zurabio

Corporate Overview with
ZB-106 Deep-Dive

Building the Next Immunology Leader

April 2023

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Investment Highlights

Experienced and proven leadership

- Experienced drug developers and asset hunters led by ex-Arena leaders
- Product approvals & commercialization
- Significant corporate transactions

Multiple Phase 2 programs

- Validated mechanisms pursuing innovative new development paths
- Potential Best in Class attributes combined with strategic clinical development to enhance differentiation

Track Record of Execution

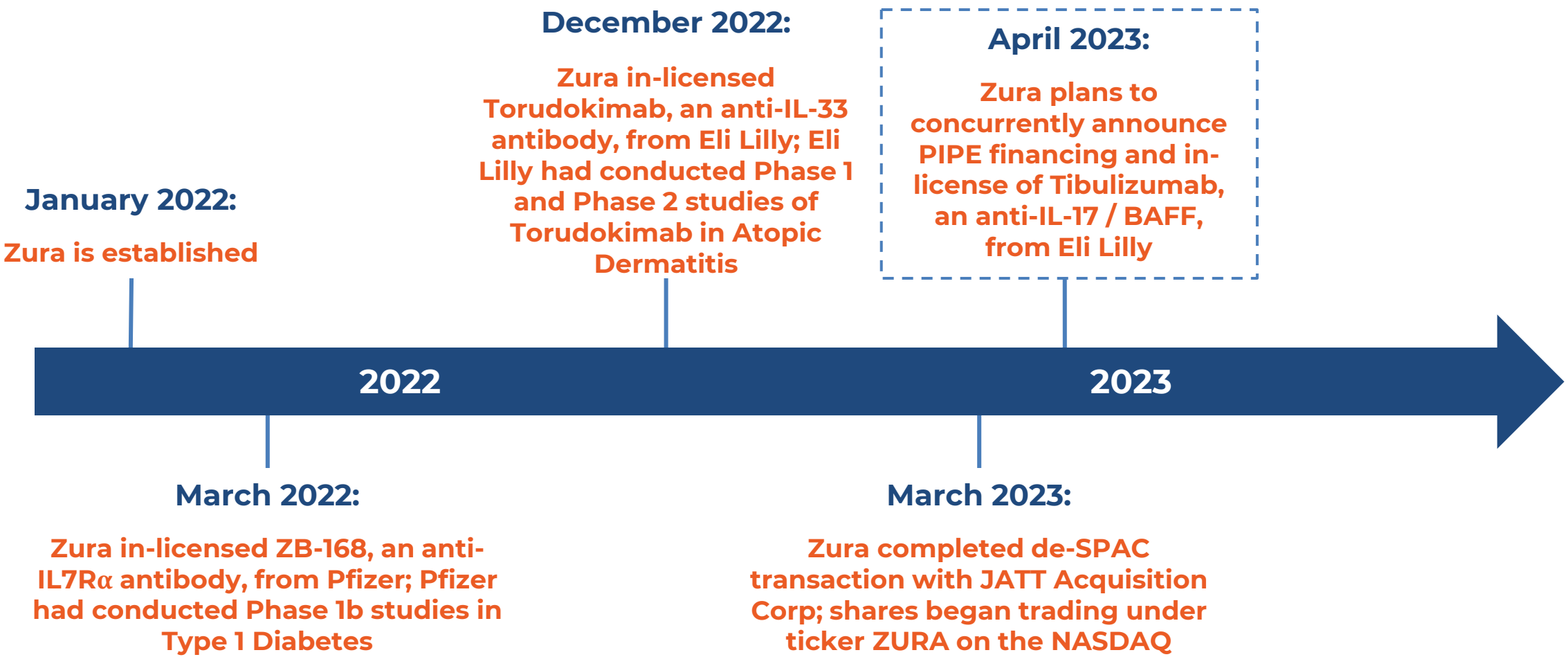
- Deep relationships with KOL community
- Prior CRO leadership history within team enhances our Sponsor status
- Recent precedent of on-time execution in crowded I&I spaces (UC, AD, etc.) includes absorbing COVID impact without delays

Strong and committed financial backing with long-term alignment

- Investors aligned with long-term commitment
- Successful PIPE transformation, including $65M in funding through Business Combination

Abbreviations: AD, Atopic Dermatitis; CRO, Contract Research Organization; KOL, Key Opinion Leader; UC, Ulcerative colitis

Zura Company Timeline



An Experienced Leadership Team from A to Z

Amit Munshi
Chairman

ARENA PHARMACEUTICALS
EPIRUS BIOPHARMACEUTICALS
PERCIVIA
AMGEN

Someit Sidhu M.D.
Chief Executive Officer and Director

McKinsey & Company
IZANA BIOSCIENCE
JATT ACQUISITION CORP
PATHIOS THERAPEUTICS

Chris Cabell M.D.
Chief Medical Officer and
Head of Research and Development

ARENA PHARMACEUTICALS
QuintilesIMS
PULMATRIX
emergent biosolutions

Kim Davis
Chief Legal Officer

ARENA PHARMACEUTICALS
kaléo
Impax
AMGEN

Verender Badial
Chief Financial Officer

JATT ACQUISITION CORP
SOCIETE GENERALE
RBS
Rothschild & Co
ABN·AMRO

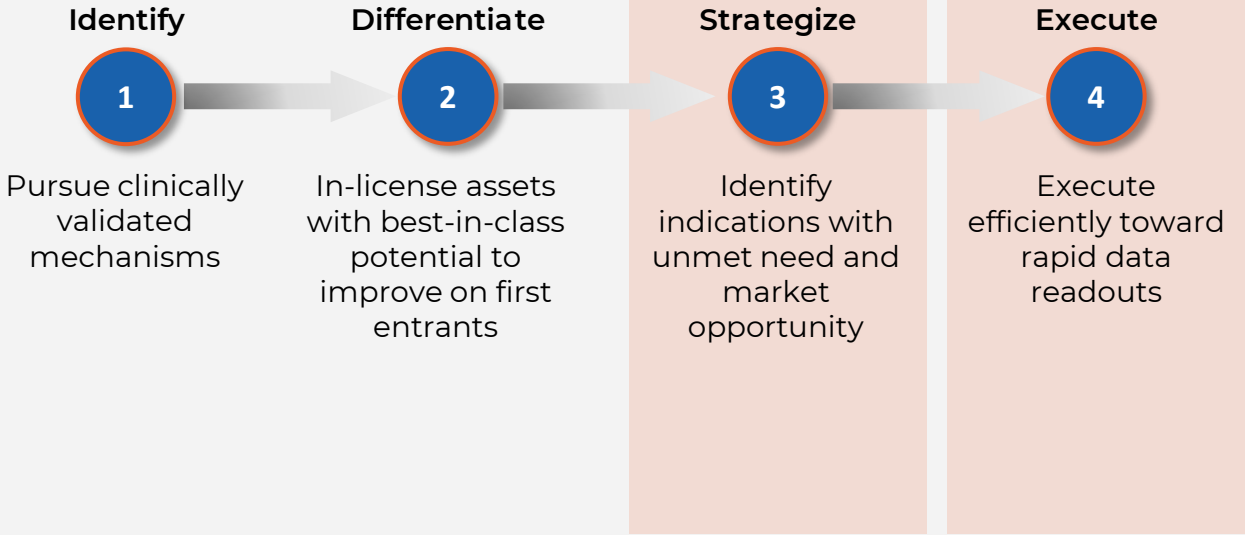
Mike Howell Ph.D.
Chief Scientific Officer and
Head of Translational Science

DermTech
MedImmune
Incyte
Boehringer Ingelheim

Gary Whale Ph.D.
Chief Technology Officer

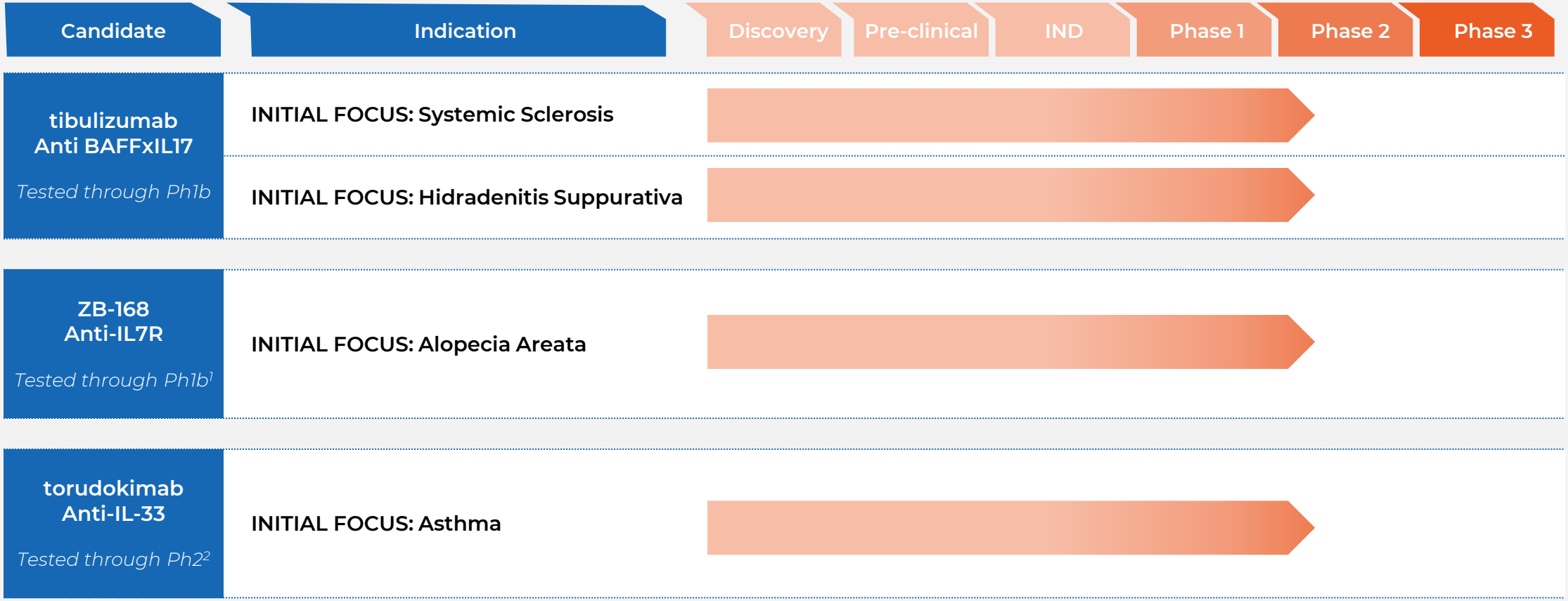
emergent biosolutions
EUSAPharma

Zura's Business Development Aligns with its Philosophy of Developing Validated Mechanisms in Novel Ways



Clinical stage pipeline targeting key immunology pathways

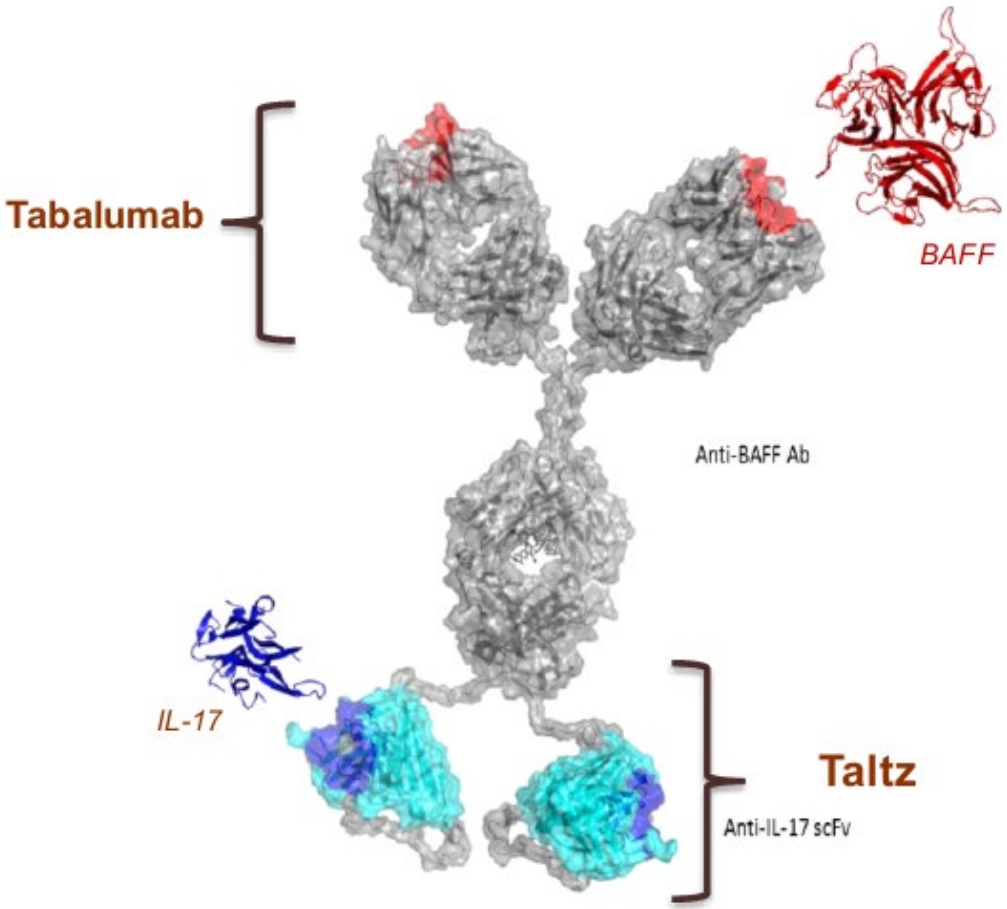
Current financing will bring in ZB-106 as lead asset into Zura



Note: Clinical development plan subject to confirmation, pending regulatory and further clinical feedback

1. Herold et al. 2019. JCI Insight, 2. Laquer et al. 2022. BrJDerm

Tibulizumab (ZB-106) is a Potential First-in-Class, Dual Antagonist Combining tabalumab and ixekizumab (TALTZ®)



ZB-106 is an IgG-scFv engineered by the fusion of ixekizumab (TALTZ®) and tabalumab[1,2]

- ZB-106 neutralizes IL-17A or BAFF regardless of whether the other binding sites are occupied
- ZB-106 binds in the same way as Taltz and tabalumab with the same number of binding sites
- Activity is mediated through direct target engagement and not ADCC
- Terminal half-life ~26 days

Robust existing clinical and non-clinical data package

- Two Phase 1b studies completed by Eli Lilly (RA and Sjögren's Syndrome)
- 78 subjects have been dosed with ZB-106
  - 57 subjects = single dose; 21 subjects = multiple dose up to 12 weeks
- Chronic toxicity studies completed with no adverse findings

Durable and deep IL-17 and BAFF signaling blockade observed with sub cutaneous dosing every 4 weeks

- At target Q4W doses BAFF and IL-17 achieve maximum receptor occupancy

Low rate of immunogenicity

- Across 78 subjects exposed to ZB-106, only 1 subject tested positive for Anti-drug Antibodies (ADAs)

Safety profile to date appears to be consistent with ixekizumab (TALTZ®) and IL-17A class

1. Liu et al. 2016. *J Inflam Research*; 2. Manetta et al. 2014. *J Inflam Research*; 3. Benschop et al. 2019. MAbs

Eli Lilly Deal Terms

Terms of Tibulizumab (ZB-106) License

Upfront:

- Mid-teens upfront cash payment for exclusive global license

Milestones:

- Single digit development milestones
- Back-end milestones triggered at approval and sales-based
- Single to low double-digit royalties on net sales

Other Key terms:

- No development in select indications (a) plaque psoriasis, (b) pediatric psoriasis, (c) genital psoriasis, (d) psoriatic arthritis, (e) ankylosing spondylitis, (f) non-radiographic axial spondylarthritis, (g) chronic spontaneous urticaria, and (h) juvenile idiopathic arthritis
- Right of first negotiation for Lilly upon completion of Phase 2b data
- Most patents expire in April 2033, but US patent expires June 2034
- Data protection is expected from marketing approval for 12 (US), 10 (EU), and 8 (JP) years

Clinical Development

Upcoming milestones

tibulizumab (BAFFx17)

- 2 clinical studies for ZB-106
- Initiate phase 2 study in **Systemic Sclerosis** (2024)
- Initiate phase 2 study in **Hidradenitis Suppurativa** (2024)

ZB-168 (IL-7Rα)

- Advance CMC program for ZB-168 ready to initiate clinical drug product at scale (H1 2024)
- Enable targeted launch of clinical trials in H2 2024 pending expected **phase 2 external catalysts in Atopic Dermatitis, Ulcerative Colitis and Sjögrens Syndrome**

torudokimab (IL-33)

- Gain FDA Regulatory feedback and alignment for torudokimab on phase 2/3 designs in Asthma (H2 2023)
- Enable launch of clinical trials in H2 2024 pending expected **phase 2 and 3 external catalysts in Asthma and COPD**

ZB-106:
Potential First-in-Class, Dual Antagonist Combining tabalumab and Taltz

Note: please see language in the Disclaimer regarding 'forward-looking statements'

IL-17 and BAFF Approved in Multiple Autoimmune Diseases

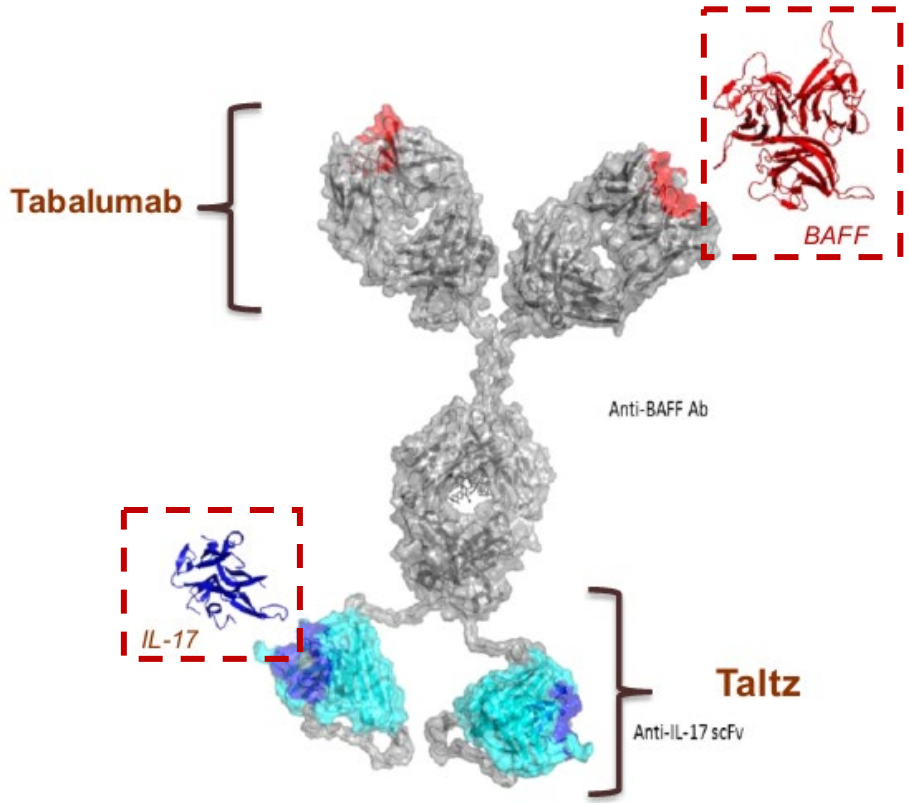
- **IL-17 and B-cell assets are widely recognized to have significant value**
- **ZB-106 represents an opportunity to pioneer a new approach to treating autoimmune diseases by directly addressing both aspects of immune inflammatory response**

		IL-17				IL-17 / BAFF	BAFF / TACI / APRIL			
	Company	NOVARTIS	Lilly	ACELYRIN	MoonLake	zurabio	GSK	RemeGen	CHINOOK THERAPEUTICS	vera therapeutics
	Asset	Cosentyx (secukinumab)	Taltz (ixekizumab)	Izokibep	Sonelokimab	ZB-106	Benlysta (belimumab)	Telitacicept	BION-1301	Atacicept
	MoA	IL-17A	IL-17A	IL-17A/A	IL-17A/F	IL-17A / BAFF	BAFF	TACI fc	APRIL	BAFF/APRIL
	Delivery	SC / IV	SC	SC	SC	SC	SC / IV	SC	SC	SC
Indications	**Plaque Psoriasis**	Approved	Approved		Ph3					
	Psoriatic Arthritis	Approved	Approved	Ph2b/3	Ph2					
	AS	Approved	Approved	Ph3						
	SLE / Lupus						Approved	App. China		
	HS	Filed		Ph2b/3	Ph2	Ph2 Ready				
	Lupus Nephritis	Ph3					Approved			Ph3
	Sjögren's							Ph3		
	IgAN							Ph2	Ph1/2	Ph2b/3
	Other			Uveitis (Ph2b/3)		Syst. Sclerosis (Ph2 Ready)	Syst. Sclerosis (Ph2)	MG (Ph3) RA (Ph3)		

Sources: Clinical Trials, Company Presentations, Wall Street Research and Evaluate Pharma.

ZB-106 is a Combination of Two Compounds that have Each Demonstrated Efficacy with an Established Safety Profile

- Taltz® (ixekizumab) is an approved anti IL-17 therapy with estimated peak sales >$3bn
- tabalumab is an anti-BAFF which has shown efficacy in some phase 3 trials



TABALUMAB

- **4,275 subjects dosed across 21 studies**
- **Statistically significant efficacy in a phase 3 study in SLE**
- **Demonstrated safety profile**

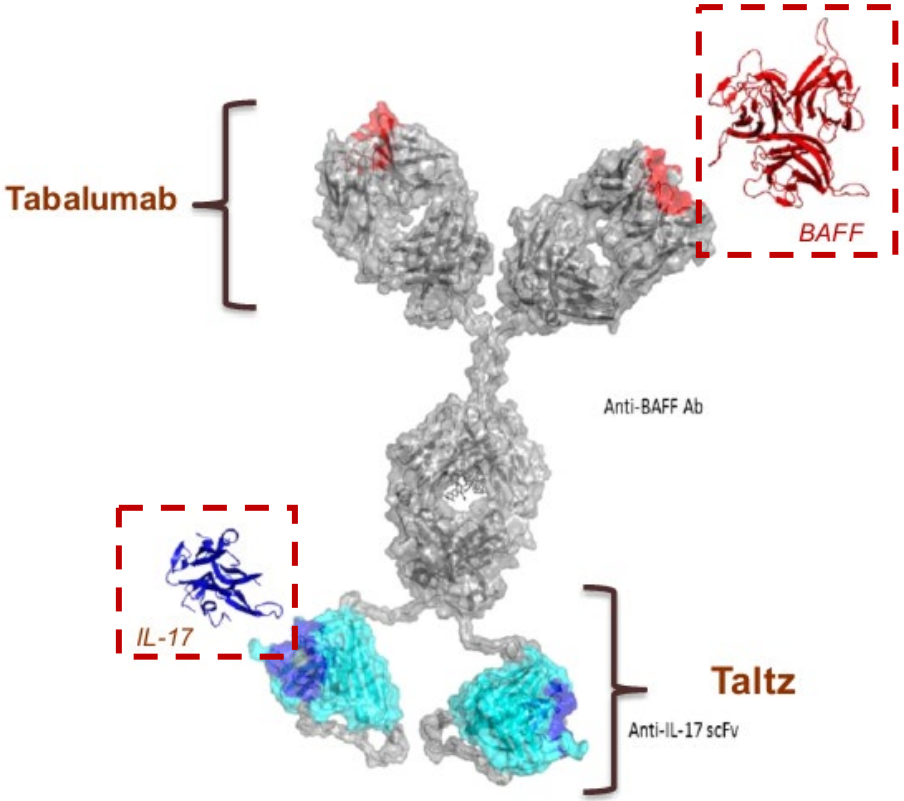
IXEKIZUMAB (TALTZ®)

- **Commercially approved drug with >$2.2bn of annual sales[1]**
- **Demonstrated efficacy in multiple indications**
- **Demonstrated safety profile**

1. Eli Lilly Annual Report

ZB-106 Has Broad Potential Therapeutic Applications

- Potential to be a first-in-class biologic in a number of autoimmune diseases where both BAFF and IL-17 are implicated in the pathology[1,2]



BAFF

- **Systemic Sclerosis**
- Sjögren's Syndrome
- Systemic Lupus Erythematosus

IL-17

- **Hidradenitis Suppurativa**
- Uveitis
- Bechet's Disease
- Lichen Planus
- Pustular Psoriasis
- Impetigo Herpetiformis
- Pityriasis Rubra Pilaris

1. Kaegi et al. 2020. Allergy; 2. Wu and Dao, 2022. JDermTreat

ZB-106 Disrupts IL-17 and/or BAFF-Mediated Inflammation

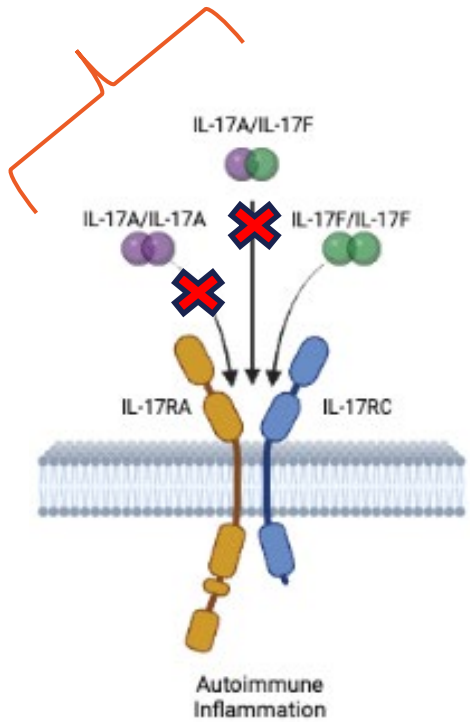
ZB-106 has the potential to treat diseases driven by B-cell and T-cell signaling

T-cell and B-cell synergy

- Multiple T-cell driven diseases remain sub-optimally treated despite the growth in “pure play” anti-IL-17 drugs
- B-cell driven diseases often have a T-cell driven autoimmune component contributing to the pathology
- Combined approach to address T-cell and B-cell drivers of autoimmunity has the potential to increase clinical benefit

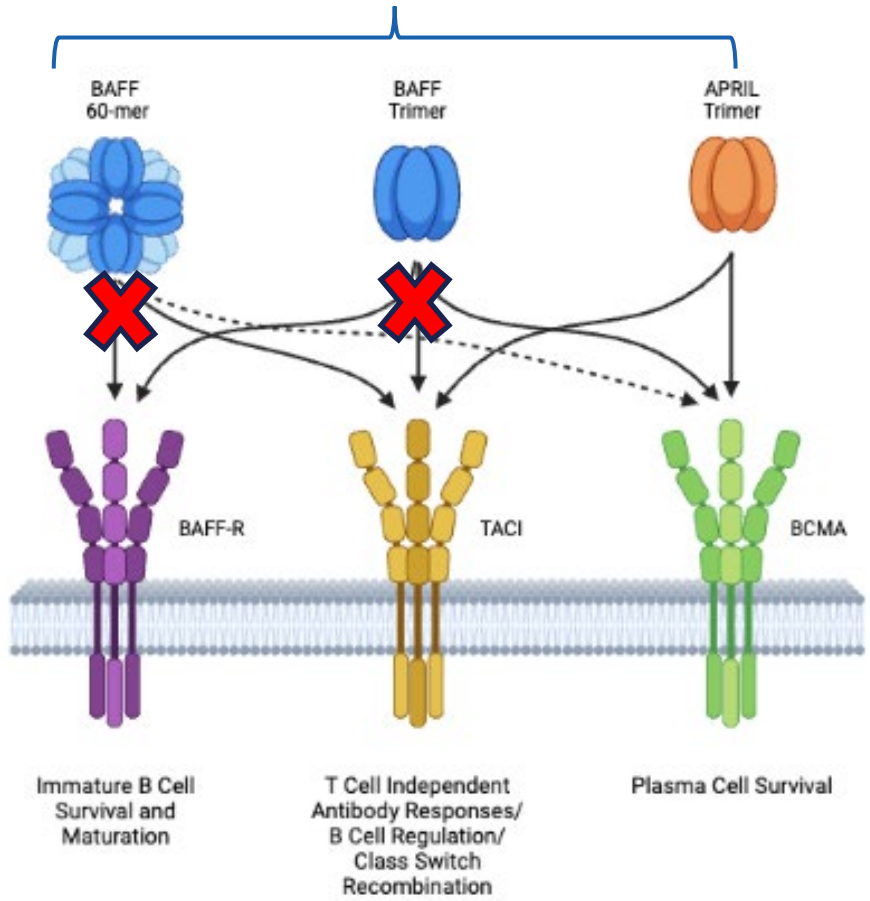
IL-17

Binds to IL-17A preventing IL-17A/A and IL-17A/F heterodimerization[1]



BAFF

Binds to BAFF trimer and BAFF 60-mer preventing binding to BAFF-R, TACI, and BCMA[2]



1. Liu et al. 2016. J Inflam Research; 2. Smulski and Eibel. 2018. FrontImmunol

Figure Generated with BioRender

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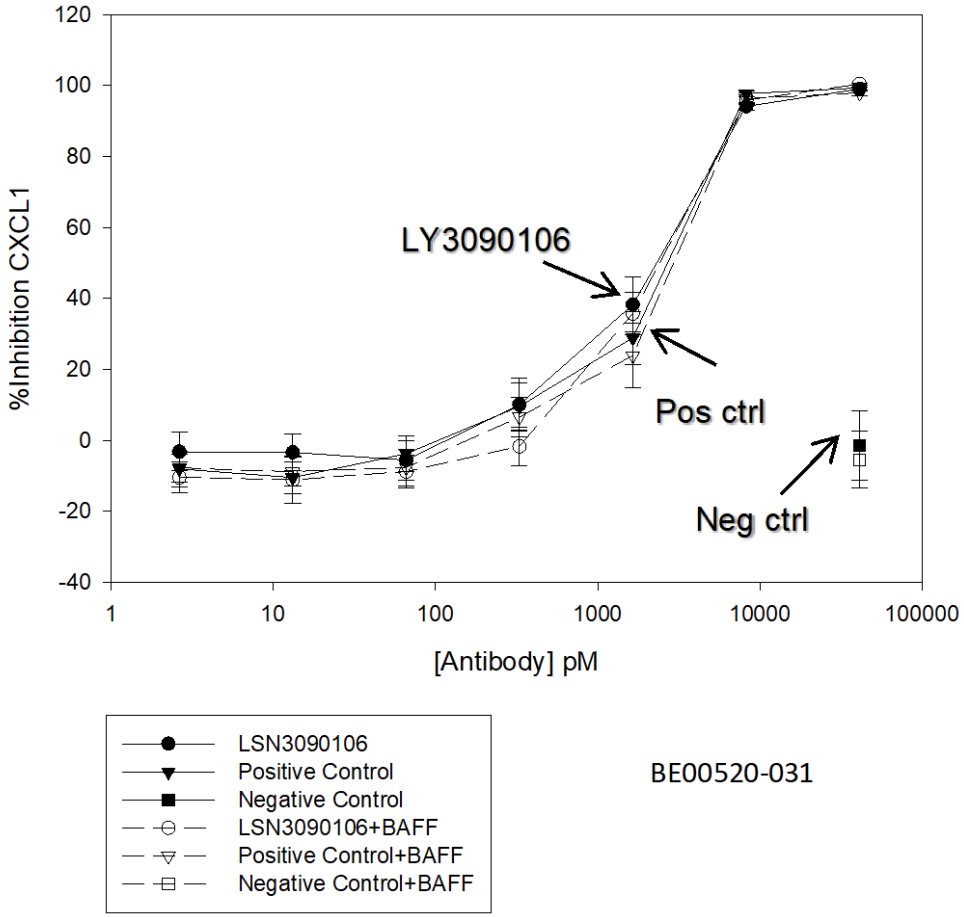
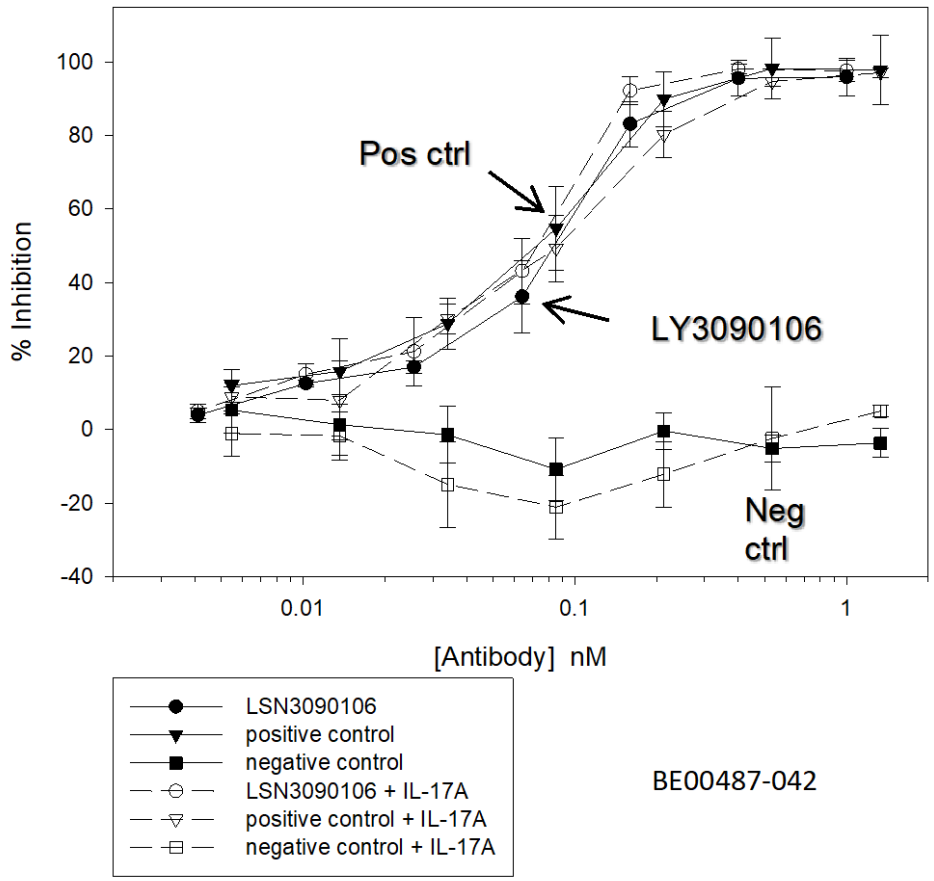
ZB-106 (LY3090106) Independently Neutralizes IL-17 or BAFF

ZB-106 inhibits IL-17 mediated CXCL1 production in HT-29 epithelial cells in a BAFF independent manner[1]



ZB-106 inhibits BAFF-mediated proliferation in T1165 cells in an IL-17 independent manner[1]

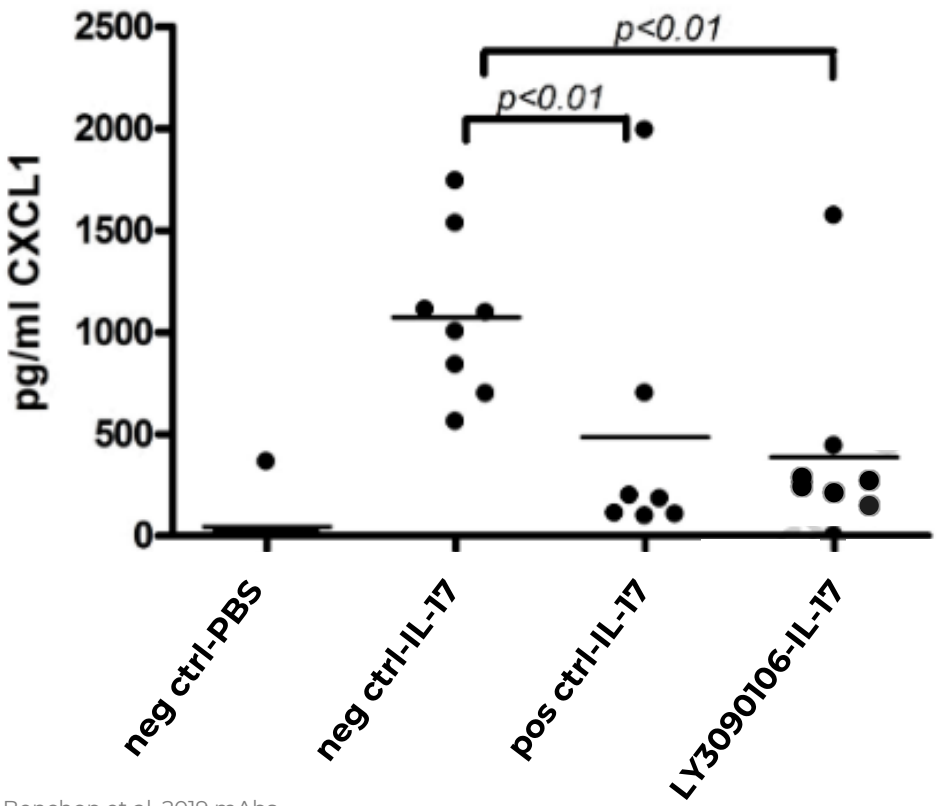


Benchop et al. 2019 mAbs.

ZB-106 (LY3090106) Inhibits IL-17 or BAFF-Mediated Inflammation

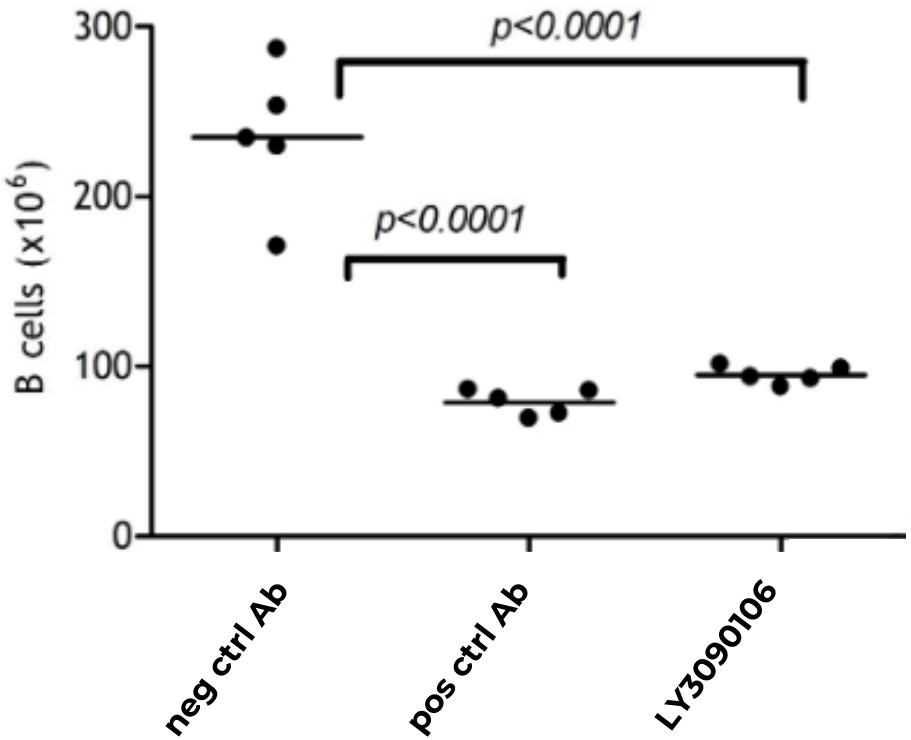
ZB-106 inhibits IL-17 mediated CXCL1 production in C57Bl/6 mice similar to ixekizumab (positive control)[1]

IL-17-induced CXCL1 Levels In-Vivo



ZB-106 reduces total B cell counts in the spleens of human BAFF transgenic mice similar to tabalumab (positive control)[1]

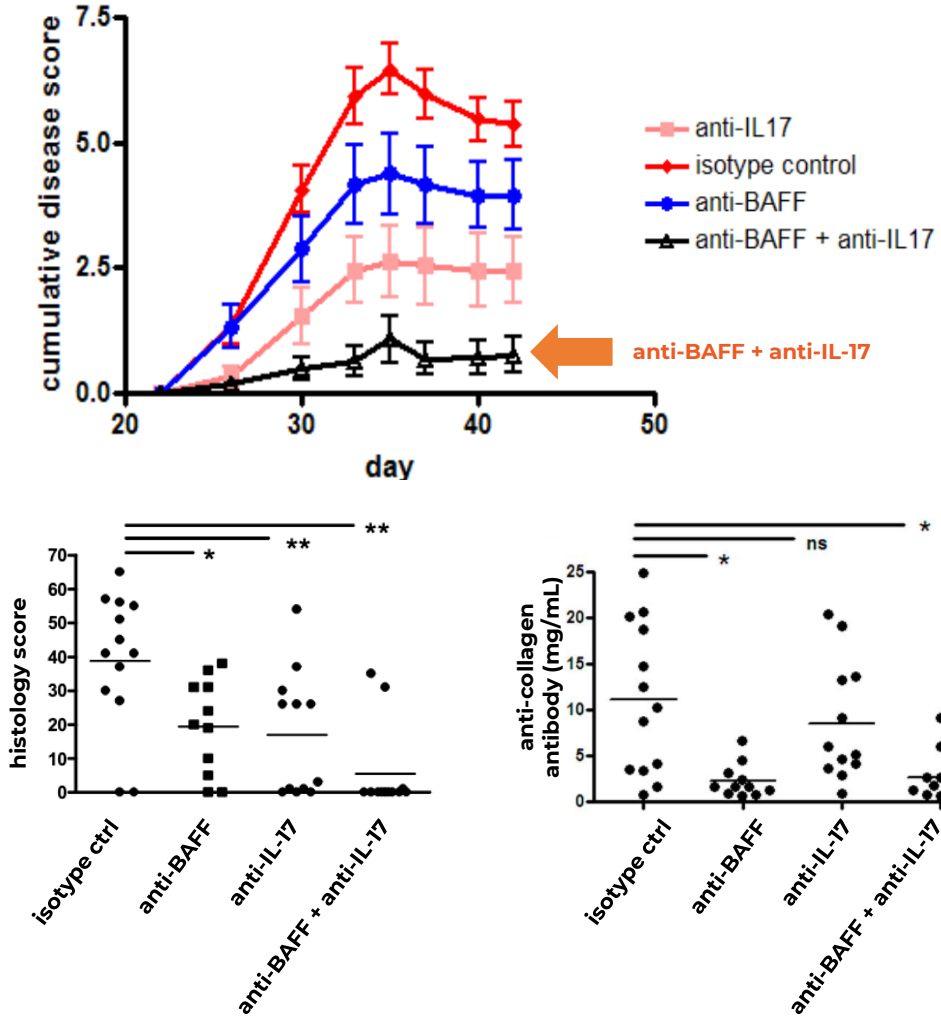
Total B Cells



Benchop et al. 2019 mAbs.

Combining IL-17 and BAFF Neutralization in a Murine Model of Arthritis Enables Improvement in Therapeutic Benefit

- Rheumatoid arthritis is a prototypic autoimmune disease where individually targeting IL-17-mediated inflammation or depleting B cells has been clinically validated
- The collagen-induced arthritis (CIA) murine model is similarly characterized by increased IL-17 production and B cells that drive disease pathogenesis
- Surrogate antibodies were used to evaluate whether neutralization of IL-17 and BAFF was superior to targeting individual pathways
- Mice were injected with anti-IL-17A and/or anti-BAFF on days 22, 29, and 36
- **Blockade of both IL-17A and BAFF was associated with reduced:**
  - **Disease severity**
  - **Inflammation in the hind paw (histology score)**
  - **Anti-collagen antibodies**



Source: Zura Internal Data, IND Briefing

ZB-106 is Clinically De-Risked Through P1b
78 Subjects/Patients Dosed Across 3 P1/1b studies

Pharmacokinetics	Pharmacodynamics	Safety and ADA
• $t_{1/2}$ is 26.9 days • Bioavailability after SC doses was 62.9% • At doses tested there is evidence of maximum target engagement with clinical safety supporting 6-fold "window" between max target engagement and max human dose tested	• In Ph1b healthy volunteer study in RA patients there was multiple impacts on PD markers • Decrease in CD20+ B-cells with higher doses generally associated with larger changes from baseline • Decrease in hs-CRP AUC was associated with higher ZB-106 AUCs	• SAD studies: No deaths or SAEs • MAD study: No deaths, single related SAE of neutropenia with resolution • Most frequent TEAE: Headache, transient neutropenia, nausea, diarrhea • No infections • In the multiple ascending dose study, one subject had TE-ADAs detected at a titer of 1:5120

Established dosing regimen

Demonstrated PD in patients in ph1b

Safety / ADA profile in line with Taltz

ZB-106 is a highly validated molecule that enables the opportunity to deliver on the promise of both IL-17 and BAFF inhibition in autoimmune disease

Source: Zura Internal Data, IND Briefing

ZB-106:
Systemic Sclerosis

Overview of Systemic Sclerosis

Disease Overview

- Systemic sclerosis (SSc) remains among the deadliest of the rheumatic diseases
- Patients with SSc often have severe disability, fibrosis-related organ failure, and die prematurely
  - Up to 50% develop interstitial lung disease (ILD), the most common cause of mortality in these patients
  - The disease has a severe impact on patients' lives, causing a variable constellation of symptoms including Raynaud's phenomenon, arthritis, painful ulcers on the fingers and toes, thickening and scarring of the skin, shortness of breath, hypertension, and severe fatigue

Unmet Need

- High unmet need remains as standard of care relies on immunosuppression therapy and biologic agents which are toxic and not well tolerated
  - Other current treatments only aim to manage symptoms and include pain relief through nonsteroidal, anti-inflammatory medications or corticosteroids
- **Two disease-modifying drugs are approved for severe lung complications of the disease (SSc-ILD)**, but no effective treatment exists that combats the disease across organ systems

Patient Population

- Varying prevalence estimates among world populations, with higher population estimates in the US than in Europe or Asia
- Data suggests ~250 per million adults in the US (80-100K patients), 233 per million in Australia (~6K patients), 88-158 per million in Western Europe
- Women are affected more frequently than men, with a female-to-male ratio of 5:1 and most commonly presents between the ages of 30-40 years

Source: Medscape, BMJ best practice

Overview of Systemic Sclerosis Potential Opportunity

Rare and life-threatening autoimmune disease characterized by tissue inflammation and fibrosis that has no disease modifying therapy

~200,000
people with SSc in US, EU and Japan[1]

40-60%
mortality in 10 years[2]

Zero
SSc-specific drugs approved

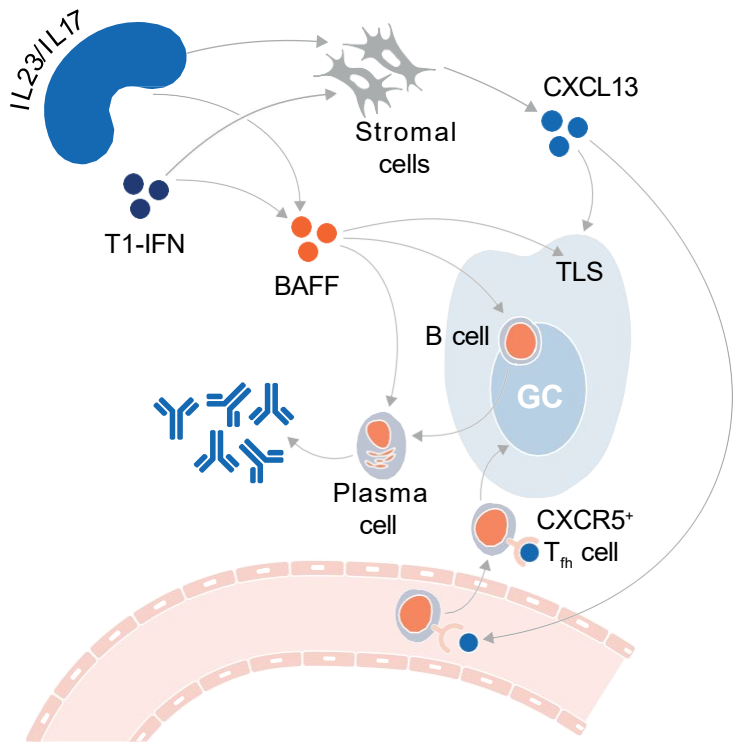
$2B+
annual potential market opportunity

1. Health Advanced, LLC; Lenabasum Commercial Market Assessment 2. Tyndall et al, 2010 3. Bergamasco, A. et al., Clin Epidemiol. 2019 Apr 18;11:257-273 4. Zura Bio internal analysis and benchmarking
5. Internal assumption based on demand research and rare disease analogues

IL-17 and BAFF Inhibition Pathways Have Shown Efficacy in Placebo Controlled Trials in Systemic Sclerosis (SSc)

IL-17 efficacy in SSc

- Brodalumab treatment in SSc leads to improved clinical outcomes[1]
- IL-17 known to play key role in the fibrotic process of various organs like lung, kidney, heart and skin
- Th17 cell–derived IL-17 was significantly higher in the skin and serum of SSc patients[2]



Role of BAFF in SSc

- Belimumab therapy shows efficacy in open label studies and one single center PBO study[3]
- Phase 2/3 initiated in SSc-ILD by GSK
- SSc patients have B cell abnormalities characterized by chronic hyper-reactivity of memory B cells[4]
- BAFF and auto-antibodies are key biomarkers in SSc[5,6]

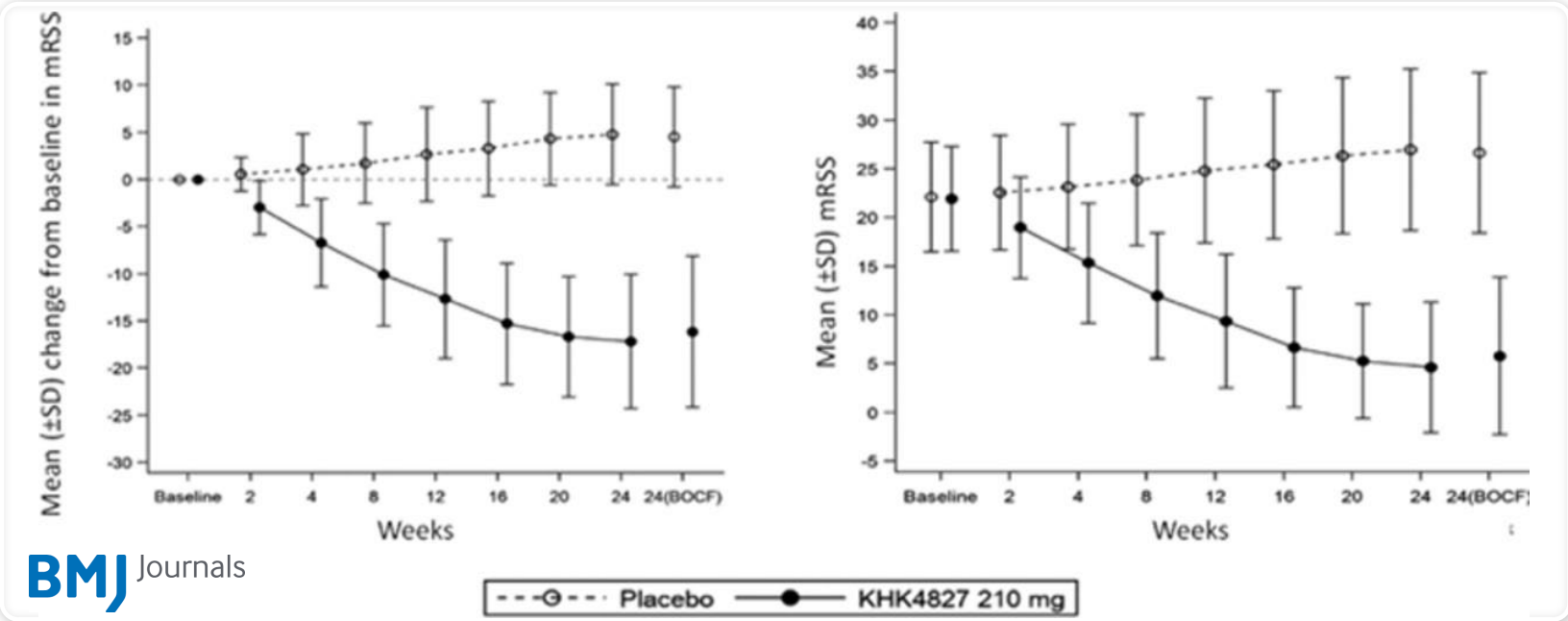
ZB-106 has the potential to treat the TH17 and BAFF components of SSc

1. Fukasawa et al. 2022. AnnalsRheumDisease; 2. Yang et al. Arthritis Research & Therapy 2014 3. Ebata et al. 2021. Lancet Rheumatol 4.Sato et al. 2004. MolImmunol.; 5. Senecal et al 2020. JSclerodermaRelatDisord; 6. Sato et al. 2000. JImmunol.

Brodalumab Treatment in SSc has Demonstrated Improved Clinical Outcomes

- Brodalumab achieved the primary endpoint (treatment difference of least square mean: −21.2 [95% CI -23.9, 18.5]; P<0.0001), and demonstrated a **rapid, sustained reduction in mRSS over 52 weeks**[1]
- The outcome of brodalumab treatment suggested its therapeutic effects on lung/respiratory functions, digital ulcers, the symptoms of gastroesophageal reflux disease, and QOL without any noteworthy safety concerns

mRSS over the 24-week follow-up (Mean +/- SD)



Source: Fukasawa et al. 2022. AnnalsRheumDisease

Belimumab Treatment in SSc has Demonstrated Improved Clinical Outcomes

Overview of Belimumab in SSc

- 52-week, investigator initiated, single center, double blind, placebo-controlled pilot study
- 20 subjects with dcSSC on MMF treated with belimumab or placebo
- No significant differences in AEs between belimumab group and placebo
- Patients in both treatment groups experienced clinical improvements in MRSS favoring belimumab (p=NS)
- All secondary endpoints favored belimumab with statistical significance in 2 endpoints: SHAQ DI and VAS Raynaud's phenomenon
- GSK recently received Orphan Drug Designation for the potential treatment of SSc with plans to initiate a phase 2/3 trial in SSc-ILD in 1H 2023

Table 2. Change in primary and secondary end points at 52 weeks*

	Belimumab + MMF (n = 9)	Placebo + MMF (n = 9)
MRSS, 0–51	–10 (–13, –9)	–3.0 (–15, –1)
SHAQ DI score, 0–3	–0.25 (–0.38, –0.25)†	0.00 (–0.13, 0.13)
VAS pain score, 0–150 mm	–10.5 (–40.5, 6.5)	–1.0 (–32.0, 0.0)
VAS RP score, 0–150 mm	–30.0 (–40.0, –14.0)‡	0.0 (–7.0, 22.0)
VAS ulcers score, 0–150 mm	–12.0 (–38.0, 1.0)	0.0 (–7.5, 4.0)
VAS breathing score, 0–150 mm	2.0 (0.0, 7.0)	0.0 (–7.0, 3.0)
VAS overall score, 0–150 mm	–14.0 (–29.0, –9.00)	–10.0 (–40.0, –6.0)
SF-36 MCS score, 0–100	7.50 (2.50, 18.50)	3.00 (0.00, 10.00)
SF-36 PCS score, 0–100	8.00 (–3.50, 19.00)	–3.00 (–3.00, 27.00)
PGA, 0–10	–4.43 (–8.05, –0.90)	–1.67 (–2.87, –0.90)
FVC, % predicted	5.00 (0.00, 8.00)	–2.00 (–6.00, 4.00)
DLco, % predicted§	2.00 (–7.00, 7.00)	0.00 (–6.00, 7.00)
CRISS score	0.61 (0.34, 0.88)	0.03 (<0.001, 0.80)

* Values are the median (interquartile range). MMF = mycophenolate mofetil; CRISS = composite response index in diffuse cutaneous systemic sclerosis (see Table 1 for other definitions).
† $P = 0.042$ versus placebo + MMF.
‡ $P = 0.029$ versus placebo + MMF.
§ Adjusted for hemoglobin level.

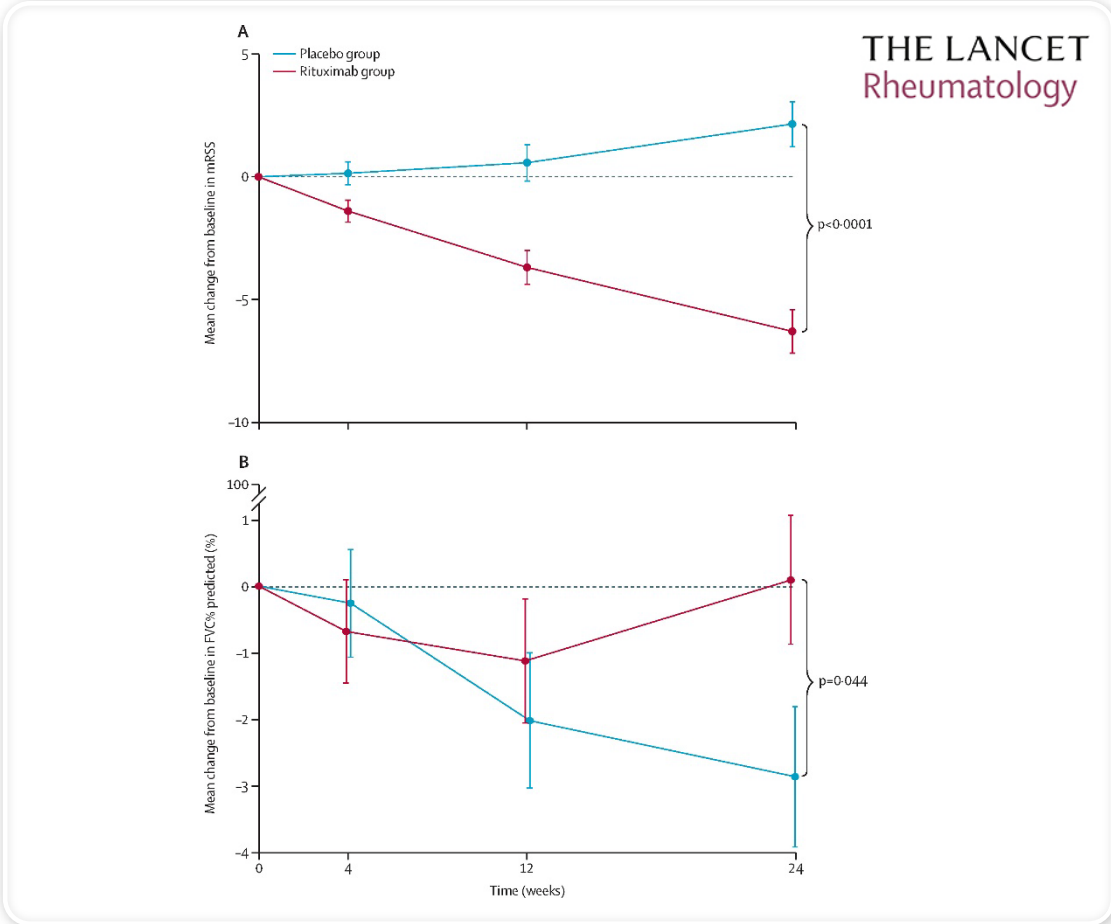
Source: Gordon et al..2018. ArthRheumatol.

B-Cell Depletion Therapy with Rituximab in SSc has Demonstrated Improved Clinical Outcomes

Rituximab in SSc shows efficacy

- Multiple Studies have shown the potential effectiveness of Rituximab in SSc – mainly open label and observational studies
- The most compelling data come from the DESIRES double blind – placebo controlled trial[1]
  - Fifty-six patients with SSc entered the study
  - The primary endpoint of mRSS change after 24 weeks of study treatment
  - Rituximab -6.30 points vs. PBO +2.14 points ($p < 0.0001$)
  - 48 / 56 subjects had SSc-ILD 2.96% FVC improvement at 24 weeks vs. PBO ($p=0.04$)

Randomized data shows improvements in SSc and SSc-ILD



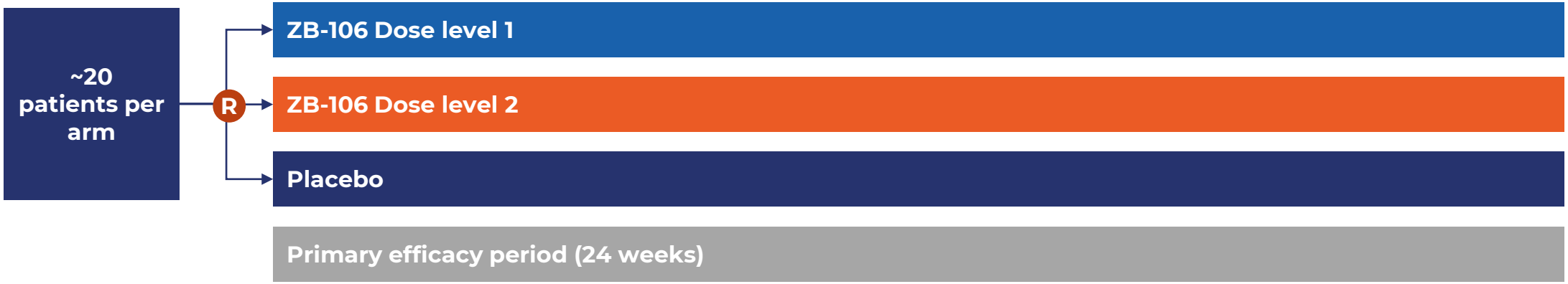
Source. Ebata et al. 2021. Lancet Rheumatol

Proposed Phase 2 Trial Design

Key inclusion criteria:

- mRSS 10-29
- Subject presented the first symptoms of sclerosis other than Raynaud's phenomenon within 60 months before enrollment

Double Blind, placebo-controlled trial



Key efficacy endpoints:

- Change mRSS
- CRISS
- SHAQ DI
- VAS (RP, Pain, Ulcers, Breathing)
- FVC, DLCO
- PK / PD assessments

Key safety endpoints:

- General Safety and Tolerability
- Severe infection
- Neutropenia
- ADA / nAb

Note: Clinical development plans are subject to ongoing review, regulatory feedback

ZB-106:
Hidradenitis Suppurativa

Overview of Hidradenitis Suppurativa

Disease Overview

- Hidradenitis Suppurativa is an inflammatory follicular skin disease
- Skin lesions develop in axillae, in the groin, and under the breasts, are formed as a result of inflammation & infection of sweat glands and are characterized by:
  - Recurrent boil-like nodules and abscesses that culminate in pus-like discharge
  - Difficult-to-heal open wounds (sinuses) and scarring.
  - Increased Th1/Th17 and B cell mediated inflammation[1-3]
  - Disproportionately affects women between adolescent age to 55 years of age[4,5]

Clinical Opportunity

- Estimated that there are **~300K** people living with Hidradenitis Suppurativa in the U.S. (1-2% global prevalence)
- Average of 7 years to diagnose globally
- High unmet need as there is only **One FDA-approved treatment for Hidradenitis Suppurativa (Humira)**
  - Current treatments only aim to manage symptoms and include palliative care such as over-the-counter eyedrops, topical cyclosporine and off-label treatments such as steroids or immunosuppressants to manage systemic symptoms

1. Moran et al. JID 2017; 2. Banerjee et al. 2017. Immunol. Invest. ; 3. Sabat et al. JACI 2022; 4. Garg et al. 2017 JAMA Dermatol; 5. Ingram. 2020. BrJDermatol

Overview of Hidradenitis Suppurativa Opportunity

Despite Multiple IL-17 Development Programs, There is Significant Opportunity to Address Unmet Need in HS

First in Class Therapy With Transformational Potential

- Known efficacy of IL17
- Strong rationale for BAFF
- Known dosing profile

Large Addressable Market

~300K Living U.S. Patients[1]

1-4% Global Prevalence[2]

$5-10B Potential market[3]

Significant Unmet Need

1 Drug Approved

Efficacy ceiling with IL17 alone

~10-20%

HiSCORE 50 Placebo-Adjusted[4]

BAFF has potential to improve clinical response vs IL17 alone - emerging early clinical data of B-cell targeted therapies

1. Epidemiology of Hidradenitis Suppurativa: Prevalence, Pathogenesis, and Factors Associated with the Development of HSInge E. Deckers &Hessel H. van der Zee &Errol P. Prens 2. Evaluate Pharma
3. Jefferies Wall Street Research 4. Cosentyx and Bimzelx Public Presentations, Publications and Research

Despite Multiple IL-17 Development Programs, There is Significant Opportunity to Address Unmet Need in HS

1 IL-17 A/F hypothesis still remains to be proven in the clinic

- IL-17 mediated inflammation is a key driver of pathophysiology in HS
- Multiple IL-17 compounds have shown efficacy, however, there were minimal differences between therapies targeting IL-17A alone versus those targeting IL-17A/F

2 Smaller therapeutics may not achieve higher efficacy or convenience

- Izokibep (IL-17A/A blocking peptide) reported improvement in a small open label study that enrolled HS patients primarily classified as Hurley Stage 2
- Data presented were similar to secukinumab Ph2 open label study in HS suggesting additional studies are needed to address the role of tissue penetration and smaller therapeutic approach

3 Despite clinical validation of IL-17, there remains a significant therapeutic gap for large number of patients

- HiSCR50 at 16 weeks tends to be ~ 15-30% (PBO adjusted), leaving substantial unmet need with opportunity for a differentiated therapy
- Addition of B-cell targeted therapies has the potential to improve overall clinical response compared with IL-17 alone

ZB-106 may address the efficacy gap raised for current IL-17 approaches in HS

Sources: Company Presentations, Publications and Research.

Public Data in Multiple P3 Studies Shows IL-17 is Clinically Validated Pathway to Treat HS

Recent HS Data

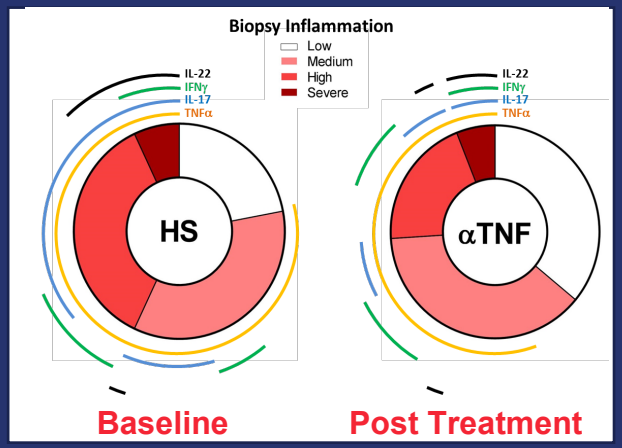
Company (Asset)		abbvie Humira	NOVARTIS Cosentyx	ucb Bimzelx	ACELYRIN Izokibep
Mechanism		TNF-α	IL-17A	IL-17A/F	IL-17A/A
Administration		SC	SC / IV	SC	SC
Phase		PIONEER I & II Phase 3	SUNSHINE & SUNRISE Phase 3	BE HEARD I & II Phase 3	Phase 2b Part A Open-Label
Dosing		40mg QW for 12W	300mg Q2W for 16W	320mg Q2W for 16W	160mg QW for 12W
Total Patients		n=633	n=360	Est. n=579	n=30
Efficacy (HiSCR50)	**Non-Placebo Adjusted**	42% - 59% at W12	42% - 45% at W16	48% - 52% at W16	71% at W12
	Placebo-Adjusted	16% - 31% at W12	11%+ at W16	19% - 20% at W16	NA
Safety / Tolerability	**Most Common AEs**	Headache 9% - 13% at W12	Headache 9% - 12% at W16	Hidradenitis 7% - 9% at W16	Injection site reactions
	Candidiasis	0% at W12[1]	0% - 3% at W12[1]	4% - 7% at W16	0% at W16[2]

Sources: Company Presentations, Publications and Research.
1. Represents data from psoriasis trial. 2. Represents safety data from psoriatic arthritis trial.

Limitations of Current Approaches in HS

Scratching the Surface

- Treatment with anti-TNFα therapy fails to downregulate inflammatory cytokines (IL-22, IL-17, IFNγ) in moderate-severe lesions[1]



- The presence of dermal tunnels increased the amount of time needed to achieve HiSCR50 with adalimumab[2]

Tunnelling into HS

- Recent literature highlights the role of dermal tunnels in the pathogenesis of HS[2,3]
- Dermal tunnels in HS are characterized by increased cellular infiltration including neutrophils; and Sabat et al. demonstrated increased BAFF production by neutrophils[3]
- Transcriptomic profiling highlights increased IL-17A and BAFF expression in dermal tunnels[3,4]
- Dermal tunnels were additionally shown to have increased numbers of B cells[3] and B cell targeting therapies are currently under investigation in HS[6]
- One significant drawback of the HiSCR is the lack of a dynamic measurement of draining tunnel; therefore, not fully capturing the anti-inflammatory properties of a therapeutic
- The International Hidradenitis Suppurativa Severity Score System (IHS4) was developed to assess inflammatory nodules (1 point), abscesses (2 points) and draining tunnels (4 points)

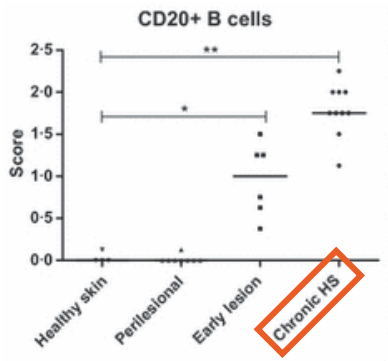
1. Moran et al. JID 2017; 2. Frew et al. 2021 Clin Exper Derm; 3. Sabat et al JACI 2023; 4. Gudjonsson et al. 2020; 5. Carmona-Rivera et al. JID 2022; 6. https://clinicaltrials.gov/ct2/show/NCT03827798

Addition of BAFF has Potential to Provide Superior Efficacy to IL-17 Alone

B-cells in HS lesions

- HS lesions have increased numbers of CD20+ B and CD138+ Plasma Cells[1]
- B-cell activating factor (BAFF) is produced by B cells, macrophages, dendritic cells, and neutrophils
- BAFF regulates B-cell survival, maturation and differentiation

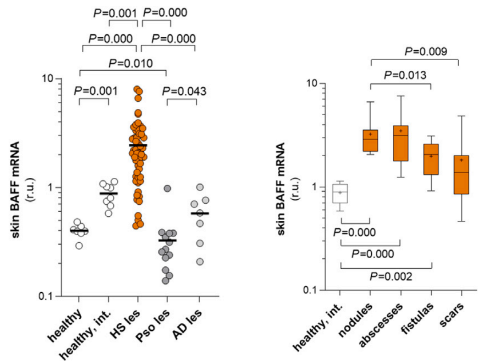
CD20+ B cells in HS Lesions



BAFF in HS

- Increased BAFF expression in HS lesions[2,3]
- Lesional upregulation of BAFF and BAFF receptors is attributed to B cells and plasma cells[2,4]
- Neutralization of BAFF with a soluble variant of BAFF-R in HS lesional explants reduced the expression of key genes associated with B and plasma cell function[2]

BAFF gene expression in HS



Clinical data in HS

- B cell depletion with rituximab provided therapeutic benefit[5]
- 4/5 cases report complete remission of HS lesions[5]

Rituximab in HS case report summary

	Complete Remission	No Remission		Complete Remission	No Remission
Number of Cases, n	4	1	**HS History (cont), n (%)**		
Female/Male (ratio)	1/3 (1:3)	1/0 (-)	**Hurley Stage[c]**		
Mean Age (stdev)	46.2 (20.5)	54 (-)	**I**	2/3 (66.7)	1/1 (100.0)
Risk Factors, n (%)[a]			**II**	1/3 (33.3)	0/1 (0.0)
Smoking History	1/2 (50.0)	1/1 (100.0)	**Comorbidities, n (%)**		
Overweight or Obese	2/2 (100.0)	1/1 (100.0)	**RA**	2/4 (50.0)	0/1 (0.0)
Family History of HS	0/2 (0.0)	1/1 (100.0)	**+ PsO**	0/4 (0.0)	1/1 (100.0)
HS History, n (%)			**PV + DDD**	1/4 (25.0)	0/1 (0.0)
HS Location[b]			**ICTO + CAAMR**	1/4 (25.0)	0/1 (0.0)
Inguinal + Back	1/3 (33.3)	0/0 (0.0)			
Inguinal + Abdomen	1/3 (33.3)	0/0 (0.0)			
Gluteal	1/3 (33.3)	0/0 (0.0)			

CAAMR, chronic active antibody-mediated rejection; *DDD*, Dowling-Degos disease ; *HS*, hidradenitis suppurativa; *ICTO*, idiopathic carpotarsal osteolysis; *PsO*, psoriasis; *PV*, pemphigus vulgaris; *RA*, rheumatoid arthritis
[a] Smoking, BMI, and family history of HS was not reported in 2/4 complete remission cases.
[b] HS location was not reported in 1/4 complete remission cases and 1/1 no remission cases.
[c] Hurley stage was not reported in 1/4 complete remission cases.

1. Van der Zee et al. 2012. Br J Derm; 2 Rumberger et al. 2020. J Inflam Research; 3.Sabat et al 2022. JACI; 4. Gudjonsson et al. 2020. JCI Insight.; 5. Seigel et al 2023. JCutanMedicSurgery

Anti-CD20+ and Anti-BAFF Treatment in HS
ZB-106 Therapeutic Potential Opportunity

Rituximab in HS

- Chimeric mAb to CD20, upon binding triggers cell death
- Used off label in a range of autoimmune diseases
- Case reports in HS (systematic review 2023)[1]
  - Majority with complete remission when treated with Rituximab did not respond to previous therapy including antibiotics and surgical excision, antibiotics alone, and isotretinoin with benzoyl peroxide.

Case report breakdown – 80% full remission[1]

Supplementary Table 1. Demographic and Clinical Features of Patients with HS Treated with Rituximab

	Complete Remission	No Remission		Complete Remission	No Remission
Number of Cases, n	4	1	HS History (cont), n (%)		
Female/Male (ratio)	1/3 (1:3)	1/0 (-)	Hurley Stage[c]		
Mean Age (stdev)	46.2 (20.5)	54 (-)	I	2/3 (66.7)	1/1 (100.0)
Risk Factors, n (%)[a]			II	1/3 (33.3)	0/1 (0.0)
Smoking History	1/2 (50.0)	1/1 (100.0)	Comorbidities, n (%)		
Overweight or Obese	2/2 (100.0)	1/1 (100.0)	RA	2/4 (50.0)	0/1 (0.0)
Family History of HS	0/2 (0.0)	1/1 (100.0)	+ PsO	0/4 (0.0)	1/1 (100.0)
HS History, n (%)			PV + DDD	1/4 (25.0)	0/1 (0.0)
HS Location[b]			ICTO + CAAMR	1/4 (25.0)	0/1 (0.0)
Inguinal + Back	1/3 (33.3)	0/0 (0.0)			
Inguinal + Abdomen	1/3 (33.3)	0/0 (0.0)			
Gluteal	1/3 (33.3)	0/0 (0.0)			

CAAMR, chronic active antibody-mediated rejection; *DDD*, Dowling-Degos disease ; *HS*, hidradenitis suppurativa; *ICTO*, idiopathic carpotarsal osteolysis; *PsO*, psoriasis; *PV*, pemphigus vulgaris; *RA*, rheumatoid arthritis
[a] Smoking, BMI, and family history of HS was not reported in 2/4 complete remission cases.
[b] HS location was not reported in 1/4 complete remission cases and 1/1 no remission cases.
[c] Hurley stage was not reported in 1/4 complete remission cases.

Implications for ZB-106

- **CD20+ B-Cells**
  - ZB-106 has direct effect reducing CD20+ B-cells
- **BAFF**
  - Increased BAFF expression in HS lesions[2,3]
  - Lesional upregulation of BAFF and BAFF receptors is attributed to B cells and plasma cells[2,4]
  - Dysregulated BAFF expression contributes to auto-immune diseases via effects on abnormal B-lymphocyte activation, proliferation, survival, and immunoglobulin secretion[5]
  - Murine models in RA provide evidence of synergistic activity of Anti-BAFF and Anti-IL-17
  - ZB-106 has been shown to have a clinical impact in diseases with elevated BAFF (e.g. SLE) with significant decreases in B-cells and serum immunoglobulins[6]
- **ZB-106 → Opportunity to improve clinical outcomes**
  - Impacting CD20+ B-cells directly
  - Inhibition of abnormal B-Cell activation and immunoglobulin secretion

Sources: 1. Seigel et al 2023. JCutanMedSurgery; 2. Rumberger et al. 2020. JInflamResearch; 3. Sabat et al 2022. JACI; 4. Gudjonsson et al. 2020. JCI Insight.; 5. Bosello et al 2007. IntJImmunopatholPharmacol ; 6. Merrill et al. 2016. Ann Rheum Dis

ZB-106 in Hidradenitis Suppurativa
Clinical Development Plan Rationale

Rationale to study HS: Opportunity for superior clinical response based upon IL-17 + BAFF inhibition

- IL-17 blockade in HS is a validated target with clear evidence of efficacy
- HiSCR50 at 16 weeks tends to be ~ 50% (placebo adjusted HiSCR50 ~15-30%), leaving substantial unmet need
- Translation data indicate an interplay between B cells and the IL-17 pathway in HS
- Case reports have shown that rituximab has an impact on HS clinical course

Dosing Rationale

- We have clear dosing windows for ZB-106
- ZB-106 clinical safety supports 6-fold “window” between max target engagement and max human dose tested

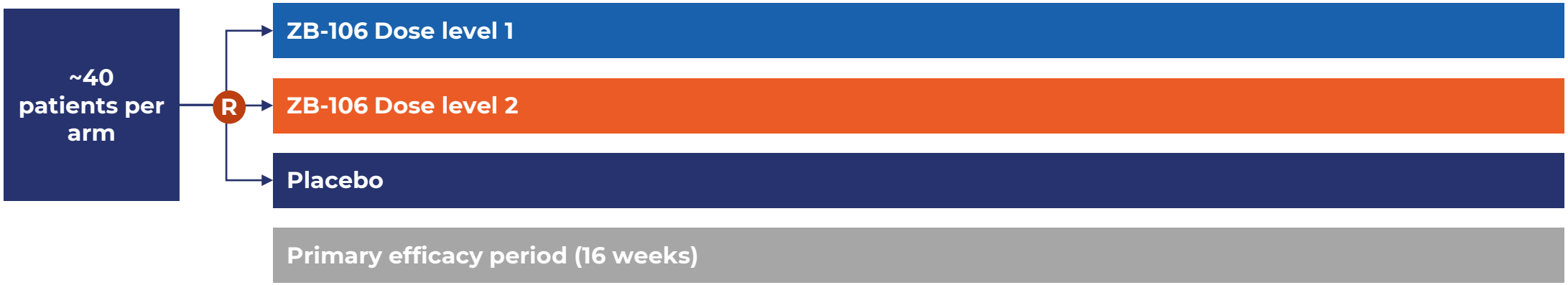
Source: Zura Internal Data

Proposed Phase 2 Trial Design

Key inclusion criteria:

- Moderate to Severe HS
- Hurley Stage II/III
- Total abscess and inflammatory count (AN) ≥ 3

Double Blind, placebo-controlled trial



Key efficacy endpoints:

- HiSCR (50, 75, 100)
- Improvement in baseline AN counts
- IHS4
- PGA
- DLQI
- PK / PD assessments

Key safety endpoints:

- General Safety and Tolerability
- Severe infection
- Neutropenia

Note: Clinical development plans are subject to ongoing review, regulatory feedback

ZB-106:
Optionality in additional indications

Multiple External Readouts Continue to Validate Both IL-17 and BAFF Pathways in Additional Diseases

	Unmet need	Evidence validating relevance of IL-17 and BAFF inhibition	External de-risking events
Sjögren's syndrome (SS)	• It is estimated that there are **250,000 – 350,000 people living with Sjögren's in the U.S.**[1] • Some estimates of the total global patient burden approach ~4M, with a smaller subset patients presenting most severely • High unmet need as there are **no FDA-approved treatments for Sjögren's**	Multiple clinical readouts have validated the BAFF Pathway in Sjögren's including • Novartis phase 2b data with BAFF-R Ianalumab (VAY736) • Remegen Telitacicept ph2 data IL-17 pathway continues to be explored pre-clinically for Sjögren's Syndrome	• Argenx efgartigimod phase 2 data in 2024 • Novartis BAFF-R Ianalumab (VAY736) phase 3 2027 • Remegen Telitacicept phase 3 readout 2027
Systemic Lupus Erythematosus (SLE)	• Systemic lupus erythematosus (SLE) is the most common form of lupus, affecting approximately 70 percent of an estimated 5 million people with lupus worldwide[2] • Approximately 170,000-200,000 Americans live with SLE. It is a chronic, incurable autoimmune disease producing autoantibodies that can attack almost any system in the body	tabalumab (BAFF) previous showed statistically significant efficacy in large 1,124 patient Ph3 study Benlysta (BAFF) is approved in SLE and Lupus Nephritis (LN) IL-17 pathway continues to be explored pre-clinically for SLE and LN	• Novartis BAFF-R Ianalumab (VAY736) • Remegen Telitacicept

Sources: Clinical Trials, Company Presentations and Wall Street Research
1. Maciel G, et al. Prevalence of Primary Sjögren's Syndrome in a US Population-Based Cohort. Arthritis care & research. 2017;69(10):1612-1616. 2. The Lupus Foundation of America

Conclusion
Investment Highlights

Experienced and proven leadership

- Experienced drug developers and asset hunters led by ex-Arena leaders
- Product approvals & commercialization
- Significant corporate transactions

Multiple Phase 2 programs

- Validated mechanisms pursuing innovative new development paths
- Potential Best in Class attributes combined with strategic clinical development to enhance differentiation

Track Record of Execution

- Deep relationships with KOL community
- Prior CRO leadership history within team enhances our Sponsor status
- Recent precedent of on-time execution in crowded I&I spaces (UC, AD, etc.) includes absorbing COVID impact without delays

Strong and committed financial backing with long-term alignment

- Investors aligned with long-term commitment
- Successful PIPE transformation, including $65M in funding through Business Combination

Abbreviations: AD, Atopic Dermatitis; CRO, Contract Research Organization; KOL, Key Opinion Leader; UC, Ulcerative colitis

ZB-168:
A Potential Best-in-Class Anti-IL7R Inhibiting Both IL7 and TSLP Pathways

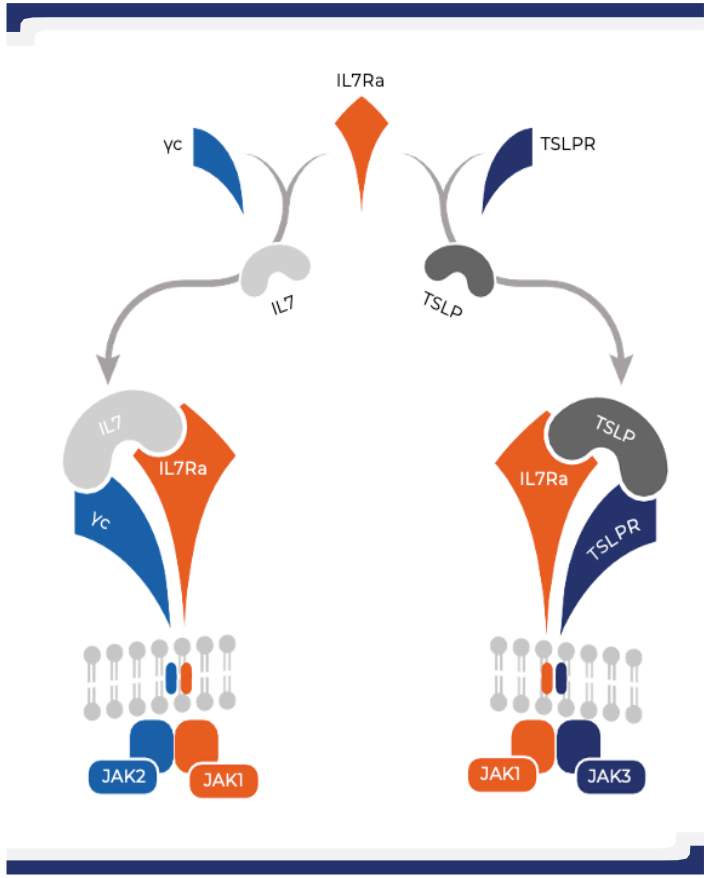
ZB-168 – Asset Overview

About ZB-168

- IL7Rα implicated in two key immune pathways[1]: IL7 and TSLP
- Only anti-IL7R program to date with human clinical data showing impact on key T-cell sub-populations[2]
- Well tolerated in >90 subjects and patients dosed in Phase 1 studies conducted by Pfizer[2, 3]
- Utility in multiple T-cell driven diseases[4]

Mechanism of Action

- Inhibition of IL7Rα blocks both IL7 and TSLP signaling[5]
- Blocking IL7Rα selectively inhibits survival/activity of effector memory and central memory CD4 and CD8 T cells while preserving the T_{regs} compartment[1, 5]
- T cell transcriptomics show downregulation of genes associated with activation, differentiation and trafficking of Th1, Th2 and Th17[6]
- Blocking TSLP reduces Asthma exacerbations in both Th2 high and low populations[7]



Indication Areas of Potential Interest

- Respiratory
- Dermatologic
- Gastrointestinal

Market Opportunity

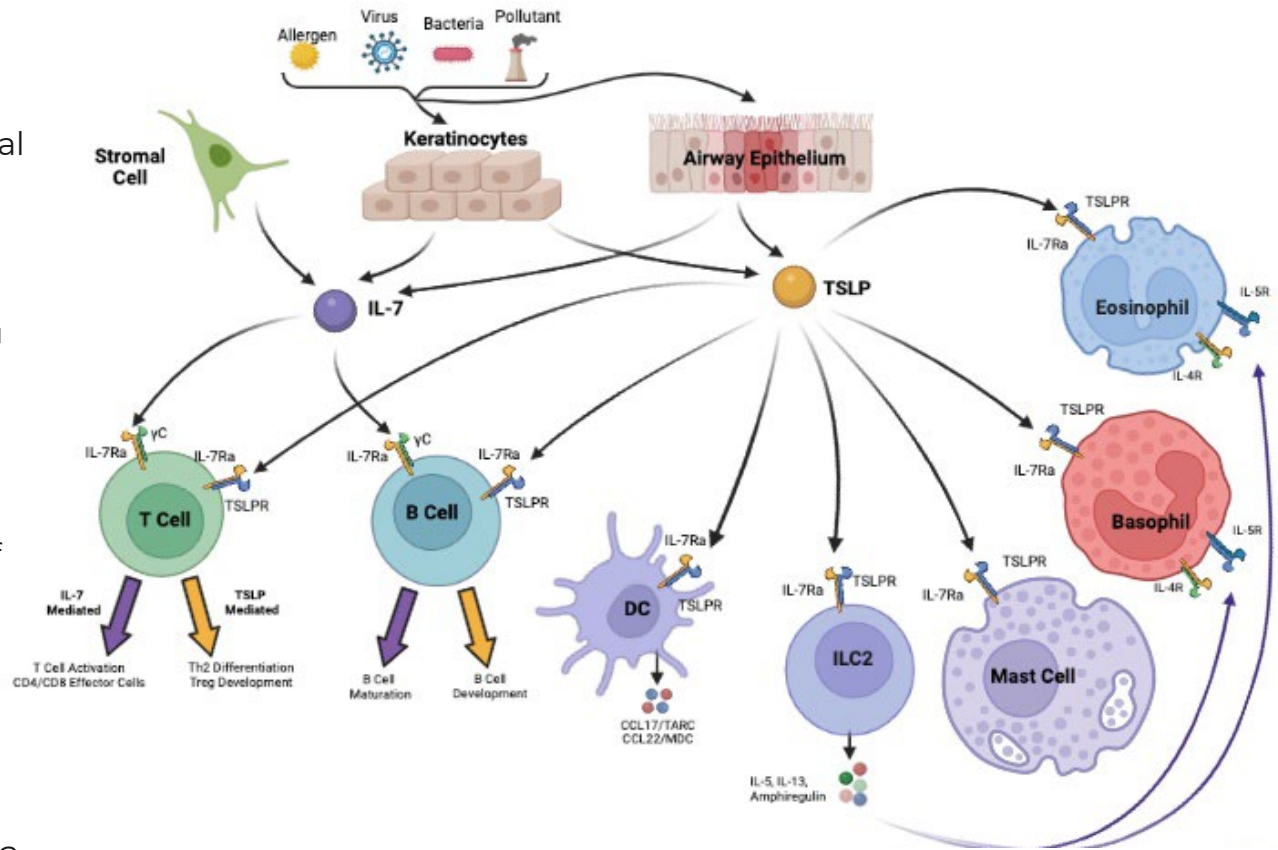
- Advances in the field further validate IL7 and TSLP in multiple type Th1 & Th2 immune disorders with significant global opportunities
- Potential 1st-in-class opportunities within several indications

1. doi: 10.1038/s41467-018-06804-y, 2. 10.1172/jci.insight.126054, 3. Clinical study report, 4. doi:10.3389/fimmu.2020.01557, 5. https://www.frontiersin.org/articles/10.3389/fimmu.2020.01557/full, 6. Herold, K. C. et a. *JCI Insight*. 2019; 4(23):e126054. 7. doi: 10.1056/NEJMoa2034975

ZB-168 Enables Broad Impact on Epithelial-Driven Inflammation by Targeting both TSLP and IL-7

TSLP and IL-7 Pathways

- Thymic stromal lymphopoietin (TSLP) is an epithelial-derived cytokine primarily expressed in the lungs, skin and gastrointestinal tract[1]
- TSLP is released from the epithelium by disease exacerbating environmental factors and is an important amplifier of Th2 immune response, including the production of IL-4, -5, -9 and -13.[1]
- TSLP inhibition is clinically validated in severe asthma and has shown positive therapeutic benefit in additional Th2 driven diseases[2,3]
- IL-7 is a growth factor that binds to the heterodimeric receptor of IL-7R:γC and is critical for the survival, development and homeostasis of central and effector memory T cells[4]
- Due to the high expression of IL-7R on T_{eff} compared to T_{reg}, inhibition results in a 20-fold greater activity in reducing T_{eff}, leading to an increase in Treg:Teff ratio[5, 6]
- Dual inhibition of TSLP and IL-7 has potential to modulate Th1, Th2, and Th17-driven inflammation in a targeted manner across a broad number of allergic and autoimmune diseases[7]



1. Ebina-Shibuy, 2022. Nat Rev Immunol, 2. Marone, 2019. Expert Opin Investig Drugs, 3. Menzies-Gow, 2020. Respir Res, 4. Chen, 2021. Frontiers Immunol, 5. Herold, 2019 JCI Insight, Graphic created in BioRender; 5. doi.org/10.3389/fimmu.2018.02692, 6. doi.org/10.1016/j.isci.2020.101421, 7. https://www.frontiersin.org/articles/10.3389/fimmu.2020.01557/full

zurabio

ZB-168 Has Broad Potential Therapeutic Applications

Inhibition of IL7R promotes a normalisation in T_{reg}:$T_{effector}$ T-cell ratios[4]

IL7

TSLP

Multiple Potential Indications in Key Therapeutic Areas

- Respiratory
- Dermatologic
- Gastrointestinal

TSLP is an early player in triggering airway inflammation via the activation of several immune cells such as dendritic cells, innate lymphoid cells, monocytes, macrophages and mast cells[5]

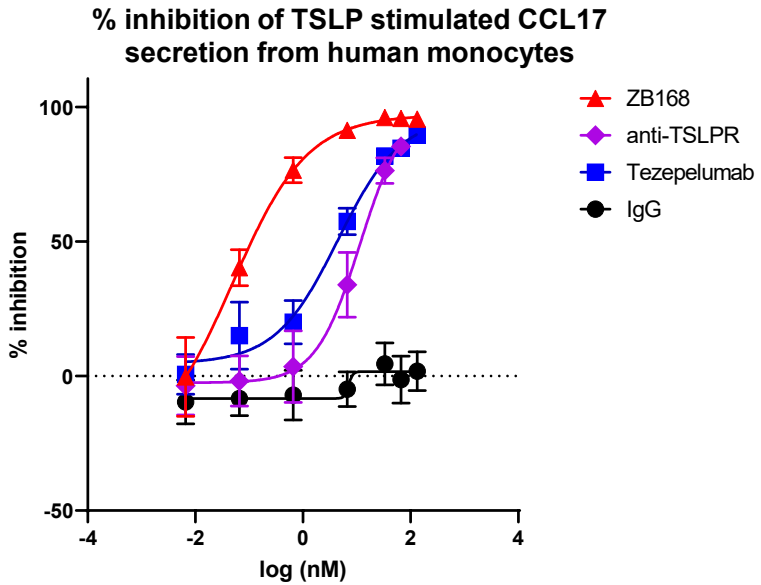
1. Eosinophilic esophagitis; 2. Chronic rhinosinusitis with nasal polyps; 3. Chronic spontaneous urticaria;
4. doi.org/10.3389/fimmu.2018.02692 & doi.org/10.1016/j.isci.2020.101421 ; 5. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6436879/

ZB-168 is Potential Best-in-Class and Only Non-Partnered IL7Rα Asset in Development

	ZB-168	ADX-914	OSE-127	GSK2618960	Tezepelumab	UPB-101
Type of Antibody	**Human**	Human	Humanised	Humanised	Human	Human
Target	**IL7Rα**	IL7Rα	IL7Rα	IL7Rα	TSLP	TSLPR[1]
Mode of Administration	**SC**[2]	SC	IV[3]	IV	SC	IV
Lead Indications	**Alopecia Areata**	Atopic Dermatitis	Ulcerative Colitis; pSS[4]	Programme inactive	Asthma, CRSwNP	Asthma
*Current Phase	**Phase 1b/2**	Phase 1b/2	Phase 2	Phase 1b	Approved	Phase 1
Humans Exposed	**HVs[5]: 60 subjects** **Patients: 33 subjects**	HVs: ~32 subjects Patients: asthma	HVs: ~63 subjects Patients: Ulcerative colitis	HVs: 18 subjects Patients: None	Patients: >1,000	HVs: 46 subjects Patients: 0

*As of September 2022; 1. Thymic stromal lymphopoietin receptor; 2. Subcutaneous; 3. Intravenous; 4. Primary Sjögren's syndrome 5. Healthy volunteers

ZB-168 is Potentially Best-in-Class Asset at TSLP Inhibition



- ZB-168 is nearly **10-fold** more potent than AZ/AMG's tezepelumab, and tezepelumab does not inhibit IL7 signaling
- ZB-168 is >**300-fold** more potent than Q32Bio / Horizon's ADX-914 in TSLP-induced markers, but similar in IL7-induced pSTA5[4]

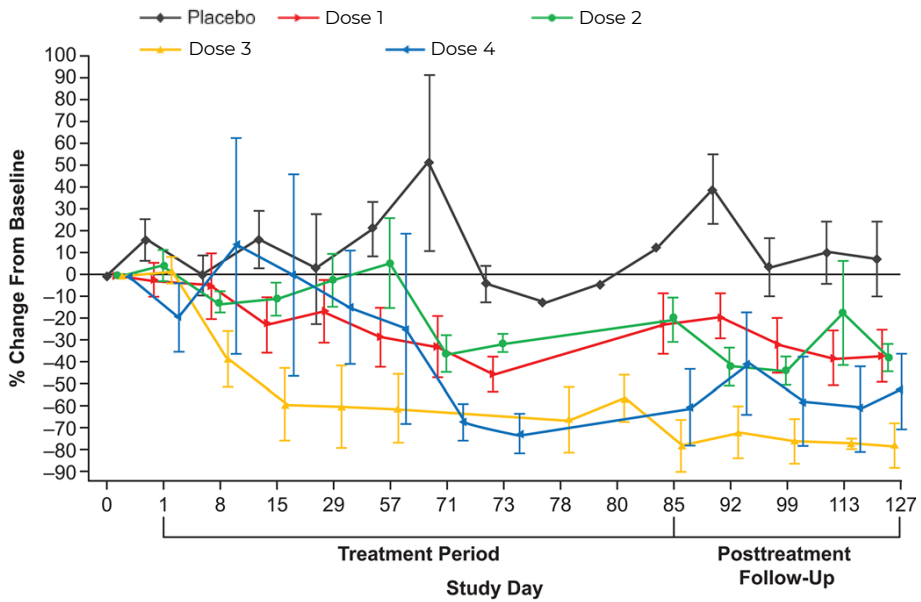
Asset	ZB-168 *(IL7Rα)*	UPB-101 (α-TSLPR)	Tezepelumab (TSLP)	ADX-914 (IL7Rα)
	IL7Rα mAb	α-TSLPR mAb	TSLP mAb	IL7Rα mAb
TSLP-Induced Signals	• 7.5 ng/ml / **0.05nM** (CCL17)[1] • 11 ng/ml / **0.07nM** (CCL22)[1] • **0.08 nM** (CCL2)[4]	• 16.1 ng/ml / **0.1nM** (CCL17)[3]	• 67 ng/ml / **0.44nM** (CCL17)[3]	• **24 nM** (CCL2)[4]
IL7-Induced Signals	• 0.46nM (pSTAT5)[2]	Neg	Neg	• 0.6 nM (IL7 at 0.25ng/ml)[4] • 2.1nM (IL7 at 2.5ng/ml)[4]

1. Zura Internal Data, 2. doi: 10.1172/jci.insight.126054, 3. doi: https://doi.org/10.1124/jpet.121.000686, 4. BMS patent https://patents.google.com/patent/WO2020154293A1/en

ZB-168 is Further Differentiated by $T_{effector}$ Cell Inhibition

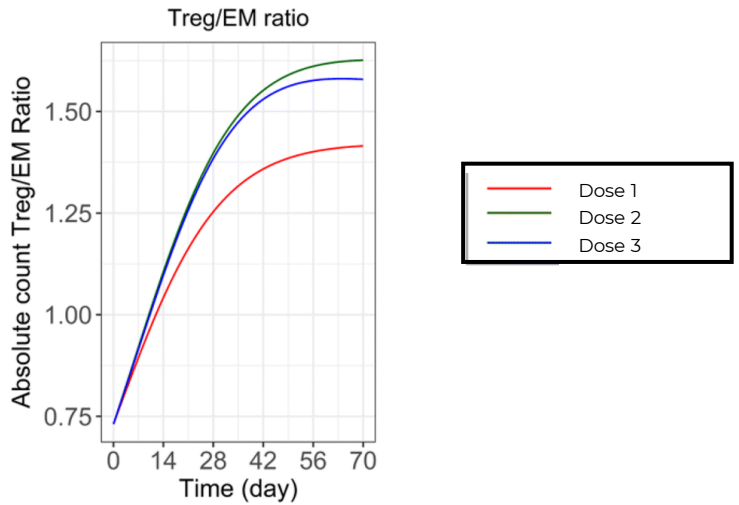
Clinical data in patients demonstrates impact on key T-cell subpopulations

CD8+ $T_{effector}$ cells[1]



- Up to 70% reduction in CD8+ $T_{effector}$ memory cells
- Similar reductions seen for naïve and central memory T-cells

Ratio of T_{reg} to $T_{effector}$ cells[2]



- Increases in ratios observed for all doses tested
- ZB-168 shows 20x greater potency for $T_{effector\ memory}$ vs T_{reg} cells

1. doi: 10.1172/jci.insight.126054, 2. doi:10.1208/s12248-019-0401-3

Summary of Clinical Data

- 93 subjects dosed with ZB-168 to date, including 33 patients with Type 1 Diabetes[1] and Multiple Sclerosis[2]
- In Phase 1 single ascending dose study, ZB-168 was generally well tolerated with no deaths and no subjects discontinued, or dose reduced due to Adverse Events (AEs)[3]
- Demonstrated significant clinically relevant biologic effects that may lead to a therapeutic benefit[1]
- Demonstrated proof of mechanism in a Phase 1b study of patients with recent onset Type 1 Diabetes (activity in inducing tolerance)[1]

1. doi: 10.1172/jci.insight.126054, 2. Internal study report, 3. Internal study report

torudokimab: Targeting Anti-IL33, an Alarmin with Potential in Multiple Indications

torudokimab – Asset Overview

About torudokimab

- IL-33 implicated in driving Th2 biology through ST2 and Th1/Th17 through alternative signaling[1]
  - Drug well tolerated in Phase 1 and 2 trials conducted by Eli Lilly[2]:
    - 141 healthy volunteers in Phase 1 study
    - 103 patients with moderate to severe atopic dermatitis
    - Utility in diseases driven by epithelial inflammation

Indication Areas of Potential Interest

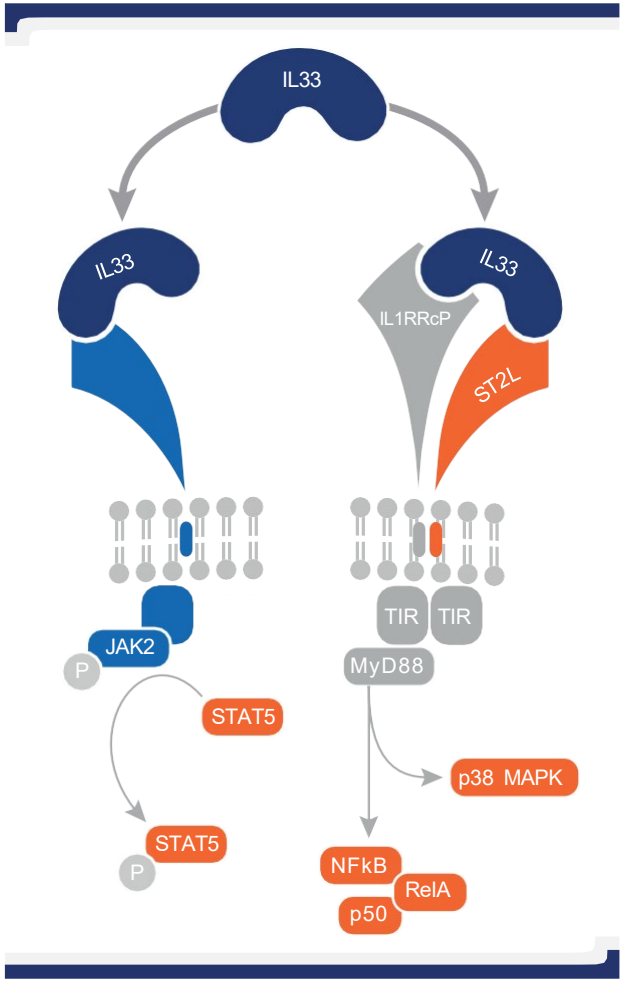
- Respiratory
- Dermatologic
- Gastrointestinal
- Orphan autoimmune

Mechanism of Action

- Inhibition of IL-33 blocks both ST2 and RAGE signaling[3]

Market Opportunity

- Advances in the field further validate IL-33 in multiple respiratory disorders with significant global blockbuster opportunities
- Potential 1st and best-in-class opportunities within multiple indications
- Validated pathways in COPD4 and asthma[5]

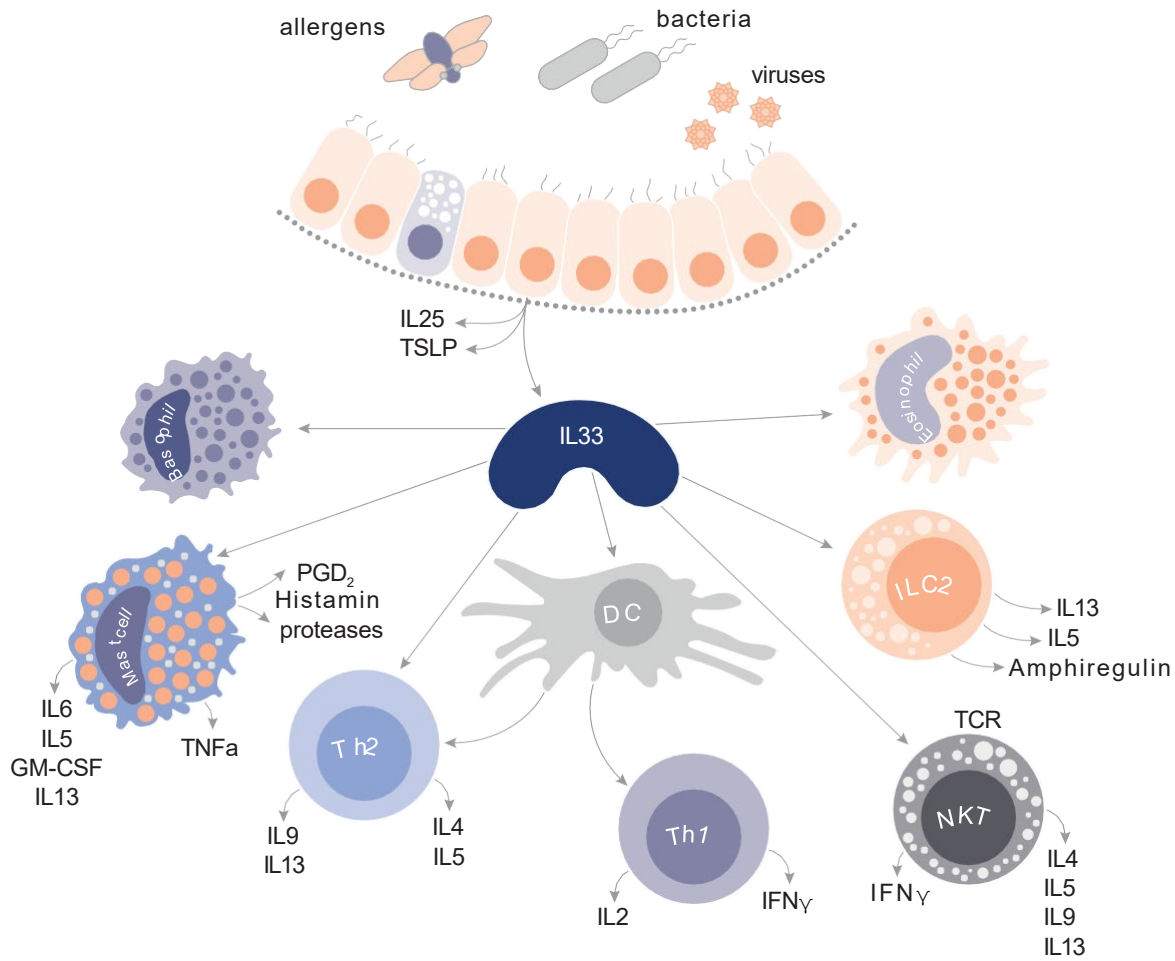


1. Suzanne Cohen et al. 2015 Nature, 2. https://clinicaltrials.gov/ct2/show/NCT03913260; https://clinicaltrials.gov/ct2/show/NCT03343587; https://clinicaltrials.gov/ct2/show/NCT03831191, Section 6.1, DSUR for period 23-Sep-2019 to 22-Sep-2020, 3. Okragly et al Journal of Inflammation Research 2021:14 3823–3835, 4 . https://doi.org/10.1016/S2213-2600(22)00005-4, 5. doi:10.1056/NEJMoa2024257

Targeting IL-33 In Epithelial Driven Diseases

IL-33 Pathway

- IL-33 is a member of the IL1 cytokine family that is constitutively expressed in structural and lining cells including fibroblasts, endothelial, and epithelial cells of skin, gastrointestinal tract, and lungs[1]
- IL-33 is released from the epithelium by disease exacerbating environmental factors and is an important amplifier of innate inflammation during exacerbations[2]
- Polymorphisms in IL33 and ST2 are associated with increased eosinophil production as well as respiratory diseases such as COPD and severe asthma.
- IL-33 inhibition clinically validated in severe asthma, COPD[3], and subsets of other epithelial disorders[4]
- Pre-clinical data demonstrates superior activity of torudokimab compared with etokimab suggesting the potential for best-in-class activity[5]
- Emerging biology suggests expanding opportunity for IL-33 in fibrotic and autoimmune conditions[6]

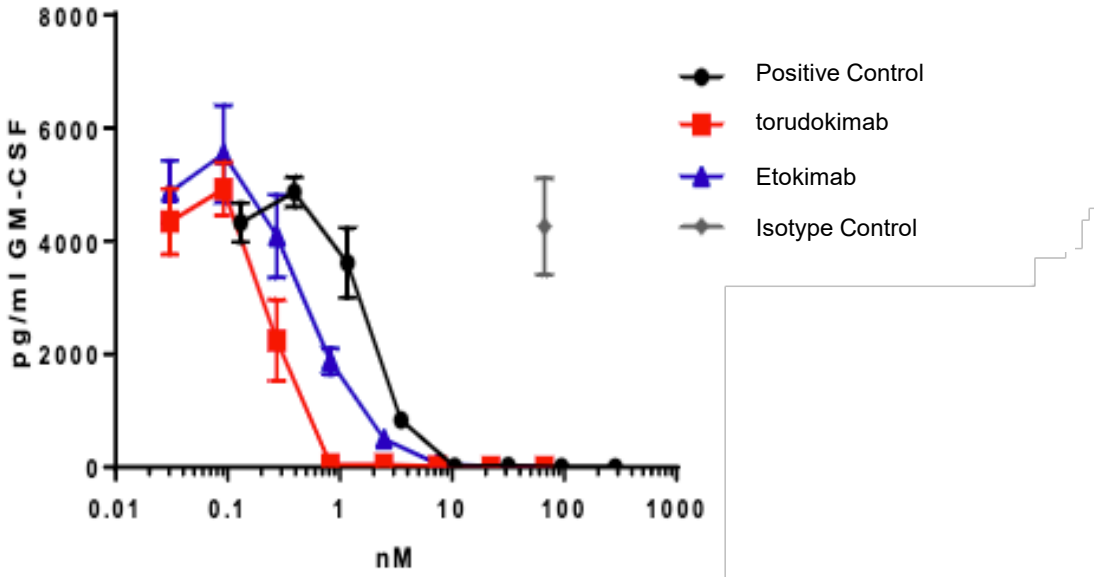


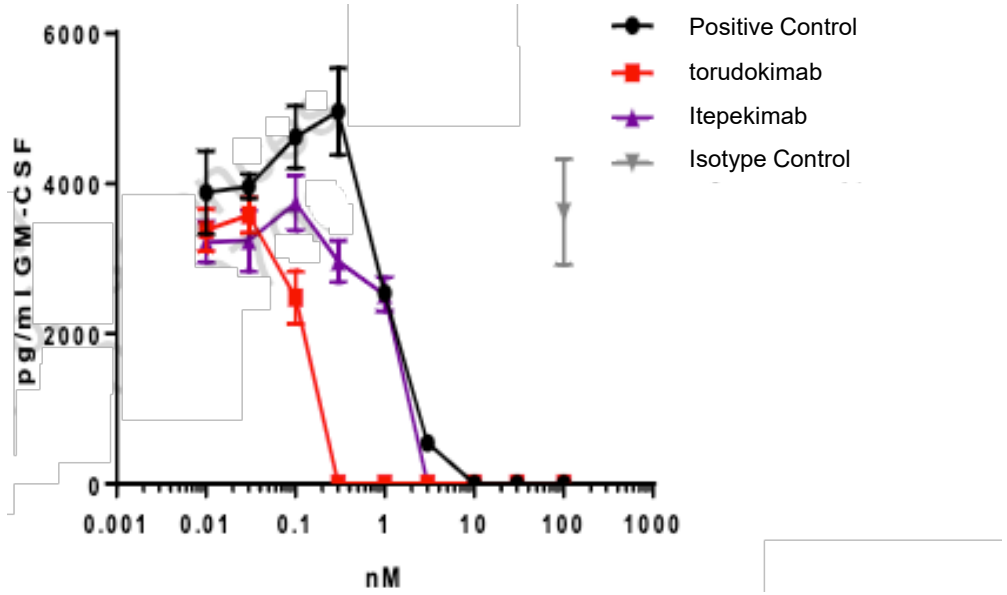
1. Chan, 2019. Frontiers Immunol, 2. doi.org/10.1016/j.cyto.2022.155891, 3. https://doi.org/10.1038/ng.323 and doi:10.1016/j.jaci.2020.04.051 , 4. .:https://doi.org/10.1016/S2213-2600(22)00005-4; doi:10.1056/NEJMoa2024257 and doi: 10.1126/scitranslmed.aax2945 , 5. Sci Trans Med., Zura Bio Internal data, 6. doi: 10.1111/imm.12174; https://doi.org/10.3389/fphys.2021.781012 and https://doi.org/10.3389/fmed.2021.739489

Note: please see language in the Disclaimer regarding 'forward-looking statements'

torudokimab Has Potential for “Best-in-Class” Activity

Torudokimab was 2.9 and 5.5 fold more potent than etokimab and itepekimab, respectively, inhibiting IL-33-induced GM-CSF production by human mast cells





Antibody	k_{on} ($M^{-1}s^{-1}$)	k_{off} (s^{-1})	k_d (pM)	Torudokimab Potency
torudokimab (LY3375880)	$1.7x10^6$	$6.7x10^{-5}$	39	
etokimab (AnaptysBio)	$9.4x10^5$	$1.2x10^{-4}$	112	2.9x
itepekimab (Regeneron)	$7.6x10^5$	$1.6x10^{-4}$	215	5.5x

Source: Zura Bio Internal data

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Note: please see language in the Disclaimer regarding ‘forward-looking statements’

Summary of Clinical Data

- >100 subjects dosed with torudokimab to date, including in a Phase 2 trial in atopic dermatitis[1]
- In Phase 1 study, torudokimab was well tolerated and no safety concerns were identified in either the SAD or MAD portions
- The target engagement evaluation suggested binding to IL-33 and treatment emergent ADA had no apparent impact on PK or target engagement
- In Phase 2 study in atopic dermatitis, torudokimab was well tolerated and no safety concerns identified; despite overall non-significant efficacy, responder analyses confirms key biomarker reductions (IL13, periostin and CCL17/TARC) and no ADA impact[1]

1. doi.org/10.1111/bjd.21631

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Corporate Overview with
ZB-106 Deep-Dive

Building the Next Immunology Leader

April 2023