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): July 27, 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 July 27, 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 July 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: July 27, 2023

ZURA BIO LIMITED

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

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Building the Next Immunology Leader

Corporate Presentation – July 2023

Forward-Looking Statements Disclaimer

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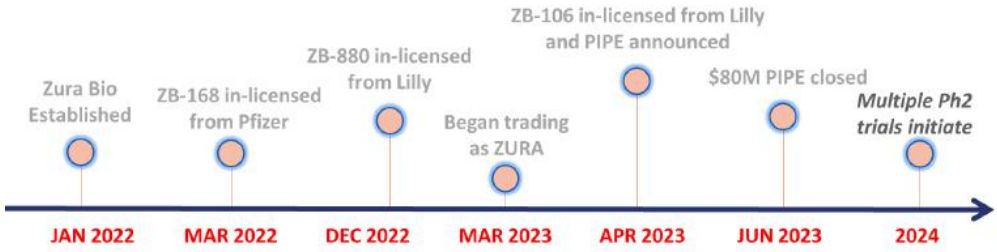
This communication includes "forward-looking statements" within the meaning of the "safe harbor" provisions of the Private Securities Litigation Reform Act of 1995. Words such as "expect," "estimate," "project," "budget," "forecast," "anticipate," "intend," "plan," "may," "will," "could," "should," "believe," "predict," "potential," "continue," "strategy," "future," "opportunity," "would," "seem," "seek," "outlook" and similar expressions are intended to identify such forward-looking statements. Forward-looking statements are predictions, projections and other statements about future events that are based on current expectations and assumptions and, as a result, are subject to risks and uncertainties that could cause the actual results to differ materially from the expected results. These statements are based on various assumptions, whether or not identified in this communication. These forward-looking statements are provided for illustrative purposes only and are not intended to 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 and uncertainties described in the "Risk Factors" sections of Zura Bio's recent filings with the SEC. These filings would identify and address other important risks and uncertainties that could cause actual events and results to differ materially from those contained in the forward-looking statements. Many of these factors are outside Zura Bio's control and are difficult to predict. Many factors could cause actual future events to differ from the forward-looking statements in this communication, including but not limited 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 ability of Zura Bio to successfully conduct research and development activities, grow and manage growth profitably, maintain relationships with customers and suppliers, and retain key employees; (4) costs related to financing transactions and the ongoing costs relating to operating as a public company; (5) changes in the applicable laws or regulations; (6) the possibility that Zura Bio may be adversely affected by other economic, business, and/or competitive factors; (7) the risk of downturns and a changing regulatory landscape in the highly competitive industry in which Zura Bio operates; (8) the impact of the global COVID-19 pandemic; (9) the potential inability of Zura Bio to raise additional capital needed to pursue its business objectives or to achieve efficiencies regarding other costs; (10) the enforceability of Zura Bio's intellectual property, including its patents, and the potential infringement on the intellectual property rights of others, cyber security risks or potential breaches of data security; and (11) other risks and uncertainties described in the Registration Statement and such other documents filed by Zura Bio from time to time with the SEC. These risks and uncertainties may be amplified by the COVID-19 pandemic or other unanticipated global disruption events, which may continue to cause economic uncertainty. Zura Bio cautions that the foregoing list of factors is not exclusive or exhaustive and not to place undue reliance upon any forward-looking 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 result of new information, future developments or otherwise, or should circumstances change, except as otherwise required by securities and other applicable laws.



- **Experienced management team** with proven ability to successfully execute and build a leading market position
- **Multiple Phase 2 ready assets** in important, underserved indications including SSc, HS and Asthma
- **Public on Nasdaq in March 2023**
- **Well capitalized with > $100M cash,* including $80M PIPE in June** (*Deep Track Capital, Great Point Partners, Suvretta Capital, others*) to support development and operations through 2026, including key data readouts

(*) As of July 2023
ABBREVIATIONS: HS, Hidradenitis suppurativa; SSc, Systemic sclerosis

Experienced management team with proven ability to successfully execute and build a leading market position

BOARD OF DIRECTORS

Amit Munshi
Chairman

Garry Neil, M.D.
Director

Jennifer Jarrett
Director

Neil Graham, M.D.
Director

Parvinder Thiara
Director

Sandeep Kulkarni, M.D.
Director

Steve Schoch
Director

Sizeable Total Addressable Market Exists Across Number of Validated Mechanisms

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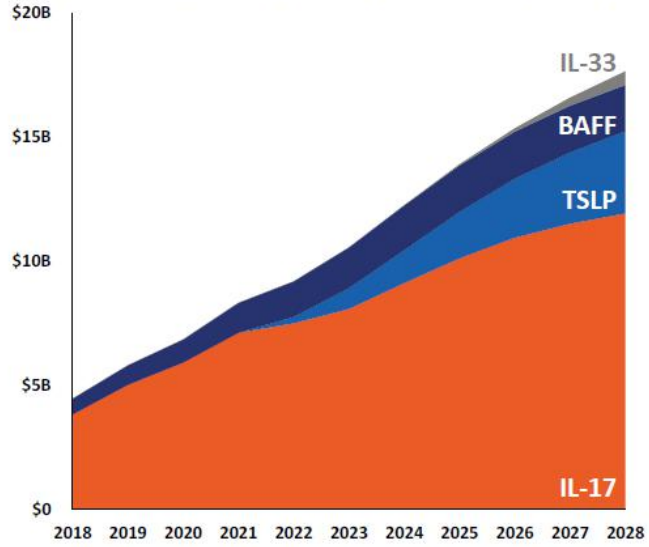
Large TAM present a ~$20B opportunity

Projected TAMs (revenue forecasts for commercial products)[1]



Mechanisms validated across an array of indications

	IL-17	TSLP	BAFF	IL-33
Mkt.	Psoriasis, Psoriatic Arthritis, Ankylosing Spondylitis	Asthma	SLE, Lupus Nephritis	–
Filed	Hidradenitis Suppurativa, Axial Spondyloarthritis	–	–	–
Ph3	Uveitis	Nasal Polyps, Eosinophilic Esophagitis	Sjögren's Syndrome, IgAN	COPD, Asthma
Ph2	–	COPD, CSU	Autoimmune Hepatitis, Systemic Sclerosis, NMO Spectrum Disorder	Atopic Dermatitis, Diabetic Kidney Disease

Sources: Evaluate Pharma. FactSet. Data as of 07/21/23.
(1) Assets included in projected TAM: Cosentyx, Taltz, Bimzelx and Siliq (IL-17), Tezspire (TSLP), Benlysta (BAFF) and itepekimab and tzorakimab (IL-33).

Clinical stage pipeline targeting key immunology pathways

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Zura Bio Program	Indication	Next Clinical Phase				Expected Key Milestone	
		Preclinical	Phase 1	Phase 2	Phase 3		
ZB-106 tibulizumab Anti-BAFFxIL-17	Systemic sclerosis Hidradenitis suppurative					Initiation of asset transfer	✓
						Open IND Rheumatology Division	✓
						Phase 2 initiation	
ZB-880 torudokimab Anti-IL-33	Asthma					Transition Asset from Lily	✓
						Gain FDA feedback on Ph 2/3 design	✓
						Ph2 initiation*	
ZB-168 Anti-IL-7R	Alopecia areata					Transition Asset from Pfizer	✓
						CMC Tech Transfer	✓
						Ph2 initiation**	

(*) pending expected phase 2 and 3 external catalysts in Asthma and Chronic obstructive pulmonary disease (COPD)
(**) pending expected phase 2 external catalysts in Atopic dermatitis (AD), Ulcerative colitis (UC) and Sjögrens syndrome (SjS)

Note: Zura Bio development plan(s) subject to review

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

ZURA'S LEAD ASSET

IgG-scFv engineered by the fusion of ixekizumab (TALTZ®) and tabalumab[1,2,3]

- Neutralizes IL-17A or BAFF regardless of other binding sites occupancy
- Binds to IL-17 and BAFF similarly to Taltz and tabalumab, respectively
- 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 participants dosed to date

Safety profile is consistent with ixekizumab (TALTZ®) and IL-17A class

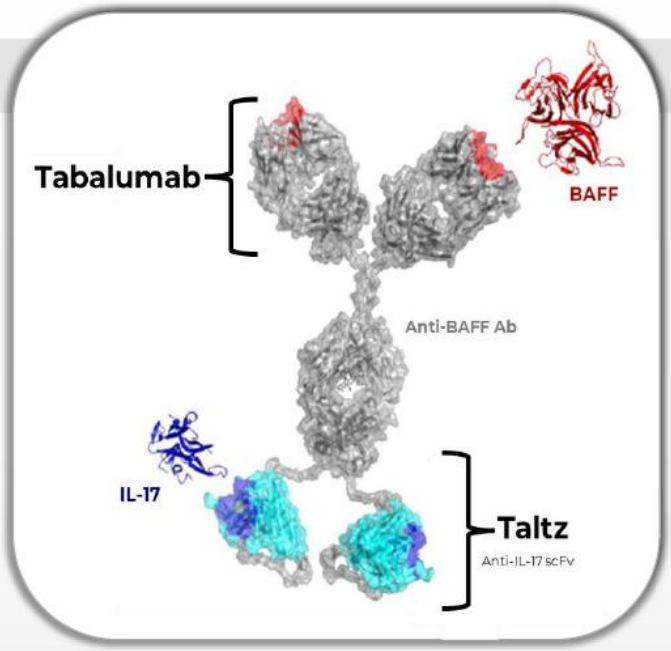
- Chronic toxicity studies completed with no adverse findings

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

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

Low rate of immunogenicity

- Only 1 participant in the MAD study positive for Anti-drug Antibodies (ADAs)



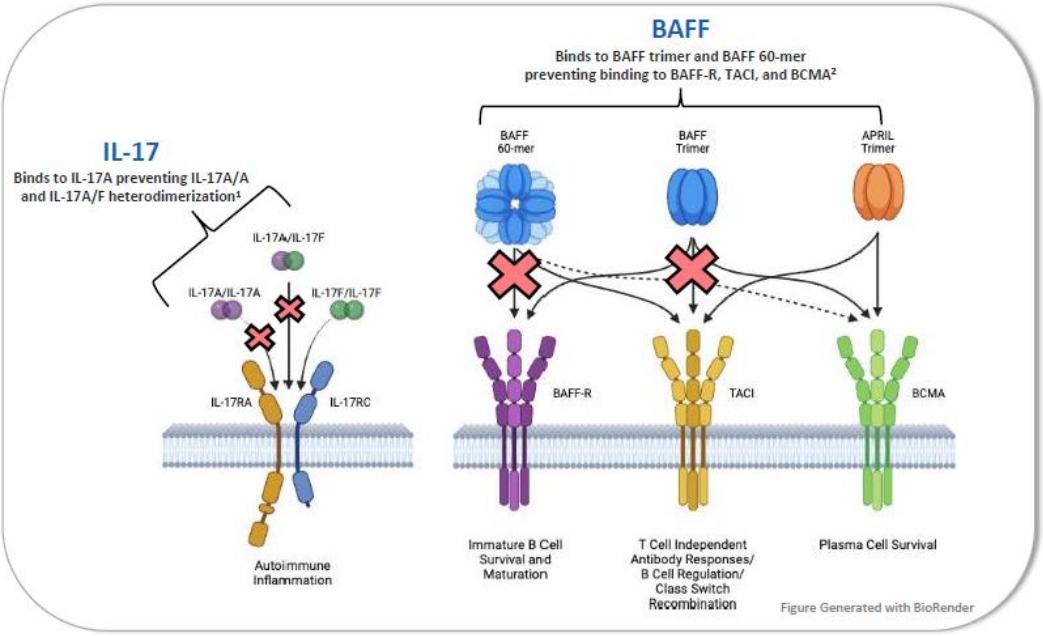
Source: 1. Liu et al. 2016. *J Inflam Research*; 2. Manetta et al. 2014. *J Inflam Research*; 3. Benschop et al. 2019. MAbs

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

POTENTIAL 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



Source: 1. Liu et al. 2016. J Inflam Research; 2. Smulski and Eibel. 2018. Frontimmunol

ZB-106

tibulizumab | Anti BAFFxIL-17 | **Systemic sclerosis (SSc)**

Potential First-in-Class, Dual Antagonist
Combining tabalumab and TALTZ®

Systemic sclerosis is a rare & life-threatening disease with insufficient therapy available

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

- One of the deadliest of the rheumatic diseases
- Associated with severe disability, fibrosis-related organ failure, and premature death
  - Up to 50% of patients develop interstitial lung disease (ILD), the most common cause of mortality in these patients
  - Severe impact on patients' lives with a variable constellation of symptoms, including Raynaud's phenomenon, arthritis, painful ulcers on fingers and toes, thickening and scarring of the skin, shortness of breath, hypertension, and severe fatigue

High unmet medical need with insufficient therapy currently available

- Standard of care relies on immunosuppression therapy and rather toxic biologic agents
- Symptom management with 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

~200,000	40-60%	Zero	$2B+
people with SSc in US, EU and Japan[1]	mortality in 10 years[2]	SSc-specific drugs approved	annual potential market opportunity

Source: Medscape, BMJ best practice 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

ZB-106 is Clinically De-Risked Through P1b

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78 Participants Dosed Across Three P1/1b studies

57 participants with single dose; 21 participants with multiple dose up to 12 weeks

PHARMACOKINETICS

- $t_{1/2}$ 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

Established dosing regimen

PHARMACODYNAMICS

- In Ph1b healthy volunteer study in RA participants 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

Demonstrated PD in participants in ph1b

SAFETY and ADA

- 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 TEAE of infection at target doses
- In the MAD study, one participant had TE-ADAs detected at a low titer

Safety / ADA profile in line with TALTZ®

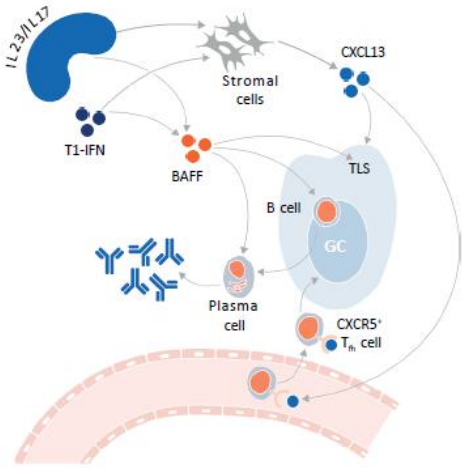
ZB-106 is a highly validated molecule that enables the opportunity to deliver on the promise of both IL-17 and BAFF inhibition in autoimmune disease

ABBREVIATIONS: MAD, multiple ascending dose; SAD, single ascending dose

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

Source: 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.

Treatments in SSc have Demonstrated Improved Clinical Outcomes

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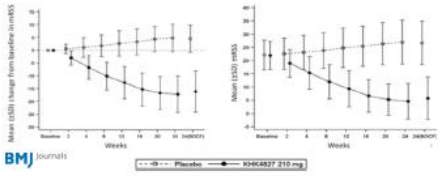
Brodalumab
IL-17 receptor antagonist

- Achieved primary endpoint of 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]
- Demonstrated therapeutic effects on lung/respiratory functions, digital ulcers, the symptoms of gastroesophageal reflux disease, and QOL without noteworthy safety concerns

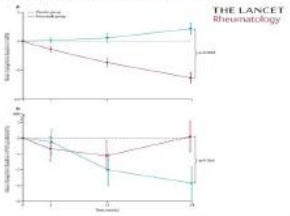


Belimumab
BAFF antagonist

- 52-week, investigator initiated, single center, double blind, placebo-controlled pilot study in 20 participants with dcSSC on MMF
- No significant difference of AEs in active and placebo arms
- Both treatment groups experienced 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
- Orphan Drug Designation granted
- P 2/3 RCT has been initiated for SSc-ILD

Rituximab
CD20 B-cell depleter

- Multiple Studies demonstrated effectiveness of Rituximab in SSc (mainly open label and observational)
- The most compelling data comes from the DESIRES double blind – placebo-controlled trial in 56 pts with SSc1
- Primary endpoint: mRSS change after 24 weeks of treatment
- Rituximab -6.30 points vs. PBO +2.14 points (p < 0.0001)
- 48 / 56 participants had SSc-ILD 2.96% FVC improvement at 24 weeks vs. PBO (p=0.04)



Source: Ebata et al. 2021. Lancet Rheumatol, Gordon et al..2018. ArthRheumatol, Fukasawa et al. 2022. AnnalsRheumDisease; Gordon et al..2018. ArthRheumatol.

Proposed Phase 2 SSc Trial Design*

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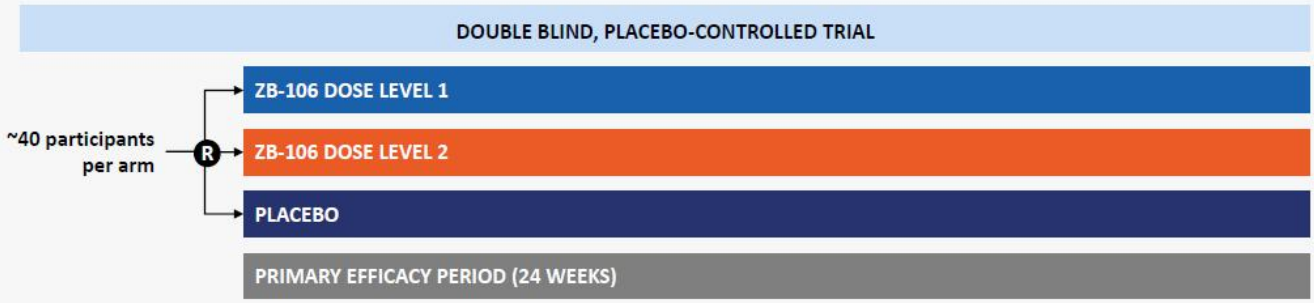
KEY INCLUSION CRITERIA:

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



KEY EFFICACY ENDPOINTS:

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

KEY SAFETY ENDPOINTS:

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

(*) Trial design is preliminary and subject to change

ZB-106

tibulizumab | Anti BAFFxIL-17 | **Hidradenitis suppurativa (HS)**

Potential First-in-Class, Dual Antagonist
Combining tabalumab and TALTZ®

Overview of Hidradenitis suppurativa (HS)

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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

~300K people

living with Hidradenitis Suppurativa in the U.S.

(1-2% global prevalence)

Average of

7 years

to diagnose globally

High unmet need

only 1 approved drug

Only Humira is FDA-approved in HS

CURRENT TREATMENTS ONLY AIM TO MANAGE SYMPTOMS AND INCLUDE PALLIATIVE CARE SUCH AS OTC EYEDROPS, TOPICAL CYCLOSPORINE AND OFF-LABEL STEROIDS OR IMMUNOSUPPRESSANTS TO MANAGE SYSTEMIC SYMPTOMS

Source: 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

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

Phase 2 data show efficacy ceiling may have been reached
Fostamatinib data provide evidence of B-Cell importance

RECENT HS DATA

Company (Asset)		abbvie *Humira*	NOVARTIS *Cosentyx*	ucb *Bimzelx*	ucb *Bimzelx*	MoonLake *Sonelokimab*	ACELYRIN *Izokibep*	rigel *Fostamatinib*
Mechanism		TNF-α	IL-17 A	IL-17 A/F	IL-17 A/F	IL-17 A/F	IL-17 A/A	SYK inhibitor
Administration		SC	SC/IV	SC	SC	SC	SC	PO
Phase		PIONEER I & II Phase 3	SUNSHINE & SUNRISE Phase 3	BE HEARD I & II Phase 3	Phase 2	Phase 2	Pase 2b Part A Open-Label	Phase 2
Dosing		40mg QW for 12W	30mg Q2W for 16W	320mg Q2W for 16W	320mg Q2W for 12W	120mg Q2W for 12W	160mg QW for 12W	150 mg BID for 12W
Total Patients		n = 633	n = 360	Est. n = 579	n=88	n=234	n=30	n=20
Efficacy (HISCR50)	Non-Placebo Adjusted	42% - 59%	42% - 45%	48% - 52%	63%	66%	71%	85%
	Placebo Adjusted	16% - 31%	11%+	19% - 20%	35%	38%	N/A	N/A
Efficacy (HISCR75)	Non-Placebo Adjusted	N/A	N/A	33-36%	50%	43%	57%	70%
	Placebo Adjusted	N/A	N/A	15%-20%	29%	29%	N/A	N/A
Safety / Tolerability	Most Common AEs	Headache 9% - 13%	Headache 9% - 12%	Hidradenitis 7% - 9%	Infections 44%	Nasopharyngitis 15%	Injection site reactions	Nausea 30%
	Candidiasis	0%[1]	0% - 3%[1]	4% - 7%	9%	10.5%	0%[2]	0%

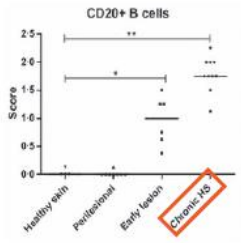
Sources: Company Presentations, Publications and Research.
1. Represents data from psoriasis trial. 2. Represents safety data from psoriatic arthritis trial.

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

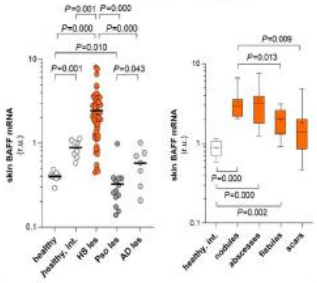
CD20+ B cells in HS Lesions



BAFF in HS

- Increased BAFF expression in HS lesions 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[2]

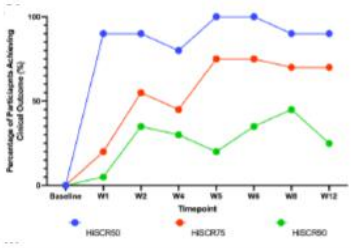
BAFF gene expression in HS



Clinical data in HS

- B cell depletion with rituximab provided therapeutic benefit[5]
- 4/5 cases report complete remission of HS lesions[5]
- Modulating B cell function using fostamatinib (SYK inhibition) provided therapeutic benefit in HS[6]

B cell disruption is efficacious in HS



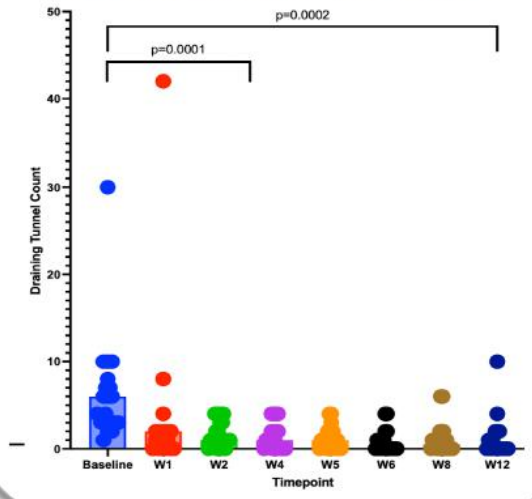
Week 12	% Achieving HiSCR50	% Achieving HiSCR75
Fostamatinib (SYK inhibition) [6]	85%	70%
Izokibep (IL-17)[3]	71%	57%

Source: 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. Jepsen et al. 2023. JAAD

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 (SYK Inhibitor) significantly reduced IHS4 scores and draining tunnel counts[5]



Source: 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

Proposed Phase 2 HS Trial Design*

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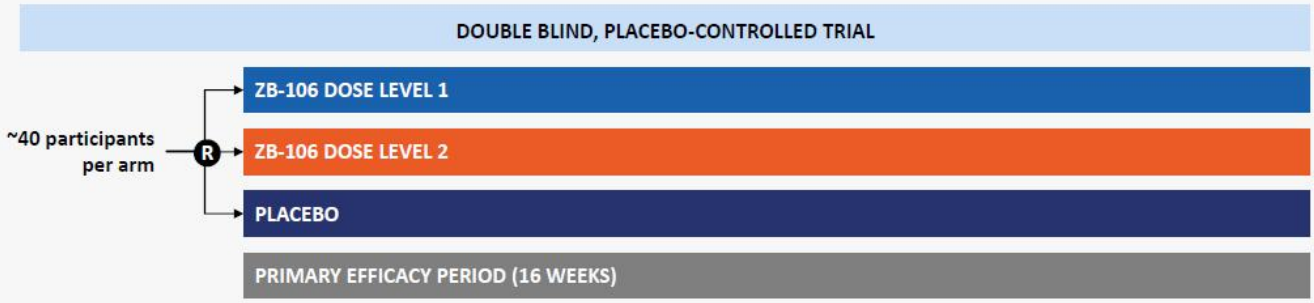
KEY INCLUSION CRITERIA:

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



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

(*) Trial design is preliminary and subject to change

ZB-168

Anti-IL-7R | **Alopecia areata (AA)**

A Potential Best-in-Class Anti-IL-7R Inhibiting Both IL-7 and TSLP Pathways

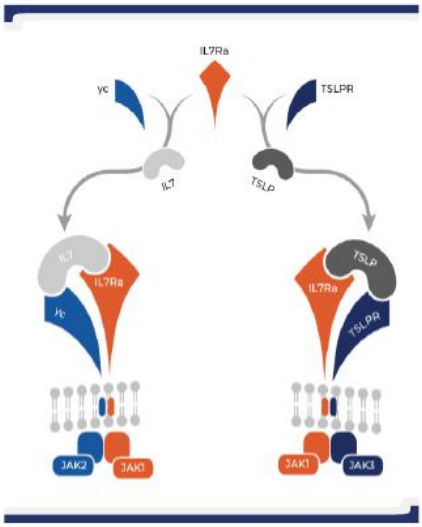
ZB-168 – Asset Overview

ABOUT ZB-168

- IL-7Rα implicated in two key immune pathways[1]: IL-7 and TSLP
- Only anti-IL-7R program to date with human clinical data showing impact on key T-cell sub-populations[2]
- Well tolerated in >90 participants and patients dosed in Phase 1 studies conducted by Pfizer[2, 3]
- Utility in multiple T-cell driven diseases[4]
- 91 subjects dosed to date

MECHANISM OF ACTION

- Inhibition of IL-7Rα blocks both IL-7 and TSLP signaling[5]
- Blocking IL-7Rα 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

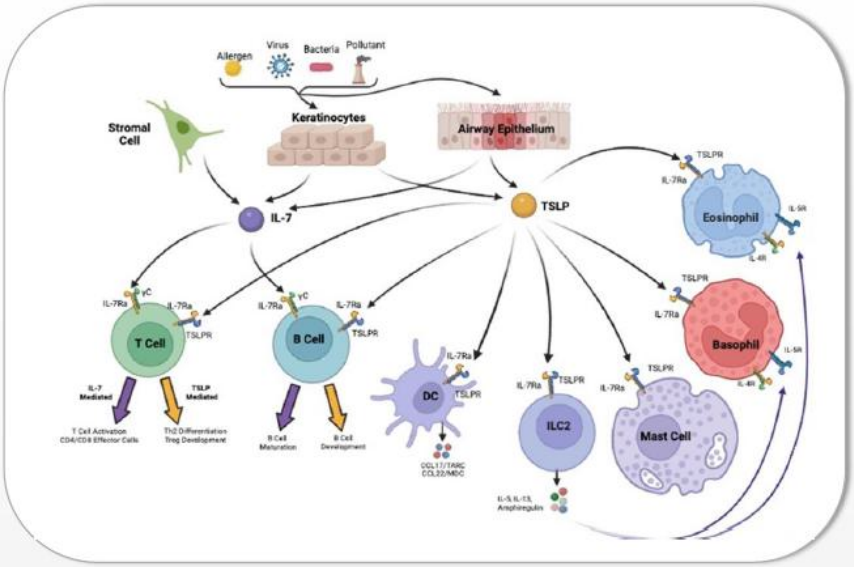
- Advances in the field further validate IL-7 and TSLP in multiple type Th1 & Th2 immune disorders with significant global opportunities
- Potential 1st-in-class opportunities within several indications

Source: 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

TSLP and IL-7 have a Broad Impact on Epithelial-Driven Inflammation

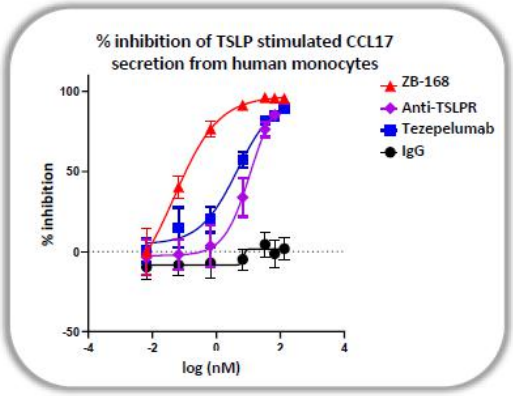
TSLP AND IL-7 PATHWAYS

- 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:gC 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]



Source: 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

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 IL-7 signaling
- ZB-168 is >**300-fold** more potent than Q32Bio / Horizon's ADX-914 in TSLP-induced markers, but similar in IL-7-induced pSTA5[4]

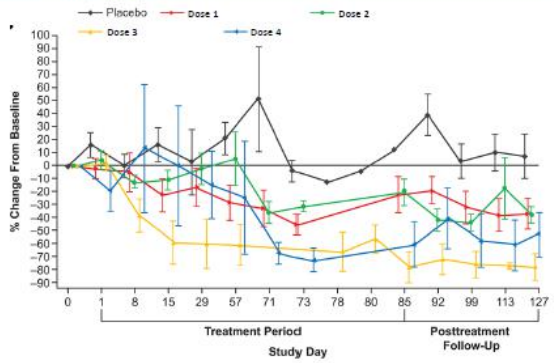
	zurabio	Upstream BIO	AstraZeneca / AMGEN	Q32 BIO / HORIZON
	ZB-168 *(IL-7Rα)*	**UPB-101 *(α-TSLPR)***	**Tezepelumab *(TSLP)***	**ADX-914 *(IL-7Rα)***
	IL-7Rα mAb	α-TSLPR mAb	TSLP mAb	IL-7Rα mAb
TSLP-Induced Signals	▪ 7.5 ng/ml / **0.05nM** (CCL17)[1] ▪ 11 ng/ml / **0.07nM** (CCL22)[1] ▪ **0.08 nM** (CCL2)[4]	16.1 ng/ml / **0.1nM** (CCL17)[3]	67 ng/ml / **0.44nM** (CCL17)[3]	**24 nM** (CCL2)[4]
IL-7-Induced Signals	0.46nM (pSTAT5)[2]	*Neg*	*Neg*	▪ 0.6 nM (IL-7 at 0.25ng/ml)[4] ▪ 2.1nM (IL-7 at 2.5ng/ml)[4]

Source: 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 Impactful on $T_{effector}$ Memory Cells

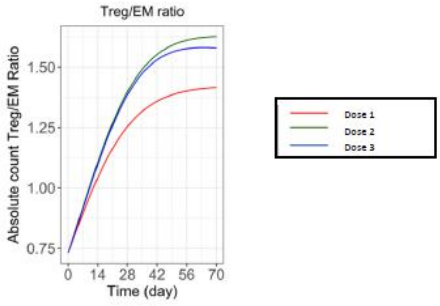
CLINICAL DATA IN PARTICIPANTS DEMONSTRATES IMPACT ON KEY T-CELL SUBPOPULATIONS

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



- Dose dependent response in impact on T-cells
- 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
- Observed increase in the Treg:TEM ratio may shift the balance from autoimmunity towards immune tolerance

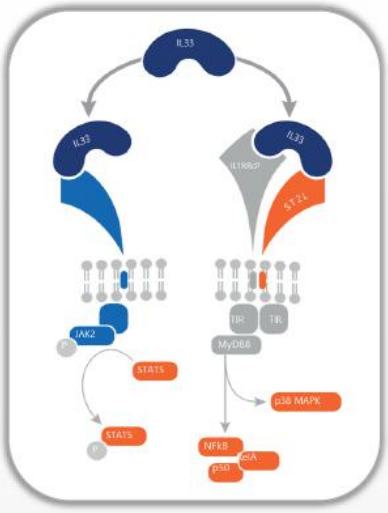
Source: 1. doi: 10.1172/jci.insight.126054, 2. doi:10.1208/s12248-019-0401-3

ZB-880

torudokimab | Anti-IL-33 | **Asthma**

A Potential Best-in-Class Anti-IL-7R Inhibiting Both IL-7 and TSLP Pathways

ZB-880 Asset Overview



About ZB-880 (torudokimab)

- IL-33 implicated in driving Th2 biology through ST2 and Th1/Th17 through alternative signaling[1]
- Well tolerated in Ph1 and Ph2 trials conducted by Eli Lilly[2]
  - 141 healthy volunteers in Ph1 study
  - 103 participants with moderate to severe atopic dermatitis in Ph2
    - Analyses confirmed key biomarker reductions (IL13, periostin and CCL17/TARC) and no ADA impact[3]
  - Potential utility in diseases driven by epithelial inflammation[1]
- The target engagement evaluation suggested binding to IL-33 and treatment emergent ADA had no apparent impact on PK or target engagement

Mechanism of Action

- Inhibition of IL-33 blocks both ST2 and RAGE signaling[4]

Initial Focus on Respiratory, Dermatologic, Gastrointestinal and Orphan Autoimmune Indications

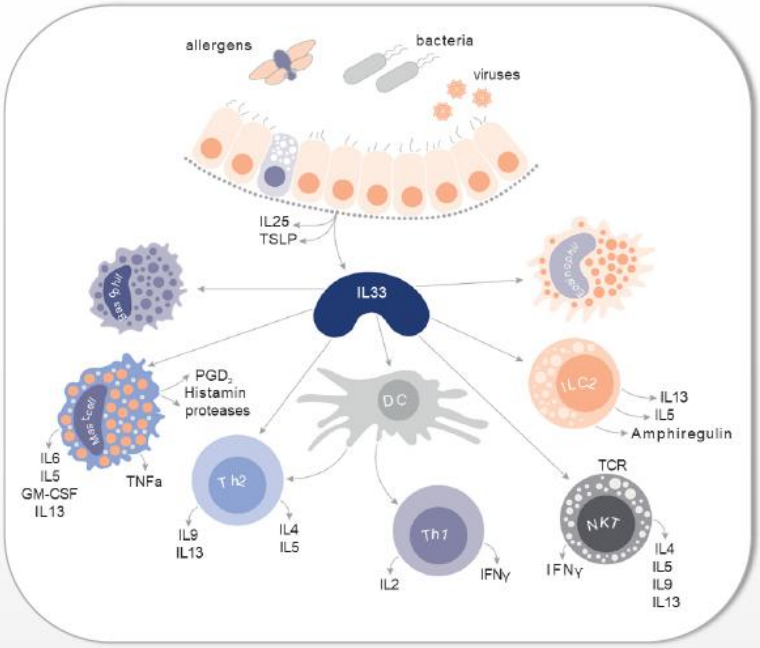
- Potential for 1st-in-class opportunities
- Validated pathways in COPD4 and asthma[5]

Source: 1. 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 doi.org/10.1111/bjd.21631 4. Okragly et al Journal of Inflammation Research 2021:14 3823–3835, 5.. doi:10.1056/NEJMoa2024257

ZB-880 IL-33 Pathway

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 IL-33 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]



Source: 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

ZB-880 Has Potential for "Best-in-Class" Activity

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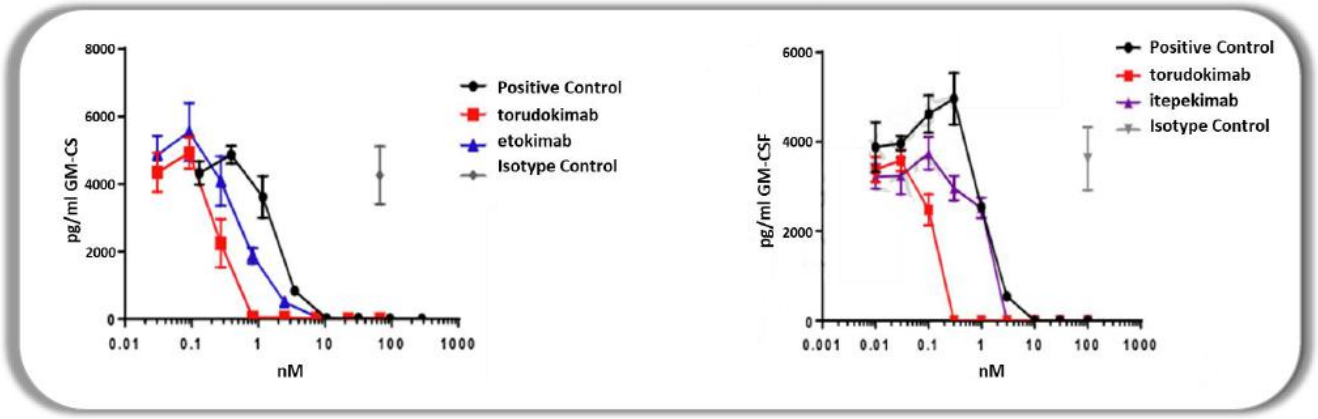
Torudokimab was 2.9 and 5.5-fold more potent than etokimab and itepekimab, respectively, inhibiting IL-33-induced GM-CSF production by human mast cells



Antibody	k_{on} ($M^{-1}s^{-1}$)	k_{off} (s^{-1})	k_d (pM)	Torudokimab Potency
torudokimab (LY3375880)	1.7×10^{6}	6.7×10^{-5}	39	
etokimab (AnaptysBio)	9.4×10^{5}	1.2×10^{-4}	112	2.9x
itepekimab (Regeneron)	7.6×10^{5}	1.6×10^{-4}	215	5.5x

Source: Zura Bio Internal data

Financial Summary and Milestones

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CASH, CASH EQUIVALENTS & INVESTMENTS

$118.2 million[1]

MARKET CAP

$398 million[2]

TOTAL SHARES OUTSTANDING

Fully Diluted, incl warrants, options[1]
66,765,163[1]

COVERING ANALYSTS

Yatin Suneja, Guggenheim Securities

Steven Seedhouse, PhD, Raymond James

Matthew Barcus, PhD, Chardan

Zura Bio Program		Indication	Expected Key Milestone	
ZB-106	tibulizumab Anti-BAFFxIL-17	Systemic sclerosis Hidradenitis suppurative	Initiation of asset transfer	✓
			Open IND Rheumatology Division	✓
			Phase 2 initiation	
ZB-880	torudokimab Anti-IL-33	Asthma	Transition Asset from Lily	✓
			Gain FDA feedback on Ph 2/3 design	✓
			Ph2 initiation*	
ZB-168	Anti-IL-7R	Alopecia areata	Transition Asset from Pfizer	✓
			CMC Tech Transfer	✓
			Ph2 initiation**	

(*) pending expected phase 2 and 3 external catalysts in Asthma and Chronic obstructive pulmonary disease (COPD)
(**) pending expected phase 2 external catalysts in Atopic dermatitis (AD), Ulcerative colitis (UC) and Sjögrens syndrome (SjS)

Note: Zura Bio development plan(s) subject to review

(1) Pro-forma adjusted for $80m private placement. Source: S-1 filling 21st July 2023: https://www.sec.gov/Archives/edgar/data/1855644/000110465923083115/tm235378-3_s1a.htm
(2) Closing price $6.62/ share (as of 26-Jul-2023) , fully diluted share outstanding incl. warrants


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