Corporate Overview

April 2026

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 and initial data from ANB033's Phase 1b clinical trial in eosinophilic esophagitis; expectations regarding the structure, infrastructure, timing and taxation of the proposed separation of companies; timing of paydown of financial obligations to Sagard; 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 projected cash runway for First Tracks Biotherapeutics. 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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Separation into two independent, publicly traded companies to unlock and maximize value to occur on April 20th

First Tracks Biotherapeutics
(Biopharma Co)

ANB033
(CD122 antagonist)

- P1b in Celiac Disease
- P1b in Eosinophilic Esophagitis

Rosnilimab
(Pathogenic T cell depleter)

- P2b completed in Rheumatoid Arthritis

ANB101
(BDCA2 modulator)

- P1 in Healthy Volunteers

Research-driven

- R&D capabilities focused on immunology targets

AnaptysBio
(Royalty Management Co)

- Protect and return value of the royalties to shareholders
- Hold and continue to manage rights to
  - Potential substantial *Jemperli* royalties from GSK
  - Potential imsidolimab royalties from Vanda
- Will retain Anaptys' net operating loss (NOL) carryforwards
- Minimal infrastructure and staff

GSK Jemperli (dostarlimab-gxly) Injection 500 mg

VANDA PHARMACEUTICALS INC. *imsidolimab*

Note: YE 2025 cash: ~$311MM, which includes the receipt in Dec. 2025 of a one-time $75MM commercial sales milestone from GSK when *Jemperli* achieved $1 billion in worldwide net sales in Nov. 2025. First Tracks Biotherapeutics to launch with $180MM of cash ($100MM from balance sheet plus $80MM gross proceeds from private offering); AnaptysBio to relaunch with ~$140MM to $145MM of net cash and investments and with a $100MM stock repurchase plan

First Tracks Biotherapeutics would retain a leading pipeline to deliver breakthroughs for patients with autoimmune diseases

	Antibody Program	Therapeutic Indication	Development Stage and Anticipated Milestones: IND Enabling / Phase 1 / Phase 2 / Phase 3
Immune Cell Modulators	**Rosnilimab** (Pathogenic T cell depleter)	**Rheumatoid Arthritis**	Late-breaking data presented at ACR 2025 Update in Q2 2026 on P3 advancement
	ANB033 (CD122 antagonist)	**Celiac Disease**	Top-line P1b data anticipated Q4 2026
		Eosinophilic Esophagitis	Top-line P1b data anticipated mid-2027
	ANB101 (BDCA2 modulator)	**Inflammatory Disease**	P1 in healthy volunteers ongoing

Significant upcoming catalysts for both *Jemperli* and imsidolimab expected within the next two years for AnaptysBio

	Antibody Program	Indication	Development Stage and Anticipated Milestones: IND Enabling / Phase 1 / Phase 2 / Phase 3 / Registrational / Commercial
AnaptysBio	***Jemperli***[1] (PD-1 antagonist) GSK	1L Endometrial Cancer	Approved in US and ex-US[2]
		1L MMR Deficient Endometrial Cancer *(chemo-free regimen)*	DOMENICA Est. primary comp. Q2 2026[4]
		2L dMMR/MSI-H Endometrial Cancer	Approved in US and ex-US
		dMMR/MSI-H Pan Tumors	Approved in US
		dMMR/MSI-H Locally-Advanced Rectal Cancer	*Commissioner's National Priority Voucher (CNPV) granted*; AZUR-1 Top-line data H2 2026[5]
		dMMR/MSI-H Perioperative Colon Cancer	AZUR-2 Top-line data 2028[5]
		Neoadjuvant MMRp/MSS Colon Cancer	AZUR-4 Est. primary comp. Q4 2026[4]
		Locally-Advanced HNSCC[3]	JADE Top-line data 2028[5]
	Imsidolimab (IL-36R antagonist) VANDA PHARMACEUTICALS INC	Generalized Pustular Psoriasis	FDA PDUFA Dec. 12, 2026

1. Not-exhaustive, does not include ADC combination opportunities (P2 combination data to be shared in H1 2026); 2. Registrational studies also ongoing in China and Japan; 3. HSNCC - Head and neck squamous cell carcinomas; 4. Per clinicaltrials.gov estimated primary completion date; 5. GSK Q4 2025 earnings

AnaptysBio
(Royalty Management Co)

Jemperli™
(dostarlimab, PD-1 antagonist)

Imsidolimab
(IL-36R antagonist)

Royalty Management Co would protect and return value of *Jemperli* and imsidolimab royalties to shareholders

Jemperli: GSK Financial Collaboration

- 2025 sales of >$1.1 billion
  - Exited 2025 with ~$1.4 billion annualized run rate[1]
  - Q4 2025 sales: $343 million (>13% QoQ growth rate)
- Significant royalties on global net sales
  - 8% ($0 to $1b), 12% ($1 - $1.5b), 20% ($1.5 - $2.5b), and 25% (>$2.5b)
- >$390 million per year in *Jemperli* royalties at GSK's peak sales guidance of >$2.7 billion[2]
  - Anaptys expects to be achieved as early as 2029
- Anticipate Sagard paydown as early as Q2 2027
- Substantial ongoing investment in additional indications for *Jemperli* monotherapy and combos
  - H2 2026: top-line data from registrational dMMR rectal trial (national priority voucher)

Imsidolimab: Vanda Financial Collaboration

- 10% royalty on global net sales
- $35 million in future milestones[3]
  - $5 million – FDA approval in GPP
  - $5 million – EMA approval in GPP
  - $25 million – $100 million annual sales milestone
- FDA BLA submitted for GPP in December 2025
  - FDA accepted the BLA filing in February 2025 with a target action date of December 12, 2026

1. GSK Q4 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."*; 3. Future regulatory and commercial milestones to be retained by First Tracks Biotherapeutics

Potential royalties 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

Sagard *Jemperli* capped non-recourse monetization

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

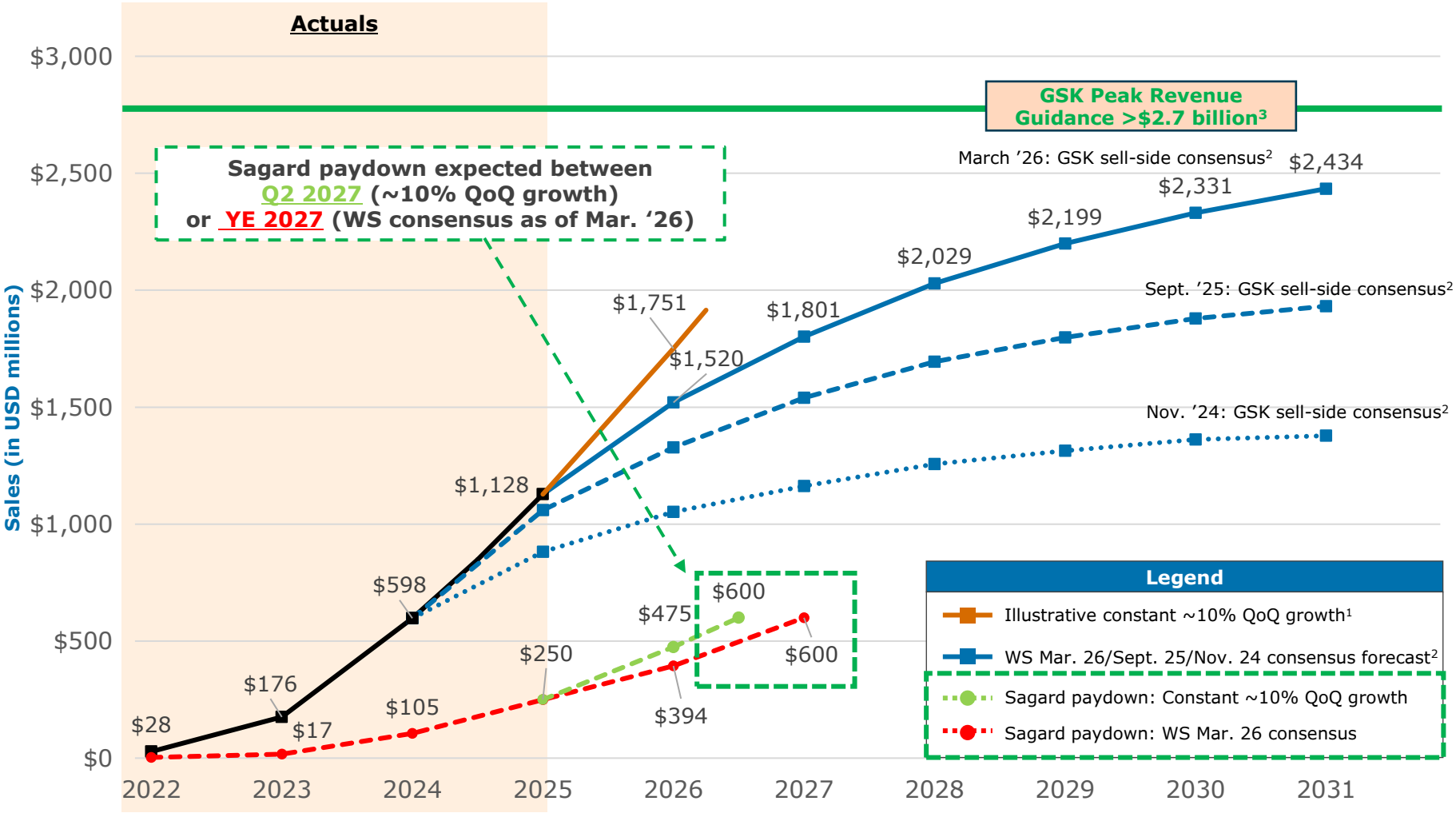
1. The following *Jemperli* milestones are also still potentially payable from GSK but contribute to Sagard paydown: $15MM on regulatory approvals
2. If cumulative $600MM not paid to Sagard by Mar. 31, 2031, the cumulative paydown increases to $675MM.
3. Forecast assumes constant ~10% quarter-over-quarter growth rate for *Jemperli* from Q4'25 through Q2'27 and milestone payments associated with filing ($5mm) and approval ($10mm) of dMMR rectal approval in the EU

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

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

Jemperli Revenue Forecasts



1. Actual *Jemperli* Q3 to Q4'25 QoQ growth was 13%, Forecast assumes illustrative constant ~10% QoQ sales growth from Q4'25 through Q2'27 and dMMR rectal approval; 2. GSK analyst consensus as of 3/9/2026 (solid blue), 9/15/2025 (dark dashed blue), and 11/26/2024 (light dashed blue) converted from GBP to USD using Q4 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

- **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
- **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

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

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 Q4 2025 earnings epidemiology report

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

Key financial terms to Anaptys

VANDA PHARMACEUTICALS INC.

Exclusive global license to Vanda
announced February 2025

$35 million future milestones[1]
$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 submitted and accepted for generalized pustular psoriasis (GPP)

Target action date of December 12, 2026[2]

Imsidolimab: two positive global Phase 3 studies in GPP

Note: $15 million payment at deal execution of $10 million upfront and $5 million for existing drug supply
1. Future regulatory and commercial milestones to be retained by First Tracks Biotherapeutics; 2. Vanda press release; 2/25/2026

First Tracks Biotherapeutics
(Biopharma Assets)

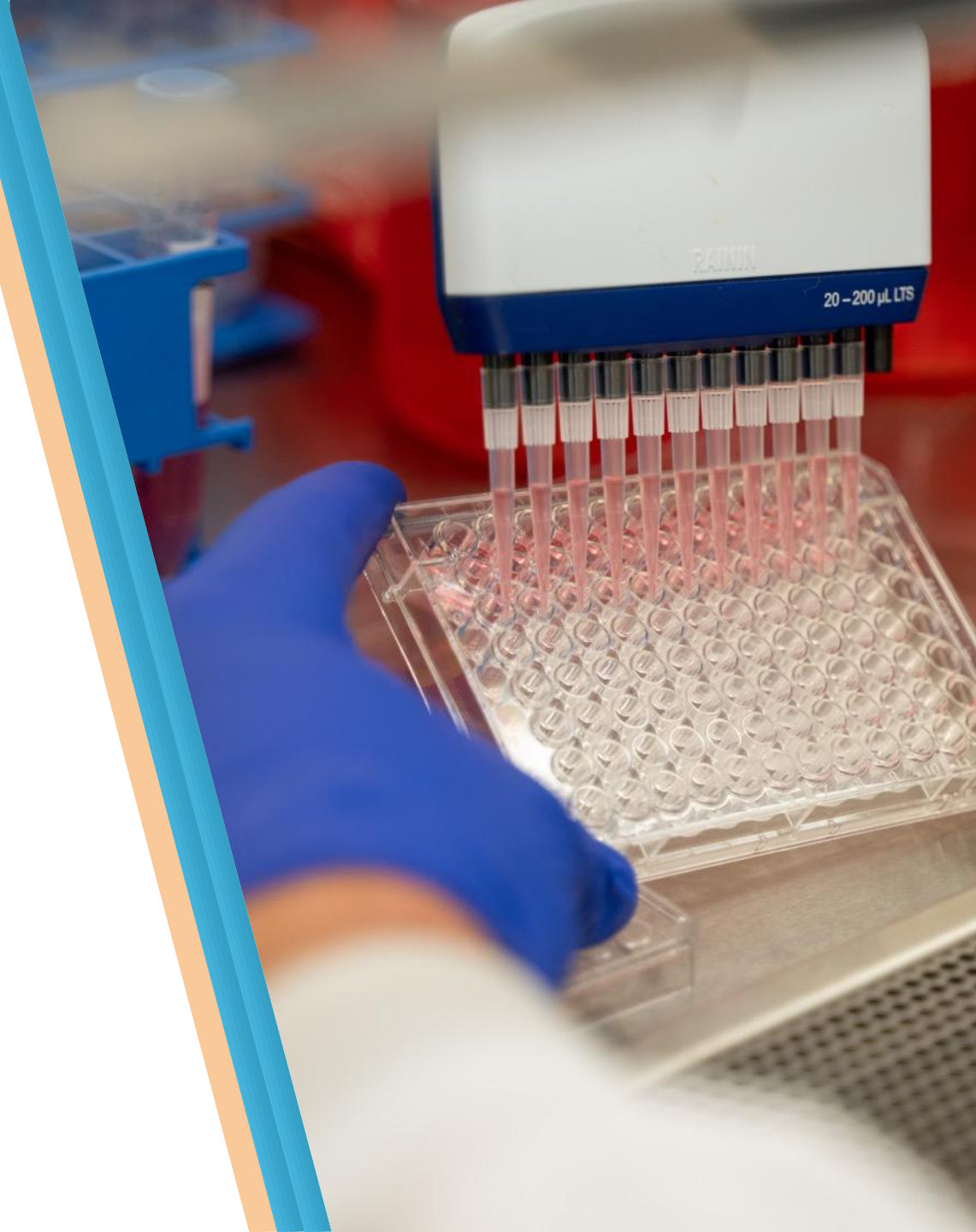
ANB033
(CD122 antagonist)

Rosnilimab
(Pathogenic T cell depleter)

ANB101
(BDCA2 modulator)

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

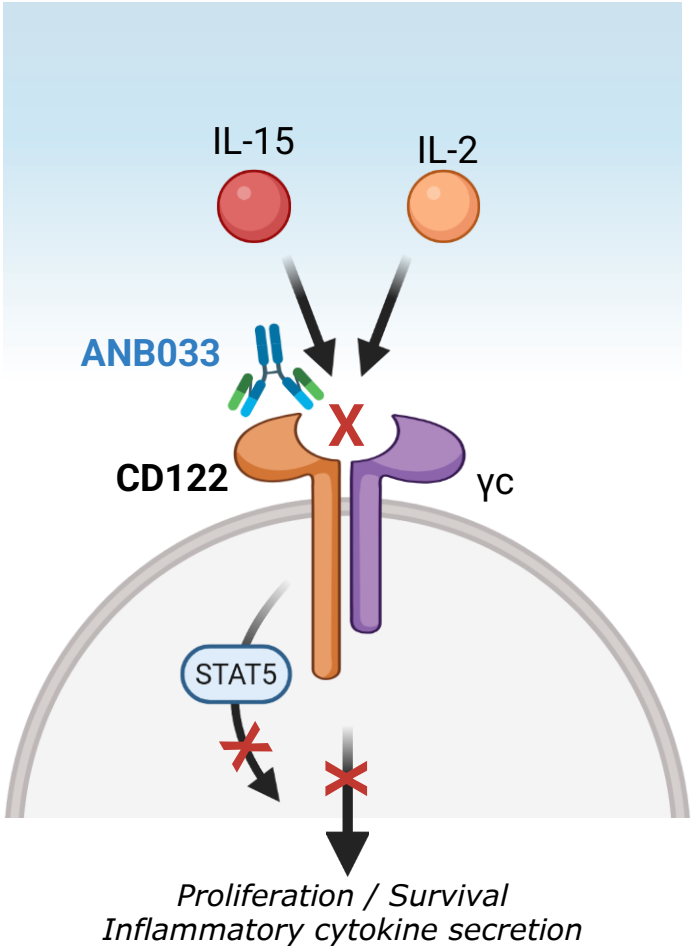
- Expressed on subsets of CD8+ and CD4+ T cells and NK cells

CD122 antagonism reduces these immune cell subsets

- Dependent on IL-15 and/or IL-2 for 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

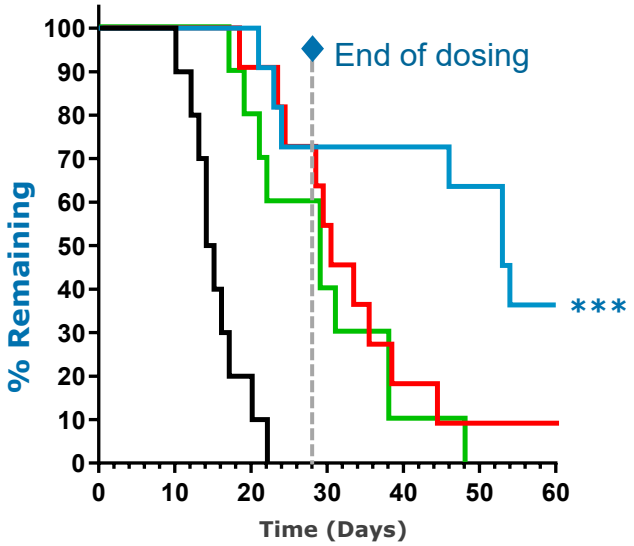
Gastroenterology	***Dermatology***	**Other Areas**
Celiac Disease (CeD)	Atopic Dermatitis	Asthma/COPD
Eosinophilic Esophagitis (EoE)	Alopecia Areata	Multiple Sclerosis
Crohn's Disease	Hidradenitis Suppurativa	Psoriatic Arthritis
Ulcerative Colitis	Lichen Planus	Type 1 Diabetes
	Vitiligo	Solid Organ Transplant

Other clinical-stage drugs targeting IL-15 or CD122

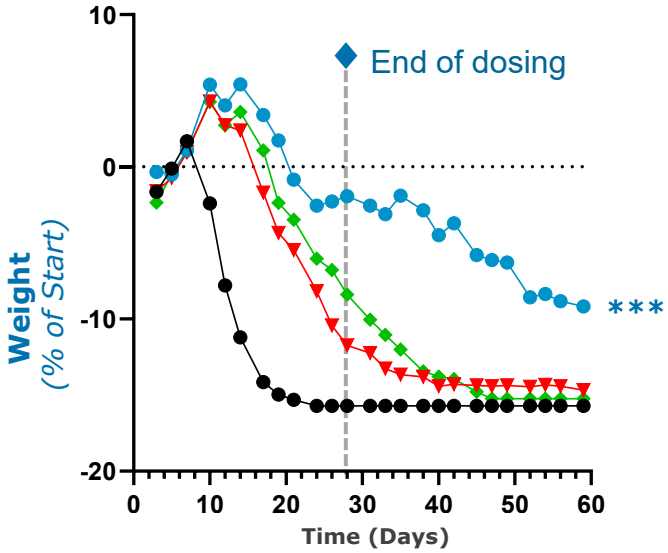
NOVARTIS	IL-15	• P1b PoC in CeD and EoE • P2a in atopic dermatitis (ongoing) • P2 in vitiligo (initiating) • *Initiating a trial in at least one other indication*
teva	IL-15	• P2a in CeD (ongoing – data Q3 2026) • P1b in vitiligo (ongoing – data Q2 2026) • *Assessing atopic dermatitis, alopecia areata, and EoE*
FORTE	CD122	• Positive P1b in CeD (P2a ongoing – data in 2026) • P1b in vitiligo (ongoing – data in H1 2026) • P1b alopecia areata (ongoing – data in 2026) • Assessing T1D

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

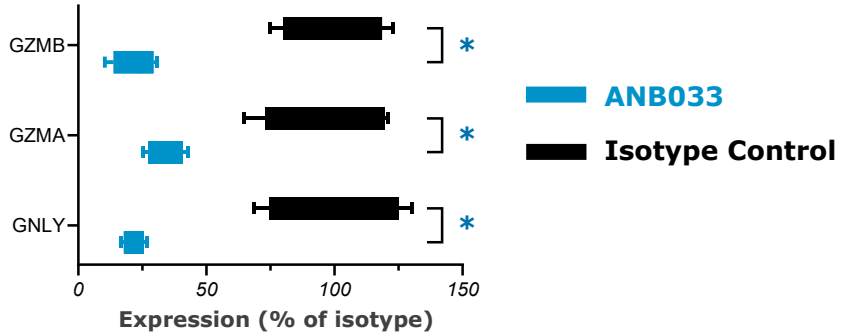
Survival



Body weight loss (carried)



Cytolytic gene expression (Day 17)



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

✓ 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

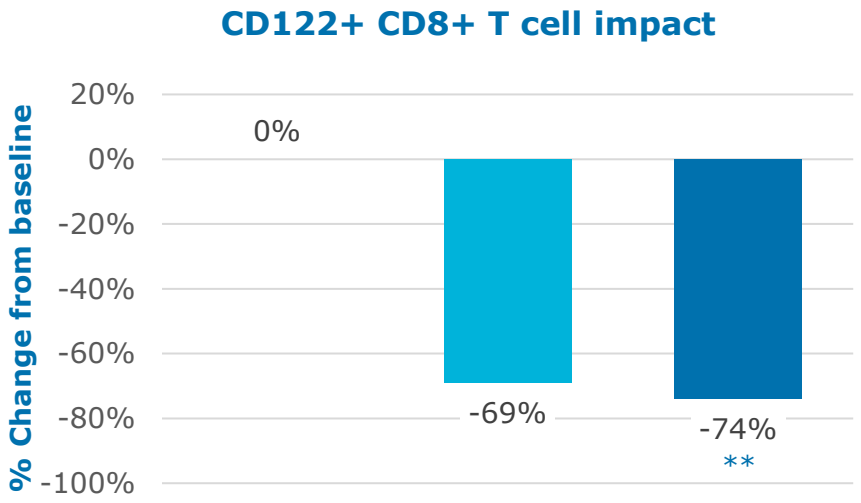
Rapid and sustained PK profile

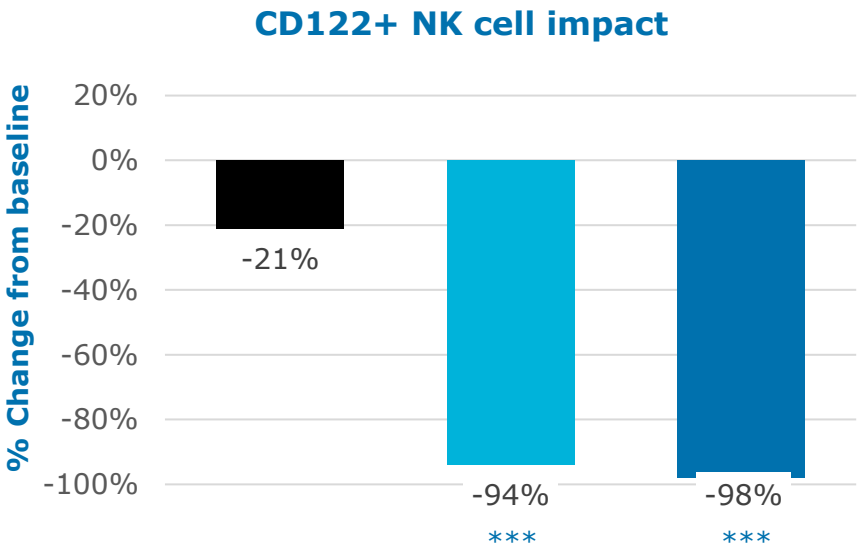
- Favorable 2 to 3-week half-life with IV and SQ dosing
- Full receptor occupancy (RO) within hours and maintained for >30 days
- Dose response observed
- Modeled to achieve >IC90 on CD8+ T cell subsets in GI tissue
- Overall, no impact on peripheral total Treg counts

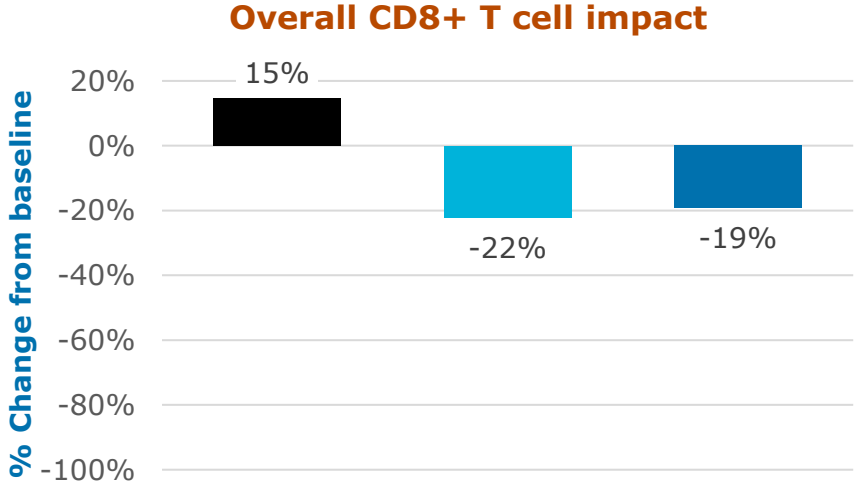
SAE = Serious adverse event

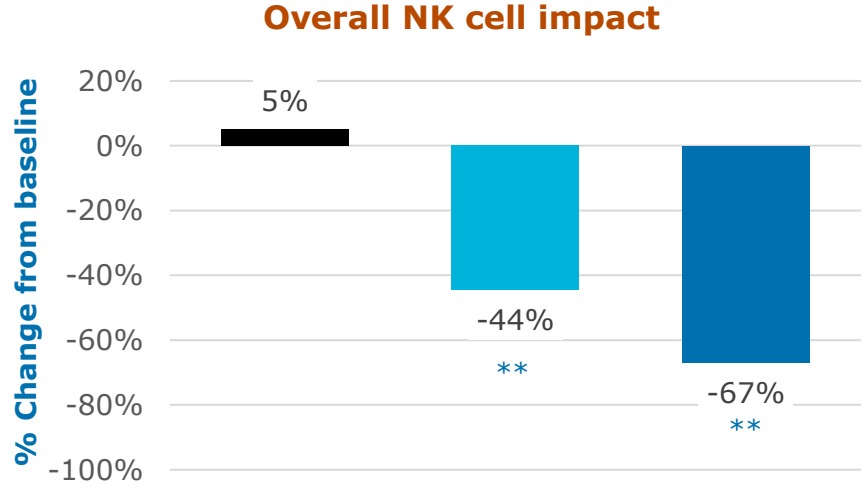
ANB033 significantly reduces CeD relevant CD8+ T cells and NK cells after single dose

Effect of ANB033 is limited to CD122 expressing cells





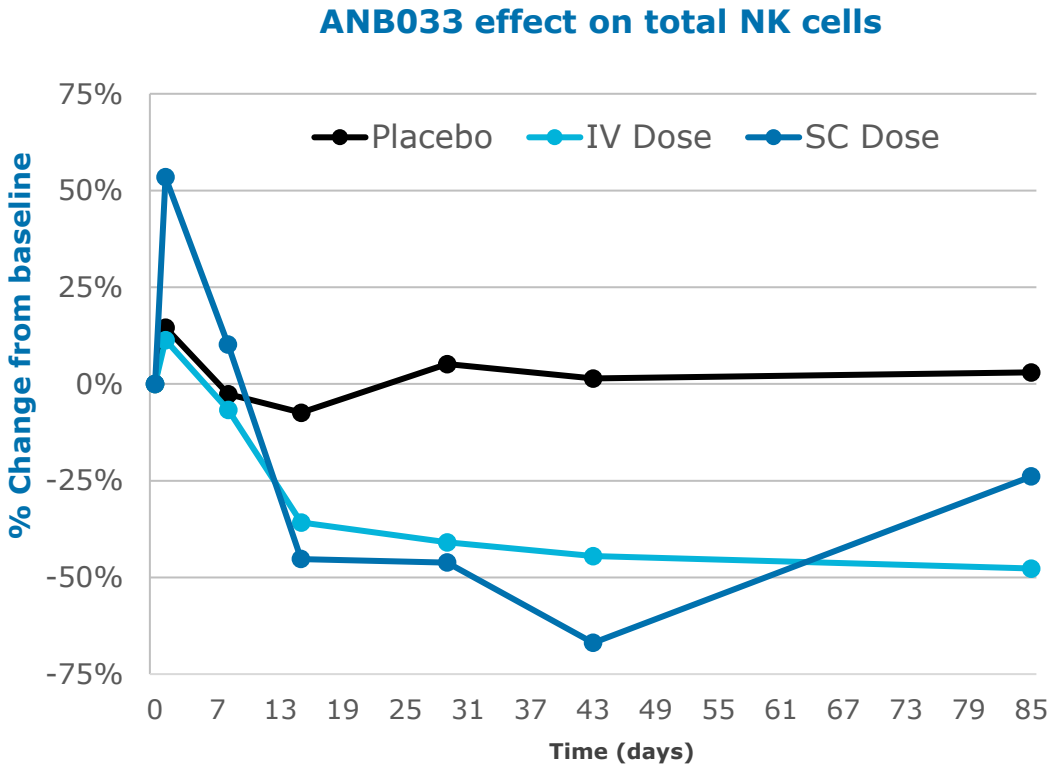


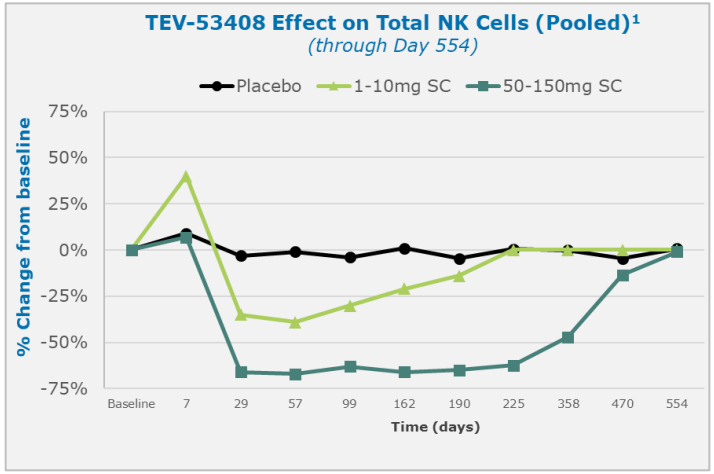


Placebo ANB033 – IV Dose ANB033 – SC Dose

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





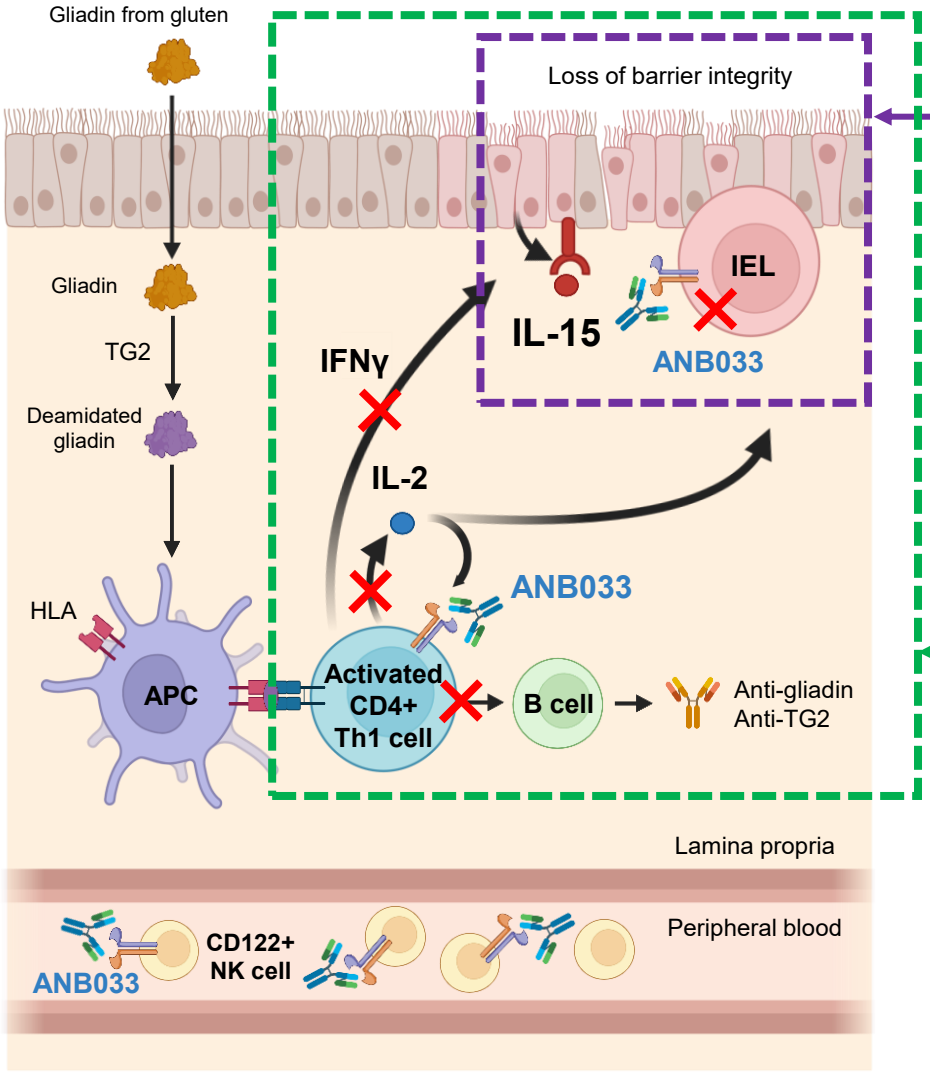
No safety signals observed in any CD122 or IL-15 trials to date after NK cell reduction

- ANB033 >50% <u>peak</u> total NK cell reduction with return towards baseline within 3 months
- TEV-53408: >50% <u>sustained</u> 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 anticipated in Sept. 2026.

ANB033's MOA is an 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

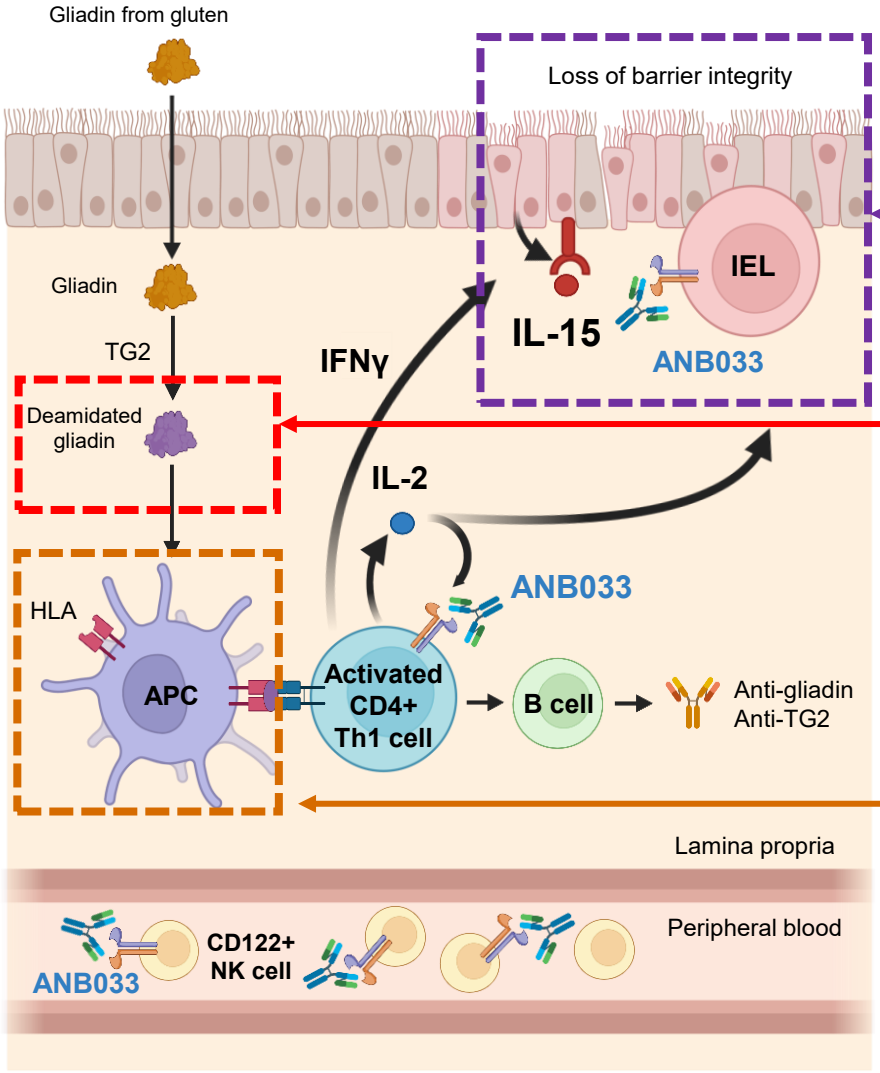
Inhibition of IL-2 signaling

- 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 the 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

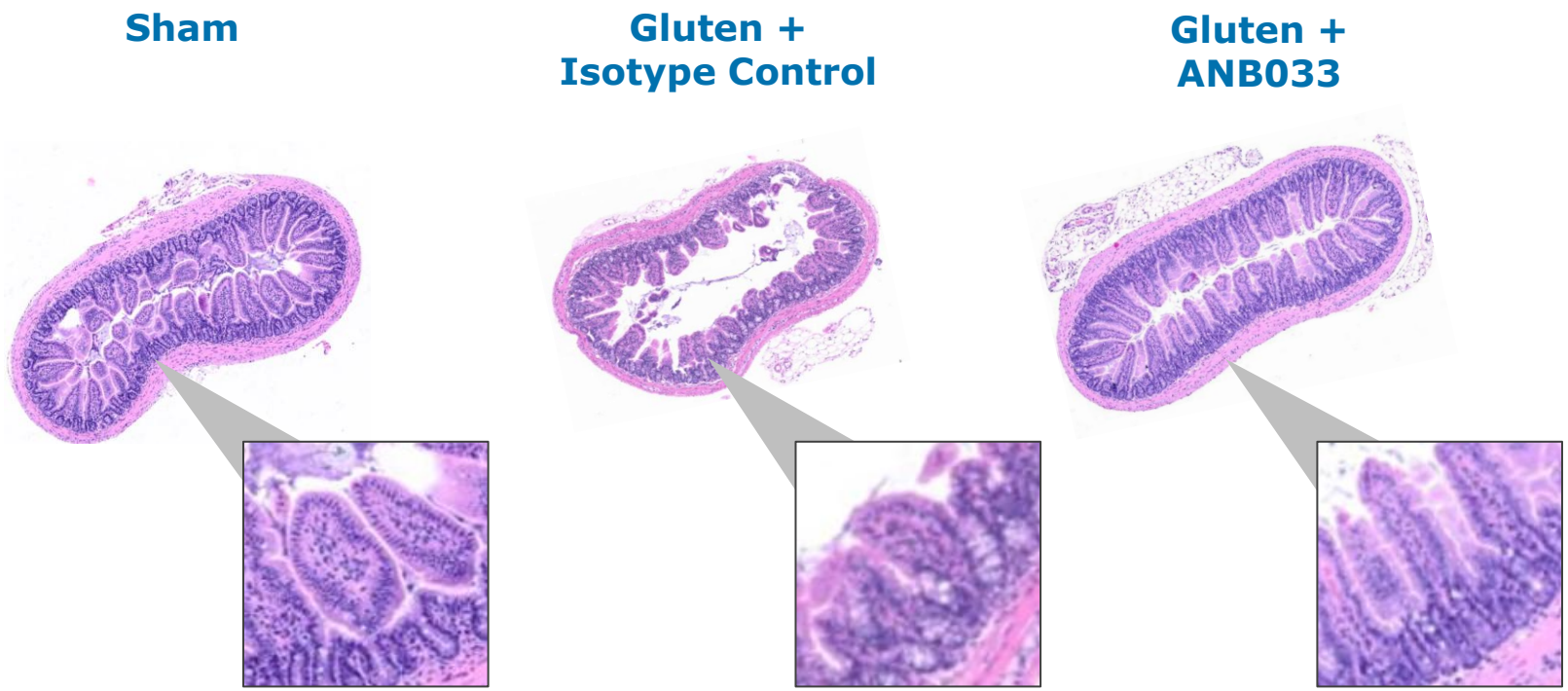
- Takeda — *P2 ongoing*, *Gluten tolerance*
- astellas — *Discontinued*, *Gluten tolerance*
- Roche — *P1 ongoing*, *HLA-DQ2.5 gluten peptide complex*
- Immunic THERAPEUTICS — *P1 ongoing*, *SIRT6 modulator*

OX-40L antagonist

- sanofi — *P2 deprioritized*

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

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



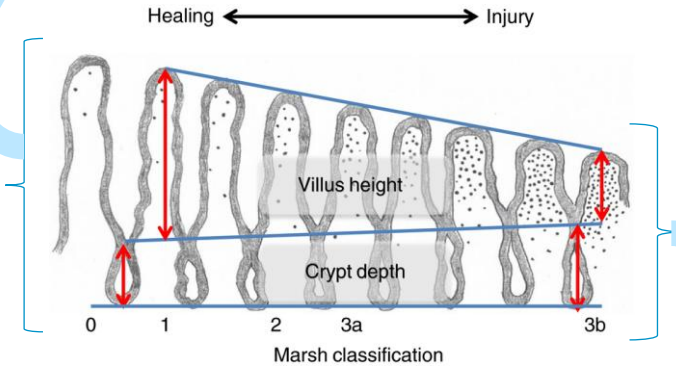
ANB033 treatment shows improved histology:
preserves villus height and crypt depth (Vh:Cd) in CeD mouse model

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 the reduction of Vh:Cd ratio compared to control

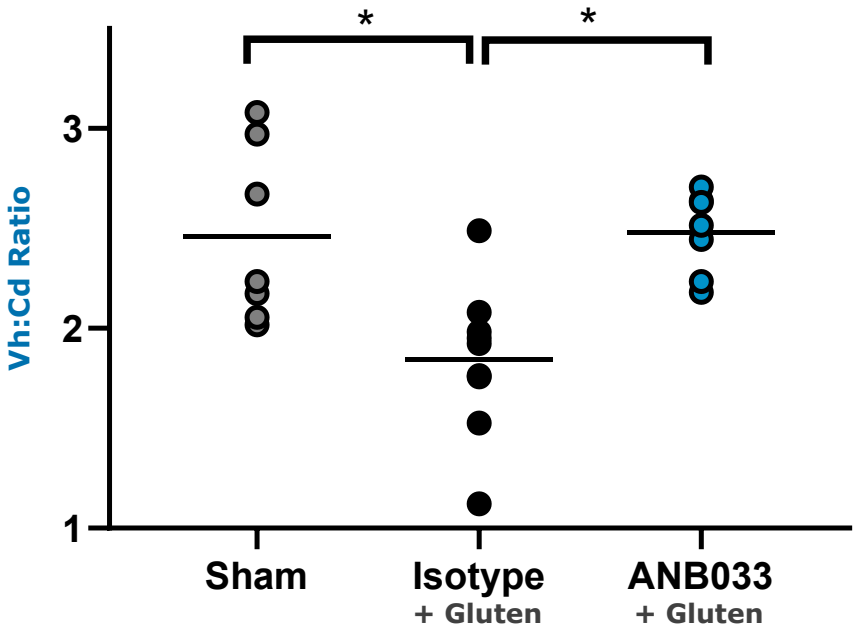
Vh:Cd ratio

Healthy tissue:
High villus height, lower crypt depth



CeD tissue:
Reduced villus height, extended crypt depth

ANB033 impact on Vh:Cd ratio



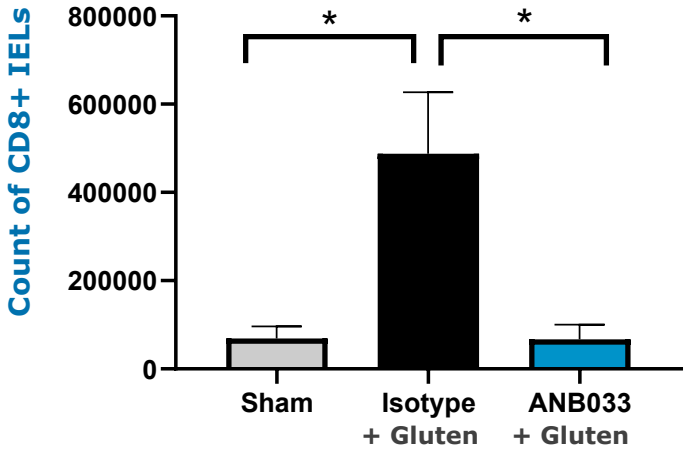
ANB033 treatment shows improved histology: preserves villus height and crypt depth (Vh:Cd) in CeD mouse model

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. * $p<0.05$.

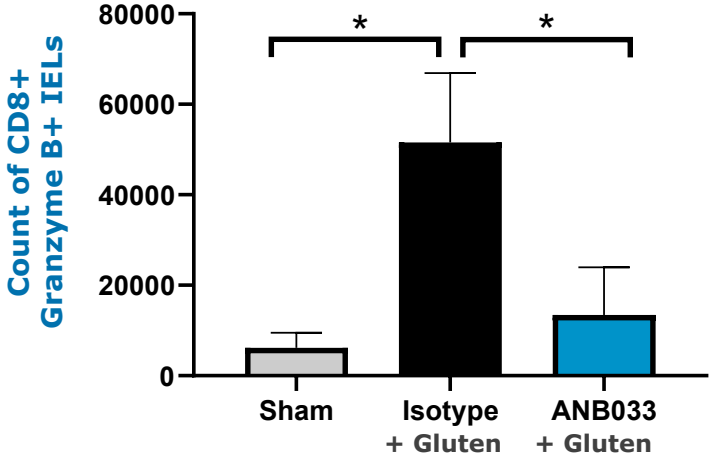
ANB033 prevents gluten-induced intestinal inflammation

Epithelial layer of small intestine

No increase in CD8+ IELs

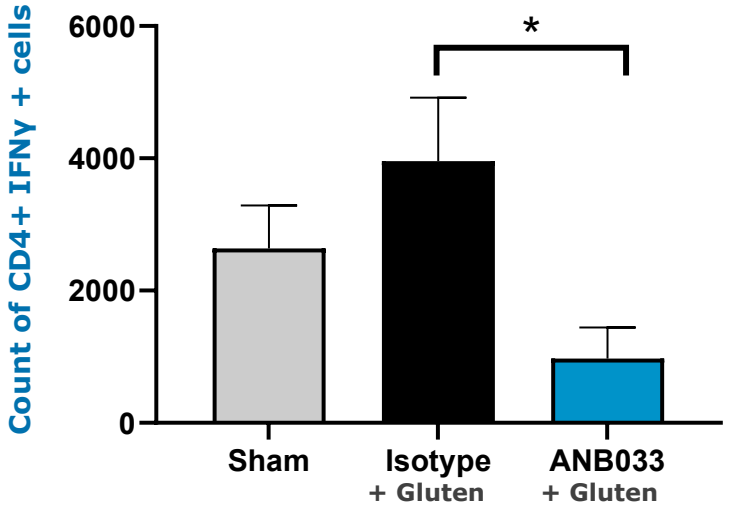


No increase in Granzyme B+ IELs



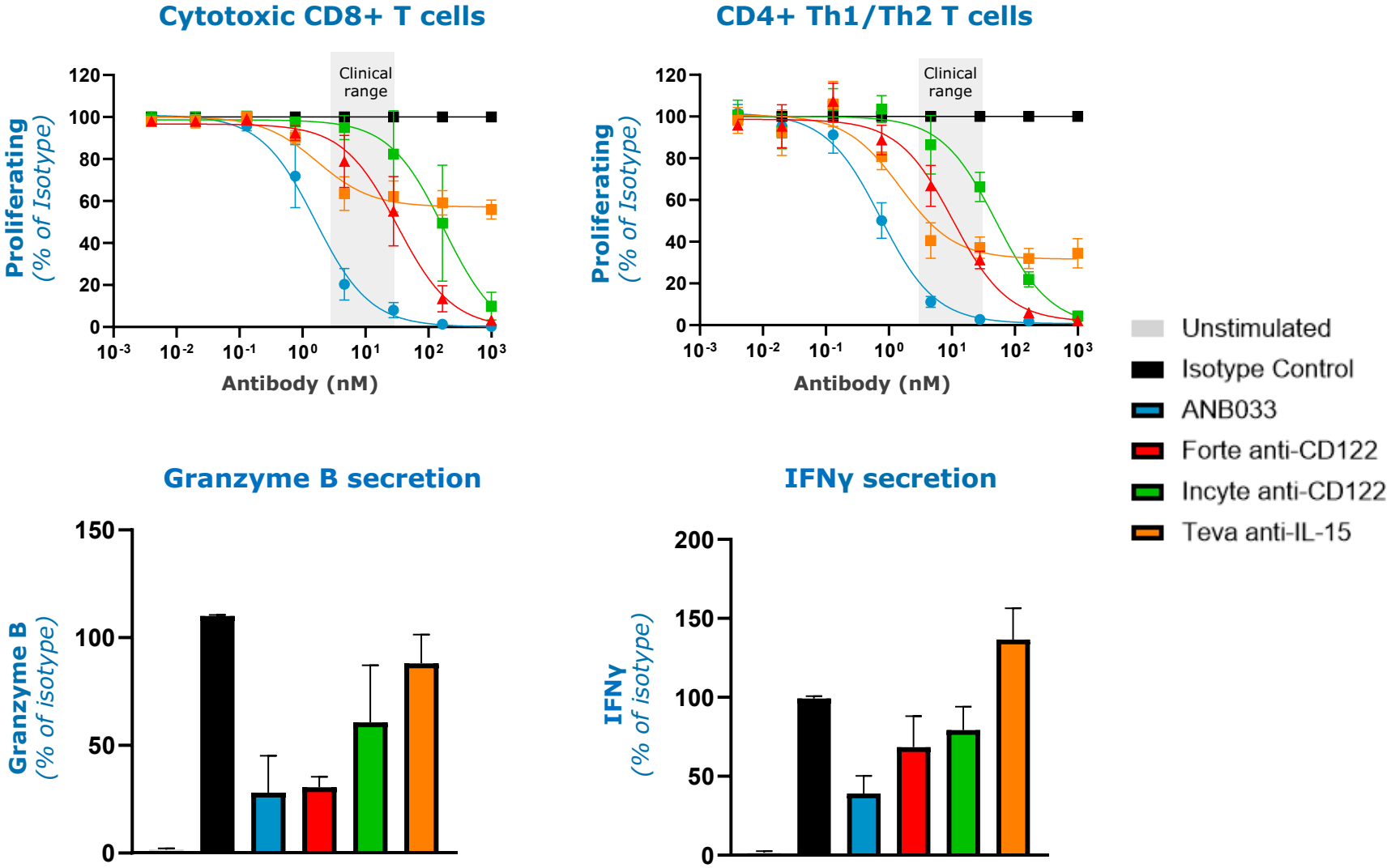
Lamina propria (LP)

No increase in CD4+ IFNγ + T cells



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), 100nM dose for all arms

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 biotech *(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

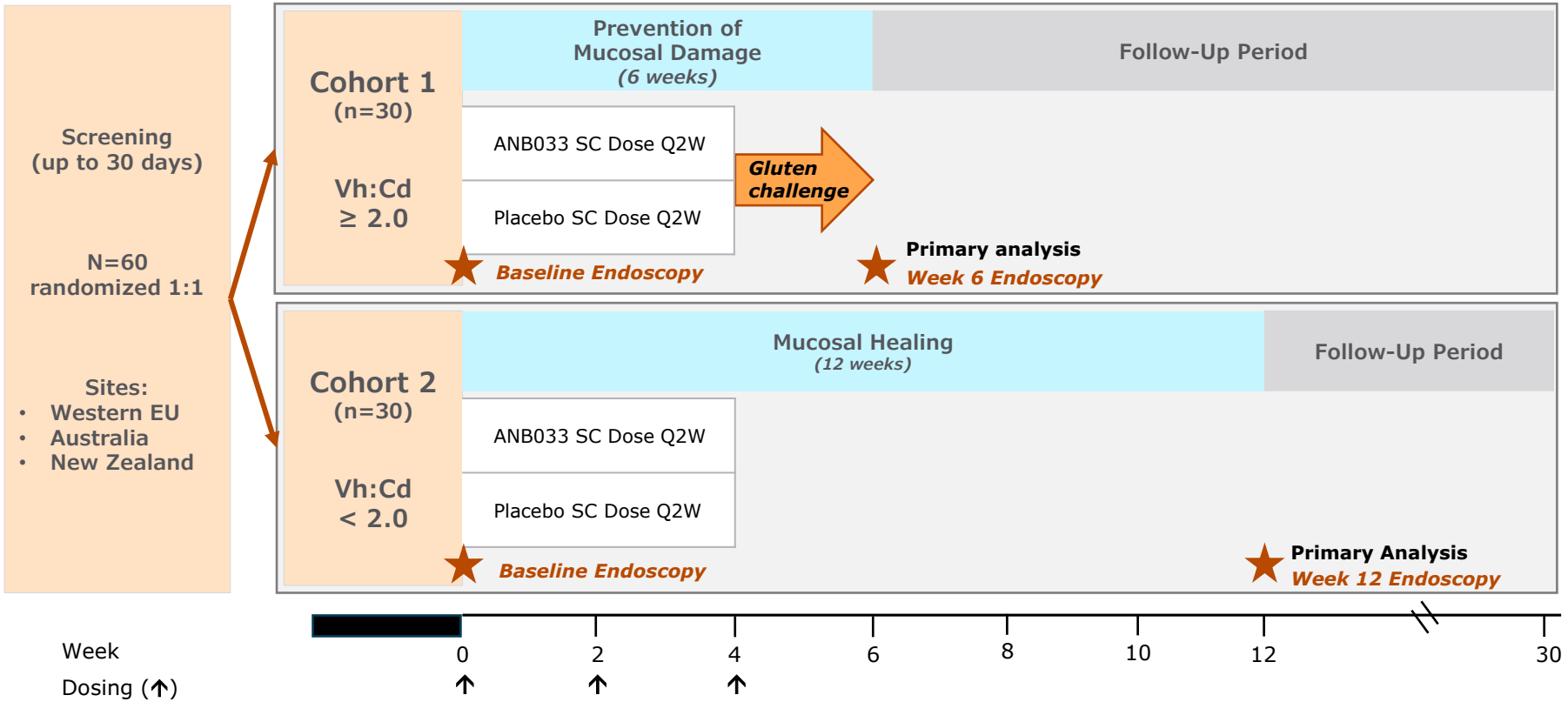
sanofi *(Phase 2b, deprioritized)*

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

GF diet = Gluten free diet.

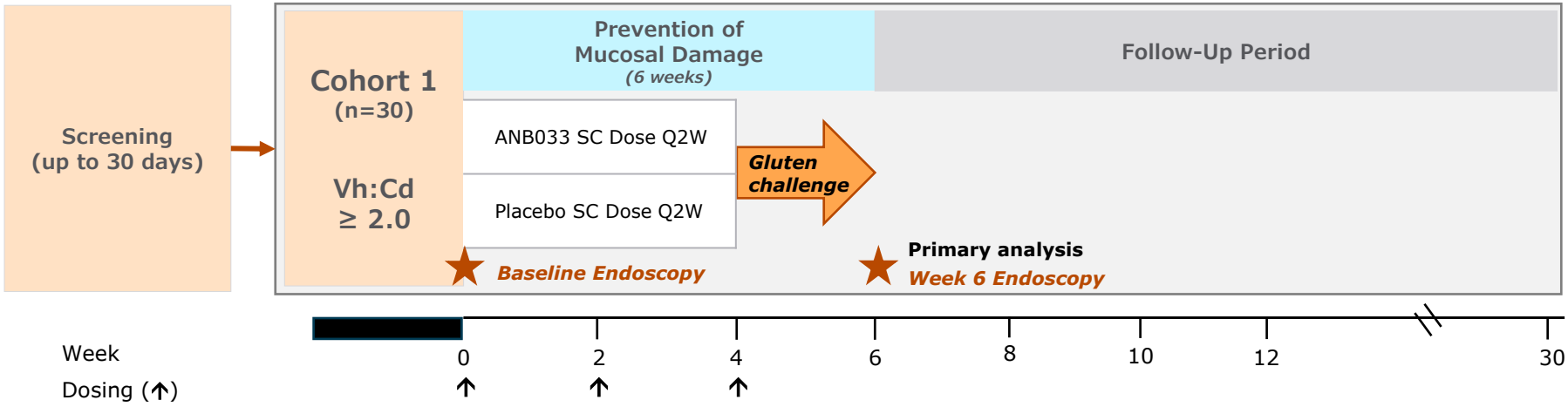
ANB033 Phase 1b trial in CeD initiated

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 • 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



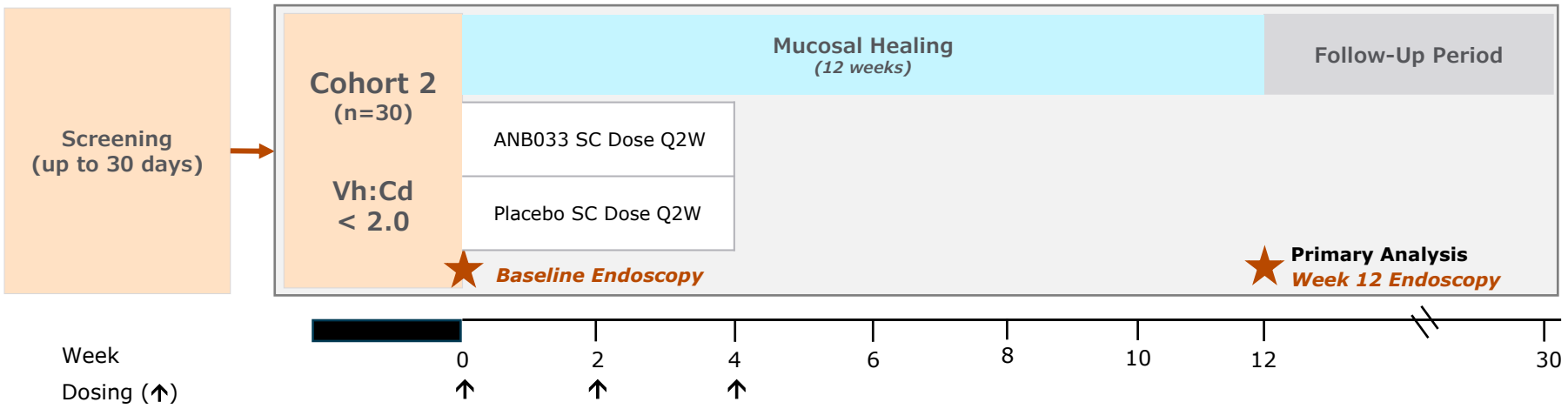
Minimal evidence of mucosal damage (Vh:Cd ≥ 2.0)

- 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



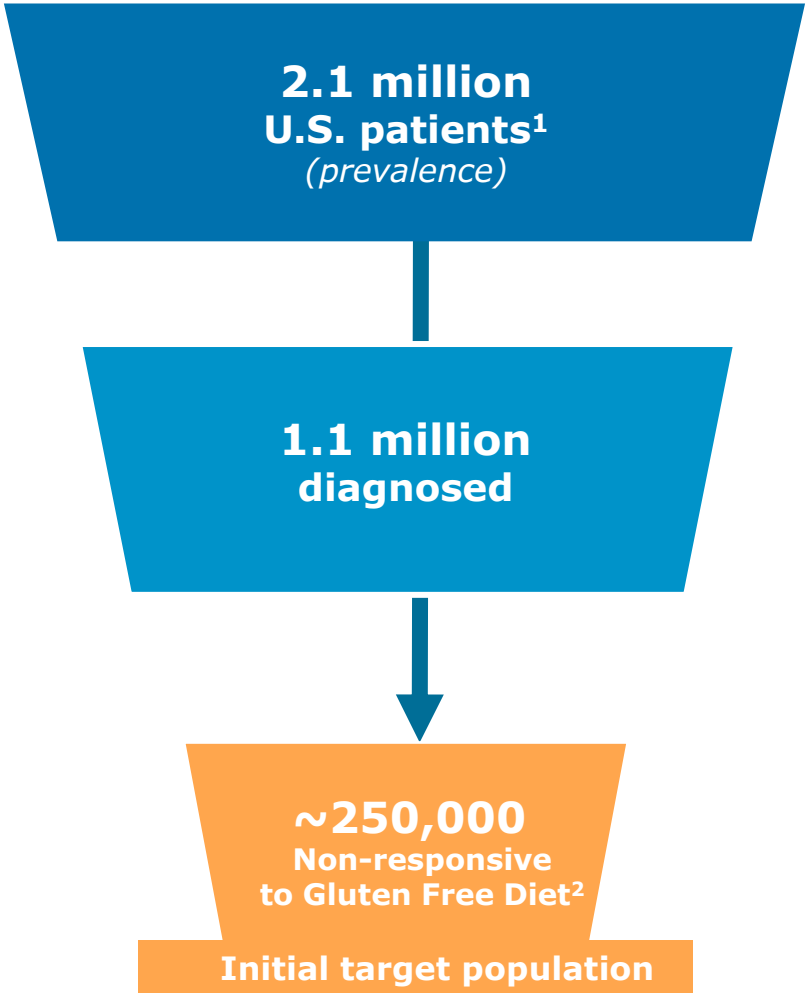
Persistent evidence of histologic CeD activity (Vh:Cd < 2.0)

- 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

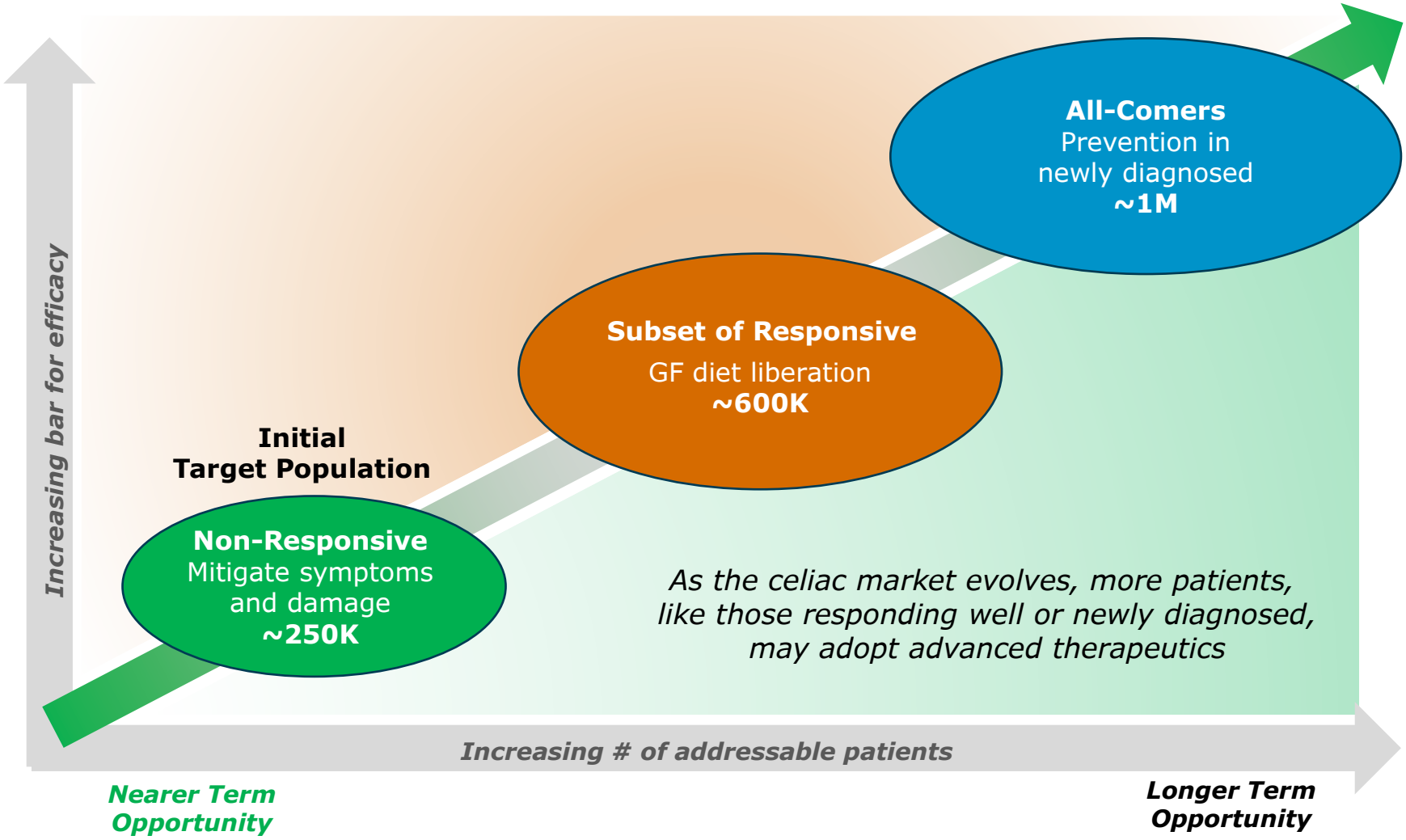
- HCPs favor MOA that targets both symptoms and histology

$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;
2. 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.

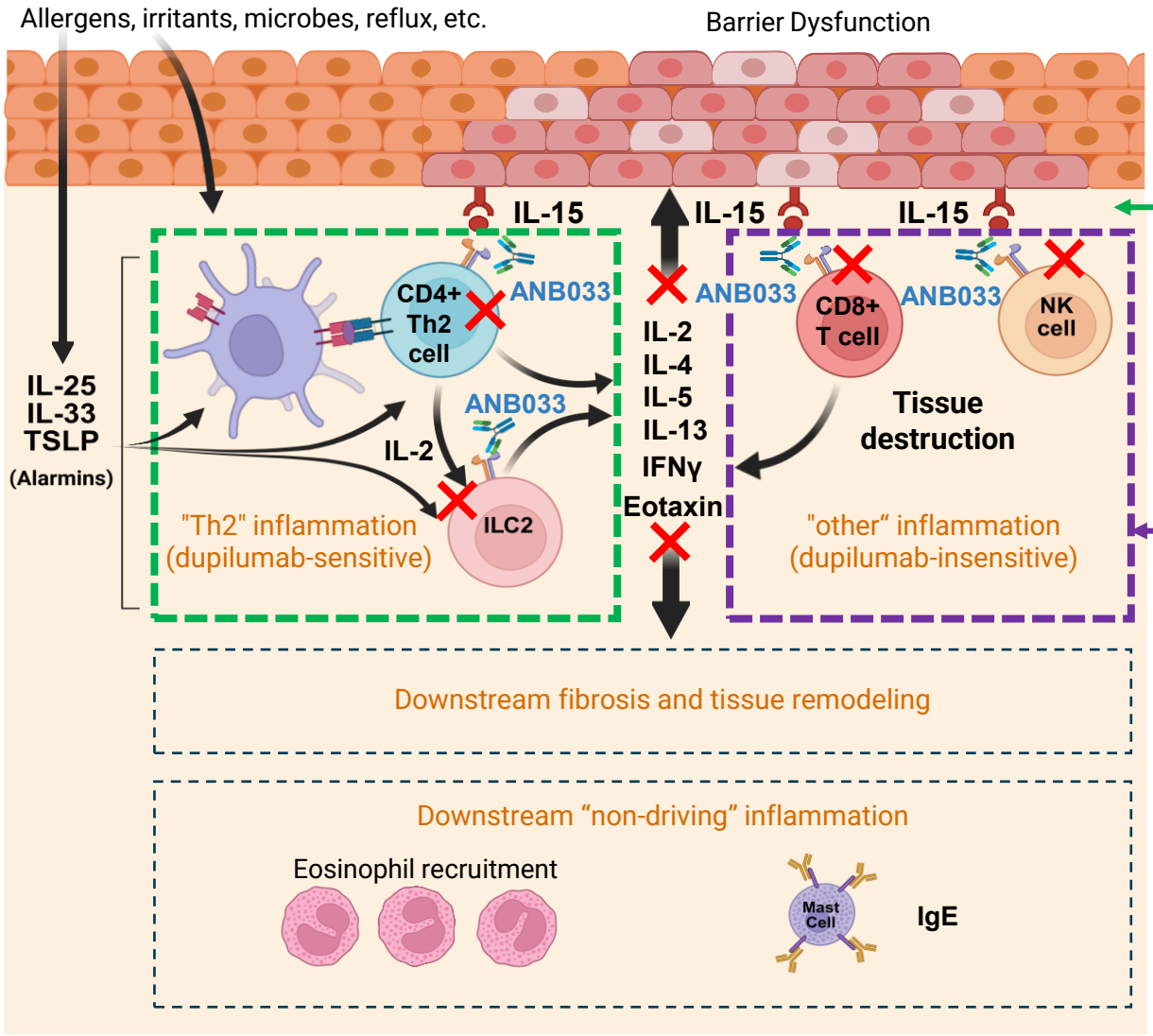
New therapies in CeD could grow market in responsive and newly diagnosed patients



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

Phase 1b trial initiated; Anticipate top-line data in mid-2027



CD4+ and ILC2 (dupilumab-sensitive)

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

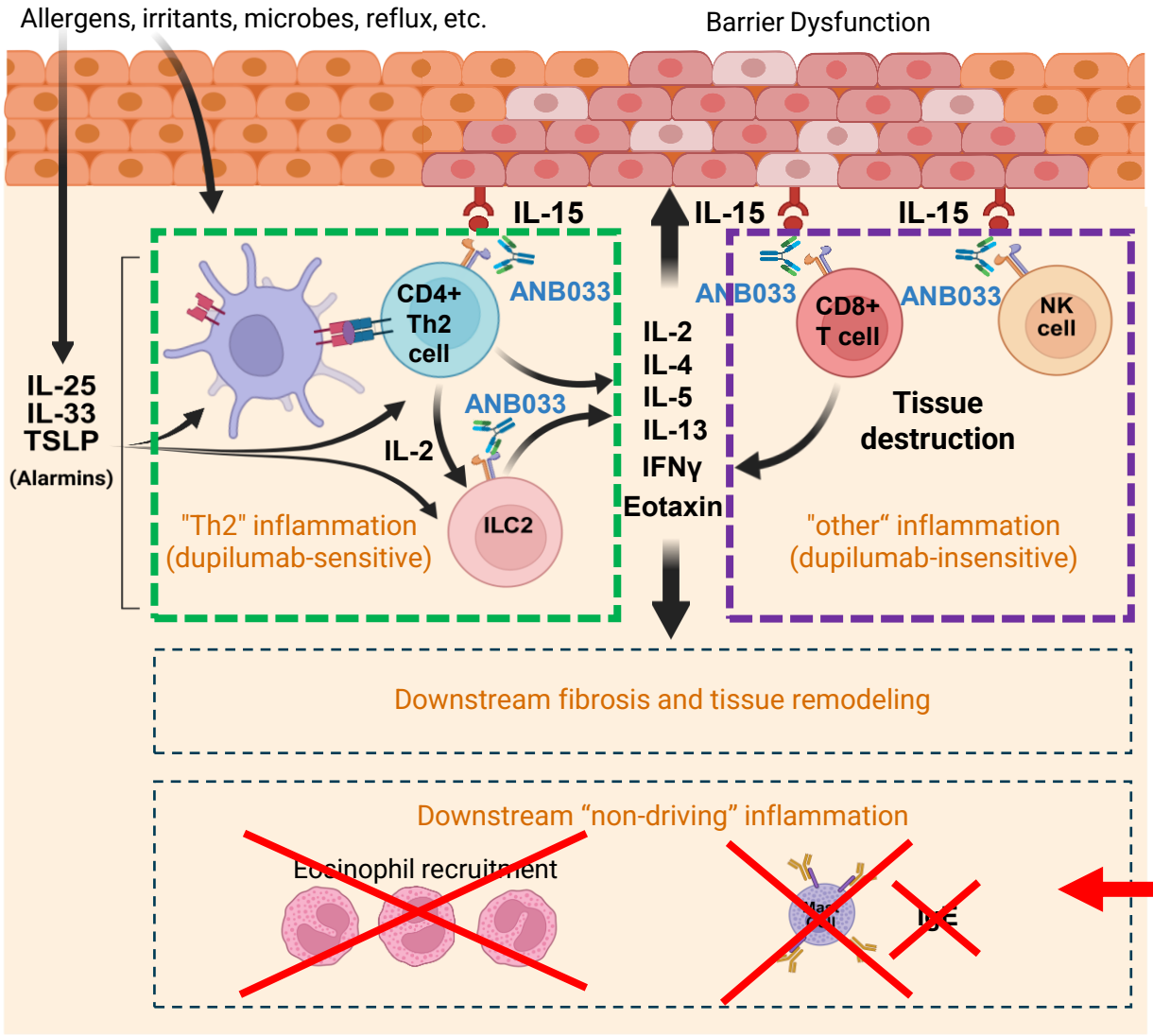
CD8+ and NK (dupilumab-insensitive)

- 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

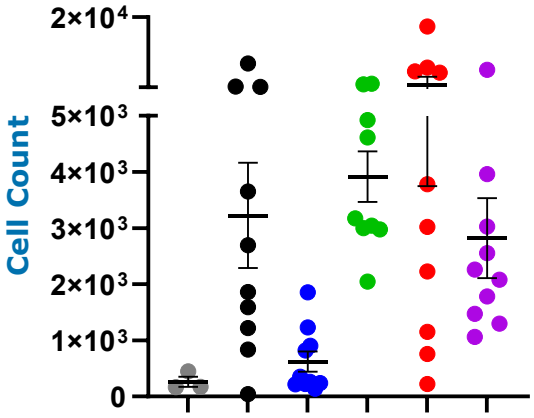
Celldex Genentech AstraZeneca

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

ANB033 prevents eosinophilia by targeting upstream inflammation

Aspergillus-induced eosinophilia

Esophageal eosinophils

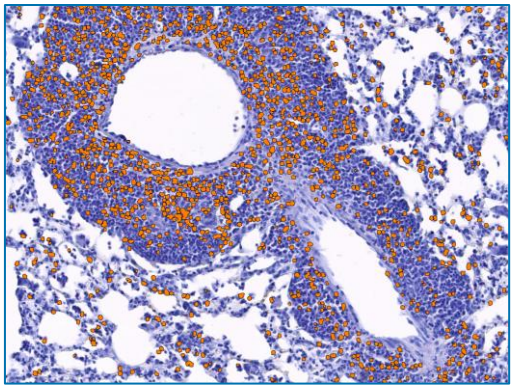


Treatment

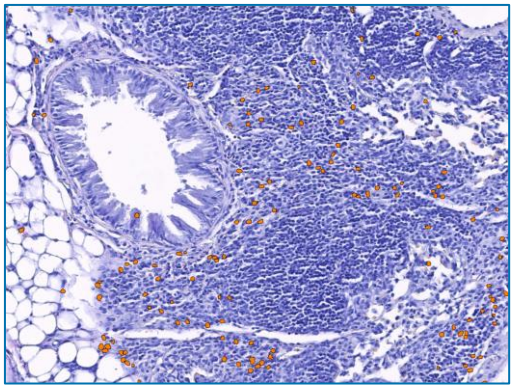
- Unchallenged
- Isotype control
- ANB033
- anti-mIL-15
- anti-mIL-13
- anti-mTSLP

Eos observed in histology

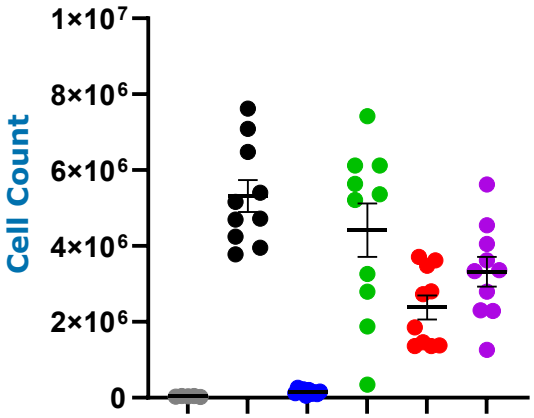
Isotype Control



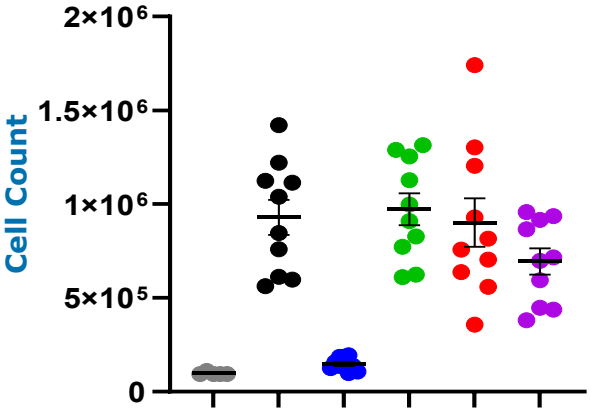
ANB033



Lung eosinophils



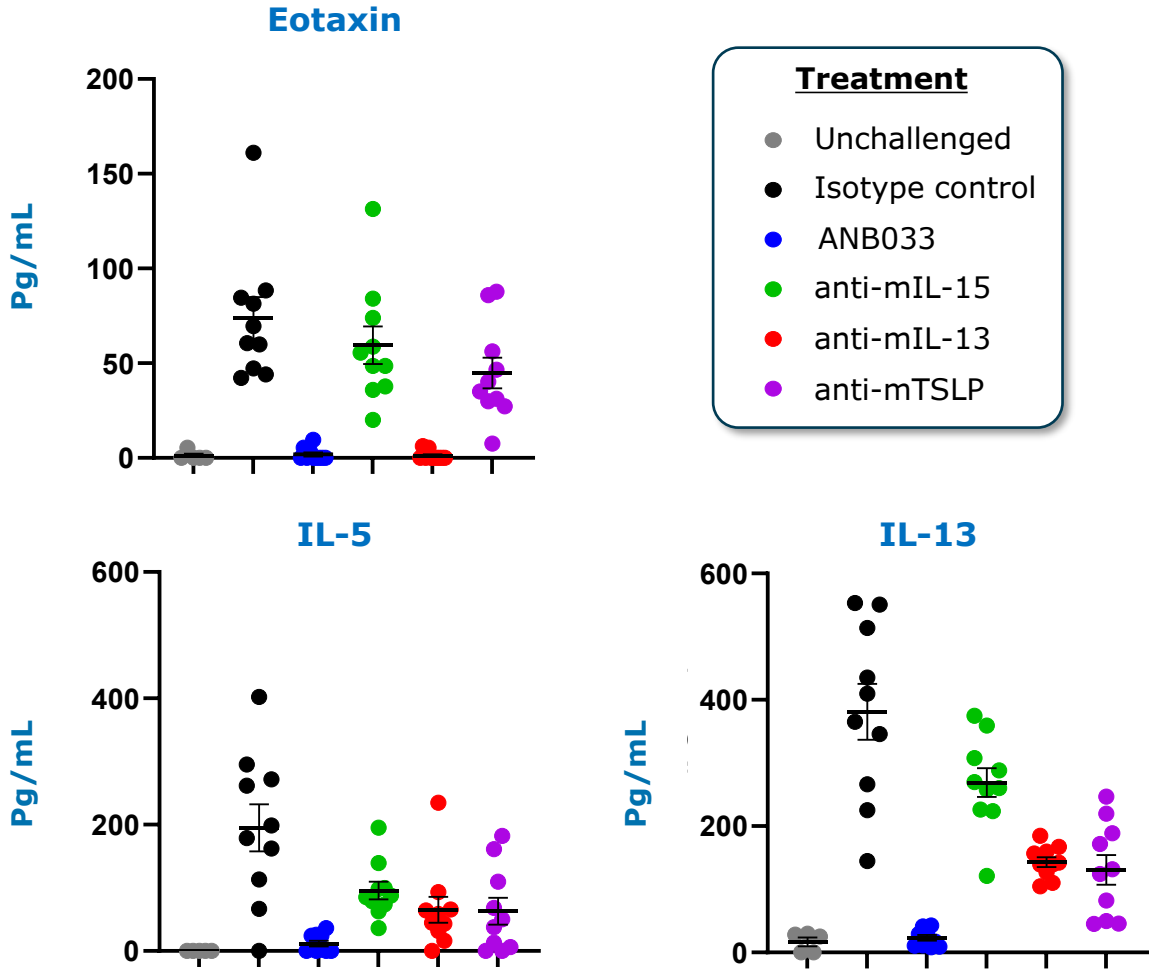
Lung ILC2s



Model of eosinophilic inflammation: Balb/c mice were challenged intranasally with *Aspergillus fumigatus* TIW for 3 weeks. The treatment regimen includes unchallenged control (PBS), isotype control, ANB033 surrogate antibody (anti-mouse CD122 antibody with similar binding epitope and affinity to ANB033), anti-mIL-15, anti-mIL-13 or anti-mTSLP, administered at 10 mg/kg BIW for 3 weeks. Tissues were assessed by flow cytometry or stained with H&E for histopathology assessment. Lung samples shown in graphic

ANB033 also prevents cytokine secretion and chemokine secretion responsible for eosinophil expansion and recruitment

Aspergillus-induced eosinophilia



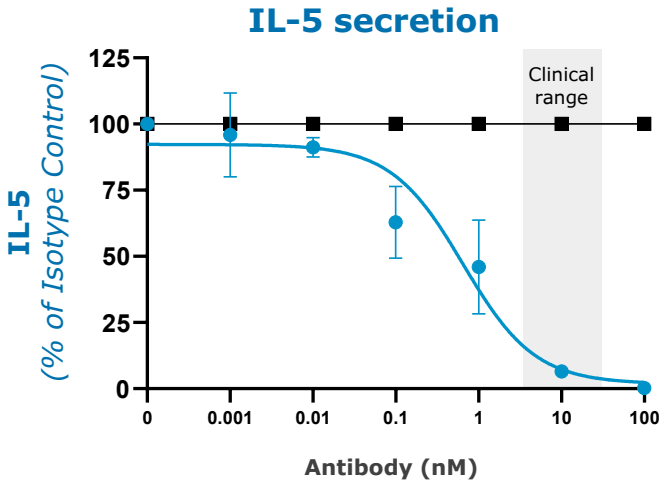
ANB033 prevents –

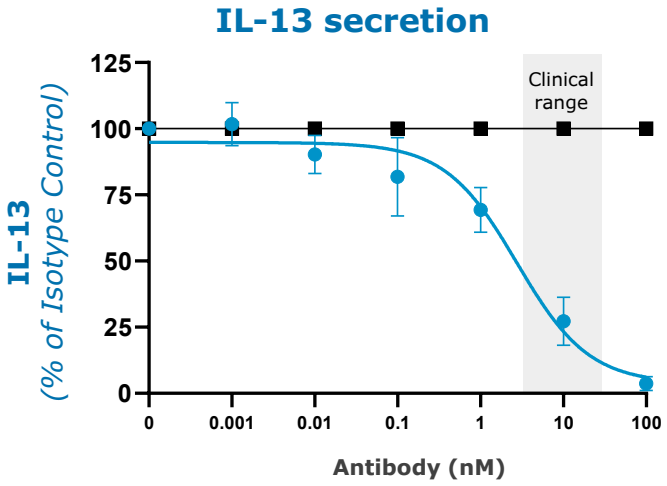
- Cytokine secretion (IL-5/IL-13) responsible for eos expansion
- Chemokine secretion (eotaxin) responsible for eos recruitment

Model of eosinophilic inflammation: Balb/c mice were challenged intranasally with *Aspergillus fumigatus* TIW for 3 weeks. The treatment regimen includes unchallenged control (PBS), isotype control, ANB033 surrogate antibody (anti-mouse CD122 antibody with similar binding epitope and affinity to ANB033), anti-mIL-15, anti-mIL-13 or anti-mTSLP, administered at 10 mg/kg BIW for 3 weeks. Detection of mIL-13 used a different epitope than neutralizing anti-mIL-13, so IL-13 bound by anti-mIL-13 is still detected via this method. Measured in Bronchial Alveolar Lavage Fluid (BALF).

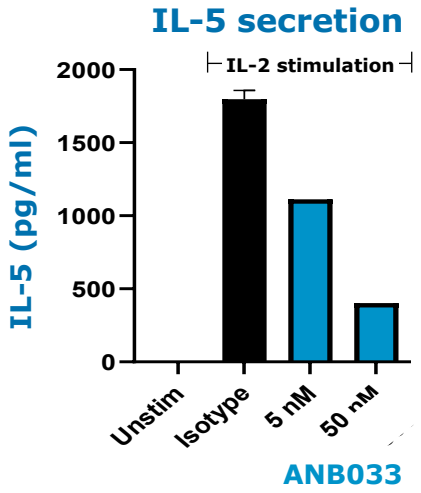
ANB033 reduces CD4+ T cell and ILC2 derived Th2 cytokines, proven drivers of EoE pathology

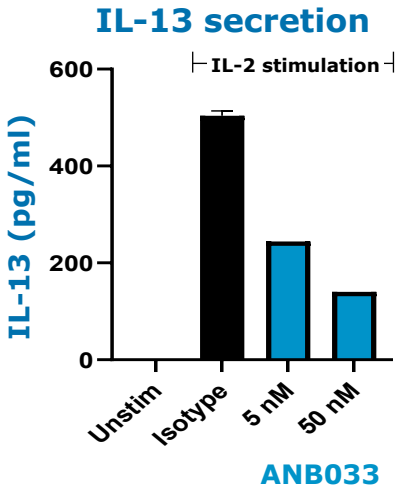
PBMC





ILC2

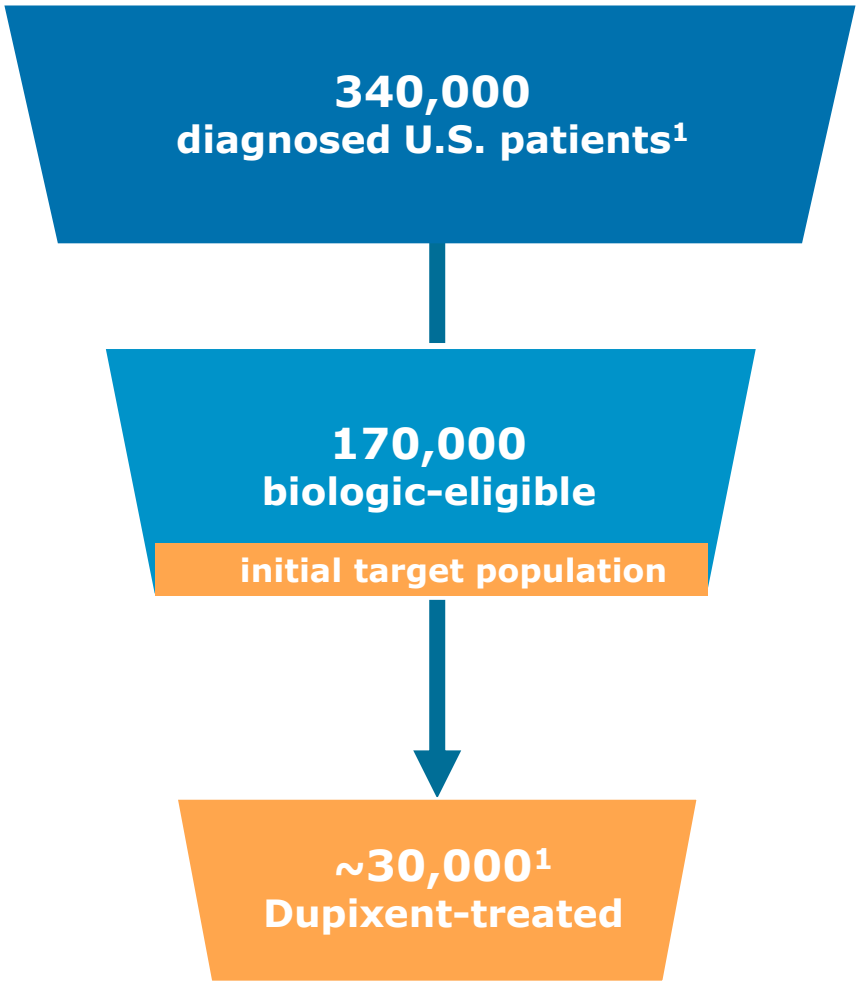




ANB033 Isotype Control

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.

EoE is a 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

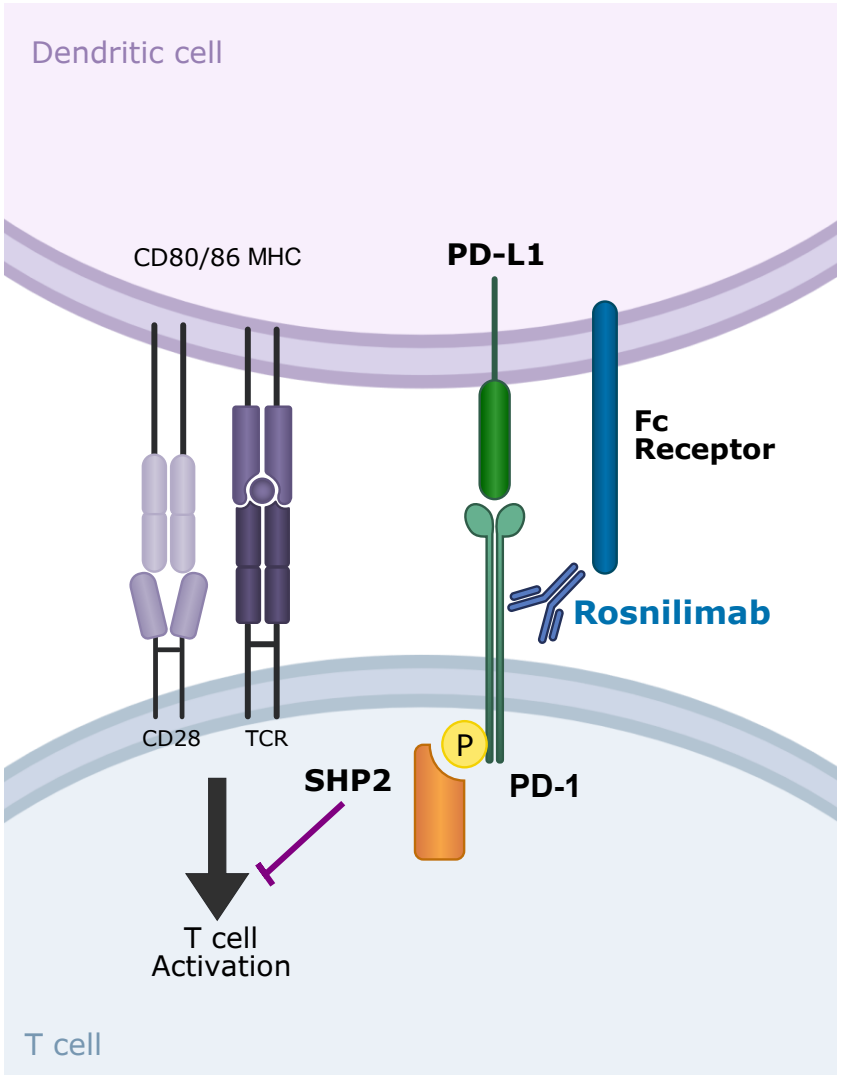
- Potential to reach IBD diagnosis and biologic penetration analogs given substantial unmet need

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)

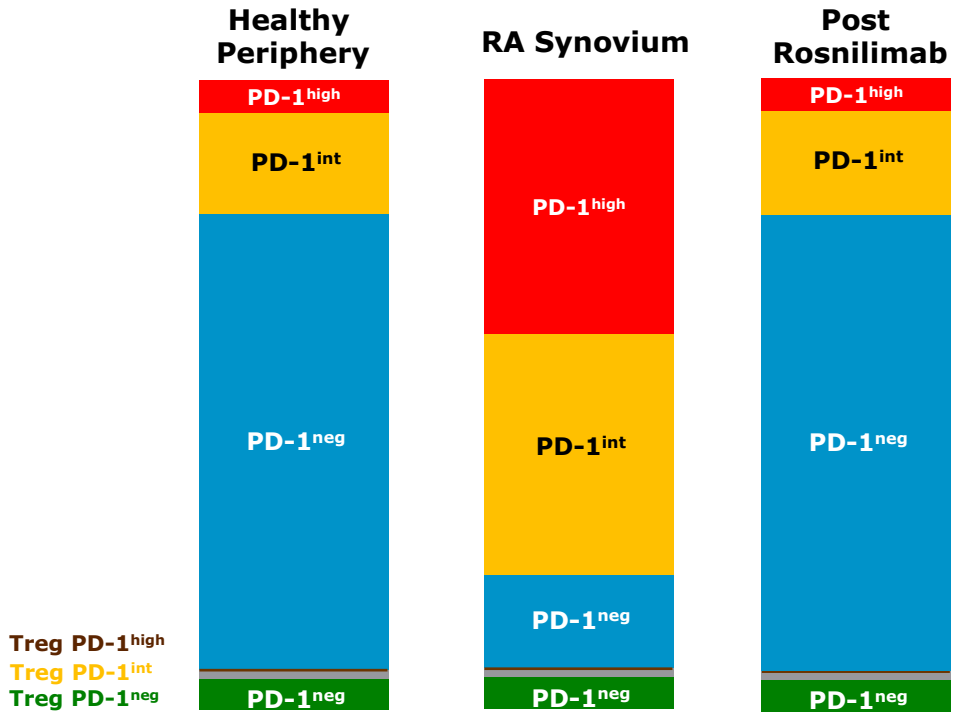
Rosnilimab selectively targets pathogenic T cells in periphery and inflamed tissue to restore immune homeostasis



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.

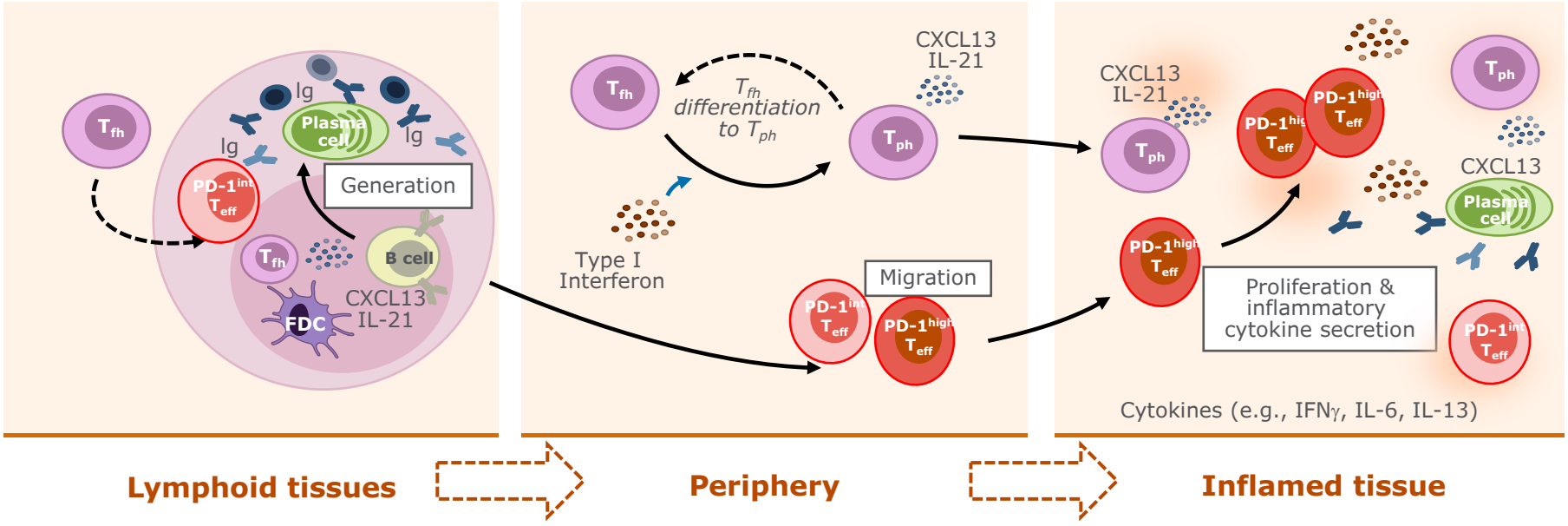
Pathogenic T_{eff} and T_{fh}/T_{ph} cells mediate autoimmune pathology

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

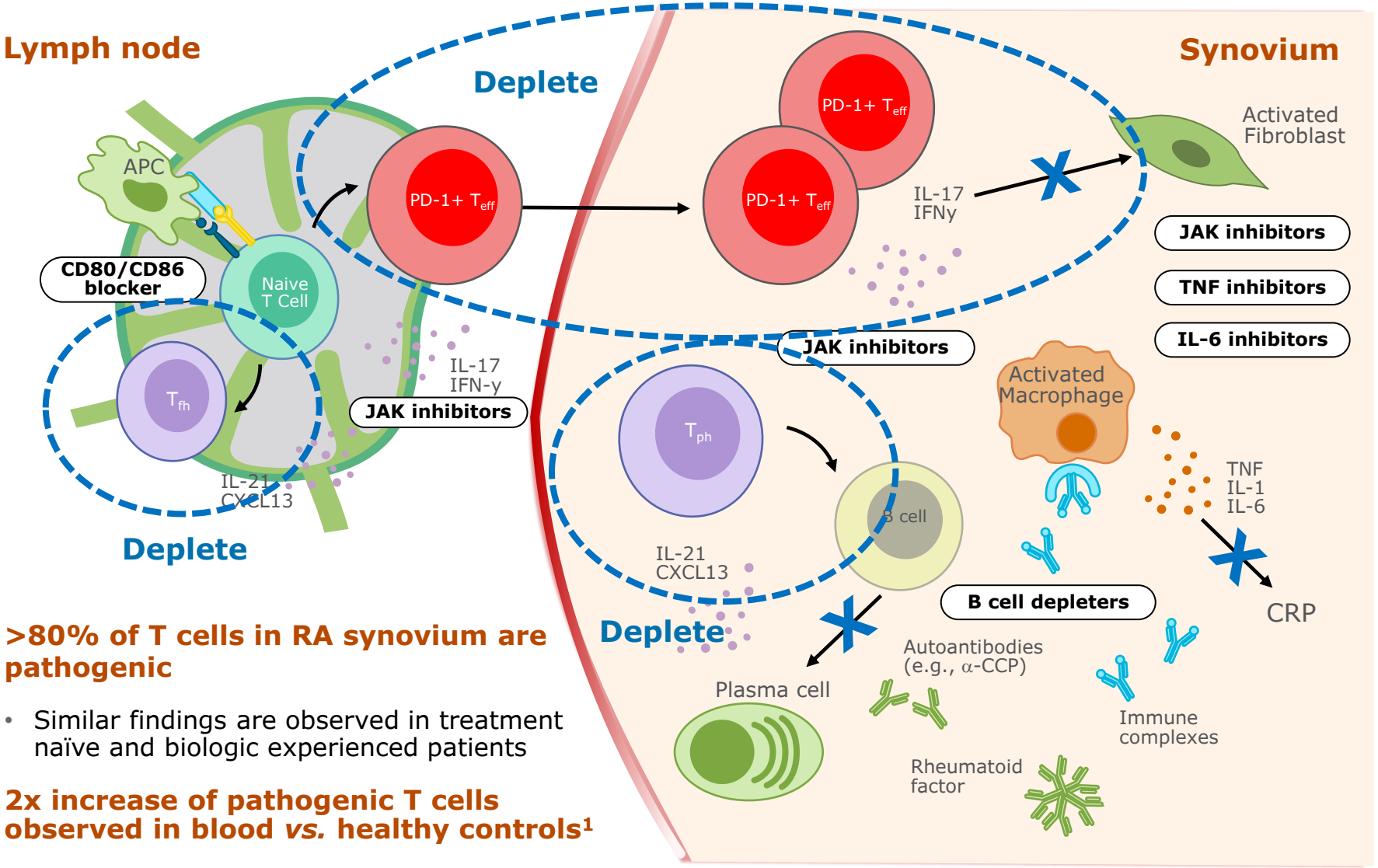
T_{eff} (effector)

- 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.

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



>80% of T cells in RA synovium are pathogenic

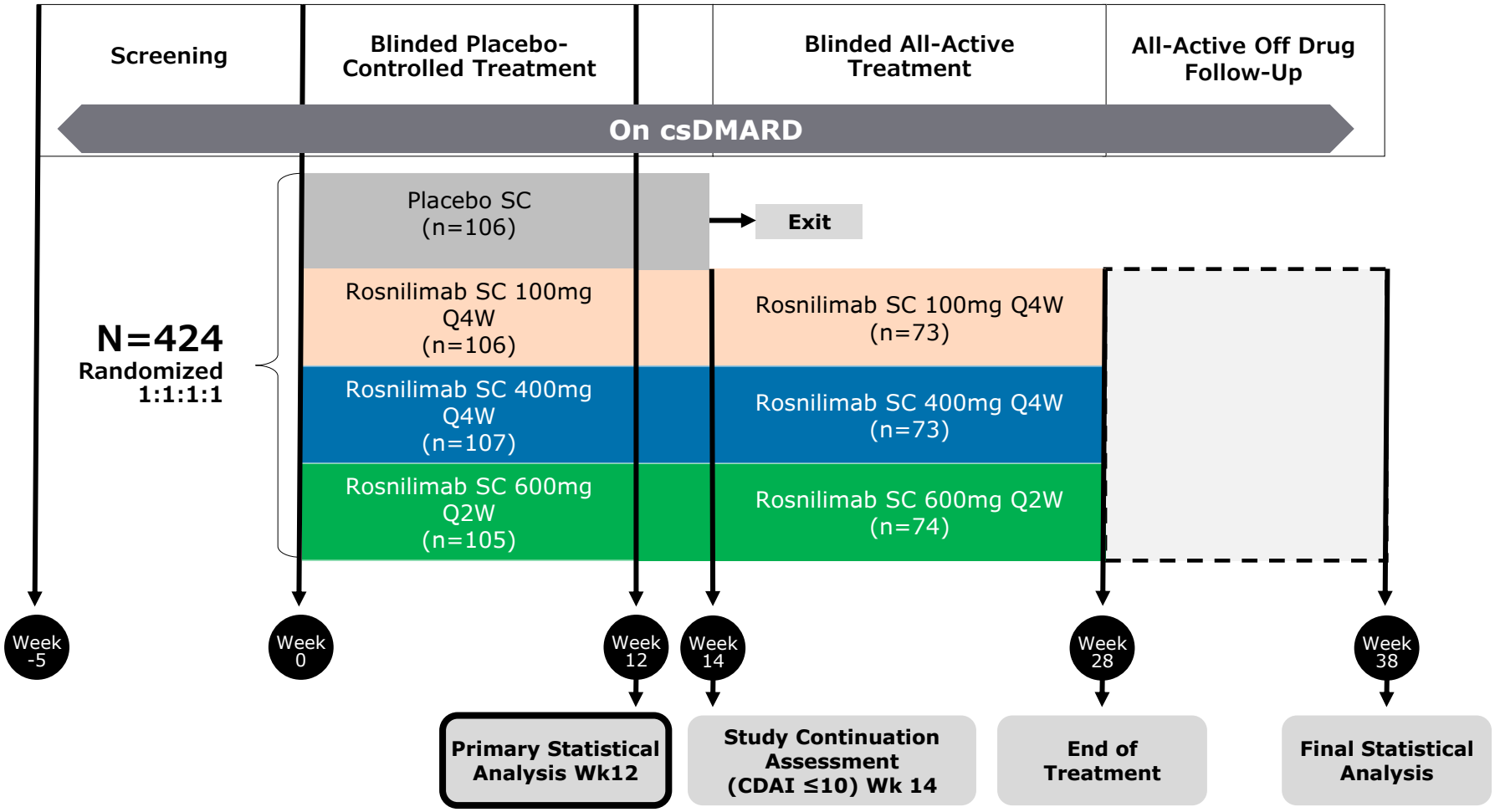
- Similar findings are observed in treatment naïve and biologic experienced patients

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



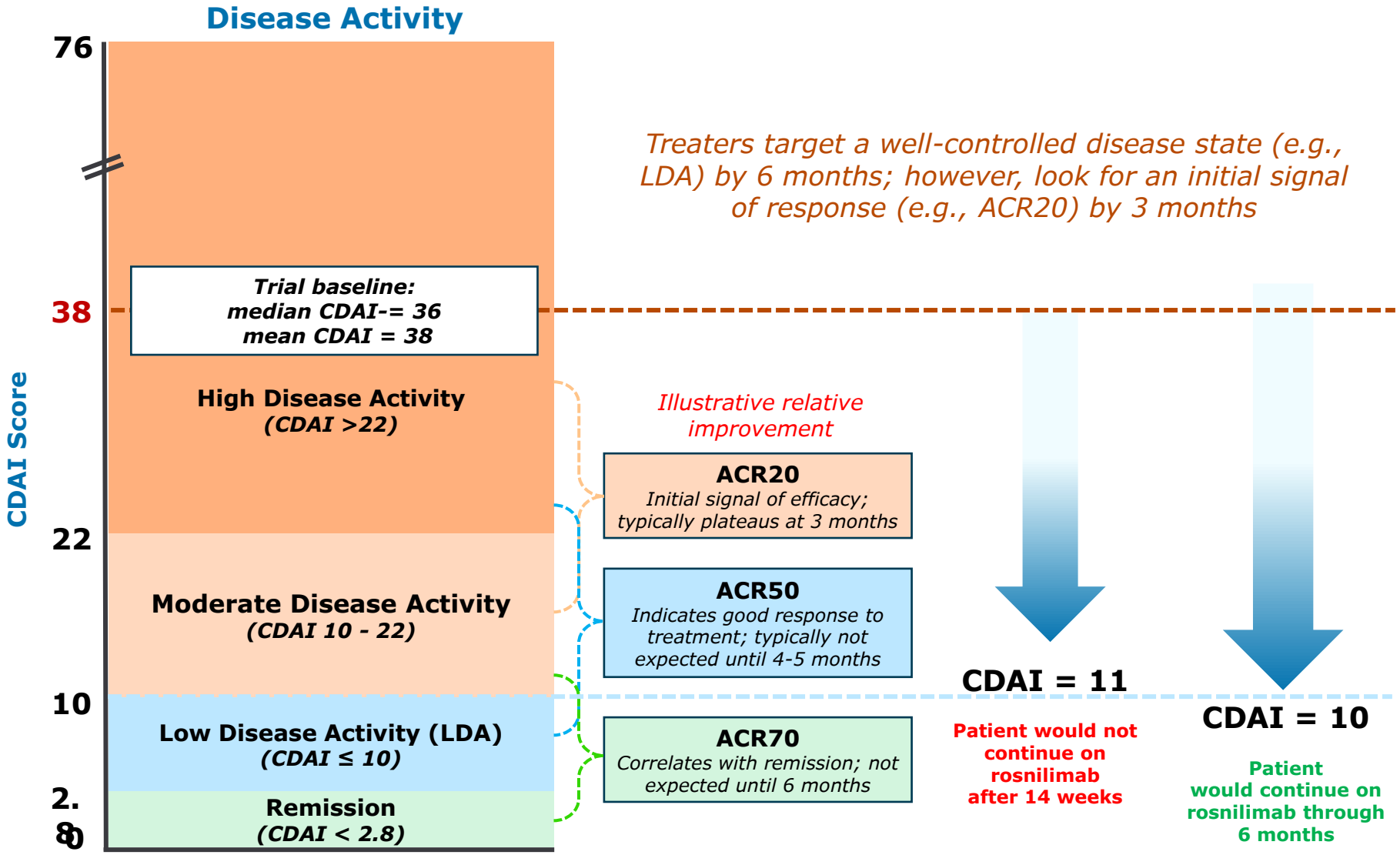
Key Inclusion Criteria – Seropositive RA, ≥6 swollen and ≥6 tender joints, hs-CRP ≥ 3mg/L during Screening, Concurrent use of 1 or 2 csDMARDs that were initiated at least 3 months before screening

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

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



Rosnilimab demonstrates best-in-disease profile in RA

Late-breaking oral presentation by Professor Paul Emery at ACR Convergence 2025

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)

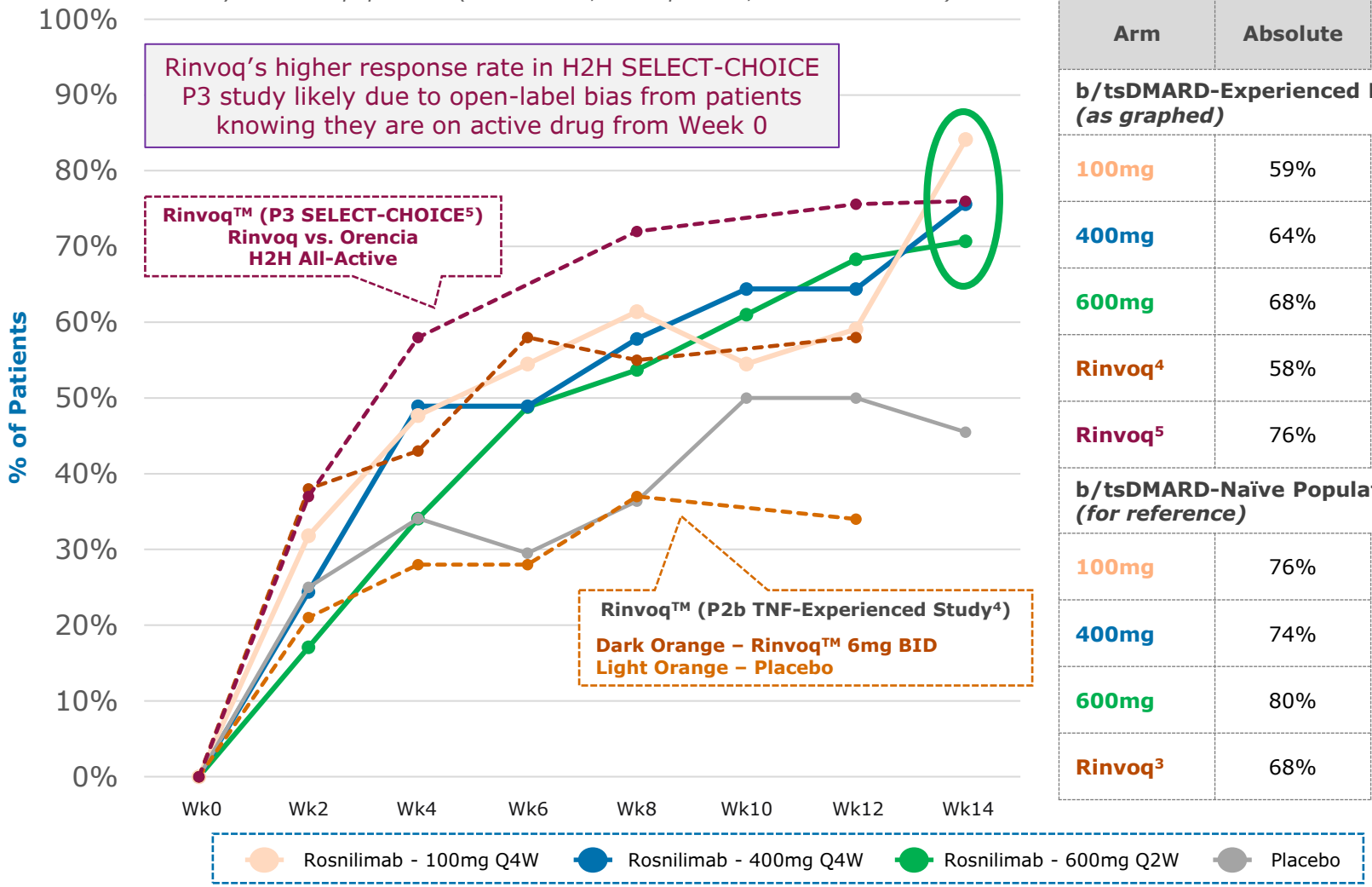
Rosnilimab, a pathogenic T cell depleter, is well-positioned for the ~$20 billion U.S. RA market which hasn't had a new mechanism approved since 2012

ACR20 response rates are comparable to Rinvoq™

Most patients had symptomatic and clinical improvement by 3 months

ACR20: b/tsDMARD-Experienced[1]

NRI analysis on ITT population (n=174 total; n=44 placebo, n=130 rosnilimab)[2]



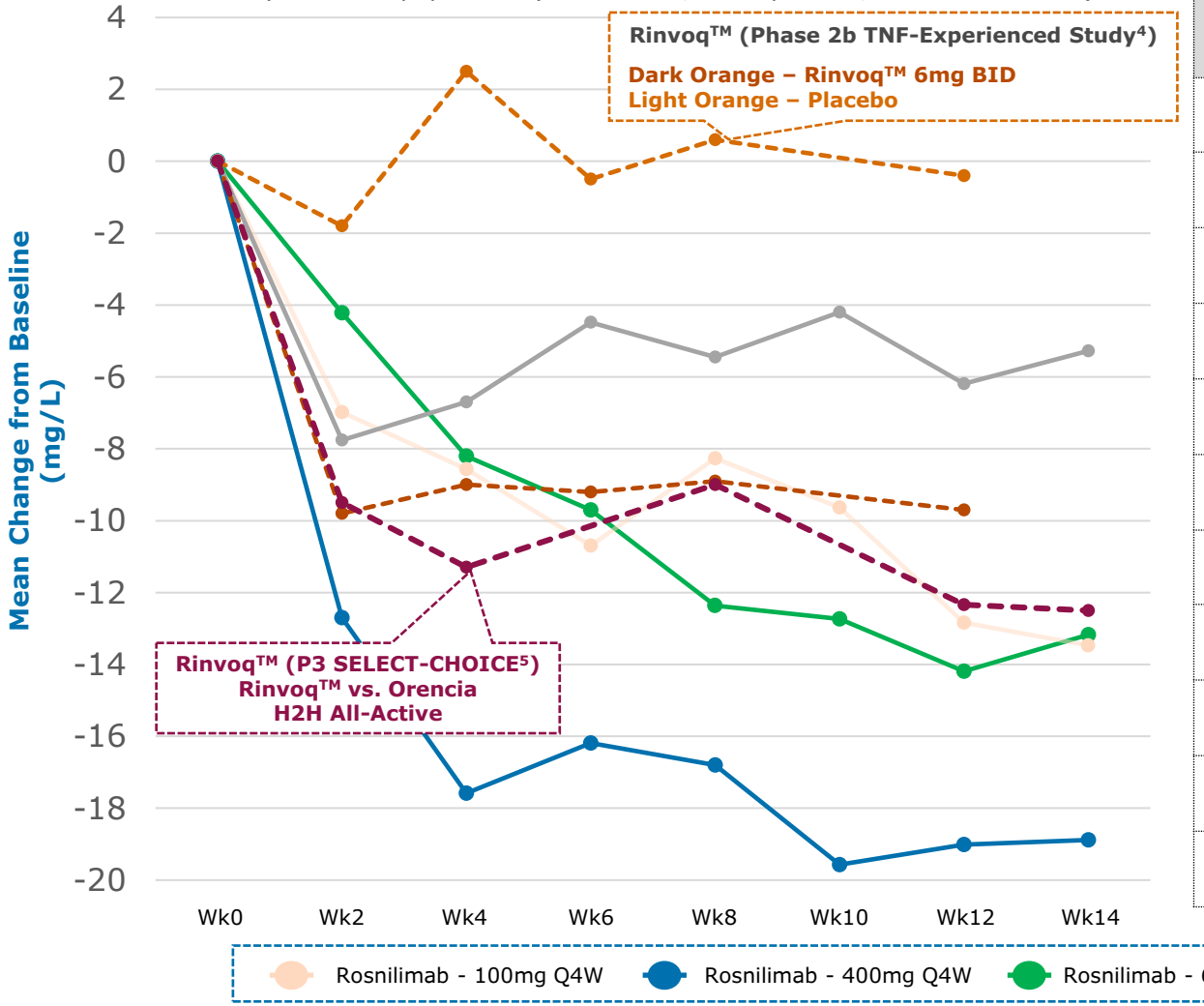
ACR20 at Week 12		
Arm	**Absolute**	**PBO Adjusted**
b/tsDMARD-Experienced Population *(as graphed)*		
100mg	59%	*9%*
400mg	64%	*14%*
600mg	68%	*18%*
Rinvoq[4]	58%	*24%*
Rinvoq[5]	76%	*N/A*
b/tsDMARD-Naïve Population *(for reference)*		
100mg	76%	*21%*
400mg	74%	*19%*
600mg	80%	*25%*
Rinvoq[3]	68%	*22%*

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)

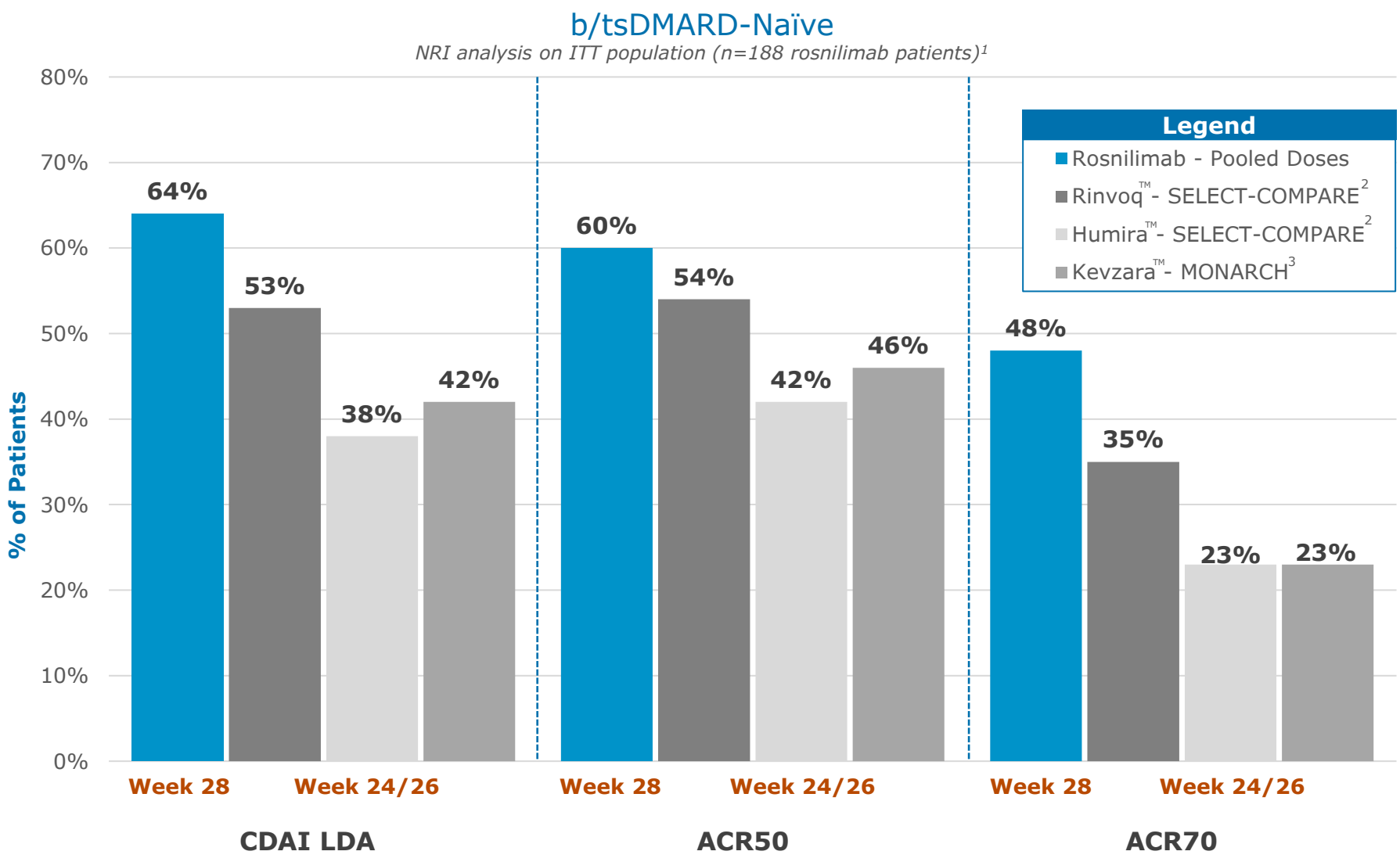


Change in CRP at Week 12		
Arm	**Baseline Mean CRP**	**PBO Adjusted**
b/tsDMARD-Experienced Population *(as graphed)*		
100mg	20.0	*-6.7*
400mg	29.4	*-12.8*
600mg	23.3	*-8.0*
Rinvoq[4]	16.0	*-9.3*
Rinvoq[5]	19.0	*N/A*
b/tsDMARD-Naïve Population *(for reference)*		
100mg	14.9	*-10.6*
400mg	14.3	*-7.0*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

Rosnilimab shows JAK-like efficacy in naïve patients

Compares favorably despite most conservative analysis and capped trial design

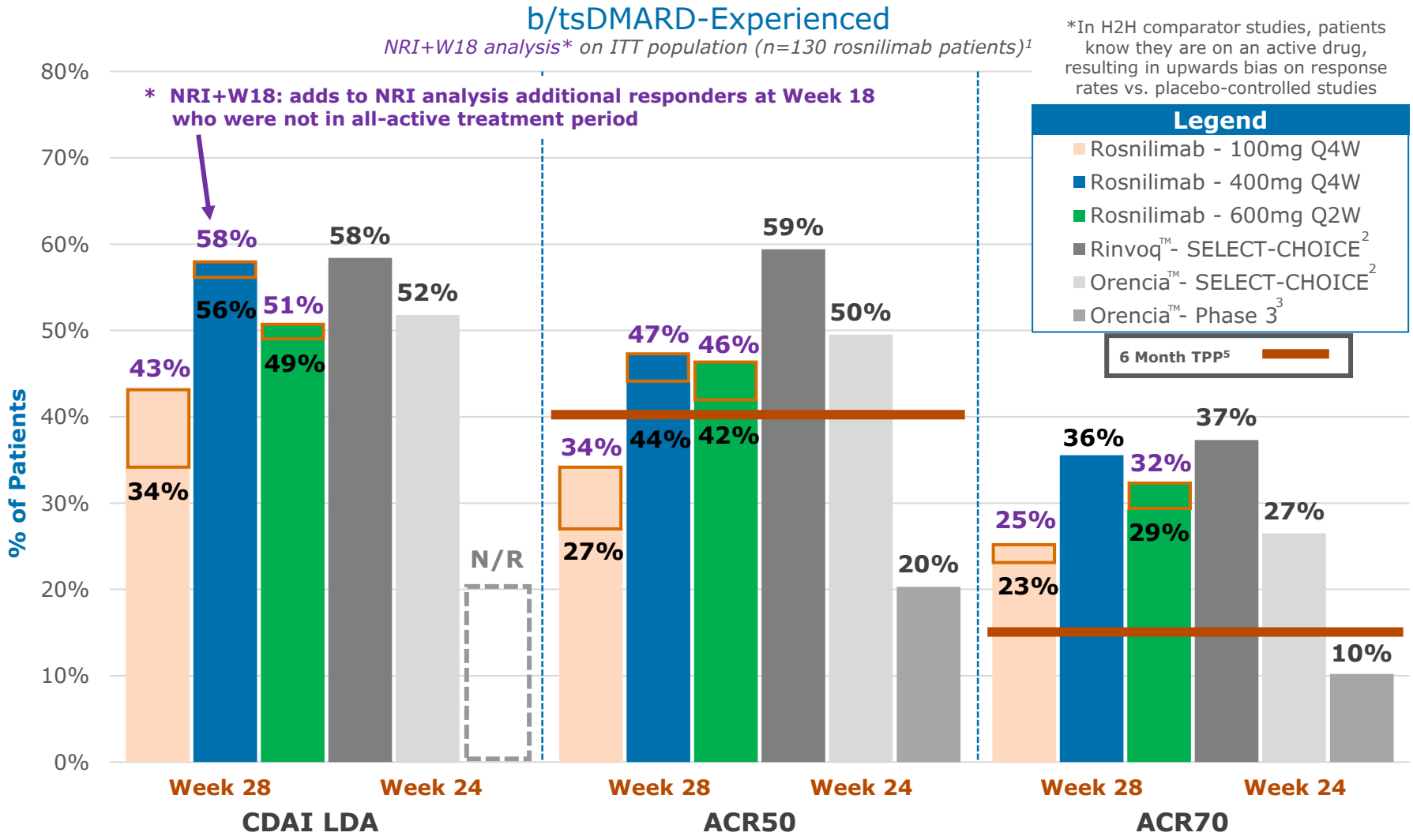


1. Non-responder imputated (NRI) analysis on intent-to-treat (ITT) of all b/tsDMARD-naïve patients randomized; b/tsDMARD-naïve population (n=62 100mg Q4W, n=62 400mg Q4W, n=64 600mg Q2W; n=188 total rosnilimab b/tsDMARD-naïve patients); 2. SELECT-COMPARE Phase 3 study; 3. Kevzara Phase 3 study; NRI data;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

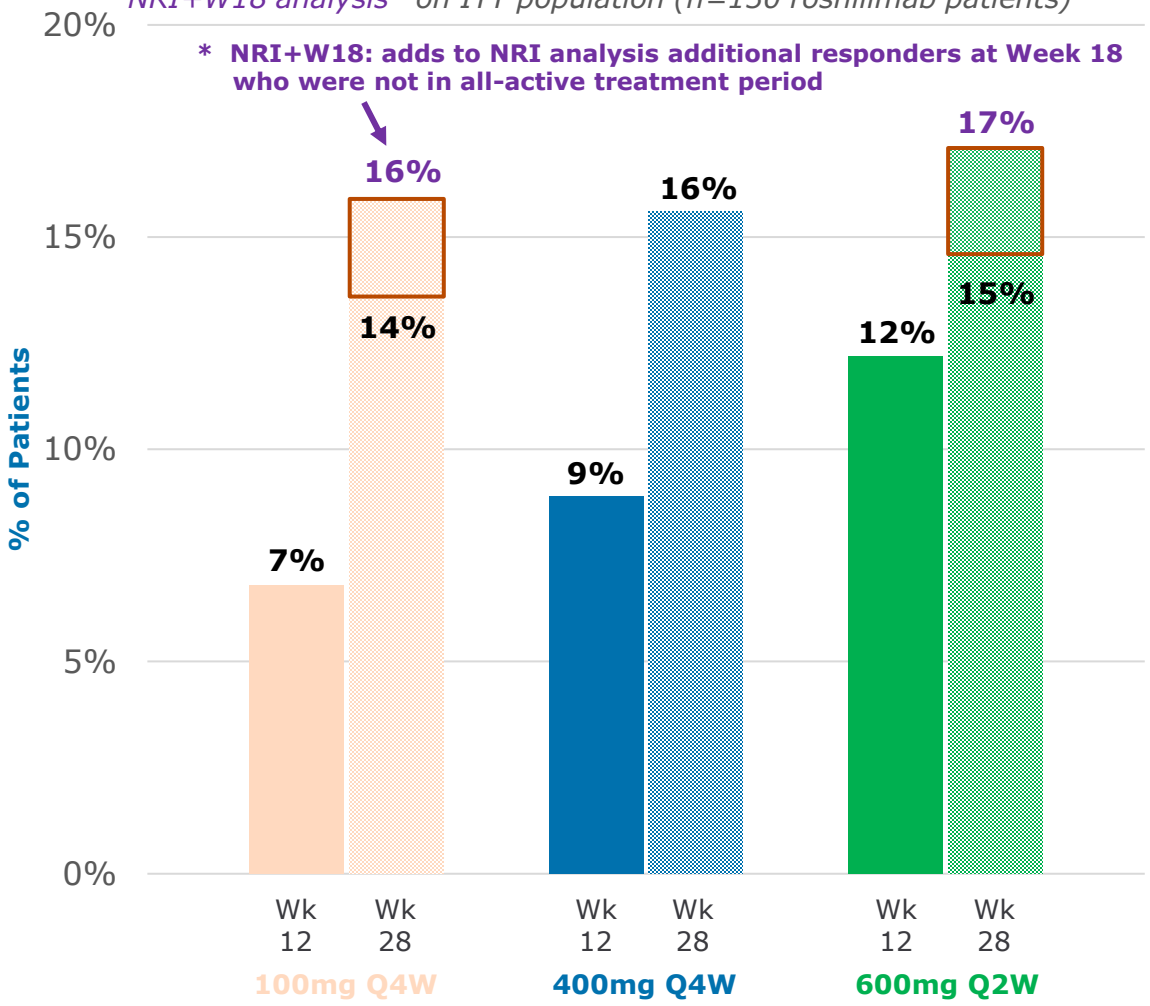
JAK-like CDAI remission rates which deepened into six months

Includes 29% with prior JAK experience

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

CDAI Remission: b/tsDMARD-Experienced

NRI+W18 analysis on ITT population (n=130 rosnilimab patients)*[1]



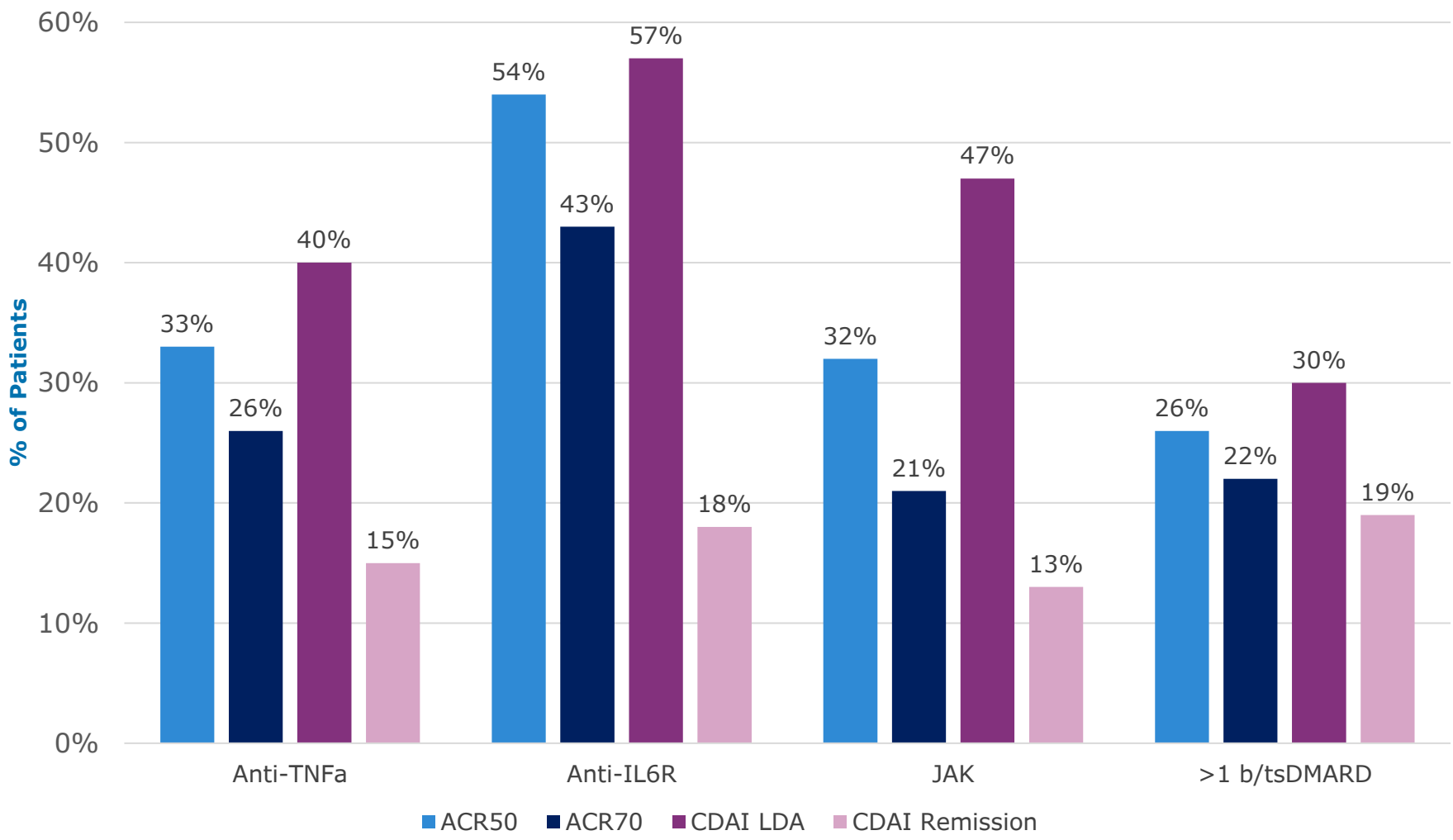
CDAI Remission at Week 28		
Arm	**NRI**	**NRI+W18**
b/tsDMARD-Experienced Population *(as graphed)*		
100mg	14%	16%
400mg	16%	16%
600mg	15%	17%
b/tsDMARD-Naïve Population		
100mg	21%	21%
400mg	18%	18%
600mg	17%	19%

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)

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

Rosnilimab Week 28 Responses Based on Prior Therapeutic Agent

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



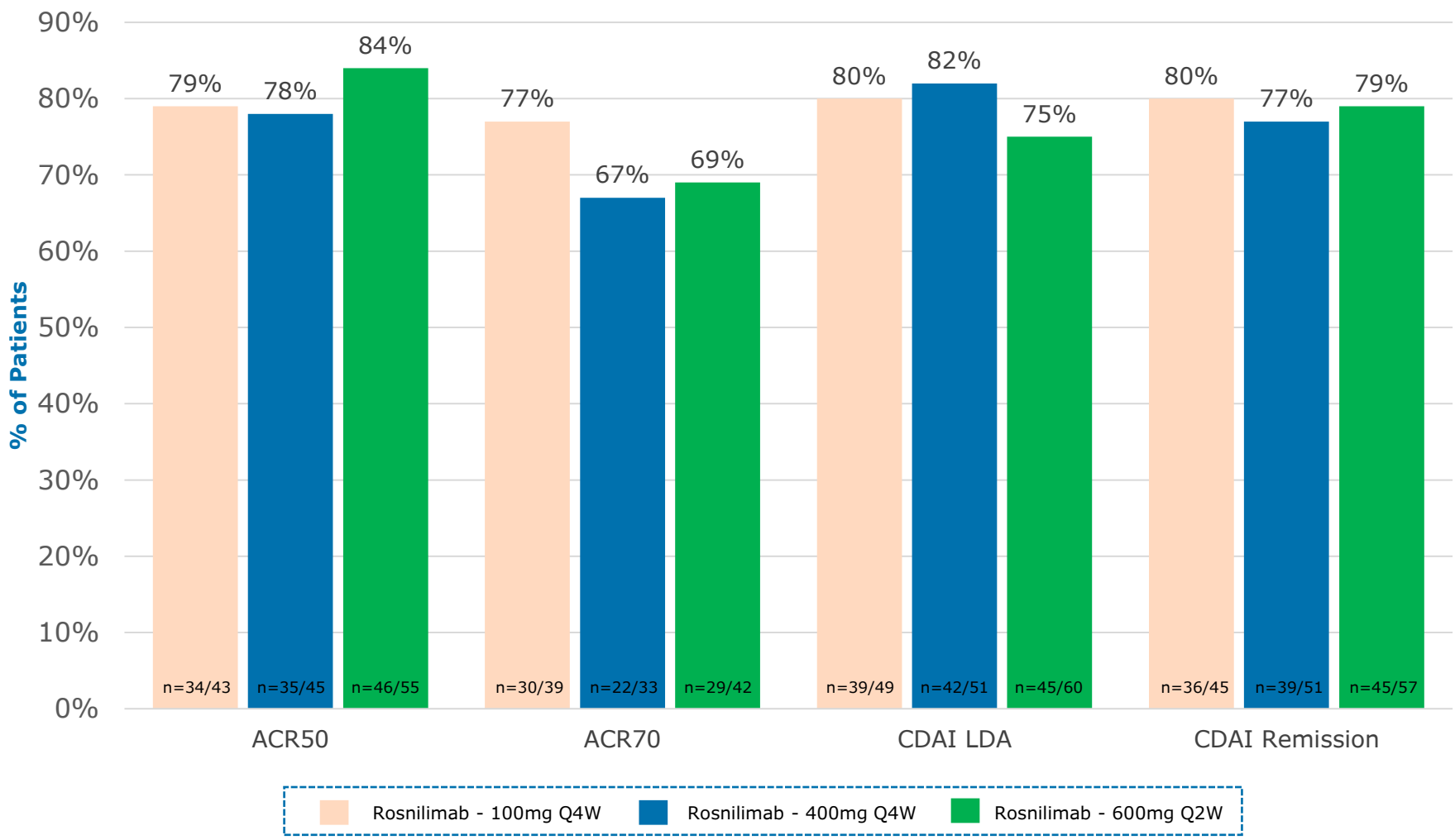
Graf et. al, "Rosnilimab, a Selective and Potent Depleter of Pathogenic T Cells, Demonstrates Efficacy, Safety and Translational Proof of Mechanism in a Rheumatoid Arthritis Phase 2B Trial", ACR Convergence, October 2025

Durable responses for 3-months off-drug

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

Rosnilimab Week 28 Responders Maintaining Response Off-Drug (Week 38)

Week 38 complete analysis



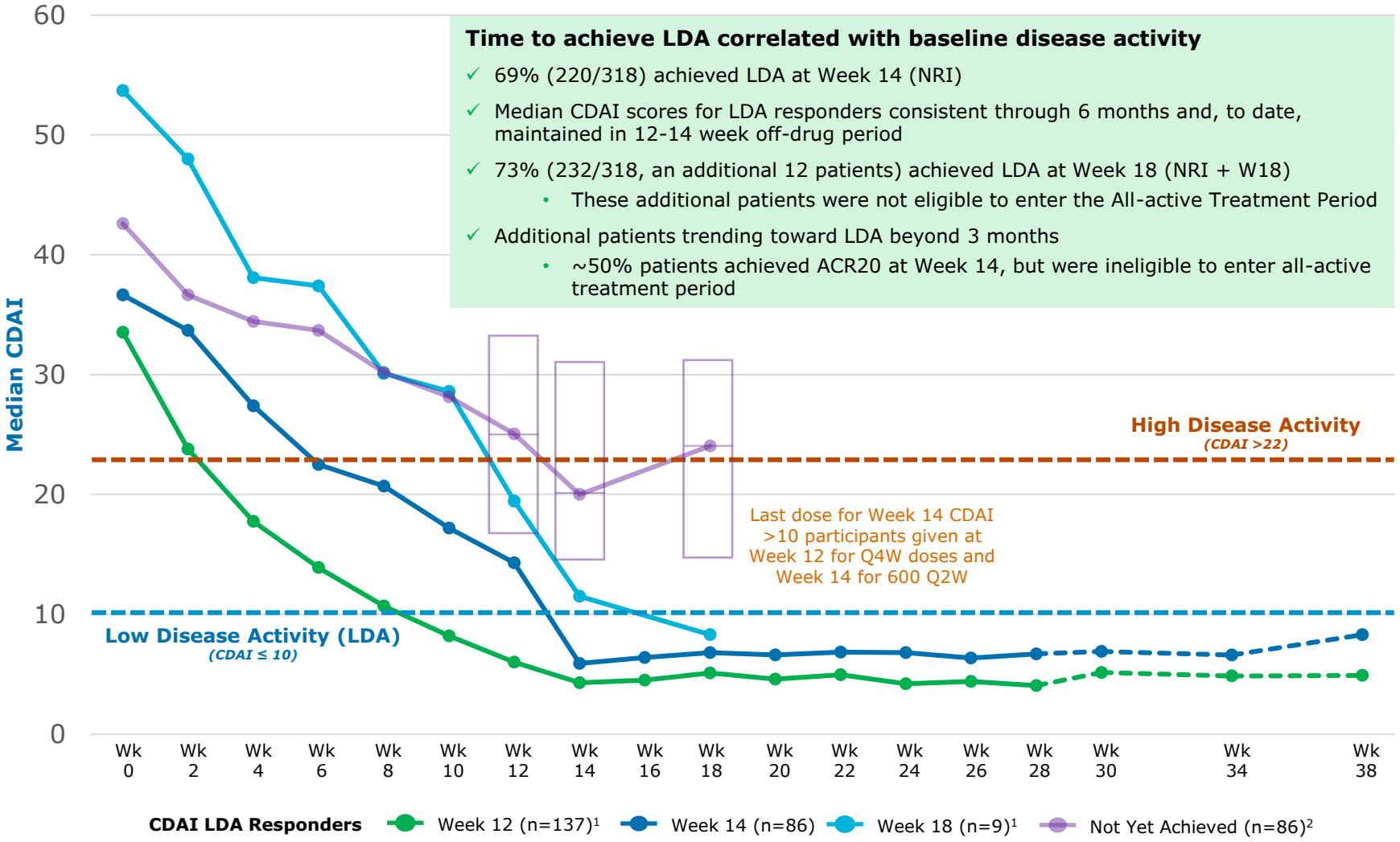
Graf et. al, "Rosnilimab, a Selective and Potent Depleter of Pathogenic T Cells, Demonstrates Efficacy, Safety and Translational Proof of Mechanism in a Rheumatoid Arthritis Phase 2B Trial", ACR Convergence, October 2025

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)

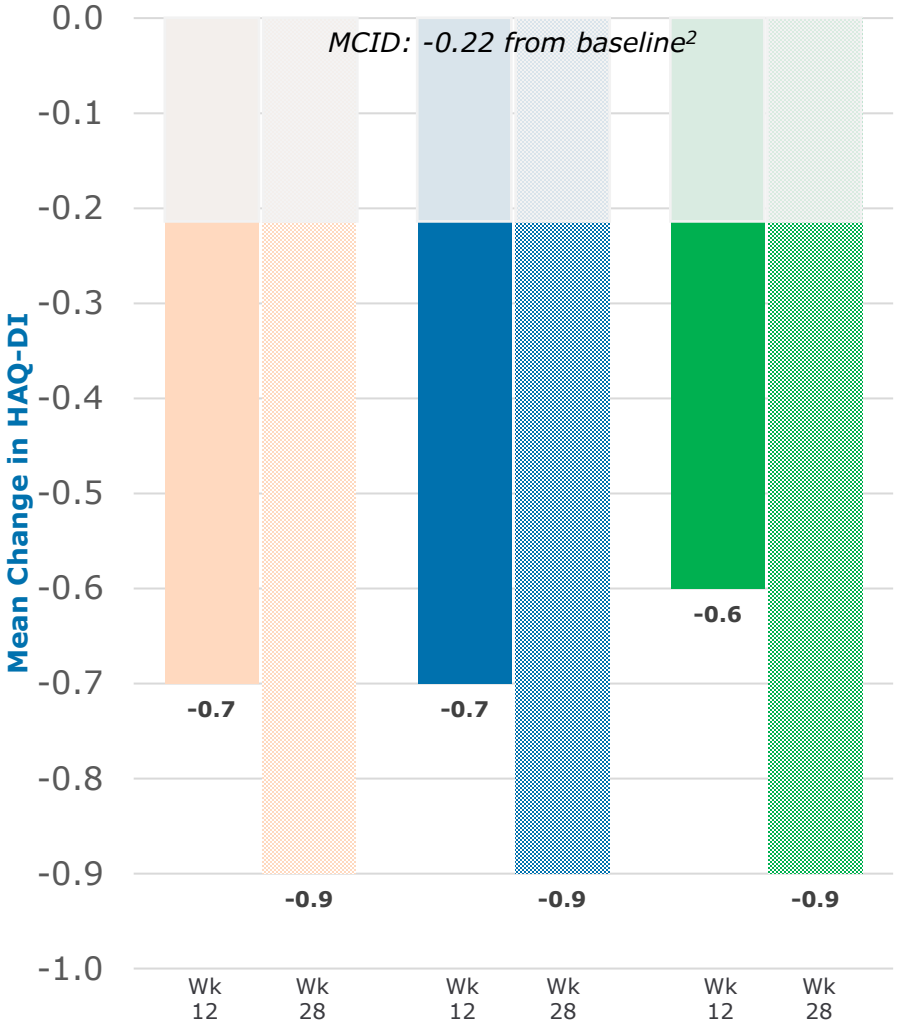


1. Green line includes 3 patients that achieved LDA at Week 12, were not CDAI LDA at Week 14, but returned to CDAI LDA at Week 18. These same 3 patients were excluded from the Light Blue line. In total 12 patients achieved CDAI LDA at Week 18. 2. Purple line includes rosnilimab patients that discontinued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

Highly meaningful clinically and symptomatic improvement across multiple PROs and CRP

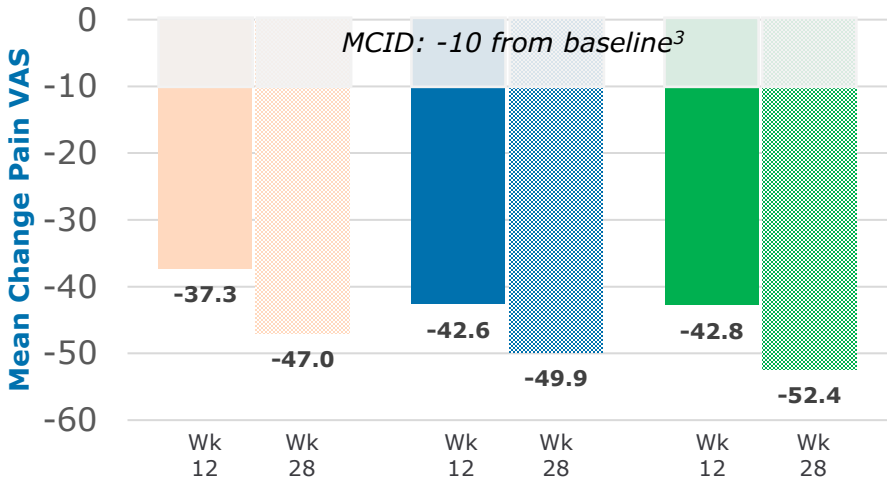
HAQ-DI: Overall

MMRM analysis on CDAI Responder Population (n=220)[1]



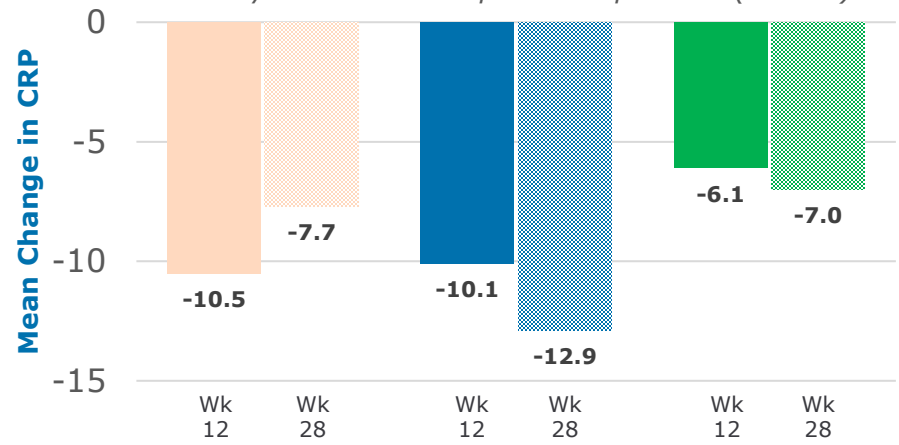
Pain VAS: Overall

MMRM analysis on CDAI Responder Population (n=220)[1]



CRP: Overall

MMRM analysis on CDAI Responder Population (n=220)[1]

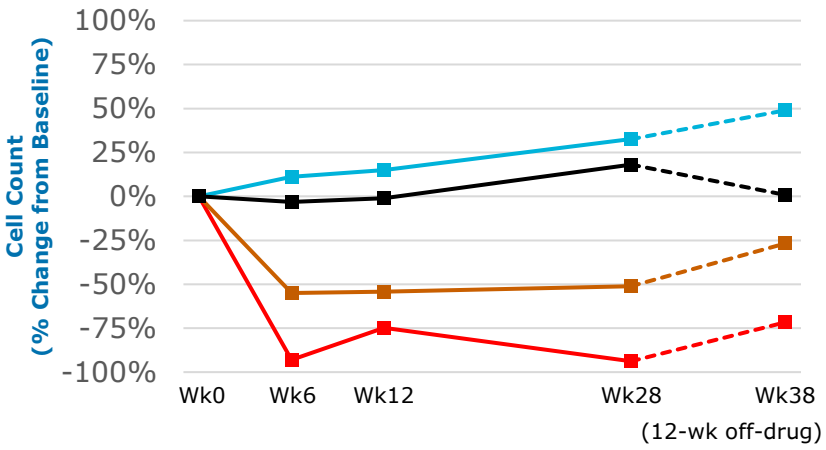


Rosnilimab - 100mg Q4W | Rosnilimab - 400mg Q4W | Rosnilimab - 600mg Q2W

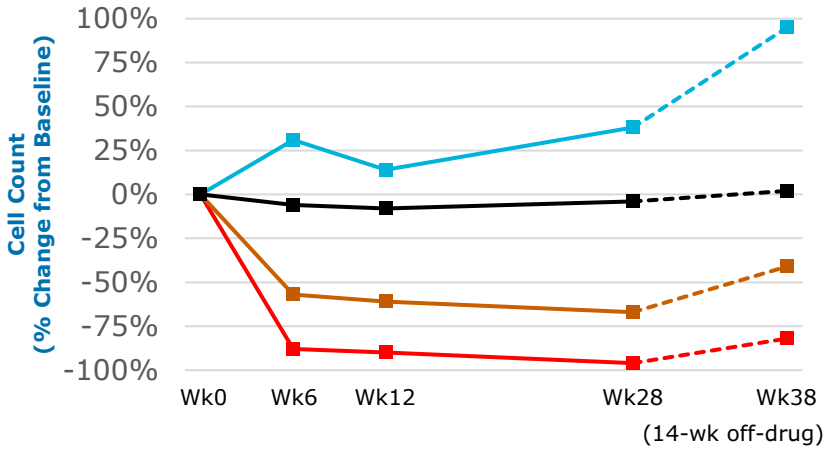
1. Mixed Model for Repeated Measures (MMRM) analysis on rosnilimab CDAI LDA responder at Week 14 population (n=220) includes naïve population (n=46 100mg Q4W, n=40 400mg Q4W, n=48 600mg Q2W; n=134 total rosnilimab patients) and experienced population (n=27 100mg Q4W, n=33 400mg Q4W, n=26 600mg Q2W; n=86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

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

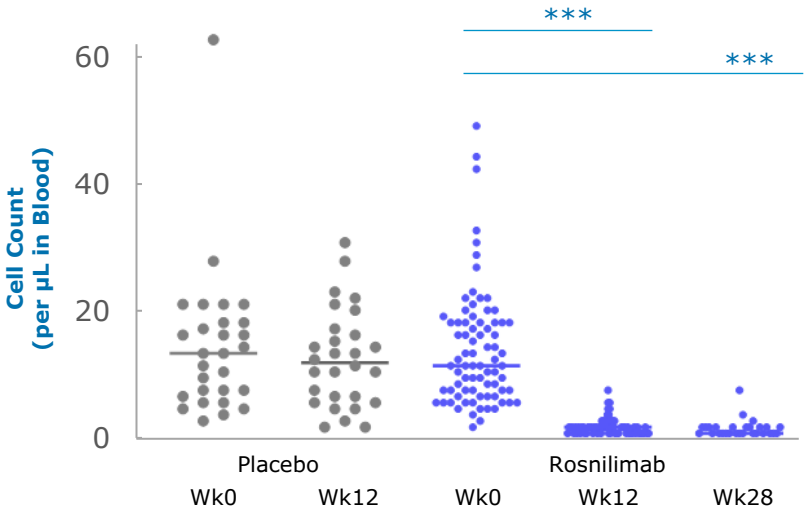
Rosnilimab 400mg Q4W T Cell Impact



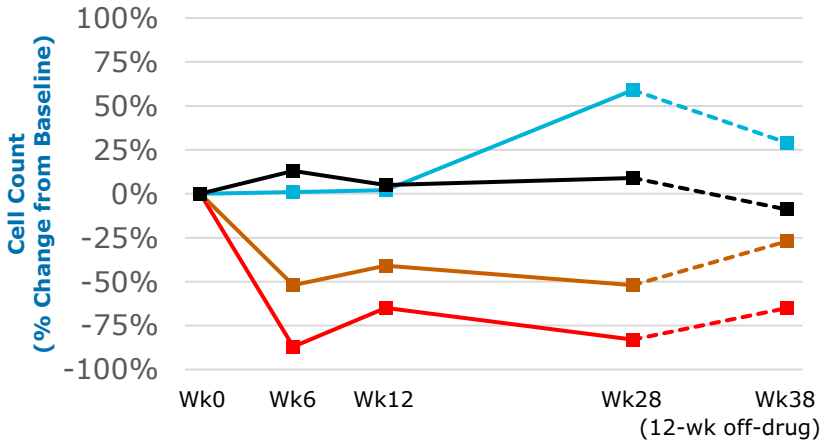
Rosnilimab 600mg Q2W T Cell Impact



Rosnilimab T_{ph} Impact – Pooled Doses

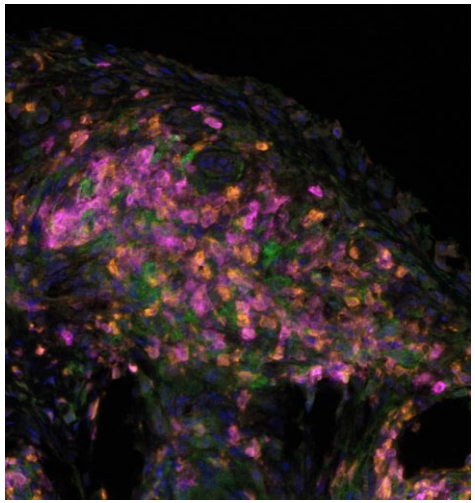
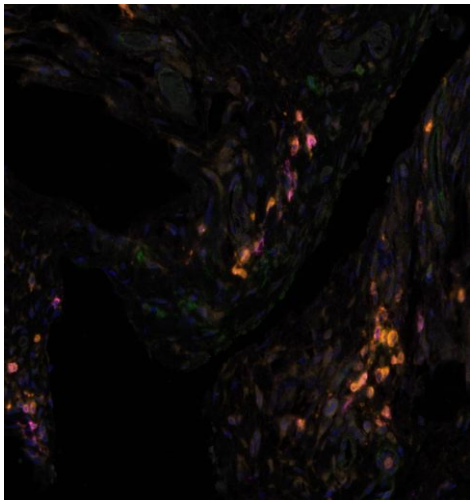
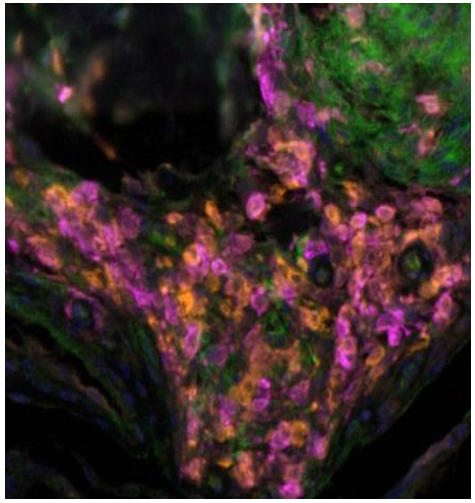
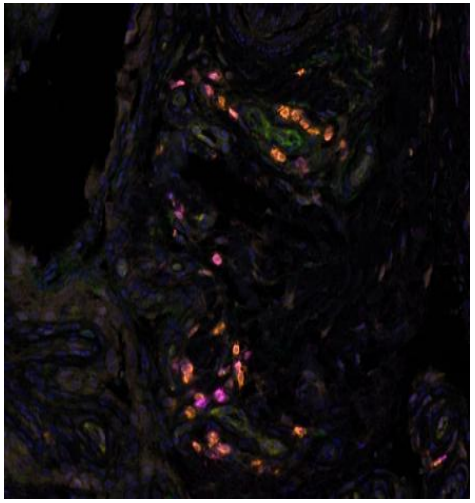


Rosnilimab 100mg Q4W T Cell Impact



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

	Baseline	Week 6
Rosnilimab 400mg Q4W		
Rosnilimab 600mg Q2W		

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

400mg/600mg doses
~90% reduction

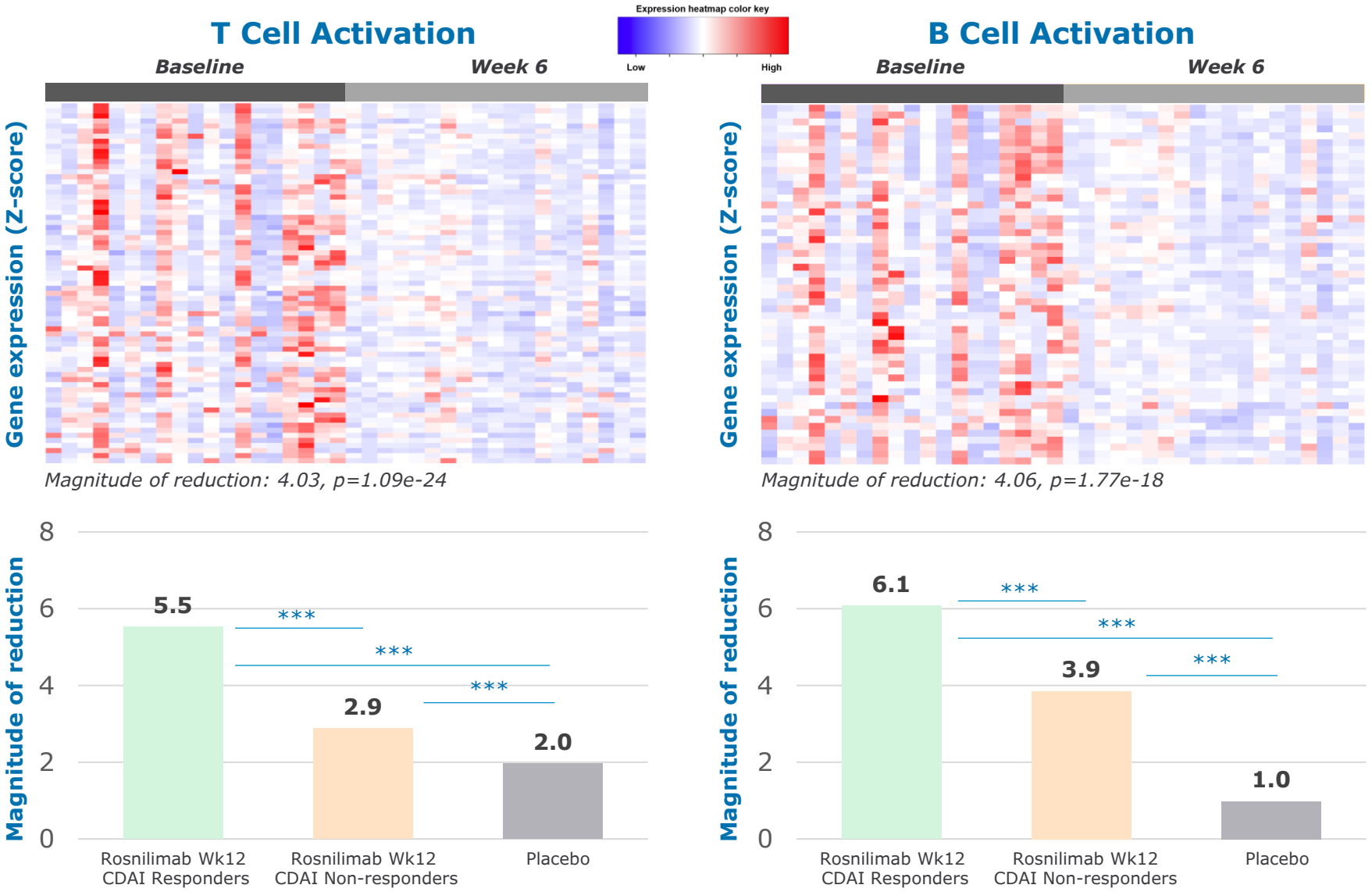
100mg dose
Inconclusive reduction

Placebo
Increased

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

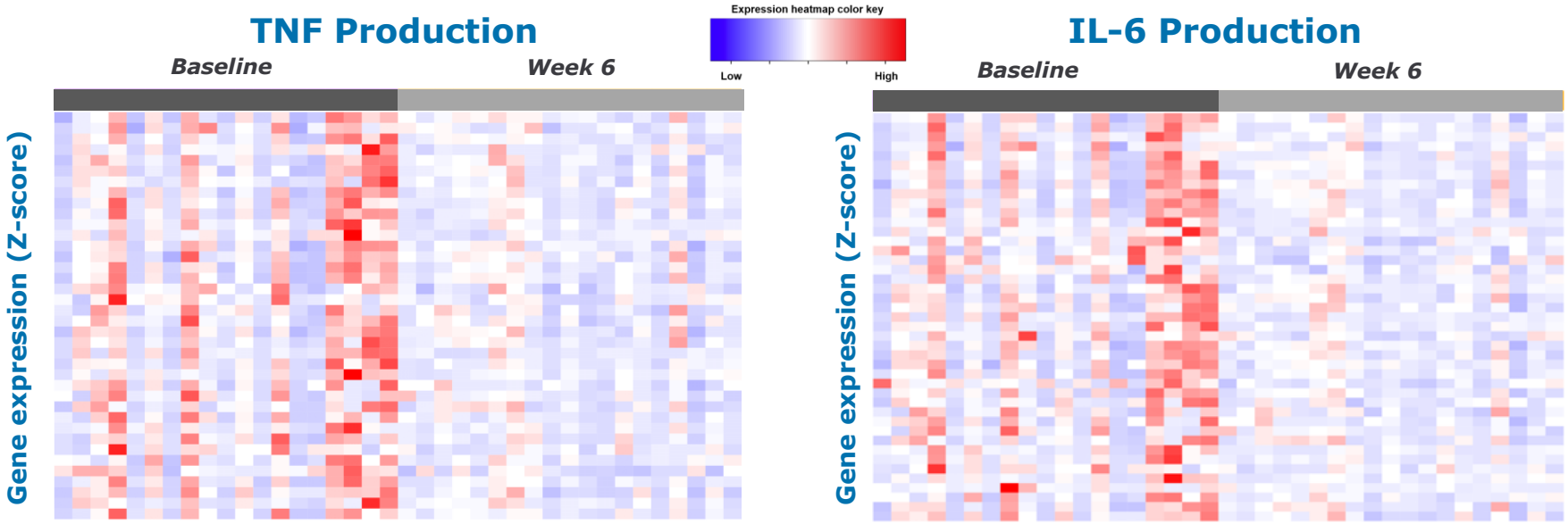
Note: Synovial biopsies of the most impacted joint taken at baseline and 6 weeks on study. Immunofluorescence performed to identify PD-1 positive cells. T_{ph} cells (PD-1+CD3+CD4+CXCR5-)

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 **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

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 is a best-in-class pathogenic T cell depleter

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

Competitive Landscape

		First Tracks Biotherapeutics Rosnilimab (IgG1k)	Lilly Peresolimab (IgG1k)	JNJ JNJ-4703 (IgG1k)	Gilead GS-0151 (IgG1 mut. FC[6])
Structural characteristics	Membrane-proximal epitope	✓	✗	✓ Limited Binding Footprint	✓
	Fc receptor binding affinity	✓	✓	✓	✓[6]
Clinical/translational outputs[1]	Peripheral (Blood) Depletion	>90%[2]	~57%[3]	~60%[5]	0%[6]
	Tissue (RA Synovium) Depletion	~90%[2]	N/A[4]	~40%[5]	0%[6]

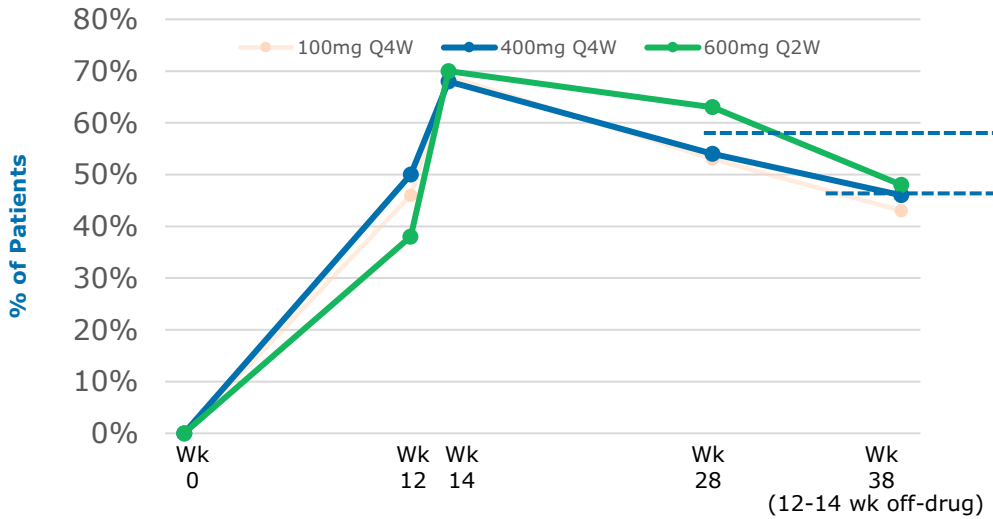
Recent Lilly patents note peresolimab's "modest" activity and disclosed more potent candidates closer to rosnilimab's profile[7]

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

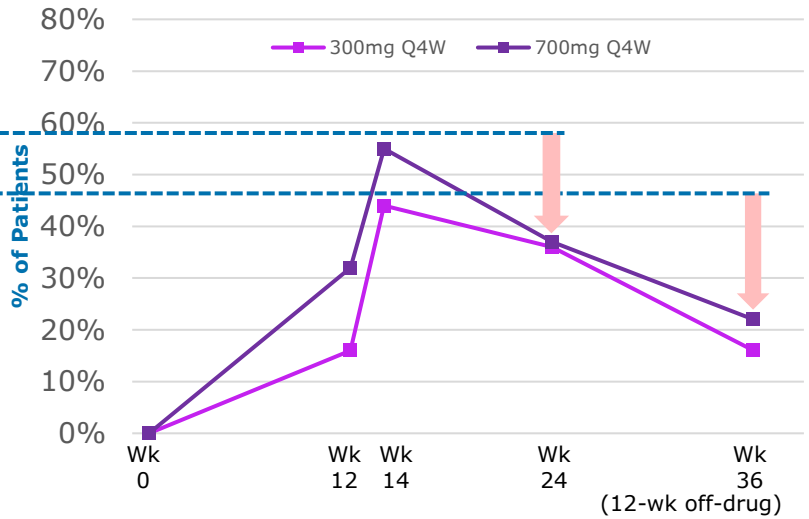
Rosnilimab P2b: CDAI LDA

NRI analysis on ITT population (n=318 rosnilimab patients)[1,2]



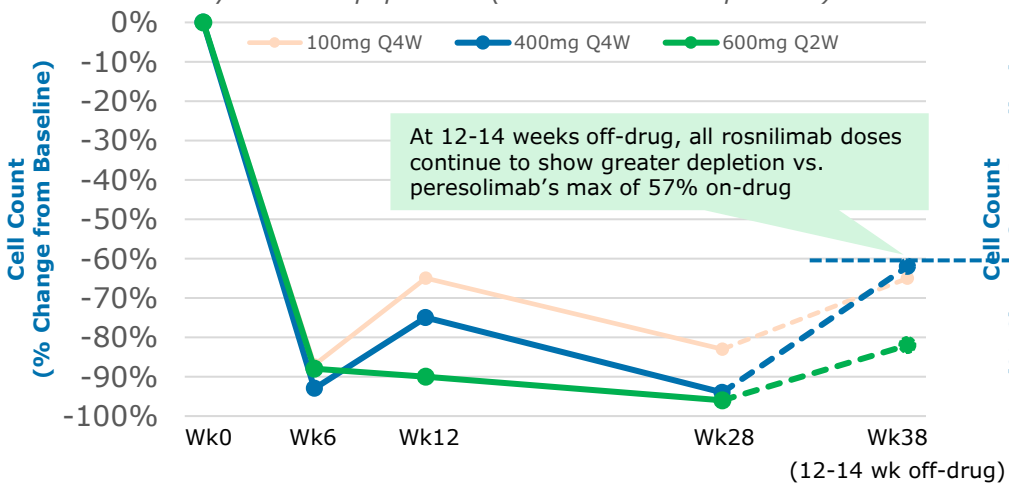
Peresolimab P2a: CDAI LDA

NRI analysis on ITT population (n=74)[3]



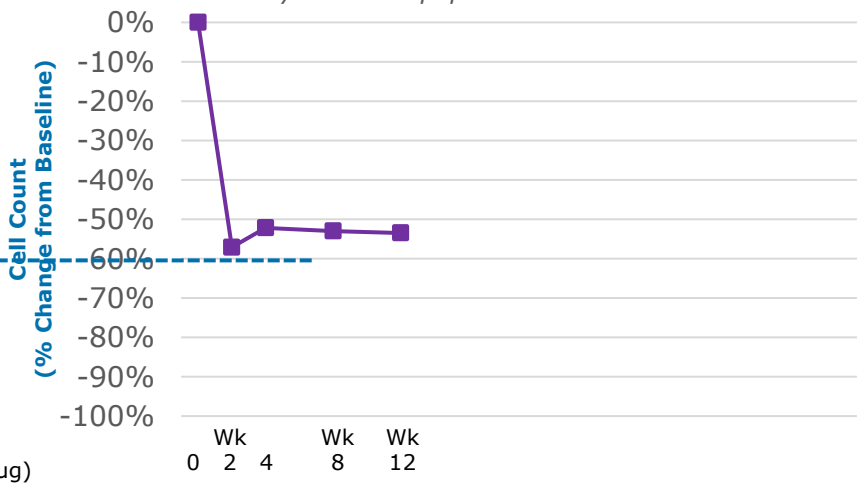
Rosnilimab P2b: PD-1^{high} T Cell Impact

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



Peresolimab P2a: PD-1^{high} T Cell Impact

NRI analysis on ITT population – Pooled doses[4]



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
Infection Rate[1]

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

2x
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

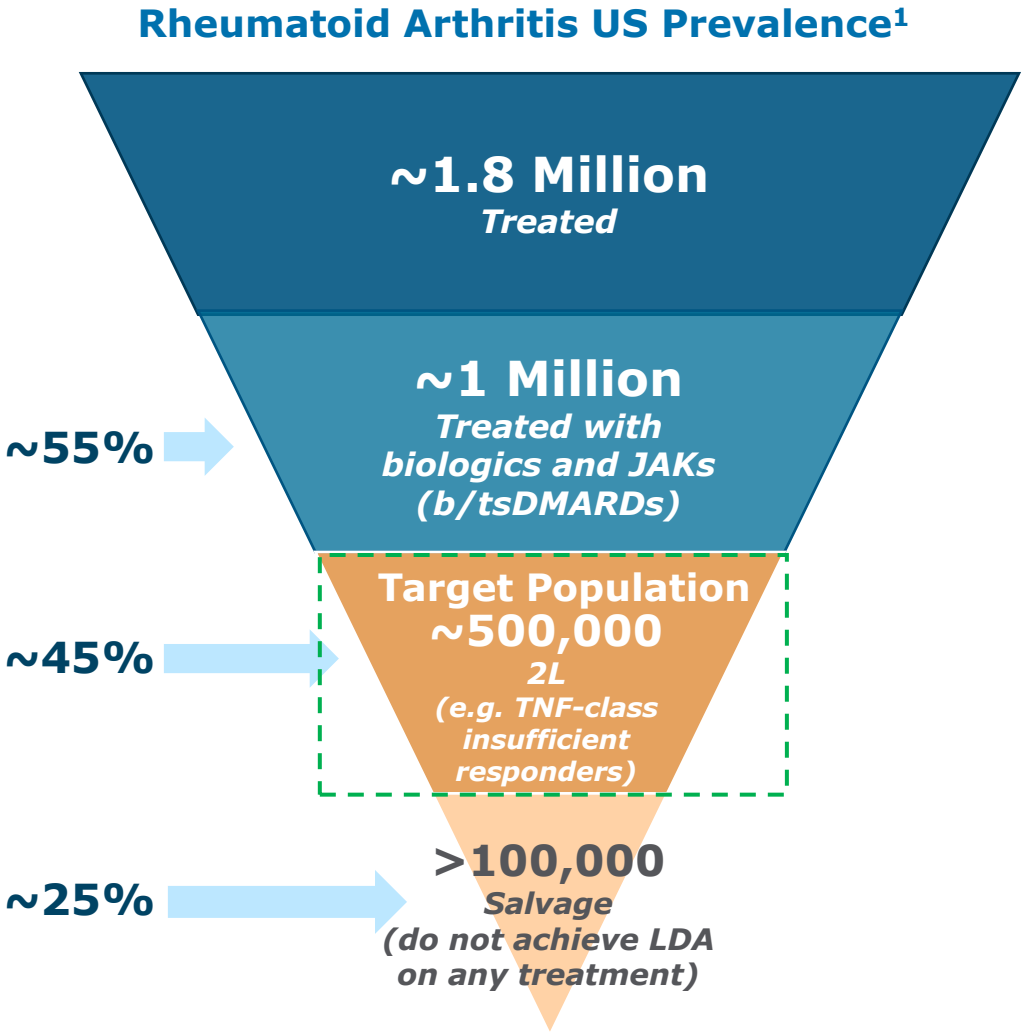
<2% dropout rate overall due to AEs through 6 months,
with only 1 dropout due to AE (headache-moderate) after 3 months

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 Q2 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

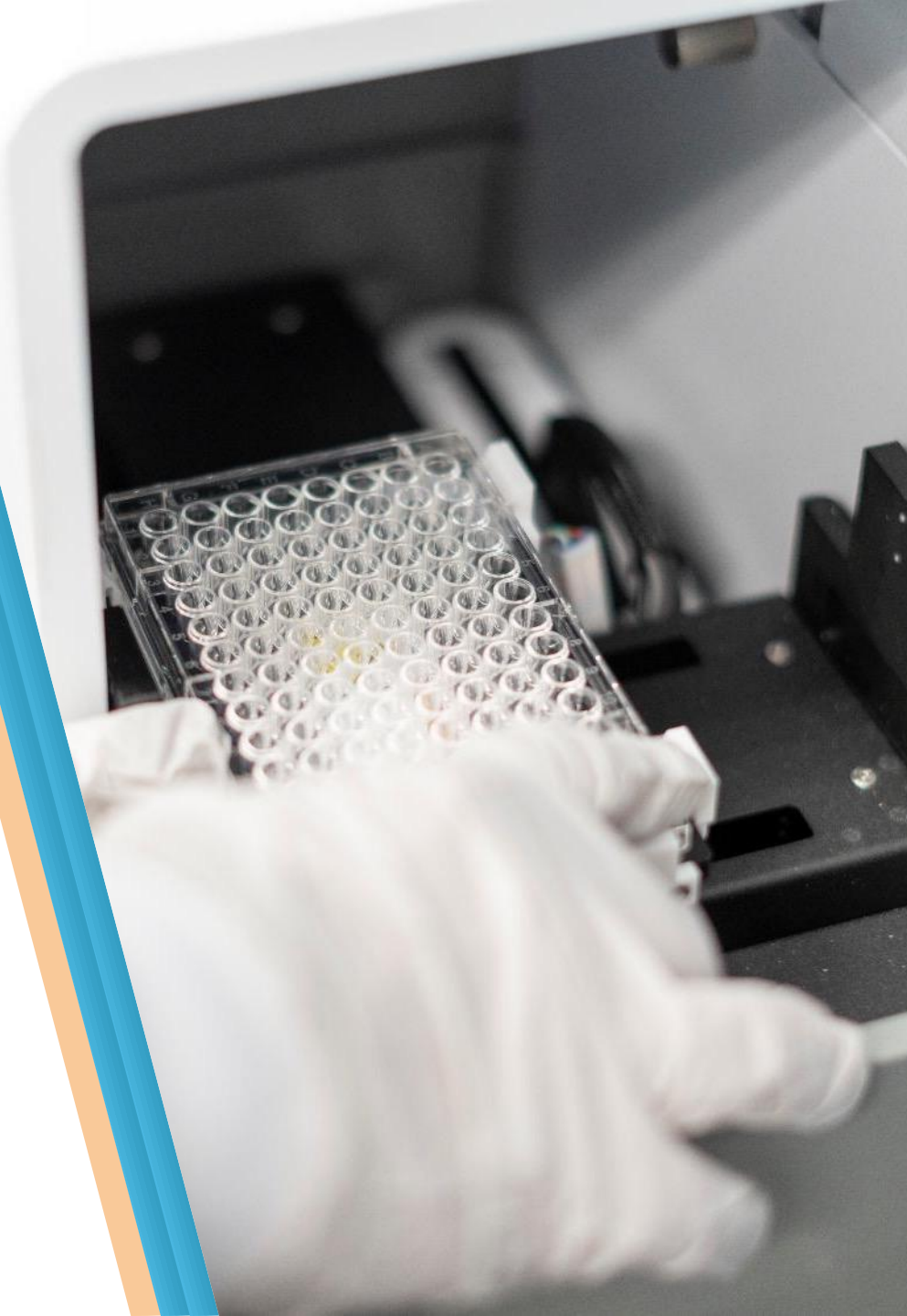
Rheumatoid Arthritis	Ulcerative Colitis
Positive Phase 2b data reported	**Top-line Phase 2 data reported**

• 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
• Late-breaking data presented at ACR 2025
	• Safe and well tolerated with similar adverse event rates vs. placebo
	  ○ Safety profile through Week 50 remains consistent with Week 12
	• Observed expected pharmacology, including ~90% depletion of pathogenic T cells
	• Lack of efficacy at Week 12 do not support further development of rosnilimab in UC
	  ○ Trial has been discontinued

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

ANB101

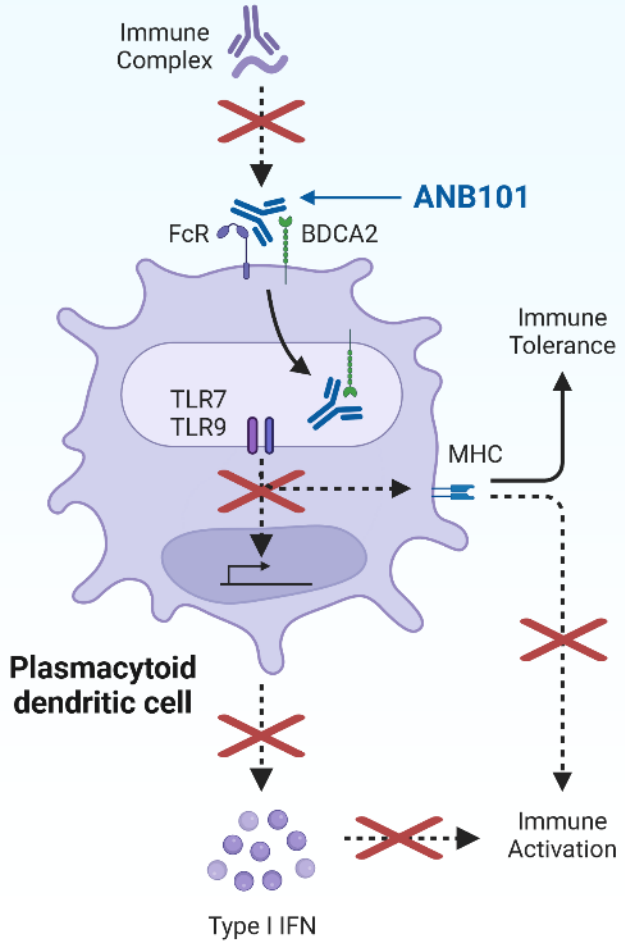
(BDCA2 modulator)



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