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: October 14, 2025
(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 7.01. Regulation FD.

On October 14, 2025, AnaptysBio, Inc. ("AnaptysBio") will host a conference call to discuss ANB033 and use a slide presentation in conjunction with the call. A copy of the presentation is filed herewith as Exhibit 99.1.

On October 14, 2025, 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 Exhibits 99.1 and 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 the 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	AnaptysBio, Inc. Investor Presentation, dated October 14, 2025.
99.2	Anaptys Corporate Presentation October 2025.
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: October 14, 2025

By: /s/ Dennis Mulroy

Name: Dennis Mulroy

Title: Chief Financial Officer


ANB033
(CD122 antagonist)
Investor Event
Oct. 14, 2025
AnaptysBio

Forward looking 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 the release of data from the Company's clinical trials, including rosnilimab's Phase 2 clinical trial in ulcerative colitis and ANB033's Phase 1b clinical trial in celiac disease; the timing of initiation of ANB033's Phase 1b clinical trial in a second indication; expectations regarding the structure, infrastructure, timing and taxation of the proposed separation of companies; whether any of the Company's product candidates will be best in class or optimized; the potential to receive any additional milestones or royalties from the GSK collaboration and timing therefor; the potential to receive any royalties or milestone payments from the Vanda Pharmaceuticals license agreement; 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.

Intention to separate into two independent, publicly traded companies to unlock and maximize value

Biopharma Co

Focus on developing and potentially commercializing therapeutics for autoimmune diseases

Rosnilimab
(Pathogenic T cell depleter)

P2b complete in Rheumatoid Arthritis

P2 in Ulcerative Colitis

ANB033
(CD122 antagonist)

P1b in Celiac Disease (CeD)

ANB101
(BDCA2 modulator)

P1 in Healthy Volunteers

Research-driven • R&D capabilities with preclinical pipeline of immunology targets

Royalty Management Co

Focus on protecting and returning value of the royalties to shareholders

- Hold and continue to manage rights to
  - Potential substantial *Jemperli* royalties from GSK
  - Imsidolimab milestones and royalties from Vanda
- Expect minimal infrastructure and staff
- Anticipate will retain Anaptys' net operating loss (NOL) carryforwards

GSK Jemperli (dostarlimab-gxly) injection 500 mg

VANDA PHARMACEUTICALS INC

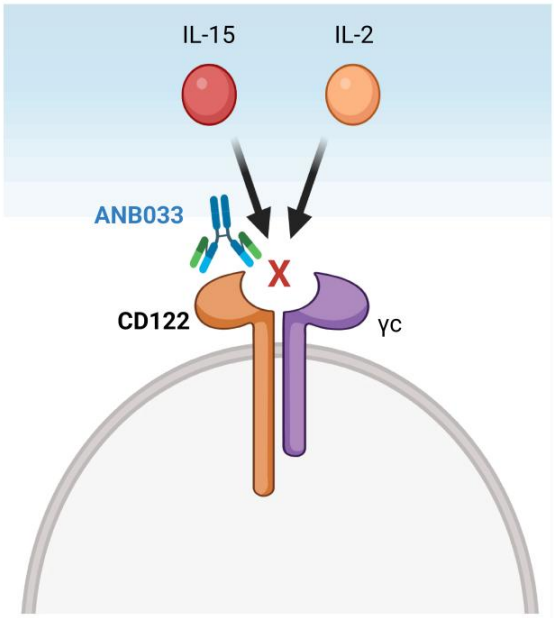
Note: Q2 2025 cash: ~$294MM. Anaptys expected cash runway through YE: 2027 includes GSK $75MM milestone for *Jemperli* $1B annual WW sales. Biopharma Co. to launch with adequate capital to fund operations for at least two years through significant potential corporate milestones. Cash runway excludes significant royalty potential from GSK or Vanda.

Targeting CD122 has strong biologic rationale

Why target CD122?

- Targeting only the IL-15 cytokine addresses inflammation, but may allow pathogenic cell survival through alternative escape mechanisms
- CD122 is the shared receptor subunit through which both IL-15 and IL-2 signal
- IL-15 and IL-2 are central cytokines in pathogenic inflammation across broad inflammatory diseases
  - Overactive signaling drives proliferation and survival of cytotoxic CD8+ and CD4+ T cell subsets and NK cells
  - Inflammatory Th1 and Th2 cytokine secretion during T cell activation

ANB033: potential best-in-class CD122 antagonist with optimized dual IL-15 and IL-2 signaling inhibition



ANB033 designed for high potency

- Specific binding epitope engineered for high affinity
- Inhibit both IL-15 and IL-2 signaling

Targets pathogenic cells in inflamed tissue

- Subsets of activated T cells
  - Cytotoxic CD8+ T cells
  - Memory CD4+ Th1/Th2 T cells
  - IELs
- ILC2s
- NK cells

Subcutaneous dosing

Broad therapeutic potential across autoimmune and inflammatory diseases

Gastroenterology	***Dermatology***	**Other Areas**
Celiac Disease	Atopic Dermatitis	Asthma
Crohn's Disease	Alopecia Areata	Multiple Sclerosis
Eosinophilic Esophagitis (EOE)	Hidradenitis Suppurativa	Psoriatic Arthritis
Ulcerative Colitis	Lichen Planus	Type 1 Diabetes
	Vitiligo	Solid Organ Transplant

Clinical-stage drugs targeting IL-15 or CD122

NOVARTIS	IL-15	• P1b PoC in CeD • P1b PoC in EoE
teva	IL-15	• P2a in CeD • P2a in vitiligo
Incyte	CD122	• P1b in vitiligo
FORTE	CD122	• Positive P1b in CeD (P2a ongoing) • P1b in vitiligo and alopecia areata • Assessing T1D

ANB033 has pipeline-in-a-product potential

ANB033: initiated initial P1b in CeD

- Multiple pathogenic drivers of disease
- Inflamed cells in CeD respond to both IL-15 and IL-2
- Targeting IL-15 / CD122 is clinically validated

Expansion opportunities

- GI, dermatology and other therapeutic areas
- Assessing potential to treat EoE

CeD Market[1]

- >250k diagnosed U.S. non-responsive CeD patients
- Gluten-free diet highly restrictive; ~50% of patients suffer anemia and fatigue
- No approved therapies
- $4 – 5bn U.S. market for CeD patients non-responsive to gluten-free diet
- Global pharma interest

1. Trinity Life Sciences Commercial Assessment HCP Primary Market Research (2025). CeD sizing reflects future US market in 2030 assuming growth in diagnosis rate based on historic trends and projected growth with entrance of novel therapies.

Agenda: ANB033 (CD122 Antagonist)

TOPIC	SPEAKER
CD122 biology and preclinical data	Martin Dahl, Ph.D., Senior Vice President, Research
Phase 1a in healthy volunteers	John Kwon, M.D., Ph.D. Vice President, Clinical Development
Drug development for CeD	Joseph Murray, M.D. Professor of Medicine Mayo Clinic College of Medicine, Rochester, MN
Phase 1b in CeD	Paul Lizzul, M.D., Ph.D. Chief Medical Officer
Commercial opportunity and next steps	Dan Faga Chief Executive Officer
Q&A	All

ANB033 blocks CD122 to inhibit pathogenic immune cells

CD122 is the beta subunit (IL-2Rβ) of the receptor for IL-15 and IL-2

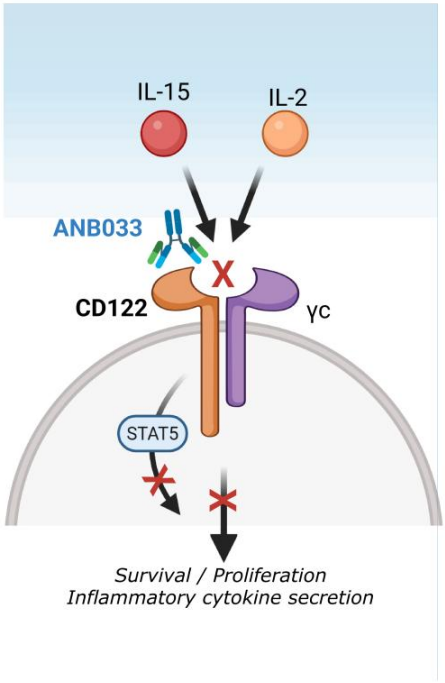
- Expressed on subsets of CD8+ and CD4+ T cells and NK cells

CD122 antagonism reduces these immune cell subsets

- Dependent on IL-15 and/or IL-2 for maintenance, proliferation and survival

Overexpressed in select diseases, including CeD gut or EoE

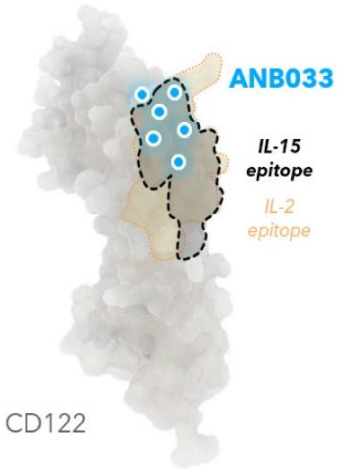
- CeD: IELs, including cytotoxic CD8+ and NK cells
- EoE: ILC2s

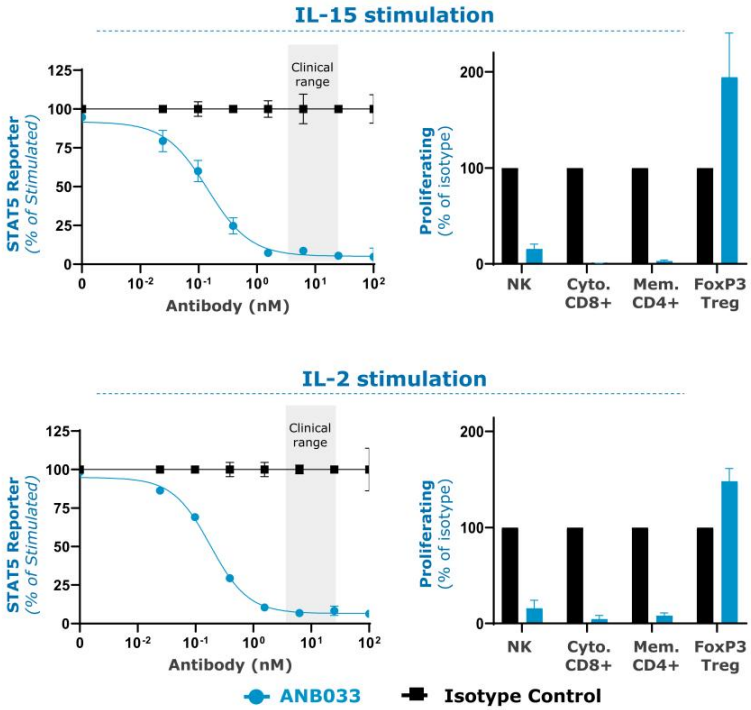


Richards et al, Cell Rep. 2025 Jul 21;44(8):116039.

ANB033's optimized epitope and high affinity lead to differentiated inhibition of IL-15 and IL-2 signaling

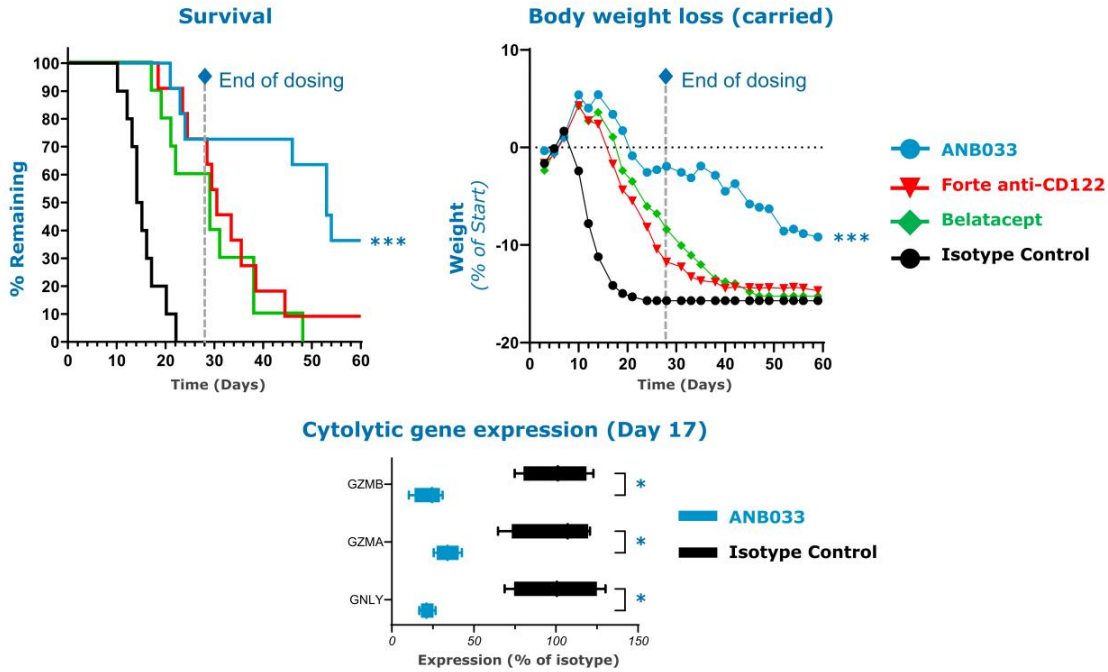
ANB033 epitope sits squarely within IL-15 and IL-2 binding footprint





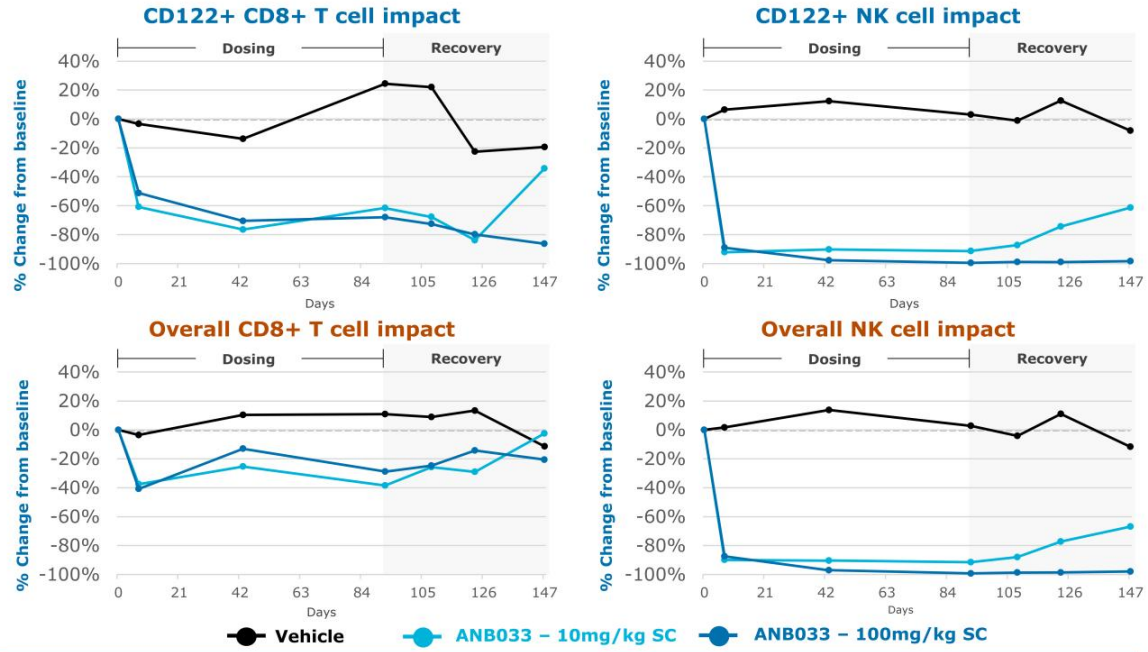
STAT5 luciferase reporter assay run in HEK293 cells that express CD122 and CD132, stimulated with IL-15 or IL-2; Cyto. = cytotoxic; Mem. = memory; Normal donor PBMC measuring proliferation (Ki67 staining) after stimulating with IL-15 or IL-2 for 7 days with isotype or ANB033 at 28 nM (N=4 donors).

ANB033 shows strong survival benefit and reduced cytolytic gene expression in aggressive GvHD mouse model



GvHD (severe phenotype) model using human IL-15 transgenic mice that support human T cell and NK cell engraftment. 60-day study. Mice dosed 3 mg/kg BIW (belatacept 75 µg TIW) through Day 28. N=10 per group (isotype control and Belatacept) or 11 per group (test articles). *** Survival: ANB033 statistically significant vs isotype control (P<0.0001), Belatacept (P=0.003), Forte anti-CD122 (first achieved on Day 38, p=0.031, with significance deepening through Day 60, P=0.0032) log-rank Mantel-Cox test; Body weight loss: ANB033 statistically significant vs isotype control (p<0.001), Belatacept (p=0.0016), Forte anti-CD122 (first achieved on Day 28, p=0.037, with significance deepening through Day 60, P=0.0003), Unpaired Student's t-tests. Gene expression data generated from purified human immune cells isolated from spleen on day 17. * p<0.05 Unpaired Student's t-tests.

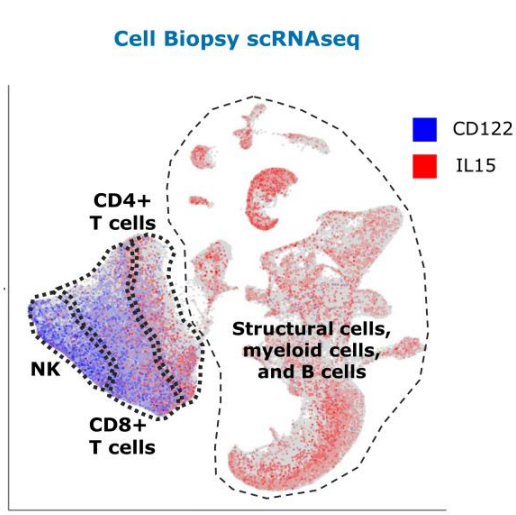
ANB033 reduces key CD122+ target cells in cynomolgus monkeys

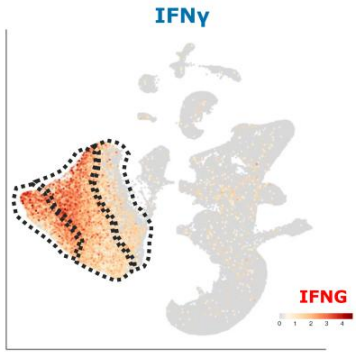


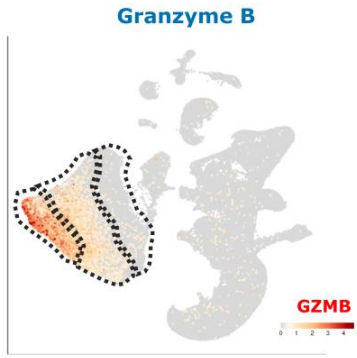
- 10-fold safety margin from cyno monkey toxicology to human in "clinical range" of dosing

Cyno NK cells identified by expression of CD159a, which unlike in humans, virtually all are CD122+.

CeD is characterized by a dense infiltration of inflammatory CD122+ immune cells







Dense CD122 expression by infiltrating immune cells

- IFNγ expression: cytotoxic CD8+ T cells, CD4+ T cells, NK cells
- Granzyme B expression: CD8+ T cells, NK cells

CD122+ cells are increased 50-150% compared to healthy

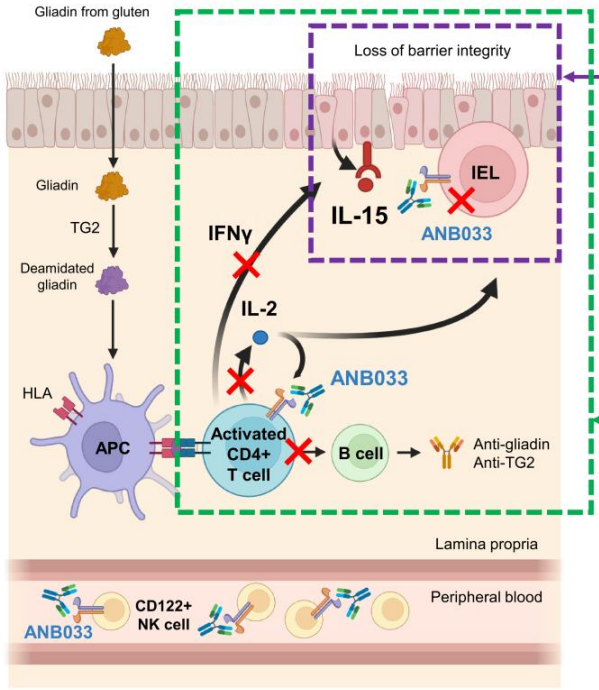
Broad IL15 expression by structural cells

- Epithelial cells, myeloid cells and lymphocytes

Richards et al, Cell Rep. 2025 Jul 21;44(8):116039; UMAP of single-cell RNA-seq data from celiac disease (CeD) intestine, colored by co-expression of gene pairs: Expression values are SCTransform-normalized; a color scale is used: red or blue: high gene expression, gray = low expression.

ANB033's MOA ideal fit for targeting CeD inflammation

CeD marked by excessive IL-15 and IL-2 production which perpetuates disease



Inhibition of IL-15 signaling

- IL-15 induces proliferation of IELs
  - Majority of IELs are CD122+ T cells
- Inhibiting IL-15 signaling reduces IELs
  - Reduces epithelial cell destruction
  - Restores barrier integrity

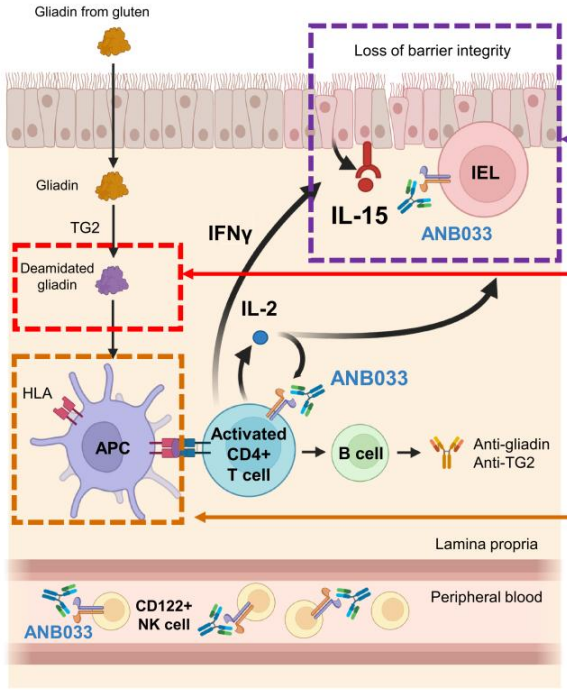
Inhibition of IL-2 signaling

- IL-2 stimulates
  - CD4 effector memory T cell activation and proliferation
  - IFNγ production leading to IL-15 secretion
- Inhibiting IL-2 signaling reduces
  - Gluten-responsive CD4 T cell expansion
  - Inflammatory cytokine secretion
  - Downstream B cell-mediated antibody responses

Adapted from Dieckman et al. (2022) Curr. Opin. Pharmacol. 66:102268.

Previous approaches have not addressed multiple pathogenic drivers of CeD

However, a CD122 antagonist targets both key pathogenic drivers of CeD



IL-15 antagonists: Clinical PoC

- teva — *P2 ongoing*
- NOVARTIS — *P1b PoC*
- AMGEN — *Lacked potency*

Non-immune cell targeting

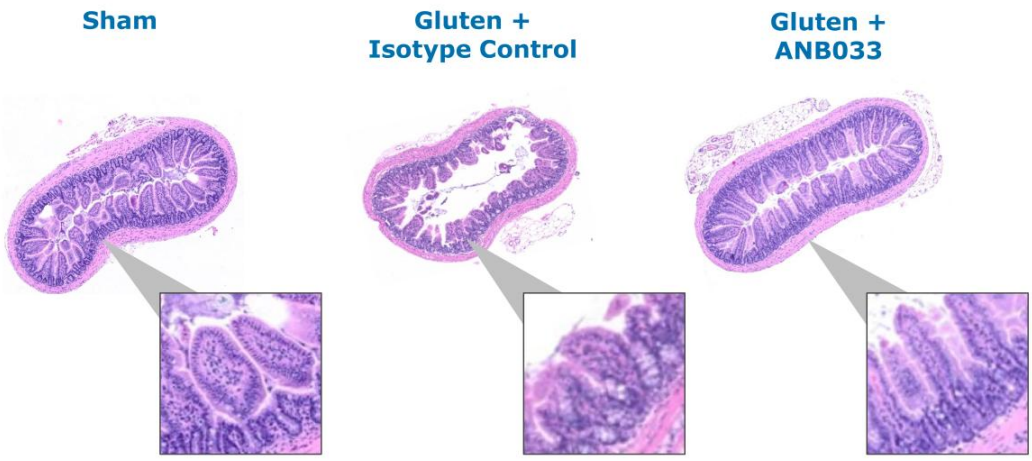
- Takeda — *P2 ongoing; Gluten tolerance*
- astellas — *Discontinued; Gluten tolerance*
- Roche — *P1 ongoing; HLA-DQ2.5 gluten peptide complex*
- Immunic THERAPEUTICS — *P1 ongoing; SIRT6 modulator*

OX-40L antagonist

- sanofi — *P2 ongoing*

Adapted from Dieckman et al. (2022) Curr. Opin. Pharmacol. 66:102268.

ANB033 prevents key CeD histologic manifestation of gluten-induced villous atrophy



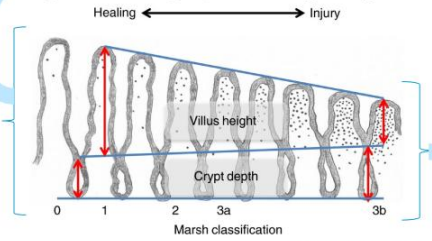
ANB033 treatment shows improved histology:
preserves villus height and crypt depth (Vh:Cd) in CeD mouse model

Note: HuDQ8-D^{d}-villin-IL-15tg mice on a gluten-free diet are challenged with gluten, and CeD features are analyzed on day 30. The treatment regimen includes a sham (no gluten), isotype control and ANB033 surrogate antibody (anti-mouse CD122 antibody with similar epitope and affinity to ANB033) administered at 10 mg/kg BIW.

ANB033 significantly prevents reduction of Vh:Cd ratio compared to control

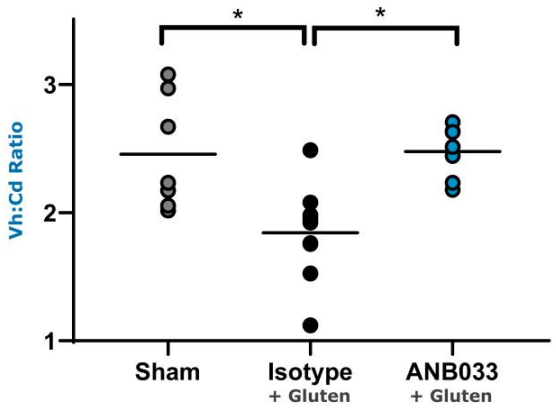
Vh:Cd ratio

Healthy tissue:
High villus height, lower crypt depth



CeD tissue:
Reduced villus height, extended crypt depth

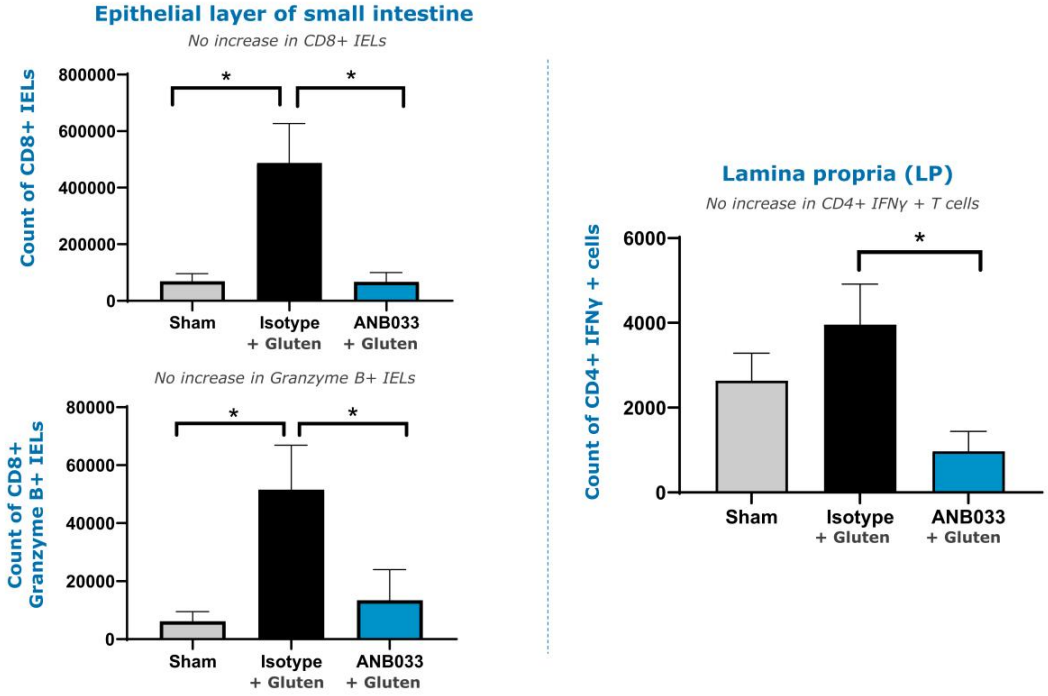
ANB033 impact on Vh:Cd ratio



ANB033 treatment shows improved histology: preserves villus height and crypt depth (Vh:Cd) in CeD mouse model

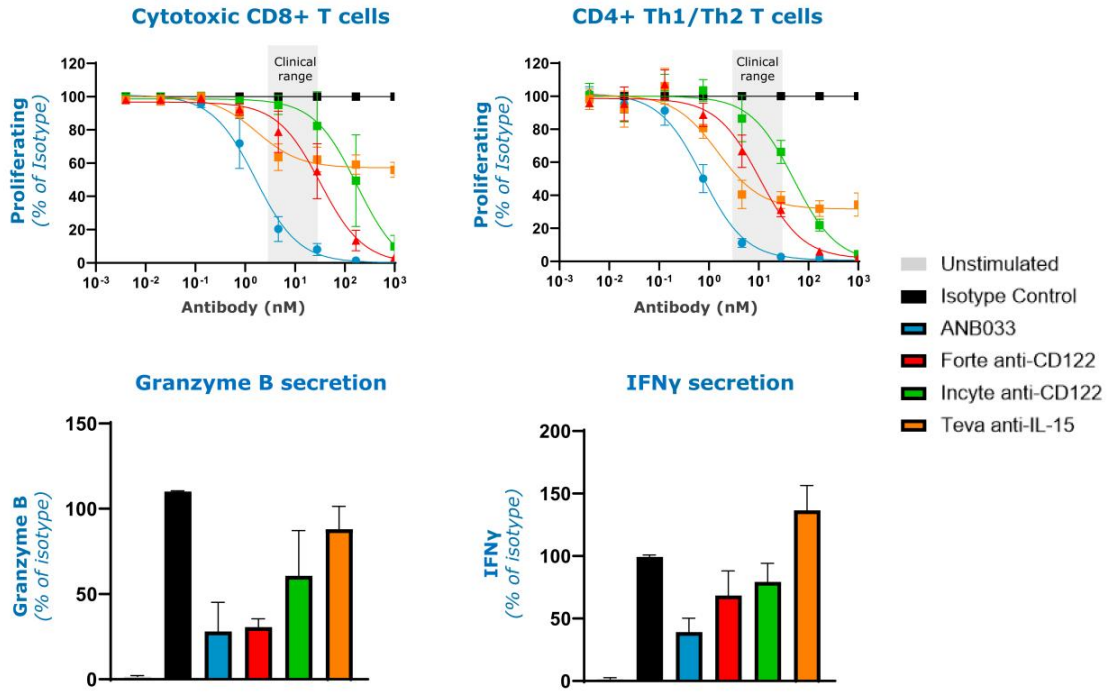
Note: HuDQ8-D^{d}-villin-IL-15tg mice on a gluten-free diet are challenged with gluten, and CeD features are analyzed on day 30. The treatment regimen includes a sham (no gluten), isotype control and ANB033 surrogate antibody (anti-mouse CD122 antibody with similar epitope and affinity to ANB033) administered at 10 mg/kg BIW. * $p<0.05$.

ANB033 prevents gluten-induced intestinal inflammation



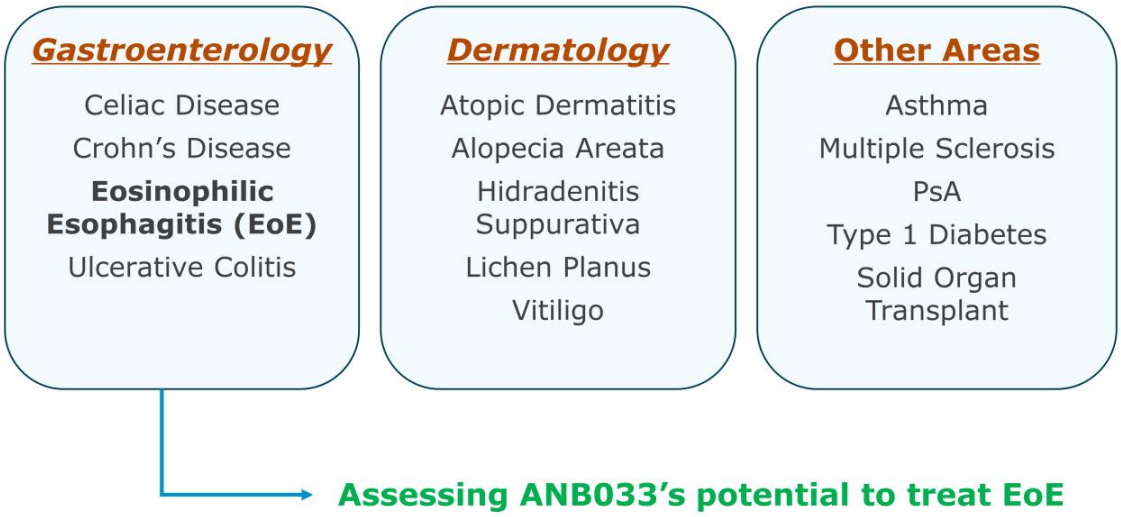
Note: HuDQ8-D^{d}-villin-IL-15tg mice on a gluten-free diet are challenged with gluten, and CeD features are analyzed on day 30. The treatment regimen includes a sham (no gluten), isotype control and ANB033 surrogate antibody (anti-mouse CD122 antibody with similar epitope and affinity to ANB033) administered at 10 mg/kg BIW. IFNγ + CD4 T cells and GrzB+ CD8+ T cells enumerated by intracellular flow cytometry.

ANB033 shows differentiated impact in CeD patient-derived PBMCs compared to competing anti-IL-15s and CD122s



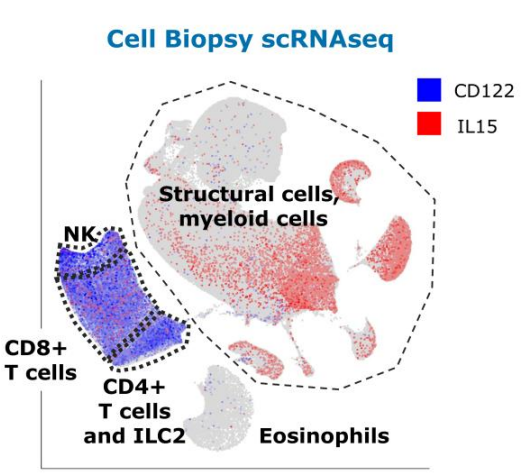
Top Panel: PBMC from CeD donors measuring proliferation (Ki67 staining), stimulated for 7 days with IL-15 + IL-2 (N=4 donors).
Bottom Panel: PBMC from CeD donors stimulated for 3 days with anti-CD3 and anti-CD28 (N=4 donors).

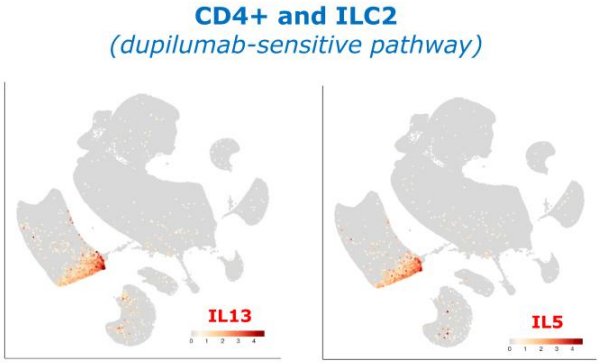
Broad therapeutic potential across autoimmune and inflammatory diseases

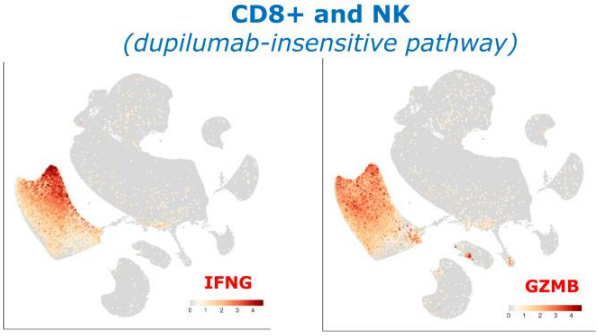


Assessing ANB033's potential to treat EoE

EoE is also characterized by a dense infiltration of immune cells presenting CD122+ expression







Dense CD122 expression by infiltrating CD8+ T cells, CD4+ T cells, NK cells

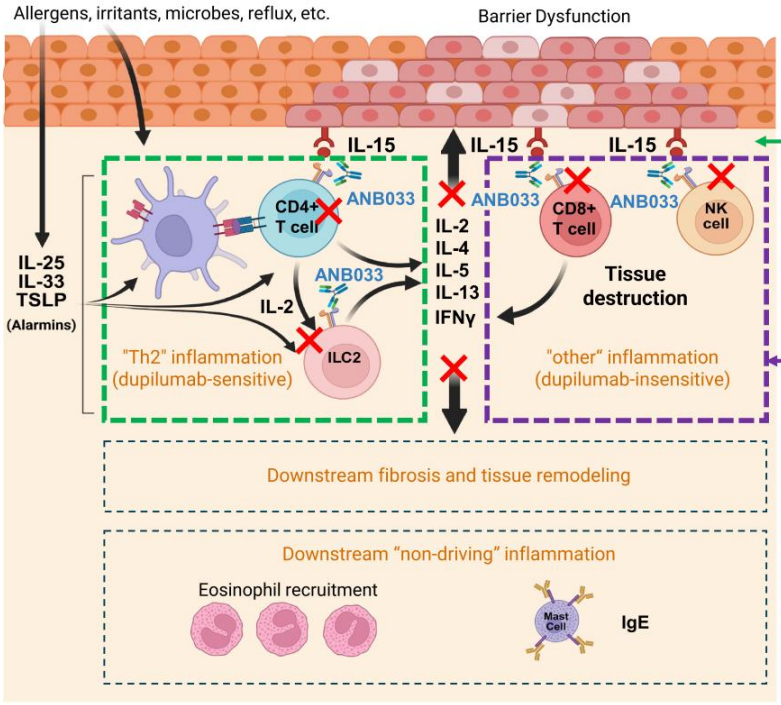
- Across both dupilumab-sensitive and insensitive mechanisms

Broad IL15 expression by structural cells

- Epithelial cells, myeloid cells and lymphocytes

Ding et al, Nature Commun. 2024 Apr 18;15(1):3344; UMAP of single-cell RNA-seq data from EoE biopsies, colored by expression of gene pairs: Expression values are SCTransform-normalized; a color scale is used: red or blue = high gene expression, gray = no expression.

Similar to CeD, ANB033 targets multiple drivers of EoE biology addressing both dupilumab sensitive and insensitive pathways



CD4+ and ILC2 (dupilumab-sensitive)

- Reduces CD4+ T cell proliferation
- Reduces IL-5/13 from CD122+ ILC2 and CD4+ T cells

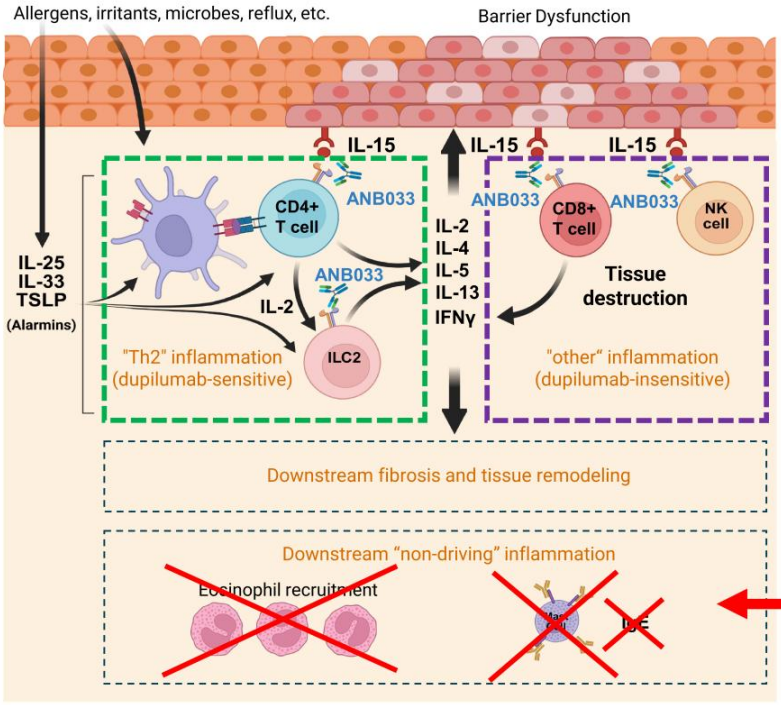
CD8+ and NK (dupilumab-insensitive)

- Inhibiting IL-15 signaling reduces –
  - IFNγ
  - Granzyme B
  - CD8+ T cell proliferation
  - NK cells

Upstream pathways lead to downstream eosinophilic infiltration into tissue

Adapted from Discepolo et. al. Gastroenterology. 2024; 167:90-103.

Mechanisms that target only downstream signals of inflammation have not been successful in EoE



Approved

sanofi

Dupilumab (anti-IL-4Ra)

Has PoC

NOVARTIS Calypso biotech

GIA-632 (anti-IL-15)

Ongoing

AMGEN

tezepelumab (anti-TSLP)

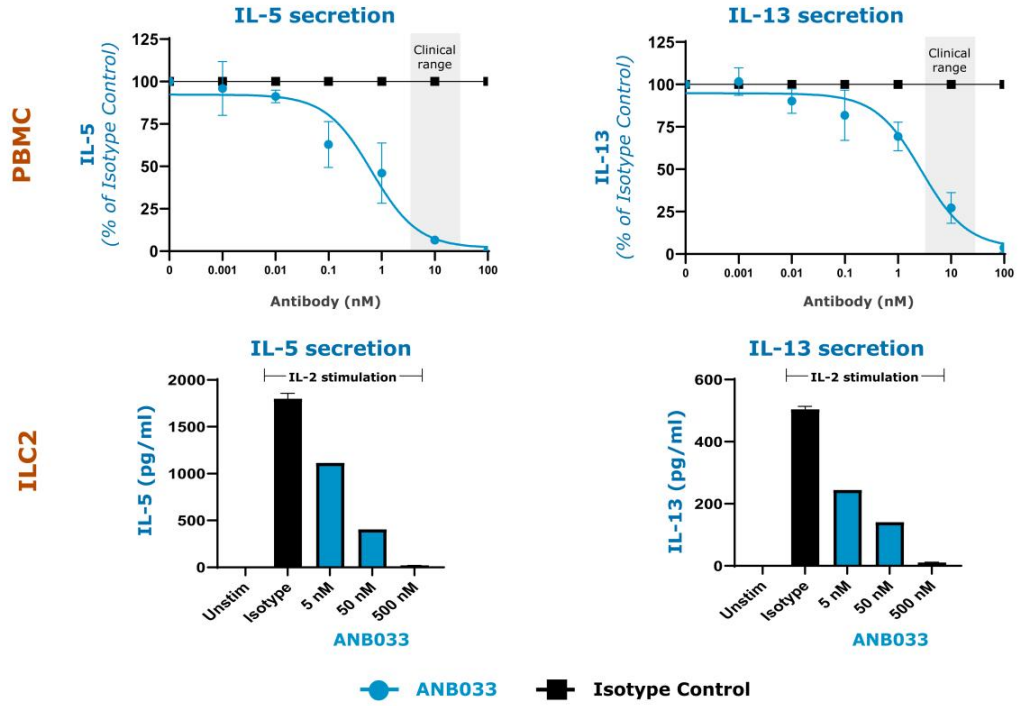
Failed mechanisms

anti-cKIT, anti-IL-5Ra, anti-IgE

Celldex Genentech AstraZeneca

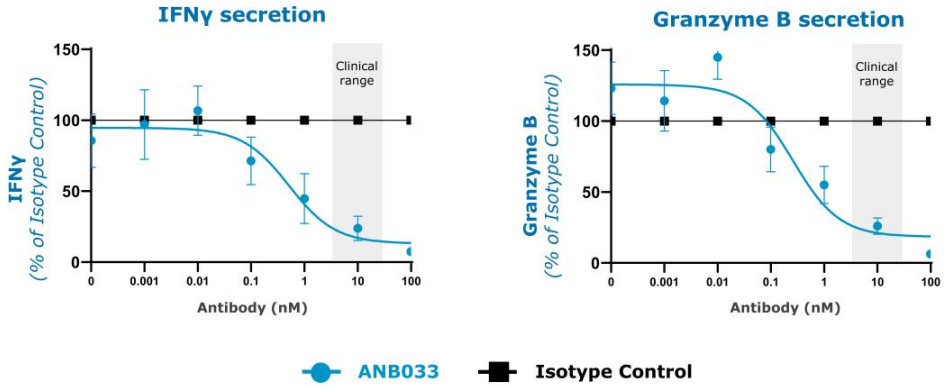
Adapted from Discepolo et. al. Gastroenterology. 2024; 167:90-103.

ANB033 reduces CD4+ T cell and ILC2 derived Th2 cytokines, proven drivers of EoE pathology



Top Panel: Human healthy PBMC were activated by anti-CD3/CD28 for 3 days; n=4 donors shown.
Bottom Panel: Purified human whole blood-derived ILC2 maintained in IL-33 were stimulated with IL-2 for 3 days; 1 of 6 similar representative donors shown.

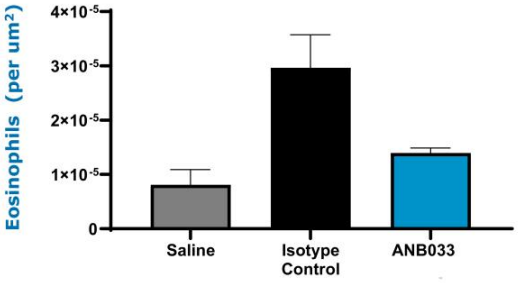
ANB033 reduces CD8+ T cell and NK cell derived Th1 cytokines and cytolytic markers



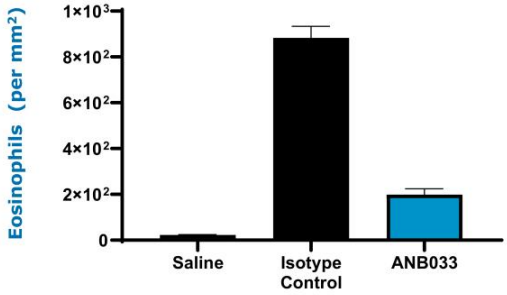
Human healthy PBMC were activated by anti-CD3/CD28 for 3 days; n=4 donors shown.

ANB033 prevents eosinophilia by targeting upstream inflammation

Esophageal eosinophils



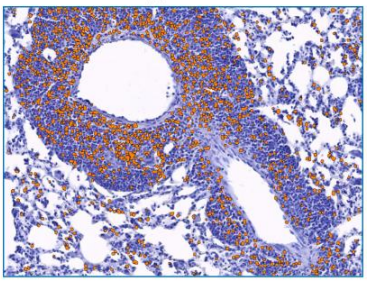
Lung eosinophils



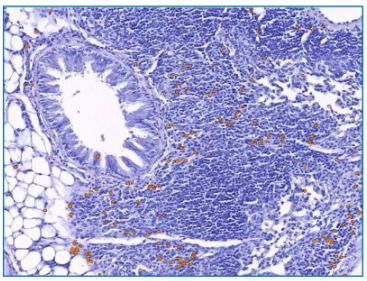
Aspergillus-induced eosinophilia

(Lung sample shown)

Isotype Control



ANB033



Model of eosinophilic inflammation: Balb/c mice were challenged intranasally with *Aspergillus fumigatus* TIW for 3 weeks. The treatment regimen includes a saline, isotype control, and ANB033 surrogate antibody (anti-mouse CD122 antibody with similar binding epitope and affinity to ANB033, administered at 10 mg/kg BIW for 3 weeks. Tissues were stained with H&E for histopathology assessment.

Agenda: ANB033 (CD122 Antagonist)

TOPIC	SPEAKER
CD122 biology and preclinical data	Martin Dahl, Ph.D., Senior Vice President, Research
Phase 1a in healthy volunteers	John Kwon, M.D., Ph.D. Vice President, Clinical Development
Drug development for CeD	Joseph Murray, M.D. Professor of Medicine Mayo Clinic College of Medicine, Rochester, MN
Phase 1b in CeD	Paul Lizzul, M.D., Ph.D. Chief Medical Officer
Commercial opportunity and next steps	Dan Faga Chief Executive Officer
Q&A	All

ANB033 Phase 1a trial ongoing in healthy volunteers

Objectives

- Safety and tolerability
- Evaluate PK and immunogenicity

Design

- All healthy volunteers have been dosed
  - ANB033: n=60
  - Placebo: n=20
- Administered both IV and SC dosing
- 10 cohorts: Four SAD IV, three SAD SC and three MAD SC
- Follow-up to ~7 months*

* The first 4 lowest SAD dose cohorts are followed through day 85; the three higher SAD dose cohorts are followed for 197 days; all MAD cohorts are followed through 218 days.

ANB033 demonstrated favorable safety, tolerability and PK profile in Phase 1a

Phase 1a results to date

✓ Safe and well tolerated

✓ No unexpected findings

✓ PK and PD support SC dosing

Favorable safety and tolerability

- No safety concerns at any dose
  - No SAEs, severe AEs, or discontinuations
  - Any adverse events mild or moderate
- No unexpected lab abnormalities
- No signs of viral infections
- No clinical pharmacology findings of concern

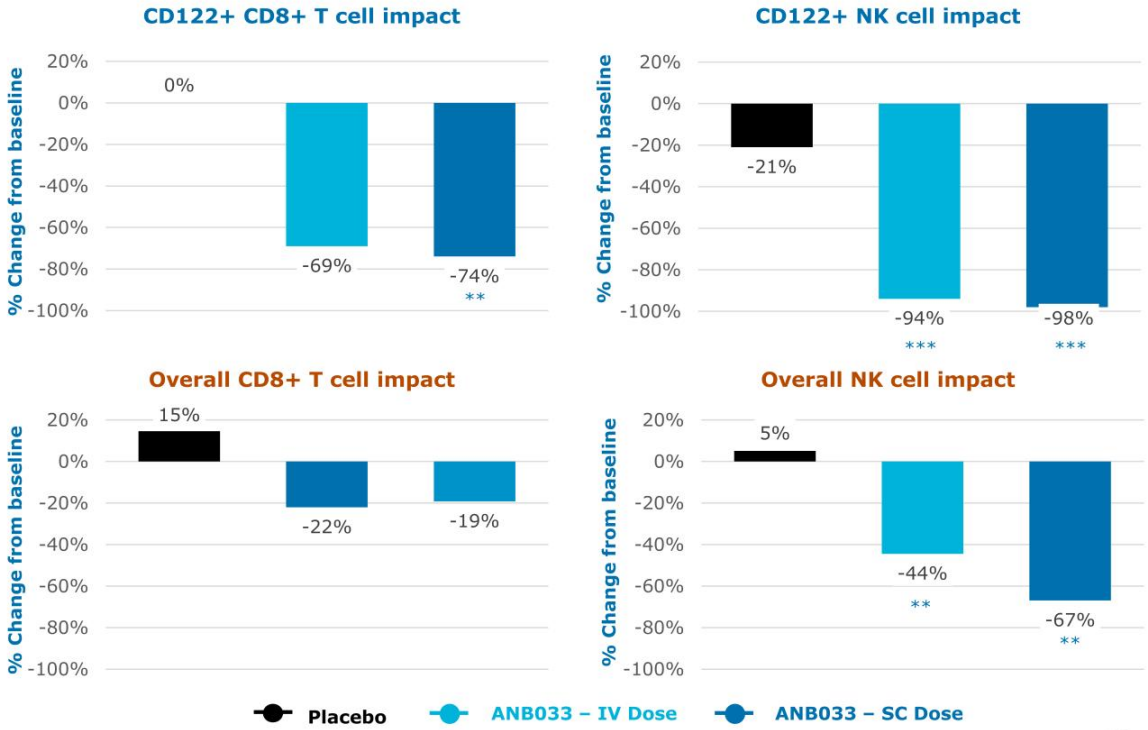
Rapid and sustained PK profile

- Favorable 2 to 3-week half-life with IV and SQ dosing
- Full receptor occupancy (RO) within hours and maintained for >30 days
- Dose response observed
- Modeled to achieve >IC90 on CD8+ T cells subsets in GI tissue
- Overall, no impact on peripheral total Treg counts

SAE = Serious adverse event

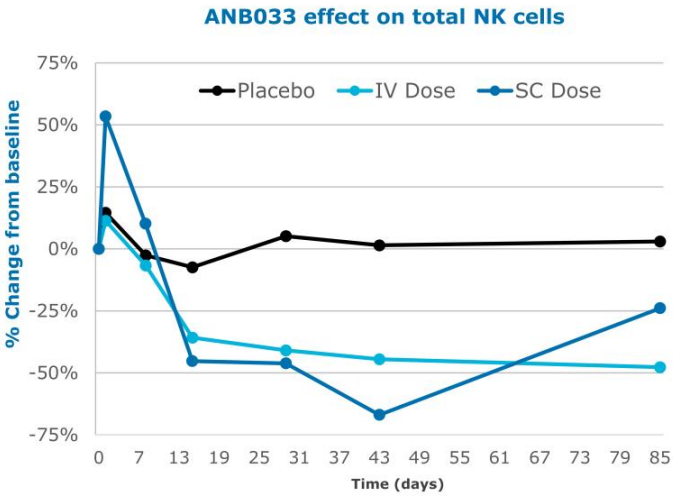
ANB033 significantly reduces CeD relevant CD8+ T cells and NK cells after single dose

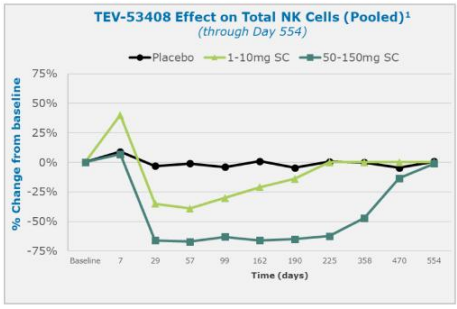
Effect of ANB033 is limited to CD122 expressing cells



Graphs reflect SAD data and maximum reductions were achieved within the first 43 days. *** p<0.001 **p<0.01

Anti-IL-15 and CD122 therapies have demonstrated sustained reduction in CD122+ NK cells with no observed safety issues





No safety signals observed in any CD122 or IL-15 trials to date after NK cell reduction

- ANB033 >50% peak total NK cell reduction with return towards baseline within 3 months
- TEV-53408: >50% sustained total NK cell reduction for 1 year with return to baseline over 18 months

1. Schnir et. al; Developing TEV-53408 for the Treatment of Celiac Disease: Summary of Preliminary Results from the First-in-Human Phase 1 Study in Healthy Volunteers, Single SC doses, DDW, May 2024. Phase 1a, single dose, study completed (n=60 TEV-53408, n=19 placebo). Moved into Phase 2a CeD trial in 48 adults while undergoing gluten challenge; primary trial completion in Sept. 2026.

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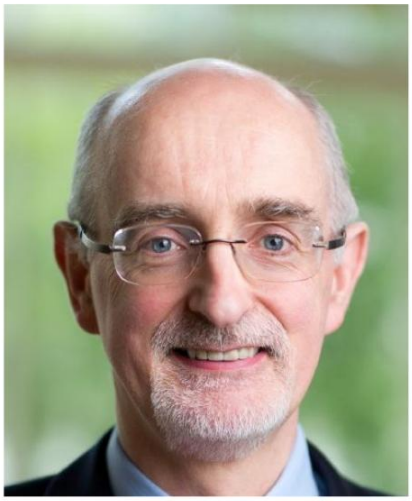
KOL discussion



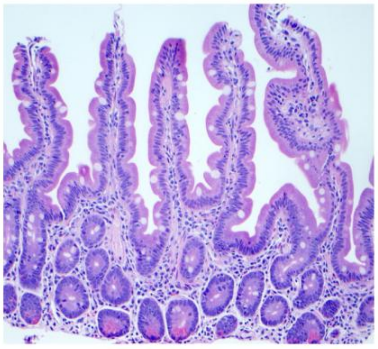
Joseph A. Murray, M.D.,
Professor of Medicine, Director, Celiac Disease Research, John and Shirley Berry Professor of Gastrointestinal Sciences, Division of Gastroenterology and Hepatology, Department of Internal Medicine, Mayo Clinic, Rochester, MN

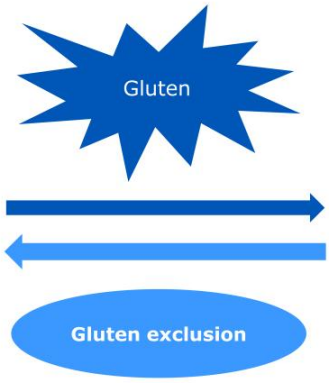
- Co-founder and past president of North American Society for Study of CeD
- Contributed to 2013 guidelines and 2019 AGA practice update on diagnosing and monitoring CeD
- Published more than 450+ peer-reviewed papers on CeD
- Past chair of AGA's Intestinal Disease Section
  - Broad experience with GI disorders, including EoE

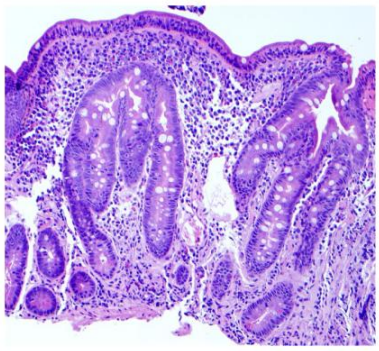
Gluten and Celiac Disease (CeD)

- Genetic predisposition
- Permanent intolerance to gluten

- Inflammation
- Villi destruction
- Crypt hyperplasia
- IEL proliferation







Normal histology

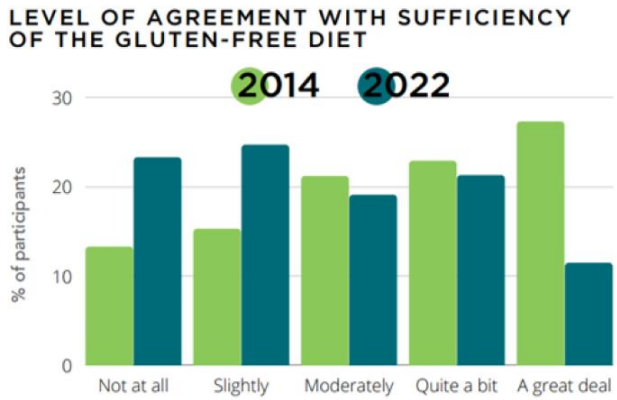
(Marsh 0)

Total villous atrophy

(Marsh 3)

Note: Marsh score -- a histological grading system – classifies severity of intestinal damage by evaluating microscopic appearance (IELs and Vh:Cd) of a small intestine biopsy. Ranges from Marsh 0 (normal villi) to Marsh 3c (complete villous atrophy), with higher scores indicating progressively more severe damage

Majority of CeD patients desire new treatment options



There has been a significant decline in satisfaction over time

- In 2022, only 12% of participants agreed "a great deal" that a gluten free diet was a sufficient treatment, compared to 27% in 2014

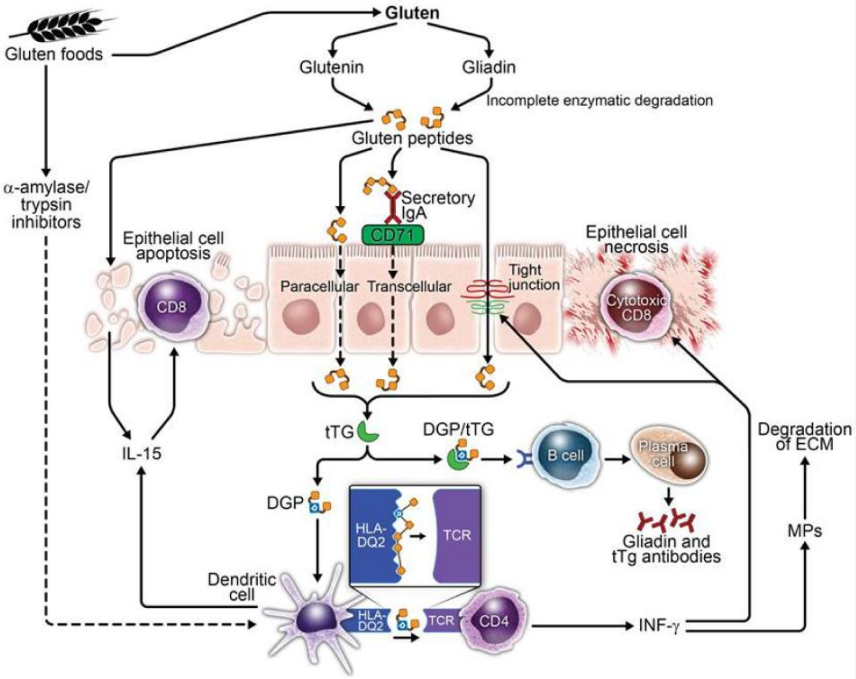
Consequences of persistent, chronic active CeD:

- GI symptoms: abdominal pain, diarrhea, constipation
- Severe malnutrition: vitamin deficiencies, anemia
- Reduced quality of life
  - Reduced social, emotional well being
  - Diminished physical functioning
- Osteoporosis
- Increased cancer risk: intestinal lymphoma, small bowel cancer

Regardless of gluten free diet, ~50% of patients suffer anemia or fatigue

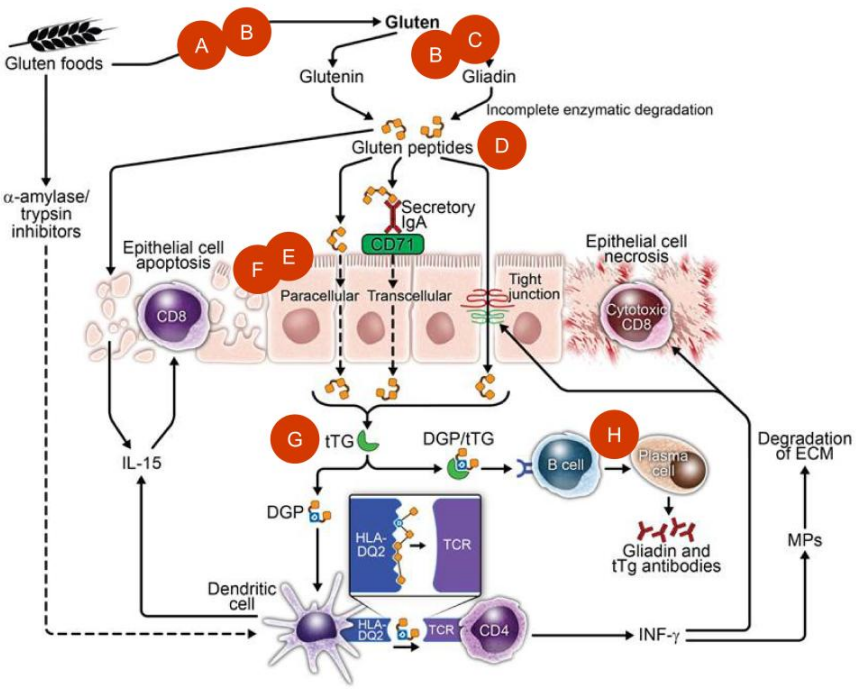
Adams et al Gastroenterology 2024;167:51-63; 1460 and 1140 CeD participants completed survey in 2014 and 2022

CeD has a multi-cell pathology



- Immune scarring is present in treated CeD
  - Epithelial-stromal-immune impacts / interactions
- IL-2, produced by CD4 T cells, elevates following gluten ingestion
  - Starting 2 hrs. after exposure
- Stressed epithelial cells express IL-15 (olmesartan)
- Key role of IL-15 contributes to:
  - Expansion of T cells, particularly IELs
  - Expansion and activation of NK cells

Some mechanisms that have been tried in CeD…



A — Toxic wheat → Non-toxic wheat

B — Enzymes + Wheat → Gluten peptide fragments

C — Protease supplement (IMGX003), Kumamax (TAK-062), and Allergan

D — Polymeric gluten binder

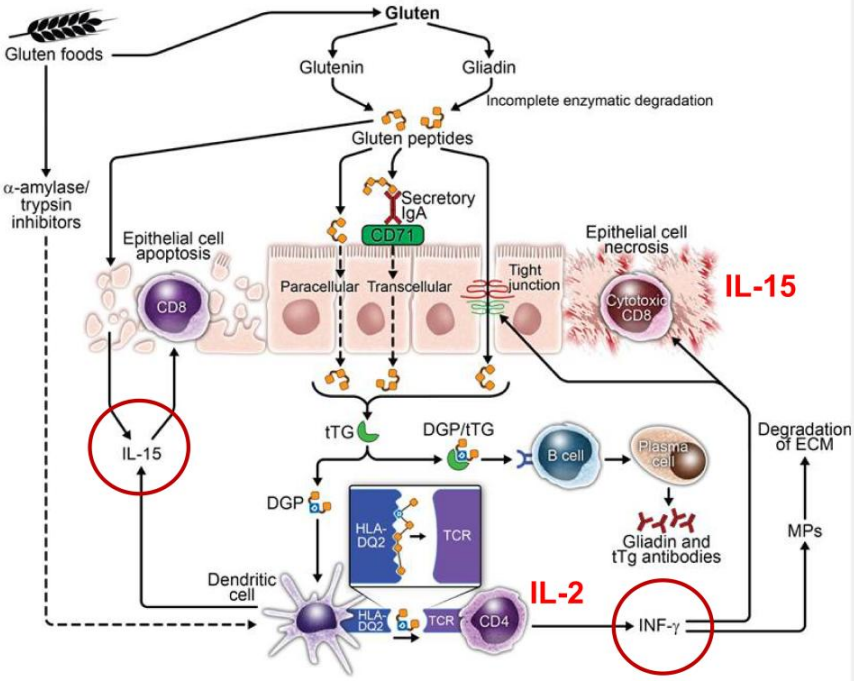
E — Probiotics (B. infantis)

F — Permeability inhibitor (larazotide acetate)

G — AntiTTG (ZED1227)

H — Nexvax

Rationale in targeting CD8 and CD4 T cells to broadly impact CeD inflammation

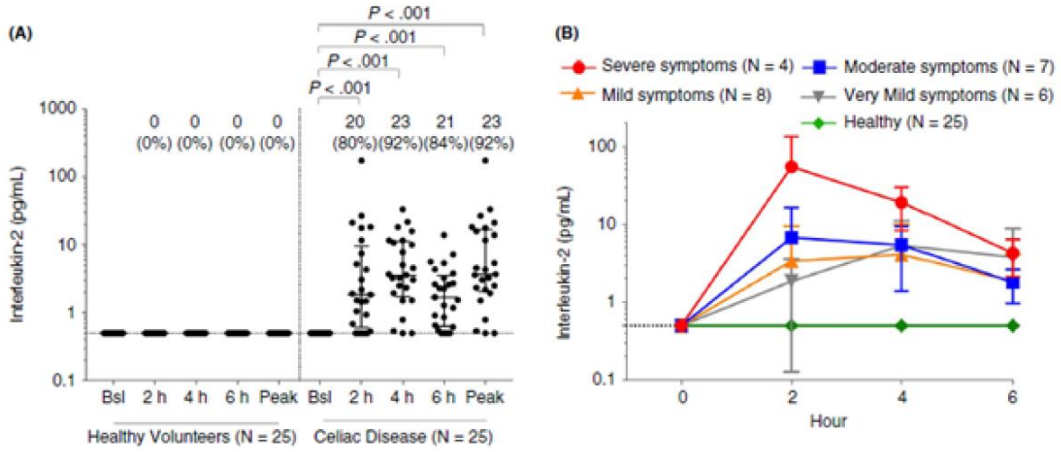


- Overactive IL-15 and IL-2 signaling drives pathogenic immune cell expansion and inflammation effects
- IL-15 is inflammatory cytokine
  - Expressed by stressed epithelial cells
  - Supports T cell and NK cell homeostasis and expansion
  - Binds intercellularly
  - IL15/IL15R transported to cell surface by stromal cells and APCs
- IL-15 expression in epithelium (mid villi), crypts and LP increased in CeD patients
- IL-2 stimulates CD4 Tem cell activation and proliferation and IFNγ production, leading to IL-15 secretion

TEM= T effector memory

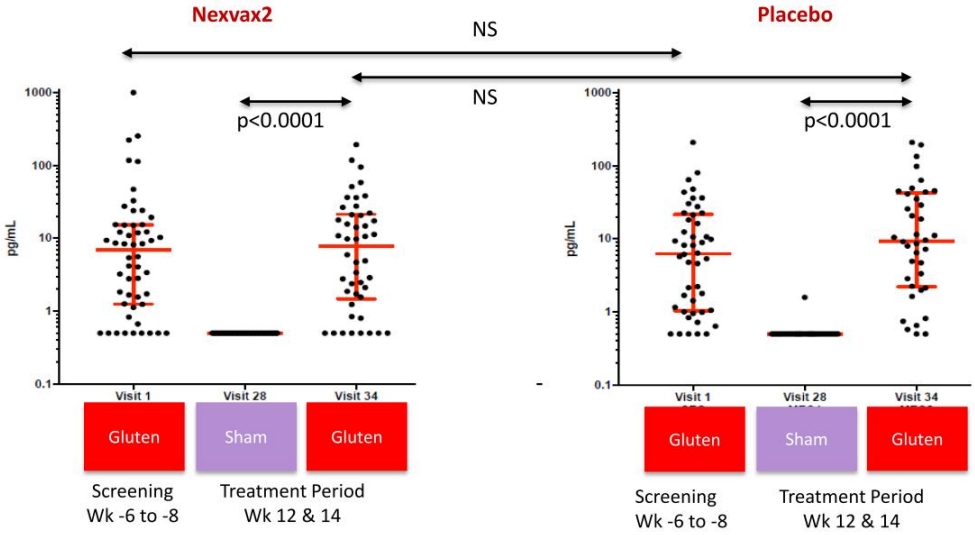
Serum IL-2, produced by CD4 T cells, elevated in CeD patients

- Increase in IL-2 seen within two hours of gluten ingestion
- Magnitude of IL-2 response correlates with severity of symptoms
- IL-2 elevation key marker for central role of CD4 T cells
- IL-2 elevation is not observed in healthy controls, CeD patients provided sham GC or non-celiac gluten sensitivity[1,2]



1 Tye-Din et al Aliment Pharmacol Ther 2019;00:1-10
2 Cartee et al Am J Gastroenterol 2022; 117:319-326

Gluten exposure induces significant serum IL-2 increase vs. sham

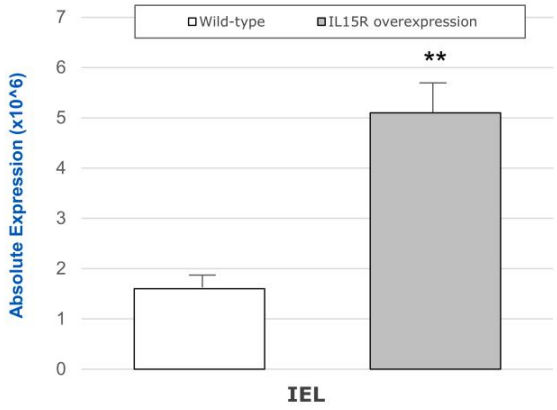


Immunotherapy aiming to restore tolerance in CD4+ T cells failed to reduce significant IL-2 induction from gluten challenge

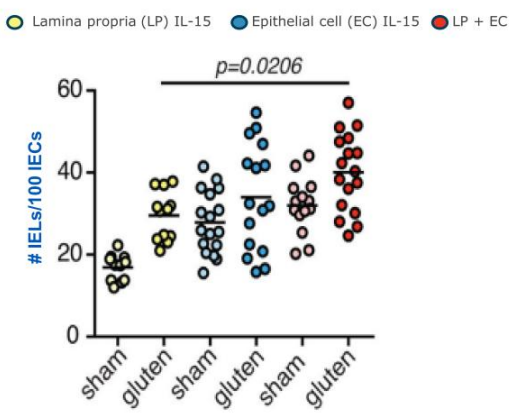
Note: Serum levels 4 h after.

IL-15 a key driver of IEL expansion

Mouse model overexpressing IL-15Rα



Three mouse models overexpressing IL-15

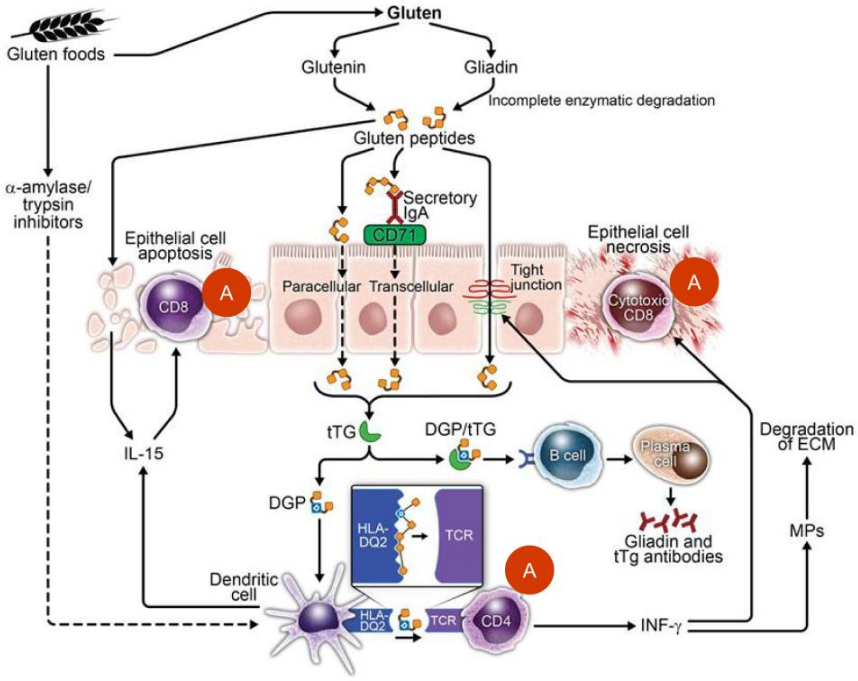


Intestinal epithelial IL-15 expression drives increases in IELs

IL-15 expression in gut drives gluten-induced increases in IELs

Left panel: Mouse model over-expressing IL-15Ra in the intestinal epithelial cell
Right panel: Mouse models with genetic predisposition to celiac disease (HLA-DQ8) and IL-15 expression driven in the epithelium and/or the lamina propria

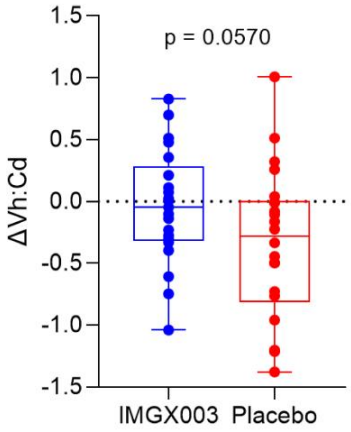
Inhibiting CD122+ CD4+ and CD8+ cells and signaling of IL-15 and IL-2



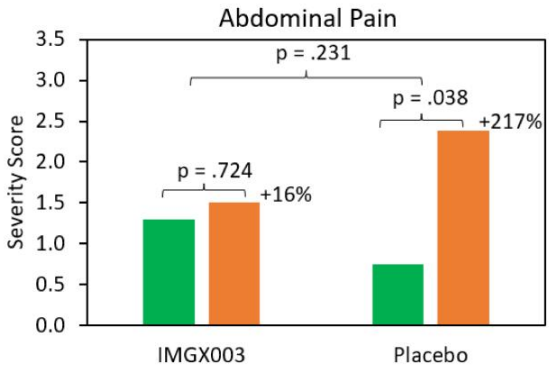
- CD122 shared receptor subunit through which both IL-15 and IL-2 signal
- Expressed on range of immune cells: CD8+ and CD4+ T cells, NK cells
- Inhibiting IL-15 and IL-2 signaling inhibits subsets of these cells, reducing inflammation and tissue destruction
- Targeting CD4 T cell activity leads to reduced inflammation and downstream immune responses, ultimately decreasing CeD consequences

A CD122 antagonist

Latiglutenase: MOA effective in preventing gluten induced injury in P1b but not in healing established disease



Latiglutenase attenuates mucosal damage by ~80% based on ratio of means for IMGX003 *vs.* placebo arms



Based on means latiglutenase attenuated abdominal pains for 2g gluten per day by 93%

This MOA was not predictive for symptomatic CeD patients with mucosal damage and failed P2 study

Oberhuber G, Granditsch G, Vogelsang H. The histopathology of celiac disease: time for a standardized report scheme for pathologists. Eur J Gastroenterol Hepatol. 1999;11(10):1185–1194

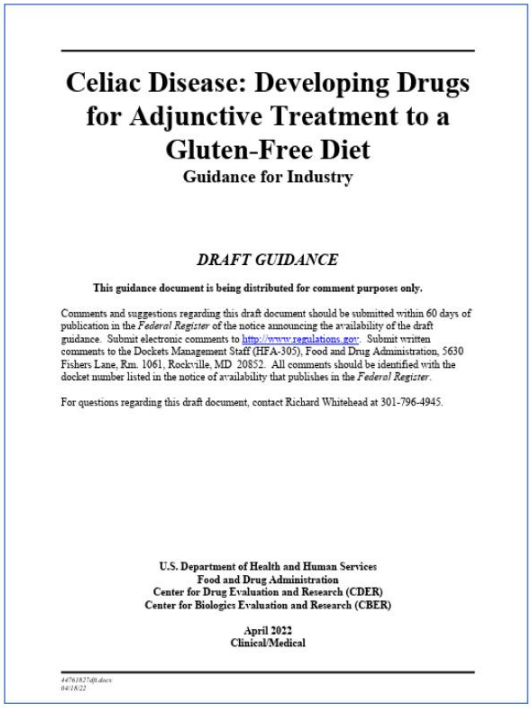
Considerations for Phase 1b CeD trial design

- Traditional P1b gluten challenge
  - Controlled symptoms and limited mucosal damage at baseline
- P2b and P3 population targets symptomatic CeD patients with mucosal damage
- Drugs whose MOA breaks down gluten (e.g., latiglutenase), have shown positive effects in P1b gluten challenge
  - Not predictive for P2b population
- For immunologic MOAs that treat inflammation, a P1b trial may be predictive of P2b
  - Could directly result in mucosal healing
- Tolerating gluten in P1b gluten challenge is difficult, leading to non-compliance

For drugs with upstream immunologic MOA, early P1b trial data that demonstrate mucosal healing and/or protection from gluten challenge, provide key foundation for further clinical development

Perspectives on FDA guidance for CeD

- GREAT Conference*
  - Significant public engagement at advocacy meetings (Beyond Celiac 2024)
- **Alignment on P2/P3 target population: symptomatic CeD despite a GFD***
  - **Need to prevent symptoms of gluten exposure in CeD patients and improvement of histologic injury**
- FDA DRAFT guidance available for registrational trials –
  - Inclusion criteria: evidence that disease is active and causing symptoms
  - Co-primary endpoints: both symptoms (PRO) **and** histology (various)
    - Both must be met as a whole



Celiac Disease: Developing Drugs for Adjunctive Treatment to a Gluten-Free Diet

Guidance for Industry

DRAFT GUIDANCE

This guidance document is being distributed for comment purposes only.

Comments and suggestions regarding this draft document should be submitted within 60 days of publication in the *Federal Register* of the notice announcing the availability of the draft guidance. Submit electronic comments to http://www.regulations.gov. Submit written comments to the Dockets Management Staff (HFA-305), Food and Drug Administration, 5630 Fishers Lane, Rm. 1061, Rockville, MD 20852. All comments should be identified with the docket number listed in the notice of availability that publishes in the *Federal Register*.

For questions regarding this draft document, contact Richard Whitehead at 301-796-4945.

U.S. Department of Health and Human Services
Food and Drug Administration
Center for Drug Evaluation and Research (CDER)
Center for Biologics Evaluation and Research (CBER)

April 2022
Clinical/Medical

GREAT Conference= The Group on Research, Education, and Training (GREAT) provides professional development to, and fosters the exchange of information and ideas among the faculty and administrative leaders of biomedical Ph.D., M.D.-Ph.D. and postdoctoral programs; GFD= Gluten free diet

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Symptomatically controlled CeD patients present with range of histologic activity

Histology *(Vh:Cd ratio)*

3.5 — 2.5 — 2.0 — 1.5 — 1.0 — 0.1

Normal — Mucosal damage — Abnormal

Symptoms

Asymptomatic — Symptom controlled — Poorly controlled

Symptomatically controlled on GF diet

Gluten challenge
Phase 1 population

teva *(Phase 1b)* | NOVARTIS Calypso *(Phase 1b)* | FORTE *(Phase 1b/2a)*

Nearly all P1b/P2a studies only assess ability **to prevent** gluten-induced mucosal injury

- Gluten challenge: patients with higher Vh:Cd ratios (>2.5 or >2.0)

Persistent mucosal damage despite paucity of symptoms

AnaptysBio *(Phase 1b)*

Added additional cohort to P1b **to inform on potential to heal mucosa** in patients with existing histologic mucosal damage and further derisk 2b

Symptomatic on GF diet

Non-responsive

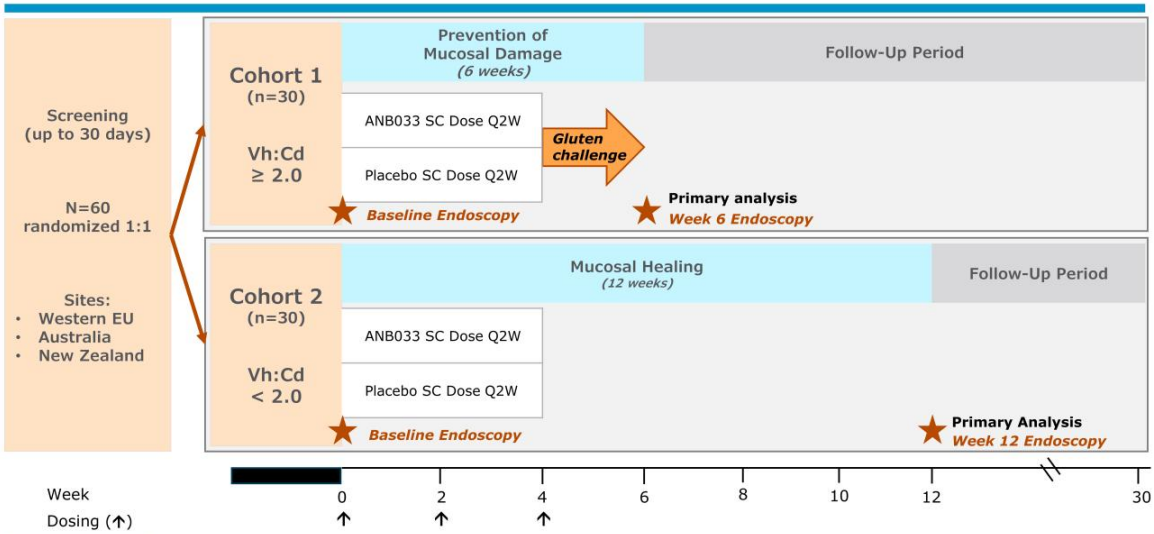
sanofi *(Phase 2b)*

Goal of P2b or P3 to assess if drug can heal damaged mucosa and restore normal symptomatology

GF diet = Gluten free diet.

ANB033 Phase 1b trial in CeD initiated

Anticipate top-line data by year-end 2026



Safety	Safety and tolerability in adult participants with well-controlled CeD
Clinical PK	PK and immunogenicity
Efficacy	• Change from baseline in Vh:Cd ratio • IEL count • PROs, including Celiac Disease Symptom Diary (CDSD)
Biomarkers	Characterize ANB033 effects on circulating biomarkers, including robust translational plan

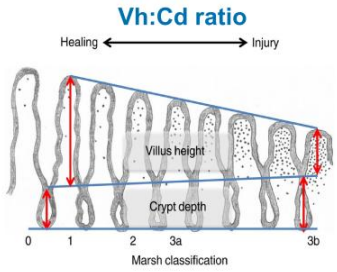
CeD quantitative histology and qualitative symptom assessments

Histology *(Vh:Cd ratio)*

3.5	2.5	2.0	1.5	1.0	0.1
Normal		Mucosal damage			Abnormal

Quantitative histology measures villus height (Vh), crypt depth (Cd) and intra-epithelial lymphocyte count (IEL per 100 enterocytes) to assess histologic changes

- Vh:Cd ratio
- IEL count

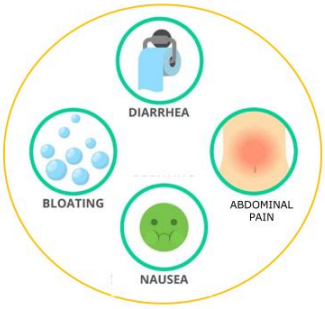


Symptoms

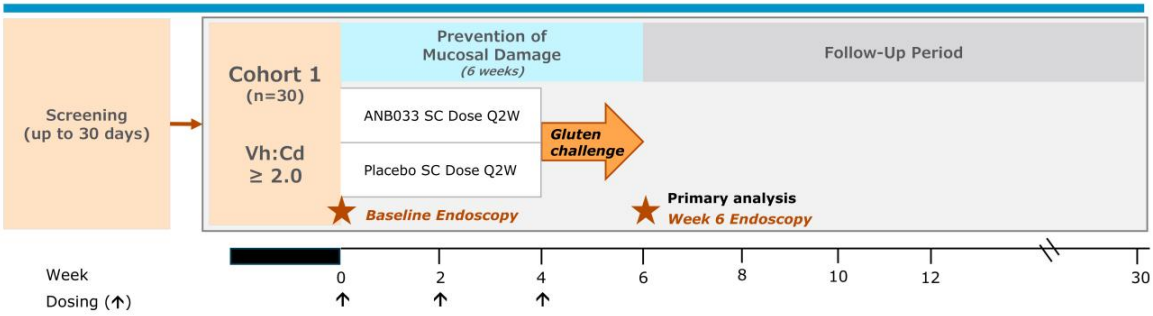
Asymptomatic	Symptom controlled	Poorly controlled

Qualitative evaluation to track changes during treatment or gluten exposure

- CeD Symptom Diary (CDSD) monitors symptoms, including
  - Severity: nausea, abdominal pain, tiredness, and bloating
  - Frequency: vomiting, diarrhea, and bowel movements



Cohort 1 (Vh:Cd ≥ 2.0) is a gluten-challenge to assess prevention of mucosal damage



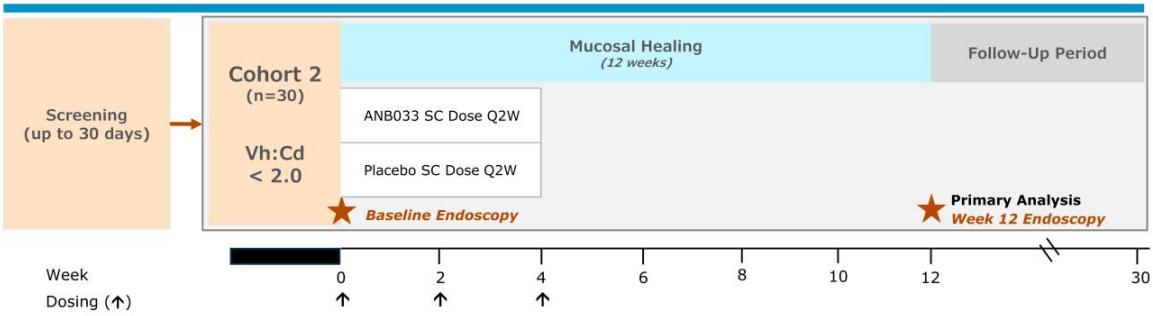
Minimal evidence of mucosal damage (Vh:Cd ≥ 2.0)

- Symptom-controlled CeD patients
- Receive GC after pre-treatment with ANB033 vs. PBO

- ANB033 dose at Week 0, 2, 4 (pre-treatment)
- Gluten challenge allows for controlled induction of mucosal damage
  - Beginning Week 4, 6g gluten dose daily (study supplied cookie) for two weeks through Week 6
- Endoscopy at Week 6
  - Assess prevention of gluten-induced mucosal damage

GC = Gluten challenge

Cohort 2 (Vh:Cd < 2.0) assesses ability to heal mucosal damage in symptom-controlled patients



Persistent evidence of histologic CeD activity (Vh:Cd < 2.0)

- Symptom-controlled CeD patients
- Substantial mucosal damage already present (no gluten-challenge)
- *Proxy: nonresponsive patients*

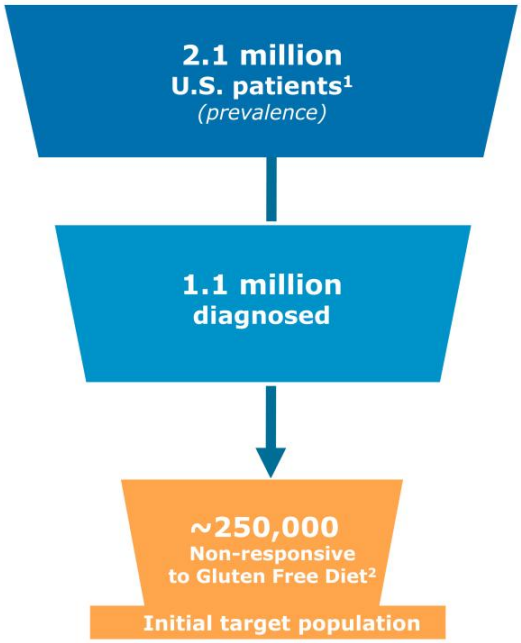
- ANB033 dose at Week 0, 2, 4
- Endoscopy at Week 12
  - Assess healing 8 weeks after last ANB033 dose
  - Maximize healing time given ANB033 prolonged tissue exposure and PD properties

GC = Gluten challenge

Agenda: ANB033 (CD122 Antagonist)

TOPIC	SPEAKER
CD122 biology and preclinical data	Martin Dahl, Ph.D., Senior Vice President, Research
Phase 1a in healthy volunteers	John Kwon, M.D., Ph.D. Vice President, Clinical Development
Drug development for CeD	Joseph Murray, M.D. Professor of Medicine Mayo Clinic College of Medicine, Rochester, MN
Phase 1b in CeD	Paul Lizzul, M.D., Ph.D. Chief Medical Officer
Commercial opportunity and next steps	Dan Faga Chief Executive Officer
Q&A	All

Potential blockbuster opportunity for ANB033 in non-responsive CeD



High disease burden

- Debilitating symptoms, social isolation
- Disease awareness driving growth
- No approved therapies

CD122s differentiated from other Tx in development

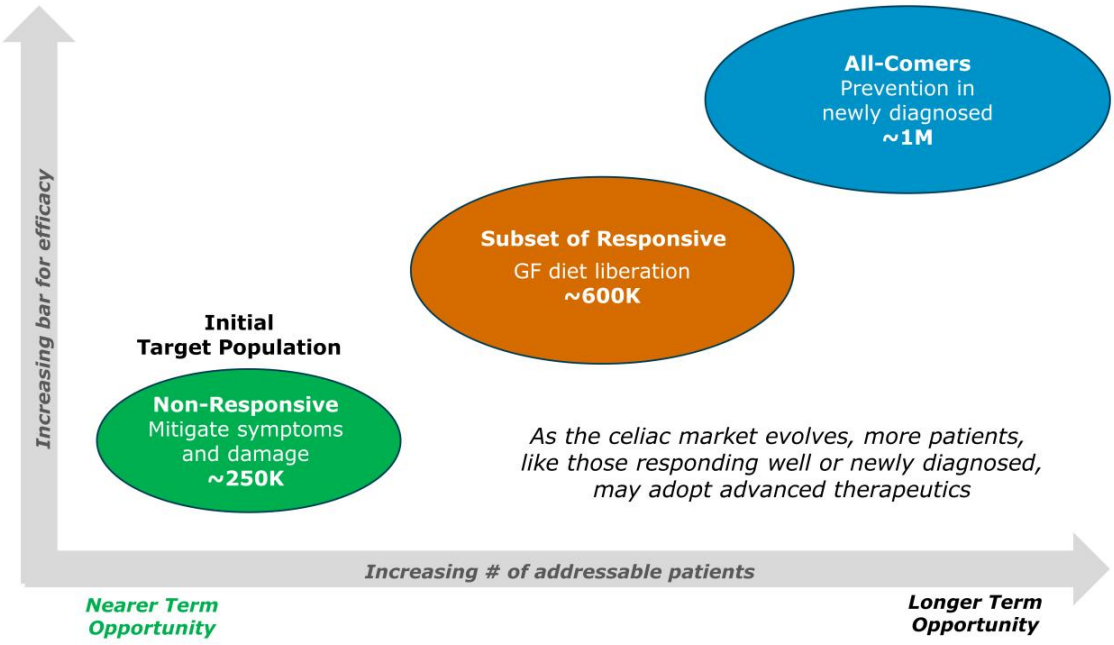
- HCPs favor MOA that targets both symptoms and histology

$4-5B U.S. market in patients non-responsive to gluten-free diet

- Potential to reach IBD diagnosis and biologic penetration analogs given substantial unmet need
- Expect reimbursement with limited utilization management

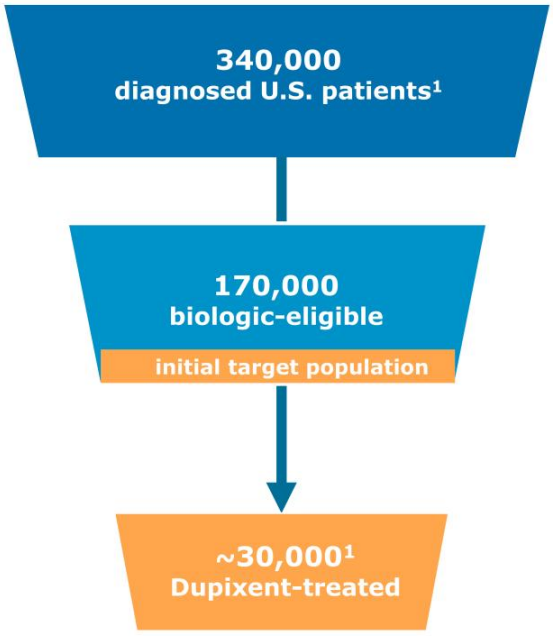
1. Singh et al. (2018), Choung et al. (2016), Katz et al. (2011), Trinity Life Sciences Commercial Assessment HCP Primary Market Research (2025). CeD sizing reflects future US market in 2030 assuming growth in diagnosis rate based on historic trends and projected growth with entrance of novel therapies i2. Leffler et al. (2007), Abhijeet et al. (2016), Aggarwal et al. (2025) Mahadev et al. (2017, Trinity Life Sciences Commercial Assessment HCP Primary Market Research (2025) Percent of CeD non-responders to Gluten Free Diet with or without villous atrophy.

New therapies in CeD could grow market in responsive and newly diagnosed patients



1. Singh et al. (2018), Choung et al. (2016), Katz et al. (2011), Leffler et al. (2007), Abhijeet et al. (2016), Aggarwal et al. (2025) Mahadev et al. (2017, Trinity Life Sciences HCP Primary Market Research (2025)] CeD sizing reflects future US market in 2041 assuming growth in diagnosis rate based on historic trends and projected growth with entrance of novel therapies.

Assessing potential to treat EoE: significant market with increasing prevalence and unmet need



Significant unmet need with limited approved therapies

- ~50% PPI or steroid non-responsive or intolerant
- Dupixent QW approved in 2022
- 20-30% Dupixent non-responsive

Increasing disease recognition with >8% CAGR[1,2]

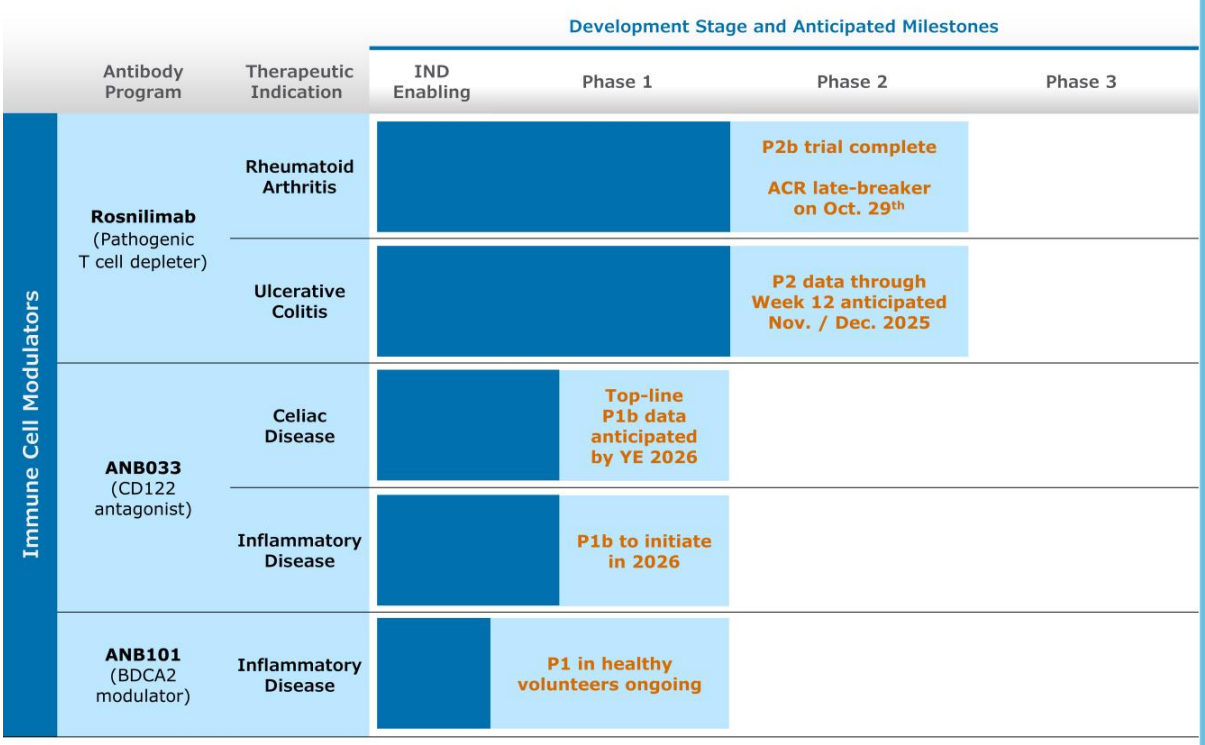
- Heightened rates of endoscopic procedures and biopsies

~$5B+ U.S. sales anticipated by 2030

- Potential to reach IBD diagnosis and biologic penetration analogs given substantial unmet need

1. ZS Claims analysis and KOL interviews August 2025; 2. "Prevalence and costs of eosinophilic esophagitis in the United States" (Thel 2024, Clinical Gastroenterology and Hepatology). 8% CAGR from 2019-2024; expected to continue through 2030.

ANB033, a potential best-in-class CD122 antagonist, has pipeline-in-a-product potential




Corporate
Overview
October 2025
AnaptysBio

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 the release of data from the Company's clinical trials, including initial data for rosnilimab's Phase 2 clinical trial in ulcerative colitis and ANB033's Phase 1b clinical trial in celiac disease; expectations regarding the structure, infrastructure, timing and taxation of the proposed separation of companies; timing of paydown of financial obligations to Sagard; timing of initiation of Phase 1b clinical trial in second indication with ANB033; timing of initiation of potential Phase 2 clinical trials with rosnilimab in additional indications; whether any partnership with rosnilimab will take place; the potential to receive any royalties or milestone payments from the Vanda Pharmaceuticals license agreement; whether any of the Company's product candidates will be best in class or optimized; the potential to receive any additional milestones or royalties from the GSK collaboration and timing therefor; 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.

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Intention to separate into two independent, publicly traded companies to unlock and maximize value

Biopharma Co

Focus on developing and potentially commercializing therapeutics for autoimmune diseases

Rosnilimab	ANB033	ANB101
(Pathogenic T cell depleter)	(CD122 antagonist)	(BDCA2 modulator)
P2b complete in Rheumatoid Arthritis	P1b in Celiac Disease (CeD)	P1 in Healthy Volunteers
P2 in Ulcerative Colitis		

Research-driven • R&D capabilities with preclinical pipeline of immunology targets

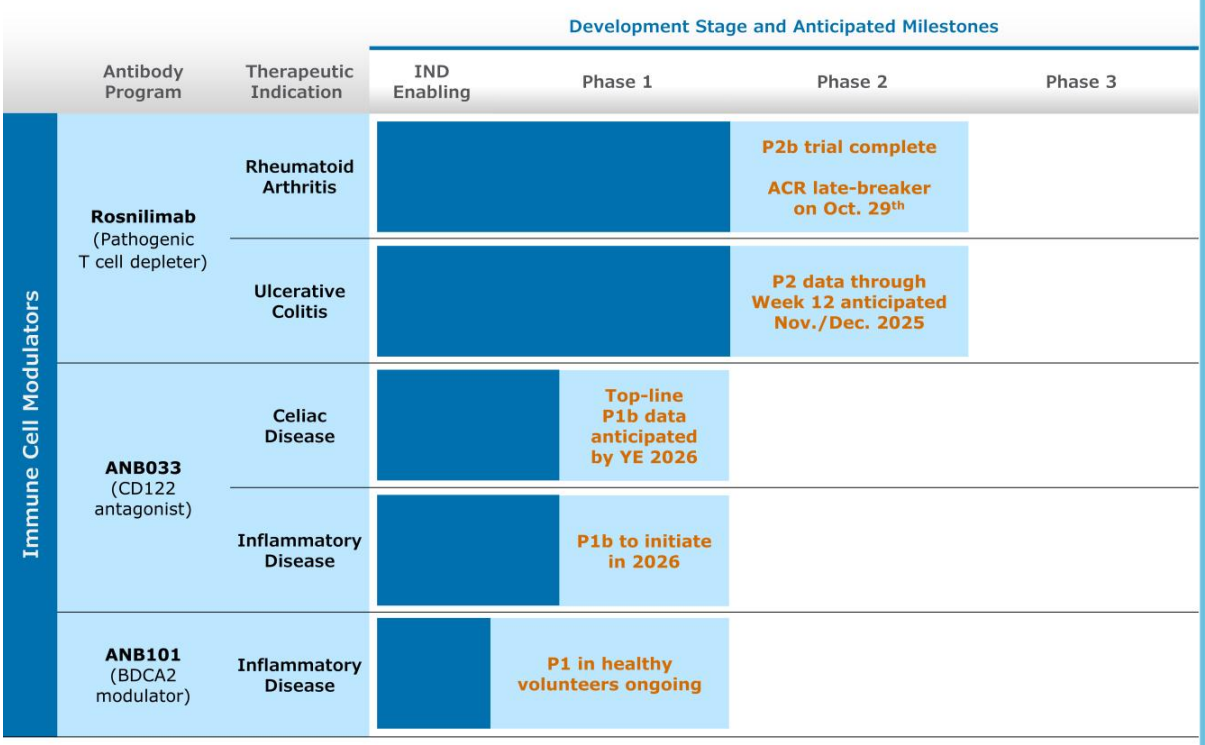
Royalty Management Co

Focus on protecting and returning value of the royalties to shareholders

- Hold and continue to manage rights to
  - Potential substantial *Jemperli* royalties from GSK
  - Imsidolimab milestones and royalties from Vanda
- Expect minimal infrastructure and staff
- Anticipate will retain Anaptys' net operating loss (NOL) carryforwards

Note: Q2 2025 cash: ~$294MM. Anaptys expected cash runway through YE: 2027 includes GSK $75MM milestone for *Jemperli* $1B annual WW sales. Biopharma Co. to launch with adequate capital to fund operations for at least two years through significant potential corporate milestones. Cash runway excludes significant royalty potential from GSK or Vanda.

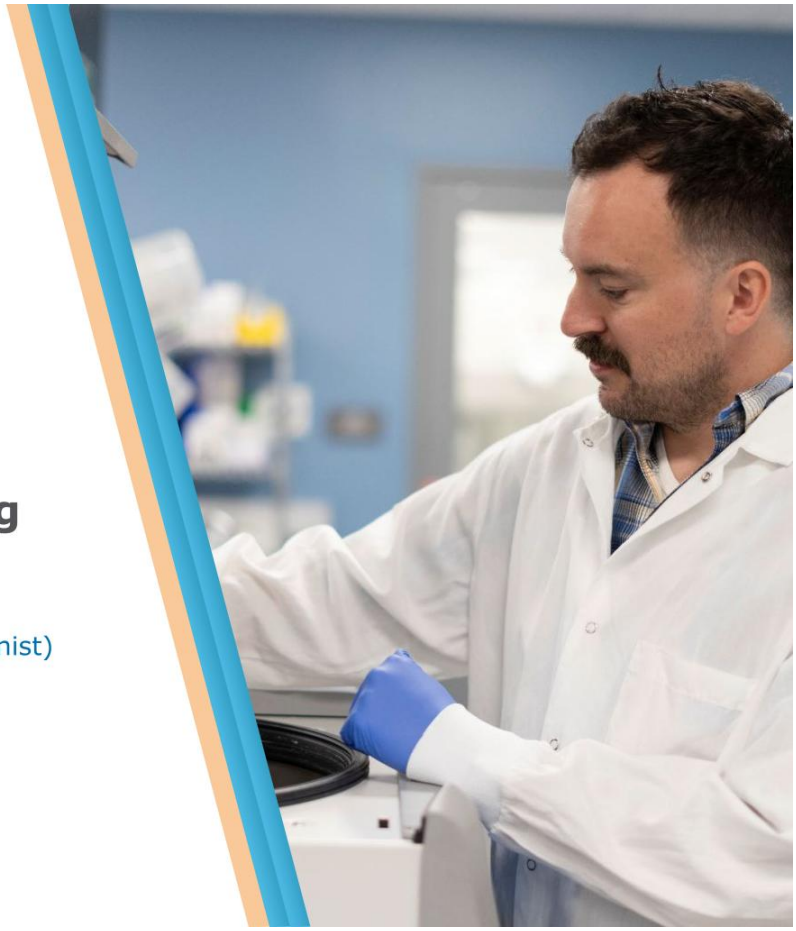
Biopharma Co would retain leading pipeline of immune cell modulating antibodies with significant upcoming catalysts



Royalty-Bearing Assets

Jemperli™
(dostarlimab, PD-1 Antagonist)

Imsidolimab
(IL-36R antagonist)



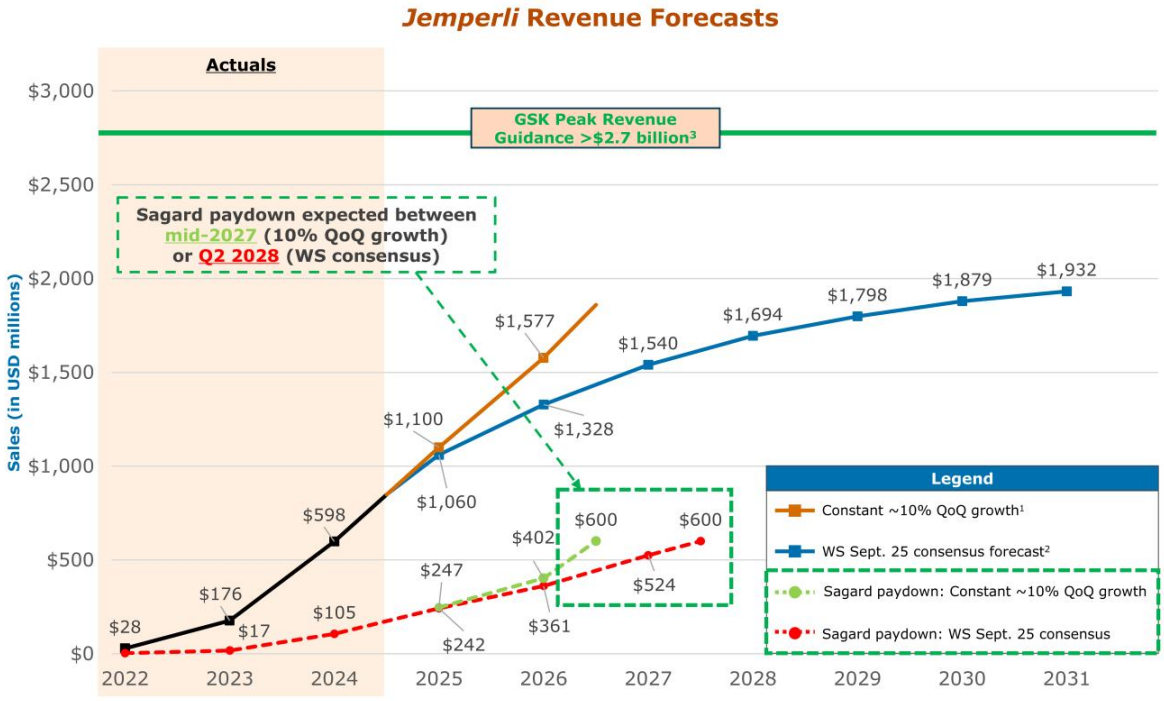
Royalty Management Co would protect and return value to shareholders

Jemperli: GSK Financial Collaboration	Imsidolimab: Vanda Financial Collaboration

• Q2 2025 sales: $262m (>19% US QoQ growth rate)
  ○ >$1b annualized run rate[1]
• Significant royalties on global net sales
  ○ 8% ($0 to $1b), 12% ($1 - $1.5b), 20% ($1.5 - $2.5b), and 25% (>$2.5b)
• Anticipate Sagard paydown between mid-2027 and Q2 2028 projected from *Jemperli's* continued strong growth rate
• Substantial ongoing investment in additional indications for *Jemperli* monotherapy and combos
  • H2 2026: top-line data from registrational dMMR rectal trial
	• 10% royalty on global net sales
	• $35 million in future milestones
	  ○ $5 million – FDA approval in GPP
	  ○ $5 million – EMA approval in GPP
	  ○ $25 million – $100 million annual sales milestone
	• FDA BLA submission for GPP expected in 2025

1. GSK Q2 2025 earnings presentation, US dollar conversion

Jemperli on a steep growth trajectory with GSK guiding to greater than £2 billion ($2.7 billion) peak monotherapy sales



1. Actual *Jemperli* Q1 to Q2'25 QoQ growth was 19%, Forecast assumes constant ~10% QoQ sales growth from Q2'25 through Q2'27 and dMMR rectal approval; 2. GSK analyst consensus as of 9/15/2025 converted from GBP to USD using Q3 2025 average exchange rate (1.35x), GSK Analyst Consensus website; 3. CEO Emma Walmsley, 2025 JP Morgan CEO Series fireside chat, 9/11/2025, *"there's no change to our peak year sales overall ambition for Jemperli, that's for sure, which is for more than £2 billion."*

GSK immuno-oncology financial collaboration

Jemperli (dostarlimab-gxly) Injection 500 mg *(PD-1 antagonist)*

Endometrial cancer (approved indications)

- **1L endometrial cancer:** Approved in US and EU for primary advanced or recurrent EC in combination with chemo
- **2L endometrial cancer:** Approved (monotherapy) in US and EU for dMMR/MSI-H recurrent or advanced EC after progressing on a platinum-containing regimen
- Significant U.S. market opportunity with 23,000 eligible diagnoses/year[1]

Colorectal cancer and dMMR pan tumors

- **MSI-H Pan Tumors:** Accelerated approval (monotherapy) in US for dMMR recurrent or advanced solid tumors that have progressed on or following prior treatment and who have no satisfactory alternative treatment options
- **Rectal cancer:** P2 AZUR-1 trial (monotherapy) in dMMR/MSI-H in locally advanced [LA] rectal cancer
  - Registrational, fully enrolled, with top-line data in H2 2026
- **Colon cancer:**
  - P3 AZUR-2 registrational, trial (monotherapy vs SoC adjuvant chemo) perioperative in patients with high-risk early-stage dMMR/MSI-H cancer
  - P2 AZUR-4 trial (dostarlimab + chemo combination) in neoadjuvant MMRp/MSS cancer

Head & Neck squamous cell carcinoma

- **LA-HNSCC:** P3 JADE registrational trial (monotherapy) sequentially after chemoradiation
  - Significant U.S. market opportunity with 54,000 eligible diagnoses/year[1]

Additional combination studies and comparative data

Liver cancer (1L HCC): P1 AMBER Cohort F trial (dostarlimab + cobolimab)

ADC combination opportunities

Head-to-Head vs. Keytruda: P2 PERLA trial (46% cORR for dostarlimab + chemo vs. 37% cORR for pembrolizumab + chemo, HR 0.70)
- *Not for registration*; data reported in December 2022

1. NCI SEER data

Potential royalties and milestones to Anaptys from GSK immuno-oncology financial collaboration

Jemperli (dostarlimab-gxly) injection 500 mg
(PD-1 antagonist)

Royalty rate (annual WW net sales)	8% - $0 to $1 billion 12% - $1.0 to $1.5 billion 20% - $1.5 to $2.5 billion 25% - >$2.5 billion
Remaining retained milestones	$75MM when annual net sales ≥ $1 billion[1]

Sagard ***"Jemperli* – only" capped non-recourse monetization**

- *Jemperli* receivables payable to Sagard until cumulative $600MM paydown by Mar. 31, 2031[1,2]
- Anticipate ~$250MM accrued to Sagard by YE 2025
- Projected cumulative $600MM paydown mid-2027 and Q2 2028[3]

1. The $75MM commercial milestone is excluded from Sagard monetization. The following *Jemperli* milestones are also still potentially payable from GSK but contribute to Sagard paydown: $15MM on regulatory approvals and $50MM on annual net sales of $750MM.

2. If cumulative $600MM not paid to Sagard by Mar. 31, 2031, the cumulative paydown increases to $675MM.

3. Forecast assumes constant ~10% QoQ sales growth from Q2'25 through Q2'27 and dMMR rectal approval and Q2 2028 derived from GSK analyst consensus as of 9/15/2025 converted to USD (1.35x conversion rate), GSK website - https://www.gsk.com/en-gb/investors/analyst-consensus/

Note: Anaptys' capped non-recourse monetizations resulted in $300MM of non-dilutive capital, including $250MM in Oct. 2021 and $50MM in May 2024.

Note: Separate sale of Anaptys' *Zejula* (niraparib) royalty interest occurred in September 2022 to DRI Healthcare Trust for $35MM upfront + $10MM potential milestone upon FDA approval of *Zejula* for the treatment of endometrial cancer, to the extent that such approval occurs on or before 12/31/25. At present, the *Jemperli* plus *Zejula* combination demonstrated significantly improved PFS in primary advanced or recurrent endometrial cancer in the RUBY Phase III trial.

Imsidolimab (IL-36R antagonist) out-licensed to Vanda

Key financial terms to Anaptys

VANDA PHARMACEUTICALS INC.

Exclusive global license to Vanda
announced February 2025

$15 million upfront payment
$10 million upfront and $5 million for existing drug supply

$35 million future milestones
$5 million – FDA approval in GPP
$5 million – EMA approval in GPP
$25 million – Achievement of $100 million WW annual net sales

10% royalties on global net sales

FDA BLA submission for generalized pustular psoriasis (GPP) expected in 2025[1]

Imsidolimab: two positive global Phase 3 studies in GPP

1. Vanda Q2 2025 earnings release/10-Q

Rosnilimab

(Pathogenic T Cell Depleter)

RA and UC in the U.S. are complementary, large commercial markets with similarly high unmet need

Treaters seek to maximize and sustain remission in maintenance phases

RA: U.S. Market

~$10bn
U.S. sales in b/tsDMARD-experienced market[1]

~500k
U.S. b/tsDMARD patients; 20-25% cycle through all classes of therapy[2]

UC: U.S. Market

$8bn ➔ >$10bn
U.S. sales in advanced therapy by 2030[3]

~300k and growing
U.S. advanced therapy patients; Cycling ramping up as advanced therapy market becomes established to avoid surgeries[3]

- Mature, non-growth market
- Established biosimilar 1L SoC (e.g. TNF)
- 2L access possible, but requires contracting
- No new MoA in the last decade

Similarities:

- Focus on achieving low disease activity or remissions long-term
- 1/3 to 1/2 relapse within 1 year following initial response at 3-6 months on induction therapy[4]
- 2L+ markets with blockbuster opportunity
- Minimal differentiation between biologics
- Assess combinations to enhance speed of induction and/or max remission rates
- **Substantial unmet need for new classes of safe, effective and durable therapies**

- Dynamic, growing market
- 1L SoC diversified with growth from branded products (IL-23p19, α4β7)
- Minimal payor management of 2L+ therapies

1. Expected by 2028 (Evaluate 29 Nov 2022); 2. Market research conducted by Ambit in 2022; 3. Market research conducted by ZS Associates in 2025; 4. Phase 3 registrational data from product labels

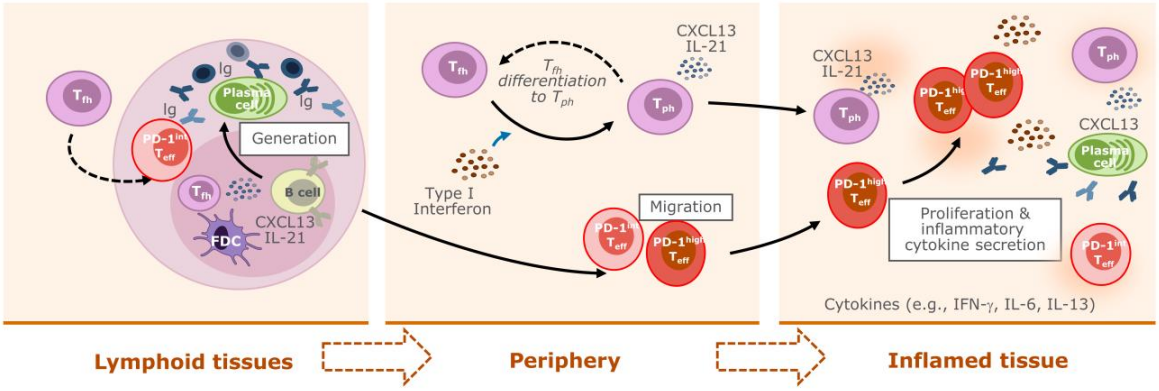
Pathogenic T_{eff} and T_{fh}/T_{ph} cells mediate autoimmune pathology

T_{fh} (follicular helper)
T_{ph} (peripheral helper)

- Secrete CXCL13 and IL-21 which recruit and mature B cells into "autoantibody secreting" plasma cells
- Depletion results in downstream effect on B cells, plasma cell generation and autoantibody levels

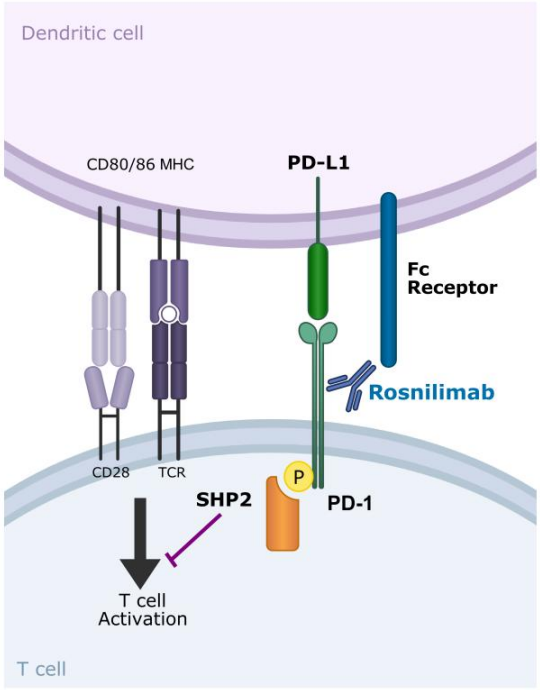
T_{eff} (effector)

- In response to stimulation, become highly activated
- Secrete inflammatory cytokines, cause tissue damage and perpetuate inflammatory cycle
- Depletion results in reduced T cell proliferation, T cell migration and cytokine secretion



Adapted from Akiyama et al, Ann Rheum Dis, 2023.

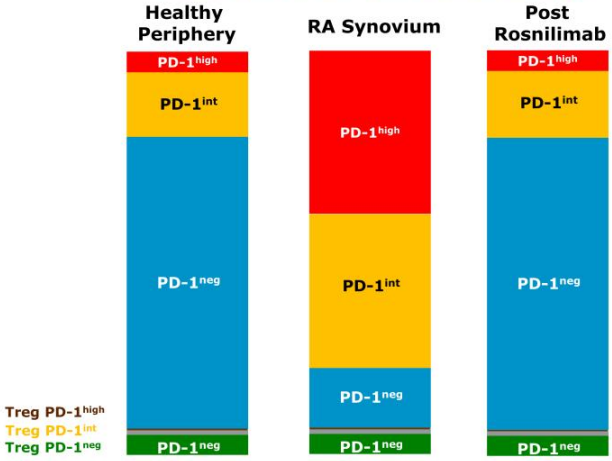
Rosnilimab selectively targets pathogenic T cells in periphery and inflamed tissue to restore immune homeostasis



Rosnilimab aims to:

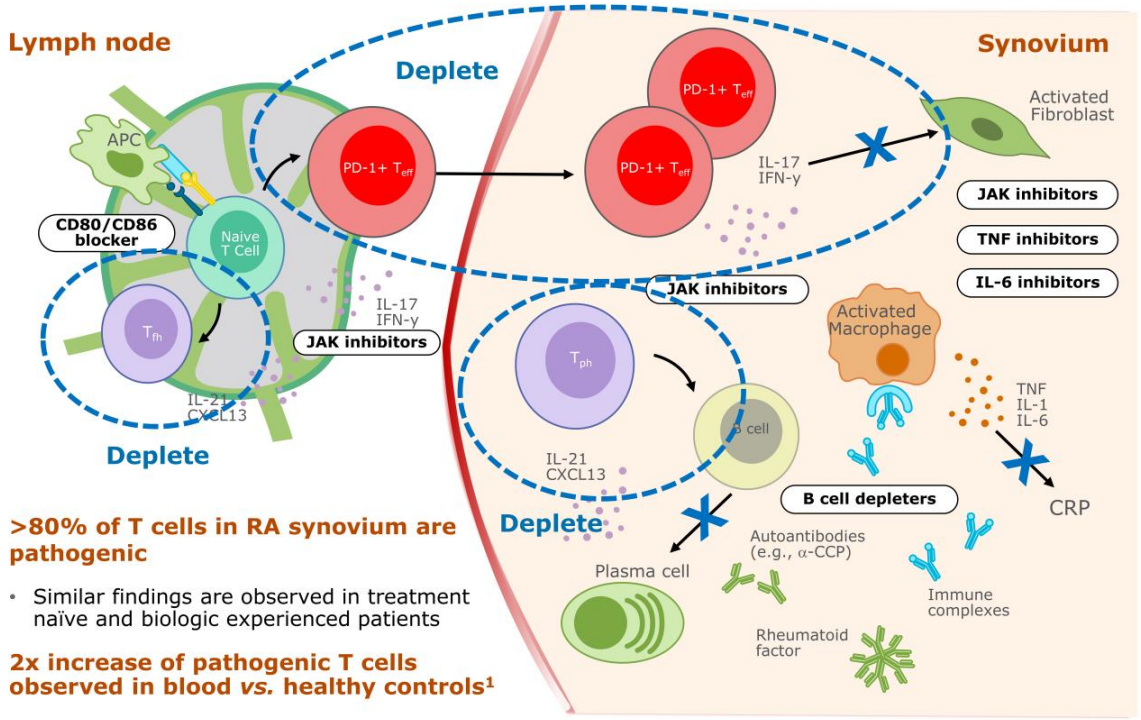
1. Leverage natural immune regulatory pathway to safely restore immune homeostasis
2. Achieve durable remission and modify disease

Illustrative T cell composition change



Effector T cells (T_{eff}): activated T cells (cytotoxic, helper, Treg); Follicular/Peripheral Helper T cells (T_{fh}, T_{ph}): support B cell differentiation and maturation.

Depleting pathogenic T cells broadly impacts multiple downstream, clinically validated drivers of RA pathogenesis



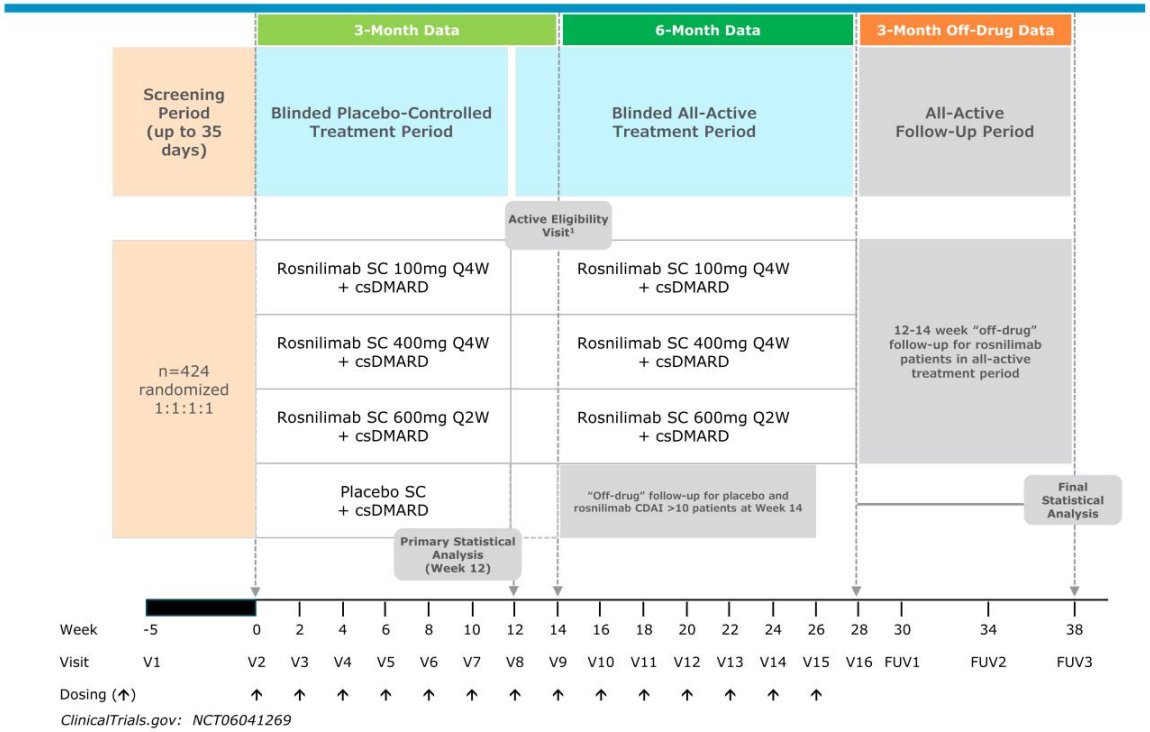
>80% of T cells in RA synovium are pathogenic

- Similar findings are observed in treatment naïve and biologic experienced patients

2x increase of pathogenic T cells observed in blood *vs.* healthy controls[1]

Adapted from Aletaha and Smolen, JAMA, 2018; 1. Chen et al, Clinical and Translational Immunology, 2024.

Rosnilimab Phase 2b trial in RA



ClinicalTrials.gov: NCT06041269

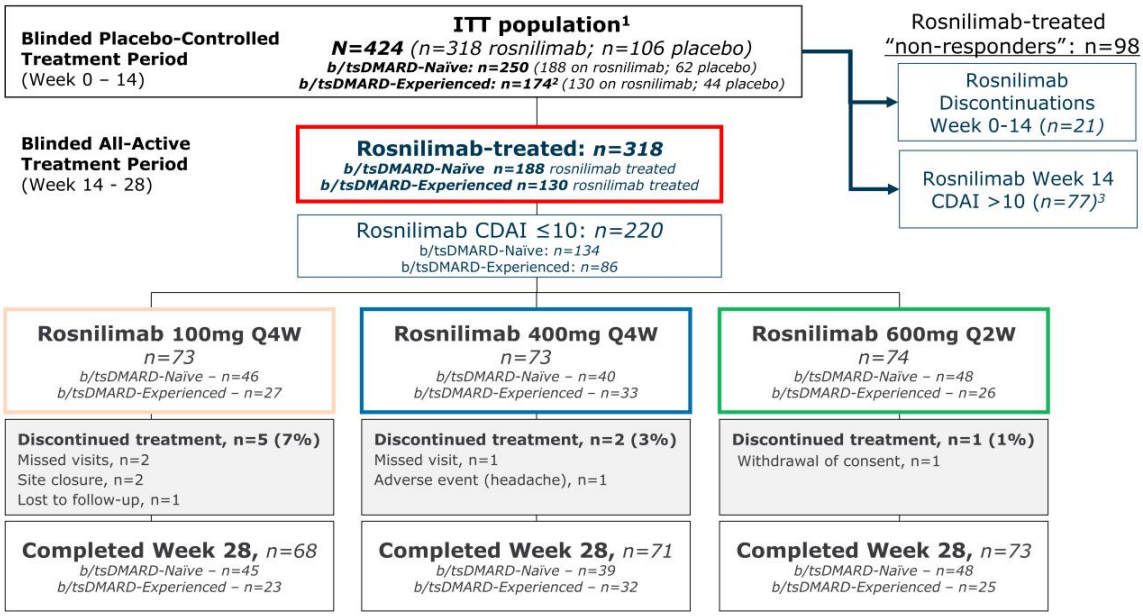
Note: All patients in trial (rosnilimab and placebo arms) are required to be on stable background csDMARD

1. Blinded study drug treatment continued for active treatment group subjects that achieved Clinical Disease Activity Index (CDAI) low disease activity (≤10)

95% completed 6-month all-active treatment period supporting rosnilimab's favorable efficacy and tolerability profile

Discontinuations
All-active treatment period (Week 14 - 28)

- ✓ 7 (of 8 total) discontinuations in all active treatment period were still in CDAI LDA at time of discontinuation
- ✓ No discontinuations due to disease progression
- ✓ Only 1 discontinuation due to AE (headache - moderate)



1. Non-responder imputated (NRI) analysis on intent-to-treat (ITT) population; b/tsDMARD-naïve population (n=62 placebo, n=62 100mg Q4W, n=62 400mg Q4W, n=64 600mg Q2W); b/tsDMARD-experienced population (n=44 placebo, n=44 100mg Q4W, n=45 400mg Q4W, n=41 600mg Q2W); 2. b/tsDMARD-experienced population included 50 patients (29% of n=174 total b/tsDMARD-experienced patients) with prior JAK experience; 3. Patients assessed at Week 14, dosed, and returned for follow-up visit at Week 18.

Rosnilimab demonstrates best-in-disease profile in RA

1

Best-in-disease profile through 6 months

- JAK-like efficacy in both 3-month placebo-controlled portion and through 6 months
- Favorable safety and tolerability, particularly when compared to standard of care
- Monthly (Q4W) dosing

2

Max response rates have not yet been observed

- Strict continuation criteria prevented patients with improvement at 3 months from continuing in this P2b trial
- Many patients beyond 3 months achieved, or were trending toward, CDAI LDA and ACR50

3

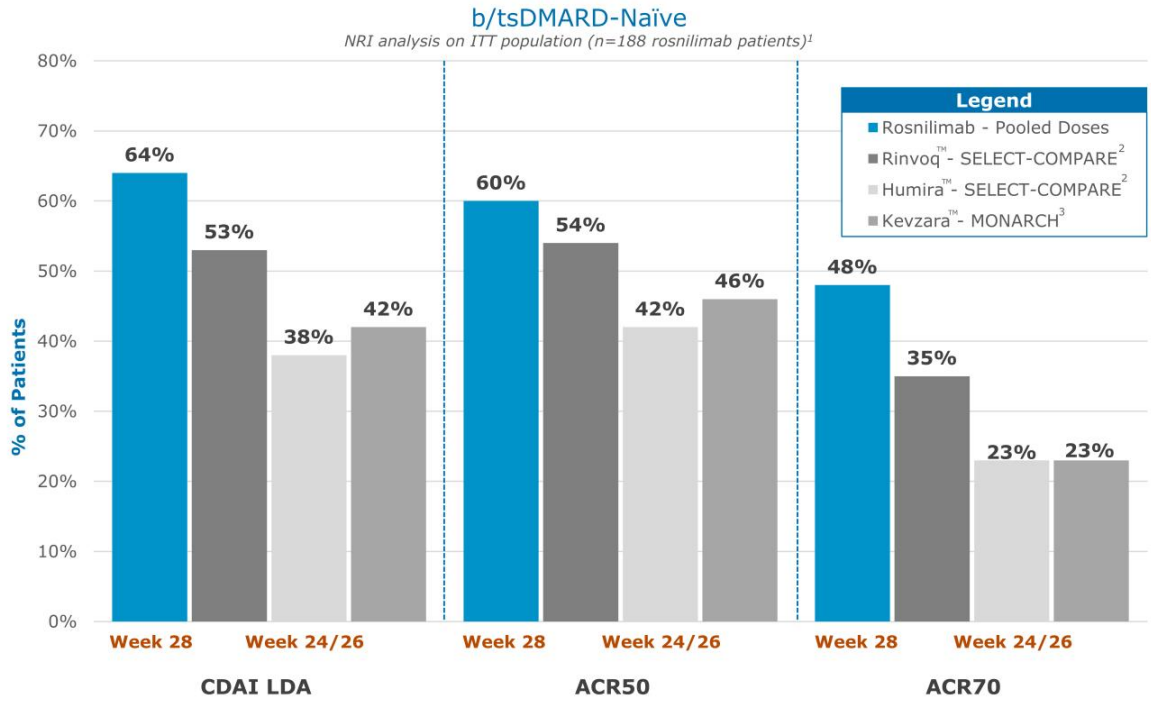
Responses durable after 6 months

- Potential for maintenance dosing with extended dosing intervals (e.g. Q8W or Q12W)

Rosnilimab, a pathogenic T cell depleter, is well-positioned for the ~$20 billion U.S. RA market which hasn't had a new mechanism approved since 2012

Rosnilimab shows JAK-like efficacy in naïve patients

Compares favorably despite most conservative analysis and capped trial design

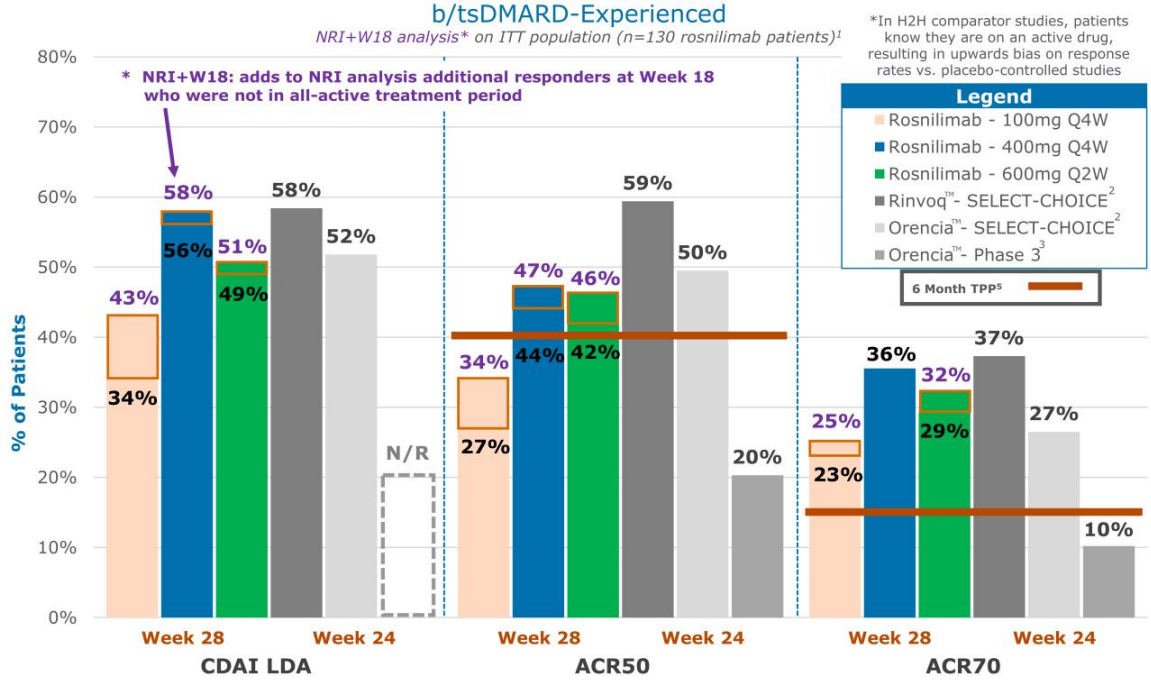


1. Non-responder imputated (NRI) analysis on intent-to-treat (ITT) of all b/tsDMARD-naïve patients randomized; b/tsDMARD-naïve population (n=62 100mg Q4W, n=62 400mg Q4W, n=64 600mg Q2W; n=188 total rosnilimab b/tsDMARD-naïve patients); 2. SELECT-COMPARE Phase 3 study; 3. Kevzara Phase 3 study; NRI data; CDAI = Clinical Diseases Activity Index; LDA = Low Disease Activity; N/R = Not Reported

Rosnilimab surpassed TPP in experienced patients and comparable at mid/high dose to JAKs in all-active H2H study*

Includes 29% with prior JAK experience

Excludes 7 patients who discontinued in all-active treatment period while in CDAI LDA

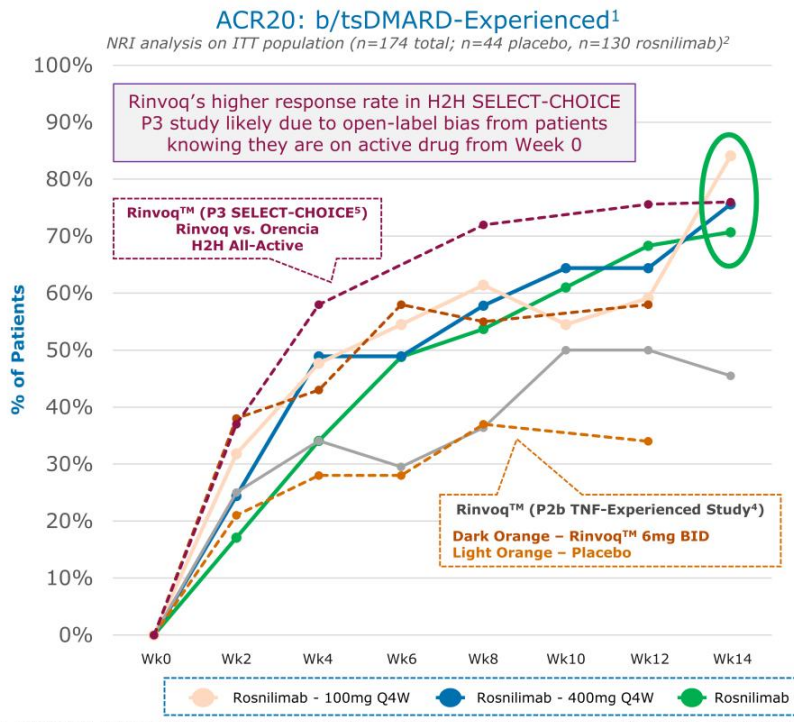


1. Non-responder imputated (NRI) analysis on intent-to-treat (ITT) of all b/tsDMARD-experienced patients randomized; b/tsDMARD-experienced population (n=44 100mg Q4W, n=45 400mg Q4W, n=41 600mg Q2W; n=130 total rosnilimab b/tsDMARD-experienced patients); 2. SELECT-CHOICE Phase 3 study; 3. Orencia Phase 3 study; NRI data; 4. Anaptys Jan. 2025 Target Product Profile (TPP);

CDAI = Clinical Diseases Activity Index; LDA = Low Disease Activity; N/R = Not Reported

ACR20 response rates are comparable to Rinvoq™

Most patients had symptomatic and clinical improvement by 3 months



ACR20 at Week 12		
Arm	**Absolute**	**PBO Adjusted**
b/tsDMARD-Experienced Population *(as graphed)*		
100mg	59%	*9%*
400mg	64%	*14%*
600mg	68%	*18%*
Rinvoq[4]	58%	*24%*
Rinvoq[5]	76%	*N/A*
b/tsDMARD-Naïve Population *(for reference)*		
100mg	76%	*21%*
400mg	74%	*19%*
600mg	80%	*25%*
Rinvoq[3]	68%	*22%*

1. b/tsDMARD-experienced population included 29% (n=50 of n=174 total experienced patients) with prior JAK experience; 2. Non-responder imputated (NRI) analysis on intent-to-treat (ITT) of all b/tsDMARD-experienced patients randomized; b/tsDMARD-experienced population (n=44 placebo, n=44 100mg Q4W, n=45 400mg Q4W, n=41 600mg Q2W; n=130 total rosnilimab b/tsDMARD-experienced patients); 3. Rinvoq™ Phase 2b MTX-IR study; 4. Rinvoq™ Phase 2b TNF-experienced study; 6mg BID (equivalent to 15mg QD); 5. SELECT-CHOICE Phase 3 study

CRP reductions are comparable to Rinvoq™

Mean Change in CRP: b/tsDMARD-Experienced[1]

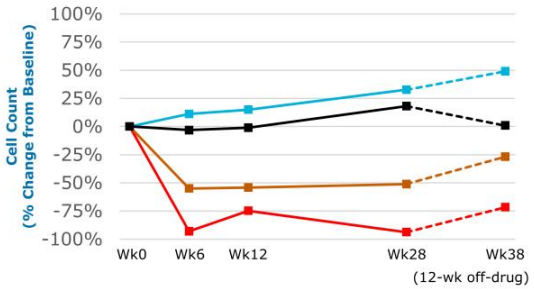
MMRM Analysis on ITT population (n=174 total; n=44 placebo, n=130 rosnilimab)

Change in CRP at Week 12		
Arm	**Baseline Mean CRP**	**PBO Adjusted**
b/tsDMARD-Experienced Population *(as graphed)*		
100mg	20.0	*-6.7*
400mg	29.4	*-12.8*
600mg	23.3	*-8.0*
Rinvoq[4]	16.0	*-9.3*
Rinvoq[5]	19.0	*N/A*
b/tsDMARD-Naïve Population *(for reference)*		
100mg	14.9	*-10.6*
400mg	14.3	*-7.0*
600mg	15.7	*-6.7*
Rinvoq[3]	17.0	*-8.4*

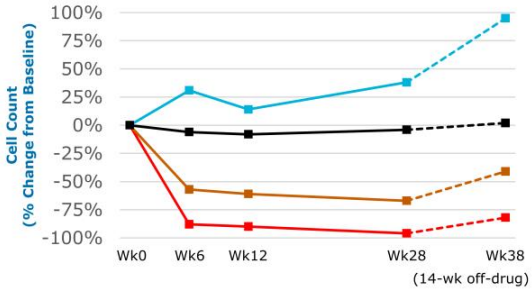
1. b/tsDMARD-experienced population included 29% (n=50 of n=174 total experienced patients) with prior JAK experience; 2. Mixed Model for Repeated Measures (MMRM) analysis on intent-to-treat (ITT) of all b/tsDMARD-experienced patients randomized; b/tsDMARD-experienced population (n=44 placebo, n=44 100mg Q4W, n=45 400mg Q4W, n=41 600mg Q2W); 3. Rinvoq™ Phase 2b MTX-IR study; 4. Rinvoq™ Phase 2b TNF-experienced study; 6mg BID (equivalent to 15mg QD) 5. SELECT-CHOICE Phase 3 study

Deep, sustained reduction of pathogenic T cells led to favorable T cell composition reflective of immune homeostasis and durable response

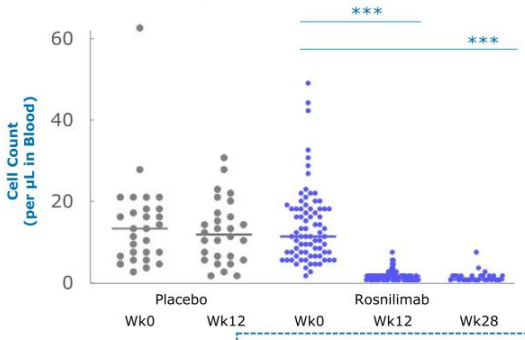
Rosnilimab 400mg Q4W T Cell Impact



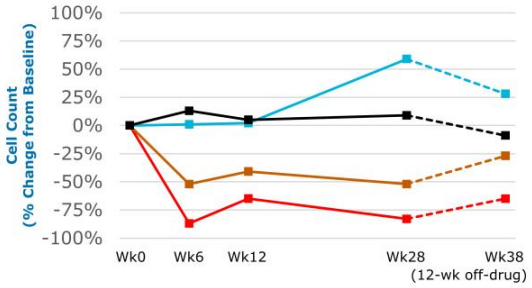
Rosnilimab 600mg Q2W T Cell Impact



Rosnilimab T_{ph} Impact – Pooled Doses

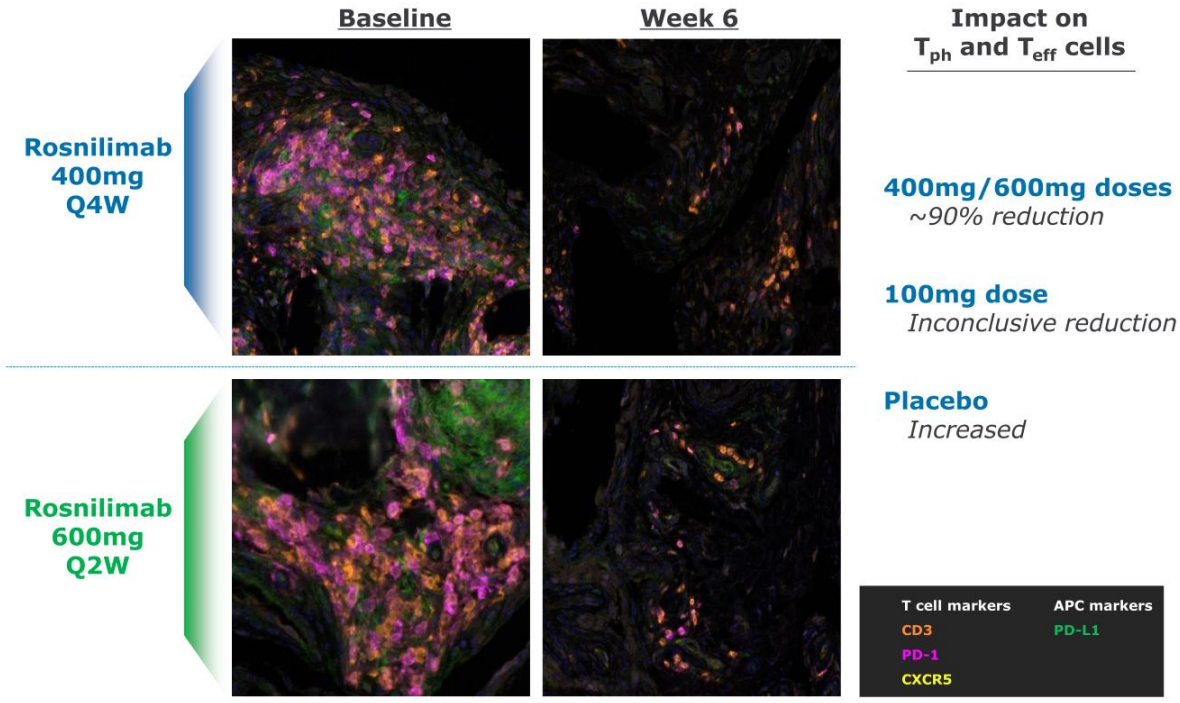


Rosnilimab 100mg Q4W T Cell Impact



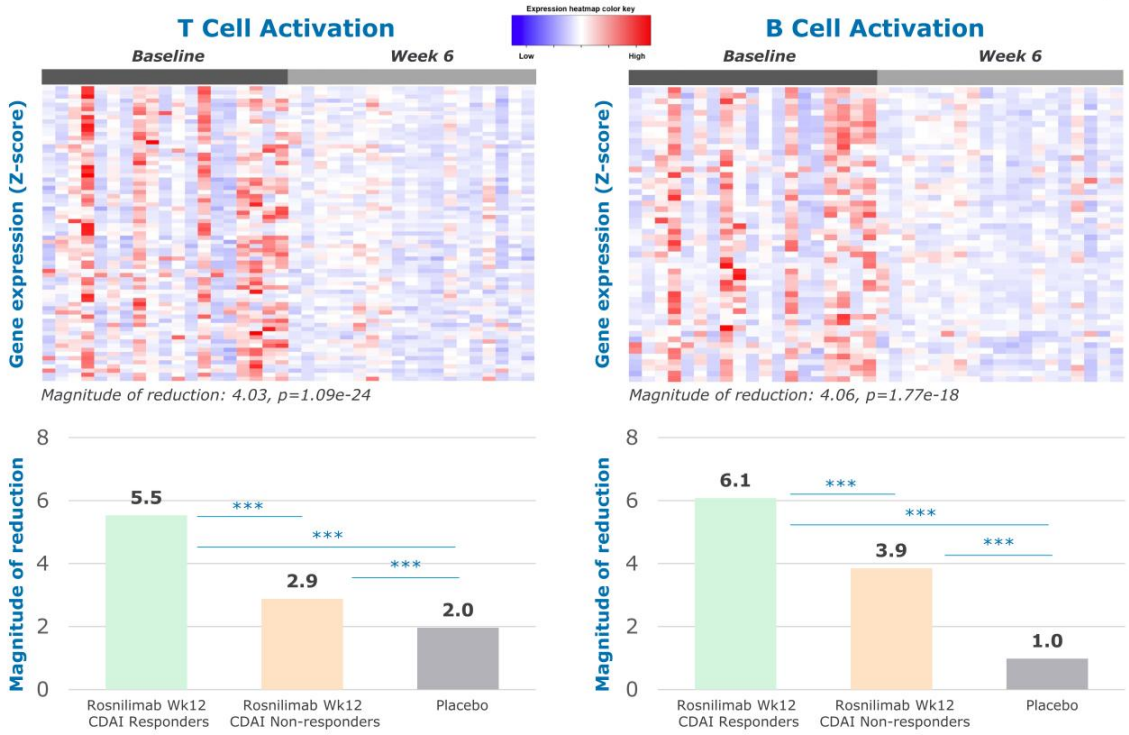
Note: data representative sample of ~50% of ITT population; T_{ph} – T peripheral helper cell defined as CD3+ CD4+ CD45RA- PD-1^{high} CXCR5-, ***p<0.001

Synovial biopsies show ~90% reduction of pathogenic T cells in the target issue



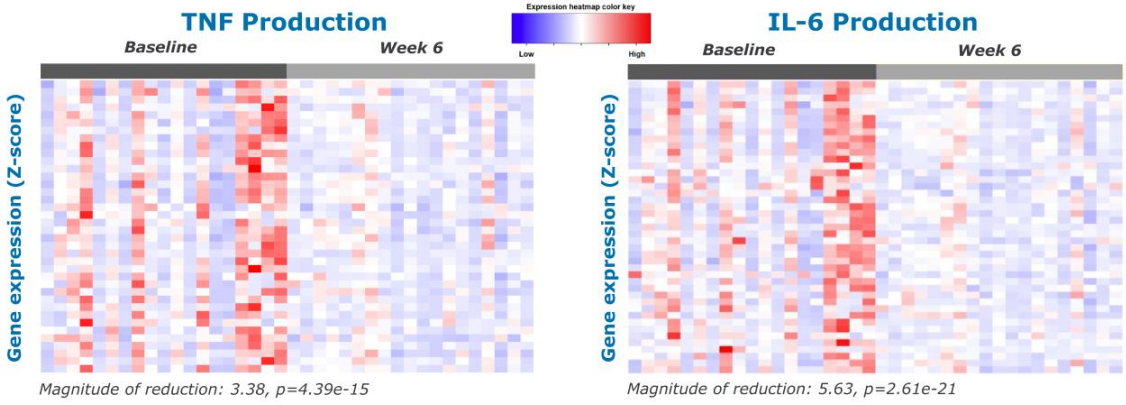
Note: Synovial biopsies of the most impacted joint taken at baseline and 6 weeks on study. Immunofluorescence performed to identify PD-1 positive cells. T_{ph} cells (PD-1+CD3+CD4+CXCR5-)

Significant reduction of T and B cell activation demonstrate on target pharmacology within the synovium



Note: Gene ontology (GO) pathway analysis performed on samples with evidence of inflammation at baseline (all rosnilimab doses pooled, n=19 paired biopsies) and with myosin normalization. Rows reflect genes with p<0.05 between Weeks 6 and 0. Magnitude of reduction defined as fold enrichment score. Rosnilimab responders achieved CDAI LDA in 3 months. ***p<0.001 for difference in fold change between baseline and Week 6 between groups.

Significant reduction of additional downstream pathways including TNF and IL-6 within the synovium



Magnitude of reduction: 3.38, p=4.39e-15

Magnitude of reduction: 5.63, p=2.61e-21

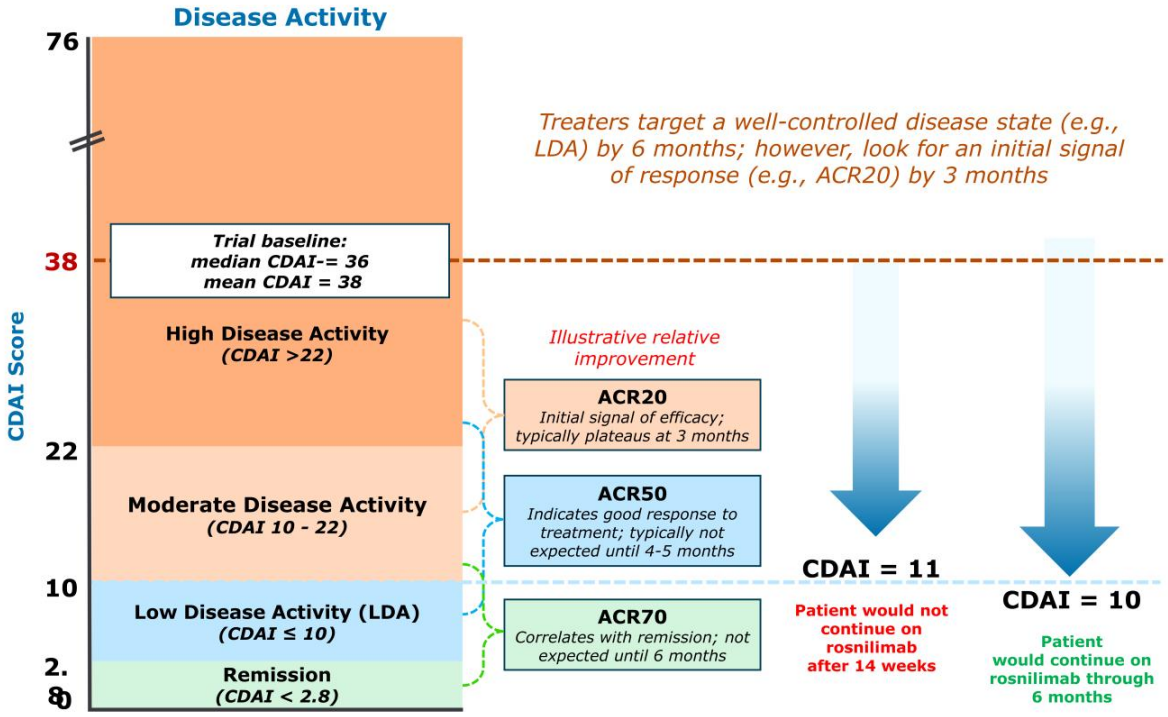
Pathway changes reflect rosnilimab's broad MOA

Significantly downregulated (p<0.05) genes of interest in RA:	**T cell activation:** IL2RA, TNFSF14 (LIGHT), CD28, CD69, CD40L, ICOS, CD226, ZAP70, TCF7, IRF1 **B cell activation:** IL7R, CD27, CD79A, BTK, SYK, IL21R **TNF and IL-6 production**: MYD88, PTPN22, LILRB1, LILRB2, NOD2, CCR2, NLRC3, IRAK3, IL1RAP, IL6R, IL17RA **Mediators of RA structural damage**: MMP1, MMP3, and RANK-L **IBD-related genes:** NOD2, TREM1, IL12RB, IFNGR1, S100A8

Note: Gene ontology (GO) pathway analysis performed on samples with evidence of inflammation at baseline (all rosnilimab doses pooled, n=19 paired biopsies) and with myosin normalization. Rows reflect genes with p<0.05 between Weeks 6 and 0. Magnitude of reduction defined as fold enrichment score.

LDA requirement at 14 weeks to continue on rosnilimab was a high bar for patients with baseline high disease activity

95% of trial participants had high disease activity (CDAI > 22) at baseline

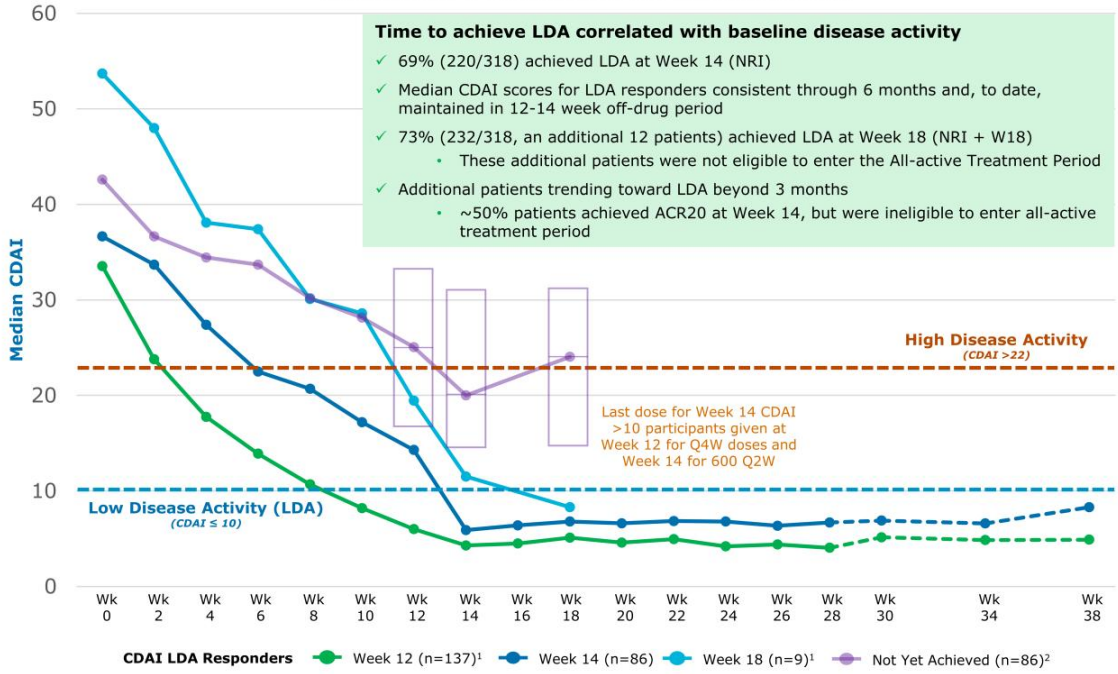


Max response was not achieved in this Phase 2b trial

On average, patients with higher disease activity take longer to achieve CDAI LDA

Median Change from Baseline in CDAI

NRI analysis on ITT population (n=318 rosnilimab patients)

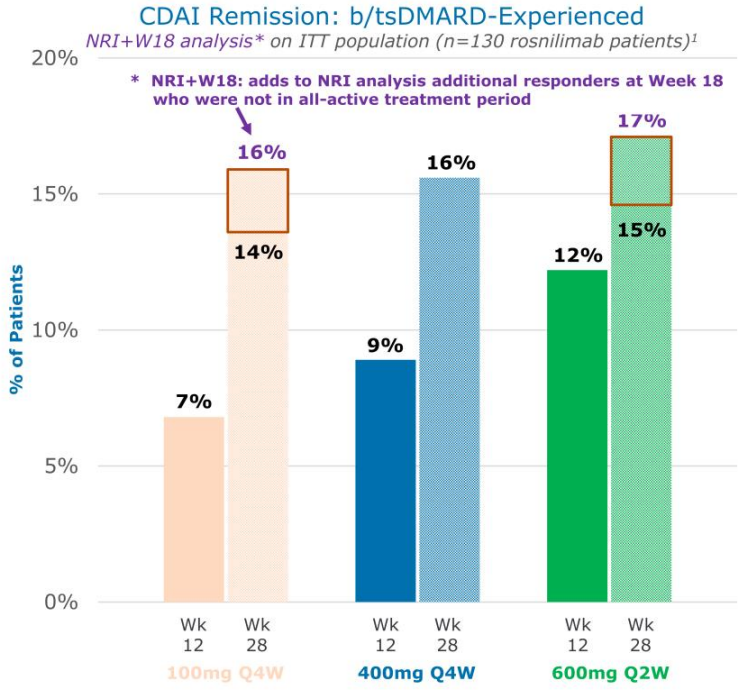


1. Green line includes 3 patients that achieved LDA at Week 12, were not CDAI LDA at Week 14, but returned to CDAI LDA at Week 18. These same 3 patients were excluded from the Light Blue line. In total 12 patients achieved CDAI LDA at Week 18. 2. Purple line includes rosnilimab patients that discontinued treatment before Week 14 (n=21). Purple box plot for "Not Yet Achieved" population for 25th percentile, median and 75th percentile values.

JAK-like CDAI remission rates which deepened into six months

Includes 29% with prior JAK experience

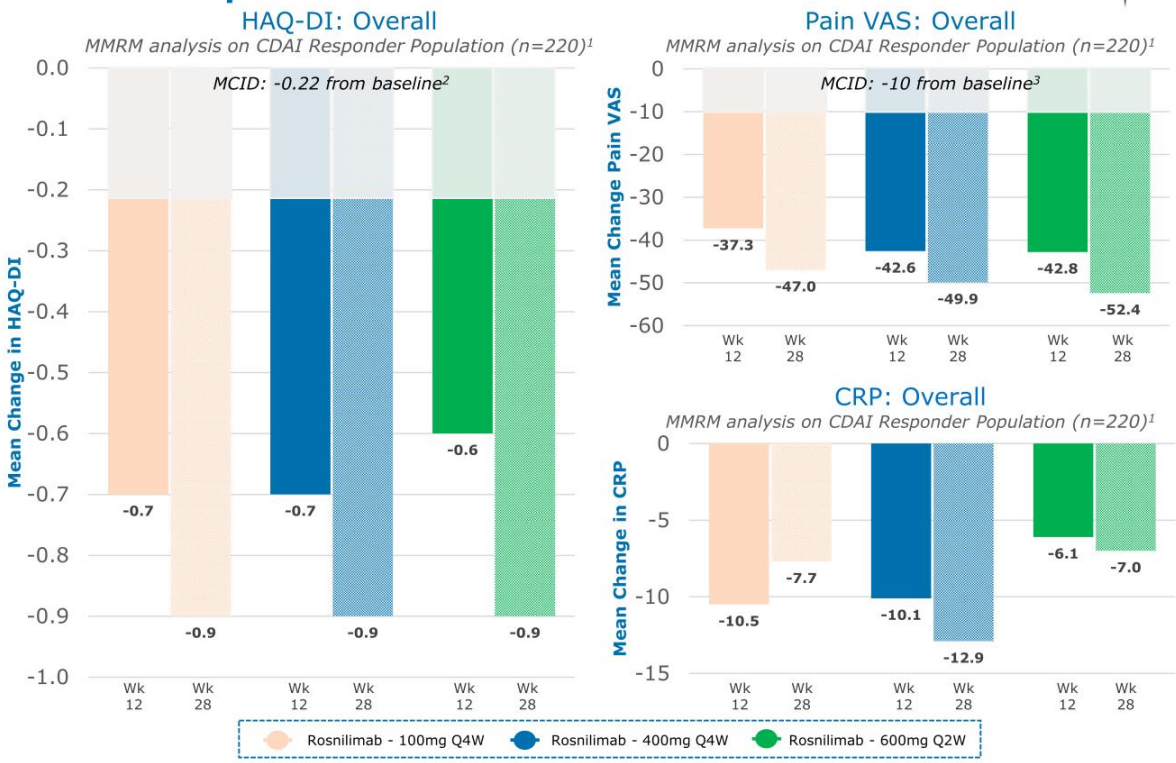
Excludes 2 patients who discontinued in the all-active treatment period while in CDAI remission



CDAI Remission at Week 28		
Arm	NRI	NRI+W18
b/tsDMARD-Experienced Population *(as graphed)*		
100mg	14%	16%
400mg	16%	16%
600mg	15%	17%
b/tsDMARD-Naïve Population		
100mg	21%	21%
400mg	18%	18%
600mg	17%	19%

1. Non-responder imputated (NRI) analysis on intent-to-treat (ITT) of all b/tsDMARD-experienced patients randomized; b/tsDMARD-experienced population (n=44 100mg Q4W, n=45 400mg Q4W, n=41 600mg Q2W; n=130 total rosnilimab b/tsDMARD-experienced patients)

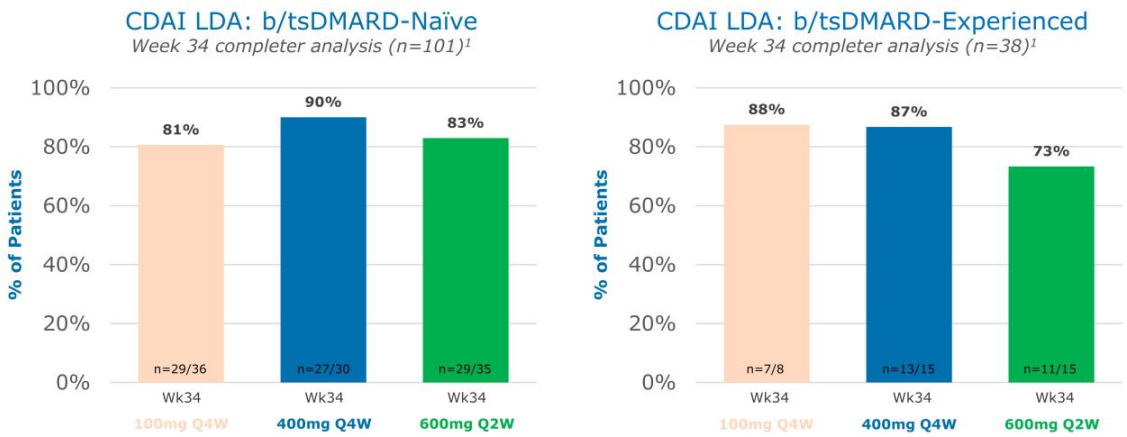
Highly meaningful clinically and symptomatic improvement across multiple PROs and CRP



1. Mixed Model for Repeated Measures (MMRM) analysis on rosnilimab CDAI LDA responder at Week 14 population (n=220) includes naïve population (n=46 100mg Q4W, n=40 400mg Q4W, n=48 600mg Q2W; n=134 total rosnilimab patients) and experienced population (n=27 100mg Q4W, n=33 400mg Q4W, n=26 600mg Q2W; n=86 total rosnilimab patients); 2. Behrens et. al, BMC Rheumatology, Dec. 2019; 3. Strand et. al, Journal of Rheumatology, Aug. 2011

Durable responses for at least 2-months off-drug

83% of Week 28 CDAI LDA responders were still in response at Week 34



Most patients who did not sustain CDAI LDA remained near the cutoff of CDAI=10

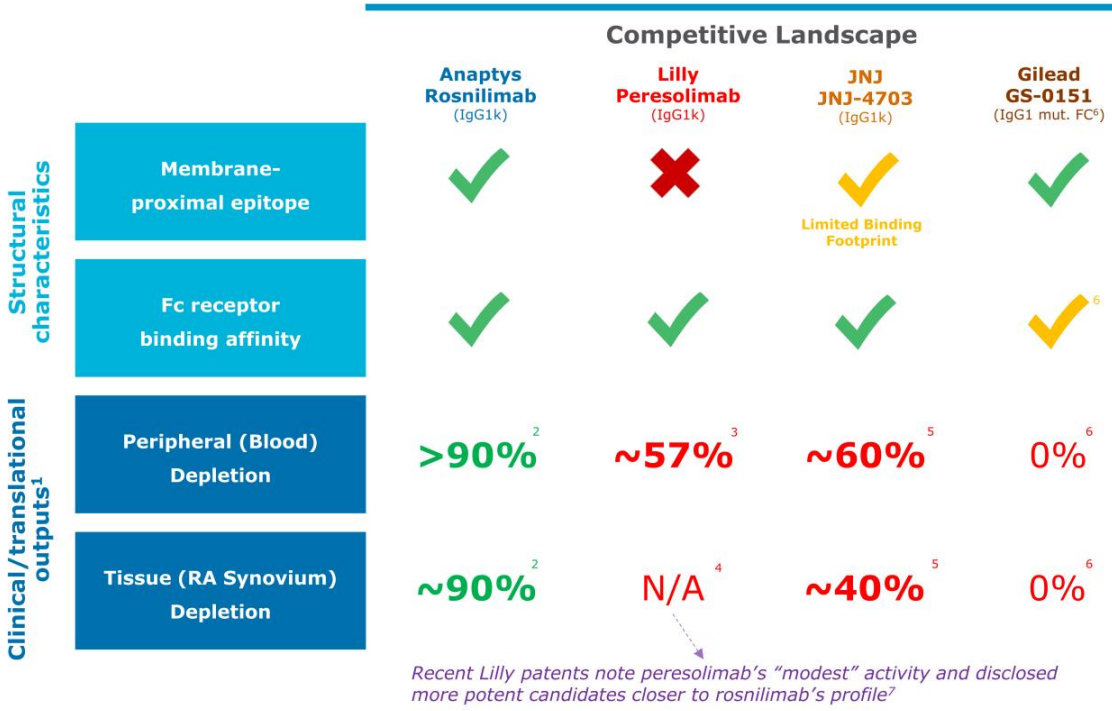
Only 23 of 139 (17%) patients of Week 28 CDAI LDA responders were not CDAI LDA (≤10) at Week 34:

- 25% (6/23) were CDAI <11
- Median CDAI = 13
- 91% (21/23) were CDAI <22 (e.g. remained CDAI moderate disease activity)

1. ~75% of patients who completed Week 28 (n=212) had reached Week 34 as of the March 11, 2025 data cutoff; this responder analysis represents patients who were in CDAI LDA, as of Week 28, relative to their CDAI status as of Week 34

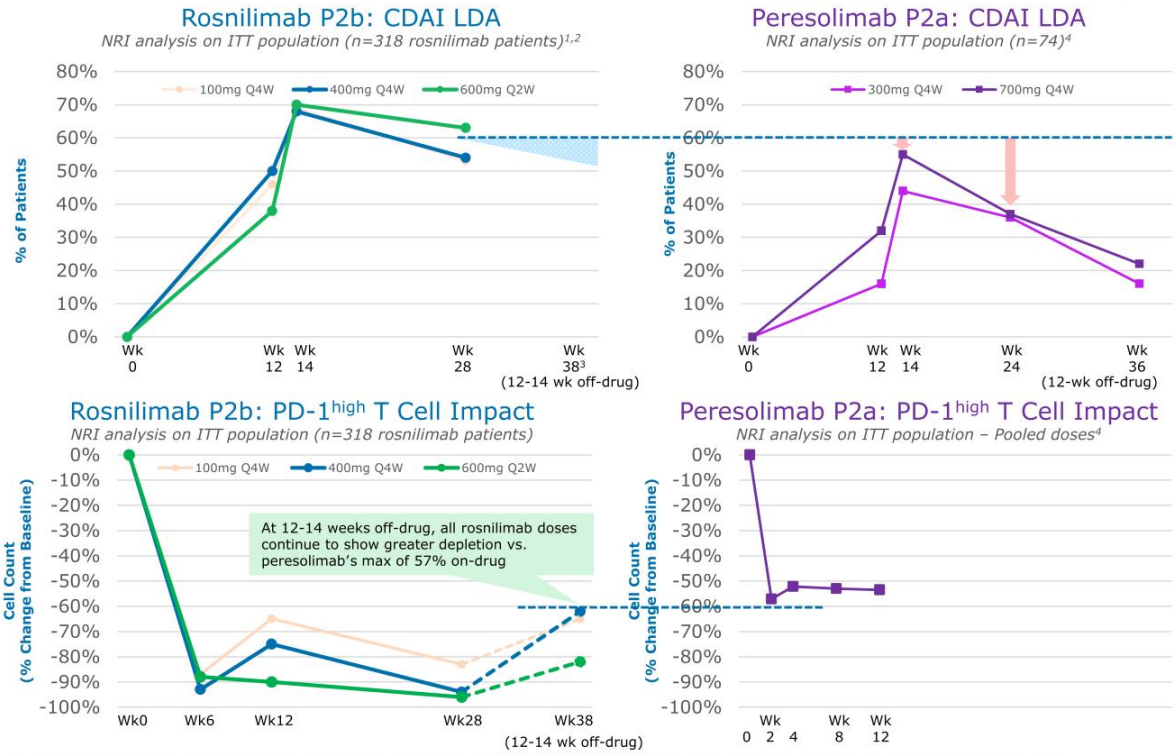
Rosnilimab is a best-in-class pathogenic T cell depleter

Competitors lack ability to potently deplete pathogenic T cells to restore immune homeostasis



1. From in-human Phase 1/2 clinical trials in RA; 2. Phase 2b RENIOR trial in RA for 400mg Q4W and 600mg Q2W doses; 3. Phase 2a trial in RA, Tuttle et. al, NEJM, May 2023, Supplemental Appendix;4. Not yet reported; 5. Phase 1b trial in RA, Ling et. al, EULAR 2025, June 2025; 6. Fc binding to FcγRIIb only, lacks any depletion activity; 7. Eli Lilly patents; WO2024196694A2 and WO2024040206A

LDA response rates and durability for rosnilimab are differentiated from Lilly's peresolimab



1. Non-responder imputated (NRI) analysis on intent-to-treat (ITT) of all 318 rosnilimab patients randomized; 2. At Week 28, 53% (100mg Q4W), 54% (400mg Q4W), and 63% (600mg Q2W) rosnilimab patients were in CDAI LDA (57% pooled); 3. Off-drug follow-up period ongoing; 4. Tuttle et. al, NEJM, May 2023, Supplemental Appendix, At Week 28, 36% (300mg Q4W) and 37% (700mg Q4W) peresolimab patients were in CDAI LDA

RA patients have significant co-morbidities which are further exacerbated with treatment

Increased co-morbidity rate in RA patients vs. general population

2x	2-3x	2x
Infection Rate[1]	DVT, PE, and MACE Risk[1,2]	Malignancy Rate[3]

Black box warnings for increasing SAE incidence of commercial products have not impeded blockbuster sales

HUMIRA® adalimumab	ORENCIA® (abatacept)	RINVOQ® upadacitinib	Rituxan® Rituximab
$4.5B RA sales[4]	**$3.6B RA sales[4]**	**$2.3B RA sales[4]**	**~$1B RA sales**
Black box warning		**Black box warning**	**Black box warning**
~30% infection rate vs. 28% placebo[5]	~54% infection rate vs. 48% placebo[5]	~20% infection rate vs. 18% placebo[5]	~39% infection rate vs. 34% placebo[5]
~0.7% MACE rate vs. 0.4% placebo[5]	~0.2% MACE rate vs. 0.5% placebo[5]	~3.4% MACE rate vs. 2.5% placebo[5]	~1.7% MACE rate vs. 1.3% placebo[5]
		~4.2% malignancy rate vs. 2.9% placebo[5]	

1. Avina-Zubieta et al., A&R, 2008, 2. Fazal et al., BMC Rheumatology, 2024, 3. Smitten et al., ART, 2008, 4. Evaluate Pharma 2023 WW RA sales, 5. Phase 3 registrational data from product labels.

Rosnilimab well tolerated with no safety signals

<2% dropout rate overall due to AEs through 6 months,
with only 1 dropout due to AE (headache-moderate) after 3 months

Study Period	Week 0 through Week 12 (N=424)				Week 0 through Week 28 (N=424)			
	Participants with Adverse Events, n (%)				Participants with Adverse Events, n (per 100 PY)*			
	Placebo (n=106)	100mg Q4W (n=106)	400mg Q4W (n=107)	600mg Q2W (n=105)	Placebo (n=106)	100mg Q4W (n=106)	400mg Q4W (n=107)	600mg Q2W (n=105)
Any AE	**36 (34%)**	**51 (48%)**	**48 (45%)**	**38 (36%)**	**39 (125.6)**	**73 (260.9)**	**66 (206.5)**	**52 (149.1)**
Any SAE[1]	1 (1%)	1 (1%)	1 (1%)	3 (3%)	1 (2.4)	2 (3.8)	2 (3.7)	4 (7.7)
Any Drug-Related SAE	1 (1%)	0 (0%)	0 (0%)	0 (0%)	1 (2.4)	0 (0)	0 (0)	0 (0)
Severe AE[2]	2 (2%)	1 (1%)	0 (0%)	4 (4%)	2 (4.8)	4 (7.5)	1 (1.9)	4 (7.8)
Drug-Related AE	18 (17%)	13 (12%)	18 (17%)	17 (16%)	18 (48.8)	17 (36.1)	28 (62.0)	19 (41.7)
AE Leading to Treatment Discontinuation	1 (1%)	1 (1%)	2 (2%)	2 (2%)	1 (2.4)	1 (1.9)	3 (5.6)	2 (3.8)
Infections	14 (13%)	24 (23%)	21 (20%)	12 (11%)	16 (41.5)	41 (98.7)	39 (89.4)	31 (67.6)
Serious	1 (1%)	1 (1%)	0	0	1 (2.4)	1 (1.9)	1 (1.9)	1 (1.9)
Opportunistic[3]	2 (1.9%)	0 (0%)	0 (0%)	0 (0%)	2 (4.8)	1 (1.9)	1 (1.8)	1 (1.9)
Participants with any AEs > 5%								
Headache	4 (4%)	7 (7%)	6 (6%)	4 (4%)	4 (9.7)	10 (19.9)	10 (19.4)	5 (9.8)
Upper respiratory tract infection	1 (1%)	7 (7%)	2 (2%)	3 (3%)	2 (4.8)	14 (27.8)	7 (13.4)	10 (19.6)
Nasopharyngitis	4 (4%)	5 (5%)	5 (5%)	0	4 (9.6)	9 (17.5)	8 (15.4)	1 (1.9)
Elevated ALT (alanine aminotransferase)[4]	1 (1%)	4 (4%)	3 (3%)	3 (3%)	1 (2.4)	8 (15.5)	5 (9.5)	4 (7.8)

** Measured as an exposure adjusted incidence rate (per 100 patient years)*

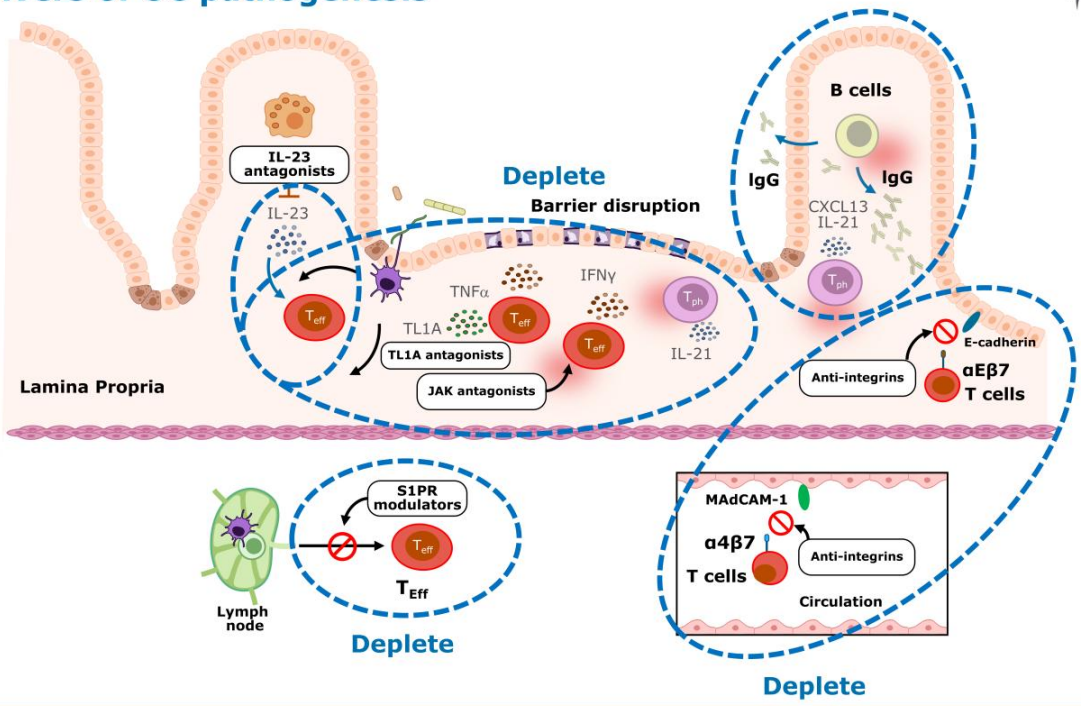
1. SAEs (severe unless otherwise noted): RSV – moderate (600mg Q2W); anaphylaxis from wasp sting (600mg Q2W); ureter stone (600mg Q2W); cholecystitis / pericardial effusion (600mg Q2W); meniscus tear – moderate (400mg Q4W); diverticulitis – moderate (400mg Q4W); embolic ischemic stroke (100mg Q4W); pneumonia – mild (100mg Q4W); cellulitis/diarrhea (placebo)

2. Severe AEs (excluding SAEs): RA flare (600mg Q2W); blood creatine phosphokinase increase (400mg Q4W); endometriosis (100mg Q4W); alanine aminotransferase increased/aspartate aminotransferase increase (100mg Q4W); flu/headache (100mg Q4W); macular degeneration/retinal hemorrhage (placebo)

3. Values shown are for herpes zoster, none were severe and are the only opportunistic infection reported.

4. No patient met the predefined protocol liver function test stopping criteria. Only one ALT elevation was severe, which was at a single time point, and resolved without interruption of therapy. None were serious, all were transient, resolved without interruption of therapy, and had an outcome of recovered/resolved or recovering/resolving.

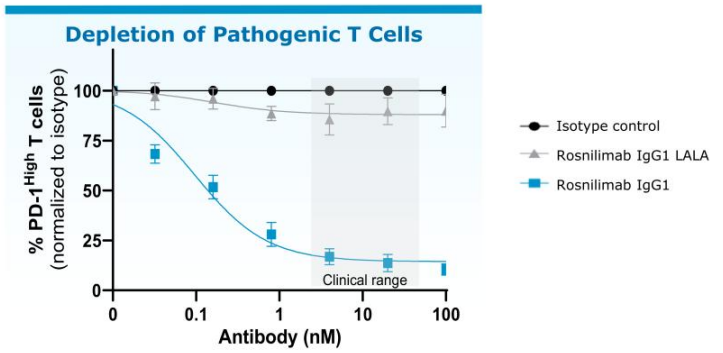
Pathogenic T cells broadly impact multiple clinically validated drivers of UC pathogenesis

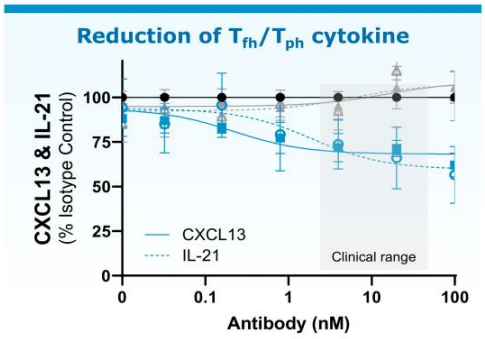


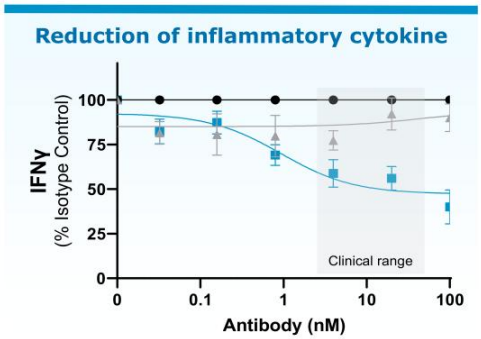
>40% of T cells in lamina propria in UC are pathogenic
2x increase of pathogenic T cells observed in blood *vs.* healthy controls[1]

Adapted from Gastroenterology & Hepatology Volume 18, Issue 8 August 2022. 1. Chen et al, Clinical and Translational Immunology, 2024.

Rosnilimab's potent depletion of pathogenic T cells reduces T cell proliferation and inflammatory cytokines that disrupt barrier function



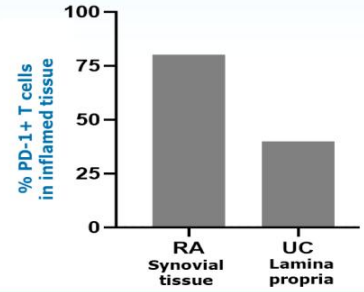




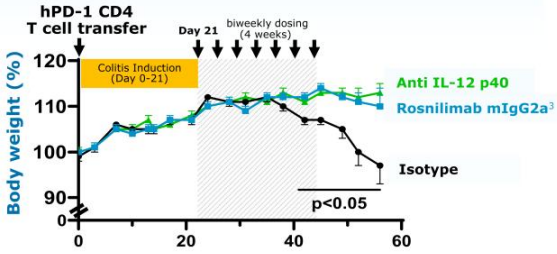
Parmley et. al. UEGW 2024. October 2024; Anti-CD3+ anti-CD28 stimulation of UC patient PBMCs for assessment of depletion and agonism MOA, representative data from N=6 donors; Rosnilimab IgG1 LALA included to demonstrate importance of Fc effector function.

T_{ph} impact seen in RA Phase 2b trial relevant to UC biology and correlates to reduction of remission

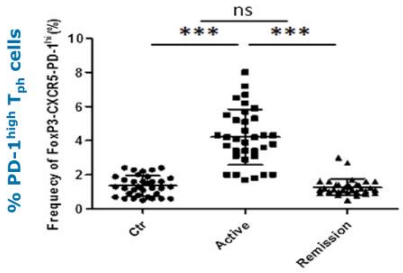
Pathogenic T cells are prevalent in inflamed tissue and periphery in RA and UC



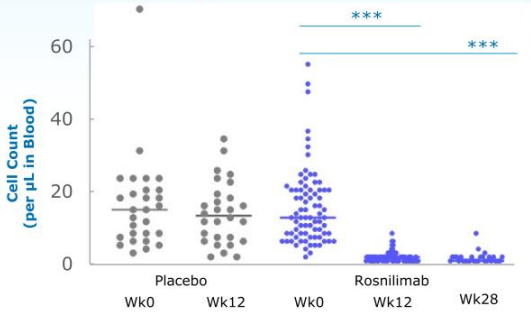
Therapeutic dosing of rosnilimab demonstrated efficacy in a murine model of colitis



T_{ph} cells are reduced with remission in UC[1,2,4]



Rosnilimab T_{ph} impact in RA Phase 2b



Parmley et. al. UEGW 2024. October 2024

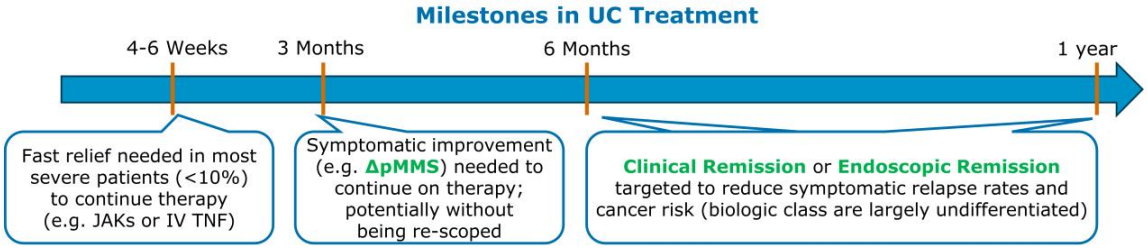
1. PD-1^{high} T_{ph} cells defined by CD3+CD4+CD45RA-PD-1+TIGIT+ICOS+CXCR5-. Long et al, Immunology Letters 233 (2021) 2-10.; 2. Rao et al, Nature, 2017. *** p<0.001, * p<0.05; 3. Rosnilimab formatted to mIgG2a to mediate effector function in mice. Suzuki et al., Sci. Immunol. 8, eadd4947 (2023); 4. Reduction of Tfh/Tph cells should impact plasma cell generation and autoantibody levels, including anti-microbial IgG antibodies that are contributing to colonic inflammation and barrier disruption: Uzzan et al, Nature, 2022.

Treaters target a well controlled disease state (e.g., endoscopic remission) in 6-12 month maintenance phase

However, treaters look for a signal of response (e.g., improved clinical symptoms) by 3+ month induction phase

UC TPP guidance

- 3 months: ΔMMS vs. placebo (primary endpoint) is statistically significant
  - Adequate symptomatic improvement to stay on drug to potential remission (by 6+ months)
- 6 months: "IL-23-like" clinical remission and endoscopic remission as measured by imputed ITT (not maintenance responder analysis)
- 6 – 12 months: Better durability than biologics, where 1/3 to 1/2 relapse within 1 year

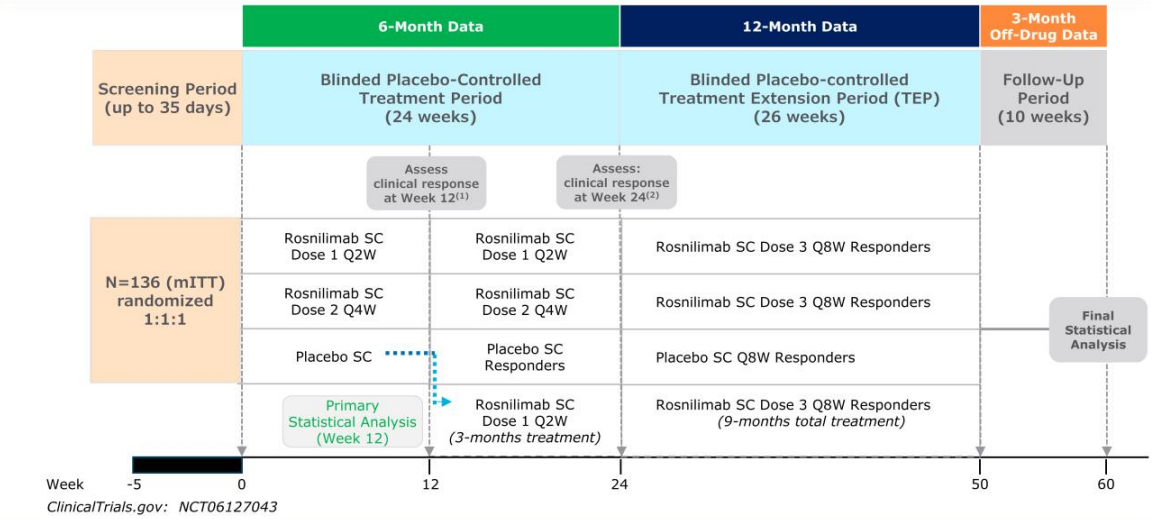


Increasingly, differentiation to be driven by 6+ month objective endpoints

AbbVie: focus on "extended induction" (~6 months) to maximize clinical response on IL-23p19's
J&J: primary endpoint of DUET TNF + IL-23p19 combo study is clinical remission at Week 48

Rosnilimab Phase 2 in moderate-to-severe UC

Enrollment completed; On track for top-line data through Week 12 in Nov/Dec 2025



ClinicalTrials.gov: NCT06127043

- **Primary endpoint: Change in mMS[3] (stool frequency + rectal bleeding + endoscopy score) vs. placebo at Week 12**
  - Assesses symptomatic and objective clinical improvement at 3 months
- **"Treat-through" design aligned with clinical practice to give a real-world assessment of remission at 6 months**
  - Distinctly different from trials with an enriched responder analysis in 6 or 12-month maintenance studies

1. Week 12 visit: All patients, regardless of study arm, treat-through to Week 24 and remain blinded to treatment arm. Placebo-treated patients who achieved partial modified Mayo score (pmMS) clinical response or at Week 12 remain on placebo, while placebo-treated patients who are non-responders on pmMS are crossed over to the subcutaneous high-dose rosnilimab treatment arm; 2. Week 24 visit: All patients, regardless of study arm, can opt into the TEP to Week 50 if achieved pmMS clinical response by Week 24. All non-responders proceed to study follow-up; 3. mMS = modified Mayo score

Next steps for rosnilimab

Rheumatoid Arthritis	Ulcerative Colitis

Positive Phase 2b data reported
• Best-in-disease profile
• Favorable safety and tolerability
• JAK-like efficacy through 6 months
  ○ Max response rates not yet observed due to trial design
• Sustained 12-14 week off-drug responses through 9 months
• ACR late-breaker on Oct. 29th
	Phase 2 data through Week 12 anticipated in Nov./Dec. 2025
	• Blinded surveillance data suggest favorable safety and tolerability profile
	• TPP guidance
	  ○ 3-months – Stat sig on primary endpoint (ΔMMS)
	  ○ 6-months – "IL-23-like" clinical and endoscopic remission measured by imputed ITT
	  ○ 6-12 months – Better durability than biologics where 1/3 to 1/2 relapse within 1 year

- Additional potential activities in 2026+
  - P3 enablement: drug supply scale-up and end-of-phase 2 regulatory interactions
  - Potentially initiate P2 studies in additional indications

- *Assessing strategic paths, including:*
  - *Partnership to develop in all indications, including RA and UC, or*
  - *Independently advance one Phase 3 indication, following UC data*
- *Outcome could impact how economic value of rosnilimab is allocated between Royalty Management Co and Biopharma Co*

ANB033

(CD122 antagonist)

ANB033 blocks CD122 to inhibit pathogenic immune cells

CD122 is the beta subunit (IL-2Rβ) of the receptor for IL-15 and IL-2

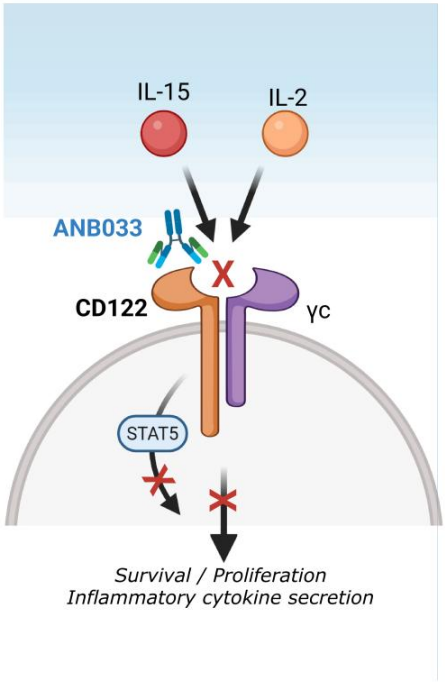
- Expressed on subsets of CD8+ and CD4+ T cells and NK cells

CD122 antagonism reduces these immune cell subsets

- Dependent on IL-15 and/or IL-2 for maintenance, proliferation and survival

Overexpressed in select diseases, including CeD gut or EoE

- CeD: IELs, including cytotoxic CD8+ and NK cells
- EoE: ILC2s



Richards et al, Cell Rep. 2025 Jul 21;44(8):116039.

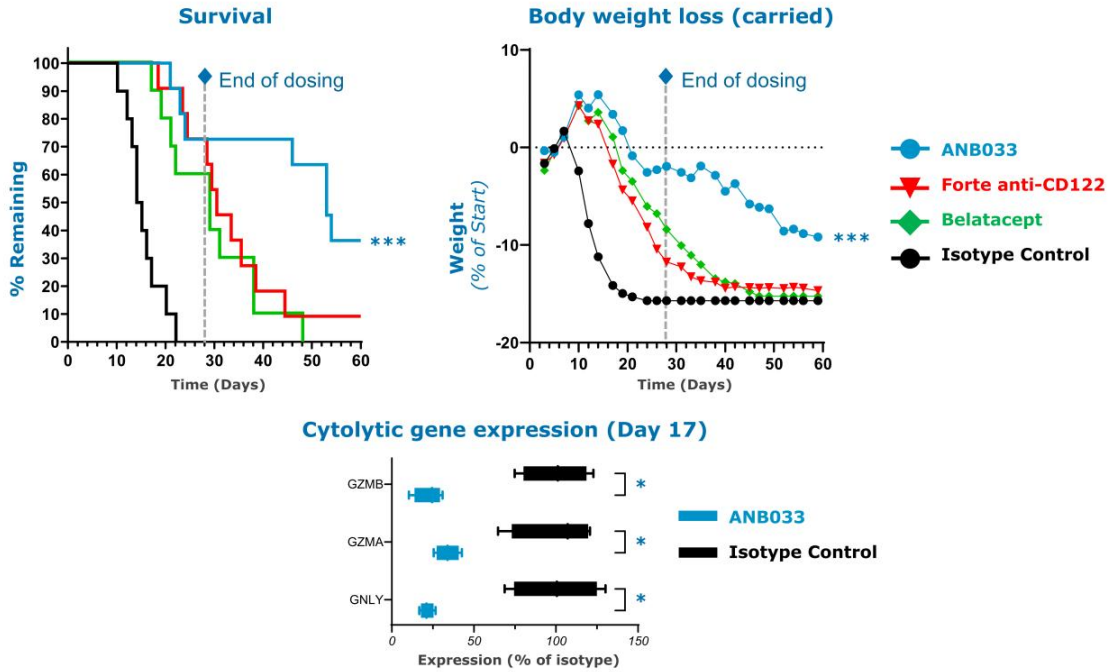
Broad therapeutic potential across autoimmune and inflammatory diseases

Gastroenterology	***Dermatology***	**Other Areas**
Celiac Disease	Atopic Dermatitis	Asthma
Crohn's Disease	Alopecia Areata	Multiple Sclerosis
Eosinophilic Esophagitis (EOE)	Hidradenitis Suppurativa	Psoriatic Arthritis
Ulcerative Colitis	Lichen Planus	Type 1 Diabetes
	Vitiligo	Solid Organ Transplant

Clinical-stage drugs targeting IL-15 or CD122

NOVARTIS	IL-15	• P1b PoC in CeD • P1b PoC in EoE
teva	IL-15	• P2a in CeD • P2a in vitiligo
Incyte	CD122	• P1b in vitiligo
FORTE	CD122	• Positive P1b in CeD (P2a ongoing) • P1b in vitiligo and alopecia areata • Assessing T1D

ANB033 shows strong survival benefit and reduced cytolytic gene expression in aggressive GvHD mouse model



GvHD (severe phenotype) model using human IL-15 transgenic mice that support human T cell and NK cell engraftment. 60-day study. Mice dosed 3 mg/kg BIW (belatacept 75 µg TIW) through Day 28. N=10 per group (isotype control and Belatacept) or 11 per group (test articles). *** Survival: ANB033 statistically significant vs isotype control (P<0.0001), Belatacept (P=0.003), Forte anti-CD122 (first achieved on Day 38, p=0.031, with significance deepening through Day 60, P=0.0032) log-rank Mantel-Cox test; Body weight loss: ANB033 statistically significant vs isotype control (p<0.001), Belatacept (p=0.0016), Forte anti-CD122 (first achieved on Day 28, p=0.037, with significance deepening through Day 60, P=0.0003), Unpaired Student's t-tests. Gene expression data generated from purified human immune cells isolated from spleen on day 17. * p<0.05 Unpaired Student's t-tests.

ANB033 Phase 1a trial ongoing in healthy volunteers

Objectives

- Safety and tolerability
- Evaluate PK and immunogenicity

Design

- All healthy volunteers have been dosed
  - ANB033: n=60
  - Placebo: n=20
- Administered both IV and SC dosing
- 10 cohorts: Four SAD IV, three SAD SC and three MAD SC
- Follow-up to ~7 months*

* The first 4 lowest SAD dose cohorts are followed through day 85; the three higher SAD dose cohorts are followed for 197 days; all MAD cohorts are followed through 218 days.

ANB033 demonstrated favorable safety, tolerability and PK profile in Phase 1a

Phase 1a results to date

✓ Safe and well tolerated

✓ No unexpected findings

✓ PK and PD support SC dosing

Favorable safety and tolerability

- No safety concerns at any dose
  - No SAEs, severe AEs, or discontinuations
  - Any adverse events mild or moderate
- No unexpected lab abnormalities
- No signs of viral infections
- No clinical pharmacology findings of concern

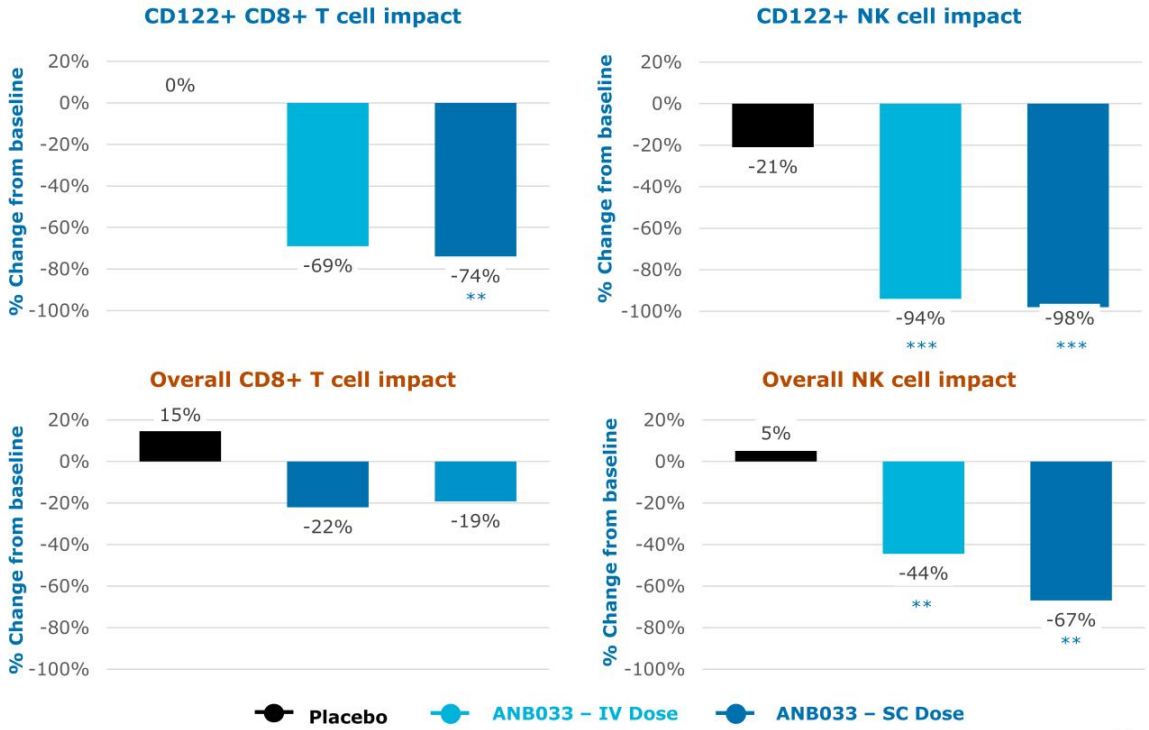
Rapid and sustained PK profile

- Favorable 2 to 3-week half-life with IV and SQ dosing
- Full receptor occupancy (RO) within hours and maintained for >30 days
- Dose response observed
- Modeled to achieve >IC90 on CD8+ T cells subsets in GI tissue
- Overall, no impact on peripheral total Treg counts

SAE = Serious adverse event

ANB033 significantly reduces CeD relevant CD8+ T cells and NK cells after single dose

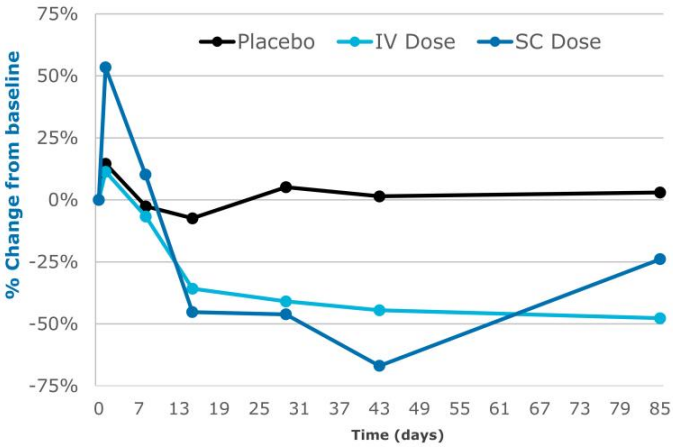
Effect of ANB033 is limited to CD122 expressing cells

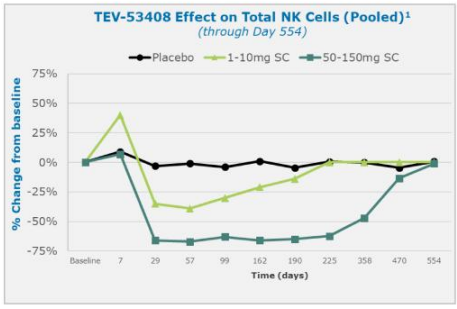


Graphs reflect SAD data and maximum reductions were achieved within the first 43 days. *** p<0.001 **p<0.01

Anti-IL-15 and CD122 therapies have demonstrated sustained reduction in CD122+ NK cells with no observed safety issues

ANB033 effect on total NK cells





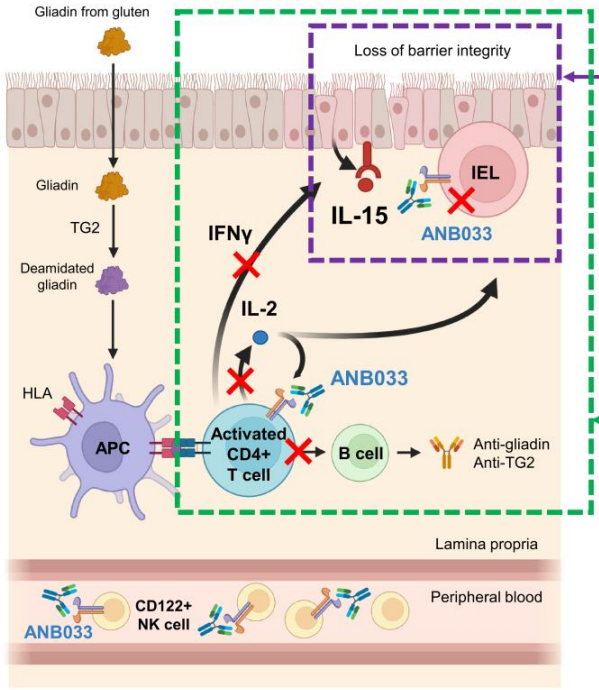
No safety signals observed in any CD122 or IL-15 trials to date after NK cell reduction

- ANB033 >50% peak total NK cell reduction with return towards baseline within 3 months
- TEV-53408: >50% sustained total NK cell reduction for 1 year with return to baseline over 18 months

1. Schnir et. al; Developing TEV-53408 for the Treatment of Celiac Disease: Summary of Preliminary Results from the First-in-Human Phase 1 Study in Healthy Volunteers, Single SC doses, DDW, May 2024. Phase 1a, single dose, study completed (n=60 TEV-53408, n=19 placebo). Moved into Phase 2a CeD trial in 48 adults while undergoing gluten challenge; primary trial completion in Sept. 2026.

ANB033's MOA ideal fit for targeting CeD inflammation

CeD marked by excessive IL-15 and IL-2 production which perpetuates disease



Inhibition of IL-15 signaling

- IL-15 induces proliferation of IELs
  - Majority of IELs are CD122+ T cells
- Inhibiting IL-15 signaling reduces IELs
  - Reduces epithelial cell destruction
  - Restores barrier integrity

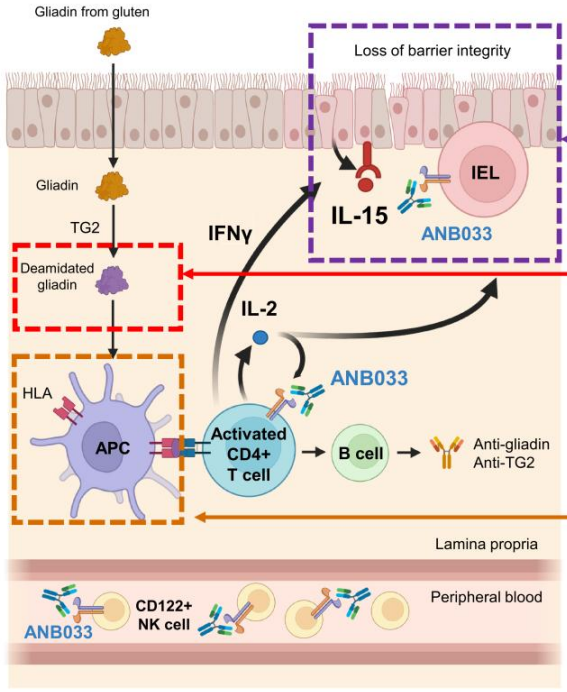
Inhibition of IL-2 signaling

- IL-2 stimulates
  - CD4 effector memory T cell activation and proliferation
  - IFNγ production leading to IL-15 secretion
- Inhibiting IL-2 signaling reduces
  - Gluten-responsive CD4 T cell expansion
  - Inflammatory cytokine secretion
  - Downstream B cell-mediated antibody responses

Adapted from Dieckman et al. (2022) Curr. Opin. Pharmacol. 66:102268.

Previous approaches have not addressed multiple pathogenic drivers of CeD

However, a CD122 antagonist targets both key pathogenic drivers of CeD



IL-15 antagonists: Clinical PoC

teva — *P2 ongoing*

NOVARTIS — *P1b PoC*

AMGEN — *Lacked potency*

Non-immune cell targeting

Takeda — *P2 ongoing*
Gluten tolerance

astellas — *Discontinued*
Gluten tolerance

Roche — *P1 ongoing*
HLA-DQ2.5 gluten peptide complex

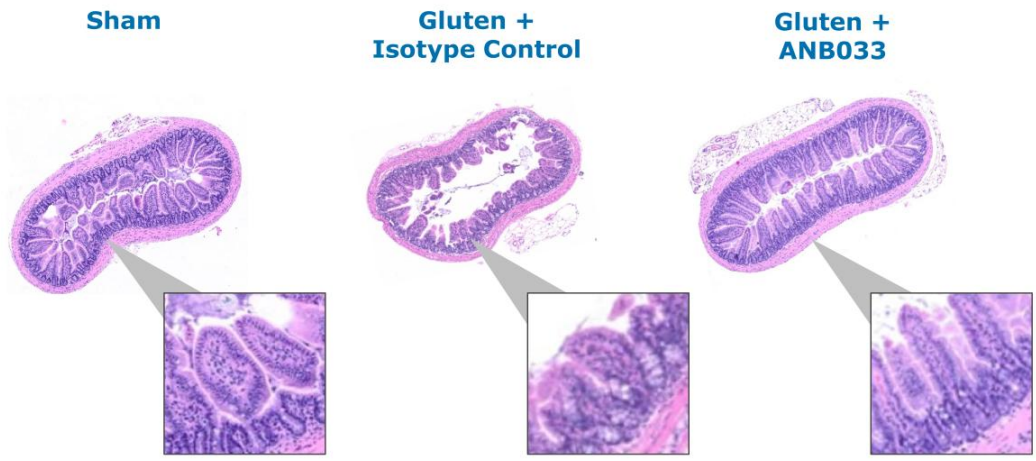
Immunic THERAPEUTICS — *P1 ongoing*
SIRT6 modulator

OX-40L antagonist

sanofi — *P2 ongoing*

Adapted from Dieckman et al. (2022) Curr. Opin. Pharmacol. 66:102268.

ANB033 prevents key CeD histologic manifestation of gluten-induced villous atrophy



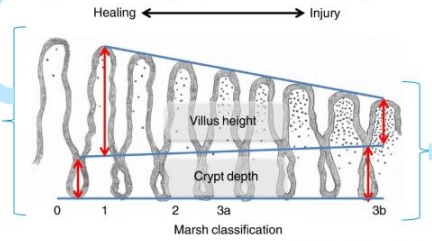
ANB033 treatment shows improved histology:
preserves villus height and crypt depth (Vh:Cd) in CeD mouse model

Note: HuDQ8-D^{d}-villin-IL-15tg mice on a gluten-free diet are challenged with gluten, and CeD features are analyzed on day 30. The treatment regimen includes a sham (no gluten), isotype control and ANB033 surrogate antibody (anti-mouse CD122 antibody with similar epitope and affinity to ANB033) administered at 10 mg/kg BIW.

ANB033 significantly prevents reduction of Vh:Cd ratio compared to control

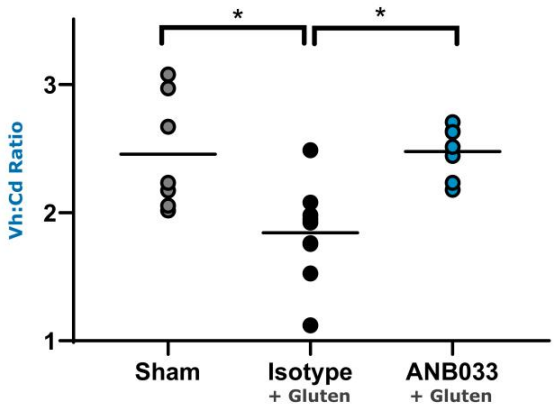
Vh:Cd ratio

Healthy tissue:
High villus height, lower crypt depth



CeD tissue:
Reduced villus height, extended crypt depth

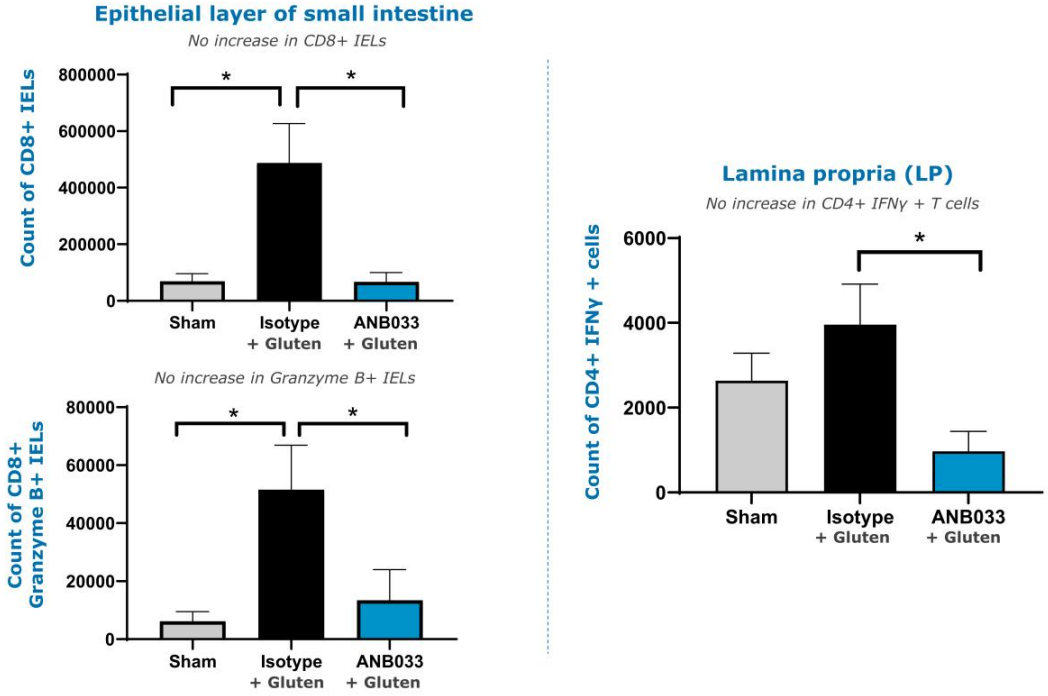
ANB033 impact on Vh:Cd ratio



ANB033 treatment shows improved histology: preserves villus height and crypt depth (Vh:Cd) in CeD mouse model

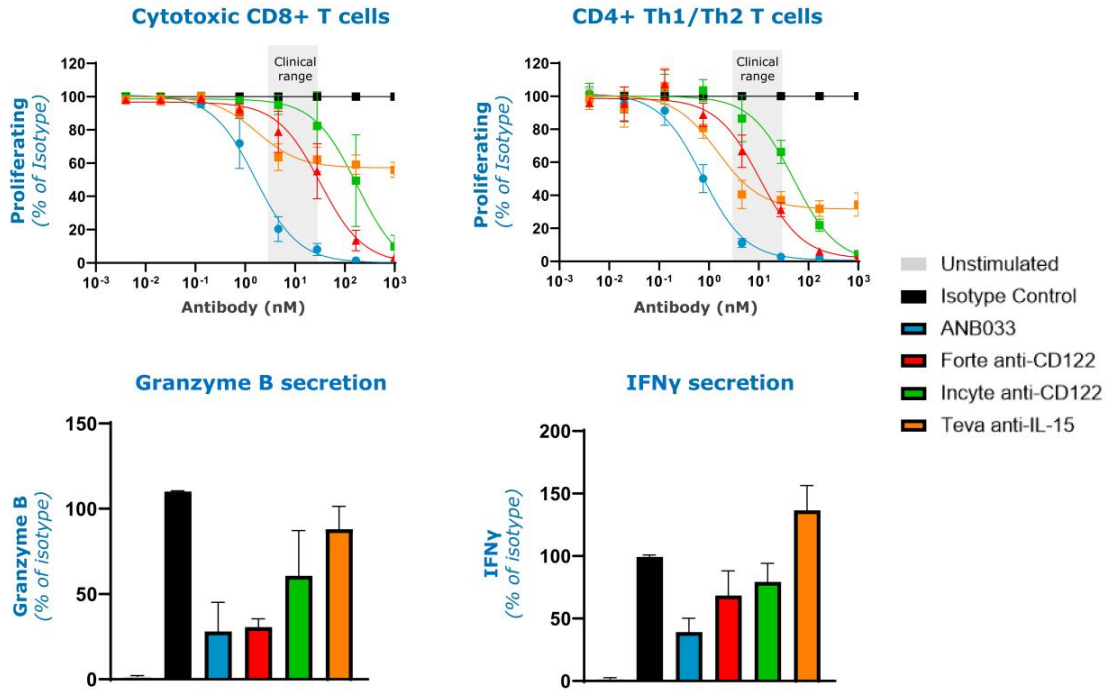
Note: HuDQ8-D^{d}-villin-IL-15tg mice on a gluten-free diet are challenged with gluten, and CeD features are analyzed on day 30. The treatment regimen includes a sham (no gluten), isotype control and ANB033 surrogate antibody (anti-mouse CD122 antibody with similar epitope and affinity to ANB033) administered at 10 mg/kg BIW. * p<0.05.

ANB033 prevents gluten-induced intestinal inflammation



Note: HuDQ8-D^{d}-villin-IL-15tg mice on a gluten-free diet are challenged with gluten, and CeD features are analyzed on day 30. The treatment regimen includes a sham (no gluten), isotype control and ANB033 surrogate antibody (anti-mouse CD122 antibody with similar epitope and affinity to ANB033) administered at 10 mg/kg BIW. IFNγ + CD4 T cells and GrzB+ CD8+ T cells enumerated by intracellular flow cytometry.

ANB033 shows differentiated impact in CeD patient-derived PBMCs compared to competing anti-IL-15s and CD122s



Top Panel: PBMC from CeD donors measuring proliferation (Ki67 staining), stimulated for 7 days with IL-15 + IL-2 (N=4 donors).
Bottom Panel: PBMC from CeD donors stimulated for 3 days with anti-CD3 and anti-CD28 (N=4 donors).

Symptomatically controlled CeD patients present with range of histologic activity

Histology *(Vh:Cd ratio)*

3.5 — 2.5 — 2.0 — 1.5 — 1.0 — 0.1

Normal | Mucosal damage | Abnormal

Symptoms

Asymptomatic | Symptom controlled | Poorly controlled

Symptomatically controlled on GF diet

Gluten challenge
Phase 1 population

teva *(Phase 1b)* | NOVARTIS Calypso *(Phase 1b)* | FORTE *(Phase 1b/2a)*

Nearly all P1b/P2a studies only assess ability **to prevent** gluten-induced mucosal injury

- Gluten challenge: patients with higher Vh:Cd ratios (>2.5 or >2.0)

Persistent mucosal damage despite paucity of symptoms

AnaptysBio *(Phase 1b)*

Added additional cohort to P1b **to inform on potential to heal mucosa** in patients with existing histologic mucosal damage and further derisk 2b

Symptomatic on GF diet

Non-responsive

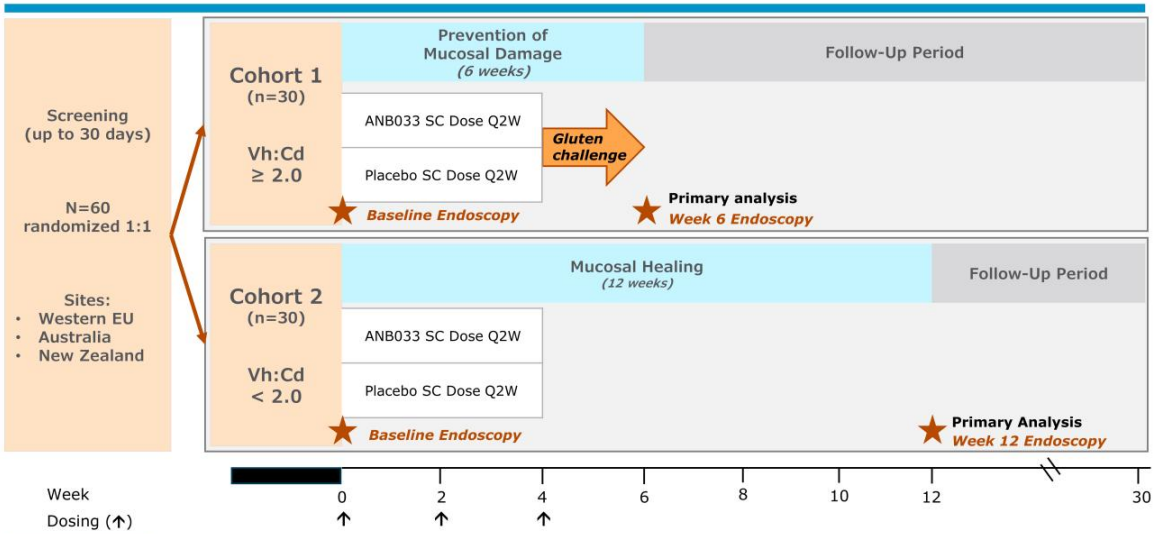
sanofi *(Phase 2b)*

Goal of P2b or P3 to assess if drug can heal damaged mucosa and restore normal symptomatology

GF diet = Gluten free diet.

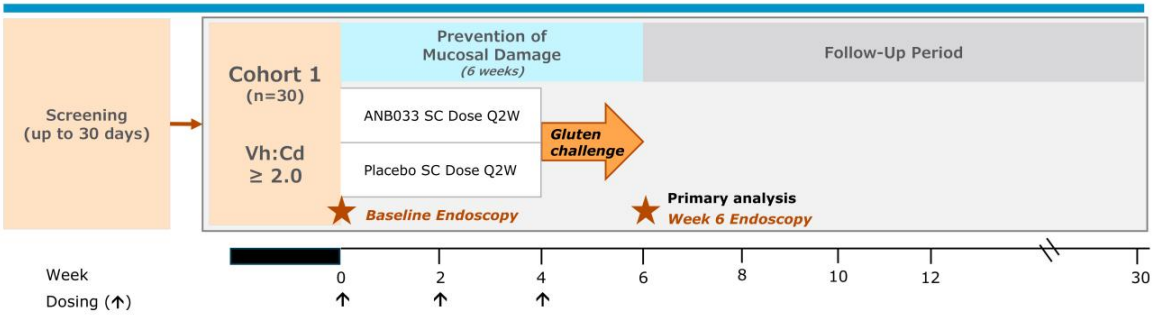
ANB033 Phase 1b trial in CeD initiated

Anticipate top-line data by year-end 2026



Safety	Safety and tolerability in adult participants with well-controlled CeD
Clinical PK	PK and immunogenicity
Efficacy	• Change from baseline in Vh:Cd ratio • IEL count • PROs, including Celiac Disease Symptom Diary (CDSD)
Biomarkers	Characterize ANB033 effects on circulating biomarkers, including robust translational plan

Cohort 1 (Vh:Cd ≥ 2.0) is a gluten-challenge to assess prevention of mucosal damage



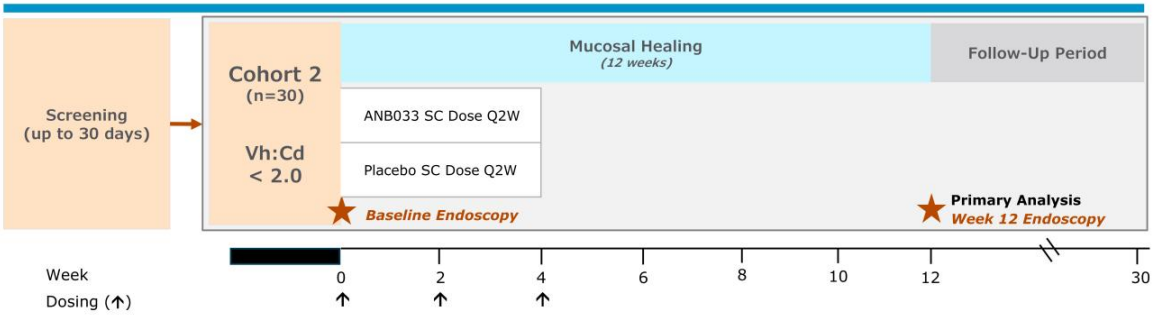
Minimal evidence of mucosal damage (Vh:Cd ≥ 2.0)

- Symptom-controlled CeD patients
- Receive GC after pre-treatment with ANB033 vs. PBO

- ANB033 dose at Week 0, 2, 4 (pre-treatment)
- Gluten challenge allows for controlled induction of mucosal damage
  - Beginning Week 4, 6g gluten dose daily (study supplied cookie) for two weeks through Week 6
- Endoscopy at Week 6
  - Assess prevention of gluten-induced mucosal damage

GC = Gluten challenge

Cohort 2 (Vh:Cd < 2.0) assesses ability to heal mucosal damage in symptom-controlled patients



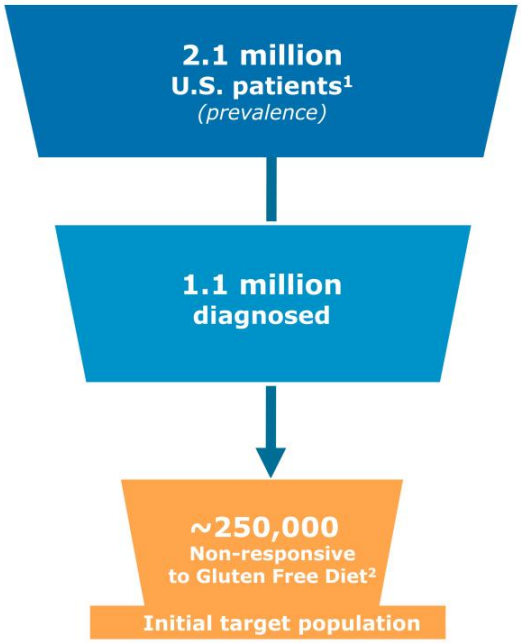
Persistent evidence of histologic CeD activity (Vh:Cd < 2.0)

- Symptom-controlled CeD patients
- Substantial mucosal damage already present (no gluten-challenge)
- *Proxy: nonresponsive patients*

- ANB033 dose at Week 0, 2, 4
- Endoscopy at Week 12
  - Assess healing 8 weeks after last ANB033 dose
  - Maximize healing time given ANB033 prolonged tissue exposure and PD properties

GC = Gluten challenge

Potential blockbuster opportunity for ANB033 in non-responsive CeD



High disease burden

- Debilitating symptoms, social isolation
- Disease awareness driving growth
- No approved therapies

CD122s differentiated from other Tx in development

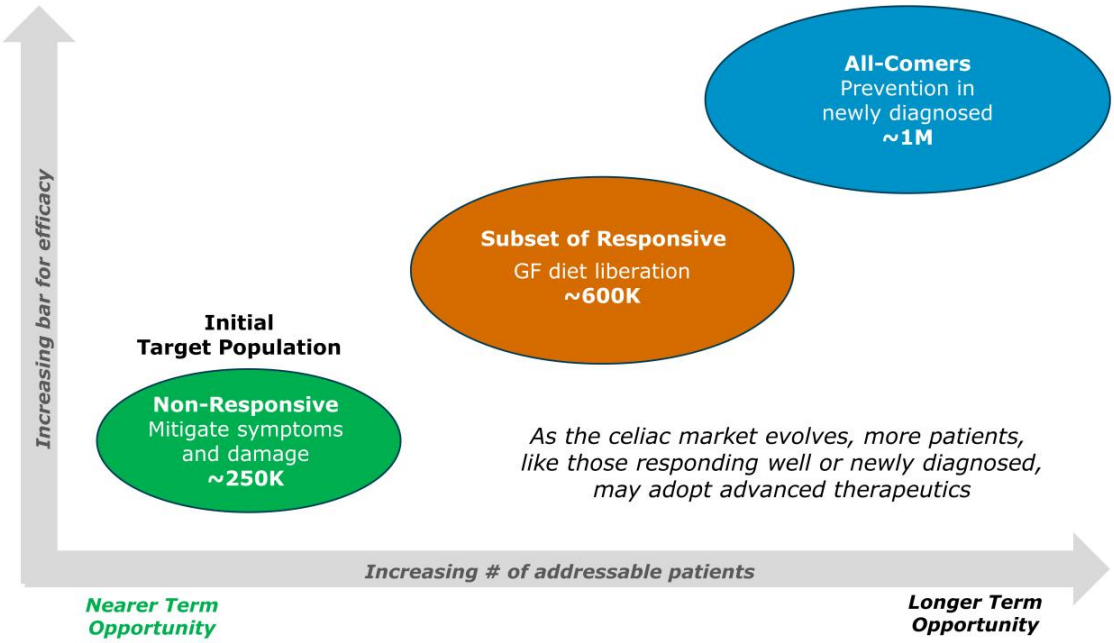
- HCPs favor MOA that targets both symptoms and histology

$4-5B U.S. market in patients non-responsive to gluten-free diet

- Potential to reach IBD diagnosis and biologic penetration analogs given substantial unmet need
- Expect reimbursement with limited utilization management

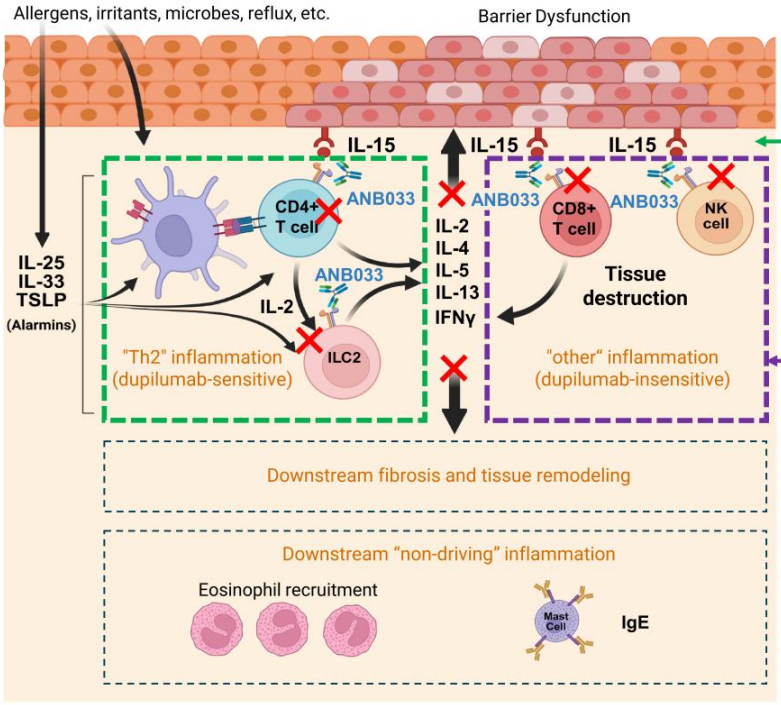
1. Singh et al. (2018), Choung et al. (2016), Katz et al. (2011), Trinity Life Sciences Commercial Assessment HCP Primary Market Research (2025). CeD sizing reflects future US market in 2030 assuming growth in diagnosis rate based on historic trends and projected growth with entrance of novel therapies
i2. Leffler et al. (2007), Abhijeet et al. (2016), Aggarwal et al. (2025) Mahadev et al. (2017, Trinity Life Sciences Commercial Assessment HCP Primary Market Research (2025) Percent of CeD non-responders to Gluten Free Diet with or without villous atrophy.

New therapies in CeD could grow market in responsive and newly diagnosed patients



1. Singh et al. (2018), Choung et al. (2016), Katz et al. (2011), Leffler et al. (2007), Abhijeet et al. (2016), Aggarwal et al. (2025) Mahadev et al. (2017, Trinity Life Sciences HCP Primary Market Research (2025)] CeD sizing reflects future US market in 2041 assuming growth in diagnosis rate based on historic trends and projected growth with entrance of novel therapies.

Similar to CeD, ANB033 targets multiple drivers of EoE biology addressing both dupilumab sensitive and insensitive pathways



CD4+ and ILC2 (dupilumab-sensitive)

- Reduces CD4+ T cell proliferation
- Reduces IL-5/13 from CD122+ ILC2 and CD4+ T cells

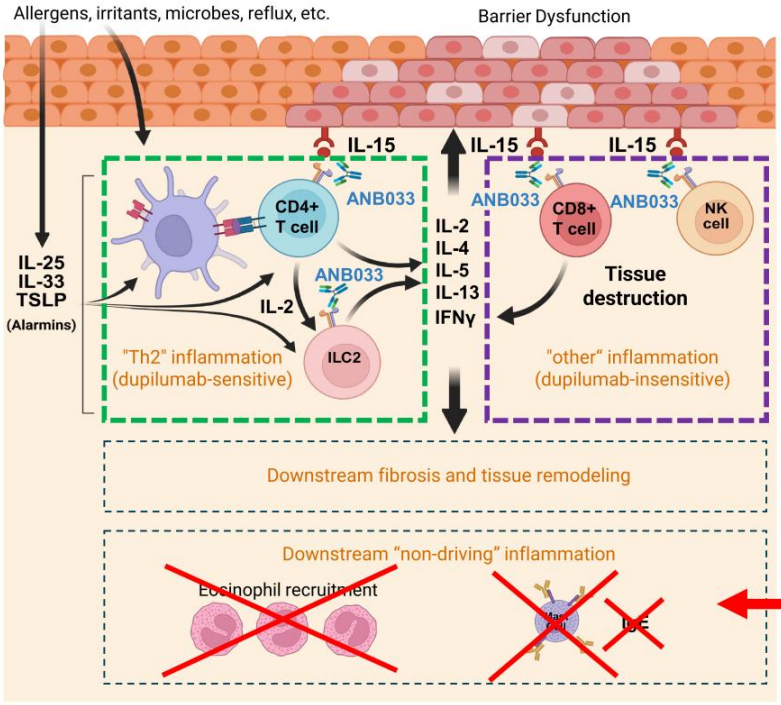
CD8+ and NK (dupilumab-insensitive)

- Inhibiting IL-15 signaling reduces –
  - IFNγ
  - Granzyme B
  - CD8+ T cell proliferation
  - NK cells

Upstream pathways lead to downstream eosinophilic infiltration into tissue

Adapted from Discepolo et. al. Gastroenterology. 2024; 167:90-103.

Mechanisms that target only downstream signals of inflammation have not been successful in EoE



Approved

sanofi

Dupilumab (anti-IL-4Ra)

Has PoC

NOVARTIS Calypso biotech

GIA-632 (anti-IL-15)

Ongoing

AMGEN

tezepelumab (anti-TSLP)

Failed mechanisms

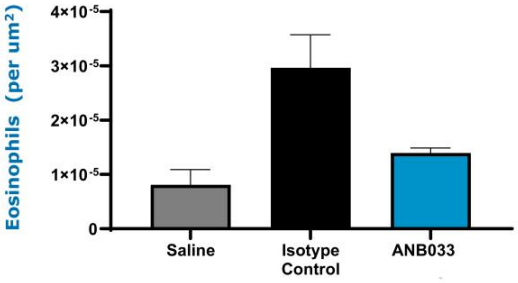
anti-cKIT, anti-IL-5Ra, anti-IgE

Celldex Genentech AstraZeneca

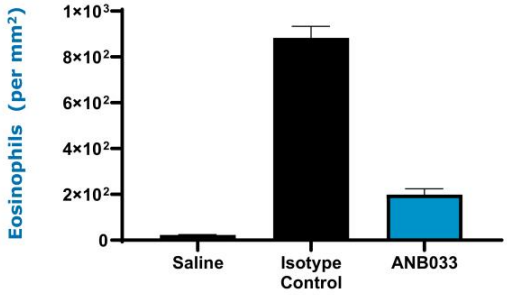
Adapted from Discepolo et. al. Gastroenterology. 2024; 167:90-103.

ANB033 prevents eosinophilia by targeting upstream inflammation

Esophageal eosinophils



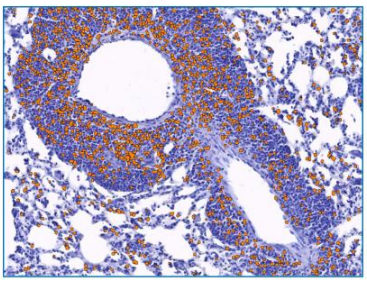
Lung eosinophils



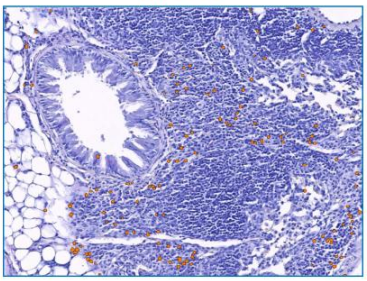
Aspergillus-induced eosinophilia

(Lung sample shown)

Isotype Control

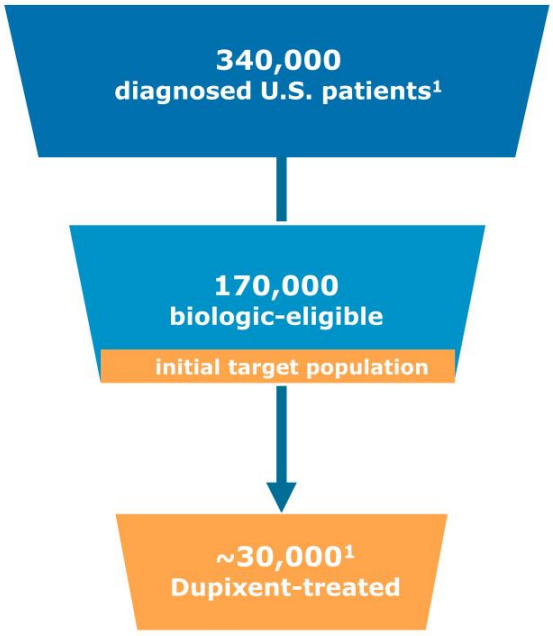


ANB033



Model of eosinophilic inflammation: Balb/c mice were challenged intranasally with *Aspergillus fumigatus* TIW for 3 weeks. The treatment regimen includes a saline, isotype control, and ANB033 surrogate antibody (anti-mouse CD122 antibody with similar binding epitope and affinity to ANB033, administered at 10 mg/kg BIW for 3 weeks. Tissues were stained with H&E for histopathology assessment.

Assessing potential to treat EoE: significant market with increasing prevalence and unmet need



Significant unmet need with limited approved therapies

- ~50% PPI or steroid non-responsive or intolerant
- Dupixent QW approved in 2022
- 20-30% Dupixent non-responsive

Increasing disease recognition with >8% CAGR[1,2]

- Heightened rates of endoscopic procedures and biopsies

~$5B+ U.S. sales anticipated by 2030

- Potential to reach IBD diagnosis and biologic penetration analogs given substantial unmet need

1. ZS Claims analysis and KOL interviews August 2025; 2. "Prevalence and costs of eosinophilic esophagitis in the United States" (Thel 2024, Clinical Gastroenterology and Hepatology). 8% CAGR from 2019-2024; expected to continue through 2030.

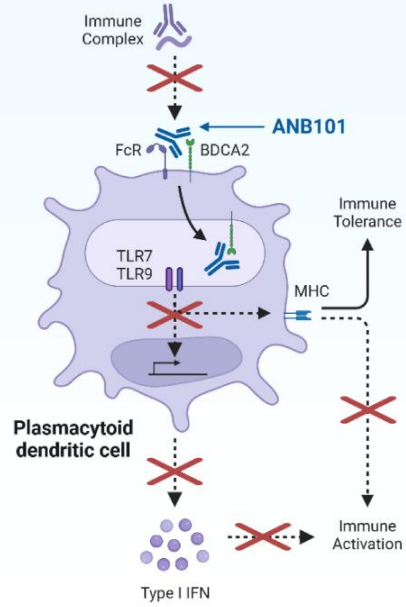
ANB101

(BDCA2 modulator)

ANB101: BDCA2 modulator of plasmacytoid dendritic cell (pDC) function

Phase 1 trial ongoing in healthy volunteers

BDCA2 is a molecule specifically expressed on pDCs



ANB101 will potently inhibit interferon secretion and immune activation

Activated pDCs bridge innate and adaptive immunity

- Secrete Type I IFN (1000x increase over other cell types)
- Present antigens to adaptive immune system

pDCs enriched in tissue in rheumatology and other inflammatory diseases

- BDCA2 modulator mechanistic proof-of-concept (Biogen's litifilimab) in SLE / CLE

ANB101: BDCA2 modulator

- Potent and sustained internalization of BDCA2 on pDC cell surface
- Profound inhibition of interferon secretion reduces inflammation

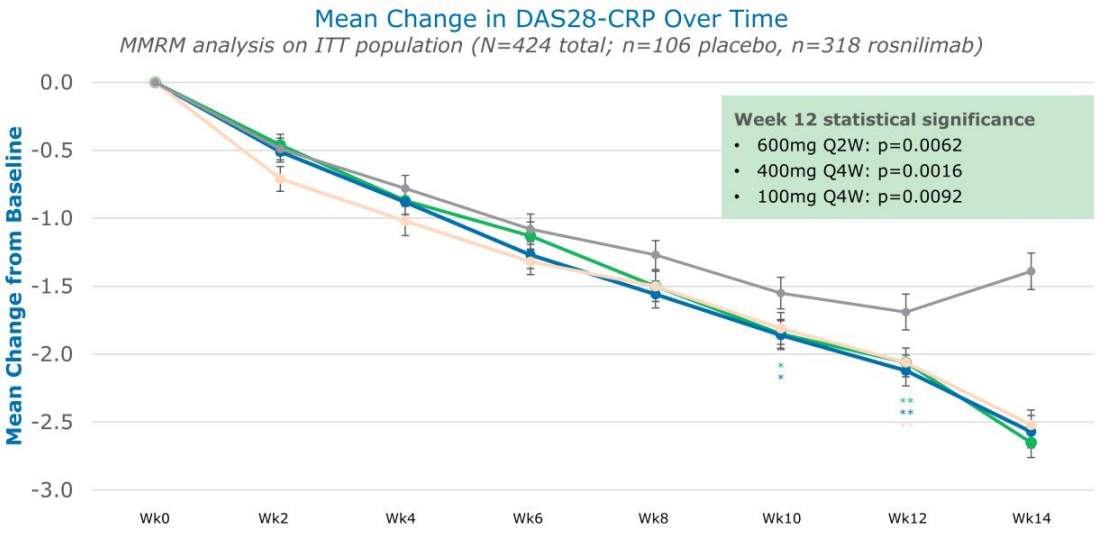
Appendix

Baseline RA disease characteristics and demographics

Baseline Characteristic	Placebo (n=106)	100mg Q4W (n=106)	400mg Q4W (n=107)	600mg Q2W (n=105)	Overall (N=424)
Age, years, mean (SD)	58 (11)	57 (10)	57 (12)	56 (11)	57 (11)
Female, n (%)	83 (78%)	79 (75%)	79 (74%)	80 (76%)	321 (76%)
Weight (kg), mean (SD)	78 (17)	78 (19)	81 (19)	77 (16)	78 (18)
Geographic region, n (%) US Ex-US	 35 (33%) 71 (67%)	 34 (32%) 72 (68%)	 35 (33%) 72 (67%)	 26 (25%) 79 (75%)	 130 (31%) 294 (69%)
Race, n (%) White Black or African American Asian Other	 102 (96%) 3 (3%) 0 (0%) 1 (1%)	 102 (96%) 1 (<1%) 0 (0%) 3 (4%)	 103 (96%) 4 (4%) 0 (0%) 0 (0%)	 101 (96%) 4 (4%) 0 (0%) 0 (0%)	 408 (96%) 12 (3%) 0 (0%) 4 (1%)
Duration of disease, years, mean (SD)	11 (9)	11 (10)	9 (8)	10 (9)	10 (9)
DAS28-CRP, mean (SD)	5.7 (0.8)	5.6 (0.8)	5.7 (0.9)	5.7 (0.8)	5.6 (0.8)
CDAI, mean (SD) CDAI >22, n (%)	37.9 (10.2) 101 (95%)	37.2 (10.6) 101 (95%)	37.1 (10.6) 102 (95%)	38.6 (11) 100 (95%)	37.7 (10.6) 404 (95%)
TJC68, mean (SD)	23 (13)	22 (12)	22 (12)	23 (13)	22 (12)
SJC66, mean (SD)	14 (7)	15 (7)	14 (7)	16 (9)	15 (8)
CRP, mean (SD)	16 (22)	17 (20)	21 (26)	19 (28)	18 (24)

DAS28-CRP – Disease Activity Score 28-C-reactive protein; CDAI – Clinical Disease Activity Index; TJC68 – tender joint count, 68 joints; SJC66 – swollen joint count, 66 joints; CRP – high-sensitivity C-reactive protein

Rosnilimab met primary endpoint of mean change from baseline in DAS28-CRP at Week 12 for all active doses

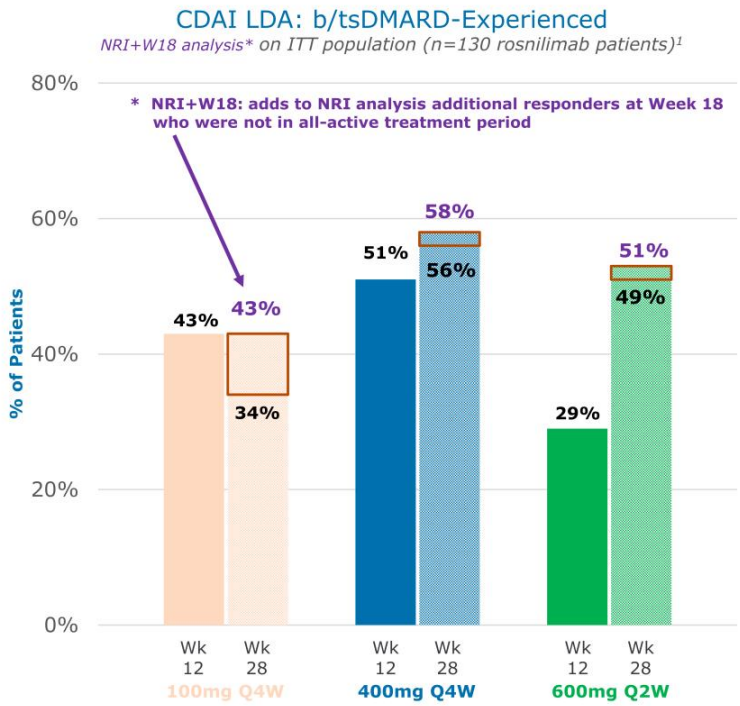


- All rosnilimab doses statistically significant at Week 12
- All rosnilimab doses continue to improve into Week 14 with no evidence of flattening
- Following Week 14 visit, placebo patients proceeded to post treatment follow-up

Rosnilimab - 100mg Q4W | Rosnilimab - 400mg Q4W | Rosnilimab - 600mg Q2W | Placebo

1. Mixed Model for Repeated Measures (MMRM) analysis on intent-to-treat (ITT) population; b/tsDMARD-naïve population (n=62 placebo, n=62 100mg Q4W, n=62 400mg Q4W, n=64 600mg Q2W); b/tsDMARD-experienced population (n=44 placebo, n=44 100mg Q4W, n=45 400mg Q4W, n=41 600mg Q2W); DAS28-CRP based on differential weighting of individual measures, including patient's general health, CRP and a count of 28 swollen and tender joints, with a score ranging from 0 to 9.4. **p<0.01, *p<0.05, Standard error (SE) used to present figures of least squares mean changes from baseline.

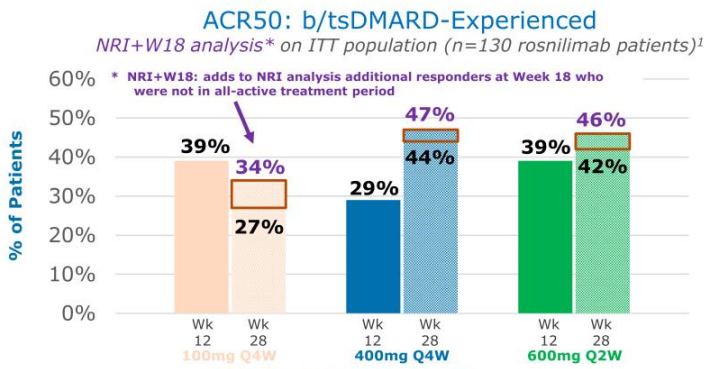
Demonstrated JAK-like CDAI LDA rates by 6 months

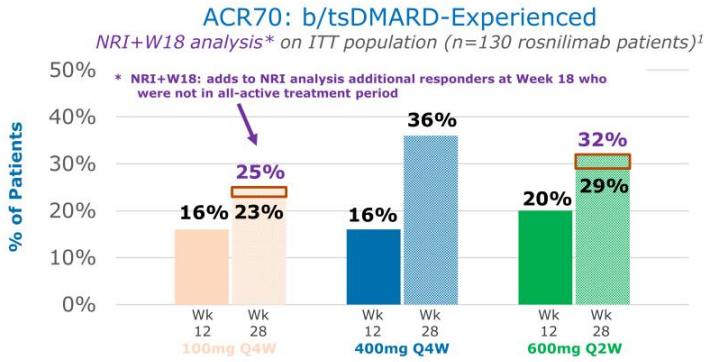


CDAI LDA at Week 28		
Arm	NRI	NRI+W18
b/tsDMARD-Experienced Population *(as graphed)*		
100mg	34%	43%
400mg	56%	58%
600mg	49%	51%
b/tsDMARD-Naïve Population		
100mg	66%	71%
400mg	53%	55%
600mg	72%	75%

1. Non-responder imputated (NRI) analysis on intent-to-treat (ITT) of all b/tsDMARD-naïve patients randomized; b/tsDMARD-experienced population (n=44 placebo, n=44 100mg Q4W, n=45 400mg Q4W, n=41 600mg Q2W; n=130 total rosnilimab b/tsDMARD-experienced patients)

Demonstrated JAK-like ACR70 rates which deepened into 6 months





ACR50 at Week 28		
Arm	NRI	NRI+W18
b/tsDMARD-Experienced Population *(as graphed)*		
100mg	27%	34%
400mg	44%	47%
600mg	42%	46%
b/tsDMARD-Naïve Population		
100mg	58%	61%
400mg	52%	53%
600mg	69%	75%

ACR70 at Week 28		
Arm	NRI	NRI+W18
b/tsDMARD-Experienced Population *(as graphed)*		
100mg	23%	25%
400mg	36%	36%
600mg	29%	32%
b/tsDMARD-Naïve Population		
100mg	53%	55%
400mg	37%	37%
600mg	55%	58%

1. Non-responder imputated (NRI) analysis on intent-to-treat (ITT) of all b/tsDMARD-naïve patients randomized; b/tsDMARD-experienced population (n=44 placebo, n=44 100mg Q4W, n=45 400mg Q4W, n=41 600mg Q2W; n=130 total rosnilimab b/tsDMARD-experienced patients)