**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: November 10, 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 November 10, 2025, AnaptysBio, Inc. ("AnaptysBio") updated its corporate investor presentation, a full copy of which is attached hereto as Exhibit 99.1.

The information in this Item 7.01, including Exhibit 99.1, 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 8.01. Other Items**

On November 10, 2025, AnaptysBio issued a press release regarding top-line Phase 2 data in ulcerative colitis with rosnilimab. A copy of the press release is filed herewith as Exhibit 99.2.

**Item 9.01. Financial Statements and Exhibits.**

(d) Exhibits

| Exhibit Number | Exhibit Title or Description |
|---|---|
| 99.1 | Anaptys Corporate Presentation November 2025. |
| 99.2 | AnaptysBio, Inc. Press Release, dated November 10, 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: November 10, 2025

By: /s/Dennis Mulroy

Name: Dennis Mulroy

Title: Chief Financial Officer


Corporate
Overview
November 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 from 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; 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.

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 by YE 2026

## 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 |
| | P1b in Undisclosed Inflammatory Disease | |

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

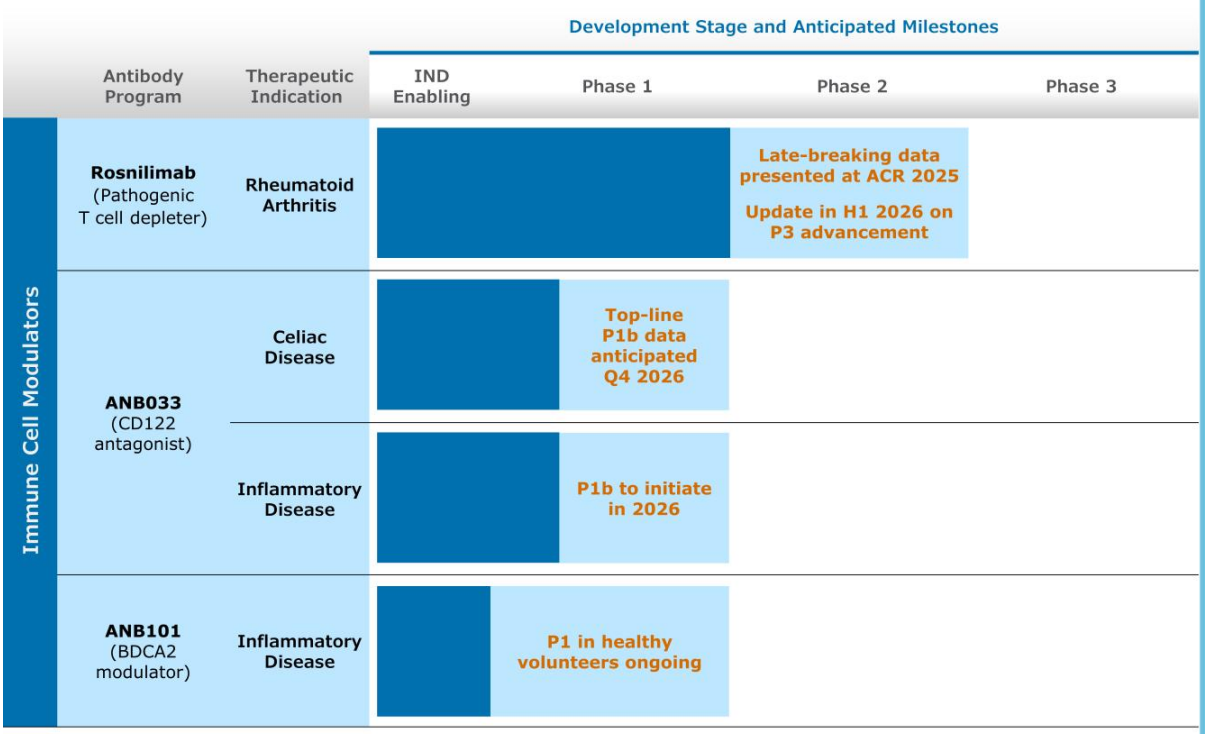
## Royalty Management Co

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: Q3 2025 cash: ~$257MM. Anticipate ending 2025 with approximately $300MM if including an anticipated accrual of a one-time $75MM commercial sales milestone in Q4 2025 from GSK once *Jemperli* achieves $1 billion in worldwide net sales.
Biopharma Co. to launch with adequate capital to fund operations for at least two years through significant potential corporate milestones

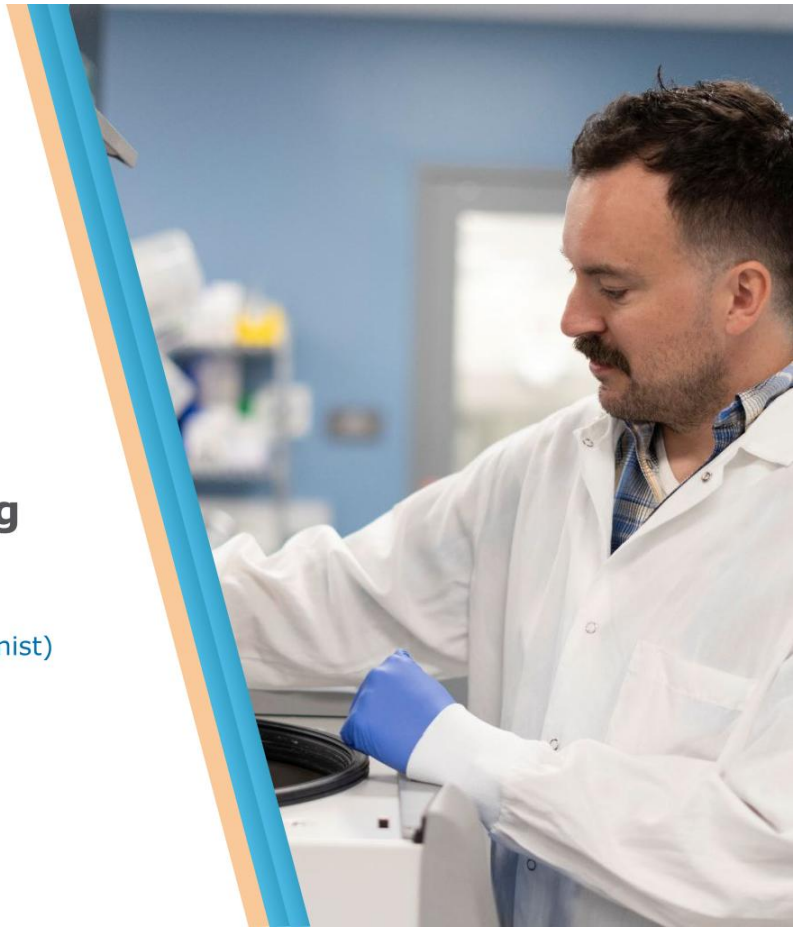
# Biopharma Co would retain leading pipeline of immune cell modulating antibodies with significant upcoming catalysts



# Royalty-Bearing Assets

*Jemperli*™
(dostarlimab, PD-1 Antagonist)

Imsidolimab
(IL-36R antagonist)

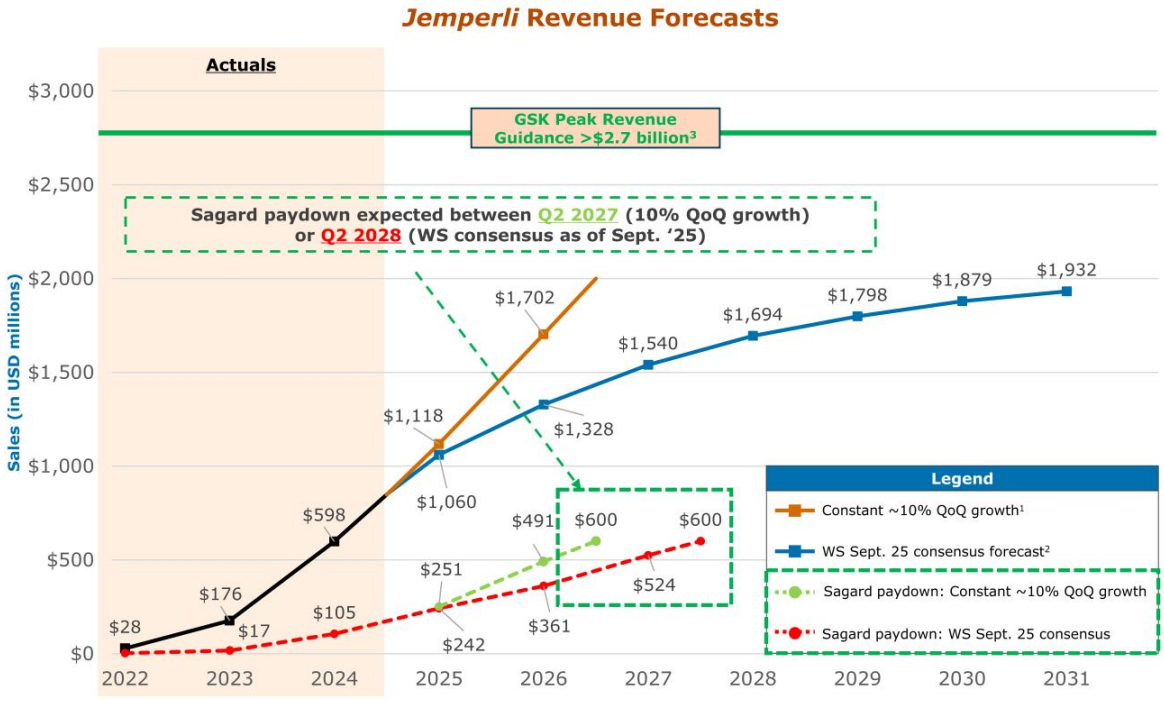


# Royalty Management Co would protect and return value to shareholders

| *Jemperli:* GSK Financial Collaboration | Imsidolimab: Vanda Financial Collaboration |
|---|---|
| • Q3 2025 sales: $303 million (>16% US QoQ growth rate)<br>  ○ >$1.2b annualized run rate[1]<br>• Significant royalties on global net sales<br>  ○ 8% ($0 to $1b), 12% ($1 - $1.5b), 20% ($1.5 - $2.5b), and 25% (>$2.5b)<br>• >$390 million per year at GSK's peak sales guidance of >$2.7 billion[2], which Anaptys expects to be achieved before 2031<br>• Anticipate Sagard paydown between Q2 2027 and Q2 2028 projected from *Jemperli's* continued strong growth rate<br>• Substantial ongoing investment in additional indications for *Jemperli* monotherapy and combos<br>  ○ H2 2026: top-line data from registrational dMMR rectal trial (national priority voucher) | • 10% royalty on global net sales<br>• $35 million in future milestones<br>  ○ $5 million – FDA approval in GPP<br>  ○ $5 million – EMA approval in GPP<br>  ○ $25 million – $100 million annual sales milestone<br>• FDA BLA submission for GPP expected in Q4 2025 |

1. GSK Q3 2025 earnings presentation, US dollar conversion
2. 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 far more than £2 billion."*

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




1. Actual *Jemperli* Q2 to Q3'25 QoQ growth was 16%, Forecast assumes constant ~10% QoQ sales growth from Q3'25 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 far 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 GSK projecting >24,000 drug-treated advanced/ recurrent endometrial cancer patients[1]
- Registrational trials ongoing in Japan and China

### 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
  - National priority voucher granted
- **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

**ADC combination opportunities**
- *P2 combination data to be shared in H1 2026*

**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. GSK Q3 2025 earnings epidemiology report

# 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<br>12% - $1.0 to $1.5 billion<br>20% - $1.5 to $2.5 billion<br>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

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 Q3'25 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

# 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**

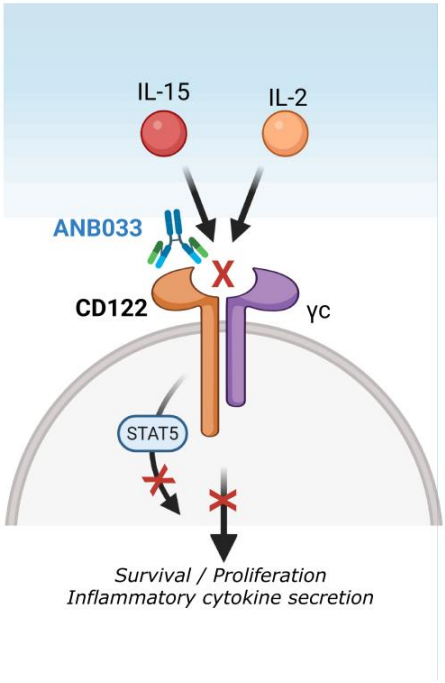
- Expressed on subsets of CD8+ and CD4+ T cells and NK cells

**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.

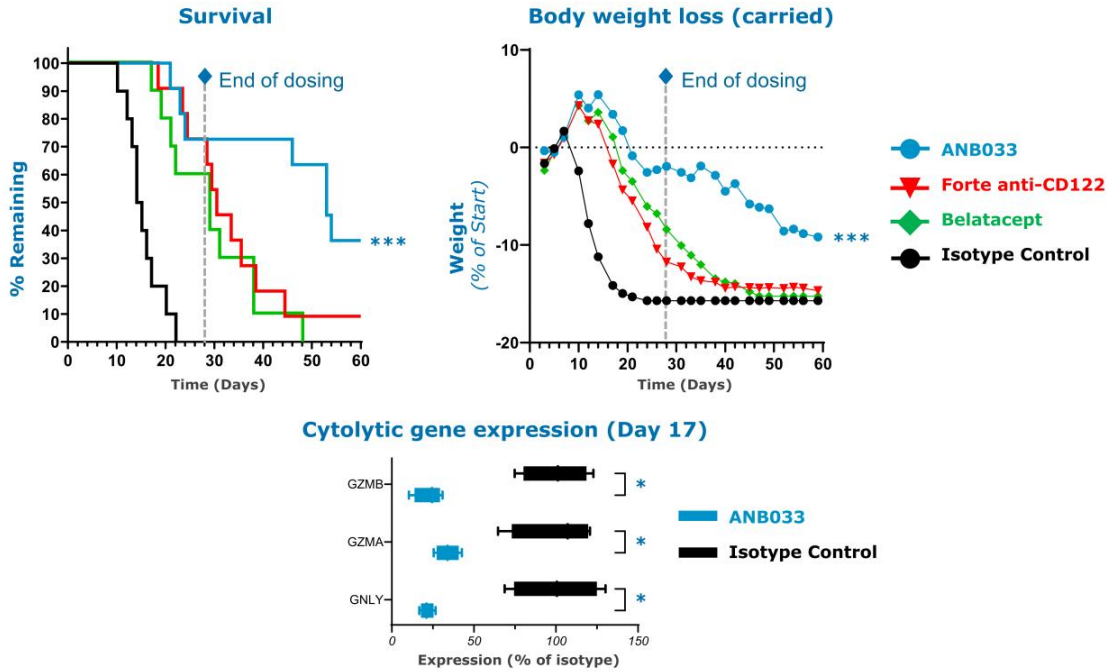
# Broad therapeutic potential across autoimmune and inflammatory diseases

| ***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<br>• P1b PoC in EoE<br>• P2a in atopic dermatitis |
| teva | IL-15 | • P2a in CeD<br>• P2a in vitiligo |
| FORTE | CD122 | • Positive P1b in CeD (P2a ongoing)<br>• P1b in vitiligo and alopecia areata<br>• 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

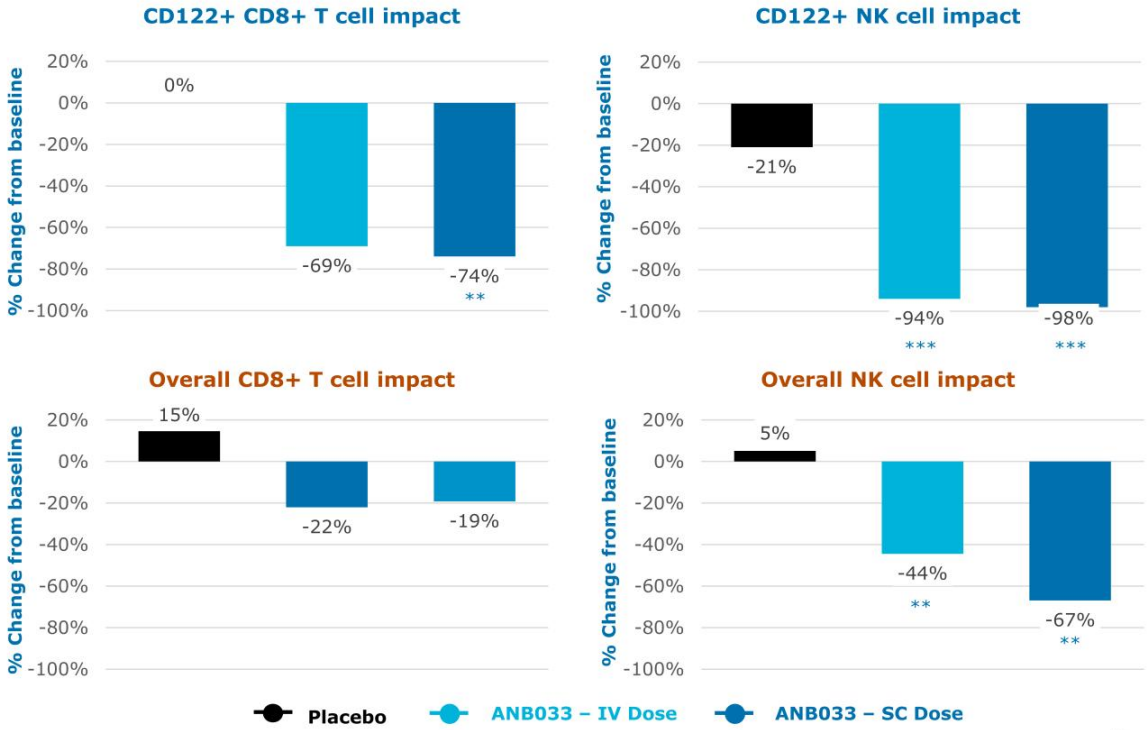
### Rapid and sustained PK profile

- 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

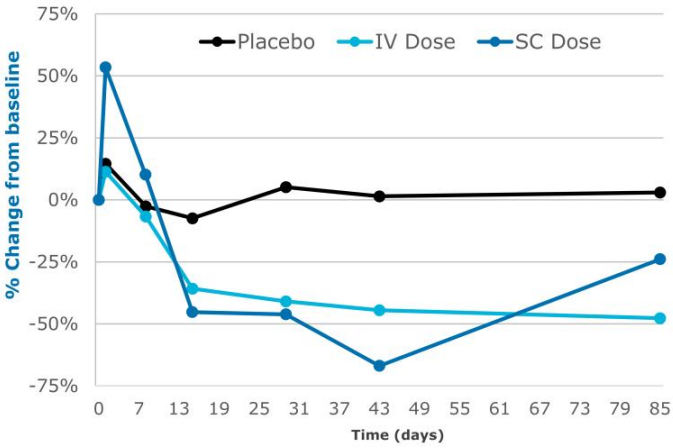
Effect of ANB033 is limited to CD122 expressing cells

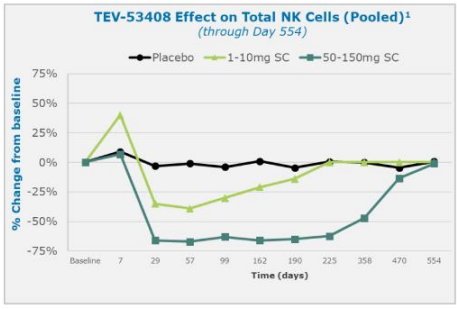



Graphs reflect SAD data and maximum reductions were achieved within the first 43 days. *** p<0.001 **p<0.01

# 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**



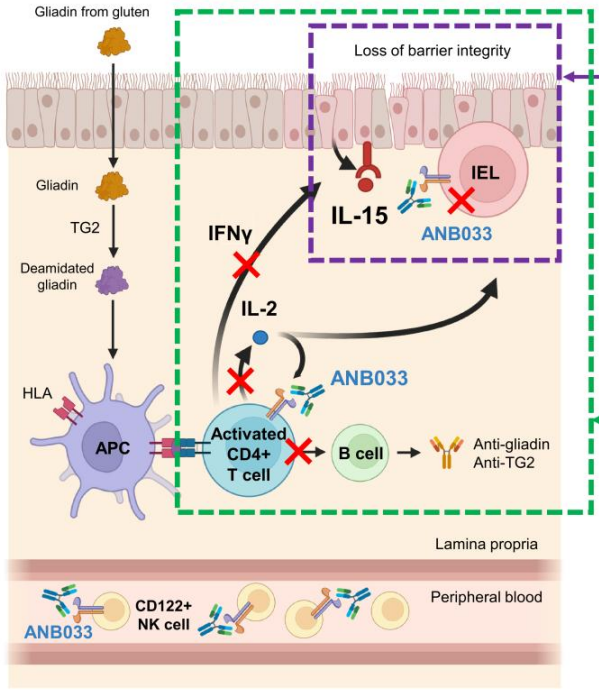

**No safety signals observed in any CD122 or IL-15 trials to date after NK cell reduction**

- 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

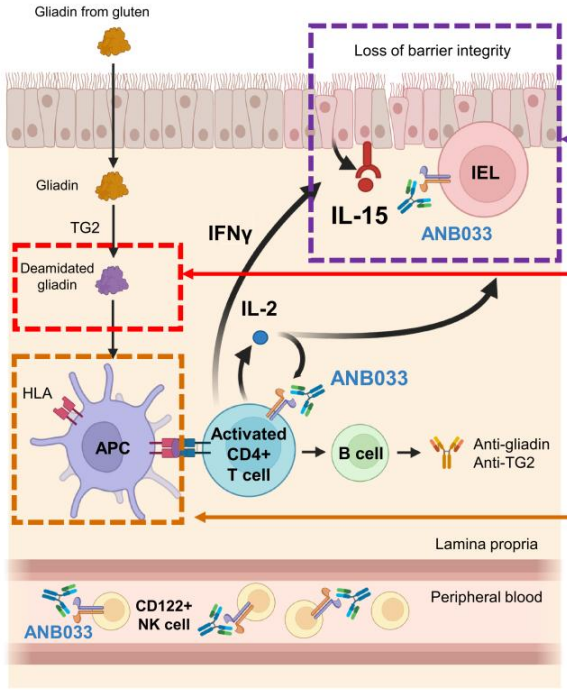
**Inhibition of IL-2 signaling**

- 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**

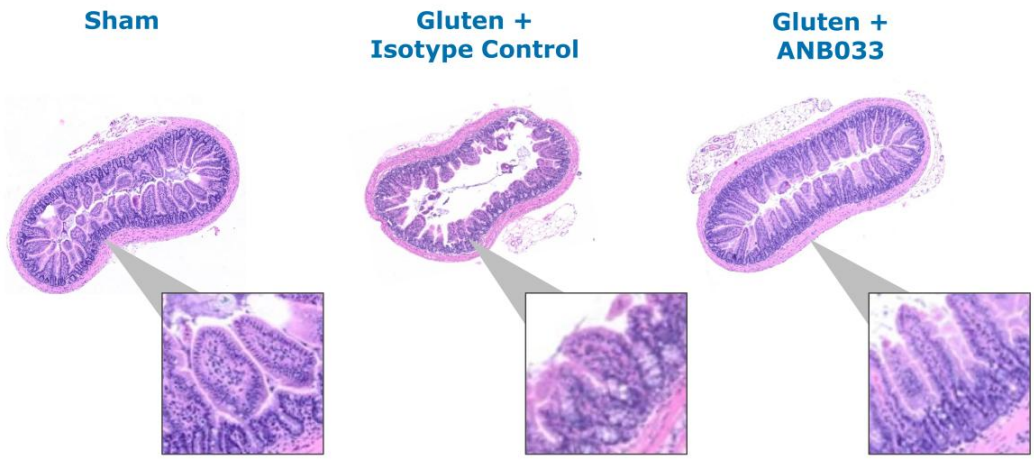
- Takeda — *P2 ongoing; Gluten tolerance*
- astellas — *Discontinued; Gluten tolerance*
- Roche — *P1 ongoing; HLA-DQ2.5 gluten peptide complex*
- Immunic THERAPEUTICS — *P1 ongoing; SIRT6 modulator*

**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



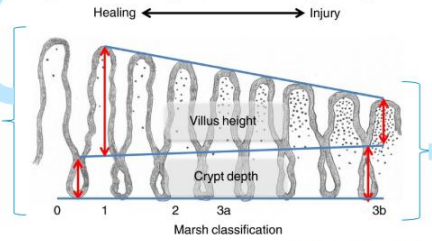

**ANB033 treatment shows improved histology:**
**preserves villus height and crypt depth (Vh:Cd) in CeD mouse model**

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

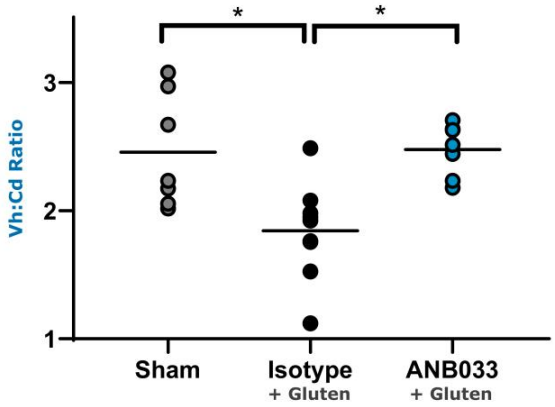
## Vh:Cd ratio

**Healthy tissue:**
High villus height, lower crypt depth




**CeD tissue:**
Reduced villus height, extended crypt depth

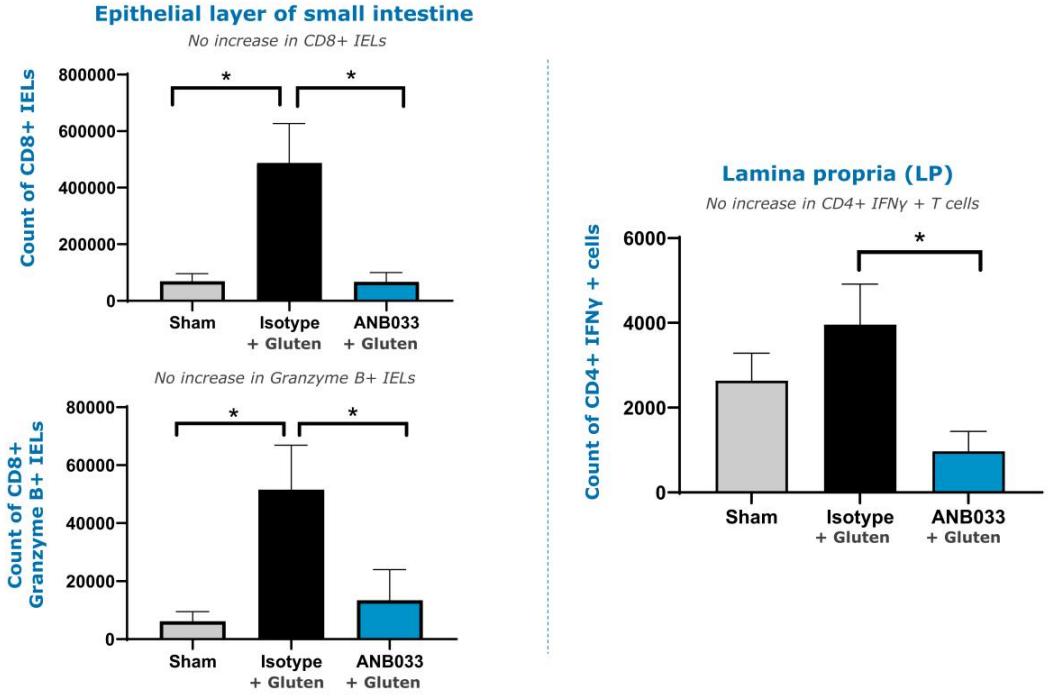
## ANB033 impact on Vh:Cd ratio




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

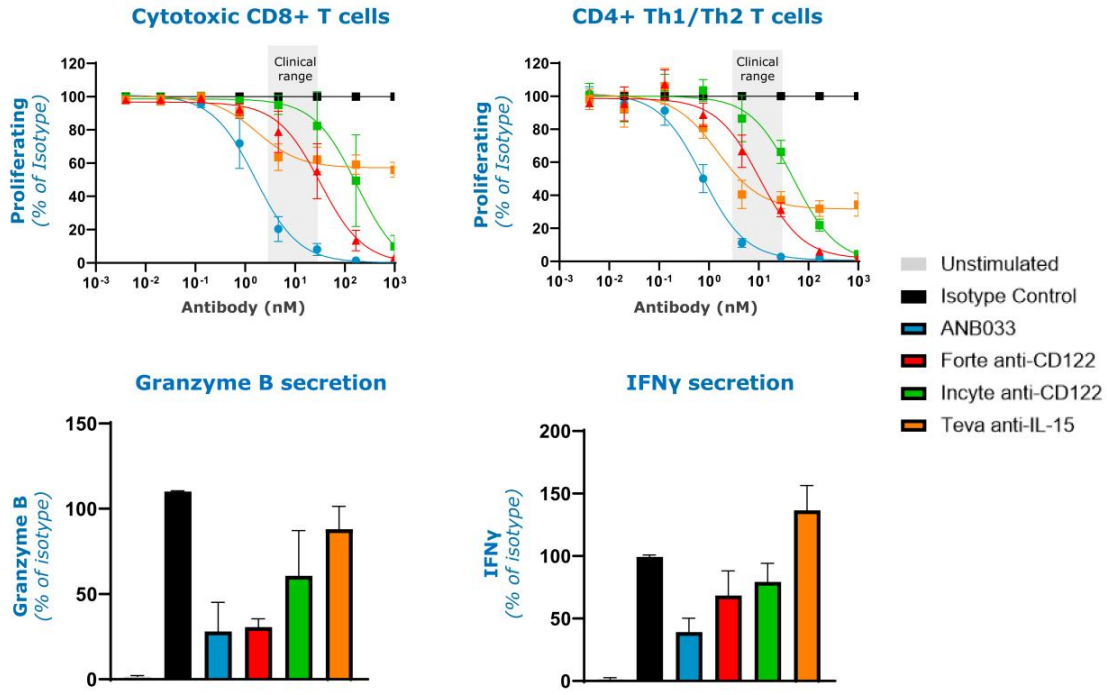
Note: HuDQ8-D$^{d}$-villin-IL-15tg mice on a gluten-free diet are challenged with gluten, and CeD features are analyzed on day 30. The treatment regimen includes a sham (no gluten), isotype control and ANB033 surrogate antibody (anti-mouse CD122 antibody with similar epitope and affinity to ANB033) administered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

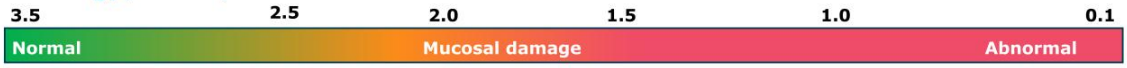
# ANB033 shows differentiated impact in CeD patient-derived PBMCs compared to competing anti-IL-15s and CD122s




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)*




**Symptoms**


**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*

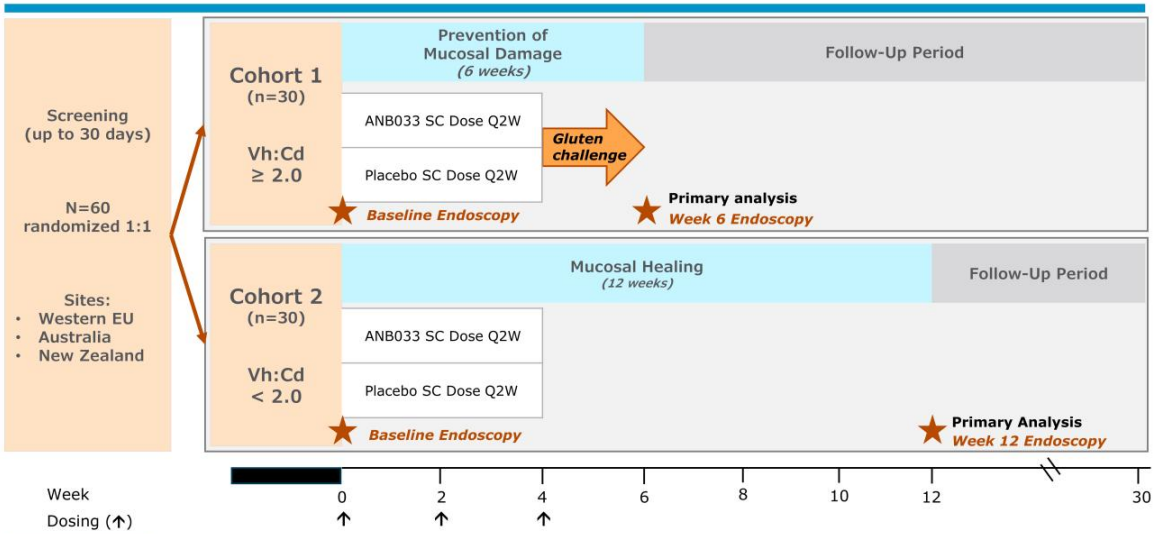
sanofi *(Phase 2b)*

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

GF diet = Gluten free diet.

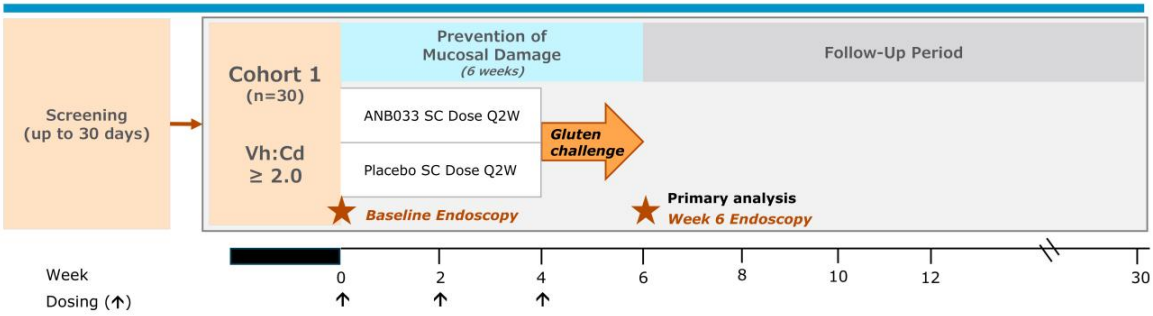
# ANB033 Phase 1b trial in CeD initiated

Anticipate top-line data in Q4 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<br>• IEL count<br>• 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



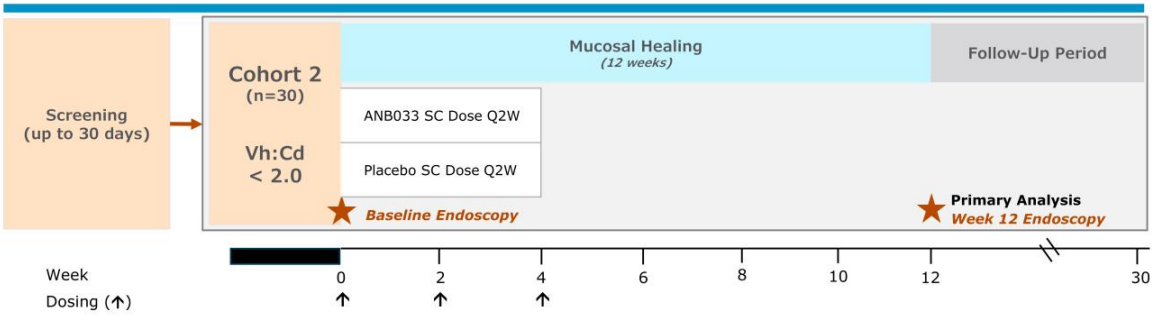

## Minimal evidence of mucosal damage (Vh:Cd ≥ 2.0)

- 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



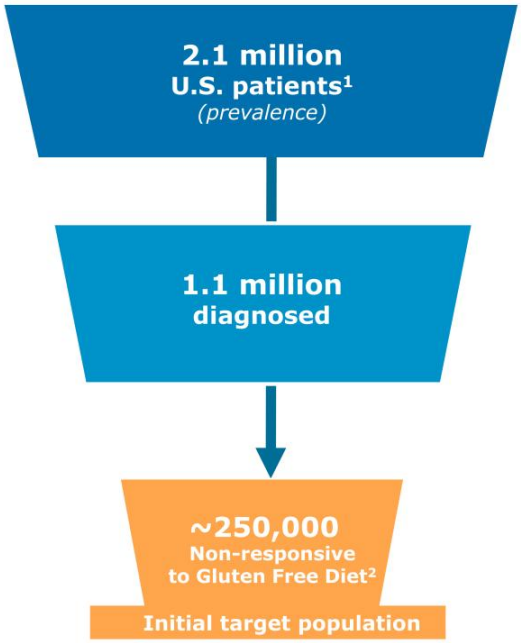

## Persistent evidence of histologic CeD activity (Vh:Cd < 2.0)

- 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**

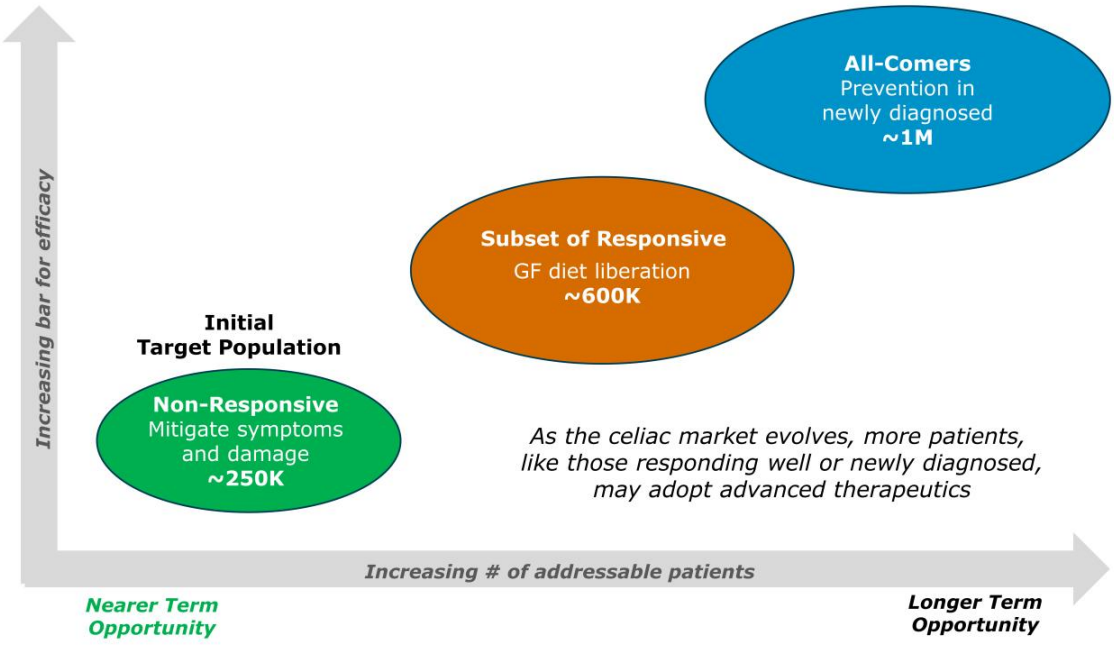
- HCPs favor MOA that targets both symptoms and histology

**$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

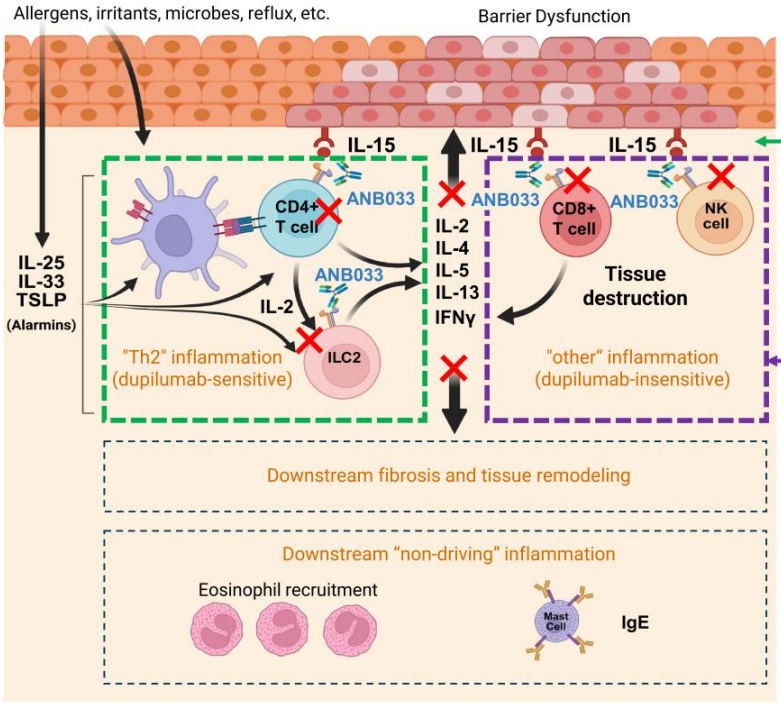
1. Singh et al. (2018), Choung et al. (2016), Katz et al. (2011), Trinity Life Sciences Commercial Assessment HCP Primary Market Research (2025). CeD sizing reflects future US market in 2030 assuming growth in diagnosis rate based on historic trends and projected growth with entrance of novel therapies 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

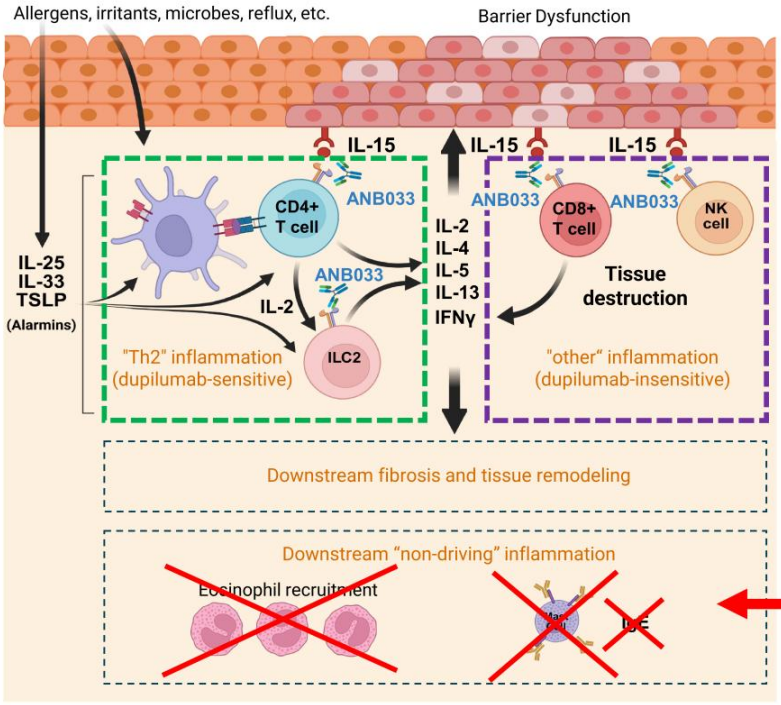
**CD8+ and NK (dupilumab-insensitive)**

- 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**

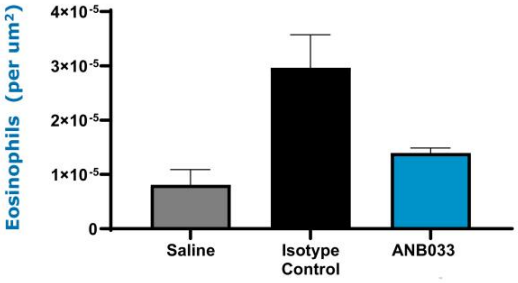
anti-cKIT, anti-IL-5Ra, anti-IgE

Celldex Genentech AstraZeneca

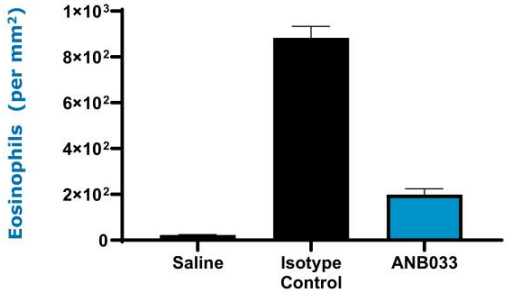
Adapted from Discepolo et. al. Gastroenterology. 2024; 167:90-103.

# ANB033 prevents eosinophilia by targeting upstream inflammation

### Esophageal eosinophils



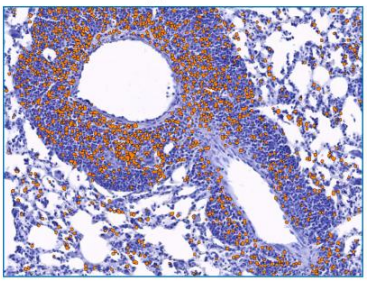

### Lung eosinophils



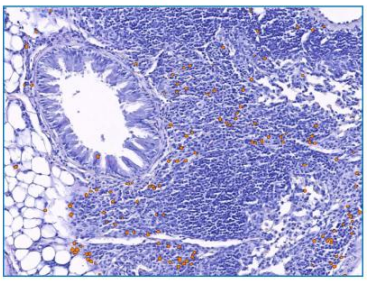

### Aspergillus-induced eosinophilia

*(Lung sample shown)*

**Isotype Control**

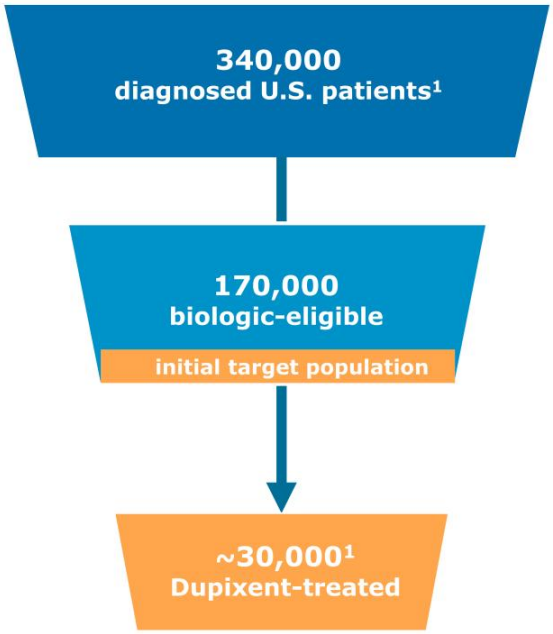


**ANB033**



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.

# Rosnilimab

*(Pathogenic T Cell Depleter)*

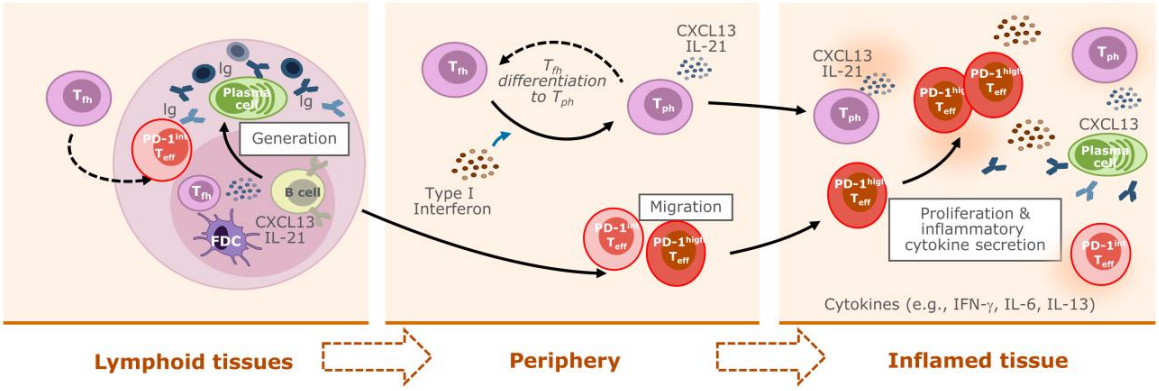
# Pathogenic $T_{eff}$ and $T_{fh}/T_{ph}$ cells mediate autoimmune pathology

**$T_{fh}$** (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

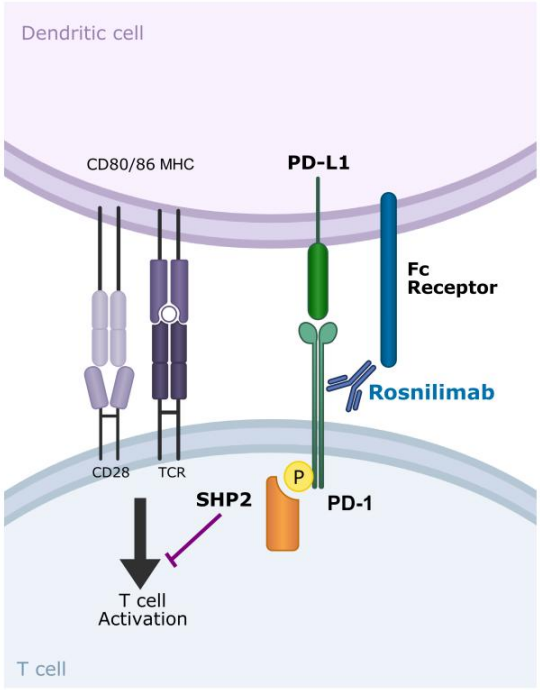
**$T_{eff}$** (effector)

- 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.

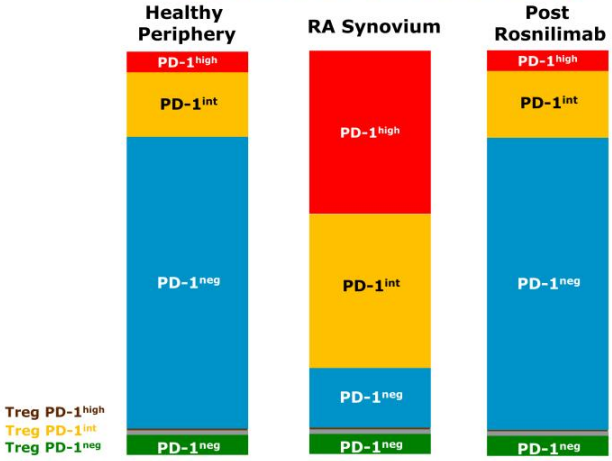
# Rosnilimab selectively targets pathogenic T cells in periphery and inflamed tissue to restore immune homeostasis




**Rosnilimab aims to:**

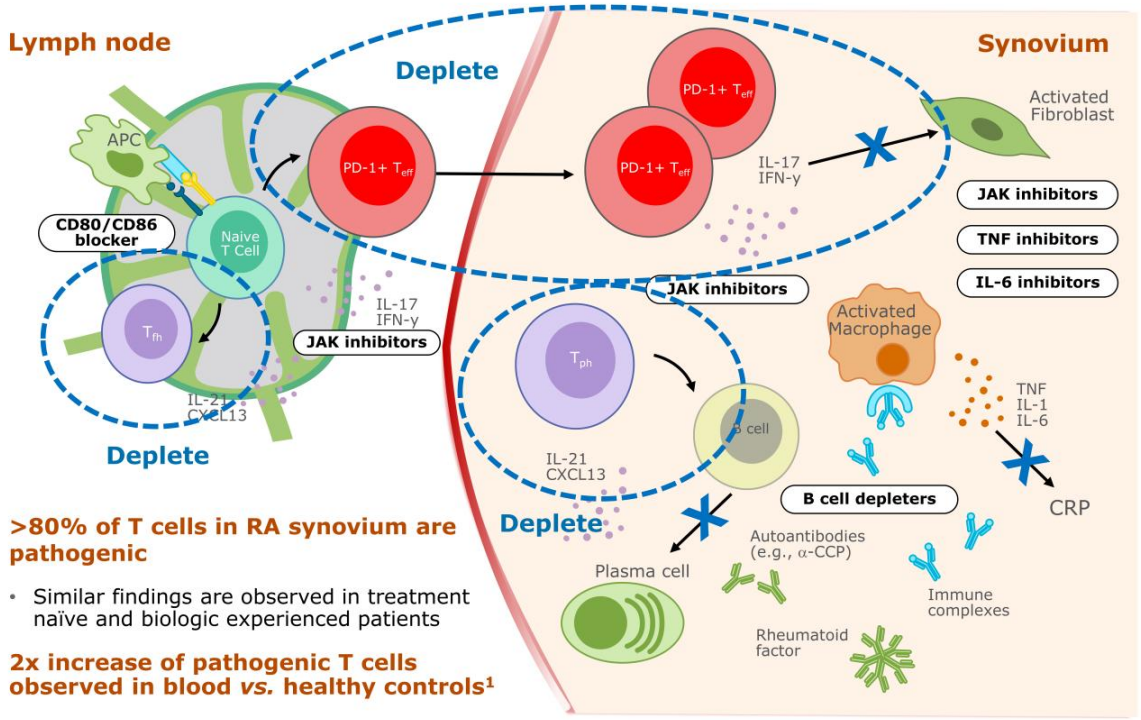
1. Leverage natural immune regulatory pathway to safely restore immune homeostasis
2. Achieve durable remission and modify disease

**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**

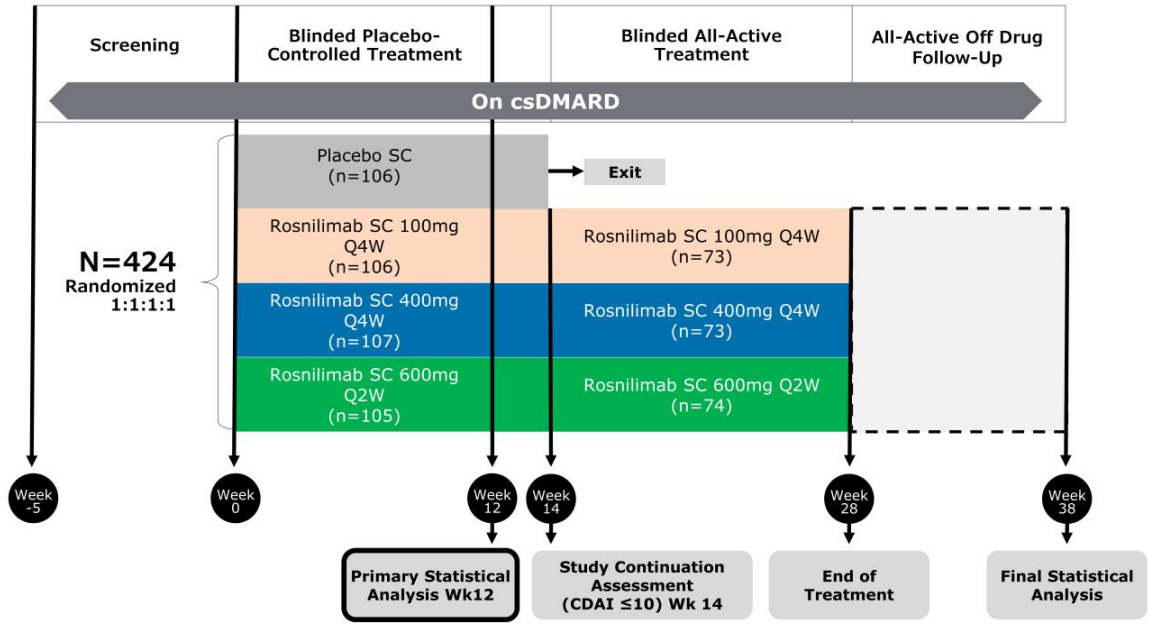
- Similar findings are observed in treatment naïve and biologic experienced patients

**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

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




**Key Inclusion Criteria** – Seropositive RA, ≥6 swollen and ≥6 tender joints, hs-CRP ≥ 3mg/L during Screening, Concurrent use of 1 or 2 csDMARDs that were initiated at least 3 months before screening

**Key Exclusion Criteria** – Inadequate response, loss of response, or intolerance to any combination of ≥ 3 b/tsDMARD classes

**Primary Endpoint** - Mean change from baseline at Week 12 for DAS28-CRP

# 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
- Similar responses observed across more stringent endpoints regardless of prior therapy type, including JAKs
- 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**

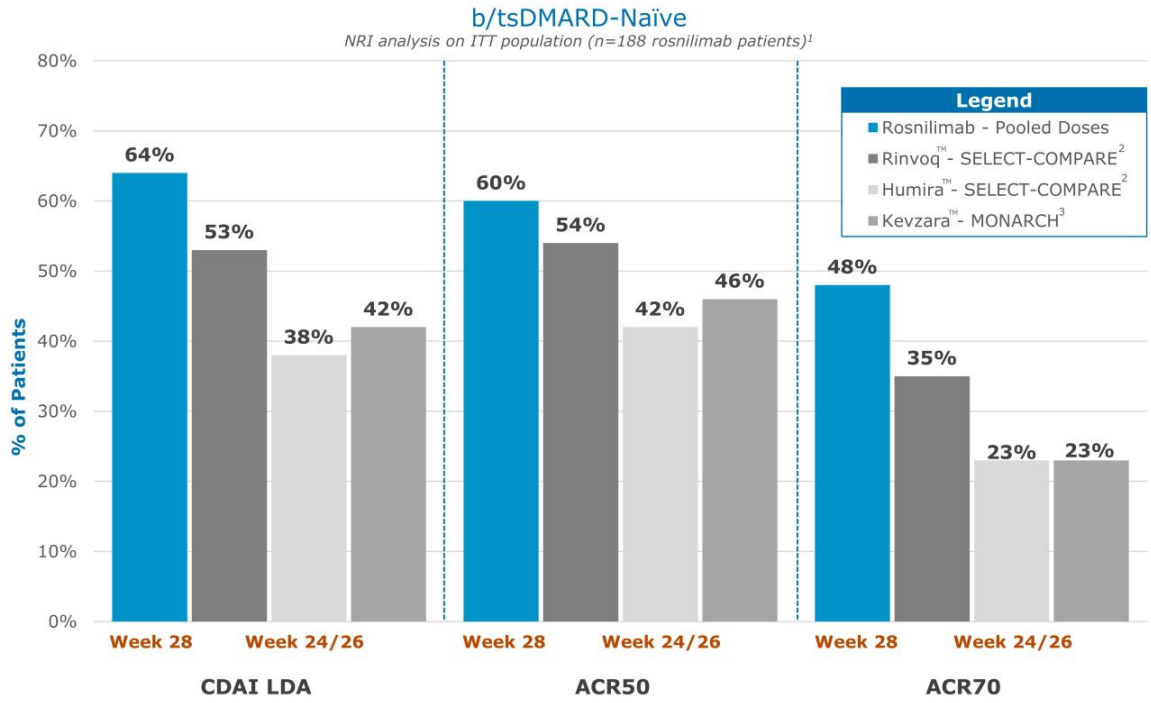
## Responses durable for at least 3-months off-drug

- 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

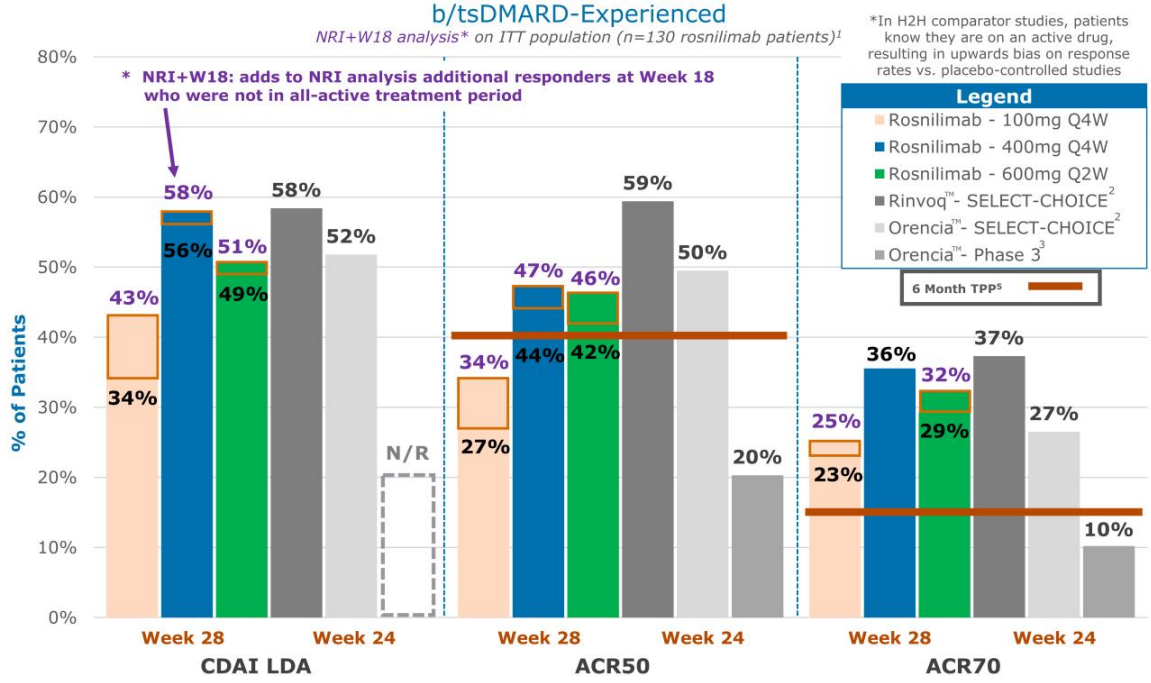



1. Non-responder imputated (NRI) analysis on intent-to-treat (ITT) of all b/tsDMARD-naïve patients randomized; b/tsDMARD-naïve population (n=62 100mg Q4W, n=62 400mg Q4W, n=64 600mg Q2W; n=188 total rosnilimab b/tsDMARD-naïve patients); 2. SELECT-COMPARE Phase 3 study; 3. Kevzara Phase 3 study; NRI data; 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

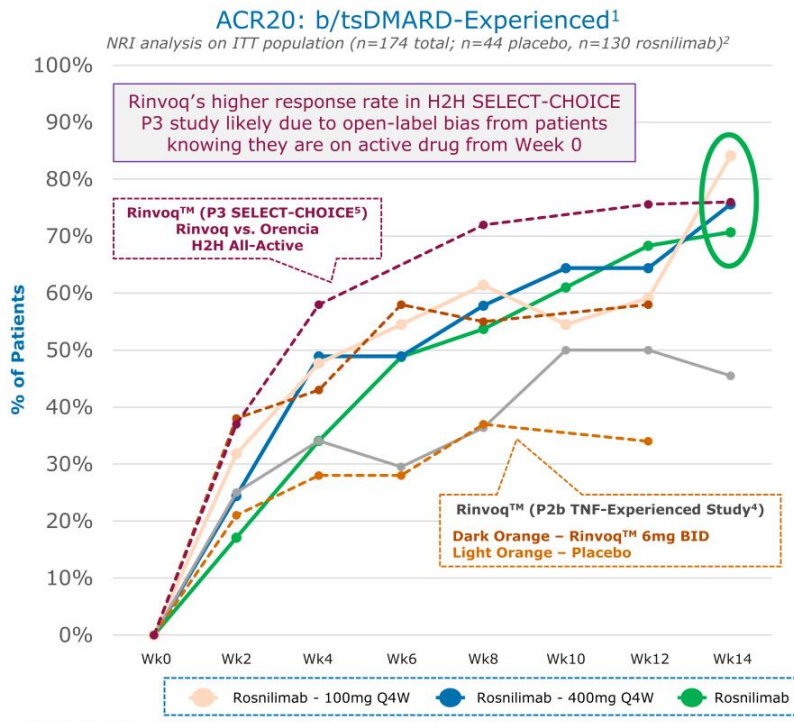



1. Non-responder imputated (NRI) analysis on intent-to-treat (ITT) of all b/tsDMARD-experienced patients randomized; b/tsDMARD-experienced population (n=44 100mg Q4W, n=45 400mg Q4W, n=41 600mg Q2W; n=130 total rosnilimab b/tsDMARD-experienced patients); 2. SELECT-CHOICE Phase 3 study; 3. Orencia Phase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

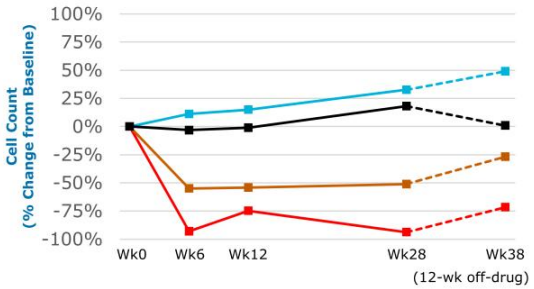
*MMRM Analysis on ITT population (n=174 total; n=44 placebo, n=130 rosnilimab)*


| 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* |

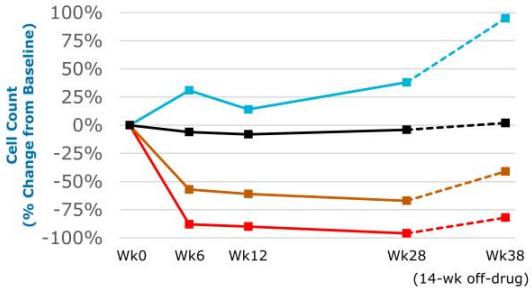
1. b/tsDMARD-experienced population included 29% (n=50 of n=174 total experienced patients) with prior JAK experience; 2. Mixed Model for Repeated Measures (MMRM) analysis on intent-to-treat (ITT) of all b/tsDMARD-experienced patients randomized; b/tsDMARD-experienced population (n=44 placebo, n=44 100mg 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

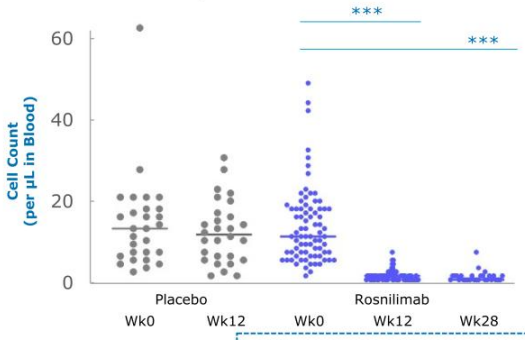
## Rosnilimab 400mg Q4W T Cell Impact



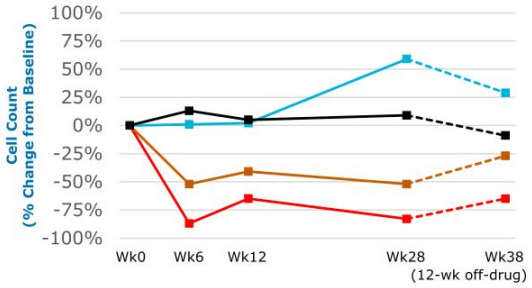

## Rosnilimab 600mg Q2W T Cell Impact




## Rosnilimab $T_{ph}$ Impact – Pooled Doses



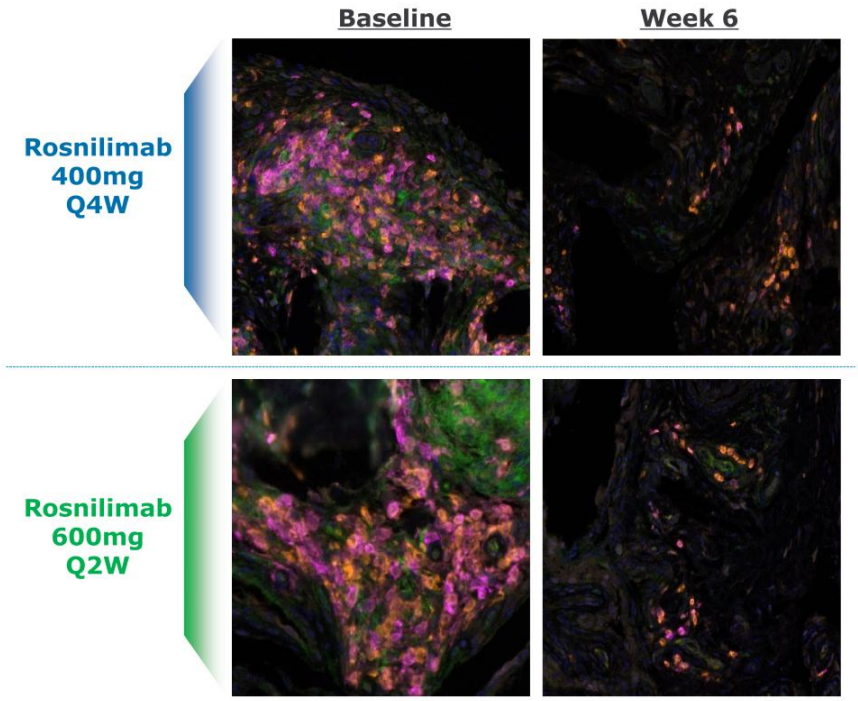

## Rosnilimab 100mg Q4W T Cell Impact





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




**Impact on $T_{ph}$ and $T_{eff}$ cells**

**400mg/600mg doses**
*~90% reduction*

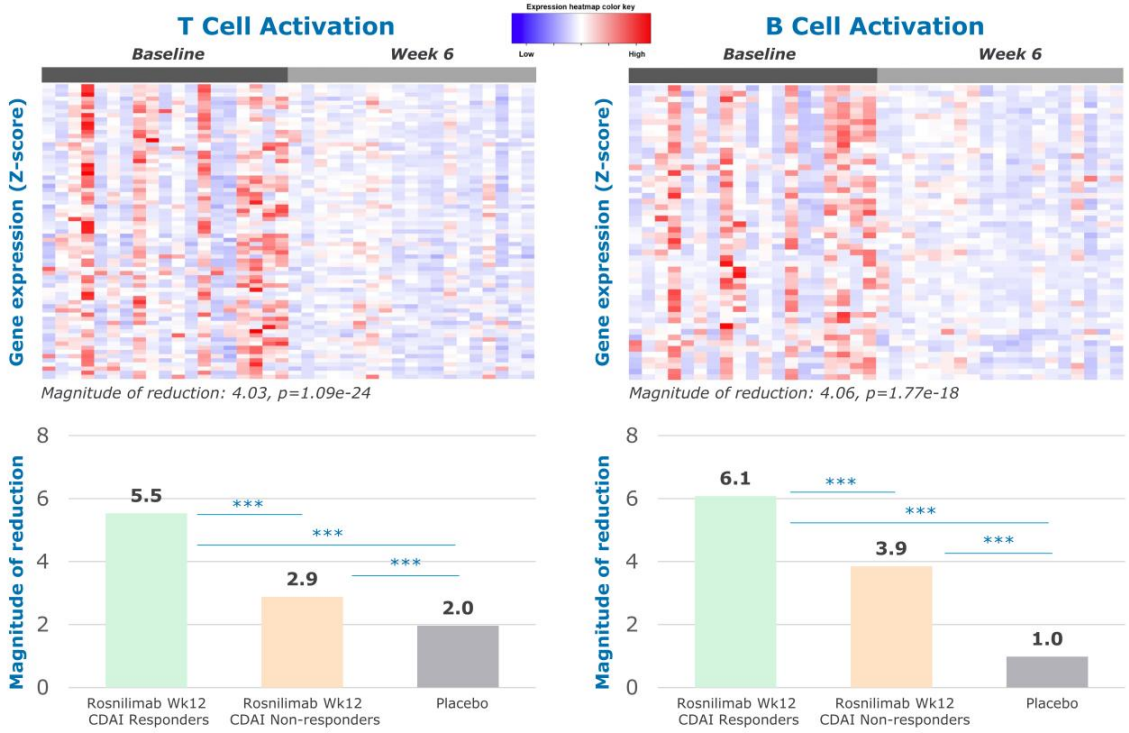
**100mg dose**
*Inconclusive reduction*

**Placebo**
*Increased*

| T cell markers | APC markers |
|---|---|
| CD3 | PD-L1 |
| PD-1 | |
| CXCR5 | |

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

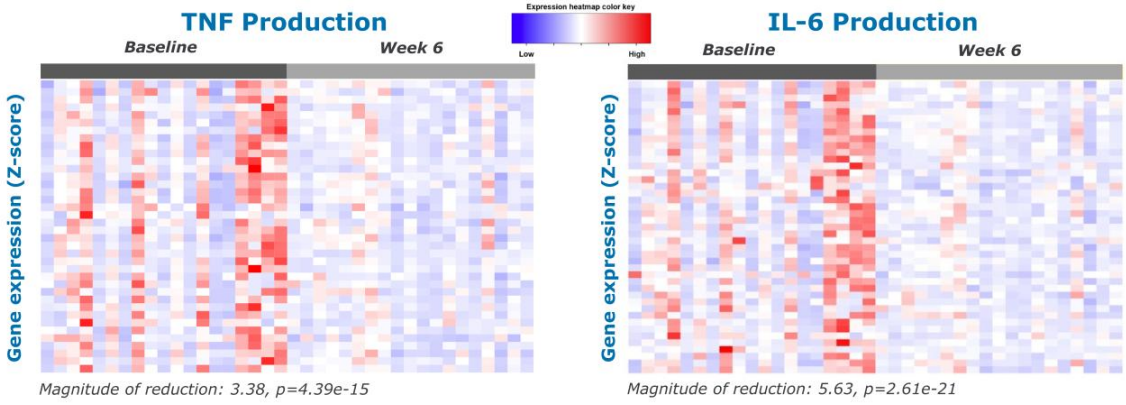






Note: Gene ontology (GO) pathway analysis performed on samples with evidence of inflammation at baseline (all rosnilimab doses pooled, n=19 paired biopsies) and with myosin normalization. Rows reflect genes with p<0.05 between Weeks 6 and 0. Magnitude of reduction defined as fold enrichment score. Rosnilimab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

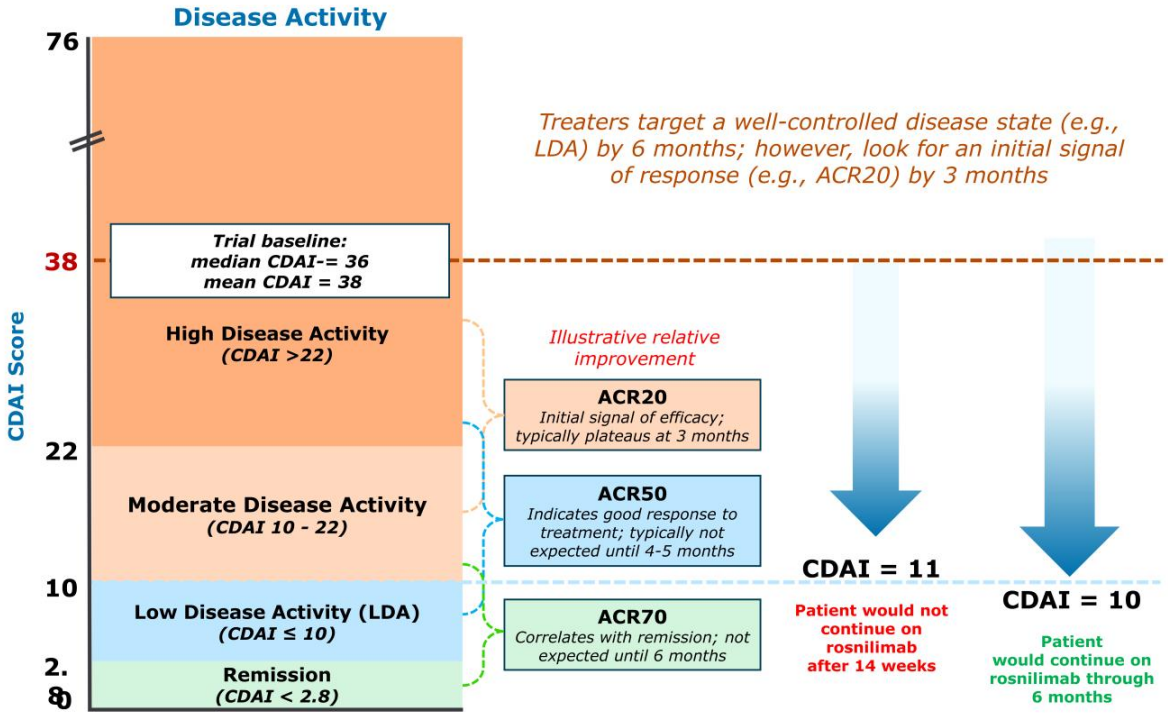
## Pathway changes reflect rosnilimab's broad MOA

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

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

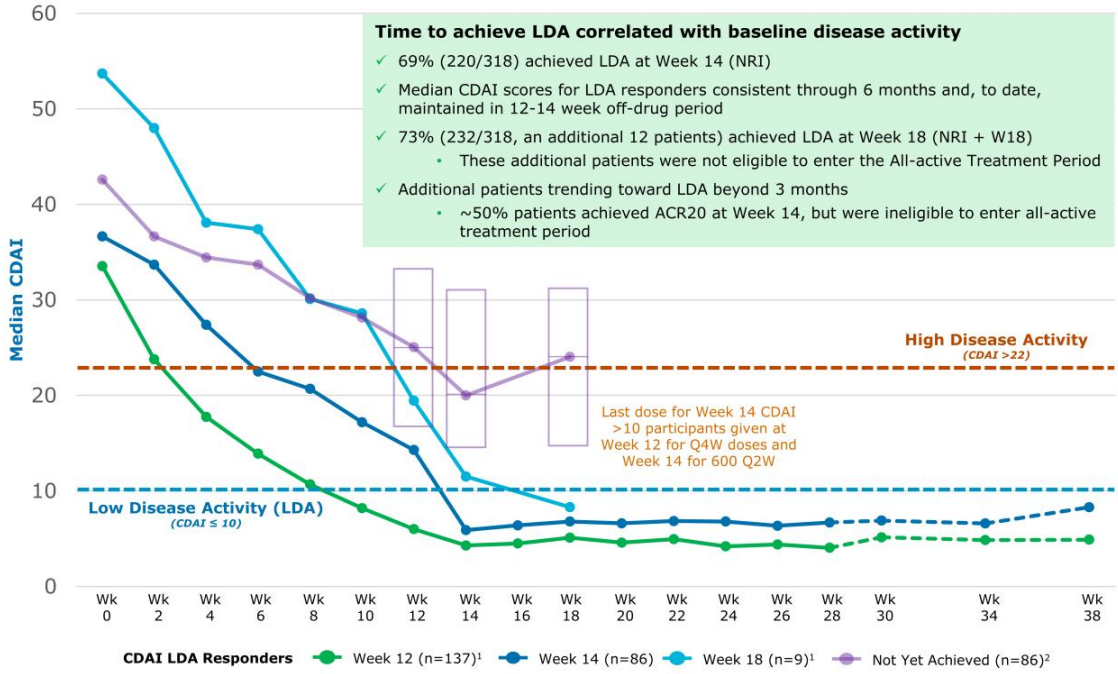


# Max response was not achieved in this Phase 2b trial

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)*

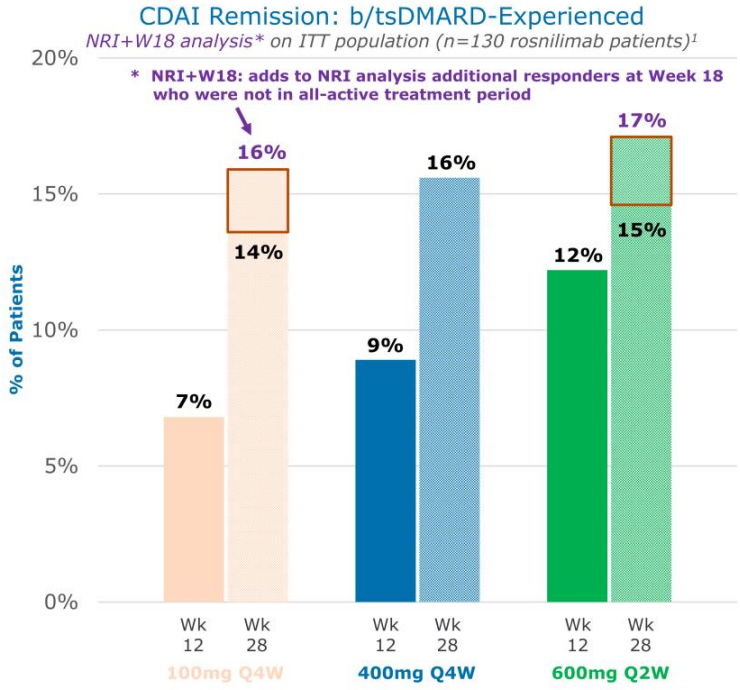



1. Green line includes 3 patients that achieved LDA at Week 12, were not CDAI LDA at Week 14, but returned to CDAI LDA at Week 18. These same 3 patients were excluded from the Light Blue line. In total 12 patients achieved CDAI LDA at Week 18. 2. Purple line includes rosnilimab patients that discontinued 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

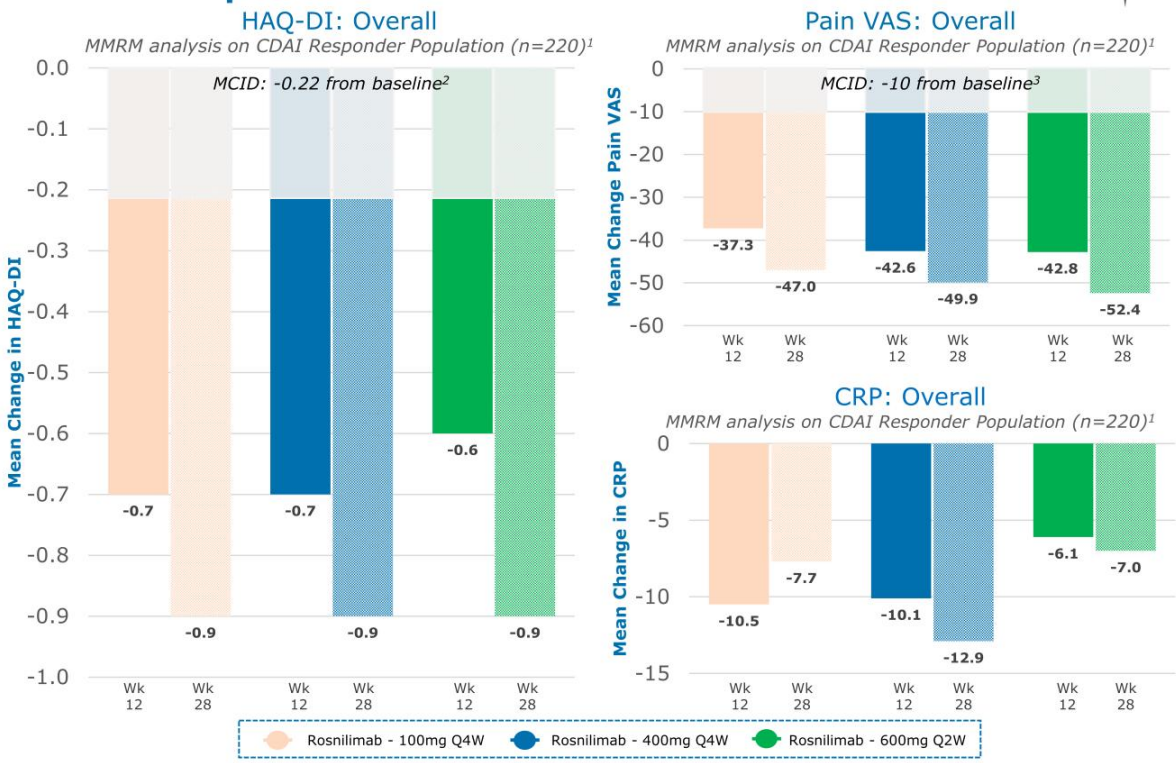
Excludes 2 patients who discontinued in the all-active treatment period while in CDAI remission




| 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% |

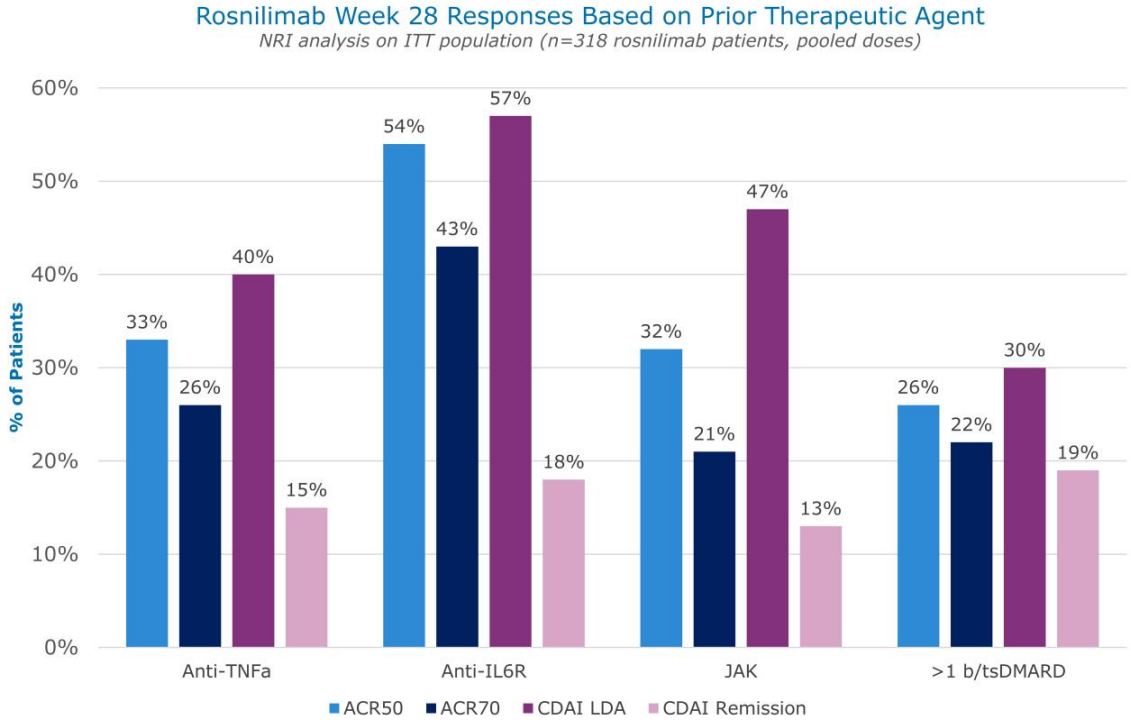
1. Non-responder imputated (NRI) analysis on intent-to-treat (ITT) of all b/tsDMARD-experienced patients randomized; b/tsDMARD-experienced population (n=44 100mg Q4W, n=45 400mg Q4W, n=41 600mg Q2W; n=130 total rosnilimab b/tsDMARD-experienced patients)

# 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

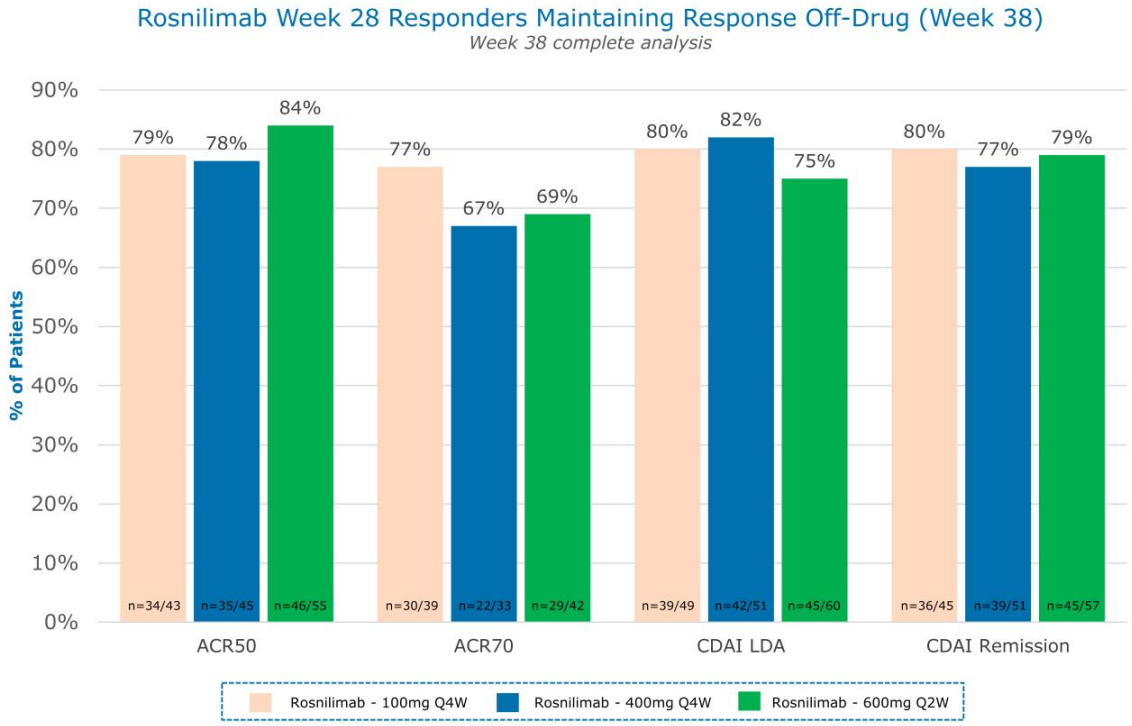
# Similar responses observed across more stringent endpoints regardless of prior therapy type, including JAKs




Graf et. al, "Rosnilimab, a Selective and Potent Depleter of Pathogenic T Cells, Demonstrates Efficacy, Safety and Translational Proof of Mechanism in a Rheumatoid Arthritis Phase 2B Trial", ACR Convergence, October 2025

# Durable responses for 3-months off-drug

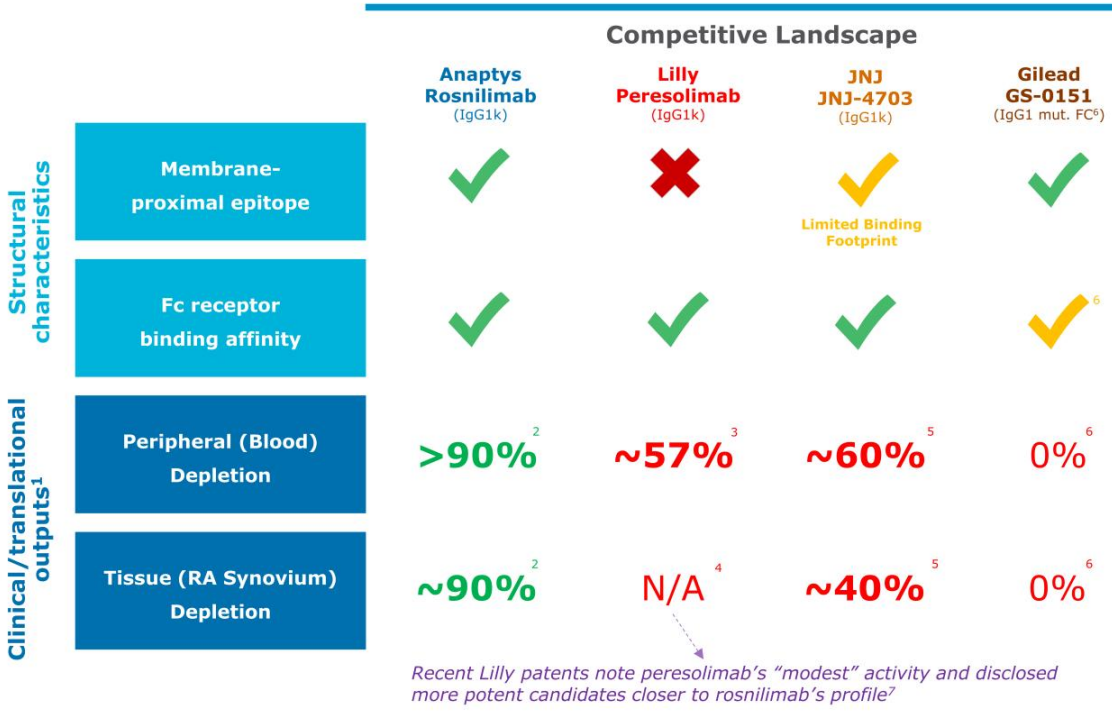
82% of Week 28 CDAI LDA responders were still in response at Week 38




Graf et. al, "Rosnilimab, a Selective and Potent Depleter of Pathogenic T Cells, Demonstrates Efficacy, Safety and Translational Proof of Mechanism in a Rheumatoid Arthritis Phase 2B Trial", ACR Convergence, October 2025

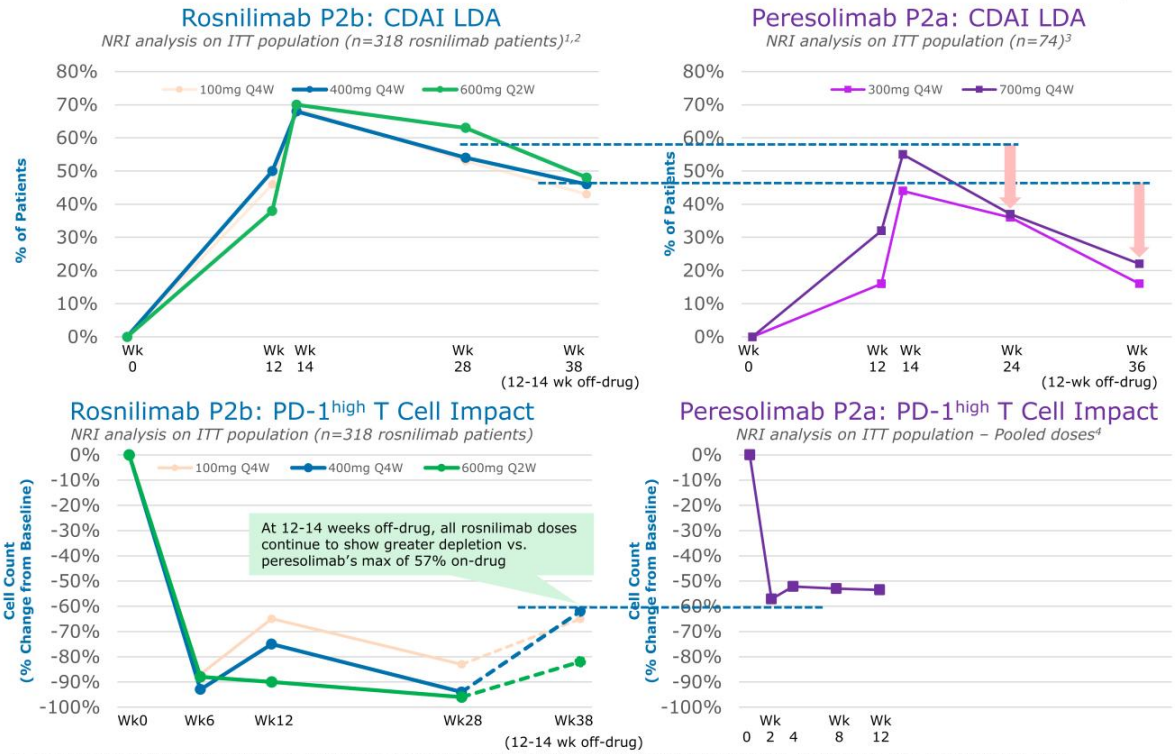
# Rosnilimab is a best-in-class pathogenic T cell depleter

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. 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

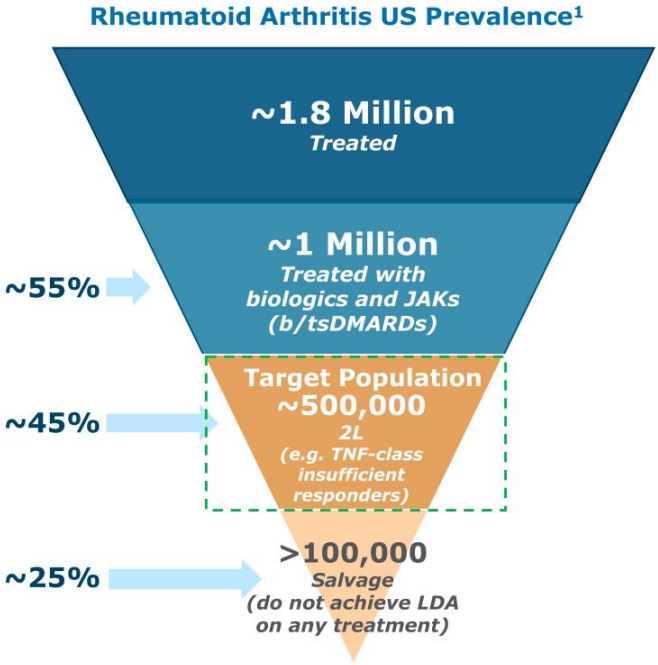
<2% dropout rate overall due to AEs through 6 months,
with only 1 dropout due to AE (headache-moderate) after 3 months

| Study Period | Week 0 through Week 12 (N=424) | | | | Week 0 through Week 38 (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%)** | **47 (152.7)** | **75 (238.3)** | **69 (190.4)** | **57 (140.1)** |
| Any SAE | 1 (1%) | 1 (1%) | 1 (1%) | 3 (3%) | 1 (2.4) | 3 (4.5) | 5 (7.3) | 4 (6.1) |
| 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%) | 1 (1%) | 0 (0%) | 4 (4%) | 3 (7.1) | 4 (6.0) | 3 (4.4) | 4 (6.1) |
| Drug-Related AE | 18 (17%) | 13 (12%) | 18 (17%) | 17 (16%) | 19 (51.2) | 17 (29.1) | 28 (49.5) | 20 (35.4) |
| AE Leading to Treatment Discontinuation | 1 (1%) | 1 (1%) | 2 (2%) | 2 (2%) | 1 (2.4) | 1 (1.5) | 3 (4.4) | 2 (3.0) |
| Infections | 14 (13%) | 24 (23%) | 21 (20%) | 12 (11%) | 23 (60.2) | 43 (87.3) | 43 (83.8) | 35 (64.7) |
| Serious | 1 (1%) | 1 (1%) | 0 | 0 | 1 (2.4) | 1 (1.5) | 1 (1.5) | 1 (1.5) |
| Opportunistic | 2 (1.9%) | 0 (0%) | 0 (0%) | 0 (0%) | 2 (4.8) | 1 (1.5) | 1 (1.5) | 1 (1.5) |
| MACE | 0 (0%) | 1 (1.5%) | 0 (0%) | 0 (0%) | 0 (0) | 1 (1.47) | 0 (0) | 0 (0) |
| Malignancies | 0 (0%) | 0 (0%) | 0 (0%) | 0 (0%) | 0 (0) | 0 (0) | 0 (0) | 0 (0) |
| Death | 0 (0%) | 0 (0%) | 0 (0%) | 0 (0%) | 0 (0) | 0 (0) | 0 (0) | 0 (0) |
| **Participants with any AEs > 5%** | | | | | | | | |
| Headache | 4 (4%) | 7 (7%) | 6 (6%) | 4 (4%) | 4 (9.6) | 10 (16.0) | 10 (15.4) | 5 (7.8) |
| Upper respiratory tract infection | 1 (1%) | 7 (7%) | 2 (2%) | 3 (3%) | 2 (4.7) | 14 (22.5) | 7 (10.6) | 12 (19.1) |
| Nasopharyngitis | 4 (4%) | 5 (5%) | 5 (5%) | 0 | 6 (14.4) | 9 (14.0) | 9 (13.8) | 5 (7.6) |
| Elevated ALT (alanine aminotransferase) | 1 (1%) | 4 (4%) | 3 (3%) | 3 (3%) | 1 (2.4) | 8 (12.4) | 4 (6.0) | 4 (6.2) |

** Exposure adjusted incidence rate per 100 person-year = 100 x (Number of subjects with AE in the given period / Total years of exposure in the given period across all subjects at risk for the treatment). All adverse events (AEs) that are summarized above are treatment emergent adverse events. SAE=serious adverse event. N – total number of subjects in analysis set, n – number of subjects in specific category*

Rosnilimab was well tolerated with no safety dose effect
Low rates of treatment discontinuation on account of TEAEs, Serious infections and opportunistic infections (herpes zoster) were balanced with no dose response; 1 MACE in 100 mg group was ischemic stroke in participant with stenosis in common carotid artery; There were no malignancies or deaths; Herpes zoster is the only opportunistic infection reported and none were severe

# RA is substantial opportunity for new class of biologics




**Target population in US generated ~$10 billion in 2021[2]**

- Rituxan/biosimilars (typically salvage therapy) achieves well over $1 billion sales in 3L+ RA despite infection risk

**Fragmented market with lack of established SOC in 2L+**

- No clear treatment of choice after failure of anti-TNFs
- No new therapeutic class launched since JAK inhibitors (Xeljanz) a decade ago (2012)

**Provides opportunity for new class to penetrate**

- Comparable or differentiated efficacy
- Durable responses
- Treatment of salvage population

1. Claims analysis to determine market size based on 5 years of claims history; 2. Evaluate Pharma; 2L = 2nd line.

# Next steps for rosnilimab

To provide an update in H1 2026 on advancement of rosnilimab in RA

**Strategic Next Steps in RA**

- Assessing potential to advance rosnilimab in RA funded by strategic or other sources of capital without diluting our royalties
- Outcome could impact how economic value of rosnilimab is allocated between Royalty Management Co and Biopharma Co

| Rheumatoid Arthritis | Ulcerative Colitis |
|---|---|
| **Positive Phase 2b data reported** | **Top-line Phase 2 data reported** |
| • Best-in-disease profile<br>• Favorable safety and tolerability<br>• JAK-like efficacy through 6 months<br>  ○ Max response rates not yet observed due to trial design<br>• Sustained 12-14 week off-drug responses through 9 months<br>• Late-breaking data presented at ACR 2025 | • Safe and well tolerated with similar adverse event rates vs. placebo<br>  ○ Safety profile through Week 50 remains consistent with Week 12<br>• Observed expected pharmacology, including ~90% depletion of pathogenic T cells<br>• Lack of efficacy at Week 12 do not support further development of rosnilimab in UC<br>  ○ Trial will be discontinued |

- Additional activities in 2026+
  - P3 enablement in RA: drug supply scale-up and end-of-phase 2 regulatory interactions

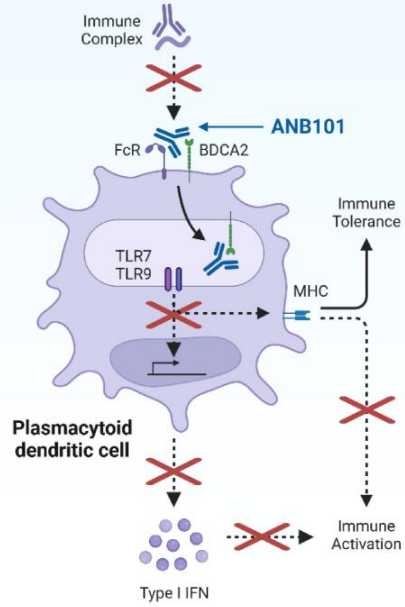
# ANB101

*(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

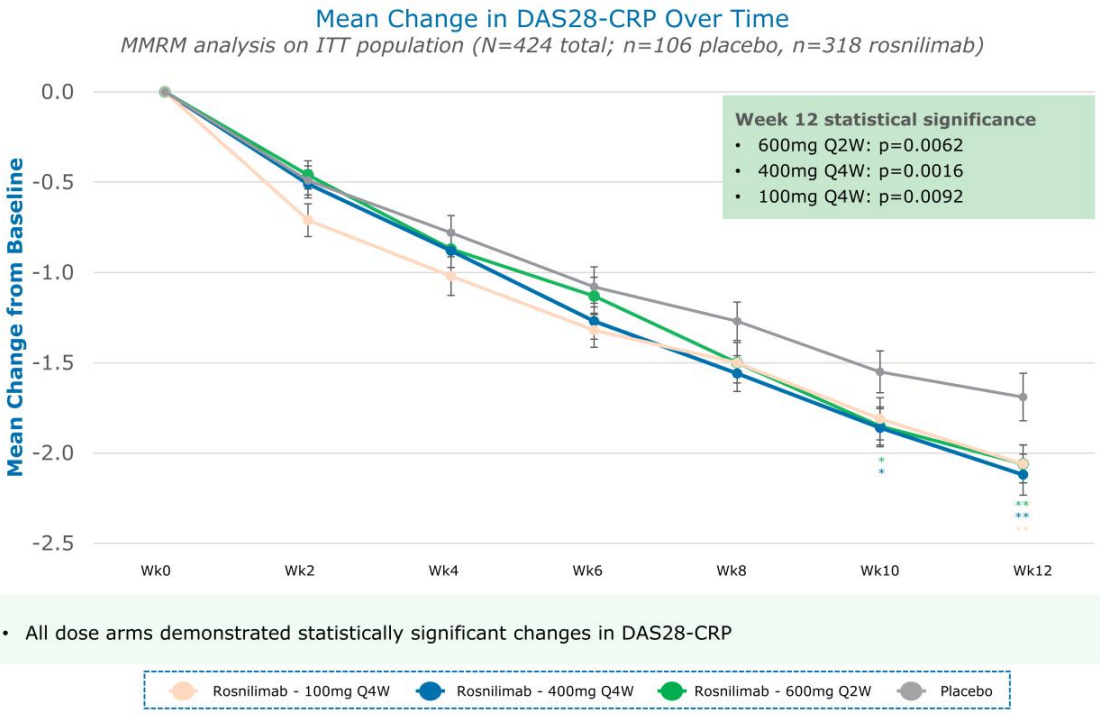
# Appendix

# 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 (%)<br>US<br>Ex-US | <br>35 (33%)<br>71 (67%) | <br>34 (32%)<br>72 (68%) | <br>35 (33%)<br>72 (67%) | <br>26 (25%)<br>79 (75%) | <br>130 (31%)<br>294 (69%) |
| Race, n (%)<br>White<br>Black or African American<br>Asian<br>Other | <br>102 (96%)<br>3 (3%)<br>0 (0%)<br>1 (1%) | <br>102 (96%)<br>1 (<1%)<br>0 (0%)<br>3 (4%) | <br>103 (96%)<br>4 (4%)<br>0 (0%)<br>0 (0%) | <br>101 (96%)<br>4 (4%)<br>0 (0%)<br>0 (0%) | <br>408 (96%)<br>12 (3%)<br>0 (0%)<br>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)<br>CDAI >22, n (%) | 37.9 (10.2)<br>101 (95%) | 37.2 (10.6)<br>101 (95%) | 37.1 (10.6)<br>102 (95%) | 38.6 (11)<br>100 (95%) | 37.7 (10.6)<br>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

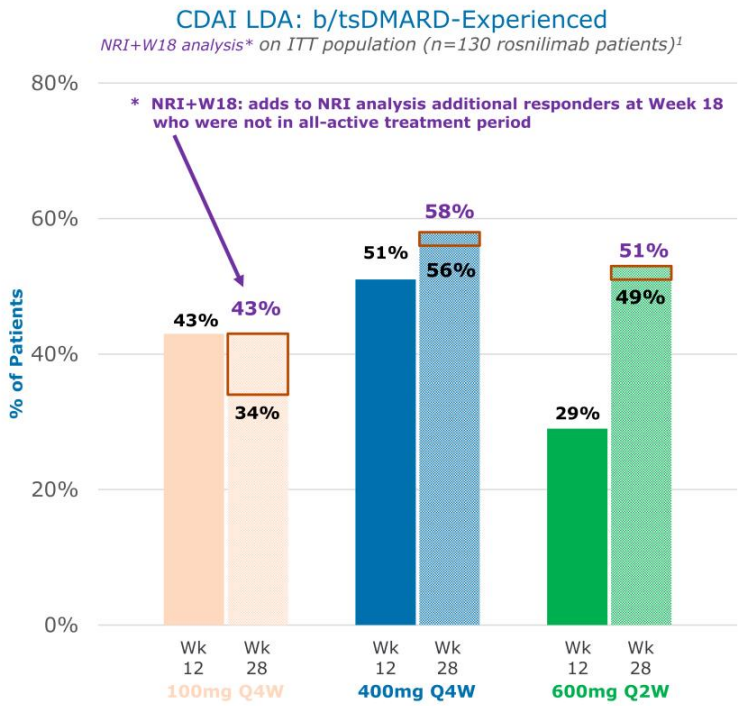



- All dose arms demonstrated statistically significant changes in DAS28-CRP

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.

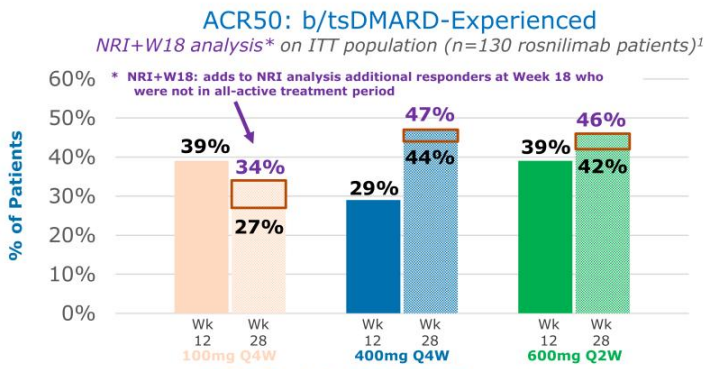
# Demonstrated JAK-like CDAI LDA rates by 6 months

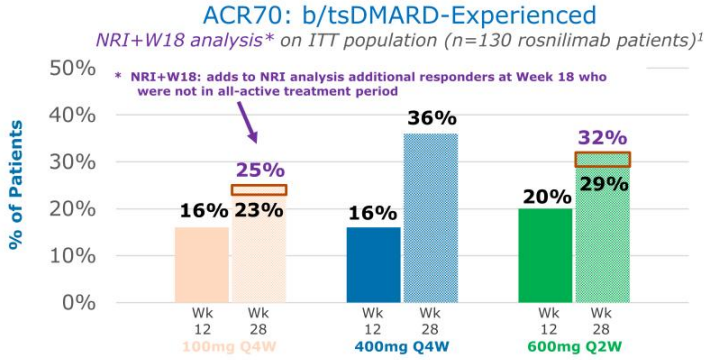



| CDAI LDA at Week 28 | | |
|---|---|---|
| Arm | NRI | NRI+W18 |
| b/tsDMARD-Experienced Population *(as graphed)* | | |
| 100mg | 34% | 43% |
| 400mg | 56% | 58% |
| 600mg | 49% | 51% |
| b/tsDMARD-Naïve Population | | |
| 100mg | 66% | 71% |
| 400mg | 53% | 55% |
| 600mg | 72% | 75% |

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)

## Anaptys Announces Phase 2 Trial of Rosnilimab Did Not Meet Primary or Secondary Endpoints at Week 12 in Moderate-to-Severe Ulcerative Colitis

- Rosnilimab was safe and well tolerated with similar adverse event rates vs. placebo
- Observed expected pharmacology, including ~90% depletion of pathogenic T cells, however lack of adequate efficacy at Week 12 does not support further development of rosnilimab in UC and trial will be discontinued, resulting in at least $10 million in savings
- Will provide update in H1 2026 on advancement of rosnilimab in RA, which would be funded by strategic or other sources of capital without diluting our royalties
- Reiterating commitment to protect and return the value of our substantial royalties to shareholders and intention to separate biopharma operations from royalty assets in 2026
- Biopharma Co will advance ANB033 through Phase 1b in celiac disease and an additional inflammatory indication, and ANB101 through Phase 1a in healthy volunteers
- Anticipate ending 2025 with ~$300 million, including anticipated accrual of one-time $75 million commercial sales milestone from GSK in Q4 2025 once *Jemperli* achieves $1 billion in global net sales

**SAN DIEGO, Nov. 10, 2025** — AnaptysBio, Inc. (Nasdaq: ANAB), a clinical-stage biotechnology company focused on delivering innovative immunology therapeutics, today announced that investigational rosnilimab was safe and well tolerated but did not meet the primary endpoint of mean change from baseline in modified Mayo Score (mMS) or key secondary endpoints of clinical response and clinical remission at Week 12 in the global Phase 2 trial for moderate-to-severe ulcerative colitis (UC). Placebo rates in the trial were within expected historical ranges. Given these results, this UC trial will be discontinued, resulting in at least $10 million in savings.

“Rosnilimab was safe and well tolerated, but we are disappointed in the lack of adequate efficacy and will discontinue the UC trial. However, we remain excited about the potential advancement of rosnilimab in RA and will provide an update in H1 2026 including funding by strategic or other sources of capital without diluting our royalties,” said Daniel Faga, president and chief executive officer of Anaptys. “ANB033, our CD122 antagonist, is in a Phase 1b for celiac disease and we plan to announce a Phase 1b in another inflammatory disease in 2026. Simultaneously, we are reiterating our intention to separate our biopharma assets from our substantial royalty assets in 2026, including *Jemperli* royalties of >$390 million per year at GSK’s peak sales guidance of >$2.7 billion[1], which Anaptys expects to be achieved before 2031.”

The Phase 2 randomized, double-blind, placebo-controlled, multicenter study evaluated the efficacy and safety of rosnilimab in UC patients. The study enrolled 136 moderate-to-severe patients across the U.S. Western and Eastern Europe with a baseline mean mMS score of 6.7 who had an inadequate response to, loss of response or intolerance to, at least one conventional or advanced UC therapy. Approximately 50% of enrolled patients had prior experience with advanced therapies and 62% had an endoscopic (MES) score of 3, indicating severe disease activity. Patients were randomized to receive either 400mg of subcutaneous (SC) rosnilimab every four weeks (Q4W), 800mg SC every two weeks (Q2W), or placebo.

Regardless of prior treatment, rosnilimab performed no better than placebo at Week 12, with clinical remission achieved by 7% of patients receiving rosnilimab 400mg Q4W or 800 mg Q2W, and 5% and 4% of patients achieving endoscopic remission, respectively. While preliminary data suggest an increase in remission rates between Week 12 and Week 24, Week 24 remission rates did not meet our six-month target product profile.

Consistent with the RA Phase 2b study, blood biomarker data in the UC study for both rosnilimab doses showed ~90% depletion of pathogenic T cells at Week 12 with preliminary data suggesting these effects were sustained through Week 24. Likewise, for patients continuing in the treatment extension period (TEP), all of whom were reassigned to a less frequent, 400mg Q8W dose regimen, these reductions were sustained through Week 50.

PD-1+ T cells were depleted in the colonic tissue at Week 12, consistent with the synovium in the RA Phase 2b study. Preliminary data show these reductions were observed equally with both induction doses, indicating the expected maximal pharmacological effect was obtained with the 400mg Q4W dose.

**Rosnilimab Well Tolerated with No Safety Signals**

Consistent with prior rosnilimab studies, a favorable safety and tolerability profile was observed, even at a 33% higher cumulative dose through 6 months in the 800mg Q2W cohort relative to the highest dose studied, 600mg Q2W, in the RA Phase 2b trial.

The data through Week 12 show –

- Most adverse events (AEs) were mild to moderate in severity
- No treatment-related serious adverse events (SAEs)
- No malignancies
- No MACE, other than one myocardial infarction in the 800mg Q2W dose cohort in a patient with hyperthyroidism, hypertension, glucose intolerance, and with evidence of preexisting cardiovascular disease by cardiac catheterization
- No serious, severe or opportunistic infections, other than a bilateral anterior uveitis by CMV in the 800mg Q2W dose cohort, diagnosed in a patient with pre-existing HIV
- No AEs of liver enzyme elevations
- Low incidence of injection site reactions and similar to placebo
- No incidence of anaphylaxis or systemic hypersensitivity

Through Week 12, adverse events occurring in $\geq$5% of participants were nasopharyngitis, ulcerative colitis, dizziness and injection site erythema, and were all mild to moderate in severity. Rosnilimab was highly tolerable, with one patient discontinuing rosnilimab due to an AE. Separately, in the placebo cohort, there was one reported death and one case of uterine leiomyoma.

The ongoing safety profile, including for patients through end-of-treatment at Week 50, remains consistent with the reported profile of all rosnilimab-treated patients through Week 12 as well as what was observed in the RA study. There have been no reported cases of malignancy or additional MACE.

“We are sincerely grateful to the patients and clinicians who participated in this trial. Despite rosnilimab not being the appropriate therapeutic option for UC, biologic insights from these data will help further inform future rosnilimab development,” said Paul Lizzul, M.D., Ph.D., chief medical officer of Anaptys.

**About Anaptys**

Anaptys is a clinical-stage biotechnology company focused on delivering innovative immunology therapeutics for autoimmune and inflammatory diseases. The company’s pipeline includes rosnilimab, a pathogenic T cell depleter, which has completed a Phase 2b trial for rheumatoid arthritis; ANB033, a CD122 antagonist, in a Phase 1b trial for celiac disease with plans to expand development into an additional indication; and ANB101, a BDCA2 modulator, in a Phase 1a trial. Anaptys has also discovered and out-licensed in financial collaborations multiple therapeutic

antibodies, including a PD-1 antagonist (*Jemperli* (dostarlimab-gxly)) to GSK and an IL-36R antagonist (imsidolimab) to Vanda Pharmaceuticals. To learn more, visit www.AnaptysBio.com or follow us on LinkedIn.

Anaptys recently announced the intent to separate its biopharma operations from its substantial royalty assets by year-end 2026, enabling investors to align their investment philosophies and portfolio allocation with the strategic opportunities and financial objectives of each company. Learn more at https://ir.anaptysbio.com/news-releases/news-release-details/anaptys-announces-intent-separate-biopharma-operations

**Forward-Looking Statements**

This press release contains forward-looking statements within the meaning of the "safe harbor" provisions of the Private Securities Litigation Reform Act of 1995, including, but not limited to: the ability to find funding for continued development of rosnilimab; expectations regarding the structure, infrastructure, timing and taxation of the proposed separation of companies; year-end cash estimates; and the potential to receive any additional milestones and royalties from the GSK collaboration, and the timing therefor. Statements including words such as “plan,” “continue,” “expect,” or “ongoing” and statements in the future tense are forward-looking statements. These forward-looking statements involve risks and uncertainties, as well as assumptions, which, if they do not fully materialize or prove incorrect, could cause its results to differ materially from those expressed or implied by such forward-looking statements. Forward-looking statements are subject to risks and uncertainties that may cause the company’s actual activities or results to differ significantly from those expressed in any forward-looking statement, including risks and uncertainties related to the company’s ability to advance its product candidates, obtain regulatory approval of and ultimately commercialize its product candidates, the timing and results of preclinical and clinical trials, the company’s ability to fund development activities and achieve development goals, the company’s ability to protect intellectual property and other risks and uncertainties described under the heading “Risk Factors” in documents the company files from time to time with the Securities and Exchange Commission. These forward-looking statements speak only as of the date of this press release, and the company undertakes no obligation to revise or update any forward-looking statements to reflect events or circumstances after the date hereof.

**Contact:**
Nick Montemarano
Executive Director, Investor Relations
858.732.0178
investors@anaptysbio.com

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1. 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 far more than £2 billion*.”; Converted from GBP to USD using Q3 2025 average exchange rate (1.35x)