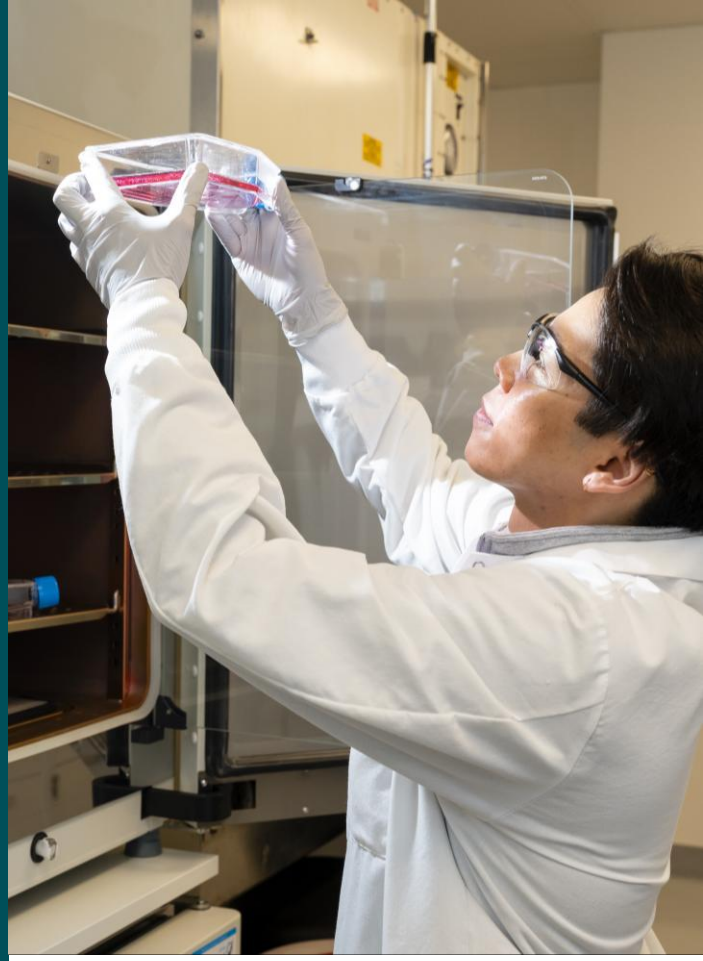
Corporate Presentation

CARVING A NEW TRAIL IN IMMUNOLOGY

May 2026

NASDAQ: TRAX



First Tracks
BIOTHERAPEUTICS

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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; whether any partnership with rosnilimab will take place; the potential to receive any milestone payments from the Vanda Pharmaceuticals license agreement; whether any of the Company's product candidates will be best in disease or best in class; 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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Developing differentiated antibody therapeutics in autoimmune and inflammatory diseases

Focused strategy to efficiently advance high-value immunology portfolio

- Data-driven development approach prioritizing differentiation, speed and capital efficiency
- Global clinical development strategy
- Deep immunology and translational R&D expertise

Lead program, ANB033, has pipeline-in-a-product potential across GI, derm and other broad autoimmune disease areas

ANB033
(CD122 antagonist)

- Phase 1b in CeD
- Phase 1b in EoE

Rosnilimab
(Pathogenic T Cell Depleter)

- Phase 2b completed in RA

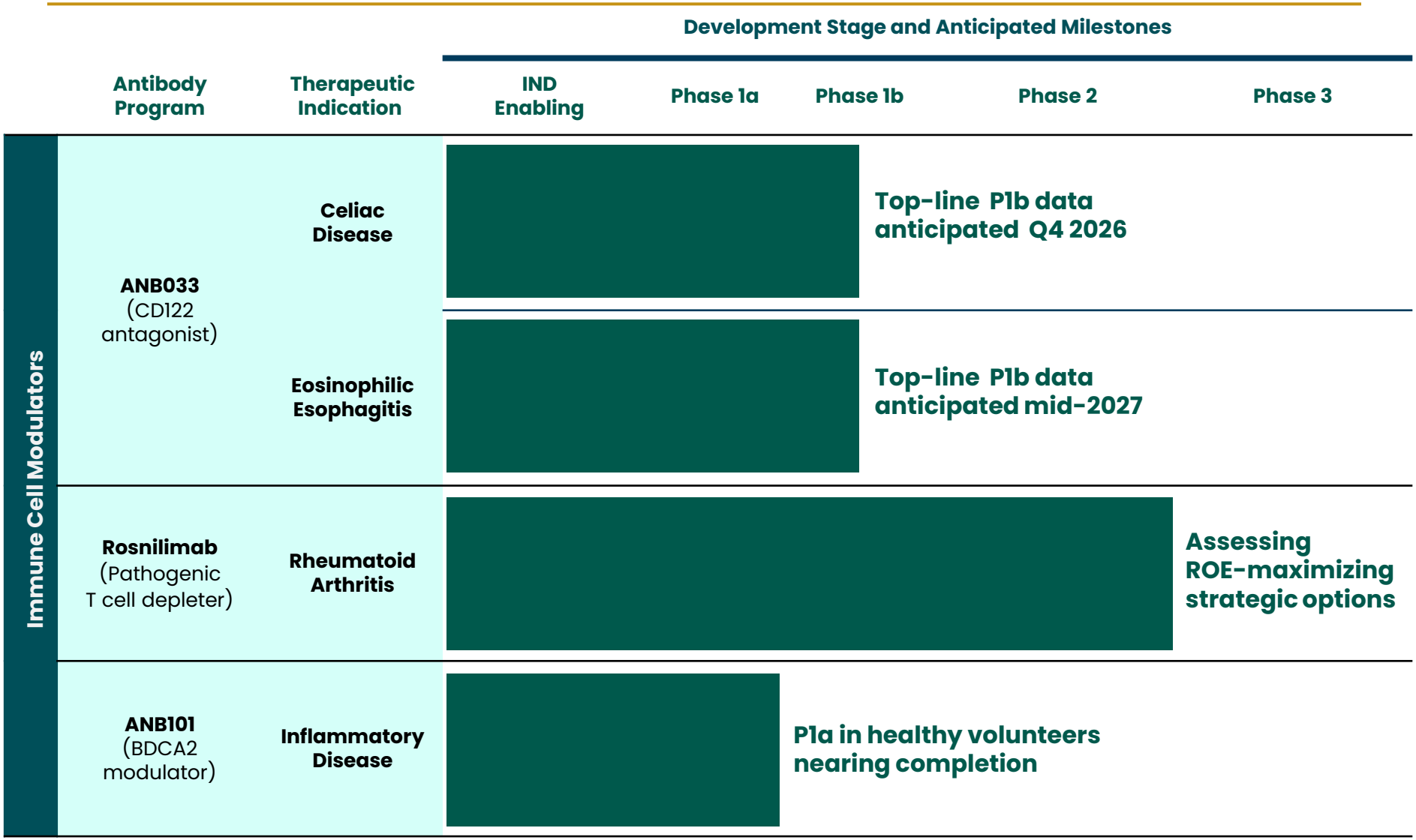
ANB101
(BDCA2 modulator)

- Phase 1a in healthy volunteers

Launched in April 2026 with $180 million in cash and a two-year cash-runway

CeD: celiac disease; EoE: eosinophilic esophagitis; RA: rheumatoid arthritis

Leading pipeline designed to deliver meaningful advances for patients with autoimmune diseases



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

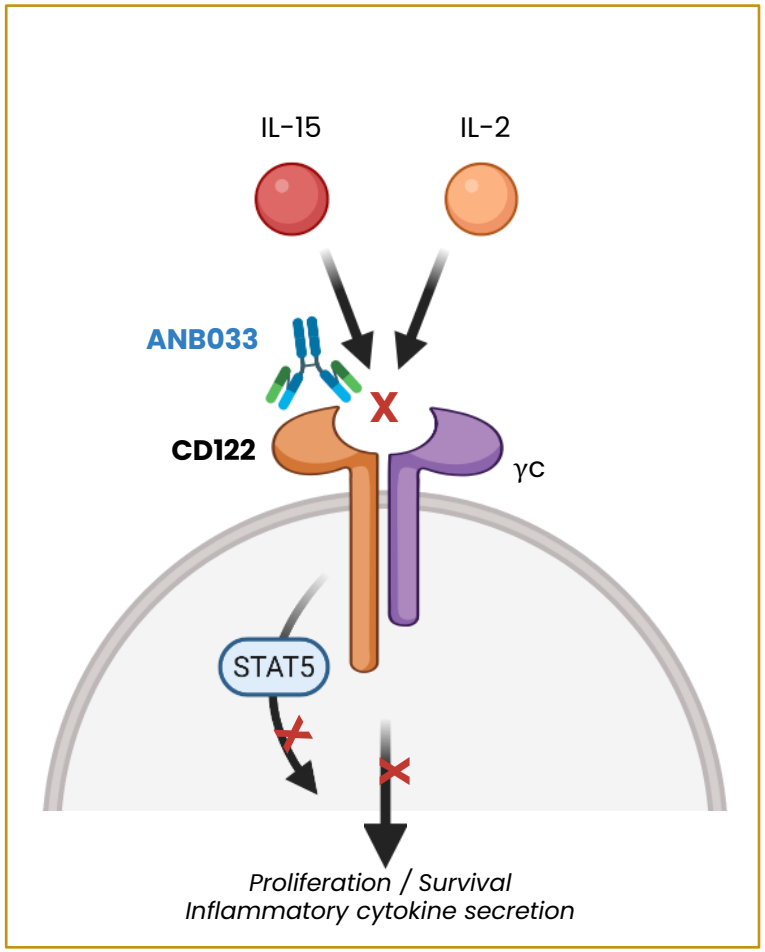
- Expressed on subsets of CD8+ and CD4+ T cells and NK cells

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.

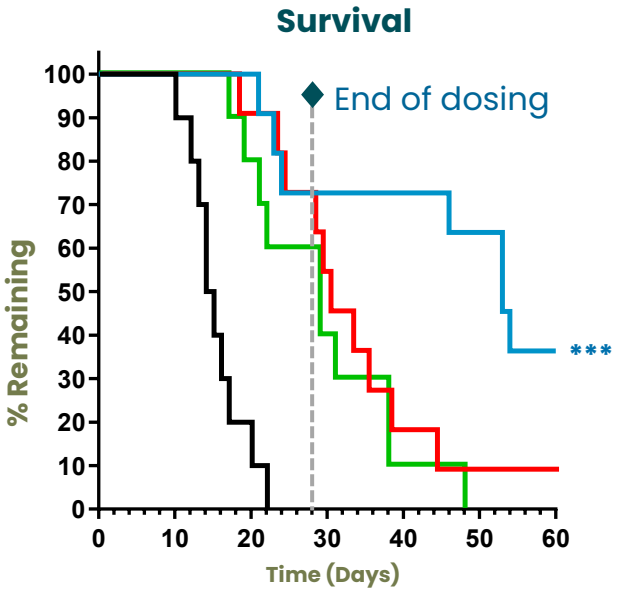
Broad therapeutic potential across autoimmune and inflammatory diseases

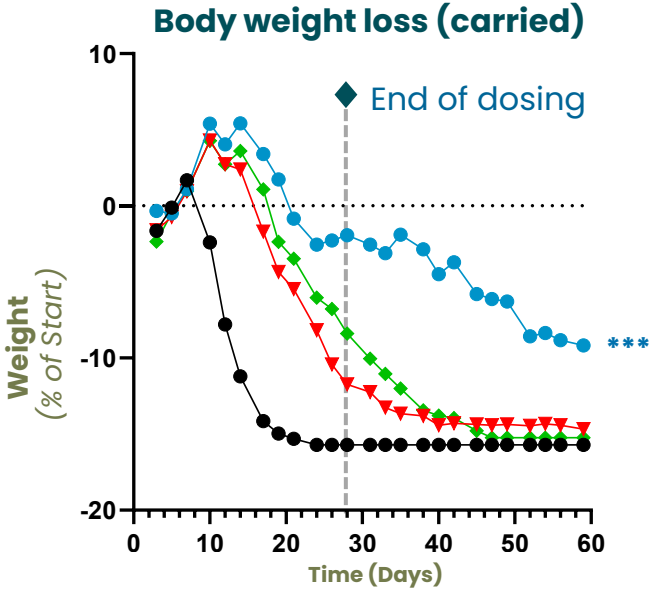
Gastroenterology	Dermatology	Other Areas
Celiac Disease (CeD)	Atopic Dermatitis	Asthma/COPD
Eosinophilic Esophagitis (EoE)	Alopecia Areata	Multiple Sclerosis
Crohn's Disease	Hidradenitis Suppurativa	Psoriatic Arthritis
Ulcerative Colitis	Lichen Planus	Type 1 Diabetes
	Vitiligo	Solid Organ Transplant

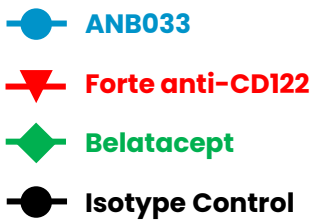
Other clinical-stage drugs targeting IL-15 or CD122

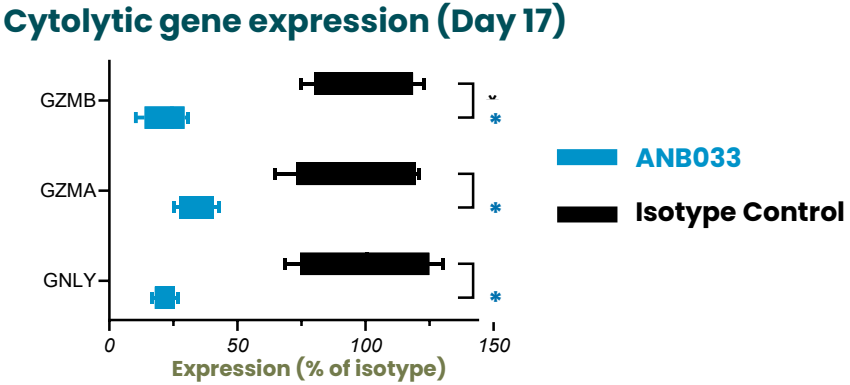
NOVARTIS	IL-15	• P1b PoC in CeD and EoE • P2a in atopic dermatitis (ongoing) • P2 in vitiligo (initiating) • *Initiating a trial in at least one other indication*
teva	IL-15	• P2a in CeD (ongoing – data Q3 2026) • P1b in vitiligo (ongoing – data Q2 2026) • Assessing atopic dermatitis, alopecia areata and EoE
FORTE	CD122	• Positive P1b in CeD (P2a ongoing – data in H2 2026) • P1b in vitiligo (ongoing – data in Q2 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









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.

First Tracks BIOTHERAPEUTICS

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

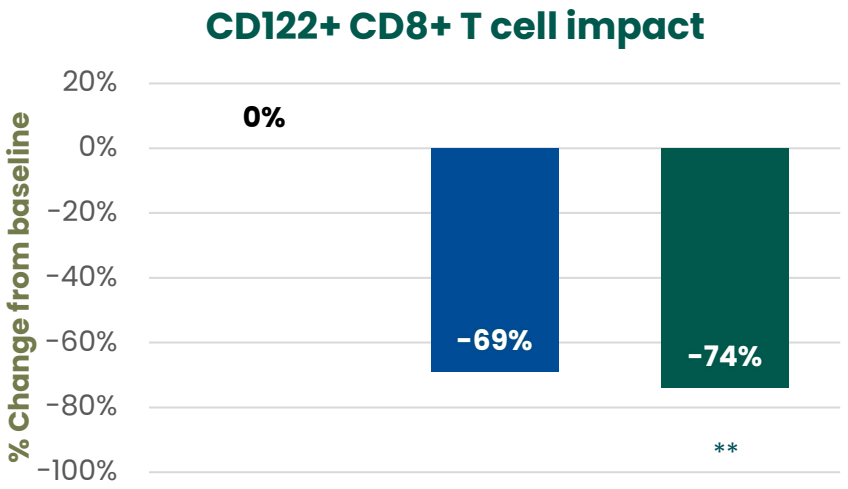
Rapid and sustained PK profile

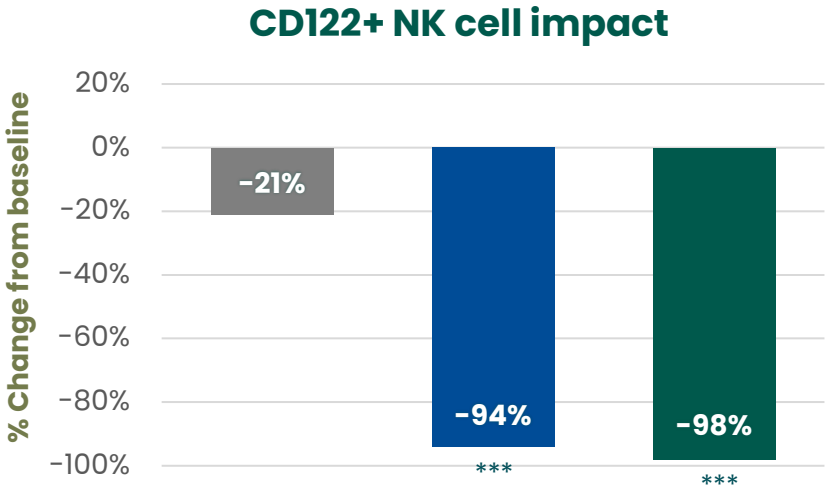
- Favorable 2 to 3-week half-life with IV and SQ dosing
- Full receptor occupancy (RO) within hours and maintained for >30 days
- Dose response observed
- Modeled to achieve >IC90 on CD8+ T cell subsets in GI tissue
- Overall, no impact on peripheral total Treg counts

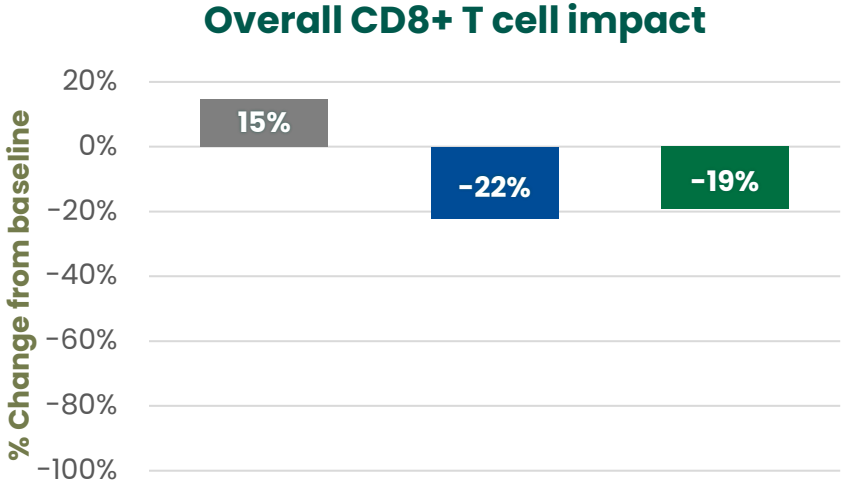
SAE = Serious adverse event.

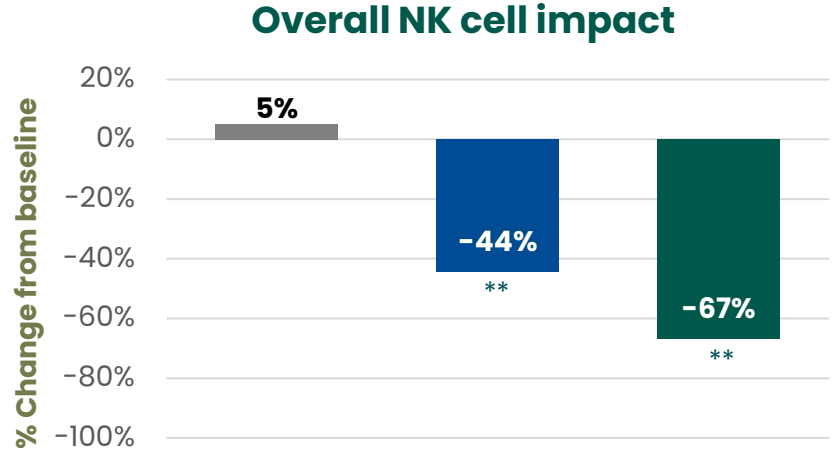
ANB033 significantly reduces CeD relevant CD8+ T cells and NK cells after single dose

Effect of ANB033 is limited to CD122 expressing cells







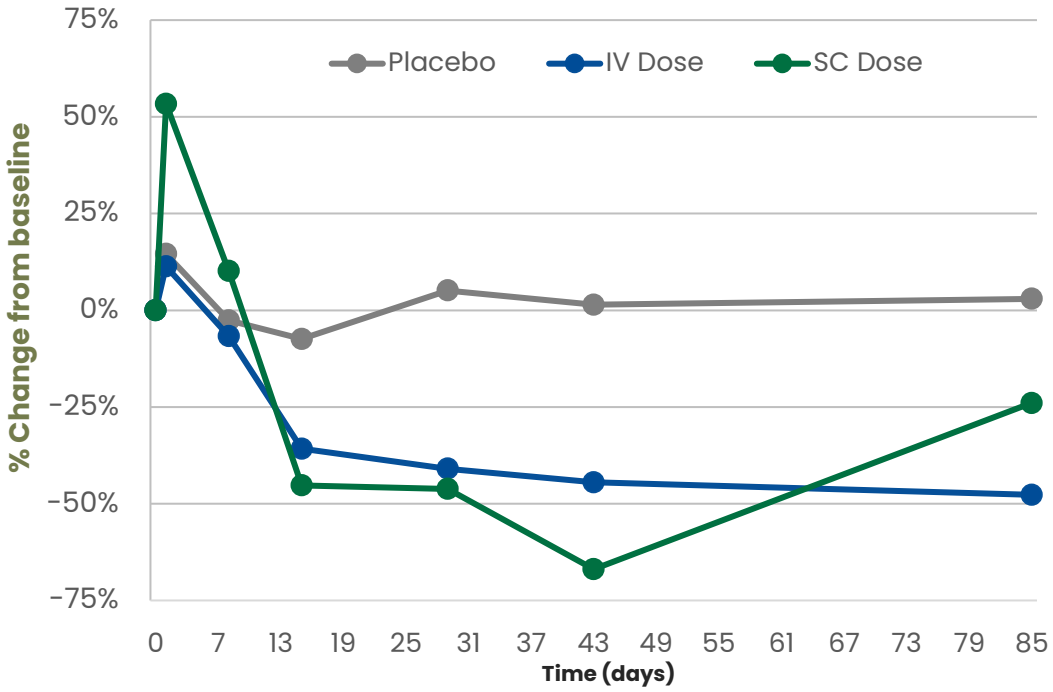


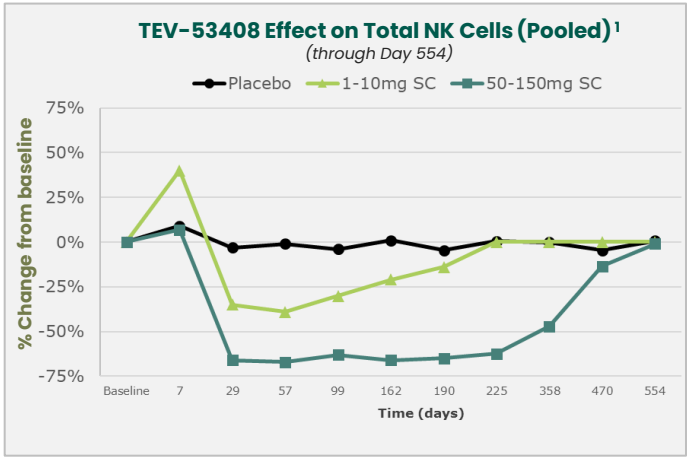
Placebo ANB033 – IV Dose ANB033 – SC Dose

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

ANB033 effect on total NK cells





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

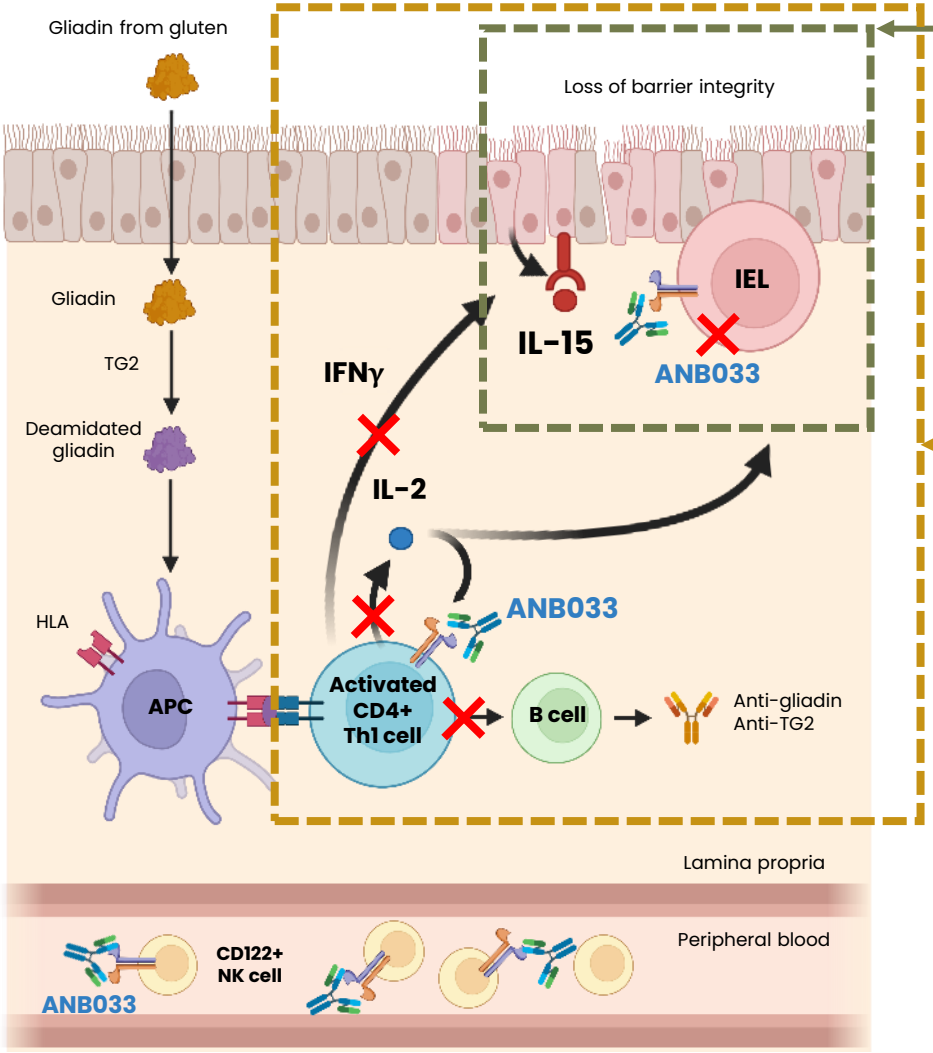
- ✓ ANB033 >50% peak total NK cell reduction with return towards baseline within 3 months
- ✓ TEV-53408: >50% sustained total NK cell reduction for 1 year with return to baseline over 18 months

First Tracks BIOTHERAPEUTICS

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 P2a CeD trial in 48 adults while undergoing GC; 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

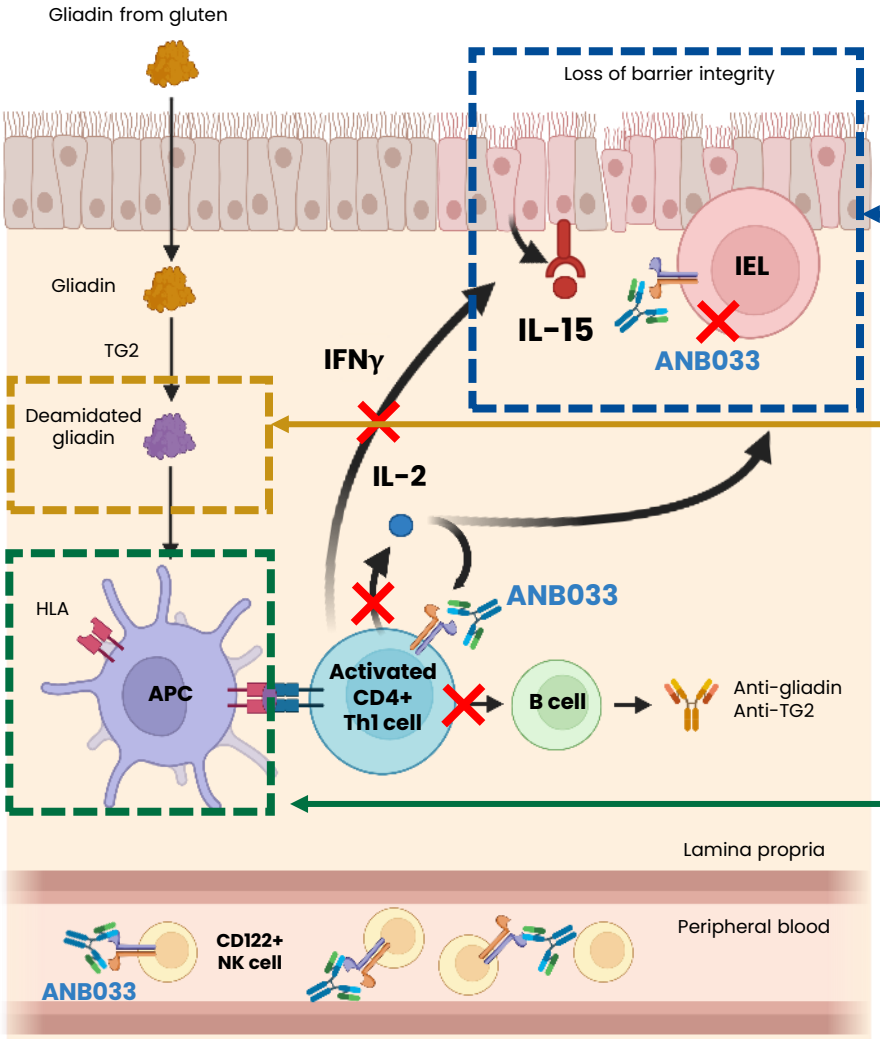
Inhibition of IL-2 signaling

- 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

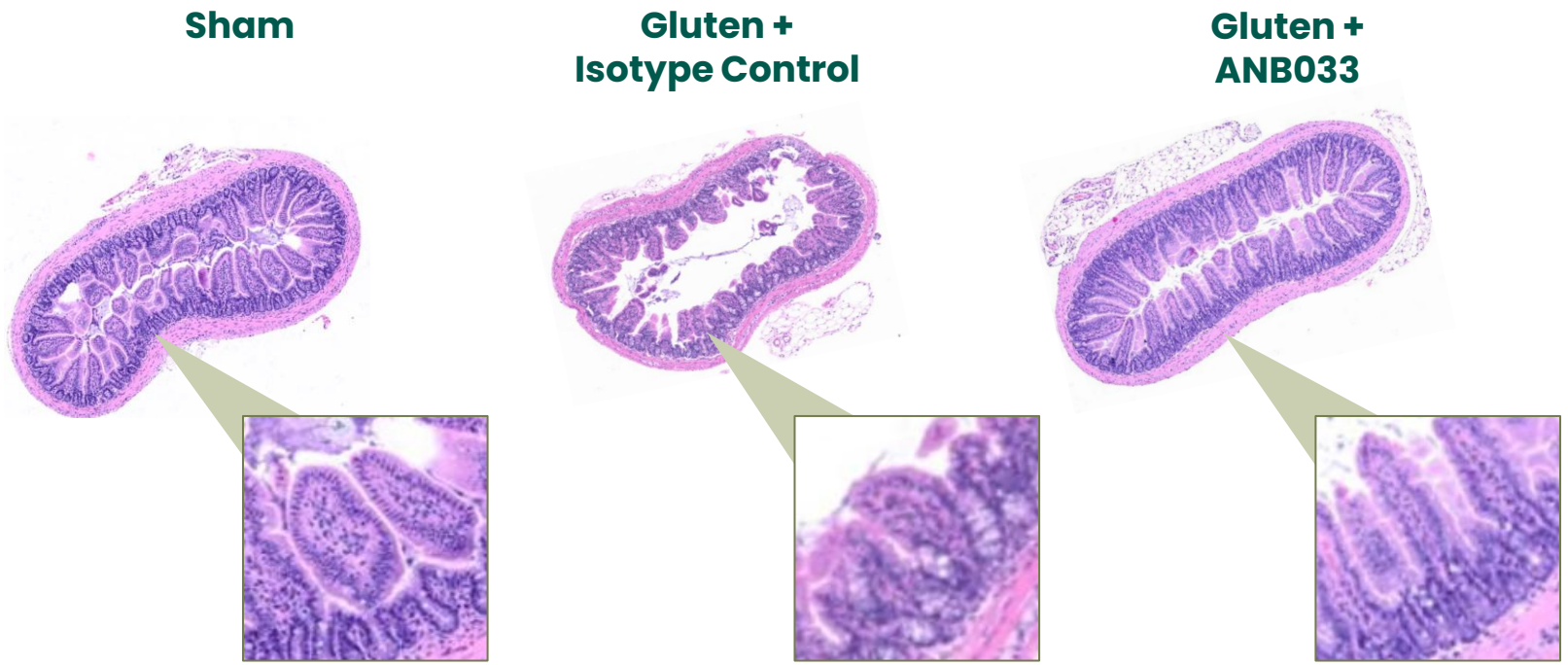
- Takeda — *P2 ongoing* — *Gluten tolerance*
- astellas — *Discontinued* — *Gluten tolerance*
- Roche — *P1 ongoing* — *HLA-DQ2.5 gluten peptide complex*
- Immunic THERAPEUTICS — *P1 ongoing* — *SIRT6 modulator*

OX-40L antagonist

- sanofi — *P2 deprioritized*

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

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



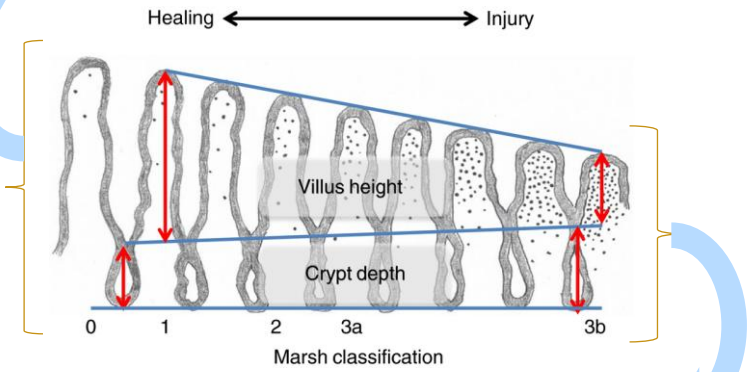
ANB033 treatment shows improved histology:
preserves villus height and crypt depth (Vh:Cd) in CeD mouse model

Note: HuDQ8-Dd-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

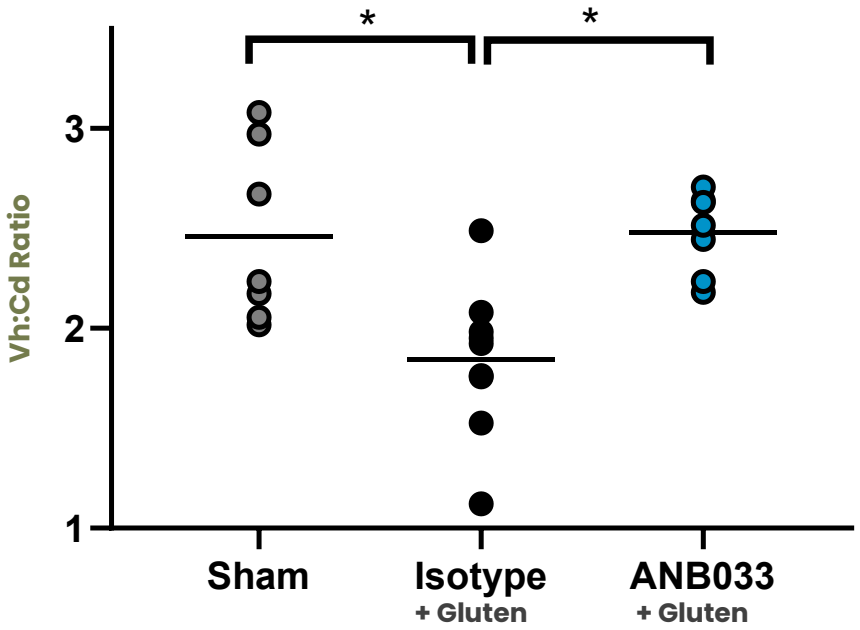
Vh:Cd ratio

Healthy tissue:
High villus height, lower crypt depth



CeD tissue:
Reduced villus height, extended crypt depth

ANB033 impact on Vh:Cd ratio



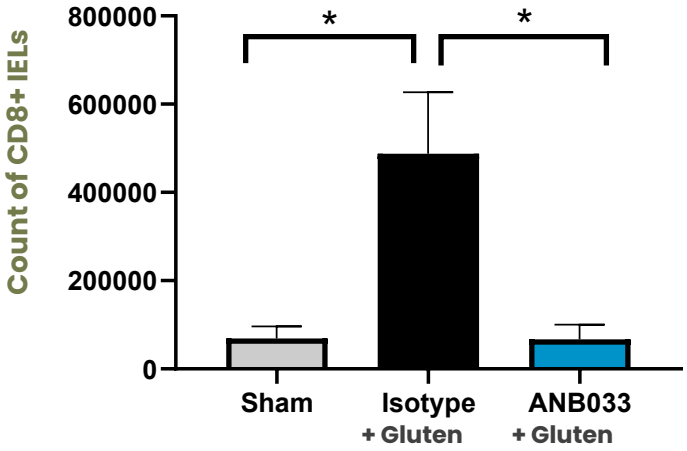
ANB033 treatment shows improved histology: preserves villus height and crypt depth (Vh:Cd) in CeD mouse model

Note: HuDQ8-Dd-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.

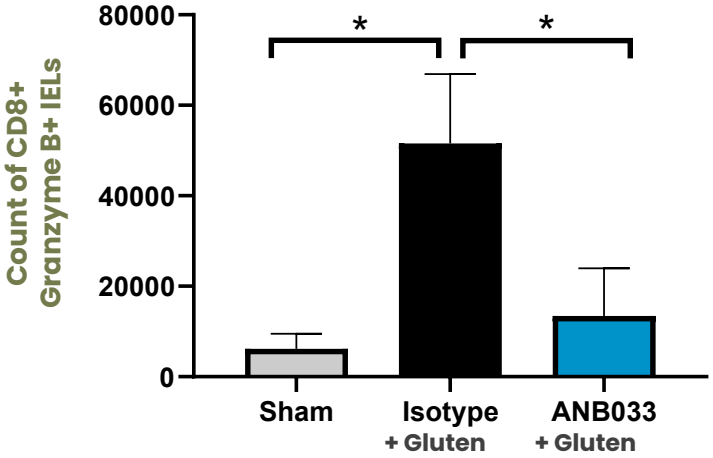
ANB033 prevents gluten-induced intestinal inflammation

Epithelial layer of small intestine

No increase in CD8+ IELs

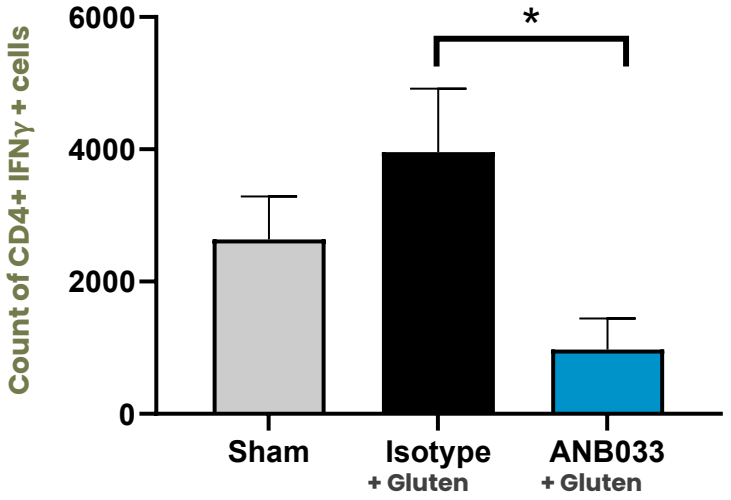


No increase in Granzyme B+ IELs



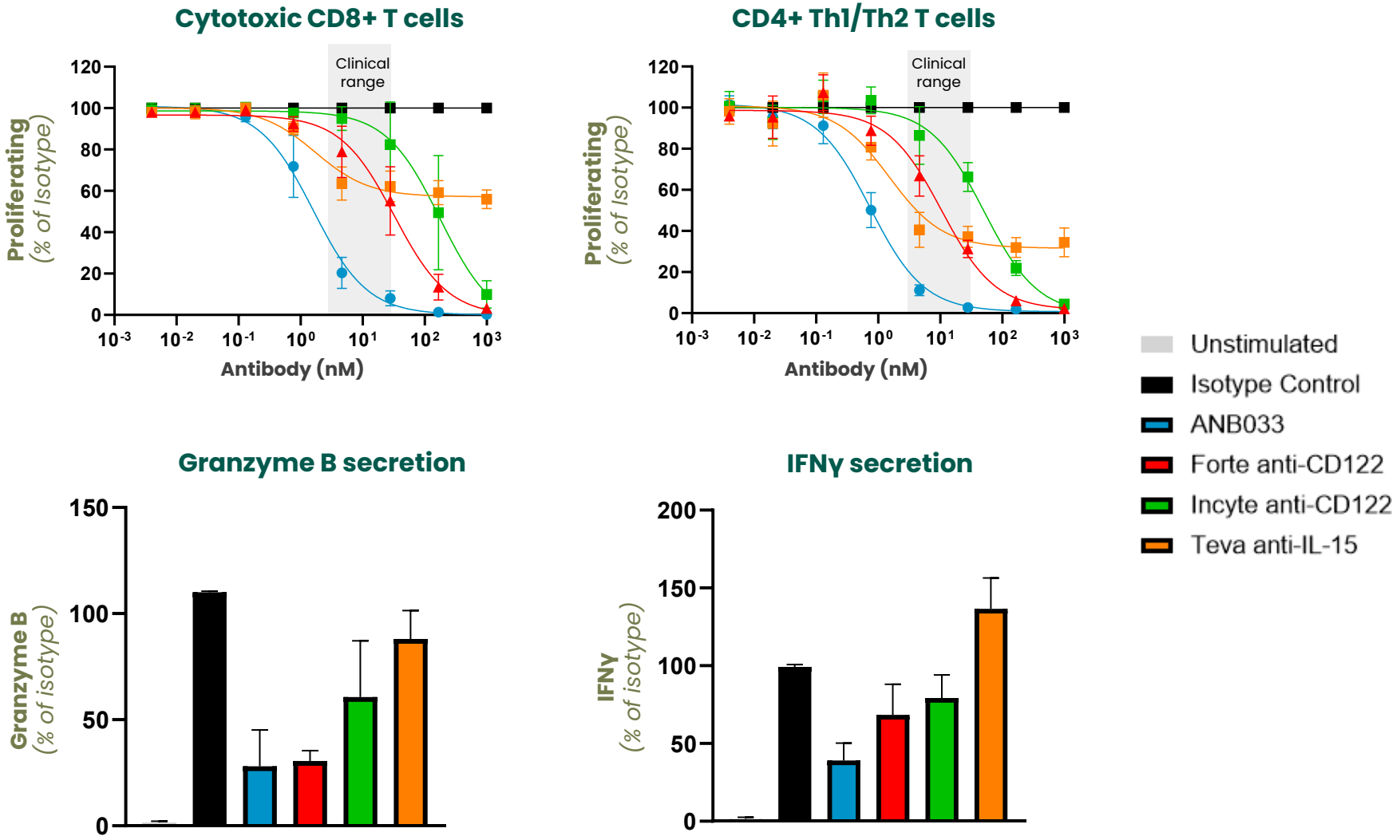
Lamina propria (LP)

No increase in CD4+ IFNγ + T cells



Note: HuDQ8-Dd-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

First Tracks BIOTHERAPEUTICS *(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

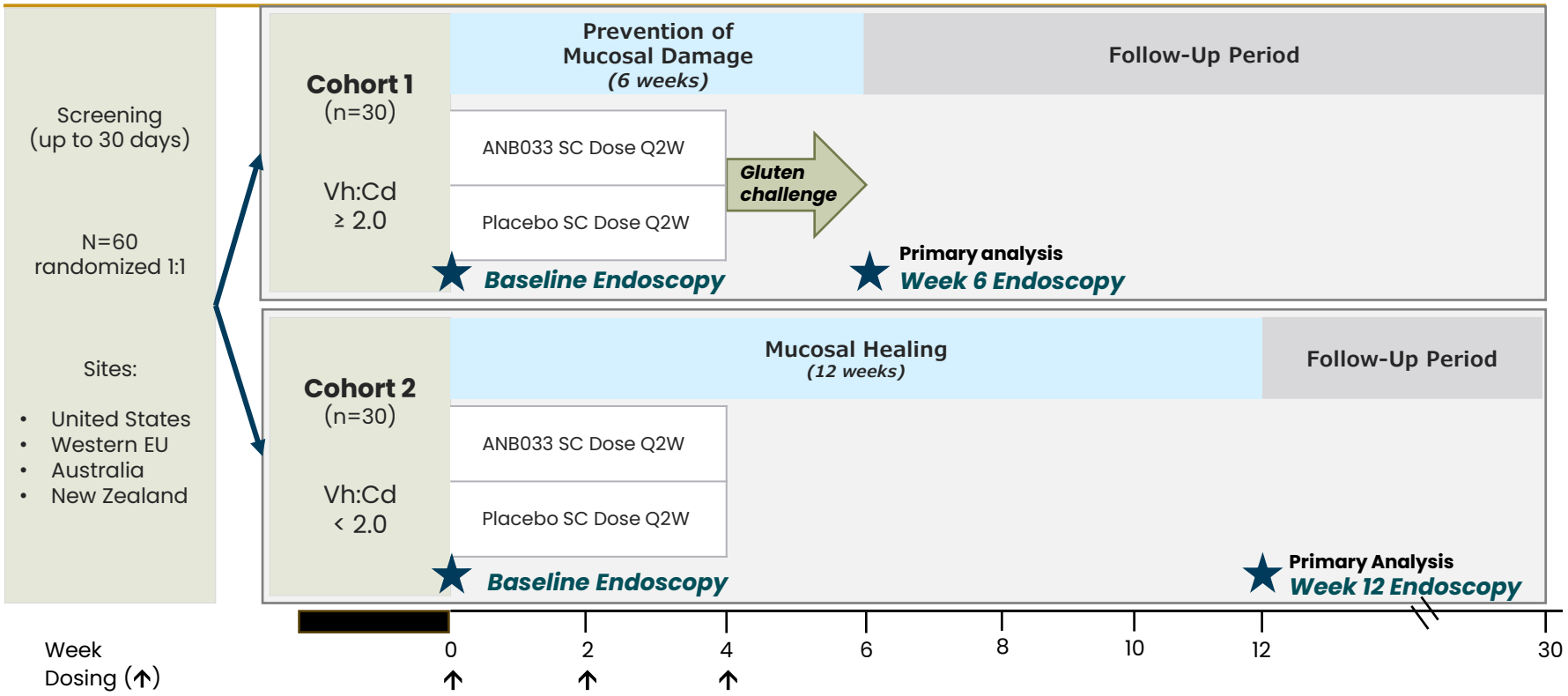
sanofi *(Phase 2b, deprioritized)*

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

GF diet = Gluten free diet.

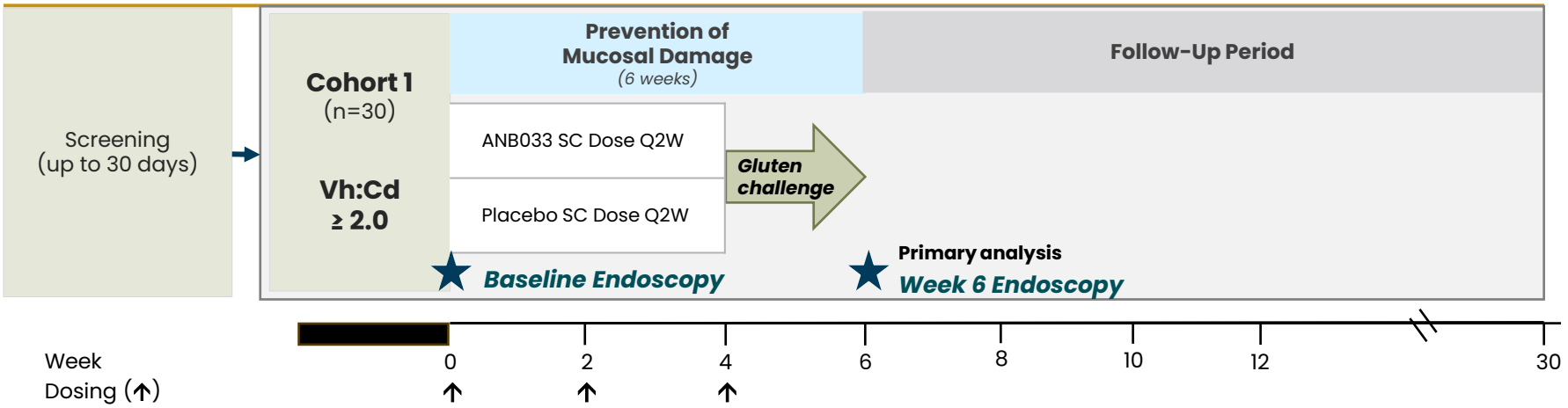
ANB033 Phase 1b trial in CeD ongoing

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



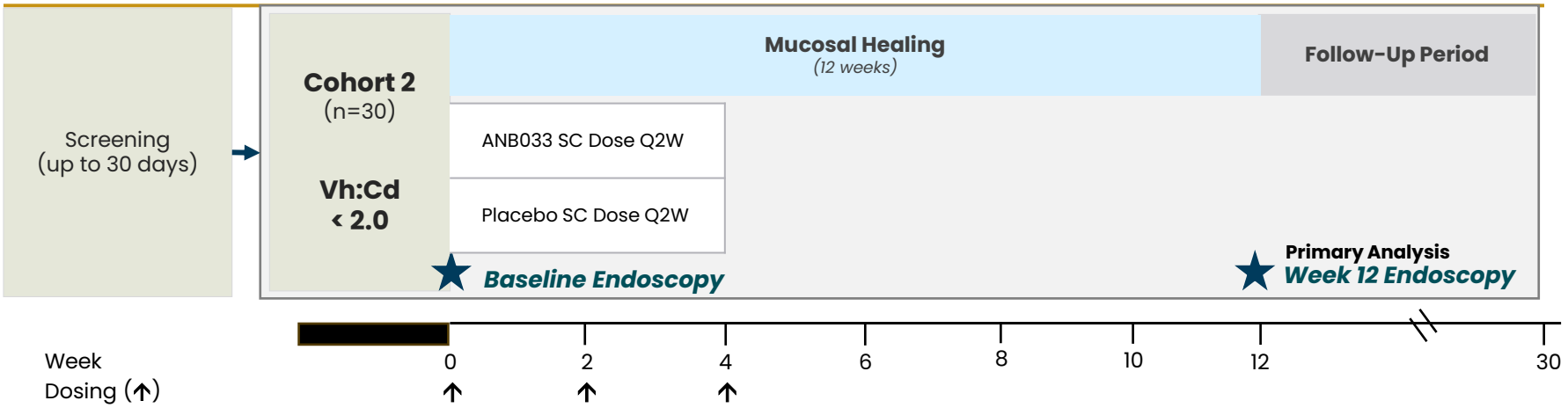
Minimal evidence of mucosal damage (Vh:Cd ≥ 2.0)

- 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



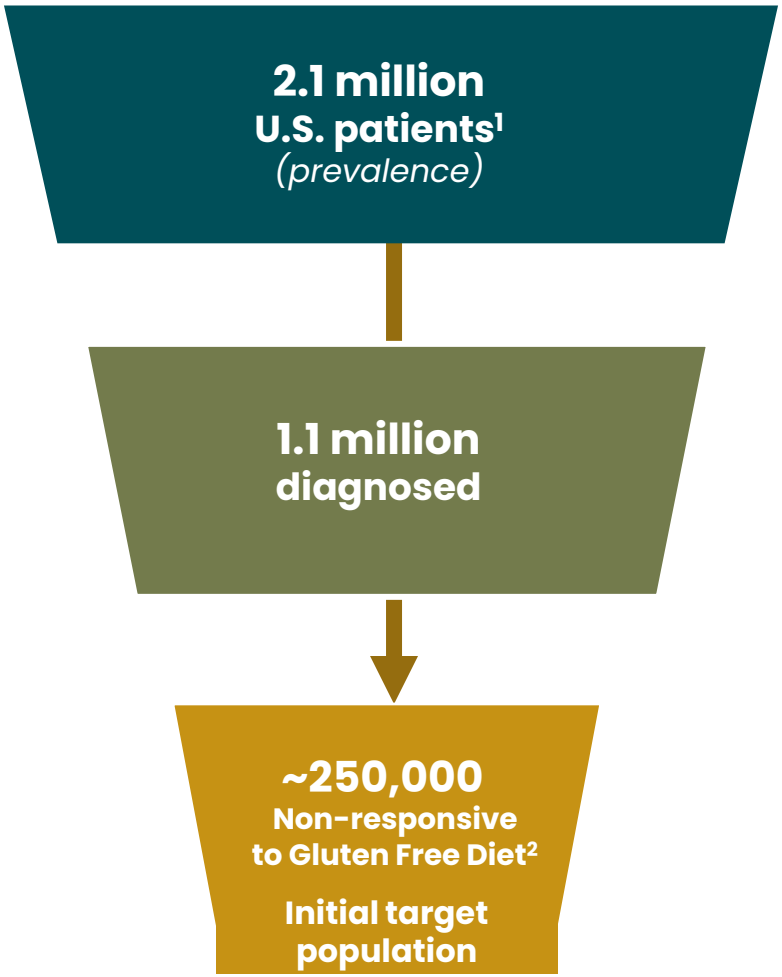
Persistent evidence of histologic CeD activity (Vh:Cd < 2.0)

- 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

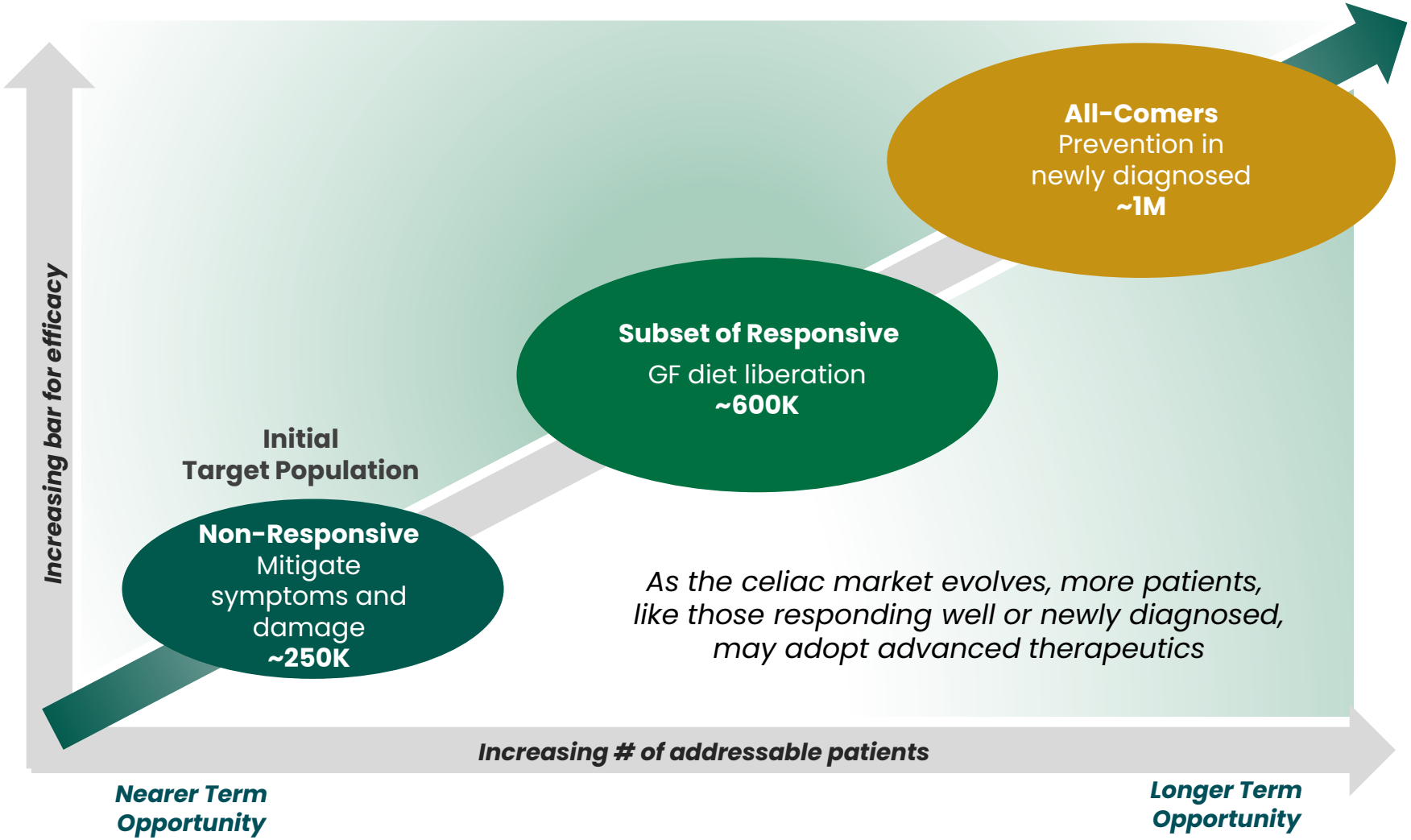
- HCPs favor MOA that targets both symptoms and histology

$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



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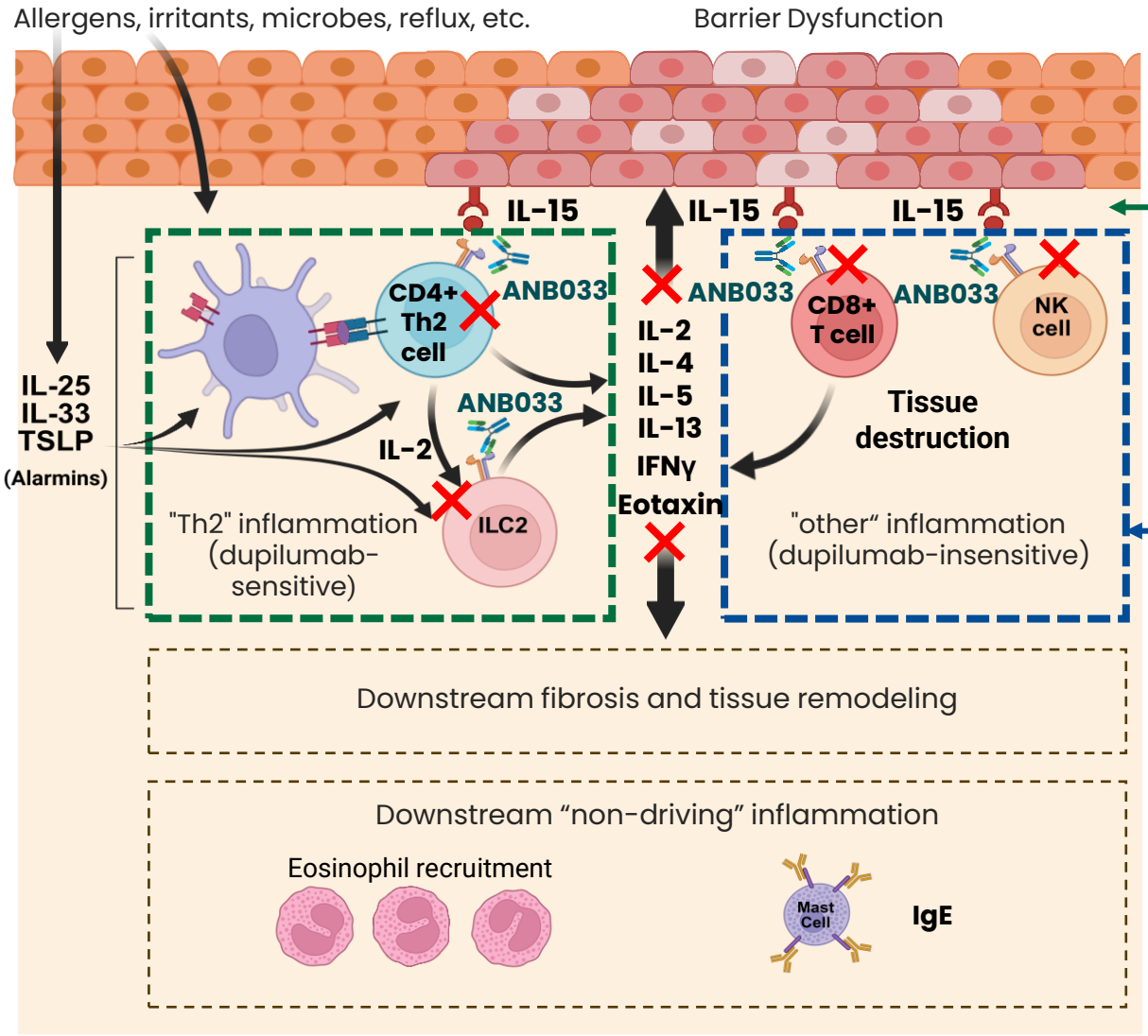
Similar to CeD, ANB033 targets multiple drivers of EoE biology addressing both dupilumab sensitive and insensitive pathways

Phase 1b trial ongoing; 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

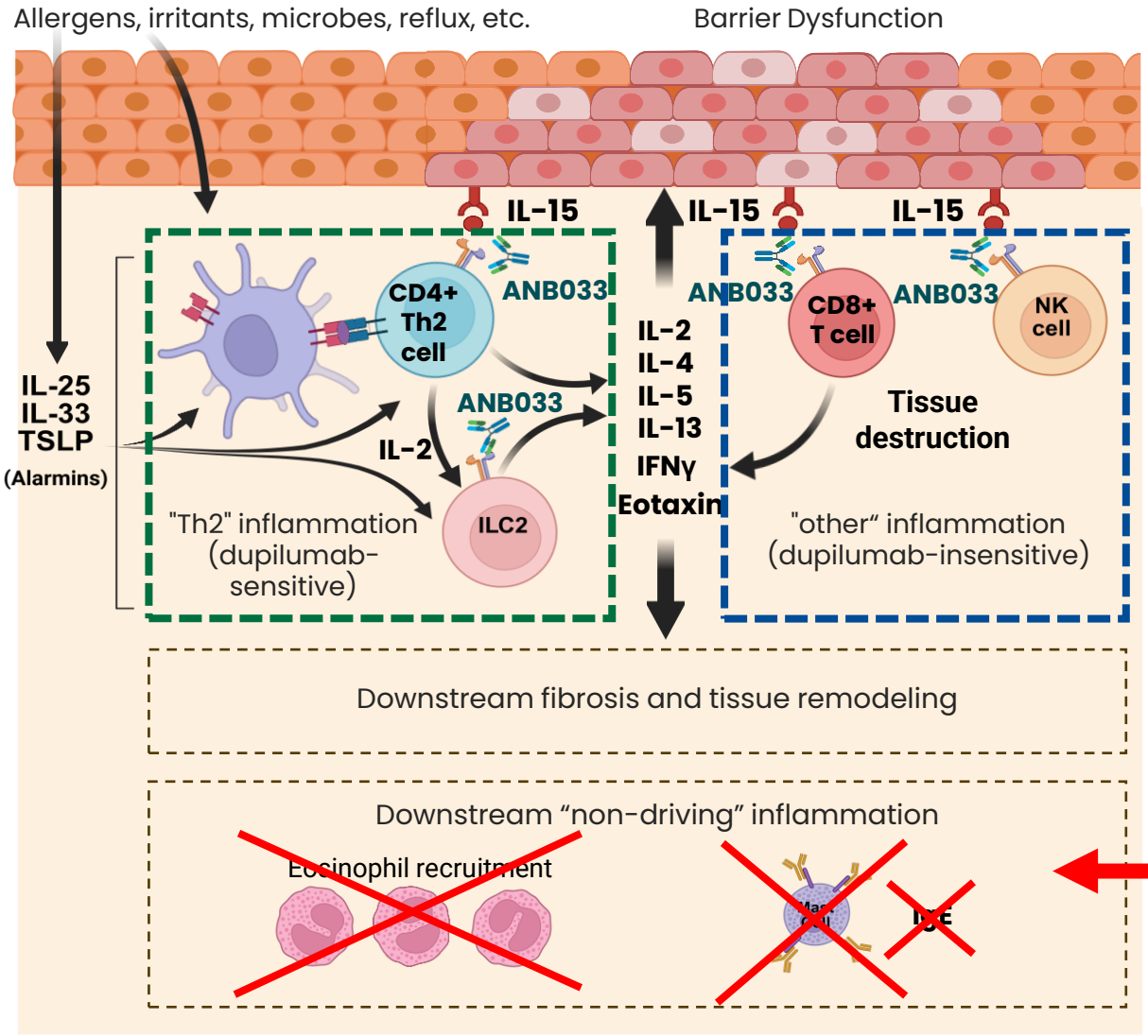
CD8+ and NK (dupilumab-insensitive)

- 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 AstraZeneca

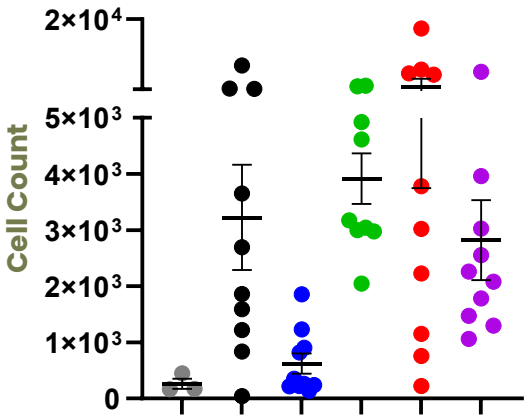
First Tracks BIOTHERAPEUTICS

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

ANB033 prevents eosinophilia by targeting upstream inflammation

Aspergillus-induced eosinophilia

Esophageal eosinophils

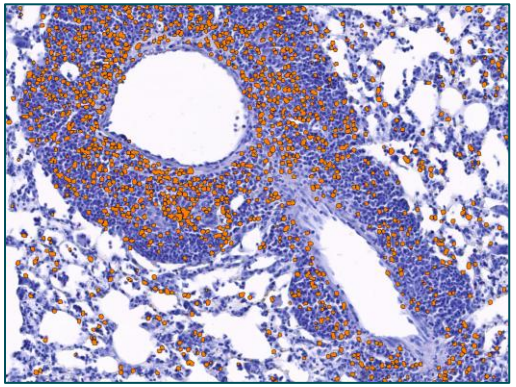


Treatment

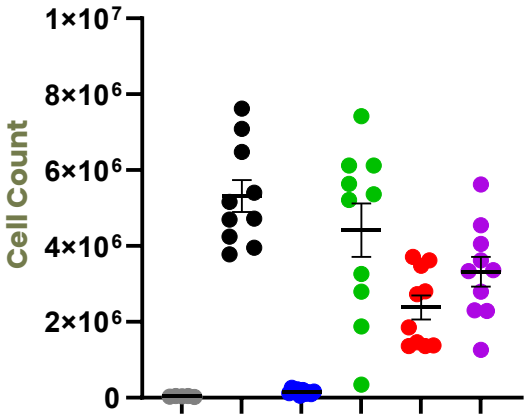
- Unchallenged
- Isotype control
- ANB033
- anti-mIL-15
- anti-mIL-13
- anti-mTSLP

Eos observed in histology

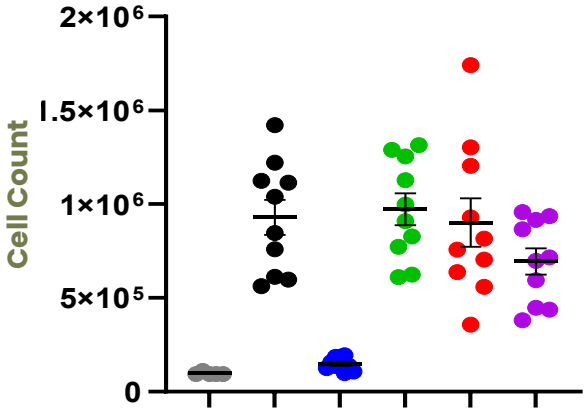
Isotype Control



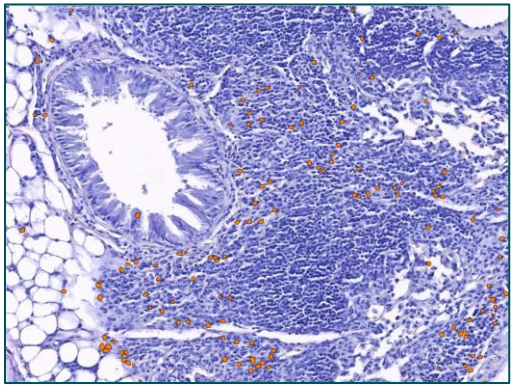
Lung eosinophils



Lung ILC2s



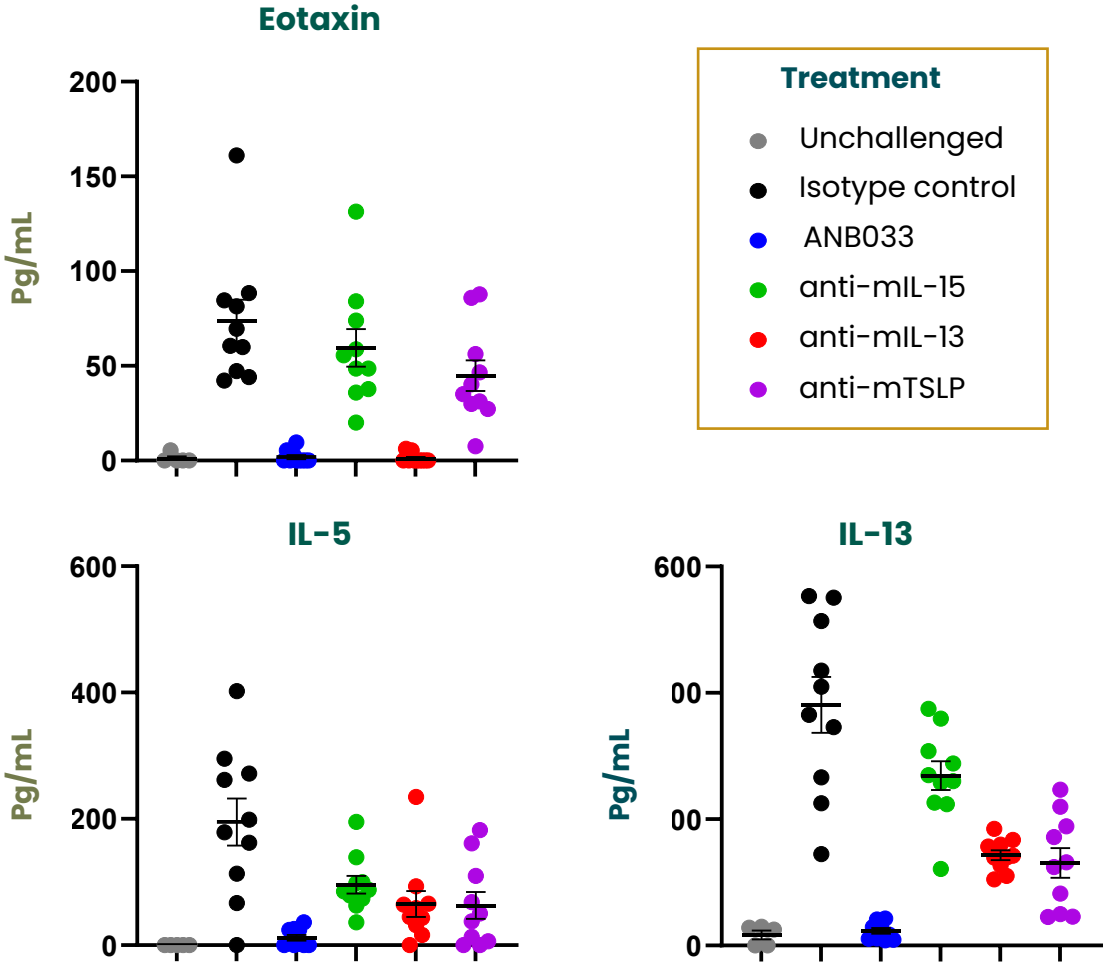
ANB033



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



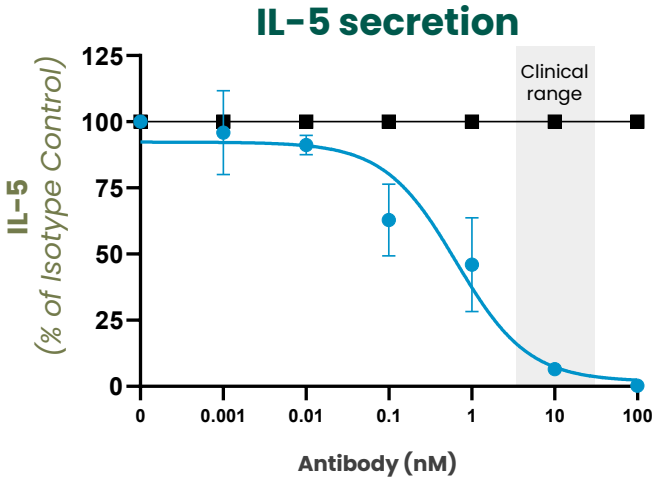
ANB033 prevents –

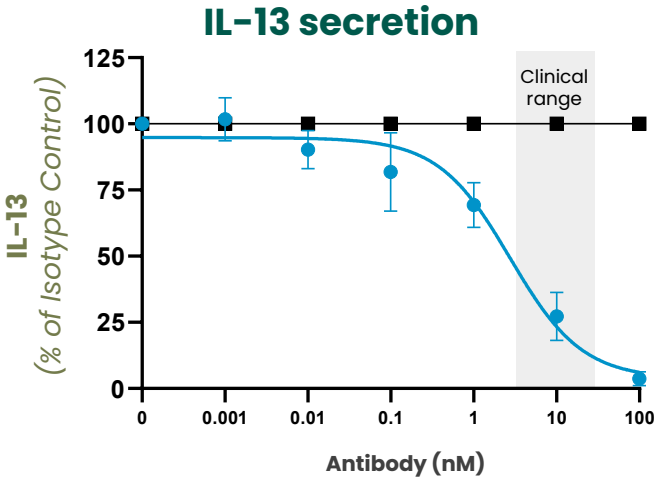
- Cytokine secretion (IL-5/IL-13) responsible for eos expansion
- Chemokine secretion (eotaxin) responsible for eos recruitment

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-mIL13, so IL-13 bound by anti-mIL-13 is still detected via this method. Measured in Bronchial Alveolar Lavage Fluid (BALF).

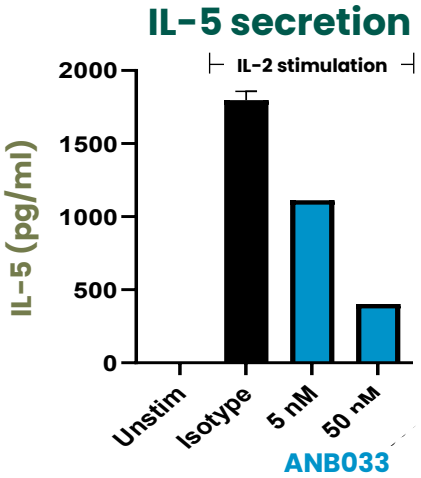
ANB033 reduces CD4+ T cell and ILC2 derived Th2 cytokines, proven drivers of EoE pathology

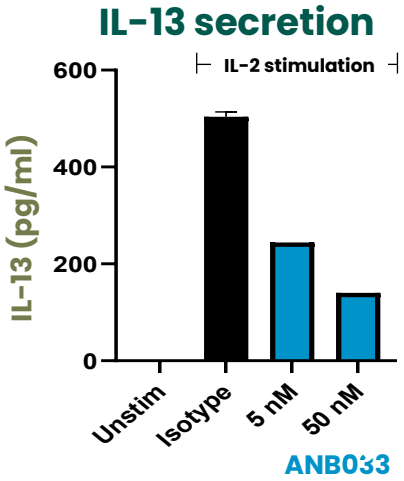
PBMC





ILC2

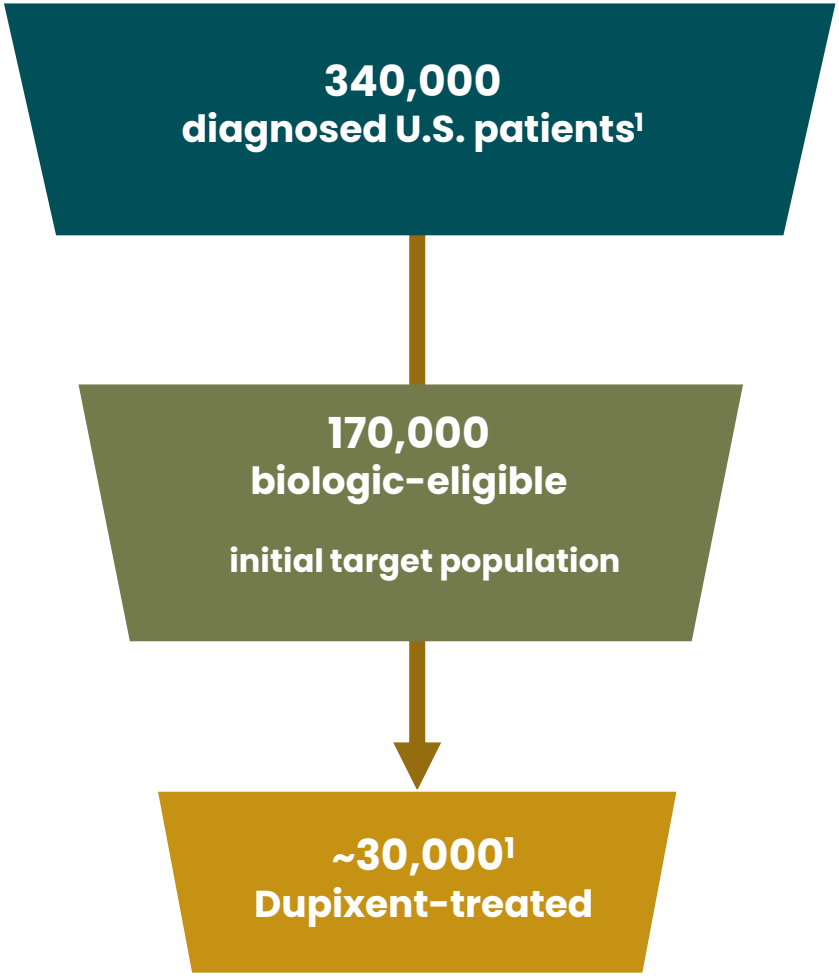




ANB033 Isotype Control

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

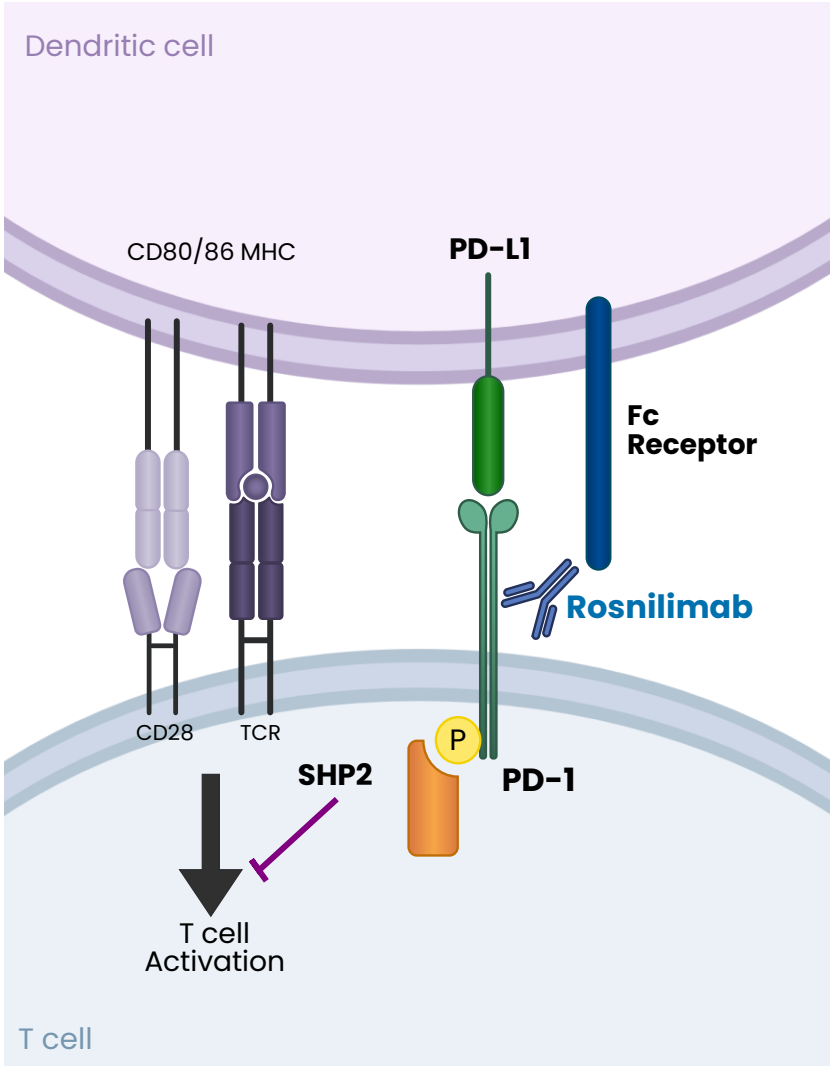
- Potential to reach IBD diagnosis and biologic penetration analogs given substantial unmet need

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

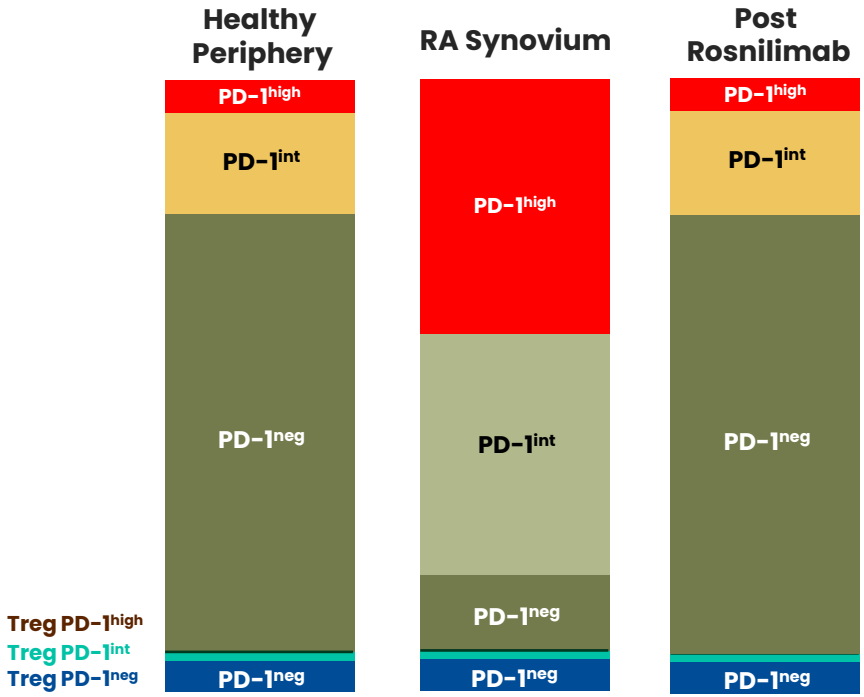
Rosnilimab selectively targets pathogenic T cells in periphery and inflamed tissue to restore immune homeostasis



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.

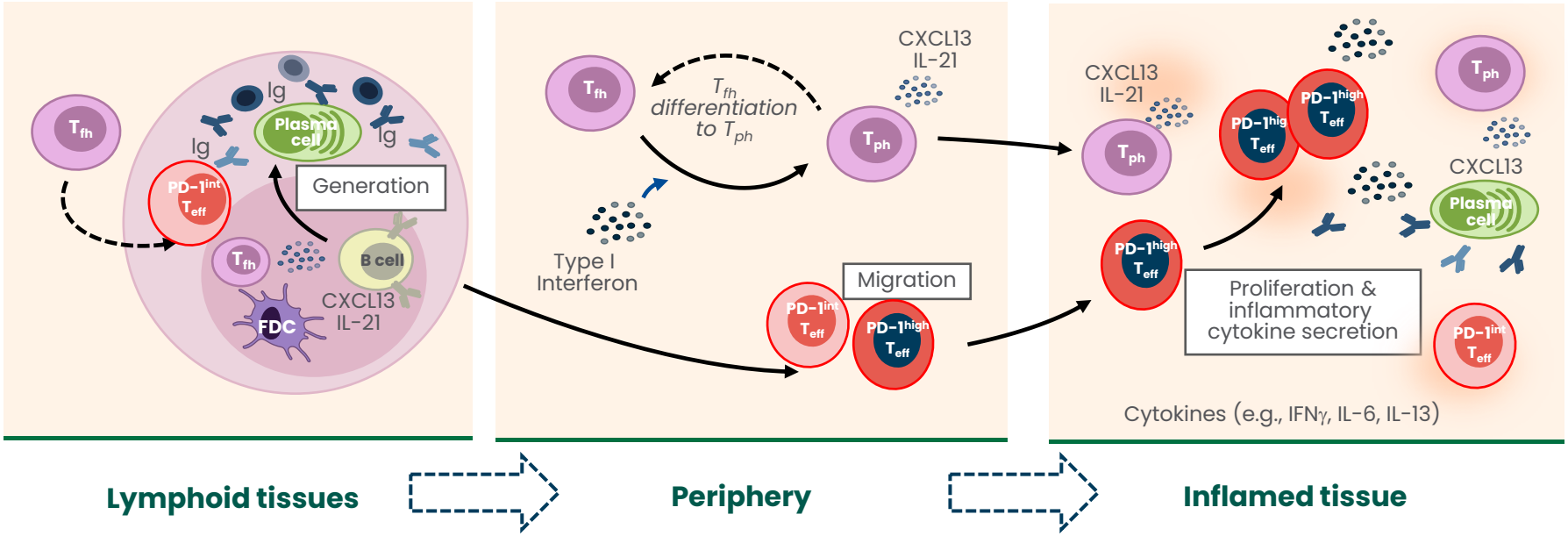
Pathogenic Teff and Tfh/Tph cells mediate autoimmune pathology

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

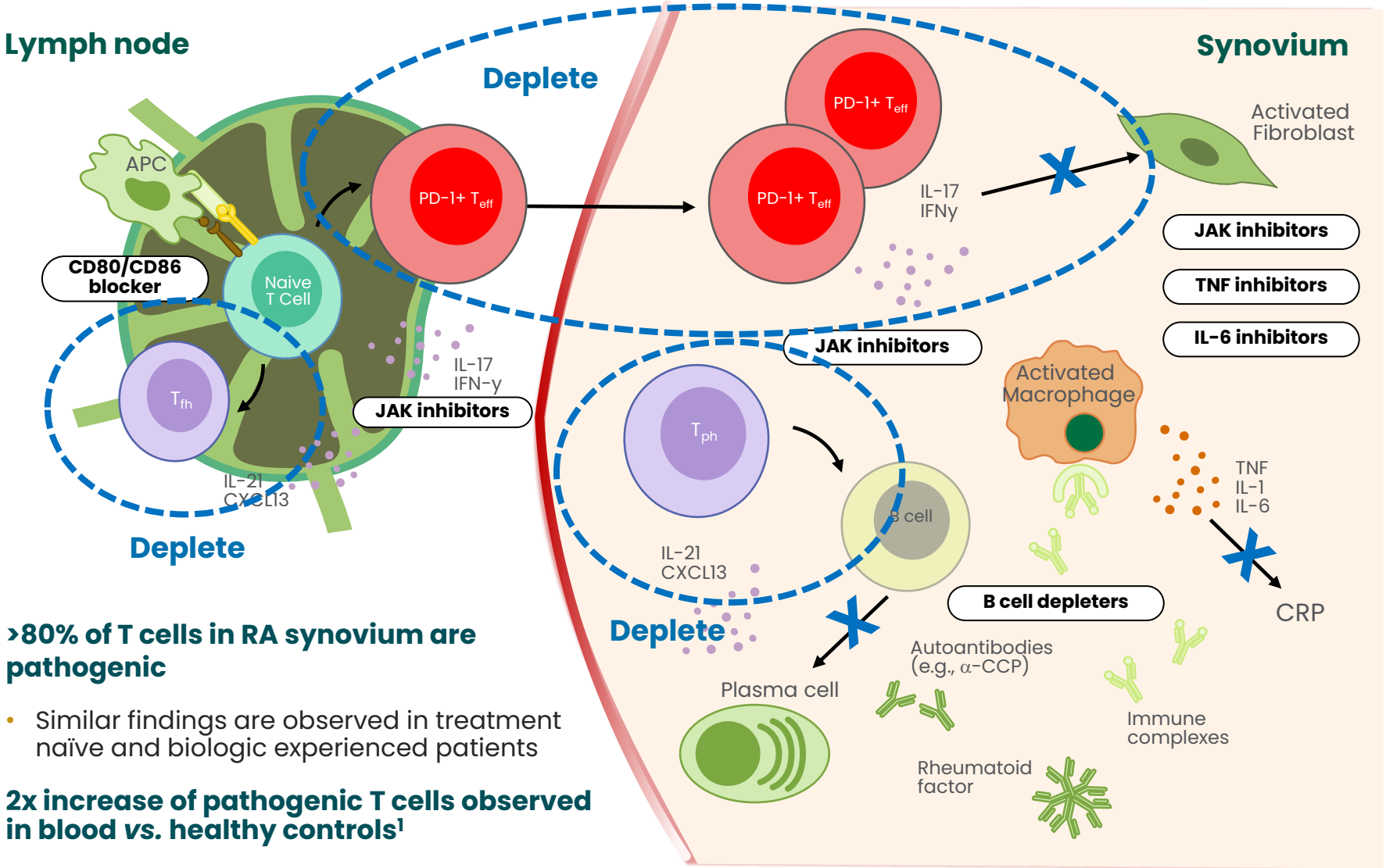
T_{eff} (effector)

- 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. 41

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

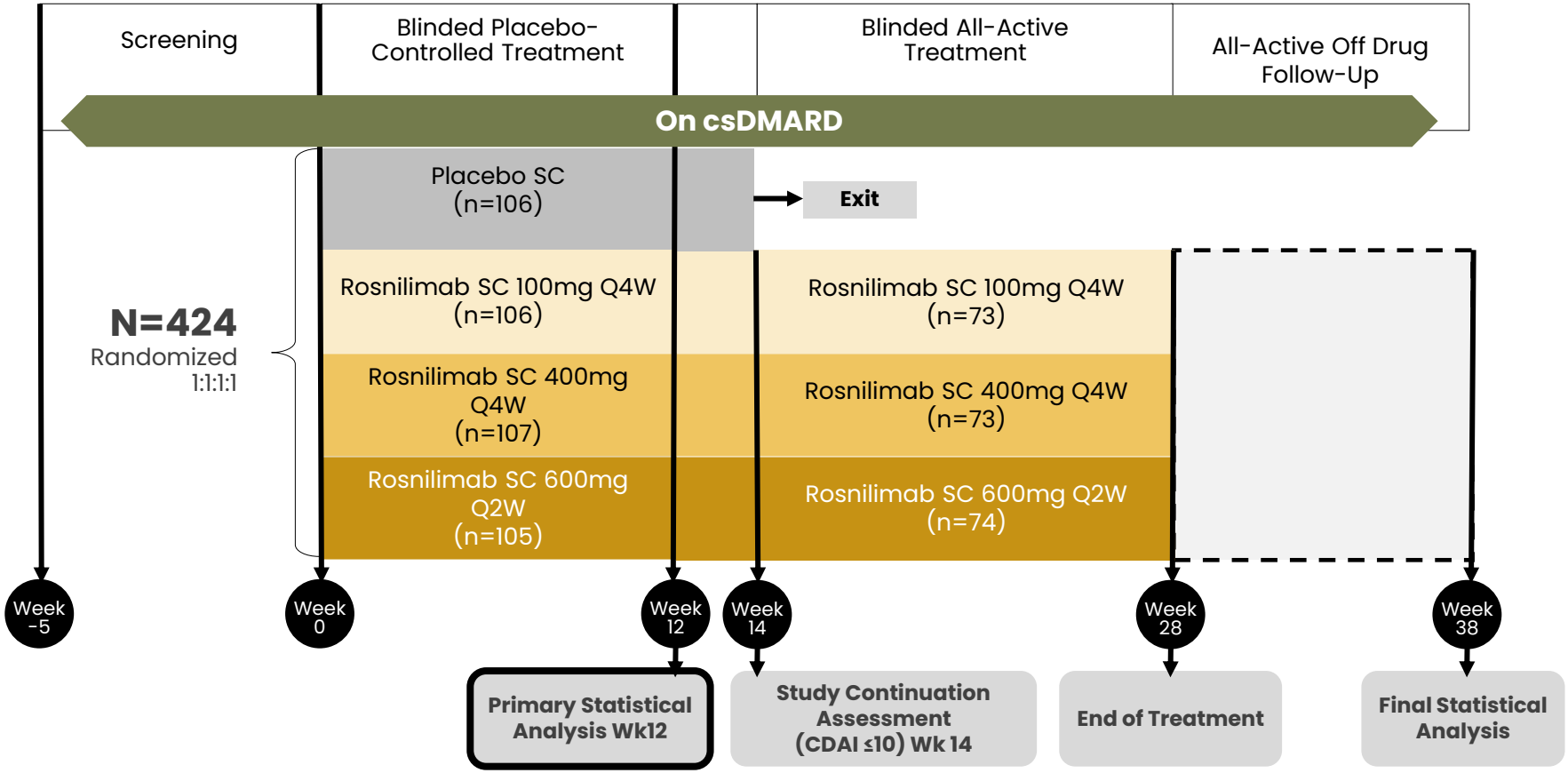
2x increase of pathogenic T cells observed in blood *vs.* healthy controls[1]

First Tracks BIOTHERAPEUTICS

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



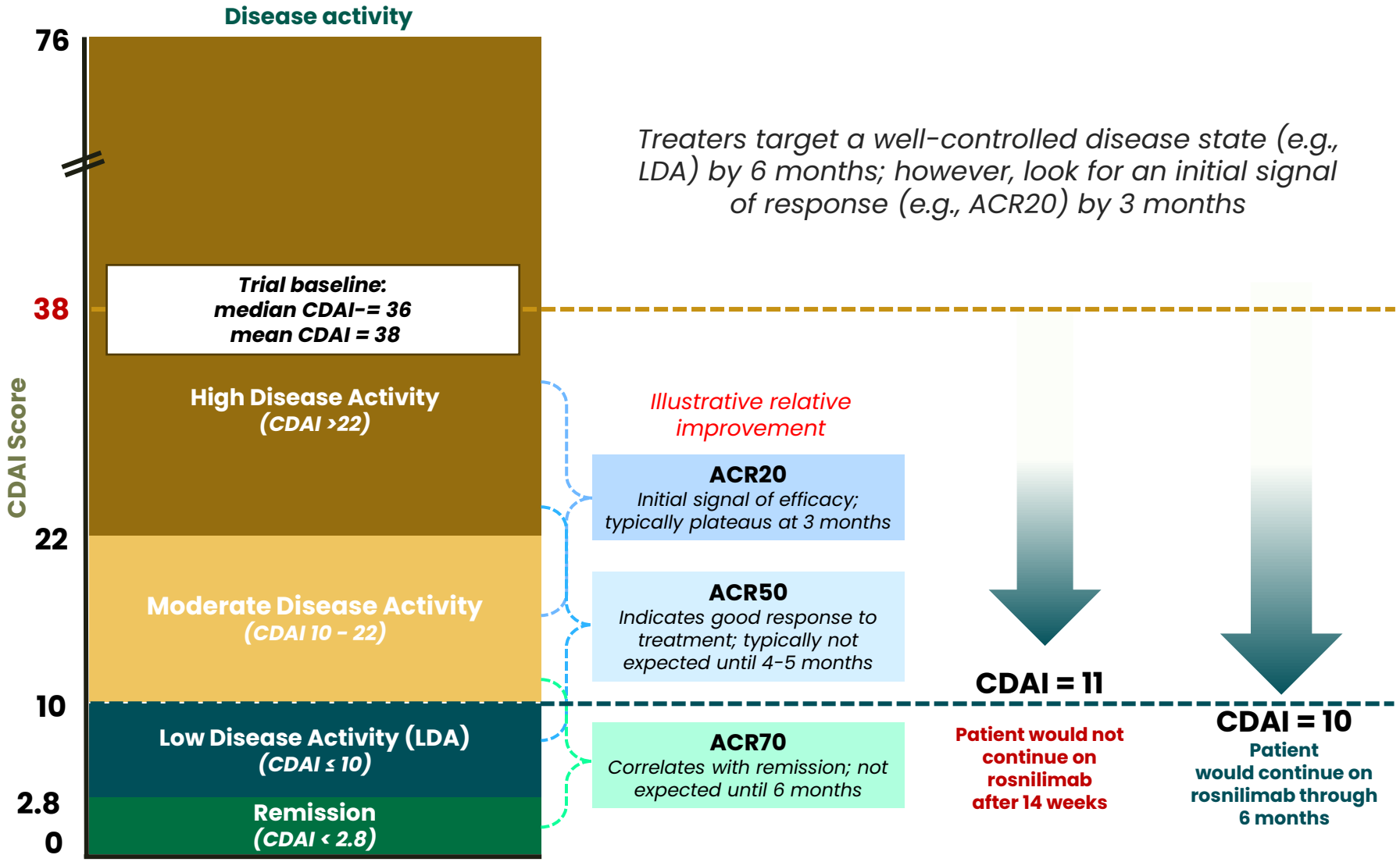
Key Inclusion Criteria – Seropositive RA, ≥6 swollen and ≥6 tender joints, hs-CRP ≥ 3mg/L during Screening, Concurrent use of 1 or 2 csDMARDs that were initiated at least 3 months before screening

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)

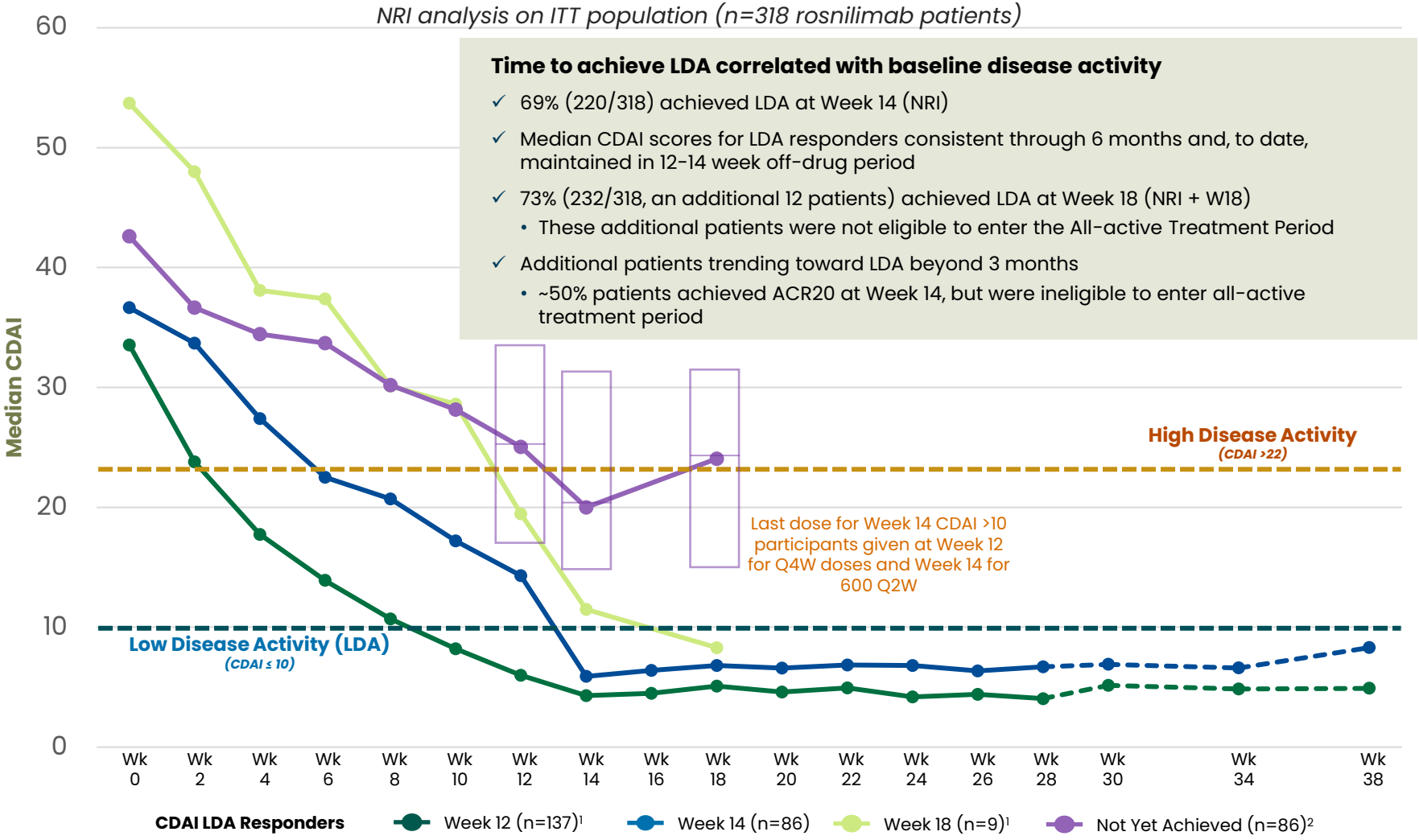
Rosnilimab, a pathogenic T cell depleter, is well-positioned for the ~$20 billion U.S. RA market which hasn't had a new mechanism approved since 2012

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)



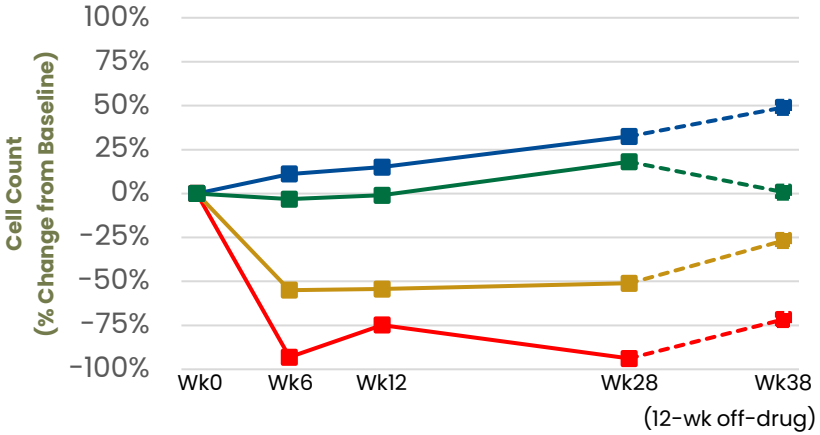
Time to achieve LDA correlated with baseline disease activity

- ✓ 69% (220/318) achieved LDA at Week 14 (NRI)
- ✓ Median CDAI scores for LDA responders consistent through 6 months and, to date, maintained in 12-14 week off-drug period
- ✓ 73% (232/318, an additional 12 patients) achieved LDA at Week 18 (NRI + W18)
  - These additional patients were not eligible to enter the All-active Treatment Period
- ✓ Additional patients trending toward LDA beyond 3 months
  - ~50% patients achieved ACR20 at Week 14, but were ineligible to enter all-active treatment period

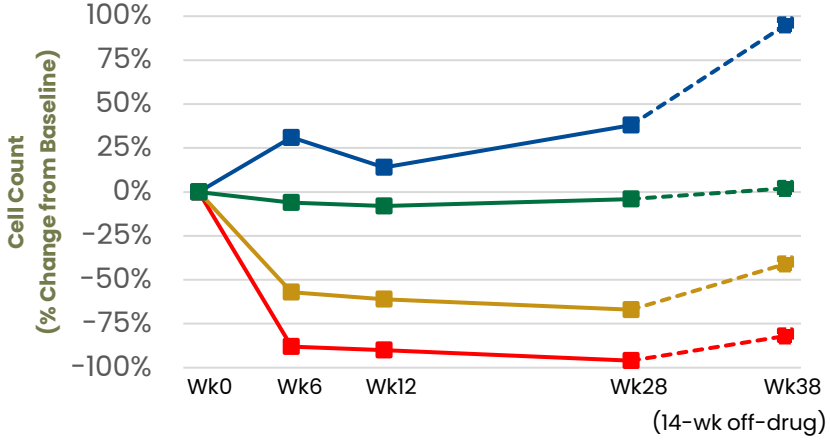
1. Green line includes 3 patients that achieved LDA at Week 12, were not CDAI LDA at Week 14, but returned to CDAI LDA at Week 18. These same 3 patients were excluded from the Light Blue line. In total 12 patients achieved CDAI LDA at Week 18. 2. Purple line includes rosnilimab patients that discontinued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

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

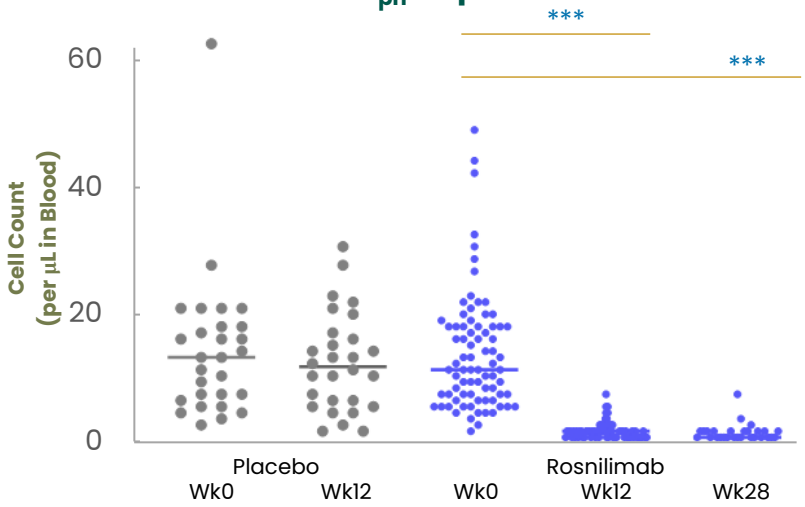
Rosnilimab 400mg Q4W T Cell Impact



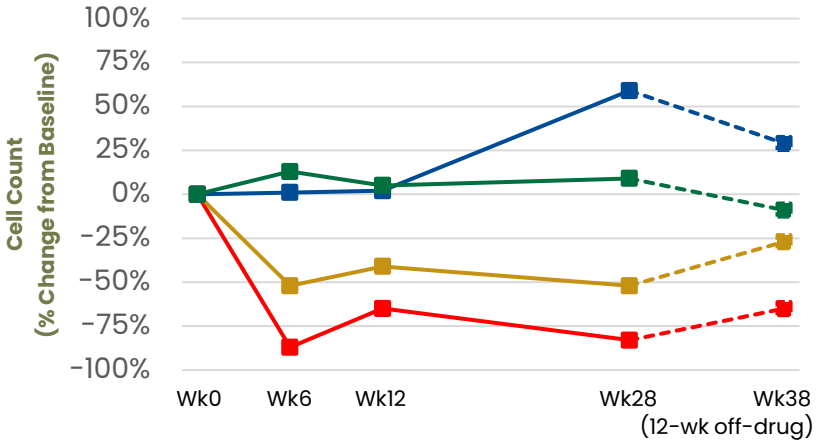
Rosnilimab 600mg Q2W T Cell Impact



Rosnilimab T_{ph} Impact – Pooled Doses



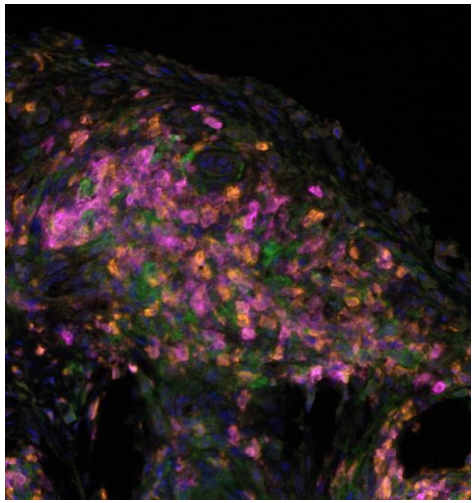
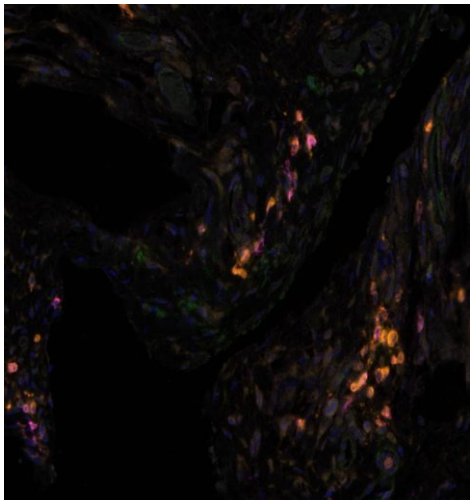
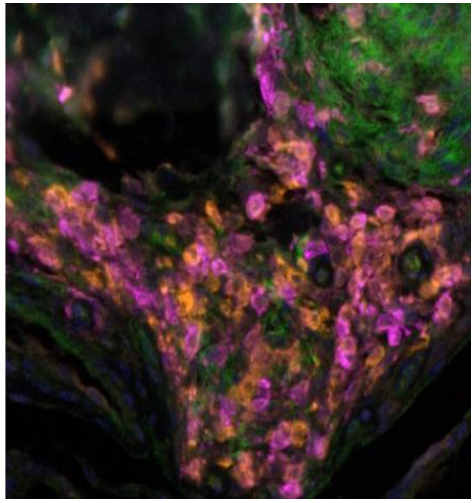
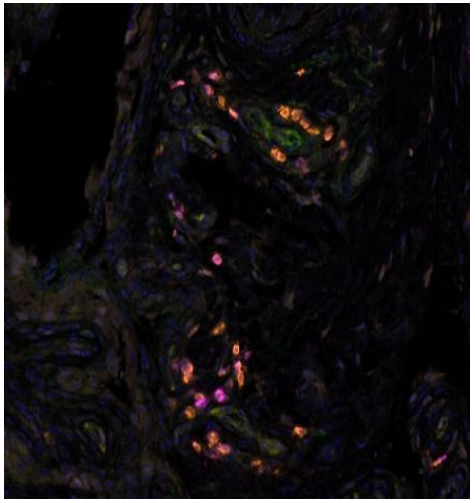
Rosnilimab 100mg Q4W T Cell Impact

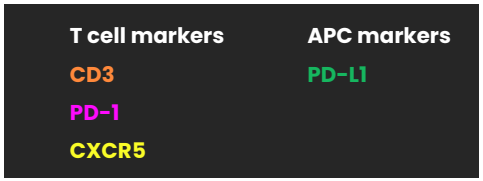


PD-1^{high} T Cells | PD-1+ T Cells | Total Treg | Total T Cells

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

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

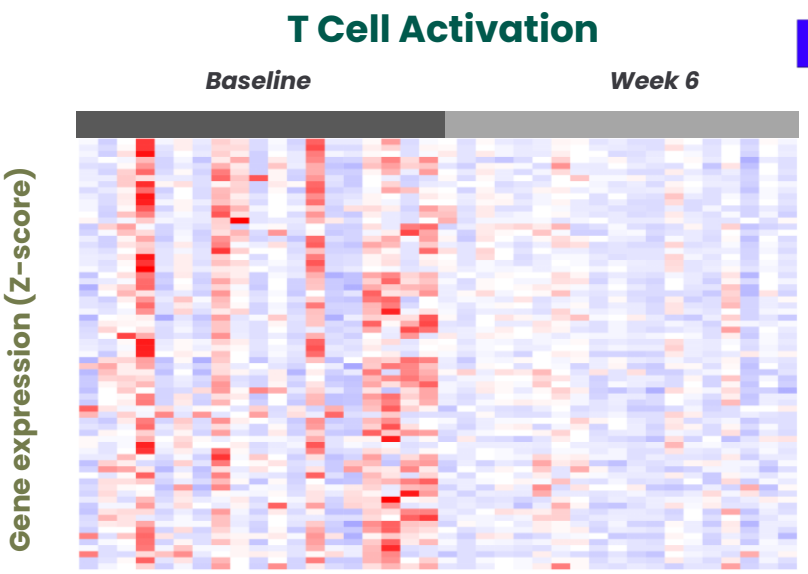
	Baseline	Week 6	Impact on T_{ph} and T_{eff} cells
Rosnilimab 400mg Q4W			**400mg/600mg doses** *~90% reduction* **100mg dose** *Inconclusive reduction*
Rosnilimab 600mg Q2W			**Placebo** *Increased*



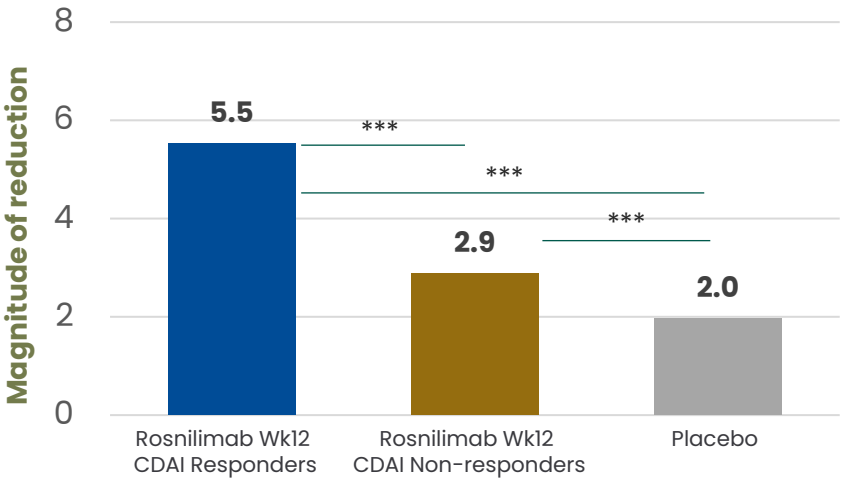
Note: Synovial biopsies of the most impacted joint taken at baseline and 6 weeks on study. Immunofluorescence performed to identify PD-1 positive cells. Tph cells (PD-1+CD3+CD4+CXCR5-).

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

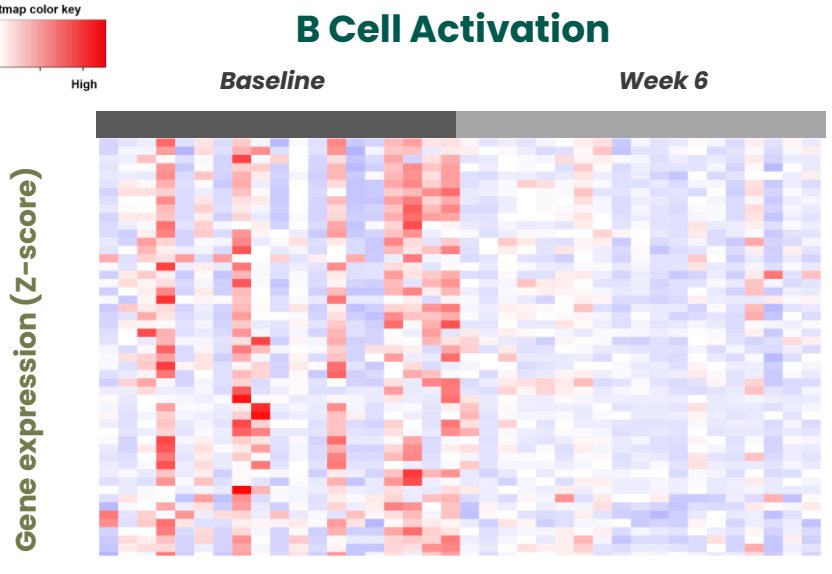
T Cell Activation



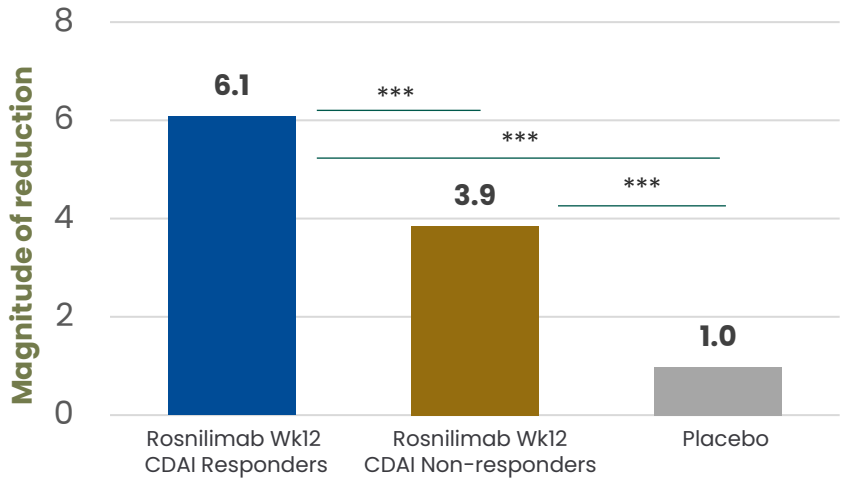
Magnitude of reduction: 4.03, p=1.09e-24



B Cell Activation



Magnitude of reduction: 4.06, p=1.77e-18



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.

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*

First Tracks BIOTHERAPEUTICS

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; No malignancies or deaths; Herpes zoster the only opportunistic infection reported and none were severe.

Next steps for rosnilimab

Rheumatoid Arthritis

Positive Phase 2b data reported

- Best-in-disease profile
- Favorable safety and tolerability
- JAK-like efficacy through 6 months
  - Max response rates not yet observed due to trial design
- Sustained 12-14 week off-drug responses through 9 months
- Late-breaking data presented at ACR 2025

- Completed End-of-Phase 2 (EOP2) meeting with FDA in Q1 2026
  - The agency provided constructive feedback on a registrational Phase 3 path in RA, a disease with significant remaining patient unmet need
- Currently assessing ROE-maximizing strategic options that may include a global partnership, out-license or asset financing to progress the potential development of rosnilimab in RA and other indications

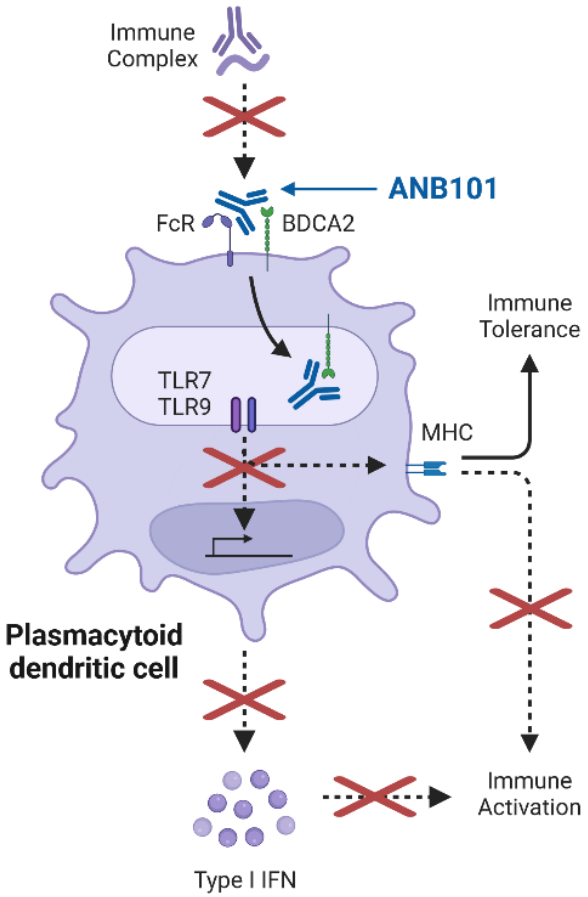
ANB101

BDCA2 modulator

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

Phase 1 trial nearing completion in healthy volunteers

BDCA2 is a molecule specifically expressed on pDCs



ANB101 potently inhibits 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