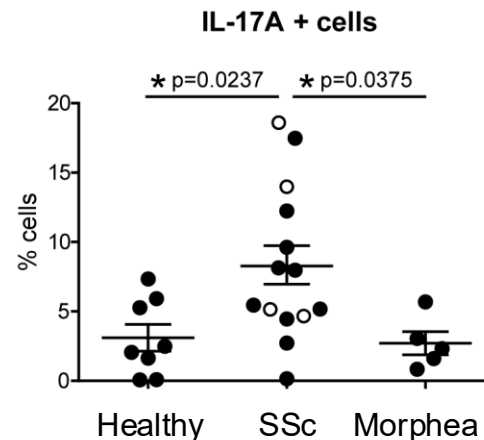


Serum IL-17A a predictor and correlate of pulmonary vascular disease^{1,3}

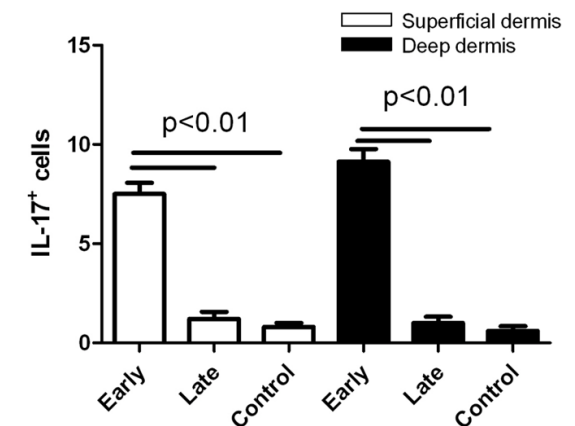


Elevations of IL-17-expressing T cells in skin correlate with early disease

IL-17A+ cells increased in SSc skin^{4,5}

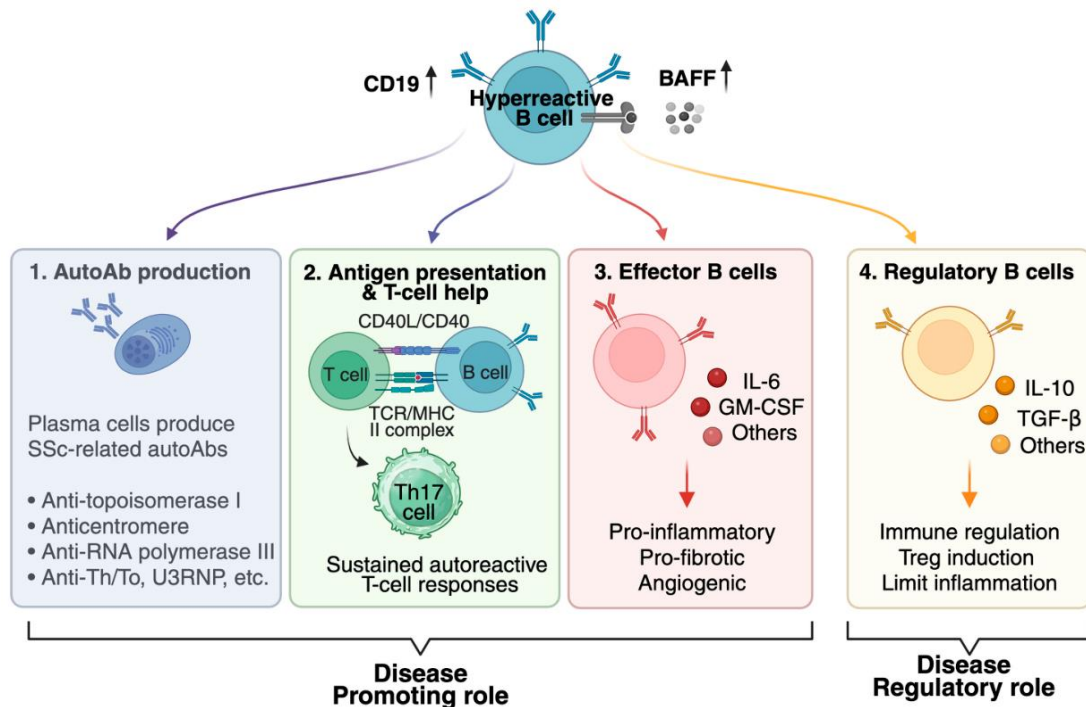


IL-17A+ cells increased in early disease skin²



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

Hypothesized disease-promoting roles of B cells³



Belimumab modulated gene profiles in post-treatment SSc skin¹

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



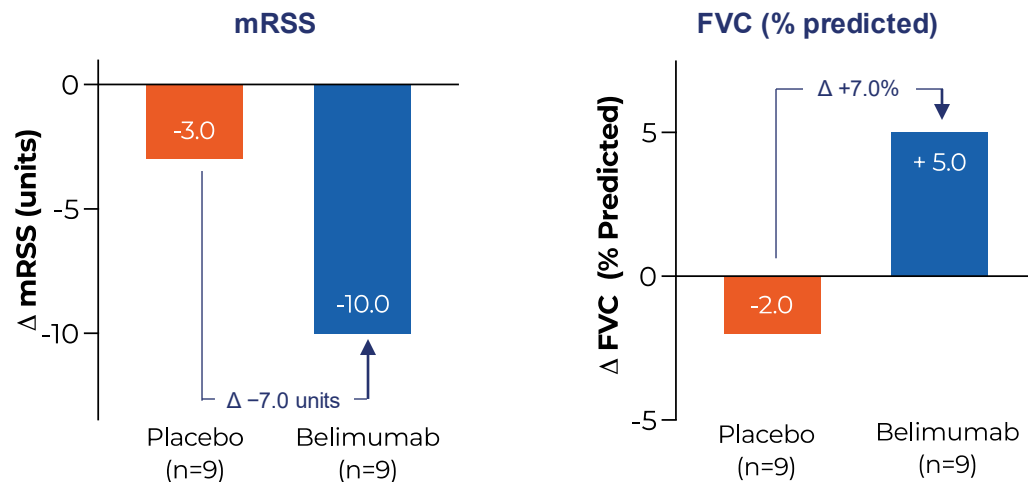
IL-17 and BAFF blockade have each shown improvement in skin and lung outcomes in SSc

BAFF ANTAGONIST

Belimumab¹

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

Phase 2 belimumab IIT (52 weeks)¹

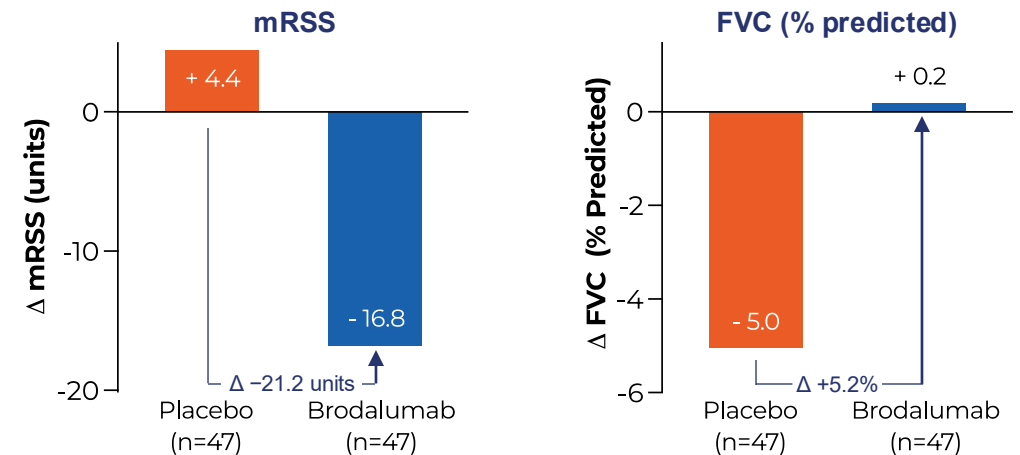


IL-17 RECEPTOR ANTAGONIST

Brodalumab²

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

Phase 3 brodalumab trial (24 weeks)²



Phase 2 study ongoing in SSc; enrollment completed



Key Inclusion Criteria

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

Endpoints and Stratification

Primary endpoint

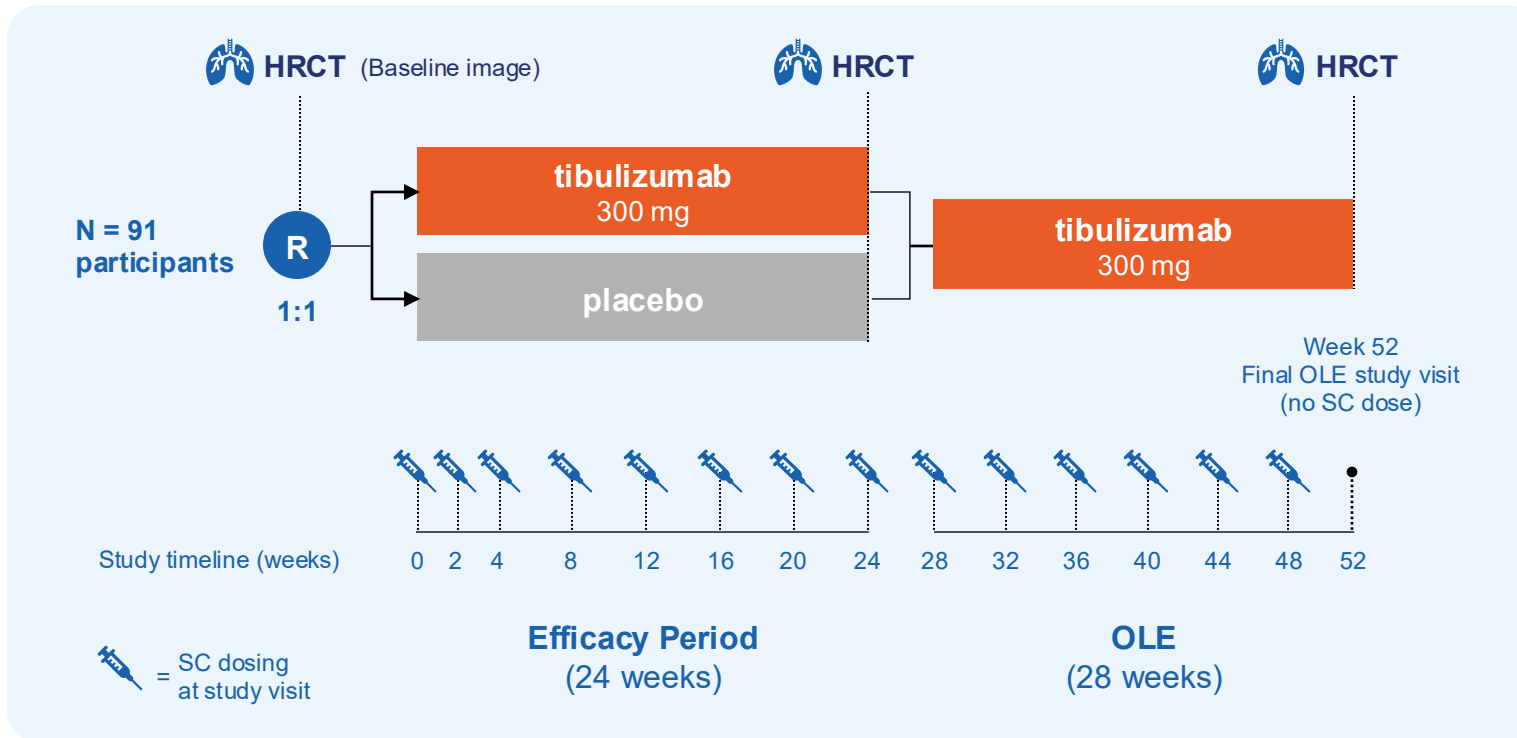
- Change from baseline in mRSS at Week 24

Additional endpoints*

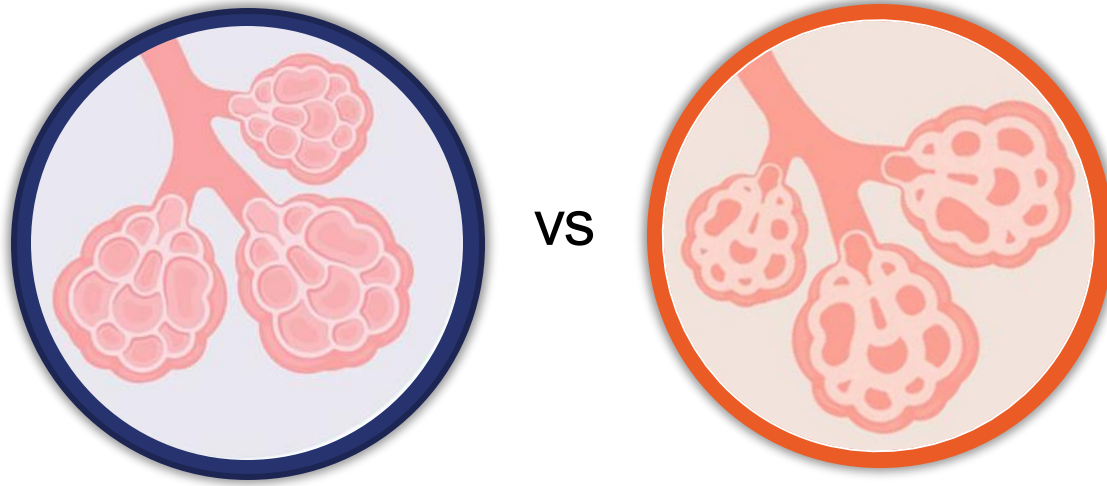
- qHRCT lung imaging
- FVC
- HAQ-DI
- Revised CRISS

Stratification factors

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



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



Healthy Lung

Fibrotic Lung

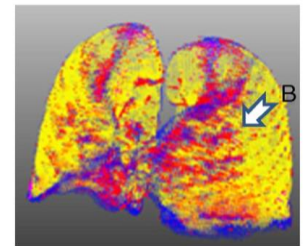
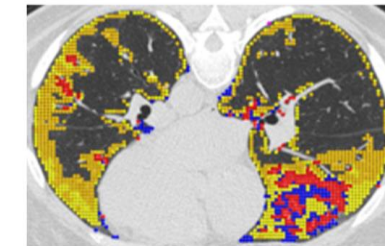
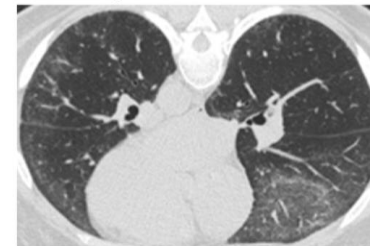
ILD includes various pulmonary disorders marked by inflammation and progressive lung fibrosis, resulting in restrictive lung function and impaired gas exchange.

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

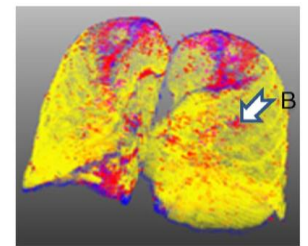
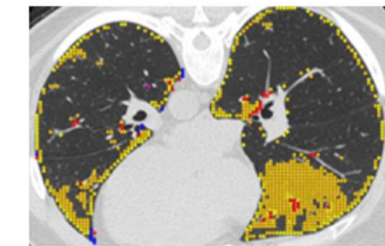
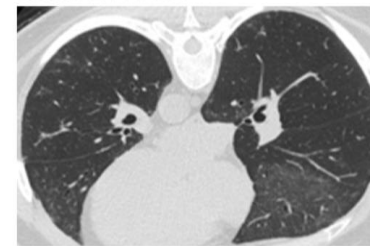
QILD by HRCT provides a sensitive measure of lung involvement, detecting changes as small as 2%.¹

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

Screening HRCT



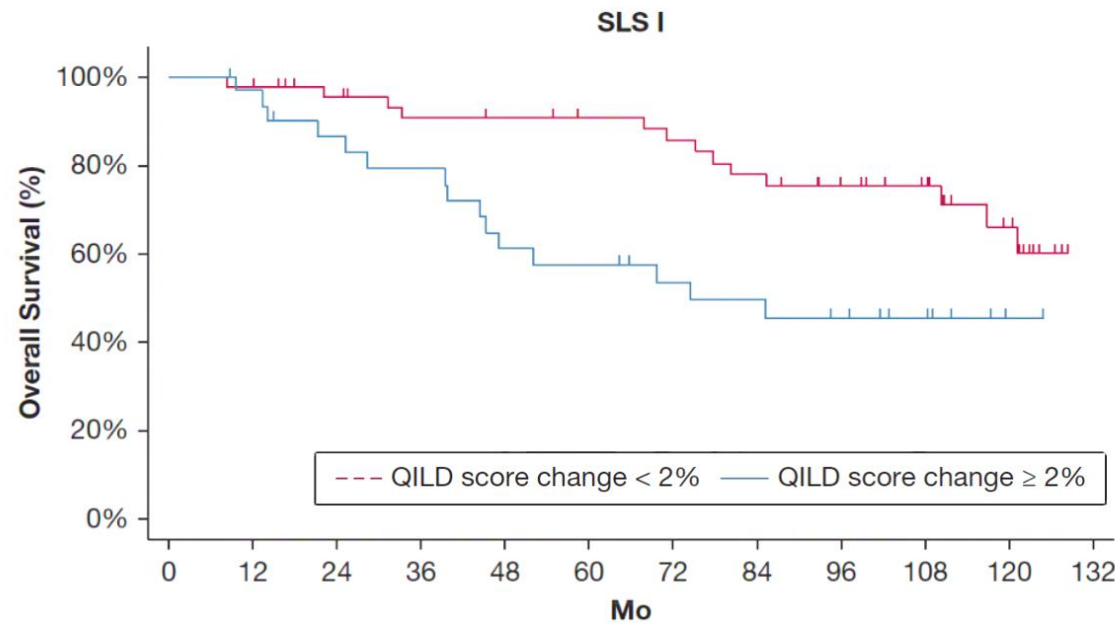
Month 24 HRCT



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

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

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



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

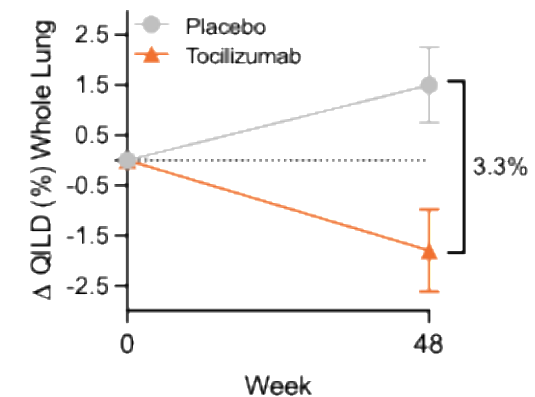
Tocilizumab Phase 3 in SSc-ILD

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

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

Patients were also evaluated by HRCT for QILD:⁵

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



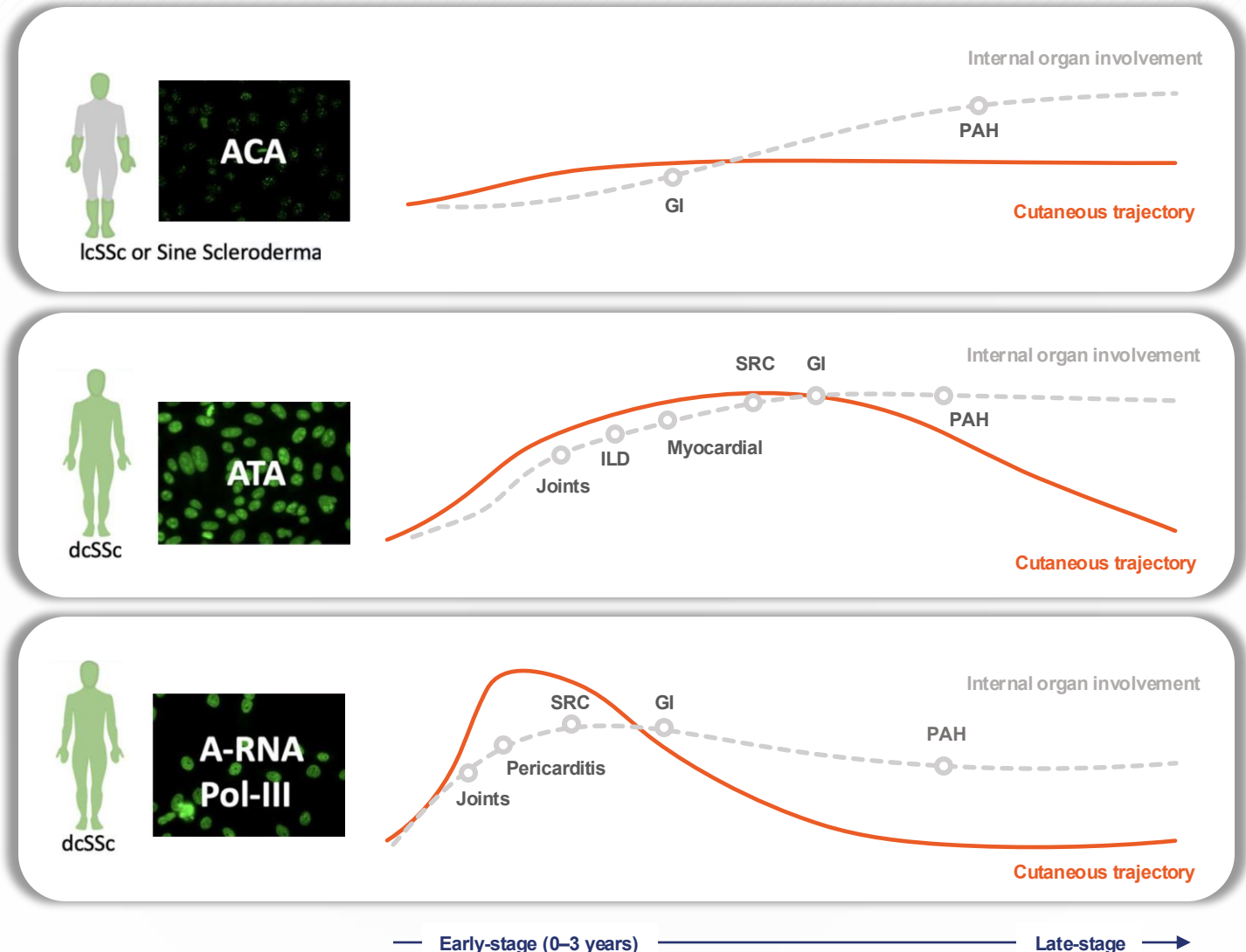
Published SSc disease trajectories validate our Phase 2 enrollment strategy

What the data show

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

Why our design fits

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





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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

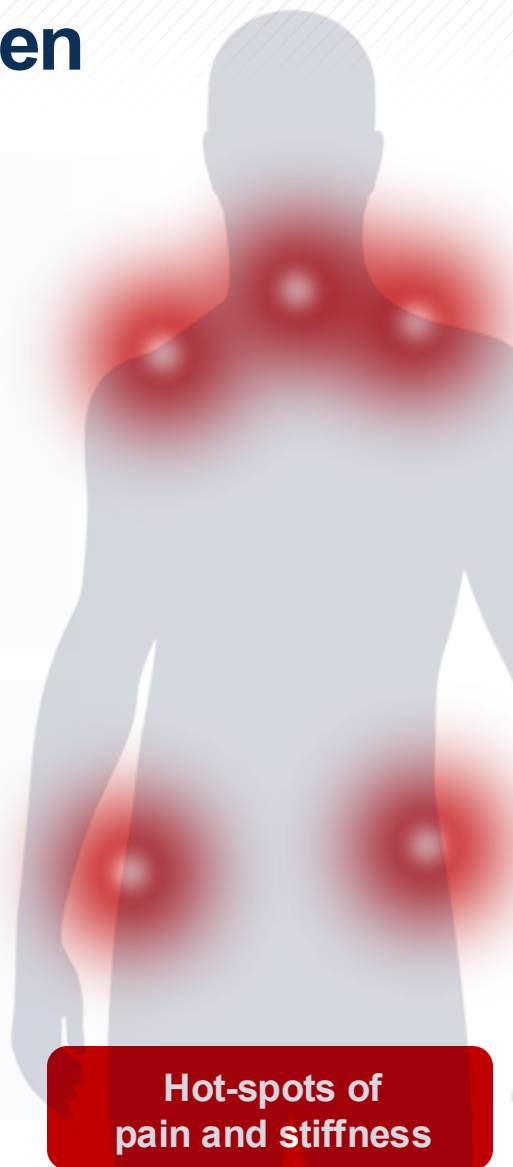
- Almost exclusively patients over 50; peak onset at ages 70–80^{2,5}
- Women are more likely to develop PMR than men⁵
- Highly comorbid population⁶

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}



Hot-spots of pain and stiffness

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



Today, only 1 FDA-approved biologic²
PMR is an underserved and underpenetrated disease

Steroid-dependency³
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

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



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

Over
50%¹

Relapse on steroid taper

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

Approx.
6 Yrs²

Median time to permanent glucocorticoid discontinuation

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

“

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

- PMR Patient⁴

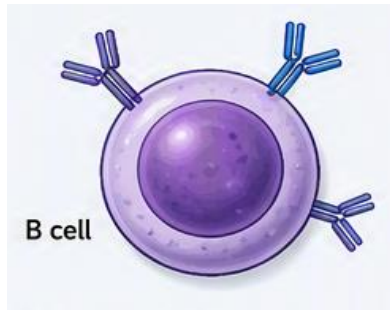
“

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

- PMR Patient⁴

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

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

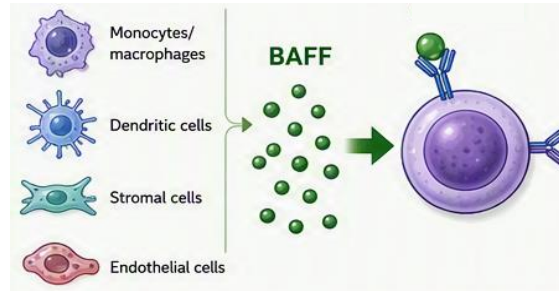


Circulating B cells:

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

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

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



Serum BAFF is:

Elevated in newly diagnosed PMR^{1,2}

Decreased during remission^{1,2}

Highly correlated with ESR, CRP, and IL-6²

↓ may lead to

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

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



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

Elevated in circulation^{3,4}

Present in synovial fluid^{3,4}

↓ may lead to

- ↑ IL-6, IL-8, TNF, and other inflammatory mediators
- ↑ Number and activation of myeloid cells

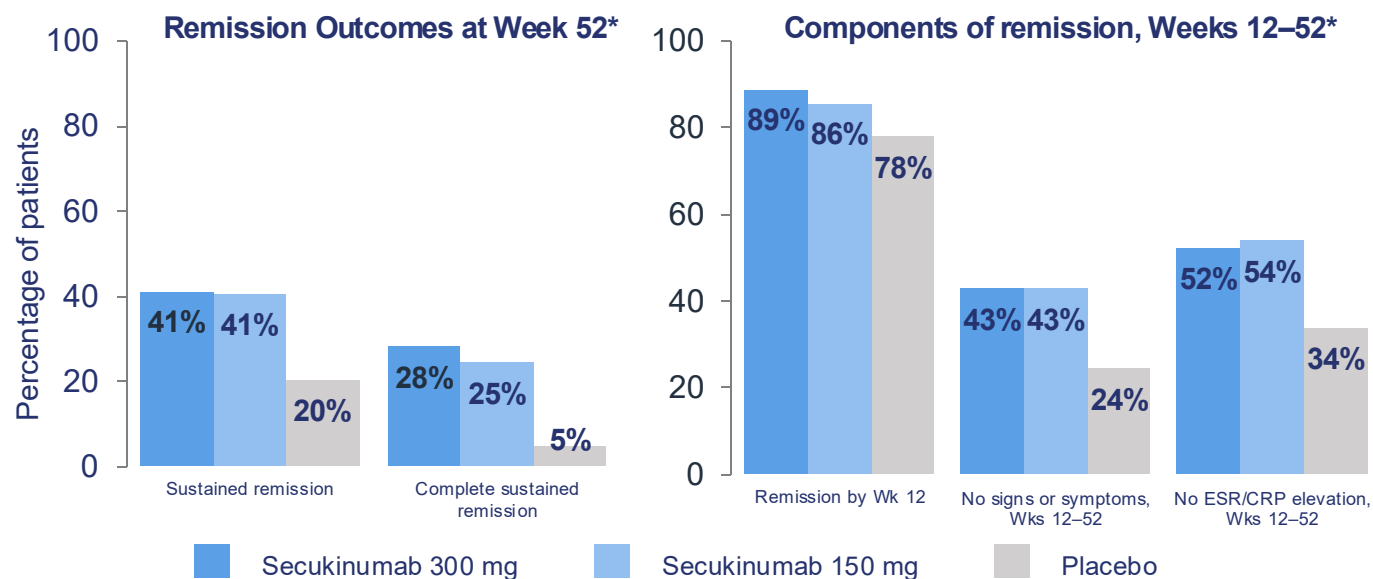
IL-17 and B-cell-targeted therapies have shown positive glucocorticoid-sparing effects in PMR

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

Secukinumab¹

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

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

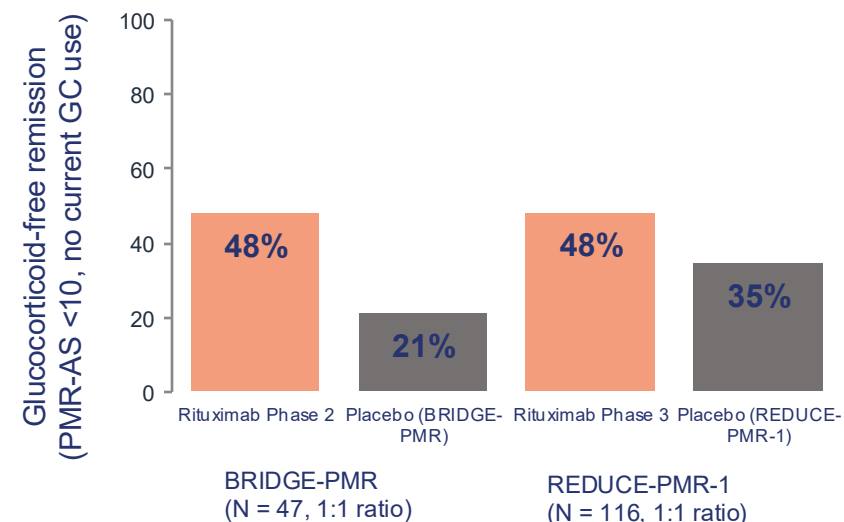


Anti-CD20 – new/relapsing PMR (Phase 2) and new-only PMR (Phase 3)

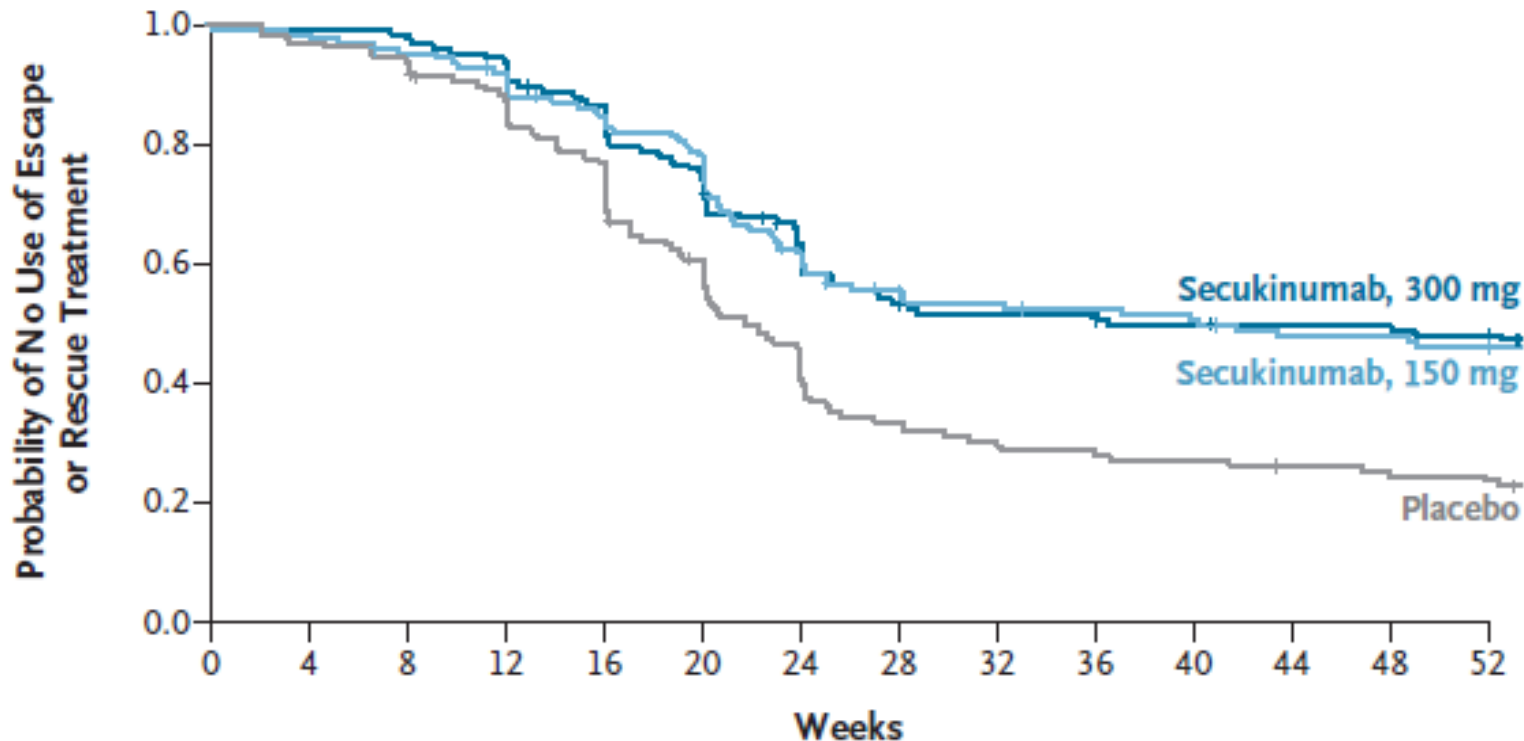
Rituximab

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

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

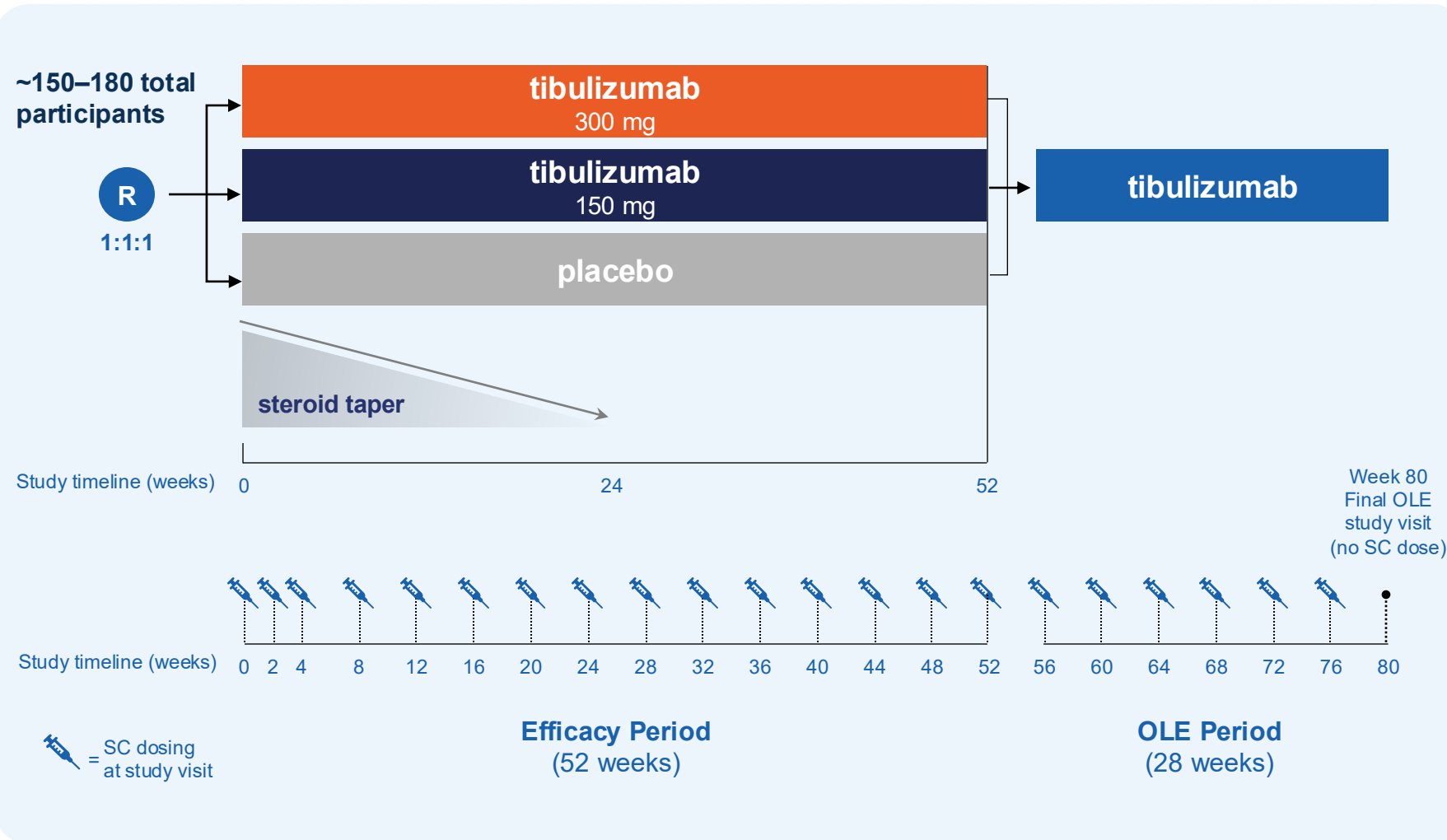


Despite IL-17 advances, the unmet need for PMR remains significant



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

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



NEXUS|PMR

Key Inclusion Criteria

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

Key Efficacy Endpoints

Primary endpoint

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

Key secondary endpoints

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



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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

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

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

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

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

IL-7

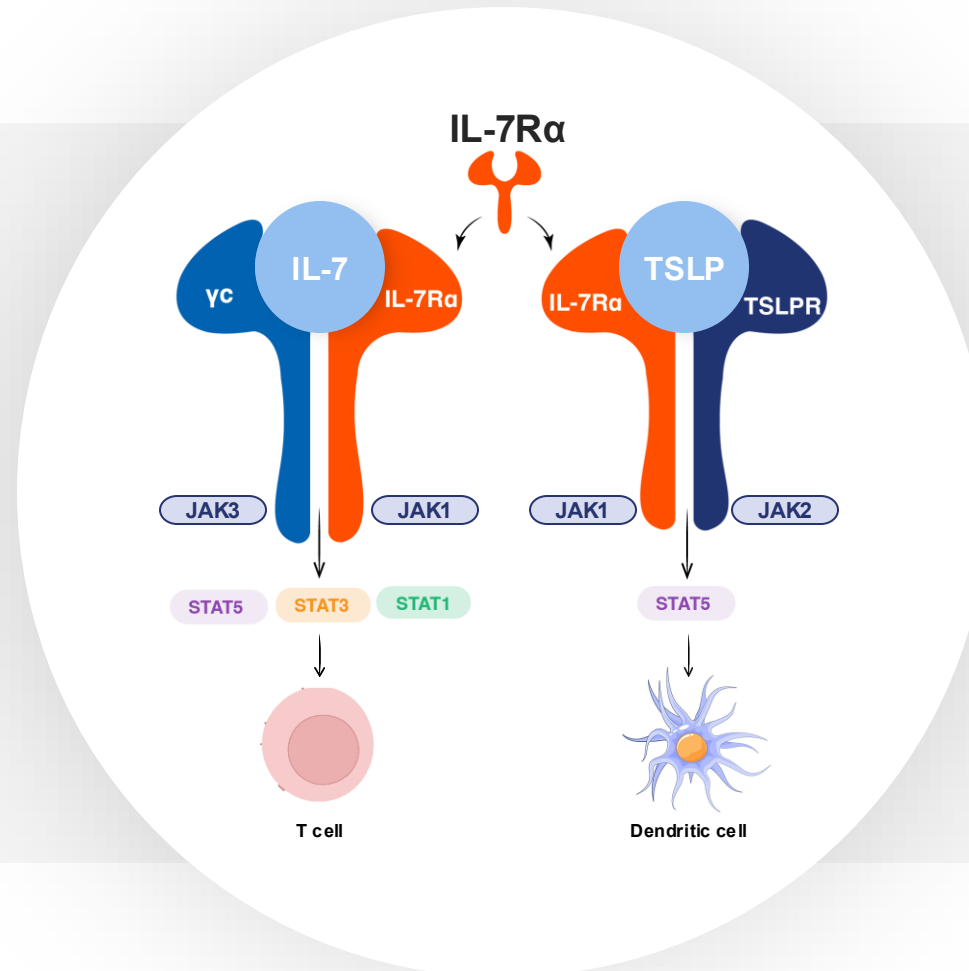
IL-7R α combines with the common gamma chain (γ c) to form the IL-7 receptor complex

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

TSLP

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

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



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

About IL-33 and torudokimab

IL-33

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

Torudokimab

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

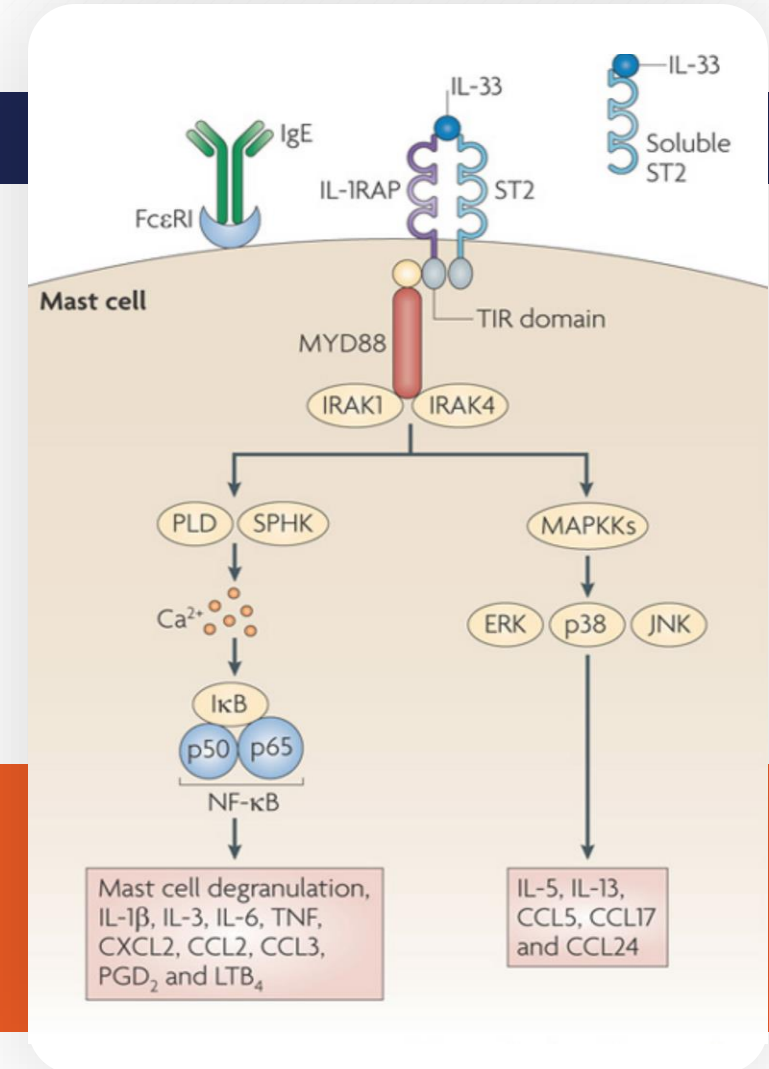
Clinical Results

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

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

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

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



Key Takeaways

Clear differentiation

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

Defined, anticipated clinical catalysts

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

Portfolio optionality

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



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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	HAQ-DI	Health Assessment Questionnaire Disability Index	mRNA	messenger RNA	RU	relative units
ACA	anti-centromere antibodies	HCP	healthcare professional	mRSS	Modified Rodnan Skin Score	SC	subcutaneous
ACR	American College of Rheumatology	HISCR50	Hidradenitis Suppurativa Clinical Response $\geq 50\%$	N	number of subjects	scFv	single-chain variable fragment
ADA	anti-drug antibody	HISCR75	Hidradenitis Suppurativa Clinical Response $\geq 75\%$	NETosis	neutrophil extracellular trap formation	SHAQ-DI	Scleroderma Health Assessment Questionnaire Disability Index
AN	abscess and inflammatory nodule	HRCT	high-resolution computed tomography	NPX	normalized protein expression	sHISCR50	Simplified Hidradenitis Suppurativa Clinical Response (50%)
A-RNA Pol-III	anti-RNA polymerase 3 antibodies	HS	hidradenitis suppurativa	NRS	Numeric Rating Scale	sHISCR75	Simplified Hidradenitis Suppurativa Clinical Response (75%)
ATA	anti-topoisomerase antibodies	I&D	incision and drainage	NS	not significant	sHISCR90	Simplified Hidradenitis Suppurativa Clinical Response (90%)
BAFF	B-cell activating factor	IgA1, D, G	immunoglobulin A1, D, and G	OLE	open-label extension	SLE	systemic lupus erythematosus
BAFF-R	B-cell activating factor receptor	IHS4	International Hidradenitis Suppurativa Severity Score System	P	p-value	SLS	Scleroderma Lung Study
BTK	Bruton's tyrosine kinase	IIT	investigator-initiated trial	PAH	pulmonary arterial hypertension	SoC	standard of care
BTKi	Bruton's tyrosine kinase inhibitor	IL-1β	interleukin 1 beta	PASI	Psoriasis Area and Severity Index	SRC	scleroderma renal crisis
CCL2/3/5/17/24	C-C motif chemokine ligand 2/3/5/17/24	IL-3	interleukin 3	PASI 90	Psoriasis Area and Severity Index 90% improvement	SSc	systemic sclerosis
CD3/8/19/20/40/45	cluster of differentiation 3/8/19/20/40/45	IL-5	interleukin 5	PBO	placebo	SSc-ILD	systemic sclerosis-associated interstitial lung disease
CFB	change from baseline	IL-6	interleukin 6	PD	pharmacodynamics	ST2	suppression of tumorigenicity 2
COPD	chronic obstructive pulmonary disease	IL-7Rα	interleukin 7 receptor alpha	PEA	primary endpoint assessment	T2D	type 2 diabetes
CRIS	Combined Response Index in Systemic Sclerosis	IL-8	interleukin 8	PGD₂	prostaglandin D2	TARC	thymus and activation-regulated chemokine
CRP	C-reactive protein	IL-10	interleukin 10	PK	pharmacokinetics	TGF-β	transforming growth factor beta
CXCL2	C-X-C motif chemokine ligand 2	IL-17	interleukin 17	pM	picomolar	Th17	T helper 17 cells
CyTOF	cytometry by time-of-flight	IL-18	interleukin 18	PMR	polymyalgia rheumatica	TLS	tertiary lymphoid structure
dcSSc	diffuse cutaneous systemic sclerosis	IL-33	interleukin 33	PMR-AS	Polymyalgia Rheumatica Activity Score	TNF	tumor necrosis factor
DLCO	diffusing capacity of the lung for carbon monoxide	ILD	interstitial lung disease	PsO	psoriasis	TNF-α	tumor necrosis factor alpha
DLQI	Dermatology Life Quality Index	JAK	Janus kinase	Q2W	once every 2 weeks	TNFi	tumor necrosis factor inhibitor
EOS	end of study	K_o	dissociation constant	Q4W	once every 4 weeks	TSLP	thymic stromal lymphopoietin
ESR	erythrocyte sedimentation rate	IcSSc	limited cutaneous systemic sclerosis	qHRCT	quantitative high-resolution computed tomography	TSLPR	thymic stromal lymphopoietin receptor
EULAR	European Alliance of Associations for Rheumatology	LPA1	lysophosphatidic acid receptor 1	QILD	quantitative interstitial lung disease	VAS	visual analog scale
FDA	Food and Drug Administration	LTB₄	leukotriene B4	QLF	quantitative lung fibrosis		
FVC	forced vital capacity	mAb	monoclonal antibody	QOL	quality of life		
GC	glucocorticoid	MMF	mycophenolate mofetil	R	randomization		
GCA	giant cell arteritis	MoA	mechanism of action	RA	rheumatoid arthritis		
GI	gastrointestinal			RNA	ribonucleic acid		



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