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: January 5, 2023
(Date of earliest event reported)

ANAPTYSBIO, INC.
(Exact Name of Registrant as Specified in Charter)

Delaware	**001-37985**	**20-3828755**
(State or Other Jurisdiction of Incorporation)	(Commission File Number)	(IRS Employer Identification No.)

10770 Wateridge Circle, Suite 210,
San Diego, CA 92121
(Address of Principal Executive Offices, and Zip Code)

(858) 362-6295
(Registrant's Telephone Number, Including Area Code)

Not Applicable
(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 (see General Instruction A.2. below):

☐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 communication pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))
☐Pre-commencement communication 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
Common Stock, par value $0.001 per share	ANAB	The Nasdaq Stock Market LLC

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (17 CFR §230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (17 CFR §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 2.02. Results of Operations and Financial Condition.

On January 5, 2023, AnaptysBio, Inc. ("AnaptysBio") issued a press release (the "Press Release") announcing certain preliminary, unaudited financial information, including that AnaptysBio expects to report that it had cash and cash equivalents and investments of greater than $575 million as of December 31, 2022. A copy of the press release is attached as Exhibit 99.1 to this Current Report on Form 8-K.

AnaptysBio's audited financial statements for the fiscal year ended December 31, 2022 are not yet available. Accordingly, the preliminary financial information included in the Press Release is an estimate subject to the completion of AnaptysBio's financial closing procedures and any adjustments that may result from the completion of the audit of AnaptysBio's financial statements. The preliminary financial information may differ materially from the actual results that will be reflected in AnaptysBio's audited financial statements when they are completed and publicly disclosed. Additional information and disclosures would be required for a more complete understanding of AnaptysBio's financial position and results of operations as of December 31, 2022.

The information in this Item 2.02, including Exhibit 99.1 to this Current Report on Form 8-K, shall not be deemed to be "filed" for purposes of Section 18 of the Securities Exchange Act of 1934, as amended, or otherwise subject to the liabilities of that section or Sections 11 and 12(a)(2) of the Securities Act of 1933, as amended. The information contained in this Item 2.02 and in the accompanying Exhibit 99.1 shall not be incorporated by reference into any registration statement or other document filed by AnaptysBio with the Securities and Exchange Commission, whether made before or after the date of this Current Report on Form 8-K, regardless of any general incorporation language in such filing (or any reference to this Current Report on Form 8-K generally), except as shall be expressly set forth by specific reference in such filing.

Item 7.01. Regulation FD.

AnaptysBio is furnishing the Press Release, a full copy of which is attached hereto as Exhibit 99.1.

On January 5, 2023, AnaptysBio updated its corporate investor presentation, a full copy of which is attached hereto as Exhibit 99.2.

The information in this item 7.01, including Exhibit 99.1 and Exhibit 99.2, shall not be deemed "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, nor shall it be deemed incorporated by reference into any other filing under the Exchange Act or Securities Act of 1933, as amended, except as expressly set forth by specific reference in such a filing.

Item 9.01. Financial Statements and Exhibits.

(d) Exhibits

Exhibit Number	**Exhibit Title or Description**
99.1	Press release issued by AnaptysBio, Inc. announcing portfolio update, dated January 5, 2023.
99.2	AnaptysBio Corporate Overview January 2023.
104	Cover Page Interactive Data File (the cover page XBRL tags are 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 thereunto duly authorized.

AnaptysBio, Inc.

Date: January 5, 2023

By: /s/Dennis Mulroy

Name: Dennis Mulroy

Title: Chief Financial Officer

AnaptysBio Announces Portfolio Update Across Best-in-Class Immune Cell Modulating Antibodies

- Advancing rosnilimab, its PD-1 agonist, into a global Phase 2b trial to treat rheumatoid arthritis with study initiation in Q3 2023
- Advancing ANB032, its BTLA agonist, into a global Phase 2b trial to treat atopic dermatitis with study initiation in Q2 2023
- Advancing rosnilimab into a second Phase 2 trial, in an indication to be announced, with study initiation anticipated by year-end 2023
- Blinded interim review of alopecia areata Phase 2a data demonstrated rosnilimab "proof of mechanism" with robust reductions in peripheral PD-1 high and PD-1+ T cells and suggests administration was generally safe and well tolerated; However, absolute SALT scores were not supportive of the target product profile and further development in alopecia areata will not be pursued
- Ended 2022 with greater than $575 million and approximately 4 years of capital

SAN DIEGO, Jan. 5, 2023 — AnaptysBio, Inc. (Nasdaq: ANAB), a clinical-stage biotechnology company focused on delivering innovative immunology therapeutics, today announced a portfolio update including initiating development of its wholly owned best-in-class immune cell modulating antibodies in autoimmune and inflammatory diseases with large and significantly underserved patient populations. With cash, cash equivalents and investments greater than $575 million as of December 31, 2022, the company anticipates having approximately 4 years of capital to execute against its non-risk adjusted research and development plan, excluding potential future royalties from its GSK immuno-oncology financial collaboration.

"We have continued to progress our strategic portfolio review and are excited to announce the near-term initiation of two global Phase 2b trials across rosnilimab, our PD-1 agonist, in rheumatoid arthritis and ANB032, our BTLA agonist, in atopic dermatitis. We believe their mechanisms of action, with the potential to restore immune balance by acting directly on cell types mediating disease pathology, have the potential to meaningfully impact large and significantly underserved patient populations," said Daniel Faga, interim president and chief executive officer of AnaptysBio. "We're well capitalized to deliver on multiple Phase 2b readouts across our wholly owned checkpoint agonists as well as to advance ANB033, our anti-CD122 antagonist, through clinical proof-of-concept."

Rosnilimab (PD-1 agonist antibody)

- Rosnilimab, its investigational wholly owned PD-1 agonist, demonstrates best-in-class activity in vitro with superior inhibition of T cell proliferation, reduction in inflammatory cytokine secretion (Th1, Th2, Th17) and depletion of PD-1+ T cells via effector function compared to Lilly PD-1 agonist

- PD-1+ T cells are clinically validated drivers of disease in rheumatoid arthritis (RA)
  - RA patient synovial biopsies have dense T cell infiltrates, with >80% of T cells expressing PD-1 and insufficient PD-L1 expression to down-regulate T cell activity
  - Rosnilimab targets multiple distinct inflammatory mechanisms addressed by approved therapies to treat RA
- Initiation in Q3 2023 of a global Phase 2b trial in moderate-to-severe RA
  - Multi-hundred patient placebo-controlled trial assessing three dose levels of subcutaneously administered rosnilimab for approximately 6 months on well-established endpoints including ACR20/50/70 and DAS28
  - Top-line interim data anticipated by mid-year 2025
- Second global Phase 2 trial, in an indication to be announced, with study initiation anticipated by year-end 2023
- Conducted a blinded interim review of alopecia areata Phase 2a data in December 2022
  - Enrolled 51 patients in a placebo-controlled trial assessing a single 400mg Q4W dosage of subcutaneously administered rosnilimab (randomized 2:1)
  - Study remains blinded with 100% of patients (n=38) through both week 20 dosing period and week 24 primary endpoint and 61% of patients (n=18) through week 32 follow-up period
- Demonstrated rosnilimab “proof of mechanism” with robust reductions in peripheral PD-1+ T cells, including PD-1 high T cell reduction of >80%, across blinded pooled rosnilimab treated and placebo patients, which is consistent with observations in the healthy volunteer Phase 1 trial of >90% in rosnilimab treated patients
- Suggests rosnilimab administration was generally safe and well tolerated
- Severity of Alopecia Tool (SALT) scores were not supportive of achieving the target product profile and further development in alopecia areata will not be pursued
  - While select patients observed changes from baseline SALT scores at week 24, no patients achieved an absolute SALT score <20
  - Interim results suggest that target efficacy was not achieved potentially due to an inadequate tested dose level, limited duration of treatment, and/or complexity of disease biology including the hair growth cycle
  - Unblinded week 32 results, tissue biopsies and additional translational data defining the extent of PD-1 modulation in the periphery and hair follicle will be available in H2 2023

ANB032 (BTLA agonist antibody)

- ANB032, its investigational wholly owned BTLA agonist, demonstrates best-in-class activity in vitro with superior inhibition of T cell proliferation and reduction in inflammatory cytokine secretion (Th1, Th2, Th17) compared to Lilly BTLA agonist
- While Th2 targeted therapies provide benefit to patients with chronic moderate-to-severe atopic dermatitis (AD), there is compelling evidence that AD is broader than a Th2 driven

disease, as Th1, Th17 and other cell types, including dendritic cells, may contribute significantly to its pathogenesis
  - ANB032 inhibits inflammatory activity of Th1, Th2 and Th17 and modulates additional cell types such as B cells and dendritic cells, with the potential for broader, deeper and more durable responses than more narrowly targeted interventions
- Initiation in Q2 2023 of a global Phase 2b trial in moderate-to-severe AD
  - IND cleared by the FDA in December 2022
  - 160 patient placebo-controlled trial assessing three dose levels of subcutaneously administered ANB032 (randomized 1:1:1:1) for 12 weeks on well-established endpoints, including EASI75 and IGA 0/1
  - Top-line interim data anticipated by year-end 2024

ANB033 (anti-CD122 antagonist antibody)

- ANB033, its investigational wholly owned anti-CD122 antagonist antibody, targets the common beta subunit shared by the IL-15 and IL-2 receptors
  - IL-15 signaling mediates the survival and maintenance of tissue resident memory T cells (T_{RM})
  - The presence of long-lived and persistent T_{RM} have been shown to drive tissue-specific immune-mediated inflammation
- IND anticipated H1 2024

Legacy clinical-stage cytokine antagonist programs available for outlicensing

- Imsidolimab, its investigational wholly owned anti-IL-36r antagonist antibody, is in Phase 3 trials for generalized pustular psoriasis (GPP)
  - Top-line data from the GEMINI-1 Phase 3 trial anticipated Q4 2023
  - Plan to outlicense imsidolimab prior to potential FDA approval
- Etokimab, its investigational wholly owned anti-IL-33 antagonist antibody, is Phase 2/3-ready for the treatment of respiratory disorders
  - No further internal investment in etokimab is being pursued

GSK immuno-oncology financial collaboration

- Dostarlimab, an anti-PD-1 antagonist antibody, cobolimab, an anti-TIM-3 antagonist antibody, and GSK4074386, an anti-LAG-3 antagonist antibody, were discovered at AnaptysBio and licensed by GSK
- JEMPERLI (dostarlimab-gxly) has the potential for a first-in-class approval in primary advanced or recurrent endometrial cancer after meeting the primary endpoint in the pivotal RUBY Phase 3 trial demonstrating JEMPERLI plus chemotherapy significantly improved PFS versus chemotherapy plus placebo
  - Regulatory submissions anticipated H1 2023
  - GSK expects to publish full results in a medical journal and present at an upcoming scientific meeting

- Dostarlimab plus ZEJULA in the pivotal FIRST Phase 3 trial in 1st line ovarian cancer is ongoing with an interim analysis expected in H2 2023
- Dostarlimab plus cobolimab plus chemotherapy vs. dostarlimab plus chemotherapy is in the pivotal COSTAR Lung Phase 3 trial in advanced non-small cell lung cancer in patients who have progressed on prior anti-PD-(L)1 therapy

About AnaptysBio

AnaptysBio is a clinical-stage biotechnology company focused on delivering innovative immunology therapeutics. It is developing immune cell modulating antibodies, including two checkpoint agonists in clinical-stage development, for autoimmune and inflammatory diseases: rosnilimab, its PD-1 agonist, in a planned Phase 2b trial for the treatment of moderate-to-severe rheumatoid arthritis; and ANB032, its BTLA agonist, in a planned Phase 2b trial for the treatment of moderate-to-severe atopic dermatitis. Its preclinical immune cell modulator portfolio includes ANB033, an anti-CD122 antagonist antibody for the treatment of autoimmune and inflammatory diseases. AnaptysBio has developed two cytokine antagonists available for outlicensing: imsidolimab, an anti-IL-36r antagonist, in Phase 3 for the treatment of generalized pustular psoriasis, or GPP, and etokimab, an anti-IL-33 antagonist that is Phase 2/3 ready. AnaptysBio has also discovered antibodies licensed to GSK in a financial collaboration for immune-oncology, including an anti-PD-1 antagonist (JEMPERLI (dostarlimab-gxly)), an anti-TIM-3 antagonist (cobolimab, GSK4069889) and an anti-LAG-3 antagonist (GSK4074386).

Forward-Looking Statements

This press release contains forward-looking statements within the meaning of the "safe harbor" provisions of the Private Securities Litigation Reform Act of 1995, including, but not limited to: the timing of initiation of the company's clinical trials, including rosnilimab's clinical trials in rheumatoid arthritis and in a second indication and ANB032's clinical trial in atopic dermatitis; the timing of the release of data from the company's clinical trials, including imsidolimab's Phase 3 clinical trial in GPP, rosnilimab's Phase 2b clinical trial in rheumatoid arthritis and ANB032's Phase 2b clinical trial in atopic dermatitis; the timing of ANB033's IND filing; statements regarding efficacy, safety and proof of mechanism from blinded data from rosnilimab's clinical trial in alopecia areata; the company's ability to find a licensing partner for imsidolimab or etokimab and the timing of any such transaction; and the company's projected cash runway. Statements including words such as "plan," "continue," "expect," or "ongoing" and statements in the future tense are forward-looking statements. These forward-looking statements involve risks and uncertainties, as well as assumptions, which, if they do not fully materialize or prove incorrect, could cause its results to differ materially from those expressed or implied by such forward-looking statements. Forward-looking statements are subject to risks and uncertainties that may cause the company's actual activities or results to differ significantly from those expressed in any forward-looking statement, including risks and uncertainties related to the company's ability to advance its product candidates, obtain regulatory approval of and ultimately commercialize its product candidates, the timing and results of preclinical and clinical trials, the company's ability to fund development activities and achieve development goals, the company's ability to protect intellectual property and other risks and uncertainties described under the

heading “Risk Factors” in documents the company files from time to time with the Securities and Exchange Commission. These forward-looking statements speak only as of the date of this press release, and the company undertakes no obligation to revise or update any forward-looking statements to reflect events or circumstances after the date hereof.

Contact:
Dennis Mulroy
AnaptysBio, Inc.
858.732.0201
dmulroy@anaptysbio.com

AnaptysBio

Corporate Overview

January 2022

Nasdaq: ANAB

Safe Harbor Statement

This presentation and any accompanying oral presentation contains forward-looking statements within the meaning of the "safe harbor" provisions of the Private Securities Litigation Reform Act of 1995, including, but not limited to: the timing of initiation of the Company's clinical trials, including rosnilimab's clinical trials in rheumatoid arthritis and in a second indication and ANB032's clinical trial in atopic dermatitis; the timing of the release of data from the Company's clinical trials, including imsidolimab's Phase 3 clinical trial in GPP, rosnilimab's Phase 2b clinical trial in rheumatoid arthritis and ANB032's Phase 2b clinical trial in atopic dermatitis; the timing of ANB033's IND filing; statements regarding efficacy, safety and proof of mechanism from blinded data from rosnilimab's clinical trial in alopecia areata; the Company's ability to find a licensing partner for imsidolimab or etokimab and the timing of any such transaction; and the Company's projected cash runway. Statements including words such as "plan," "continue," "expect," or "ongoing" and statements in the future tense are forward-looking statements. These forward-looking statements involve risks and uncertainties, as well as assumptions, which, if they do not fully materialize or prove incorrect, could cause its results to differ materially from those expressed or implied by such forward-looking statements. Forward-looking statements are subject to risks and uncertainties that may cause the company's actual activities or results to differ significantly from those expressed in any forward-looking statement, including risks and uncertainties related to the company's ability to advance its product candidates, obtain regulatory approval of and ultimately commercialize its product candidates, the timing and results of preclinical and clinical trials, the company's ability to fund development activities and achieve development goals, the company's ability to protect intellectual property and other risks and uncertainties described under the heading "Risk Factors" in documents the company files from time to time with the Securities and Exchange Commission. These forward-looking statements speak only as of the date of this presentation, and the company undertakes no obligation to revise or update any forward-looking statements to reflect events or circumstances after the date hereof.

Certain information contained in this presentation may be derived from information provided by industry sources. The Company believes such information is accurate and that the sources from which it has been obtained are reliable. However, the Company cannot guarantee the accuracy of, and has not independently verified, such information.

The trademarks included herein are the property of the owners thereof and are used for reference purposes only. Such use should not be construed as an endorsement of such products.

Best-In-Class Immune Cell Modulating Antibodies

Potential to restore immune balance across autoimmune and inflammatory diseases

Immune Cell Modulators			Cytokine Antagonists (legacy programs for outlicensing)
Rosnilimab (PD-1 agonist) P2b in Rheumatoid Arthritis	ANB032 (BTLA agonist) P2b in Atopic Dermatitis	ANB033 (CD122 antagonist) IND-enabling	Imsidolimab (IL-36r) P3 in GPP Etokimab (IL-33) P2b/3-ready in respiratory disorders
Autoimmune and inflammatory diseases including dermatology, gastroenterology and rheumatology			

Research-driven	Strong capital position	GSK immuno-oncology financial collaboration
Preclinical pipeline of immunology targets	~4 years of cash with >$575mm as of YE 2022	Significant royalty potential

Immune Cell Modulator Development Plan

Three phase 2 initiations in 2023

	Antibody Program	Therapeutic Indication	Development Stage and Anticipated Milestones				
			Lead Optimization	IND Enabling	Phase 1	Phase 2	Phase 3
Immune Cell Modulators	**Rosnilimab** (PD-1 agonist)	Rheumatoid Arthritis				P2b initiation Q3 2023 Top-line data mid 2025	
		Inflammatory Disease				Trial initiation YE 2023	
	ANB032 (BTLA agonist)	Atopic Dermatitis				P2b initiation Q2 2023 Top-line data YE 2024	
	ANB033 (CD122 antagonist)	Inflammatory Diseases		IND submission H1 2024			
Cytokine Antagonists	Legacy Programs Available for Outlicensing						
	Imsidolimab (IL-36r antagonist)	Generalized Pustular Psoriasis (GPP)				*Outlicense prior to FDA Approval*	Top-line data Q4 2023
	Etokimab (IL-33 antagonist)	Respiratory Disorders				P2b/3-ready *no further internal investment*	

All programs discovered internally using AnaptysBio's proprietary antibody platform technology

AnaptysBio

Advantages of Immune Cell Modulation in Treatment of Autoimmune and Inflammatory Diseases

Moderate-to-Severe Inflammatory Diseases are Complex, Leading to Shortcomings in SOC Therapies

Efficacy

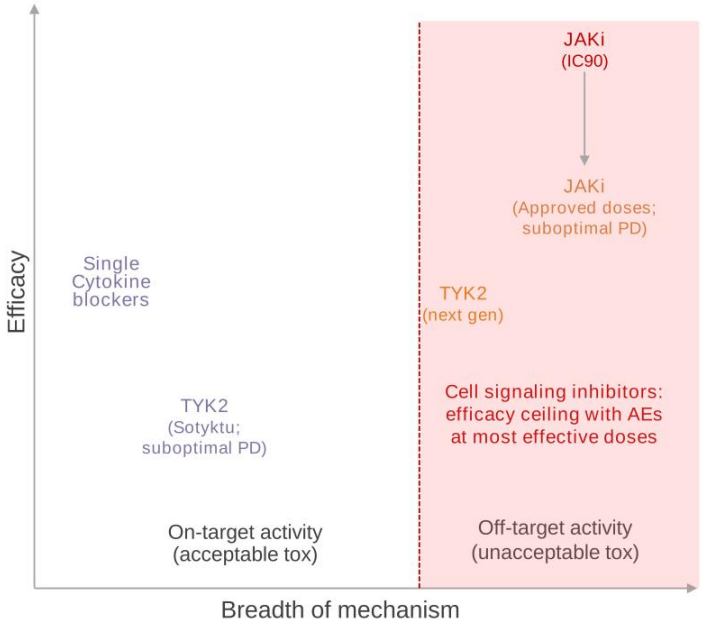
- Complex, heterogeneous diseases involve upregulation of multiple cytokines
- Treatment requires more than a single cytokine blocker

Durability

- Downstream intervention does not target underlying dysregulation
- Chronic treatment required to suppress symptoms

Safety

- Cell signaling inhibitors (e.g. JAKi) with broad activity, have significant tox
  - Black-box warning risks include major cardiac and thrombotic events



Immune Cell Modulators May Restore Immune Balance Across Moderate-to-Severe Complex Diseases

Efficacy: Act directly on dysregulation

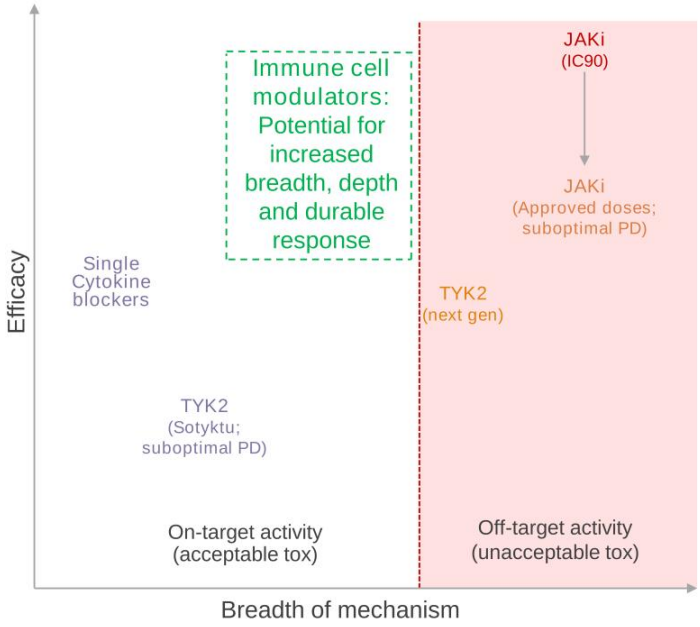
- Suppression of immune cells may increase breadth and depth of response

Durability: Longer remission

- Restore tolerance by modulating key immune cell types
- Potential for durable response

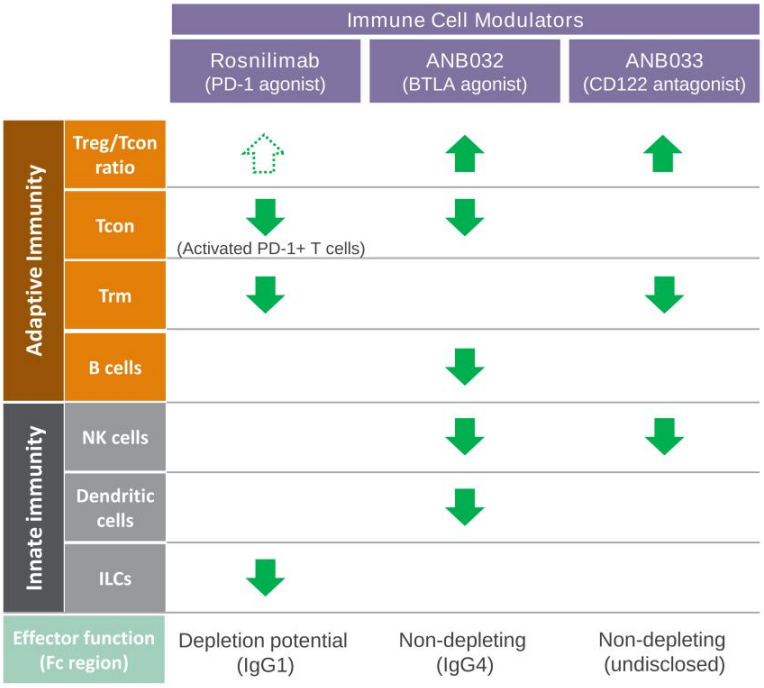
Safety: Targeting immune cells

- Preferential target expression on activated immune cells may enable wider therapeutic window
- Growing agonist class experience has been well-tolerated to date
- No evidence of carcinogenicity with checkpoint inhibitors in I&I, such as abatacept, in decades of use



Immune Cell Modulators are a Novel Therapeutic Class

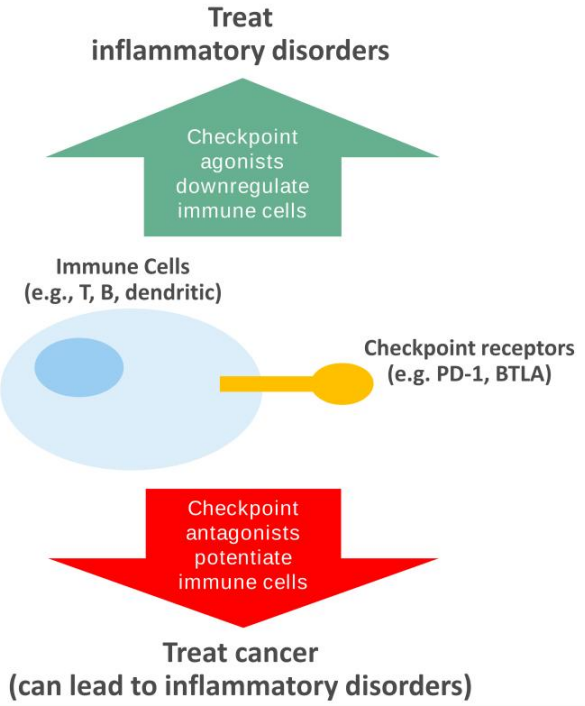
Suppress activated immune cell types to restore immune balance



Potential Mechanisms of Action

1. Bias Treg/Tcon ratio towards tolerance at sites of aberrant inflammation and systemically
2. Modulation of adaptive immunity may enable resolution of inflammatory cycle
3. Modulation of innate immunity may support return to homeostasis
4. Potential to deplete (rosnilimab) or induce death (ANB033) in pathogenically targeted activated cells

Anti-Inflammatory Checkpoint Agonist Antibodies

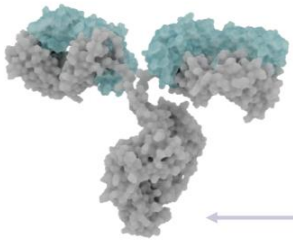


- Checkpoint agonists for PD-1 and BTLA down regulate T cell mediated immune responses
  - BTLA also present on B cells and dendritic cells
- Potential applicability across complex diseases
  - Dermatology
  - Gastroenterology
  - Rheumatology
  - Respiratory
  - Neurology
- Potential for concomitant or sequential combination with targeted immune-suppressive therapies

Combination of Characteristics Optimize Agonistic Signaling

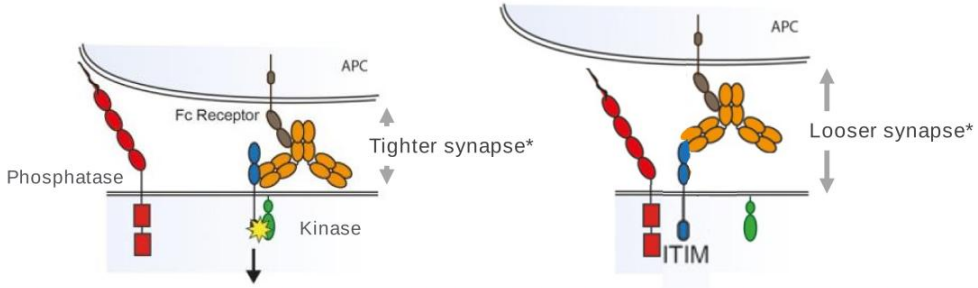
Antigen binding characteristics

- **Antigen binding epitope:**
  - Spare natural ligand binding interaction
  - Bind proximal to the membrane to enhance tight synapse formation
- **Antigen binding affinity:**
  - Optimal (not maximal affinity)



Fc binding characteristics

- **Fc receptor binding affinity:**
  - Tight synapse formation
  - Clustering of checkpoint receptors promoted via Fc anchoring
- **Effector function:**
  - Isotype selection may enable depletion (NK cell-mediated ADCC and/or monocyte-mediated ADCP)



Tight immune synapse optimizes agonistic activity by excluding large phosphatases that dephosphorylate the checkpoint receptor

*Figure adapted from Frontiers in Immunology. 2018 Oct 8; 9: 2306

AnaptysBio

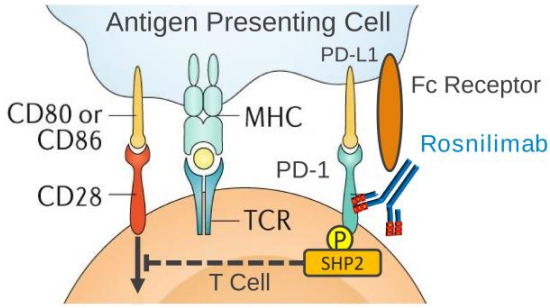
Rosnilimab (PD-1 agonist mAb)

Introduction

Rosnilimab: Best-in-Class PD-1 Agonist Antibody

Suppression of T cell mediated inflammatory diseases

- Rosnilimab: IgG1 antibody that binds to PD-1+ T cells
  - Membrane proximal binding epitope contributes to differentiated potency
- Acts on inflamed tissue and periphery to:
  - Mimic PD-L1 function to down-regulate T cell activity (agonistic signaling)
  - Deplete PD-1+ T cells (effector function)
  - Prevent migration of PD-1+ T cells to inflamed tissue by peripheral depletion
- Specific expression on activated T cell subclasses (e.g. Th1, Th2, Th17)
- Rosnilimab targets multiple distinct inflammatory mechanisms addressed by approved therapies



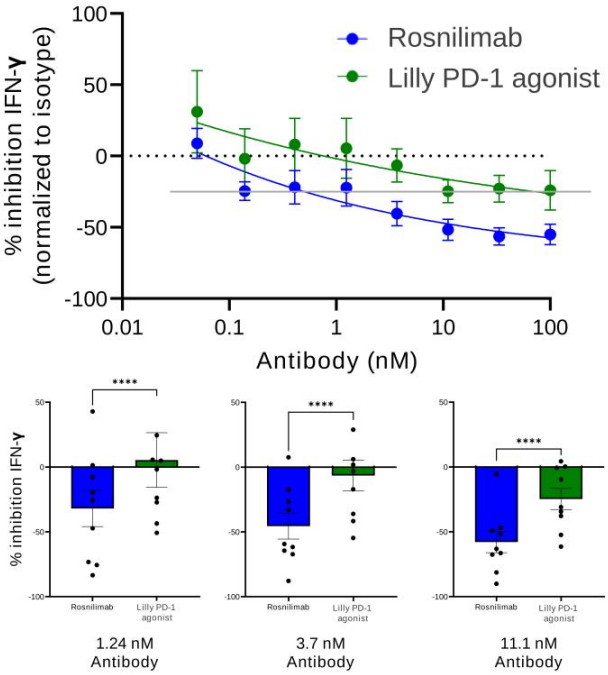
Rosnilimab MOA

Inhibit T cell proliferation and cytokine secretion	Deplete PD-1+ T cells	Prevent migration of pathogenic T cells to inflamed tissue
ORENCIA (abatacept) (CD80/86 modulator)	--	ZEPOSIA (ozanimod) (S1PR inhibitor); Entyvio (vedolizumab) (α4β7 blockers)

Rosnilimab Significantly Reduces IFNγ in Healthy PBMCs Compared to Lilly PD-1 Agonist

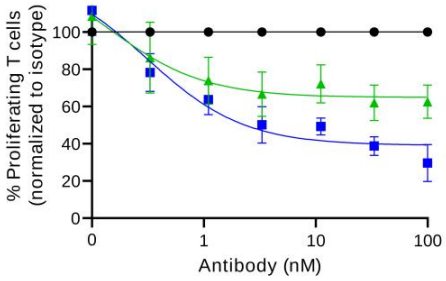
- Rosnilimab shows meaningful IFNγ inhibition beginning at low concentrations
- Potency differentiation from Lilly's PD-1 agonist is sustained across clinical concentrations

Tetanus toxoid recall stimulation assay measures antigen-specific immune cell response.
Prior immunity in majority of population due to tetanus vaccination, N=9



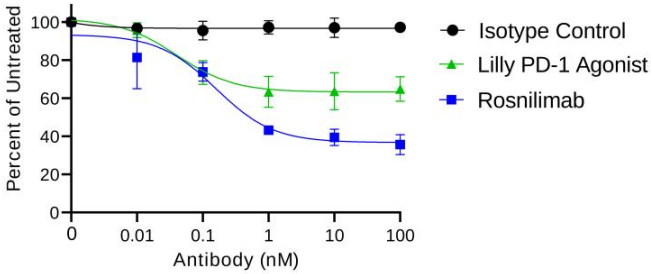
Rosnilimab Demonstrates Superior Agonistic Signaling and Depletion of PD-1+ T Cells at Clinically Relevant Concentrations

Agonistic Signaling
(Inhibition of T cell proliferation)



Anti-CD3+ anti-CD28 stimulation of PBMCs for proliferation assessment of T cells, representative data from N=4 donors

Depletion
(NK cell-mediated ADCC of PD-1+ T cells)

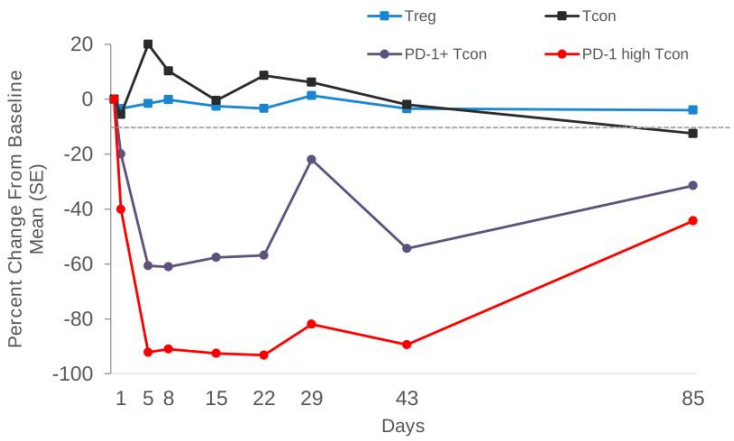


Healthy donor T cells + NK cells (1:5 ratio) + rosnilimab in in-vitro ADCC assay, representative data from N=2 donors

Complementary mechanisms of action enable potent reduction of PD-1+ T cells to restore immune balance

Rosnilimab Healthy Volunteer Phase 1 Trial Results

Potent and sustained reduction in peripheral PD-1+ T cells for >30 days in SAD



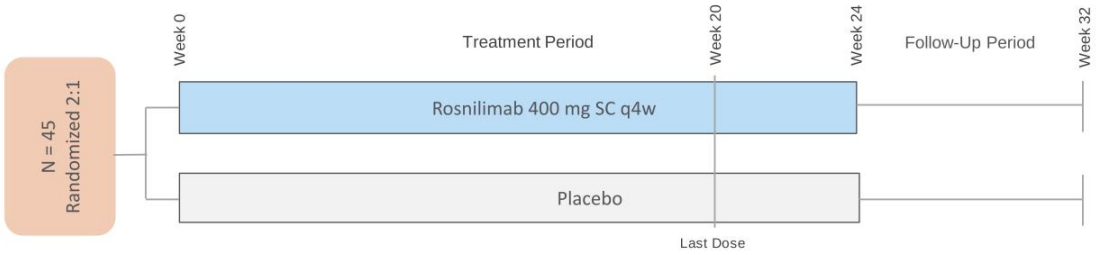
- PD-1+ T cell effect
  - >50% reduction of PD-1+ Tcon
  - >90% reduction of PD-1 high Tcon
- PD-1 negative Tcon effect
  - Minimal/no effect
- Treg effect
  - Minimal /no effect on overall Tregs
  - Positive bias to Treg/Tcon ratio

Favorable safety, pharmacokinetics and pharmacodynamic activity

- ✓ Well tolerated with no dose-limiting toxicities
- ✓ Favorable PK with ~2 week half-life with IV or SC dosing
- ✓ Full RO observed quickly and maintained for 30 days with a single dose

*Results shown from 400mg subcutaneous dose (SAD)

Rosnilimab "Proof of Mechanism" Phase 2a Trial in Moderate-to-Severe Alopecia Areata



"Proof of Mechanism" P2a Blinded Pooled Interim Data	
Peripheral T cell effect • Impact on activated T cells and Tcon / Treg ratio in blood and implications for broader inflammatory diseases through week 32	✓ Robust reductions in peripheral PD-1+ T cells observed in AA patients consistent with HV P1 trial
Change in SALT score (primary endpoint) • Commercially relevant in absolute SALT <20 (secondary endpoint)	X Modest improvement in some patients observed through week 24+; No patients with absolute SALT<20
Durability • Maintenance of change in SALT score through week 32	• TBD; patients with SALT improvement observed were stable off treatment through weeks 28 - 32
Safety and tolerability	✓ Generally safe and well tolerated

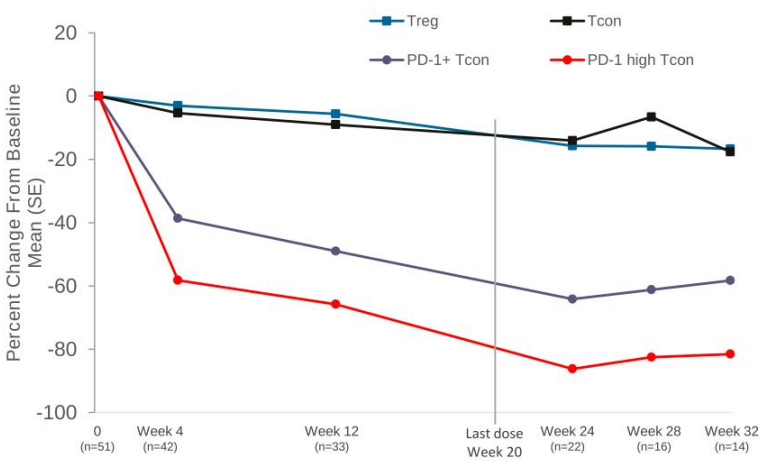
Additional P2a Data Available H2 23

- Tissue Biopsies
  - Impact on reduction of PD-1+ T cells and cytokines in hair follicle (inflamed tissue)
- Circulating Biomarkers & T Cell Phenotyping
  - Impact on reduction of PD-1+ T cells, including Tregs and Tcons, and cytokines in periphery

Rosnilimab P2a "Proof-of-Mechanism" Blinded Data

Potent and sustained reduction in peripheral PD-1+ T cells in alopecia patients consistent with HV P1 trial results

Blinded (pooled treated + placebo 2:1 randomization) Immunophenotyping Data



- PD-1+ T cell effect
  - >60% reduction of PD-1+ Tcon
  - >80% reduction of PD-1 high Tcon
- PD-1 negative Tcon effect
  - Minimal/no effect
- Treg effect
  - Minimal /no effect on overall Treg
  - Positive bias to Treg/Tcon ratio

PD-1+ T cell impact in the periphery supports proof of mechanism to advance rosnilimab into additional indications, such as rheumatoid arthritis

*Results shown at 400mg subcutaneous dose Q4W

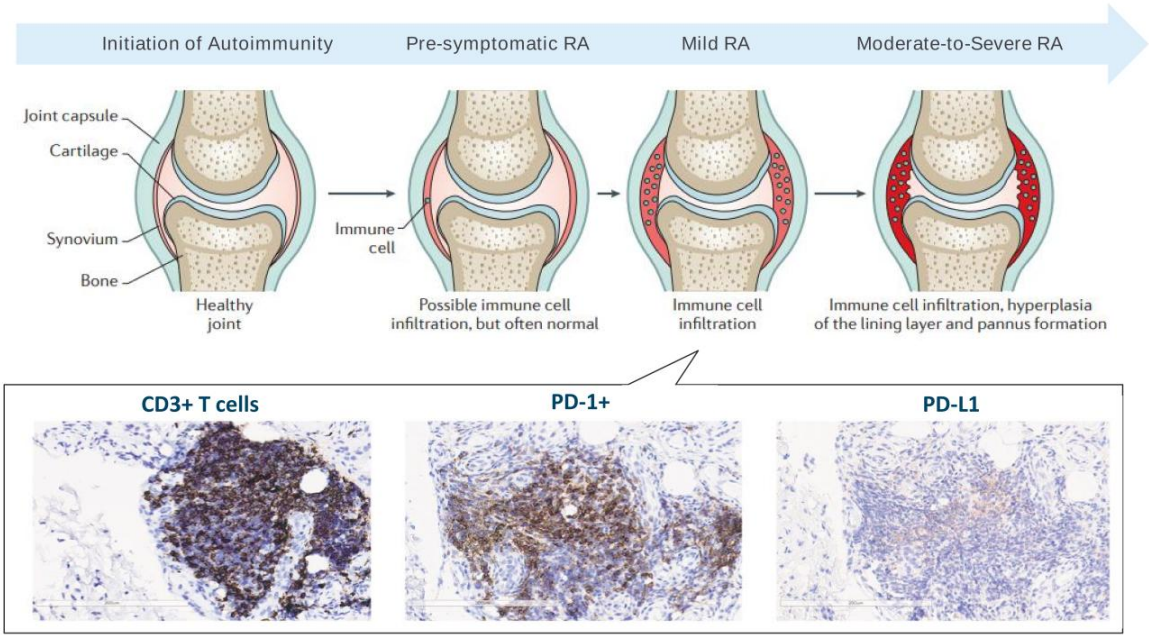
AnaptysBio

Rosnilimab (PD-1 agonist mAb)

Rheumatoid Arthritis (RA)

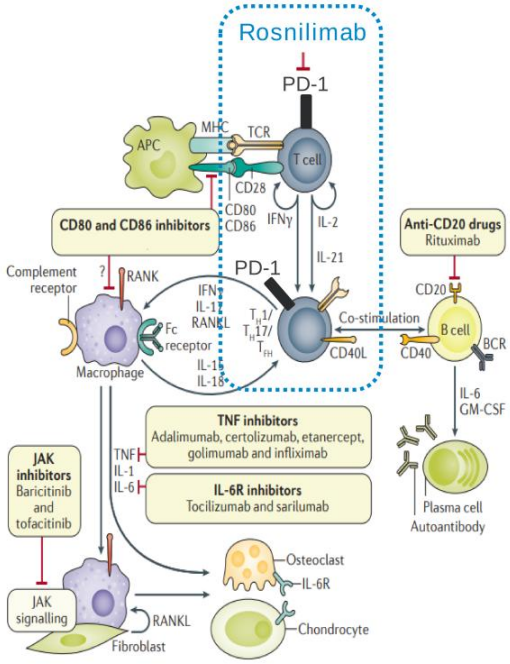
PD-1+ T Cells are Clinically Validated Drivers of Disease in RA

>80% of T cells in treatment-naive RA joints are PD-1+ and not down-regulated due to lack of sufficient PD-L1 expression



*Adapted from Nature Reviews Disease Primers volume 4, Article number: 18001 (2018) and PLoS One 2018; 13(2): e0192704

PD-1 Agonists are a Promising Novel Mechanism for the Treatment of RA

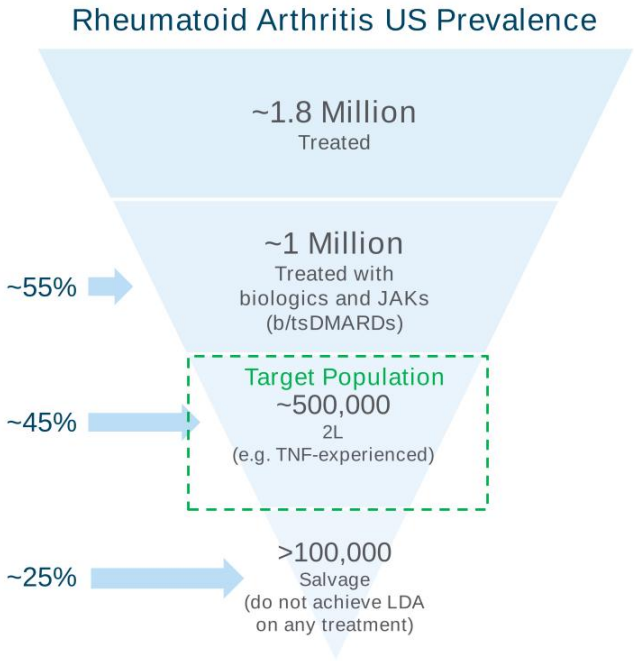


- b/tsDMARDs provide benefit by inhibiting either T cells, B cells or downstream cytokines
- However, most patients cycle through classes of therapy
  - Many patients cannot achieve LDA
  - If LDA achieved, responses often not durable
- PD-1 agonists target multiple distinct inflammatory mechanisms addressed by approved therapies to treat RA
  - Inhibit PD-1+ T cell proliferation and cytokine secretion
  - Deplete PD-1+ T cells, including impact on downstream B cells
  - Prevent migration of pathogenic PD-1+ T cells to joints

*Adapted from Nature Reviews Disease Primers volume 4, Article number: 18001 (2018), **b/tsDMARD – biologic/targeted synthetic disease modifying antirheumatic drug, LDA – low disease activity

RA is a Substantial Opportunity for a New Class of Biologics

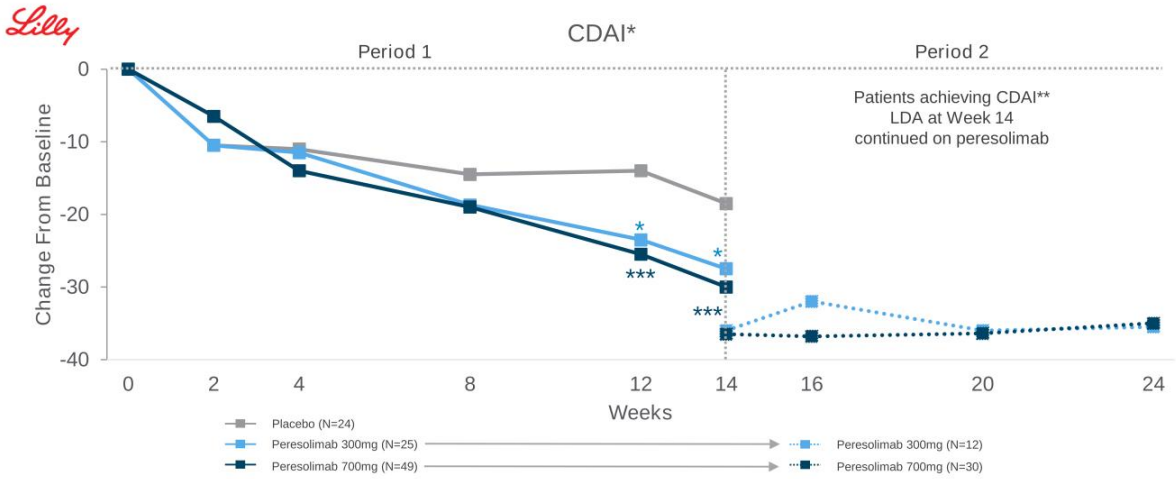
~500k patients in the US seek treatment after progressing on anti-TNF therapy



- Target population in US generated ~$10 billion in 2021
  - Rituxan (typically salvage therapy) achieves well over $1 billion sales in 3L+ RA despite infection risk
- No new therapeutic class has launched for RA since JAK inhibitors (Xeljanz) a decade ago (2012)
- Lack of established SOC in 2L+ provides opportunity for new class to penetrate
  - Comparable or differentiated efficacy
  - Durable responses
  - Treatment of salvage population

*Claims analysis to determine market size based on 5 years of claims history, ** Evaluate Pharma,***b/tsDMARD – biologic/targeted synthetic disease modifying antirheumatic drug, 2L – 2nd line

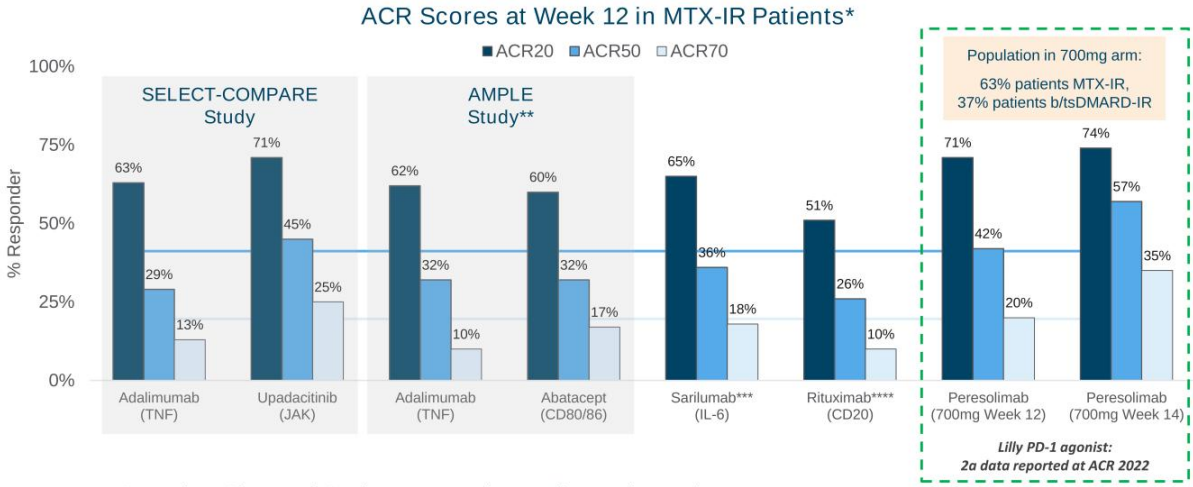
Lilly's PD-1 Agonist Showed Proof of Mechanism and Promising Efficacy in a Phase 2a RA Study

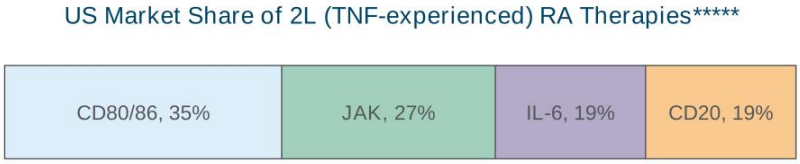


- 48% and 61% of patients achieved LDA at week 12 for 300 and 700 mg Q4Q IV doses
- Patients achieving LDA at week 12 continued treatment through week 24
  - At either dose, patient benefit was sustained
- Similar responses observed regardless of whether a patient was naïve to or had received a prior b/tsDMARD

*Week 0 though Week 24 data estimated from peresolimab (PD-1 agonist) 2022 ACR presentation,**CDAI – clinical disease activity index, LDA – low disease activity

PD-1 Agonists have the Potential to Restore Immune Balance and Differentiate from Available Treatments on ACR50/70





Fragmented market

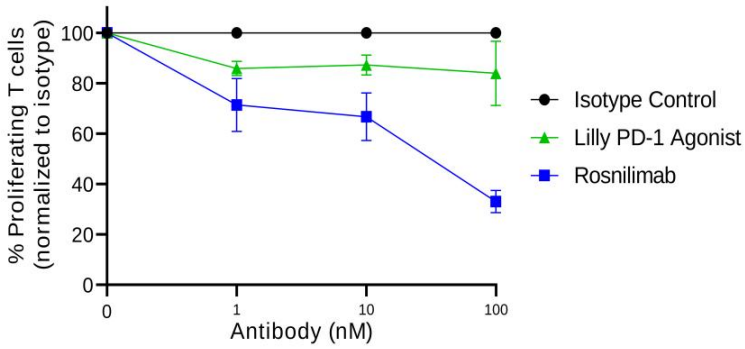
No clear treatment of choice after failure of anti-TNFs

*Patients on background MTX, **12 week data based on ACR time graph, unless otherwise noted. ***Phase 3 study versus placebo in MTX-IR patients, **** Phase 3 study versus placebo in MTX-IR patients (SERENE) at week 24, *****5 year claim history analysis

Rosnilimab Demonstrates Superior Inhibition of T Cell Proliferation in Rheumatoid Arthritis Patient-Derived PBMCs

- More potent reduction compared to Lilly PD-1 agonist is consistent between healthy PBMCs and RA patient-derived PBMCs

Inhibition of T Cell Proliferation (in RA Patient-Derived PBMCs)



Anti-CD3+ anti-CD28 stimulation of PBMCs for proliferation assessment of T cells, representative data from N=3 donors

Rosnilimab P2b Trial in Moderate-to-Severe Rheumatoid Arthritis

Initiation Q3 2023; Topline Interim Data Mid 2025

P2b Trial Objectives

- Explore broad exposure response and establish efficacious dose
- Enable initiation of a registrational phase 3 campaign
- Analyze potential predictive baseline characteristics including patient prior therapy and biomarkers

Patient Population and Endpoints

- Global, multi-hundred patient placebo-controlled trial
- Inclusive broad population of cDMARD and b/tsDMARD-experienced patients
- Standard efficacy endpoints including ACR20/50/70, DAS28 and CDAI

Dosing and Administration

- Three dose levels of rosnilimab
  - High dose is multiple fold higher than used in alopecia areata Phase 2a study
- Subcutaneous dosing to support patient self-administration
- Rosnilimab dosing duration of ~6 months

Timing and Trial Initiation

- Phase 2b trial initiation in Q3 2023
- Topline data anticipated in mid 2025

*cDMARD – conventional disease modifying antirheumatic drug, CDAI – clinical disease activity index, DAS28 – disease activity score

AnaptysBio

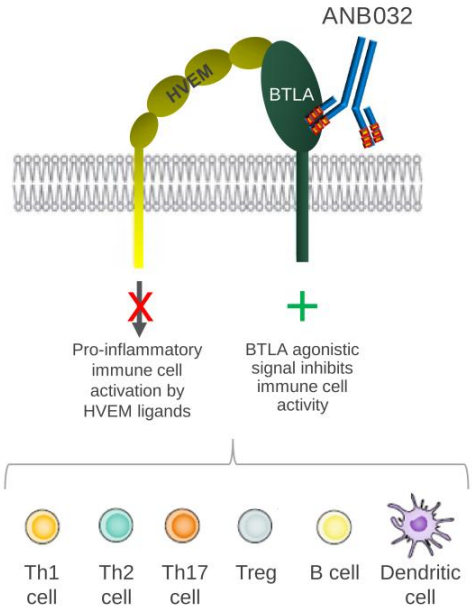
ANB032 (BTLA agonist mAb)

Introduction

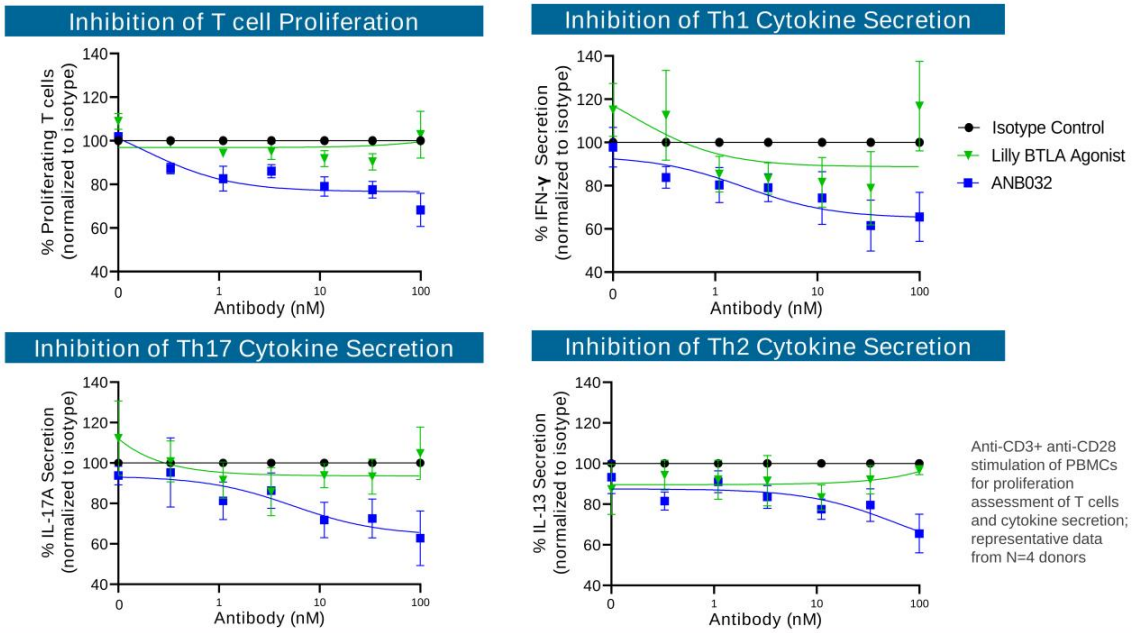
ANB032: Best-in-Class BTLA Agonist Antibody

Suppression of T and B cell mediated inflammatory diseases

- ANB032: IgG4 antibody that binds to BTLA in lymphoid (T and B) cells and dendritic cells
  - Fc receptor binding profile together with membrane proximal binding epitope contributes to differentiated potency
- ANB032 inhibits T cell proliferation and inflammatory cytokine secretion
- ANB032 demonstrated robust in vivo efficacy in animal models of GVHD
- Potential broad applicability to inflammatory disease due to breadth of BTLA expression across immune cell types



ANB032 Demonstrates Superior Agonistic Signaling in Healthy PBMCs Compared to Lilly BTLA Agonist



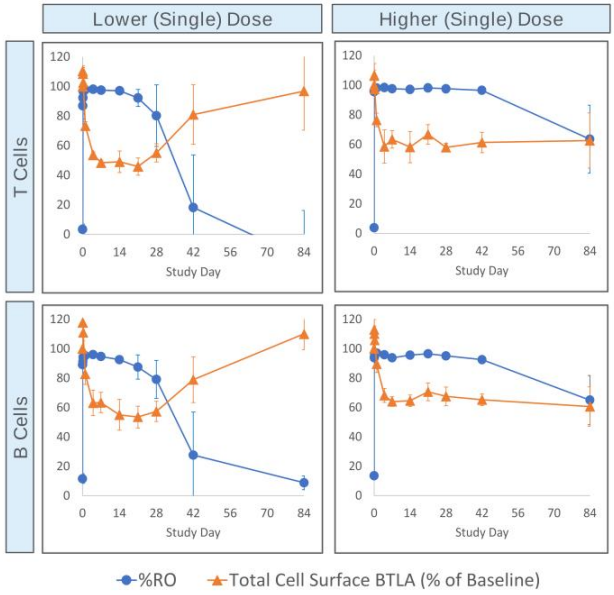
Anti-CD3+ anti-CD28 stimulation of PBMCs for proliferation assessment of T cells and cytokine secretion; representative data from N=4 donors

ANB032 shows meaningful T cell inhibition beginning at low concentrations and potency differentiation is sustained across clinical concentrations

ANB032 Phase 1 Healthy Volunteer Trial Top-Line Data

Favorable safety, pharmacokinetics and pharmacodynamic activity

- 96 healthy volunteers in randomized, double-blind, placebo-controlled SAD and MAD cohorts
- ANB032 well-tolerated with no dose limiting tox
  - Most AEs mild-to-moderate, of short duration, dose independent and resolved without sequelae
  - No Serious Adverse Events
- Favorable PK: ~2-week half-life with IV and SQ dosing
- Full receptor occupancy (RO) within hours and maintained for >30 days
- Pharmacodynamic analyses:
  - Rapid and sustained target engagement on both T cells and B cells
  - Reduction of cell surface BTLA expression
  - Duration of reduced BTLA expression persisted in a dose-dependent manner



ANB032 in humans mirrored observation in animal models of inflammation supporting potential broad treatment of T and B-cell driven inflammatory diseases

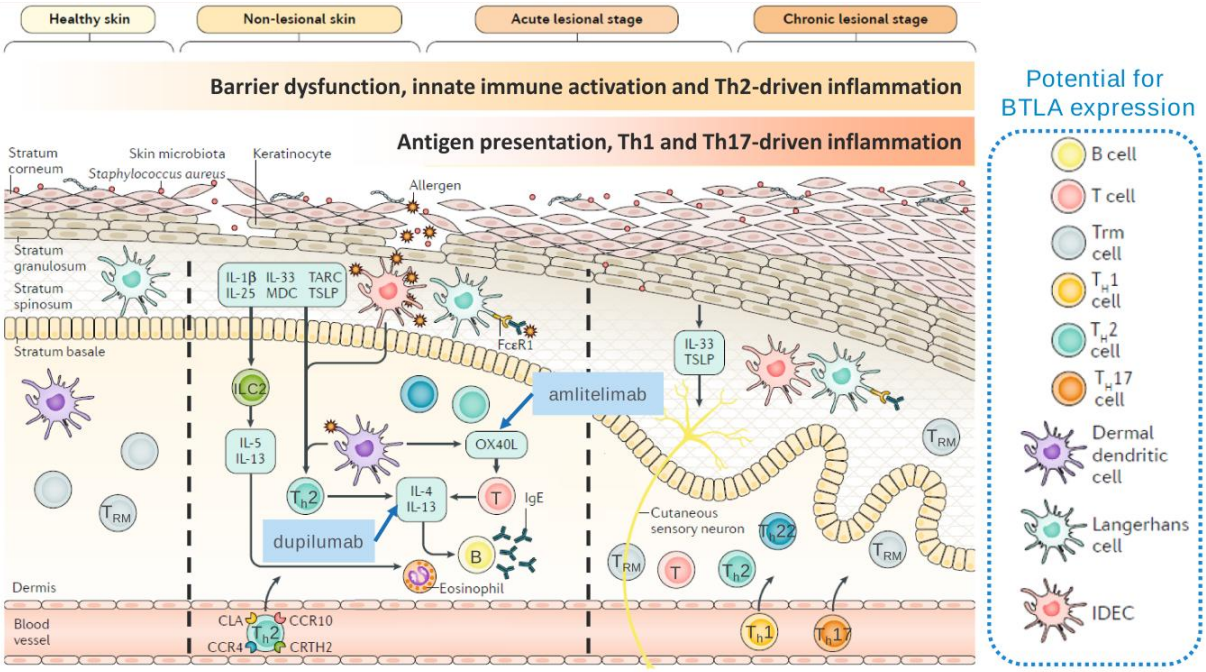
AnaptysBio

ANB032 (BTLA agonist mAb)

Atopic Dermatitis Phase 2b

Atopic Dermatitis (AD) is Broader than a Th2 Inflammatory Disease

Th2 targeting SOC provides benefit; however, other cell types including Th1, Th17 and dendritic cells may contribute significantly to AD pathogenesis

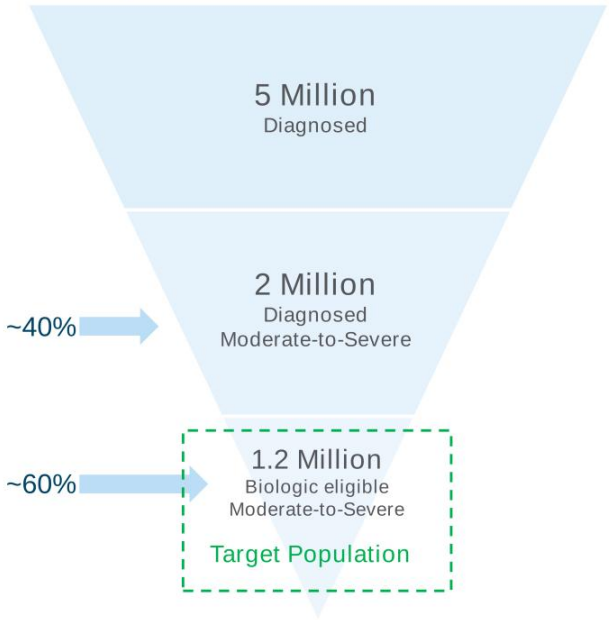


Adapted from Nature Reviews Disease Primers volume 4, Article number: 1 (2018)

AD is a Substantial Opportunity for a New Class of Biologics

~1.2m patients in the US are biologic eligible

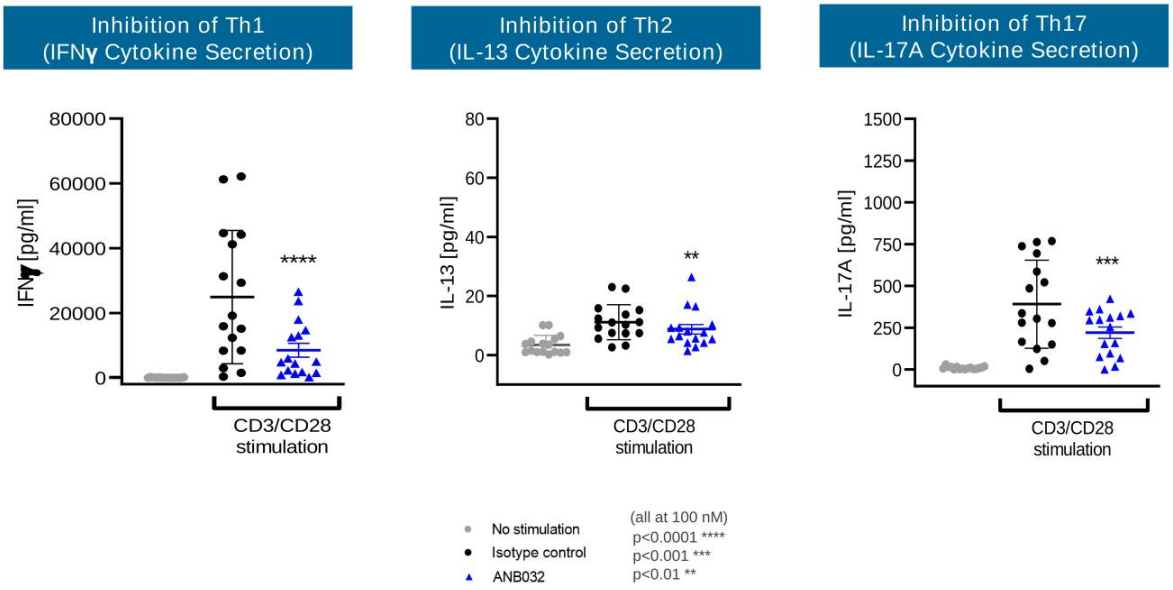
Atopic Dermatitis US Prevalence*



- Dupixent is standard of care in moderate-to-severe AD
  - >$7 billion projected global sales (2027)**
- Significant room to differentiate vs. SOC
  - Dupixent EASI75 of ~45%-50% leaves substantial opportunity for broader response, including suboptimal responding patients
  - Dupixent EASI90 of ~35% leaves substantial opportunity for deeper response
  - Potential for durable response
  - Black box warning is an impediment to use of oral JAKs

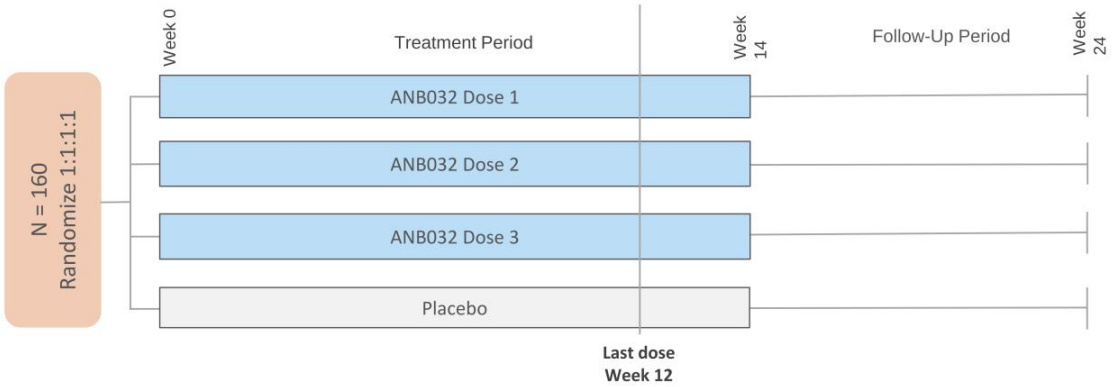
*Claims analysis to determine market size based on 5 years of claims history, ** Evaluate Pharma.

ANB032 Inhibits Th1/Th2/Th17 Cytokine Secretion in Atopic Dermatitis Patient-derived PBMCs



ANB032 P2b Trial in Moderate-to-Severe Atopic Dermatitis

Initiation Q2 2023; Topline data YE 2024



Patient Population	• Adult patients with moderate to severe atopic dermatitis • All comer population; includes patients with both Dupixent naïve and Dupixent experience
Key Endpoints	• Primary: Mean change from Baseline in EASI at Week 14 • Secondary: EASI75, IGA 0/1, PNRS, DLQI, SCORAD50, Safety • Exploratory: Th1/Th2/Th17 biomarkers, tissue biopsy

AnaptysBio

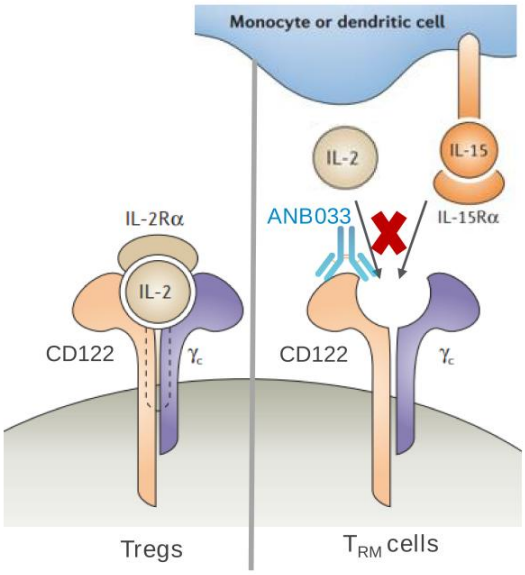
ANB033 (anti-CD122 antagonist mAb)

Autoimmune and Inflammatory Diseases

ANB033: Anti-CD122 Antagonist mAb Targets Pathogenic Tissue Resident Memory T Cells (TRM)

IND filing targeted H1 2024

CD122 is a shared beta subunit of the receptors for IL-15 and IL-2



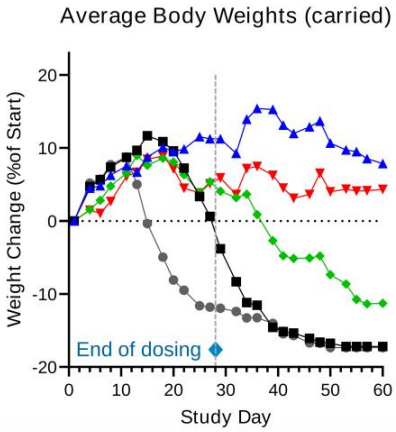
ANB033 MoA: Induce death of T_{RM} cells to achieve and maintain remission

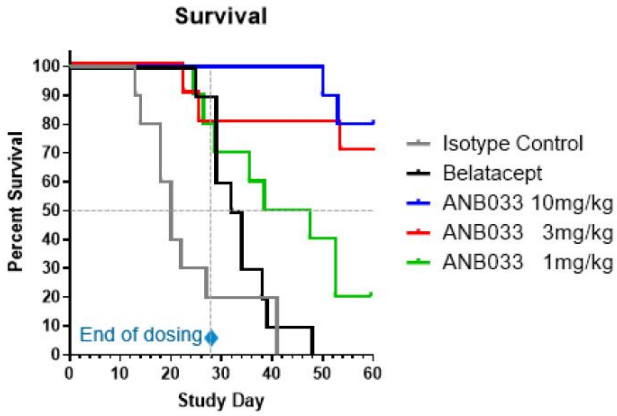
- IL-15 mediates survival of T_{RM} cells
- Both IL-15 and IL-2 mediate:
  - Proliferation of T cells
  - Inflammatory cytokine secretion (IFNγ) during T cell activation
- High affinity antagonist antibody induces death in T_{RM} cells by preferentially inhibiting the lower affinity dimeric receptor complex
  - Spares Tregs which express higher affinity IL-2 trimeric receptor complex
- Targeted elimination of T_{RM} cells may potentially drive durable responses

ANB033: Durable Survival in GVHD Model

All mice treated at high-dose survived well beyond end of dosing

- GVHD (severe phenotype) model in human IL-15 transgenic mouse supports T cell and NK cell survival
- ANB033 preclinical data suggests targeted elimination of pathogenic T_{RM} drives more potent and durable response than belatacept
  - Belatacept (GVHD standard-of-care which only impedes T cell activation) shows minimal benefit over control





GVHD model is biologically relevant to CD122 antagonist MoA with translation to inflammatory diseases driven by pathogenic TRM and Treg imbalance including rheumatology, dermatology, gastroenterology, and respiratory

Note: ANB033 treated mice dosed twice per week through Day 28

AnaptysBio

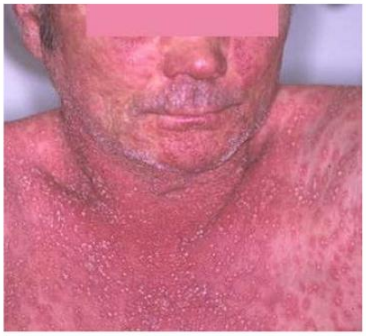
Legacy Programs for Outlicense

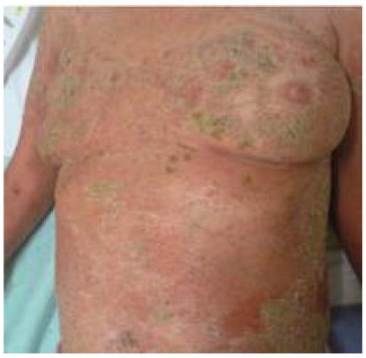
Imsidolimab (IL-36r antagonist mAb)
Etokimab (IL-33 antagonist mAb)

Generalized Pustular Psoriasis (GPP)

Systemic inflammatory disease where IL-36 pathway plays key role in pathology

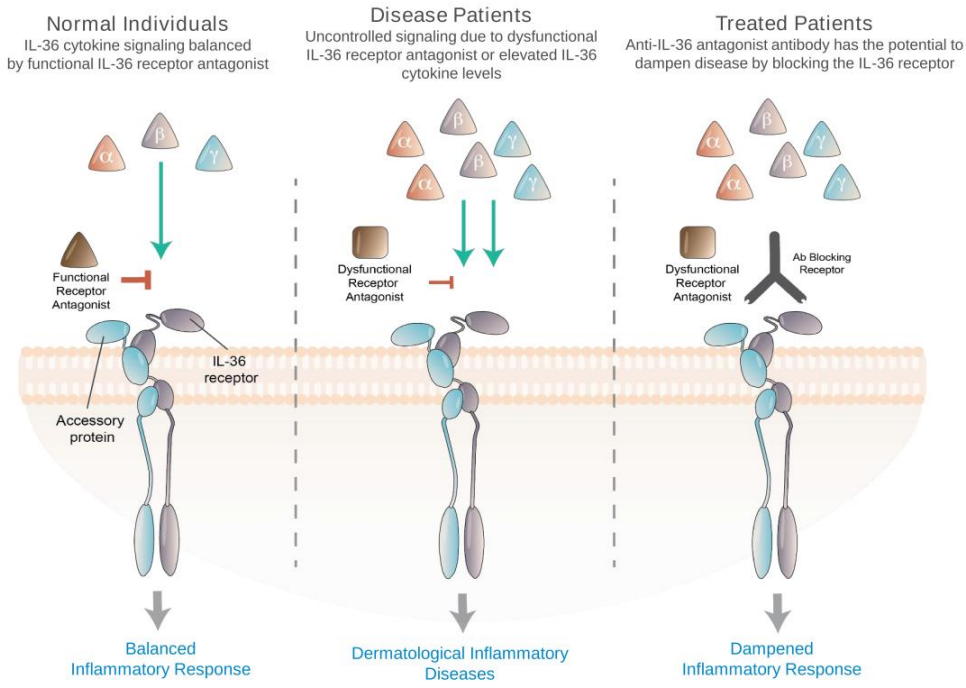
- GPP is a systemic, life-threatening inflammatory disease characterized by widespread pustules
  - Associated with unregulated IL-36 signaling
  - Patients have a high fever and elevated levels of serum CRP and inflammatory cytokines (e.g., IL-8)
  - Severe GPP patients can die from cardio-pulmonary failure, exhaustion, toxicity and infection
- GPP ICD-10 diagnostic code analysis by IQVIA assessed US prevalence during 2017-2019 timeframe
  - ~37,000 unique patients diagnosed at least once
  - ~15,000 unique patients diagnosed two or more times
- FDA has granted Orphan Drug Designation to imsidolimab for the treatment of GPP
- Worldwide registry (RADIANCE) of GPP patients ongoing
  - Increase understanding of patient journey and support enrollment of Phase 3 trial





Imsidolimab: an Anti-IL-36r Antagonist Antibody

Uncontrolled-IL-36 signaling, as well as a genetic IL-36 association, mediates GPP

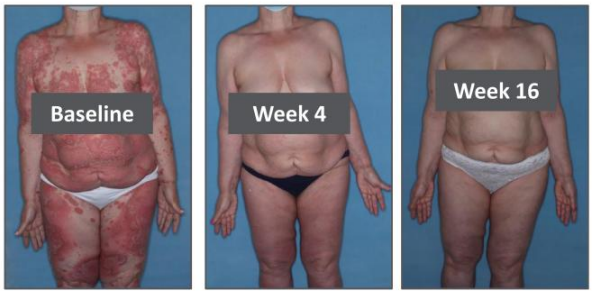


GALLOP: Imsidolimab GPP Phase 2 Trial Top-Line Data

Presented at EADV Congress October 2021

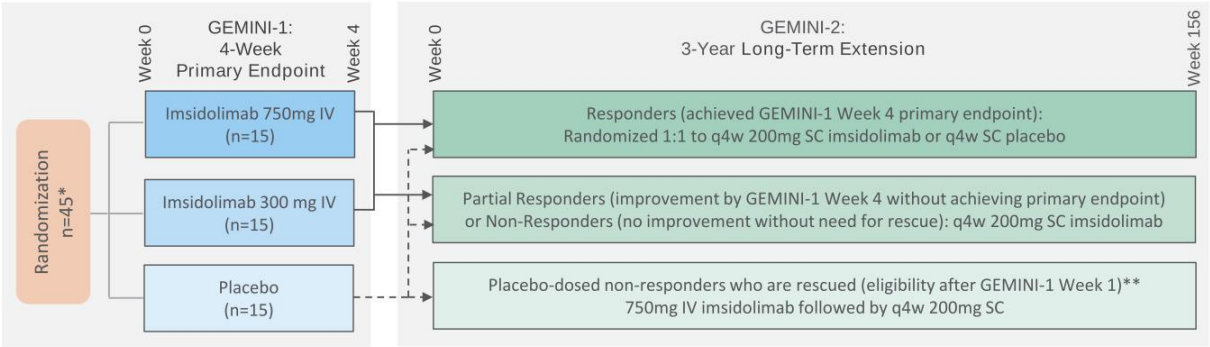
- Imsidolimab 750mg IV loading dose + 100mg SC q4w maintenance dose
- Rapid and sustained efficacy through week 16
  - 6 of 8 (75%) patients achieved primary endpoint of improvement in the clinical global impression scale (CGI)
  - Early reduction of erythema with skin pustules by week 1; sustained relative to baseline at week 16
  - GPP Physician Global Assessment (GPPGPA) demonstrated sustained efficacy through week 16
  - 2 patients dropped out of the study before Day 29 and deemed non-responders
- Imsidolimab generally well-tolerated
  - Most adverse events were mild to moderate and resolved without sequelae
  - Two subjects had serious adverse events and recovered without sequelae (sepsis due to S. aureus infection and SARS-CoV-2 injection)
- Genotypic testing indicated homozygous wild-type IL-36RN, CARD14 and AP1S3 alleles for all tested patients
  - IL-36R inhibition may be efficacious in GPP irrespective of genetic mutations

Endpoint	Baseline	Week 1 Relative to Baseline	Week 4 Relative to Baseline	Week 16 Relative to Baseline
CGI improvement **(primary endpoint)**	N/A	7 of 8 patients	6 of 8 patients	6 of 8 patients
mJDA-SI	9	-29%	-54%	-58%
Erythema with pustules (% body surface area)	24%	-60%	-94%	-98%
DLQI (MCID of -4)	16	-1	-6	-11



GEMINI-1 & 2: Imsidolimab GPP Phase 3 Trials

GEMINI-1 top-line data anticipated Q4 2023



Patient Population	• Male and female subjects 18 to 80 years of age • Clinically confirmed diagnosis of GPP as per ERASPEN definition • Baseline Generalized Pustular Psoriasis Physician Global Assessment (GPPPGA) score of at least moderate severity (3 and higher) • Active flare with pustules and erythema accounting for at least 5% of body surface area at baseline
Key Endpoints	• Primary: GPPPGA score of clear (0) or almost clear (1) at GEMINI-1 Week 4 • Key Secondary: Pustulation Rating Scale (PRS) of 0 or 1 at GEMINI-1 Week 1 • Other: Time to flare recurrence, proportion of subjects in remission, DLQI, safety

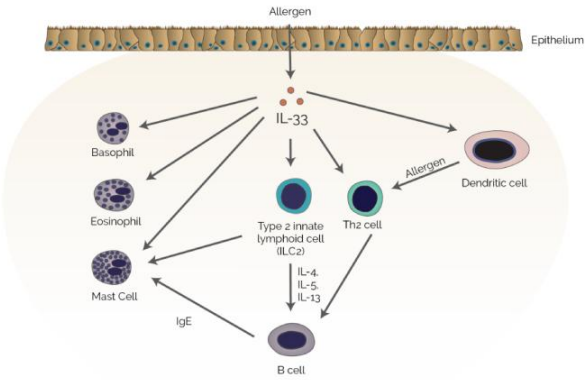
ClinicalTrials.gov: NCT05352893, NCT05366855

* 80% power calculated for GEMINI-1 using two-sized test alpha of 0.05 assuming ~40% effect size with 45 patient sample size
**Starting at week 1 in GEMINI-1, placebo patients who have not improved or are worsening are eligible to be rescued with imsidolimab

Etokimab: Phase 2b/3-Ready Anti-IL-33 Antagonist Antibody

IL-33 biology applicable to respiratory disorders

- Etokimab: IgG1 antibody that inhibits the activity of the active form of IL-33
- IL-33 is genetically associated with asthma
  - IL-33 loss-of-function mutations protect against asthma, while gain-of-function mutations increase asthma incidence
  - Translational studies have demonstrated IL-33's role as a pro-inflammatory cytokine released upon allergen contact with epithelium
- Broad commercial opportunity in respiratory and allergic conditions
  - ~1.1 million adults diagnosed in U.S. with severe asthma
  - ~550,000 million adults diagnosed in U.S. with Type 2 or Non-Type 2 COPD are inadequately controlled by standard of care
- IL-33 pathway derisked in COPD
  (positive Phase 2 data via AZ and REGN/SA)



- **IL-33 is active in its reduced form and is quickly oxidized into an inactive form as a mechanism to limit its local activity**
- **The majority of IL-33 in the body is the inactive oxidized form**

Given etokimab's MOA, it specifically inhibits only the IL-33 molecules that are driving activity and not "wasted" by binding to non-active oxidized IL-33

Etokimab is P2b/3 Ready
(drug supply on hand, preclinical toxicology, P2 data, and competitor POC data across respiratory diseases)

AnaptysBio

GSK Immuno-Oncology Financial Collaboration

JEMPERLI™ (dostarlimab, anti-PD-1 Antagonist)
Cobolimab (GSK4069889, anti-TIM-3 Antagonist)
GSK4074386 (TSR-033, anti-LAG-3 Antagonist)

Significant Potential Royalties from GSK Immuno-Oncology Financial Collaboration

JEMPERLI
(anti-PD-1 antagonist)

- JEMPERLI 8% royalties on annual net sales <$1B
- JEMPERLI $15mm regulatory and $90MM commercial milestones on annual net sales <$1B

GSK anticipates ~$1.2-2.4B peak annual sales for JEMPERLI in currently approved indications and anticipated 1L endometrial/ovarian approvals*

Sagard: JEMPERLI Capped Non-Recourse Monetization

In 2H21, ANAB received $250mm upfront in **exchange for only the above receivables until** Sagard is paid back one of the following capped returns: :

- $312.5MM (125% of upfront) by end 2026 or
- $337.5MM (135% of upfront) by end 2027 or
- $412.5MM (165% of upfront) anytime after 2027

Receivables Excluded from Sagard Monetization

- JEMPERLI 12-25% royalties on annual net sales ≥ $1B
- JEMPERLI $75mm commercial milestone on annual net sales ≥ $1B

Cobolimab
(anti-TIM-3 antagonist)

GSK4074386
(anti-LAG-3 antagonist)

- Both programs being developed in combination with JEMPERLI
- 4-8% royalties on annual net sales on each program
- $5mm clinical development, $90mm regulatory and $165mm commercial milestones on each program**

* In June 2021, GSK estimated potential peak annual global JEMPERLI sales on a non-risk adjusted basis of £1-£2 billion

** $10mm clinical development milestones remaining for GSK4074386

Note: Sale of ZEJULA (niraparib) royalty interest in September 2022 to wholly-owned subsidiary of DRI Healthcare Trust for $35mm upfront + $10mm potential milestone upon FDA approval of ZEJULA for the treatment of endometrial cancer, for which the drug is currently in a fully-enrolled ongoing RUBY Phase 3 study of dostarlimab + niraparib, to the extent that such approval occurs on or before 12/31/25.

GSK Immuno-Oncology Financial Collaboration

JEMPERLI (dostarlimab) potential first-in-class PD-1 antagonist in first line endometrial and ovarian cancer

Jemperli (dostarlimab-gxly) Injection 500 mg

(Anti-PD-1 antagonist)

Cobolimab

(Anti-TIM-3 antagonist)

GSK4074386

(Anti-LAG-3 antagonist)

Dostarlimab: 1L Endometrial Cancer

- Approved in US and EU for dMMR recurrent or advanced endometrial cancer after progressing a platinum-containing regimen
- P3 RUBY trial met primary endpoint in 1L primary advanced or recurrent endometrial cancer (Dec. 2022)
  - Regulatory submissions planned 1H 2023
  - Significant market opportunity for 30% of the 65,000 new endometrial diagnoses per year in the US*

Dostarlimab: 1L Ovarian Cancer

- P3 FIRST trial (combination of dostarlimab + niraparib) in 1L ovarian cancer
  - P3 data (interim analysis) 2H 2023
  - Significant market opportunity with ~20,000 new ovarian diagnoses per year in US*

Dostarlimab and cobolimab: NSCLC

- P3 COSTAR trial (combination of dostarlimab + cobolimab) in 2L NSCLC patients have progressed on prior therapy
  - P3 initiated Oct. 2022
  - Significant market opportunity with 237,000 new NSCLC diagnoses per year in the US*
- PERLA, H2H trial in 1L NSCLC: cORR of 46% for dostarlimab + chemo vs 37% in pembrolizumab + chemo

Additional Dostarlimab Royalty Opportunities

- Dostarlimab combination with anti-TIGIT (EOS-448/GSK'859A) in P2 for 1L NSCLC and H&NSCC
- Dostarlimab + GDK4074386 in CITRINO trial across multiple solid tumors
- Additional P1/2 dostarlimab combinations include: anti-CD96, STING agonist, and PVRIG across multiple solid tumors

*NCI SEER Data


AnaptysBio