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

FORM 8-K

CURRENT REPORT
PURSUANT TO SECTION 13 OR 15(D)
OF THE SECURITIES EXCHANGE ACT OF 1934

Date of Report (Date of earliest event reported): June 14, 2023

Zura Bio Limited

(Exact name of registrant as specified in its charter)

Cayman Islands	001-40598	98-1725736
(State or other jurisdiction of incorporation)	(Commission File Number)	(I.R.S. Employer Identification No.)

4225 Executive Square, Suite 600
La Jolla, CA 92037
(Address of principal executive offices, including zip code)

(858) 247-0520
(Registrant's telephone number, including area code)

(Former name or former address, if changed since last report)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions:

☐ Written communication pursuant to Rule 425 under the Securities Act (17 CFR 230.425)

☐ Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)

☐ Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))

☐ Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Securities registered pursuant to Section 12(b) of the Act:

Title of each class	Trading Symbol(s)	Name of each exchange on which registered
Class A Ordinary Shares, par value $0.0001 per share	**ZURA**	**The Nasdaq Stock Market**
Warrants, each whole warrant exercisable for one Class A Ordinary Share at an exercise price of $11.50 per share	**ZURAW**	**The Nasdaq Stock Market**

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (§230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (§240.12b-2 of this chapter).

Emerging growth company ☒

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act. ☐

Item 7.01 Regulation FD Disclosure.

On June 14, 2023, representatives of Zura Bio Limited, a Cayman Islands exempted company (the "Company"), began making presentations to banks and analysts using slides containing the information attached to this Current Report on Form 8-K as Exhibit 99.1 (the "Investor Presentation"), which is incorporated herein by reference. The Company expects to use the Investor Presentation, in whole or in part, and possibly with modifications, in connection with presentations to investors, analysts and others during the fiscal year ending December 31, 2023.

By filing this Current Report on Form 8-K and furnishing the information contained herein, the Company makes no admission as to the materiality of any information in this report that is required to be disclosed solely by reason of Regulation FD.

The information contained in the Investor Presentation is summary information that is intended to be considered in the context of the Company's Securities and Exchange Commission ("SEC") filings and other public announcements that the Company may make, by press release or otherwise, from time to time. The Company undertakes no duty or obligation to publicly update or revise the information contained in this report, although it may do so from time to time as its management believes is warranted. Any such updating may be made through the filing of other reports or documents with the SEC, through press releases or through other public disclosure.

The information presented in Item 7.01 of this Current Report on Form 8-K and Exhibit 99.1 shall not be deemed to be "filed" for purposes of Section 18 of the Securities Exchange Act of 1934, as amended (the "Exchange Act"), or otherwise subject to the liabilities of that section, unless the Company specifically states that the information is to be considered "filed" under the Exchange Act or specifically incorporates it by reference into a filing under the Securities Act of 1933, as amended, or the Exchange Act.

Item 9.01 Financial Statements and Exhibits.

(d) Exhibits.

Number	Description
99.1	Investor Presentation dated June 2023.
104	Cover Page Interactive Data File (embedded within the Inline XBRL document).

SIGNATURES

Pursuant to the requirements of the Securities Exchange Act of 1934, the Registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

Dated: June 14, 2023

Zura Bio Limited

By: /s/ Kim Davis
Kim Davis
Chief Legal Officer

zurabio

Corporate Overview

Building the Next Immunology Leader

Last Updated: June 2023

Disclaimer

Forward Looking Statements Disclaimer

This communication includes “forward-looking statements” within the meaning of the “safe harbor” provisions of the Private Securities Litigation
Words such as “expect,” “estimate,” “project,” “budget,” “forecast,” “anticipate,” “intend,” “plan,” “may,” “will,” “could,” “should,” “believe,” “predict
“continue,” “strategy,” “future,” “opportunity,” “would,” “seem,” “seek,” “outlook” and similar expressions are intended to identify such forward-loo
Forward-looking statements are predictions, projections and other statements about future events that are based on current expectations and a
result, are subject to risks and uncertainties that could cause the actual results to differ materially from the expected results. These statements
assumptions, whether or not identified in this communication. These forward-looking statements are provided for illustrative purposes only and
serve as, and must not be relied on by an investor as, a guarantee, an assurance, a prediction or a definitive statement of fact or probability.

Actual events and circumstances are difficult or impossible to predict and will differ from assumptions. You should carefully consider the risks ar
described in the “Risk Factors” sections of Zura Bio’s recent filings with the SEC. These filings would identify and address other important risks
could cause actual events and results to differ materially from those contained in the forward-looking statements. Many of these factors are outs
and are difficult to predict. Many factors could cause actual future events to differ from the forward-looking statements in this communication, in
to: (1) the outcome of any legal proceedings that may be instituted against Zura Bio; (2) volatility in the price of Zura Bio’s securities; (3) the abil
successfully conduct research and development activities, grow and manage growth profitably, maintain relationships with customers and supp
employees; (4) costs related to financing transactions and the ongoing costs relating to operating as a public company; (5) changes in the appli
regulations; (6) the possibility that Zura Bio may be adversely affected by other economic, business, and/or competitive factors; (7) the risk of d
changing regulatory landscape in the highly competitive industry in which Zura Bio operates; (8) the impact of the global COVID-19 pandemic; (
inability of Zura Bio to raise additional capital needed to pursue its business objectives or to achieve efficiencies regarding other costs; (10) the
Bio’s intellectual property, including its patents, and the potential infringement on the intellectual property rights of others, cyber security risks or
data security; and (11) other risks and uncertainties described in the Registration Statement and such other documents filed by Zura Bio from ti
the SEC. These risks and uncertainties may be amplified by the COVID-19 pandemic or other unanticipated global disruption events, which ma
economic uncertainty. Zura Bio cautions that the foregoing list of factors is not exclusive or exhaustive and not to place undue reliance upon an
statements, including projections, which speak only as of the date made. Zura Bio gives no assurance that it will achieve its expectations.

Zura Bio does not undertake or accept any obligation to publicly provide revisions or updates to any forward-looking statements, whether as a r
information, future developments or otherwise, or should circumstances change, except as otherwise required by securities and other applicabl

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

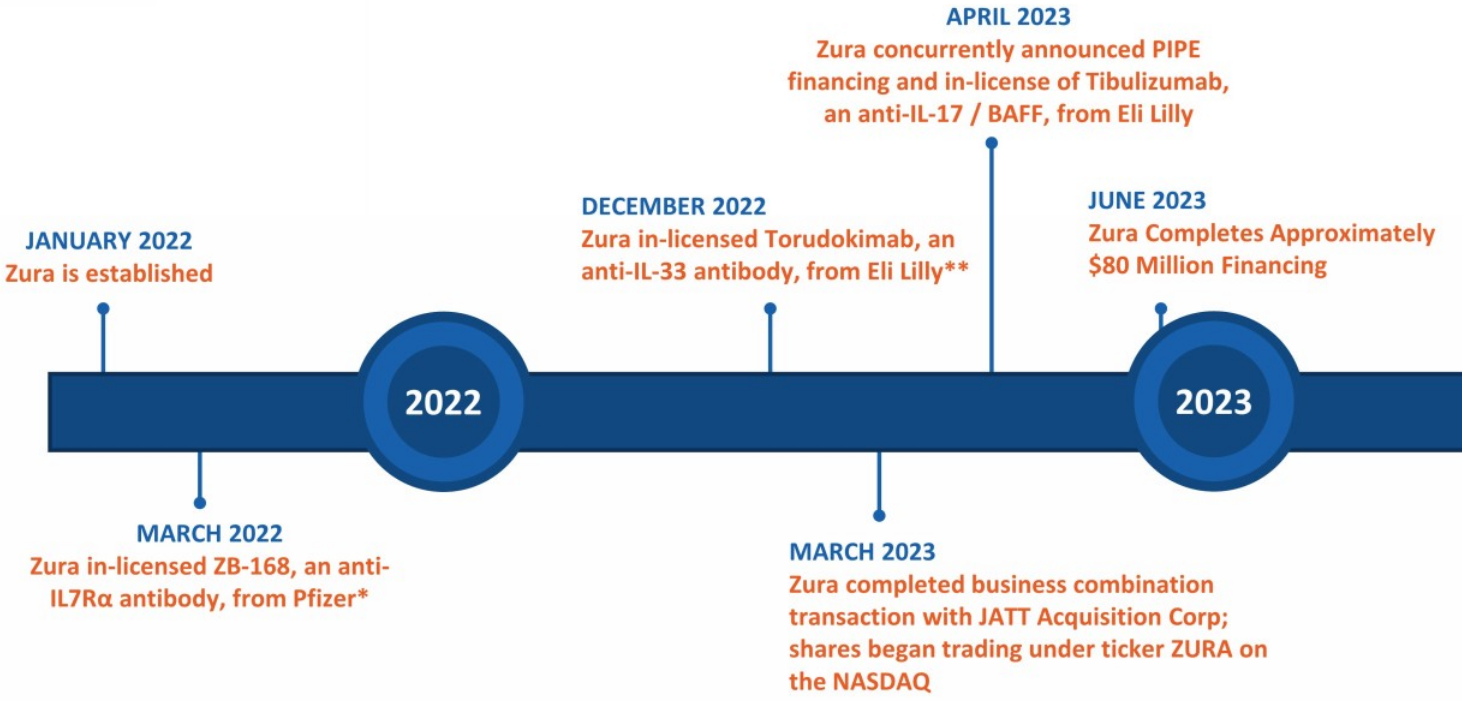
Strong and committed financial backing with long-term alignment

- Investors aligned with long-term commitment
- Successful PIPE transformation and private placement financing resulting in total cash and cash equivalents of approximately $120 million[1]

1. The Private Placement resulted in gross proceeds of approximately $80.0 million, before deducting placement agent fees and other offering expenses payable by the Company

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

Zura Company Timeline



(*) *Pfizer conducted Phase 1b studies in Type 1 Diabetes*
(**) *Eli Lilly conducted Phase 1 and Phase 2 studies of Torudokimab in Atopic Dermatitis*

An Experienced Leadership Team from A to Z

Amit Munshi
Chairman

ReNAgade THERAPEUTICS · ARENA PHARMACEUTICALS · EPIRUS · PERCIVIA · AMGEN

Someit Sidhu M.D.
Chief Executive Officer and Director

McKinsey & Company · IZANA BIOSCIENCE · JATT · PATHIOS THERAPEUTICS

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

ARENA PHARMACEUTICALS · QuintilesIMS · PULMATRIX · emergent

Kim D
Chief Lega

kaléo · AMG

Verender Badial
Chief Financial Officer

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

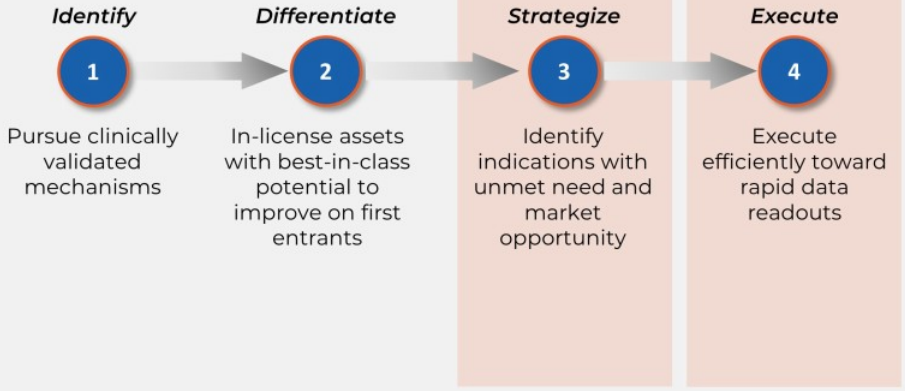
Mike Howell Ph.D.
Chief Scientific Officer and
Head of Translational Science

DermTech · MedImmune · Incyte · Boehringer Ingelheim

Gary Whale Ph.D.
Chief Technology Officer

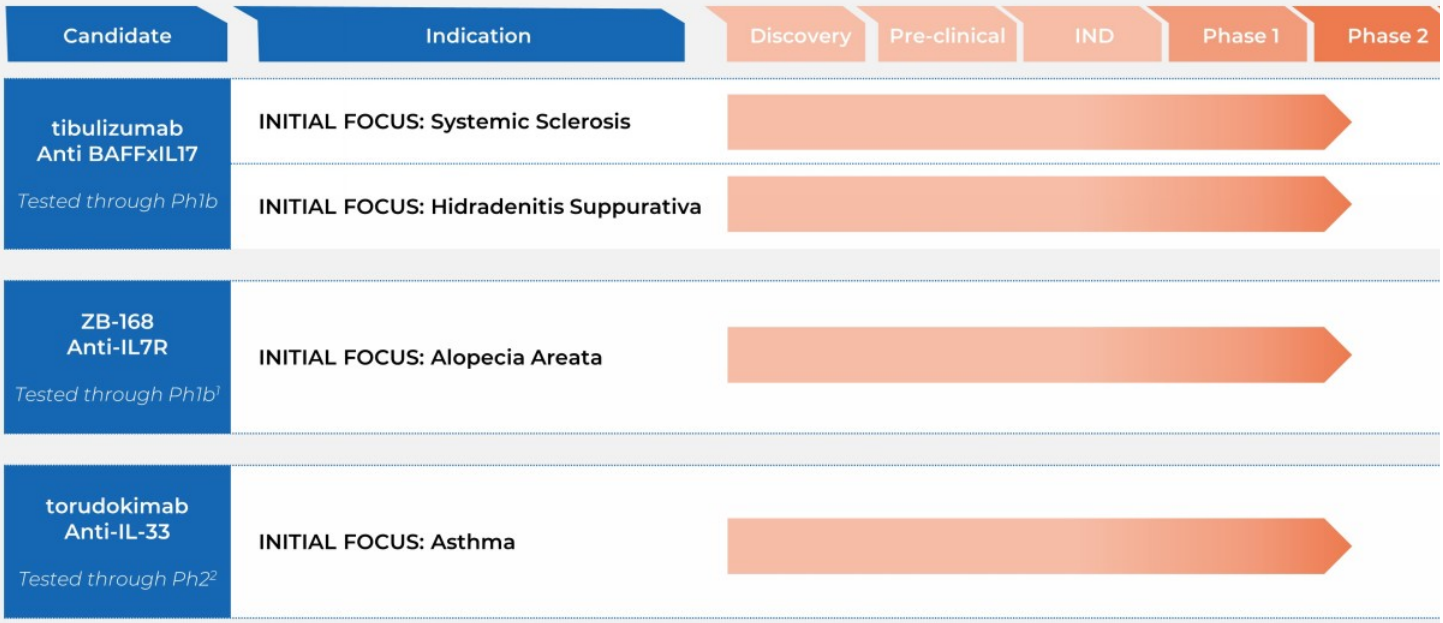
emergent · EUSAPharma

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



Clinical stage pipeline targeting key immunology pathways

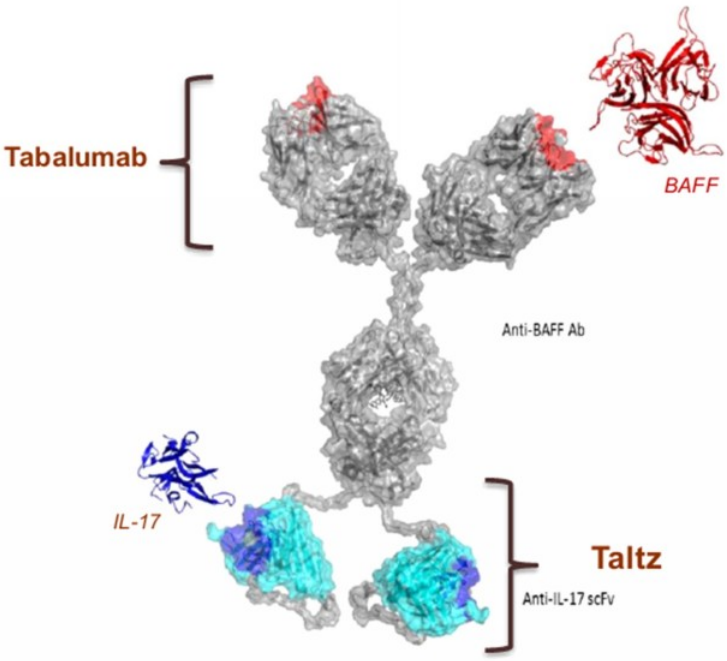
Current financing will bring in ZB-106 as lead asset for Zura



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 Antag Combining tabalumab and ixekizumab (TALTZ®)



ZB-106 is an IgG-scFv engineered by the fusion of ixekizur and tabalumab[1,2]

- ZB-106 neutralizes IL-17A or BAFF regardless of whether th are occupied
- ZB-106 binds in the same way as Taltz and tabalumab wit of binding sites
- Activity is mediated through direct target engagement ar
- Terminal half-life ~26 days

Robust existing clinical and non-clinical data package

- Two Phase 1b studies completed by Eli Lilly (RA and Sjögr
- 78 subjects have been dosed with ZB-106
  - 57 subjects = single dose; 21 subjects = multiple dose up
- Chronic toxicity studies completed with no adverse findin

Durable and deep IL-17 and BAFF signaling blockade obse cutaneous dosing every 4 weeks

- At target Q4W doses BAFF and IL-17 achieve maximum re

Low rate of immunogenicity

- Across 78 subjects exposed to ZB-106, only 1 subject teste drug Antibodies (ADAs)

Safety profile to date appears to be consistent with ixekiz 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) p arthritis, (e) ankylosing spondylitis, (f) non-radiographic axial spondylarthritis, (g) chronic spontaneous ur 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 Ato**
  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 202
- Enable launch of clinical trials in H2 2024 pending expected **phase 2 and 3 external catalysts in Asthm**
  COPD


ZE
Potential First-in-Class, Dual Ant
Combining tabalumab an
Note: please see language in the Disclaimer regarding 'forward-looking statements'

IL-17 and BAFF Approved in Multiple Autoimmune Diseases

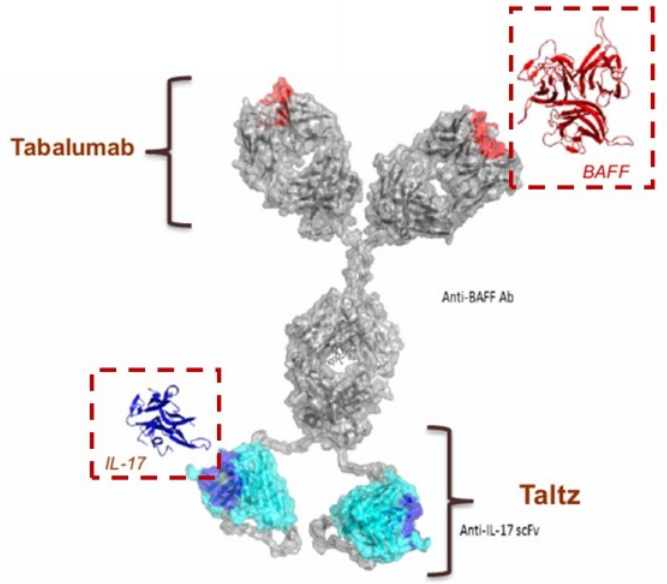
- **IL-17 and B-cell assets are widely recognized to have significant value**
- **ZB-106 represents an opportunity to pioneer a new approach to treating autoimmune diseases by directly addressi of immune inflammatory response**

		IL-17				IL-17 / BAFF	BAFF / TACI / API		
	Company	NOVARTIS	Lilly	ACELYRIN	MoonLake	zurabio	GSK	RemeGen	CHIN
	Asset	Cosentyx (secukinumab)	Taltz (ixekizumab)	Izokibep	Sonelokimab	ZB-106	Benlysta (belimumab)	Telitacicept	BION
	MoA	IL-17A	IL-17A	IL-17A/A	IL-17A/F	IL-17A / BAFF	BAFF	TACI fc	APF
	Delivery	SC / IV	SC	SC	SC	SC	SC / IV	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		
	Sjögren's							Ph3	
	IgAN							Ph2	Ph
	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 E
Demonstrated Efficacy with an Established Safety Prof

- 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 a studies
- Statistically significant a phase 3 study in SLE
- Demonstrated safety pr

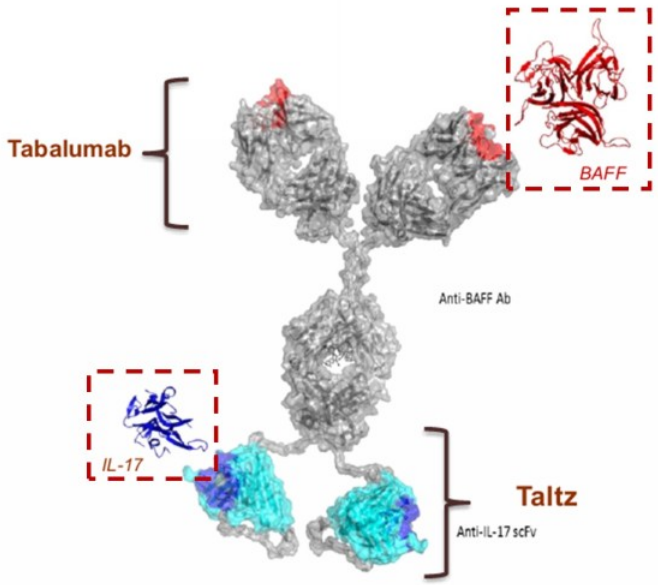
IXEKIZUMAB (TALTZ®)

- Commercially approved >$2.2bn of annual sales
- Demonstrated efficacy indications
- Demonstrated safety pr

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-1 implicated in the pathology[1,2]



BAFF

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

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

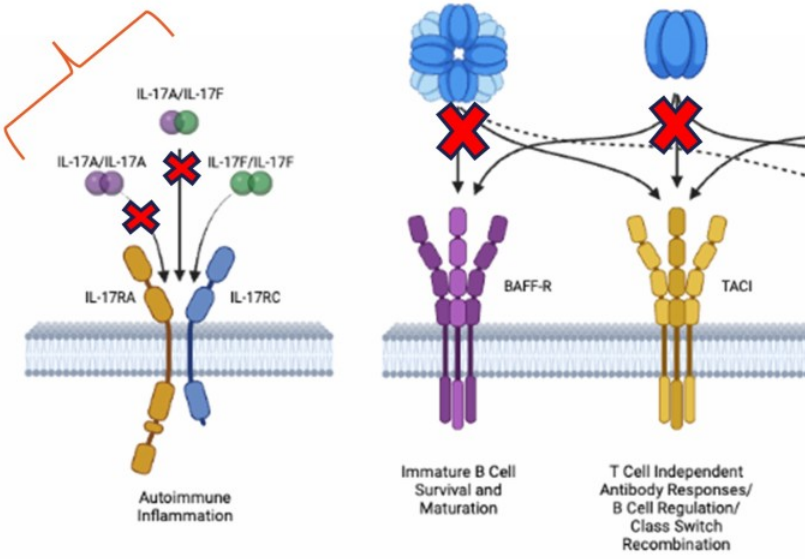
ZB-106 has the potential to treat diseases driven by B-cell and T-cell signaling

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 preve
BAFF-R, TACI, and BCMA[2]



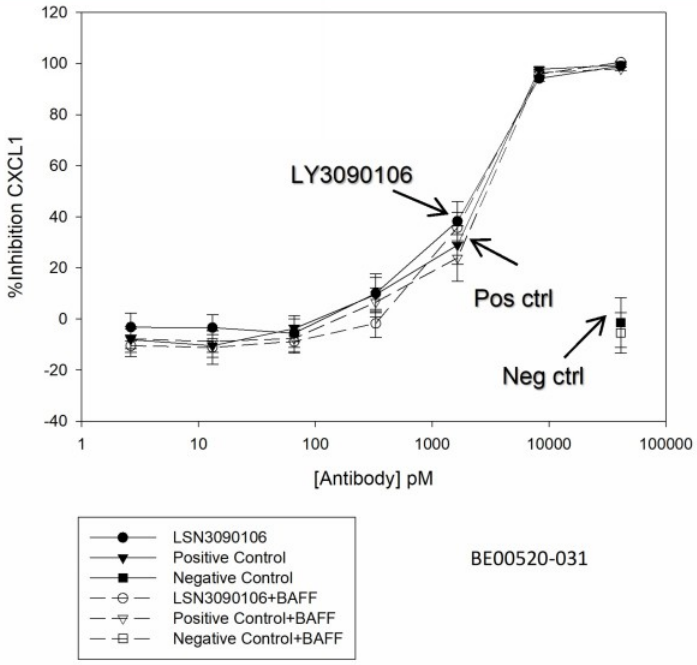
1. Liu et al. 2016. J Inflam Research; 2. Smulski and Eibel. 2018. FrontImmunol

Figure Ge

zurabio

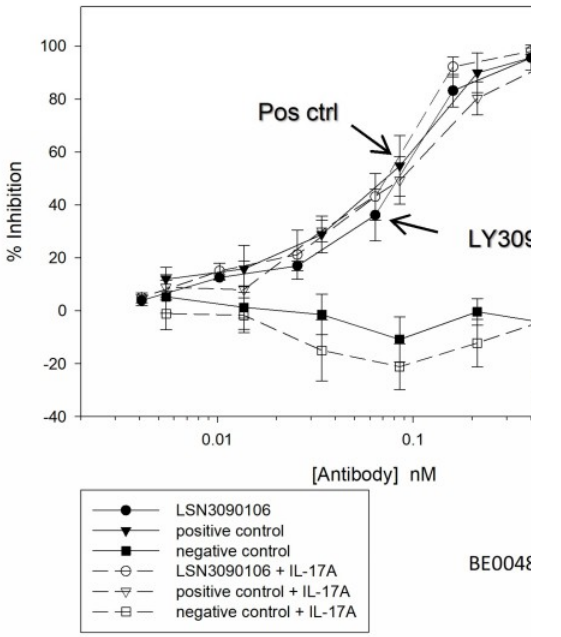
ZB-106 (LY3090106) Independently Neutralizes IL-17 or BAFF

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



BE00520-031

ZB-106 inhibits BAFF-mediated proliferation i an IL-17 independent manner[1]



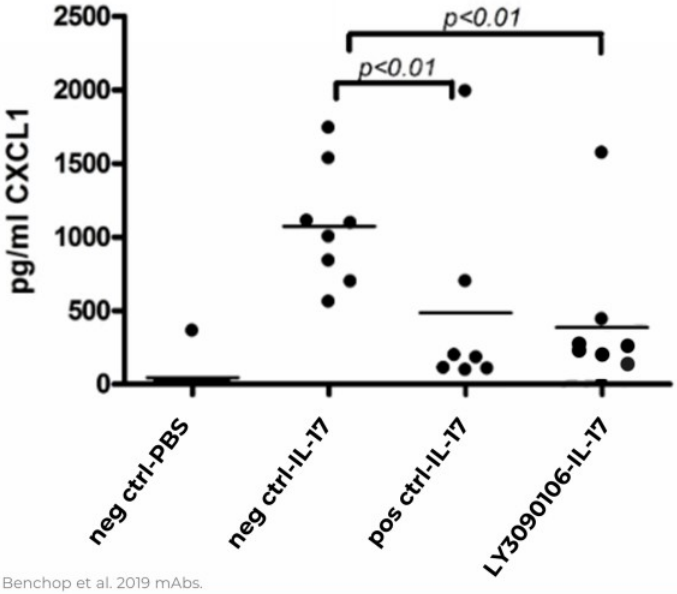
BE0048

Benchop et al. 2019 mAbs.

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

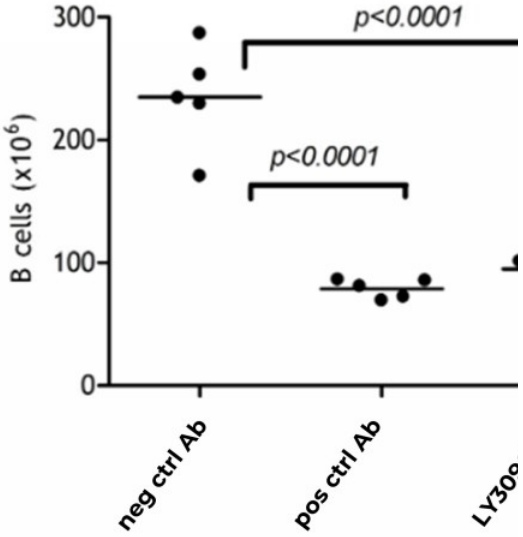
ZB-106 inhibits IL-17 mediated CXCL1 production in C57Bl/6 mice similar to ixekizumab (positive control)[1]

IL-17-induced CXCL1 Levels In-Vivo



ZB-106 reduces total B cell counts in the spleen transgenic mice similar to tabalumab (pos

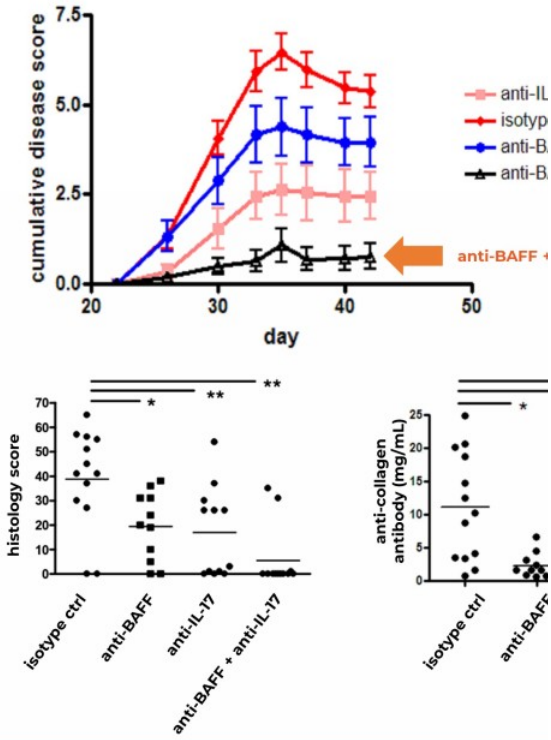
Total B Cells



Benchop et al. 2019 mAbs.

Combining IL-17 and BAFF Neutralization in a Murine Model 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 AD
• $t_{1/2}$ 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 • MAD study: No deaths, sir SAE of neutropenia with r • Most frequent TEAE: Heac transient neutropenia, nau diarrhea • No infections • In the multiple ascending study, one subject had TE detected at a titer of 1:5120

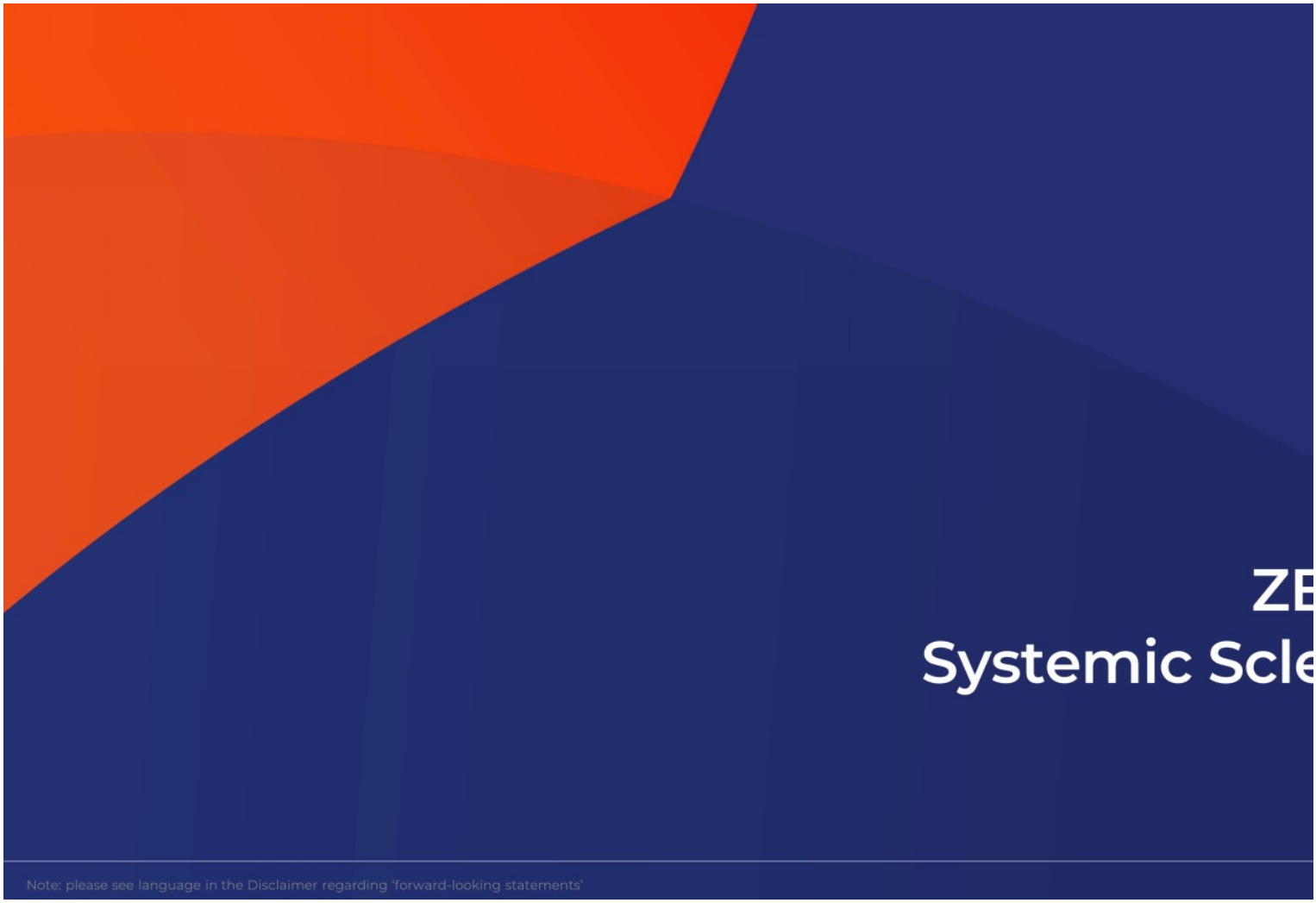
Established dosing regimen

Demonstrated PD in patients in ph1b

Safety / ADA pr line with Ta

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

Source: Zura Internal Data, IND Briefing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Systemic Scle
Note: please see language in the Disclaimer regarding 'forward-looking statements'

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 p arthritis, painful ulcers on the fingers and toes, thickening and scarring of the skin, shortness of breath, hypertension, fatigue

Unmet Need

- High unmet need remains as standard of care relies on immunosuppression therapy and biologic agents which are toxi tolerated
  - Other current treatments only aim to manage symptoms and include pain relief through nonsteroidal, anti-inflamm medications or corticosteroids
- **Two disease-modifying drugs are approved for severe lung complications of the disease (SSc-ILD)**, but no effective t 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 A
- Data suggests ~250 per million adults in the US (80-100K patients), 233 per million in Australia (~6K patients), 88-158 per Western Europe
- Women are affected more frequently than men, with a female-to-male ratio of 5:1 and most commonly presents betwee 30-40 years

Source: Medscape, BMJ best practice

Overview of Systemic Sclerosis Potential Opportunity

Rare and life-threatening autoimmune disease characterized by tissue inflammation a
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

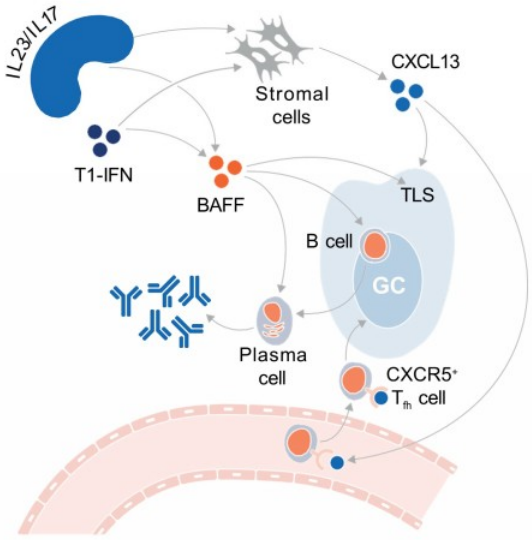
$2
annual market o

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 b
5. Internal assumption based on demand research and rare disease analogues

IL-17 and BAFF Inhibition Pathways Have Shown Efficacy in Place 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 ir

- Belimumab tl efficacy in ope and one singl study[3]
- Phase 2/3 initi by GSK
- SSc patients h abnormalities by chronic hy of memory B
- BAFF and aut are key bioma

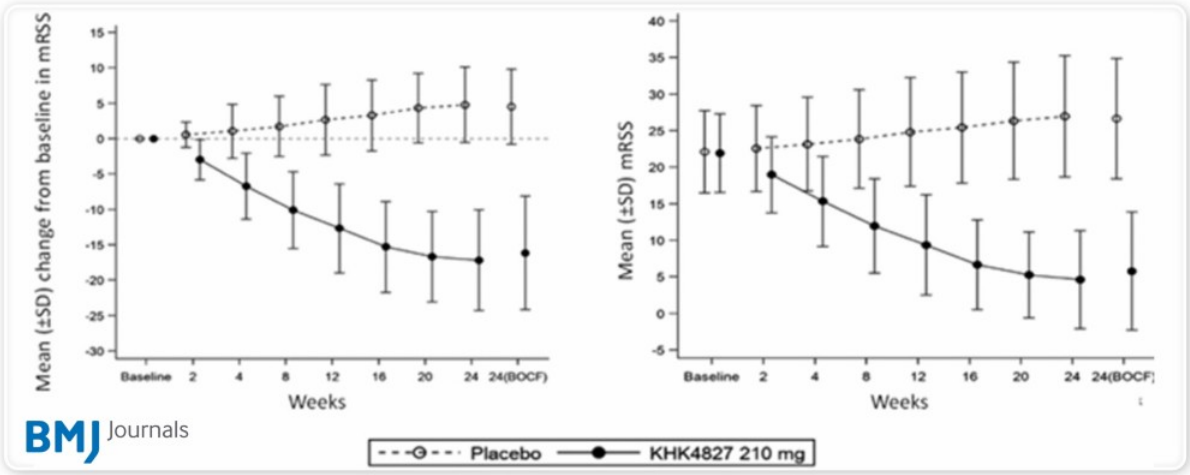
ZB-106 has the potential to treat the TH17 and BAFF components of SSc

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 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, 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

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

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

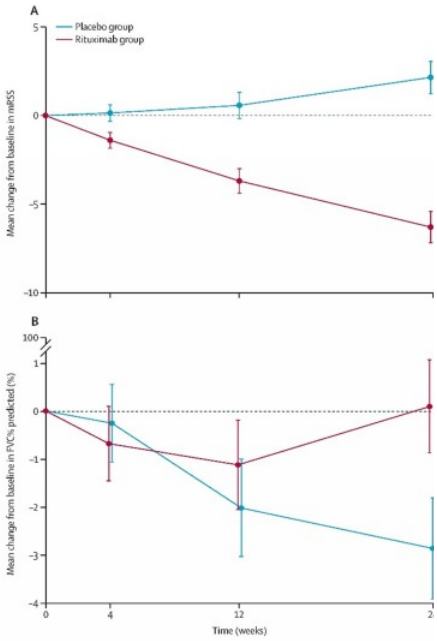
Source: Gordon et al..2018. ArthRheumatol.

B-Cell Depletion Therapy with Rituximab in SSc has Demonstrate 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 improvemen SSc-ILD

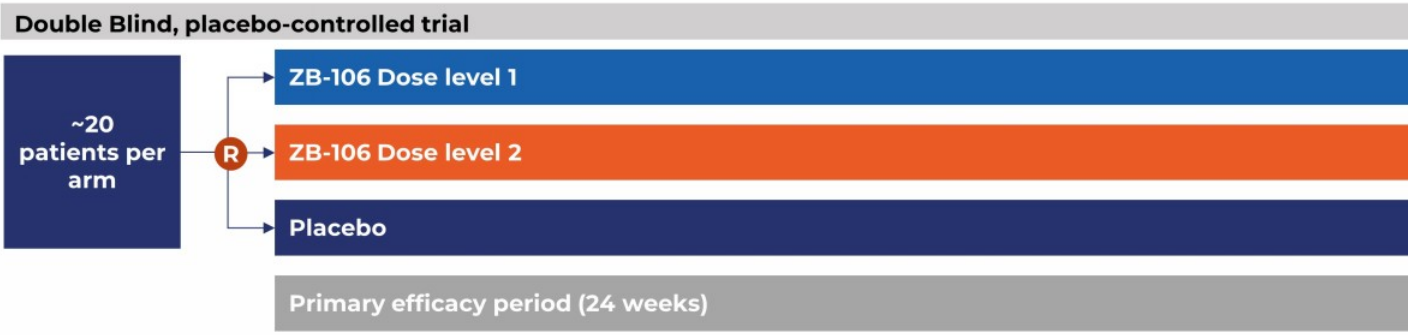


Source. Ebata et al. 2021. Lancet Rheumatol

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 enrollme



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

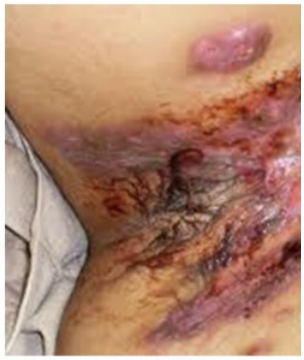
Note: Clinical development planning, including proposed trial designs, are subject to ongoing company review and regulatory feedback and therefore may change


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

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 eye topical cyclosporine and off-label treatments such as steroids or immunosuppressants to manage systemic

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 Unmet Need in HS

First in Class Therapy With Transformational Potential

- Known efficacy of IL-17
- Strong rationale for BAFF
- Known dosing profile

Large Addressable Market

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

1-4% Global Prevalence[2]

$ P r

Significant Unmet Need

1 Drug Approved

Efficacy ceiling with IL17 alone

~10-20%

HiSCORE 50 Placebo-Adjusted[4]

h in re al ea of

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. Eval
3. Jefferies Wall Street Research 4. Cosentyx and Bimzelx Public Presentations, Publications and Research

Despite Multiple IL-17 Development Programs, There is Sign 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 therap 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 p classified as Hurley Stage 2
- Data presented were similar to secukinumab Ph2 open label study in HS suggesting additional studies a 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 pat

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

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	A
Mechanism		TNF-α	IL-17A	IL-17A/F	
Administration		SC	SC / IV	SC	
Phase		PIONEER I & II Phase 3	SUNSHINE & SUNRISE Phase 3	BE HEARD I & II Phase 3	Phase
Dosing		40mg QW for 12W	300mg Q2W for 16W	320mg Q2W for 16W	160n
Total Patients		n=633	n=360	Est. n=579	
Efficacy (HiSCR50)	Non-Placebo Adjusted	42% - 59% at W12	42% - 45% at W16	48% - 52% at W16	
	Placebo-Adjusted	16% - 31% at W12	11%+ at W16	19% - 20% at W16	
Safety / Tolerability	Most Common AEs	Headache 9% - 13% at W12	Headache 9% - 12% at W16	Hidradenitis 7% - 9% at W16	Injecti
	Candidiasis	0% at W12[1]	0% - 3% at W12[1]	4% - 7% at W16	

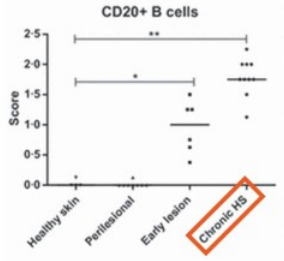
Sources: Company Presentations, Publications and Research.
1. Represents data from psoriasis trial. 2. Represents safety data from psoriatic arthritis trial.

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

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

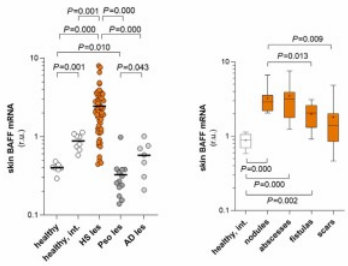
CD20+ B cells in HS Lesions



BAFF in HS

- Increased BAFF expression in HS lesions and tunnels[2-4]
- 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[4]

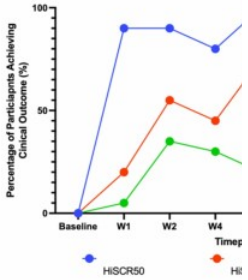
BAFF gene expression in HS



Clinical dat

- B cell depletion with ritu therapeutic benefit[5]
- 4/5 cases report complet lesions[5]
- Modulating B cell functio inhibition provided thera

B cell disruption is ef



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; 6. Jeps

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SYK Antagonism in Hidradenitis Suppurativa Demonstrates Clinical Effic with Greater Response in B-cell Predominant Disease

Rituximab

- 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]

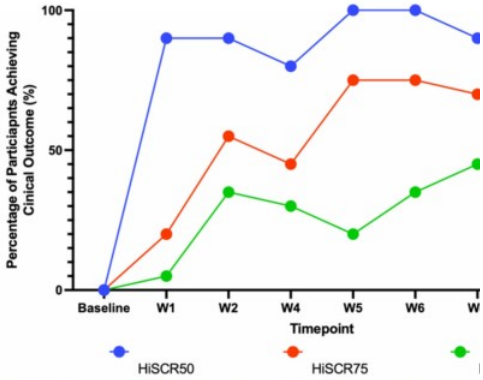
Supplementary Table 1. Demographic and Clinical Features of Patients with HS Treated with Rituximab

	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.

Fostamatinib

- Novel chemical entity targeting Spleen tyrosine
- Approved for the treatment of chronic immune (ITP) in patients who have had insufficient respo therapy
- SYK activation is dependent on BAFF-mediated
- Open label trial in 20 patients with moderate-to-



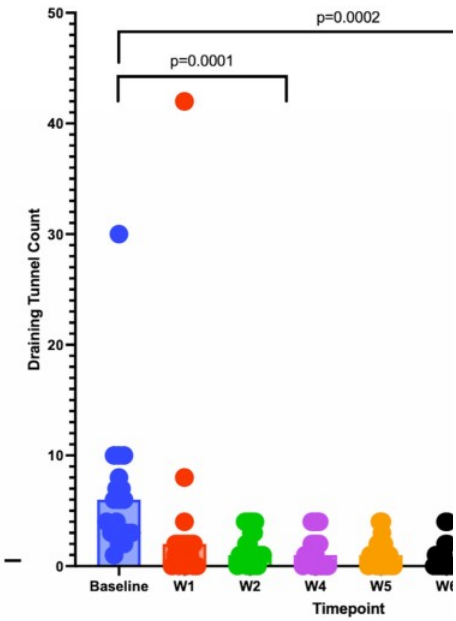
Week of Study	% Achieving HiSCR50	% Achieving HiSCR75
Week 4	85%	45%
Week 12	85%	70%
Izokibep (Acelyrin)[3]	71%	57%

Sources: 1. Seigel et al 2023. JCutanMedSurgery; 2. Jepsen et al. 2023 JAAD; 3. Late Breaking Data @ AAD

Dermal Tunnels: Role in HS Pathogenesis

- Recent literature highlights the role of dermal tunnels in the pathogenesis of HS[1,2]
- The presence of dermal tunnels increased the amount of time needed to achieve HiSCR50 with adalimumab[3]
- 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
- Dermal tunnels in HS are characterized by increased numbers of neutrophils and B cells; and Sabat et al. demonstrated increased BAFF production by neutrophils[2,4]
- The International Hidradenitis Suppurativa Severity Score System (IHS4) was developed to assess inflammatory nodules (1 point), abscesses (2 points) and draining tunnels (4 points)

Treatment with fostamatinib significantly reduced IHS4 draining tunnel cou



1. Frew et al. 2021 Clin Exper Derm; 2. Sabat et al JACI 2023; 3. Moran et al. JID 2017; 4. Gudjonsson et al. 2020; 5. Jepsen et al. 2023. JAAD.

ZB-106 in Hidradenitis Suppurativa
Clinical Development Plan Rationale

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
- 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

Source: Zura Internal Data

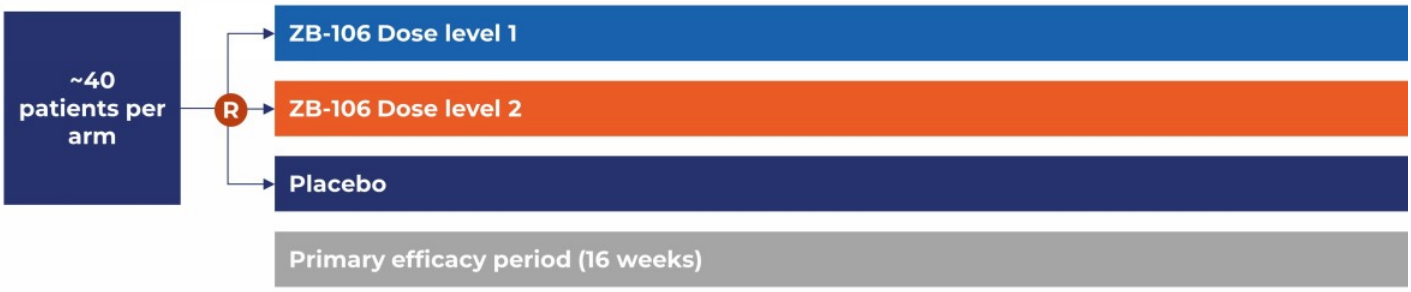
Note: Clinical development planning, including proposed trial designs, are subject to ongoing company review and regulatory feedback and therefore may change

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



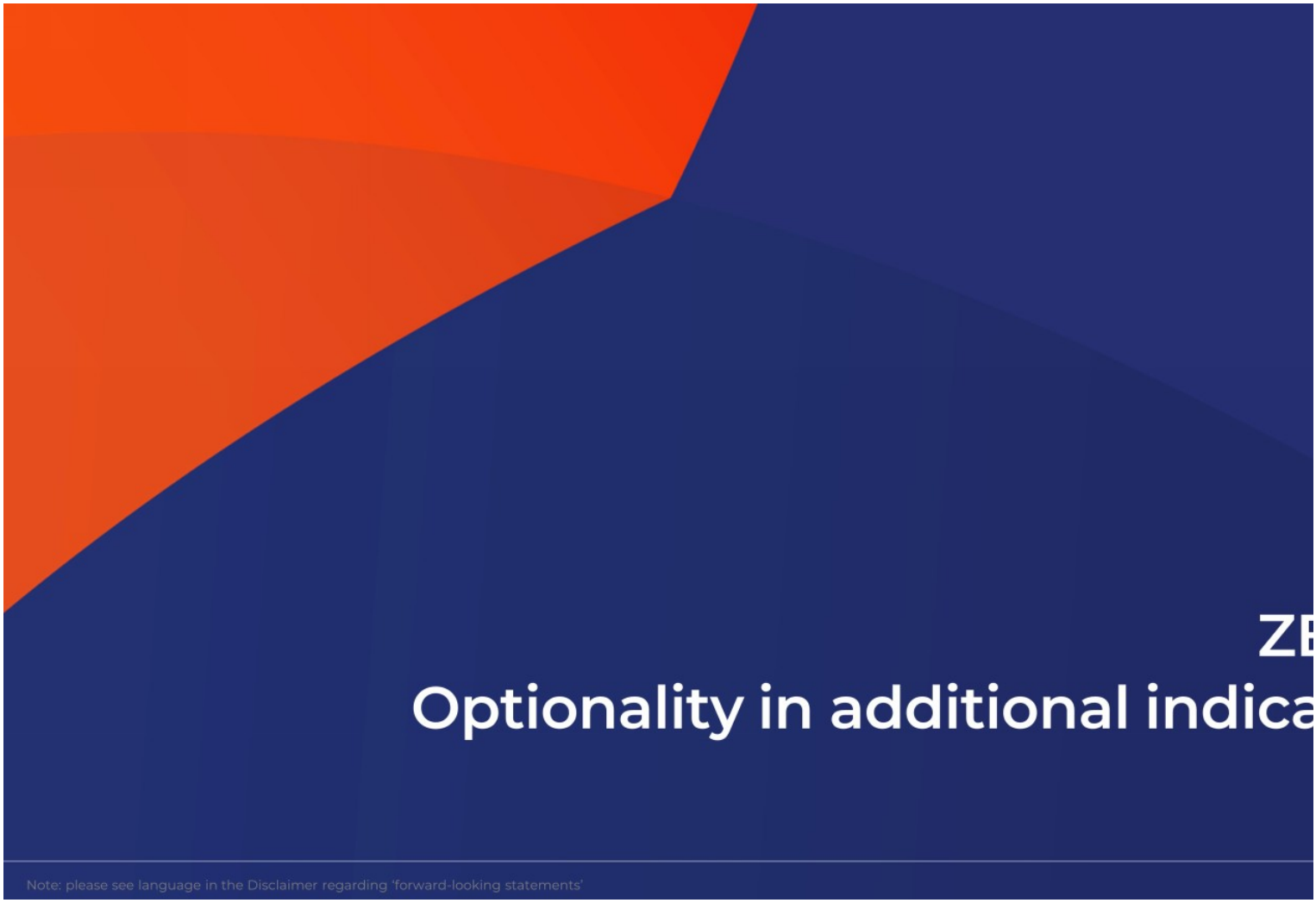
Key efficacy endpoints:

- 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 planning, including proposed trial designs, are subject to ongoing company review and regulatory feedback and therefore may change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Optionality in additional indica
Note: please see language in the Disclaimer regarding 'forward-looking statements'

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	
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	• Arg ph • No Ian ph • Re Tel rea
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	• No Ian • Re Tel

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

ZB
A Potential Best-in-Class Anti
Inhibiting Both IL7 and TSLP Path

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

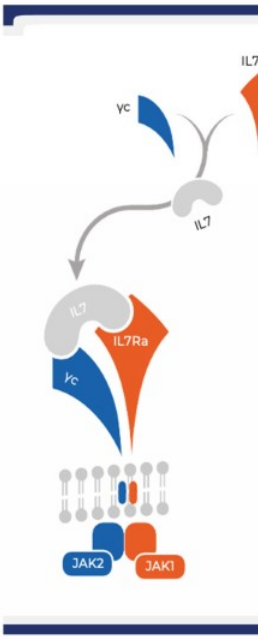
ZB-168 – Asset Overview

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, I
4(23):e126054. 7. doi: 10.1056/NEJMoa2034975

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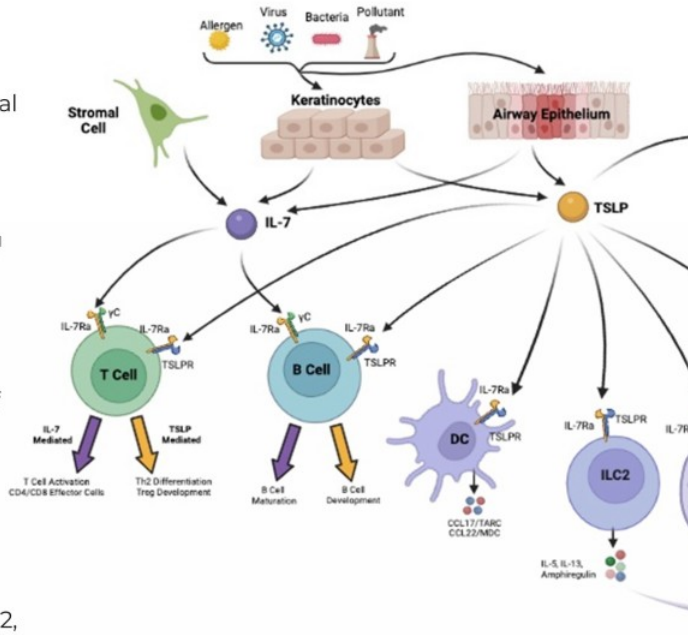
ZB-168 Enables Broad Impact on Epithelial-Driven Inflam by Targeting both TSLP and IL-7

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 In 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

ZB-168 Has Broad Potential Therapeutic Applications

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

Multiple Pote
Indications in
Therapeutic A

- Respirat
- Dermat
- Gastroin

TSLP is an early player in triggering airway inflam
the activation of several immune cells such as der
innate lymphoid cells, monocytes, macrophages a

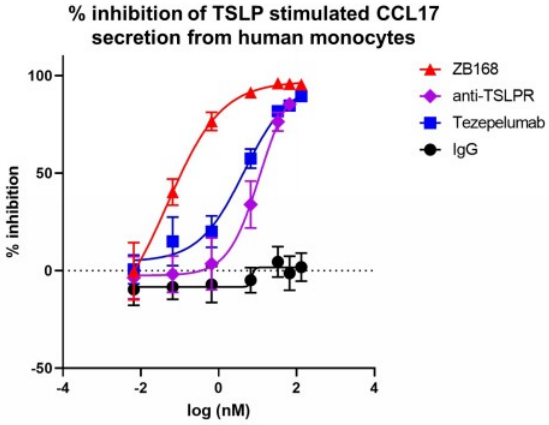
1. Eosinophilic esophagitis; 2. Chronic rhinosinusitis with nasal polyps; 3. Chronic spontaneous urticaria;
4. doi.org/10.3389/fimmu.2018.02692 & doi.org/10.1016/j.isci.2020.101421 ; 5. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6436879/

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

	ZB-168	ADX-914	OSE-127	GSK2618960	Tezepelumab	UPE
Type of Antibody	**Human**	Human	Humanised	Humanised	Human	Hur
Target	**IL7Rα**	IL7Rα	IL7Rα	IL7Rα	TSLP	TSL
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	Ast
*Current Phase	**Phase 1b/2**	Phase 1b/2	Phase 2	Phase 1b	Approved	Pha
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 Pati

*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 Inhibitio



- 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]

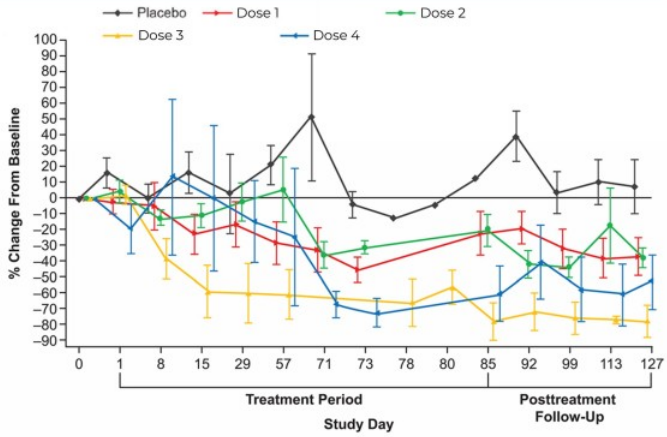
	zurabio	UpstreamBio	AstraZeneca / AMGEN
Asset	**ZB-168** ***(IL7Rα)***	**UPB-101** ***(α-TSLPR)***	**Tezepelumab** ***(TSLP)***
	IL7Rα mAb	α-TSLPR mAb	TSLP mAb
TSLP-Induced Signals	• 7.5 ng/ml / **0.05nM** (CCL17)[1] • 11 ng/ml / **0.07nM** (CCL22)[1] • **0.08 nM** (CCL2)[4]	• 16.1 ng/ml / **0.1nM** (CCL17)[3]	• 67 ng/ml / **0.44nM** (CCL17)[3]
IL7-Induced Signals	• 0.46nM (pSTAT5)[2]	Neg	Neg

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

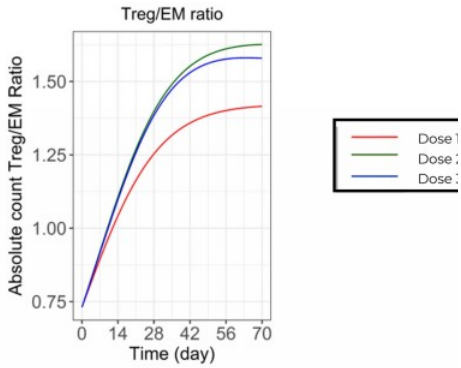
Clinical data in patients demonstrates impact on key T-cell subpopulations

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
- ZB-168 shows 20x greater potency for T_e 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 Diabete Multiple Sclerosis[2]
- In Phase 1 single ascending dose study, ZB-168 was generally well tolerated with deaths and no subjects discontinued, or dose reduced due to Adverse Events (A
- 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 o Type 1 Diabetes (activity in inducing tolerance)[1]

1. doi: 10.1172/jci.insight.126054, 2. Zura internal data, 3. Zura internal data

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Targeting Anti-IL33, an Alarmin
Potential in Multiple Indica

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

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

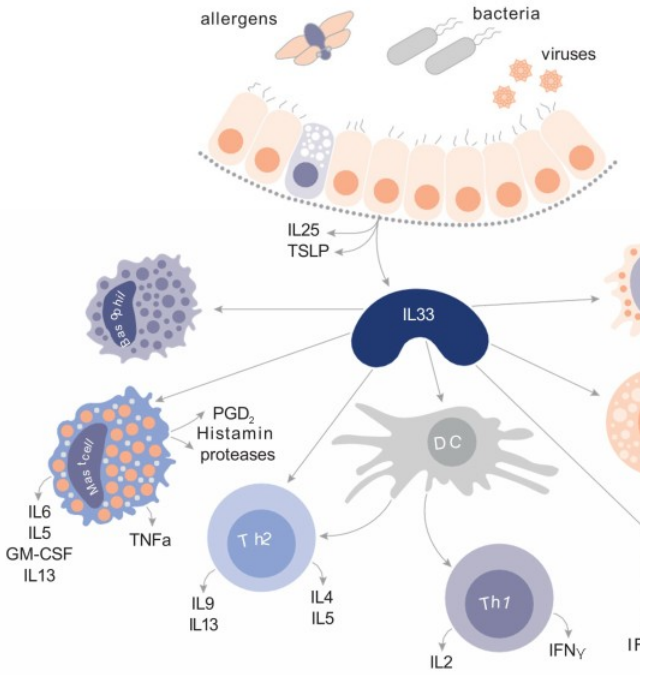
- Advances in the field further validate IL-33 in multiple respiratory disorders with significant global blockbuster opportunities
- Potential 1st and best-in-class opportunities within multiple indications
- Validated pathways in COPD4 and asthma[5]

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

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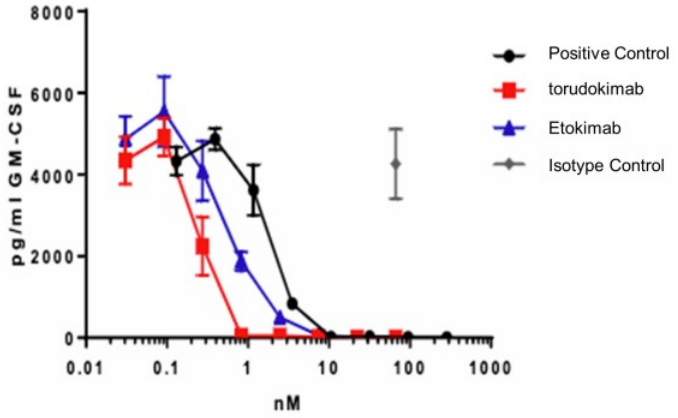
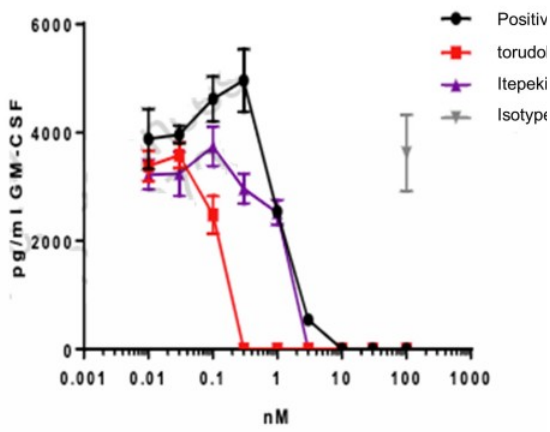
torudokimab Has Potential for “Best-in-Class” Activity

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





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

Source: Zura Bio Internal data

Summary of Clinical Data

- >100 subjects dosed with torudokimab to date, including in a Phase 2 trial in atc 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 sa 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

Building the Next Immunology Leader

Last Updated: June 2023