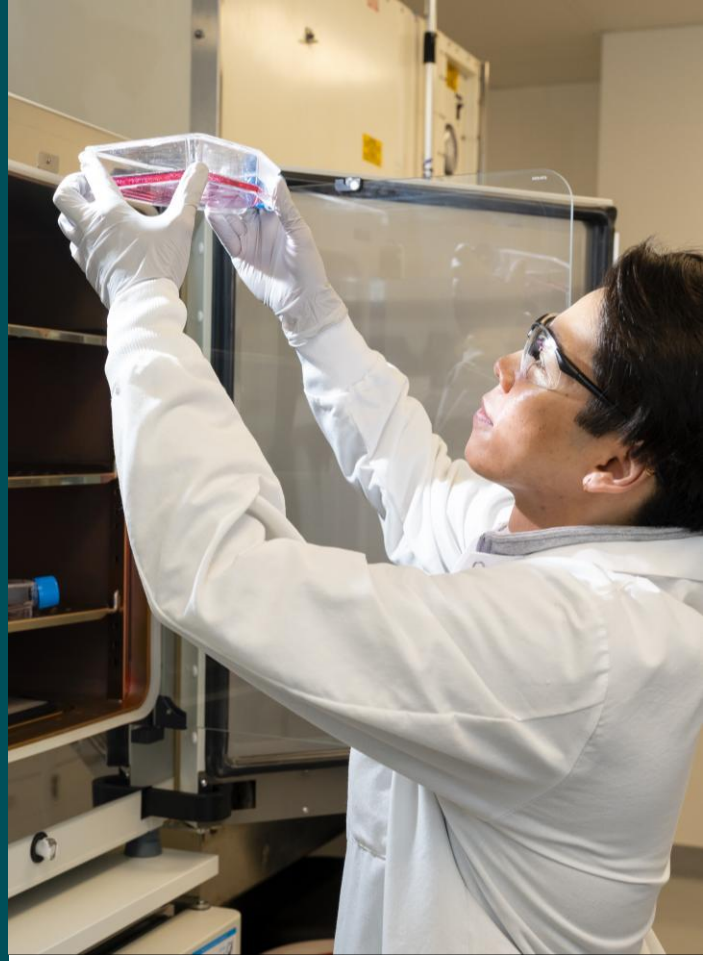
Corporate Presentation

CARVING A NEW TRAIL IN IMMUNOLOGY

November 2026

NASDAQ: TRAX



First Tracks
BIOTHERAPEUTICS

Safe harbor statement

This presentation and any accompanying oral presentation contain forward-looking statements within the meaning of the "safe harbor" provisions of the Private Securities Litigation Reform Act of 1995, including statements regarding the potential benefits, efficacy and safety profile of ANB033 and the Company's other product candidates; the Company's regulatory plans and the anticipated timing of initiation of, enrollment in, and data from, its clinical trials; strategic options for rosnilimab; and the potential market opportunity for ANB033. Statements including words such as "plan," "expect," "anticipate," "potential" or "ongoing" and statements in the future tense are forward-looking statements. Top-line results are based on a preliminary analysis and may change following a more comprehensive review of the data, and results from a Phase 1b trial may not be predictive of results in later-stage trials.

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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 gastroenterology, dermatology and other broad autoimmune disease areas

ANB033
(CD122 antagonist)

- Phase 1b in CeD
- Phase 1b in EoE
- Anticipate Phase 2 expansion in H1 2027 into vitiligo and one additional indication

Rosnilimab
(Pathogenic T Cell depleter)

- Phase 2b completed in RA

ANB101
(BDCA2 modulator)

- Phase 1a completed in healthy volunteers

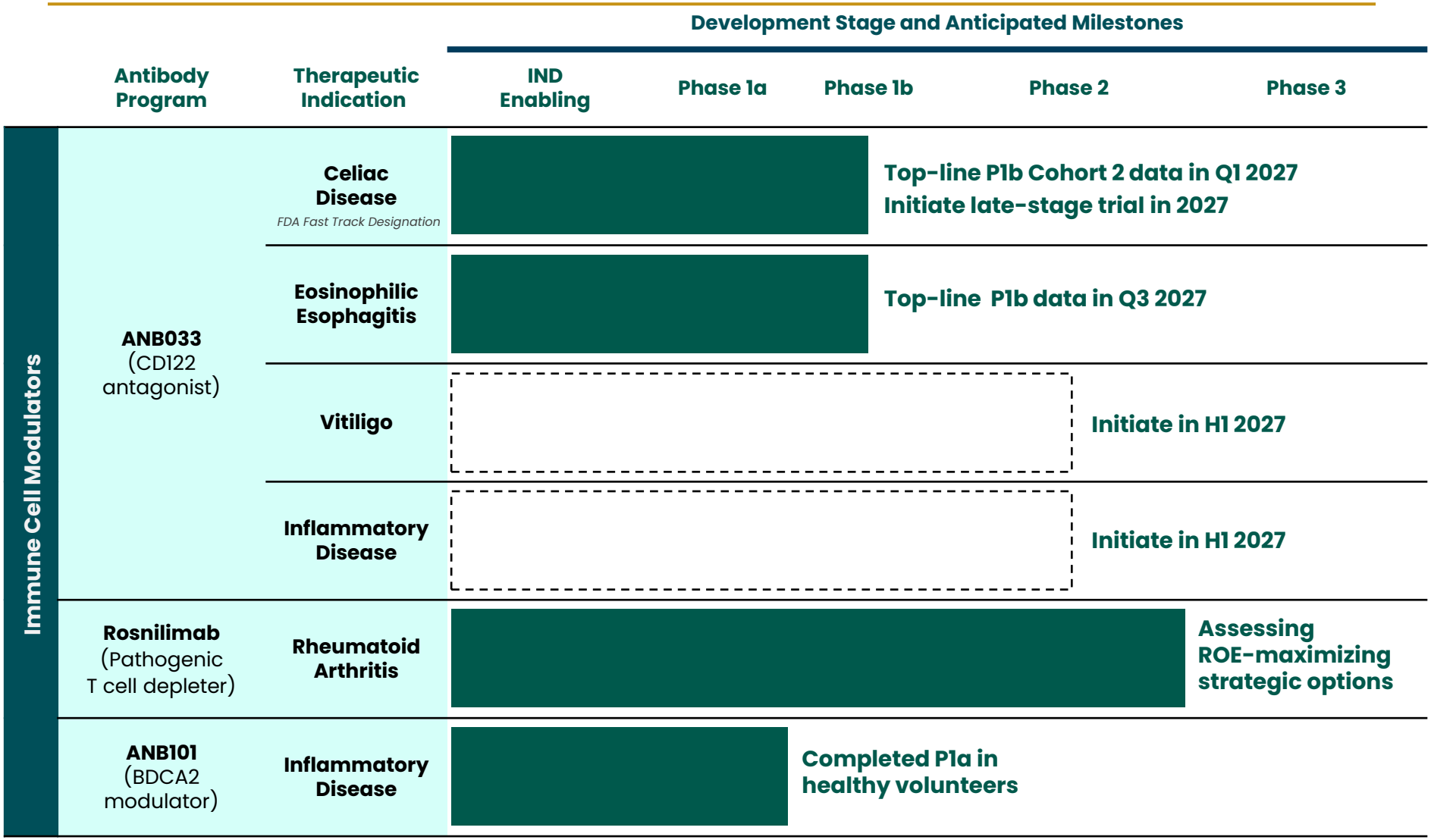
$343 million in pro forma cash[1]

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

1. Cash, cash equivalents and investments totaled $155.6 million as of Sept. 30, 2026, with pro forma cash of $343 million following net proceeds from $200 million private placement announced on Oct. 15, 2026.

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

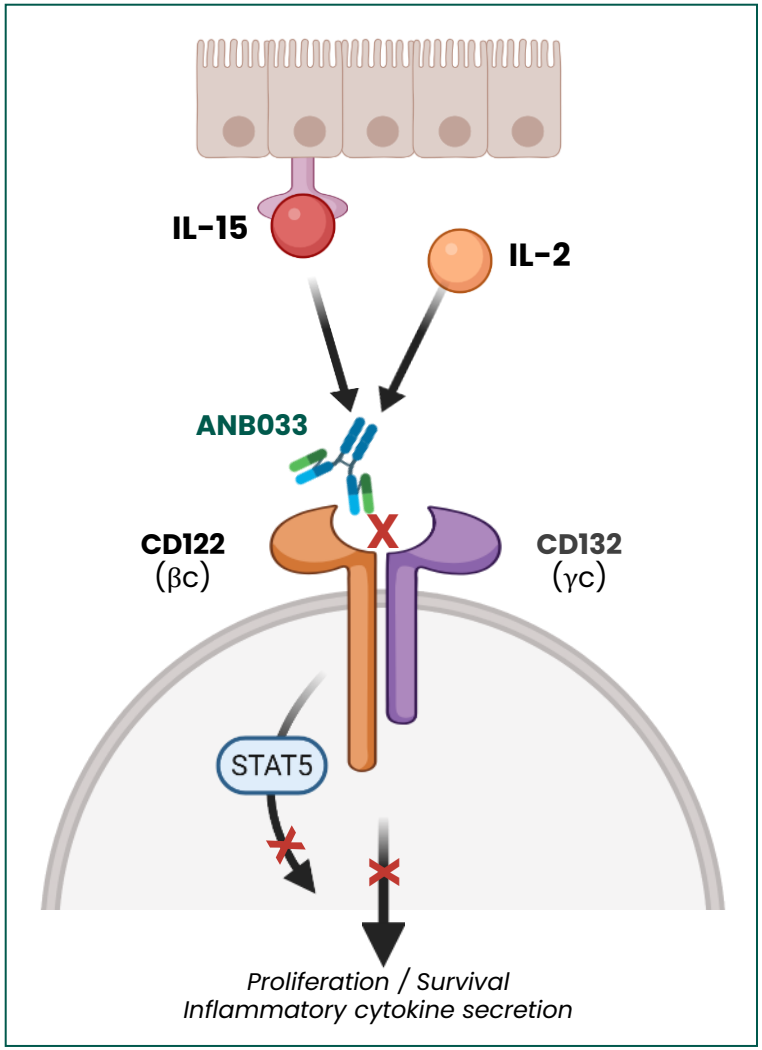
- Expressed on subsets of CD8+ and CD4+ T cells and NK cells

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



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

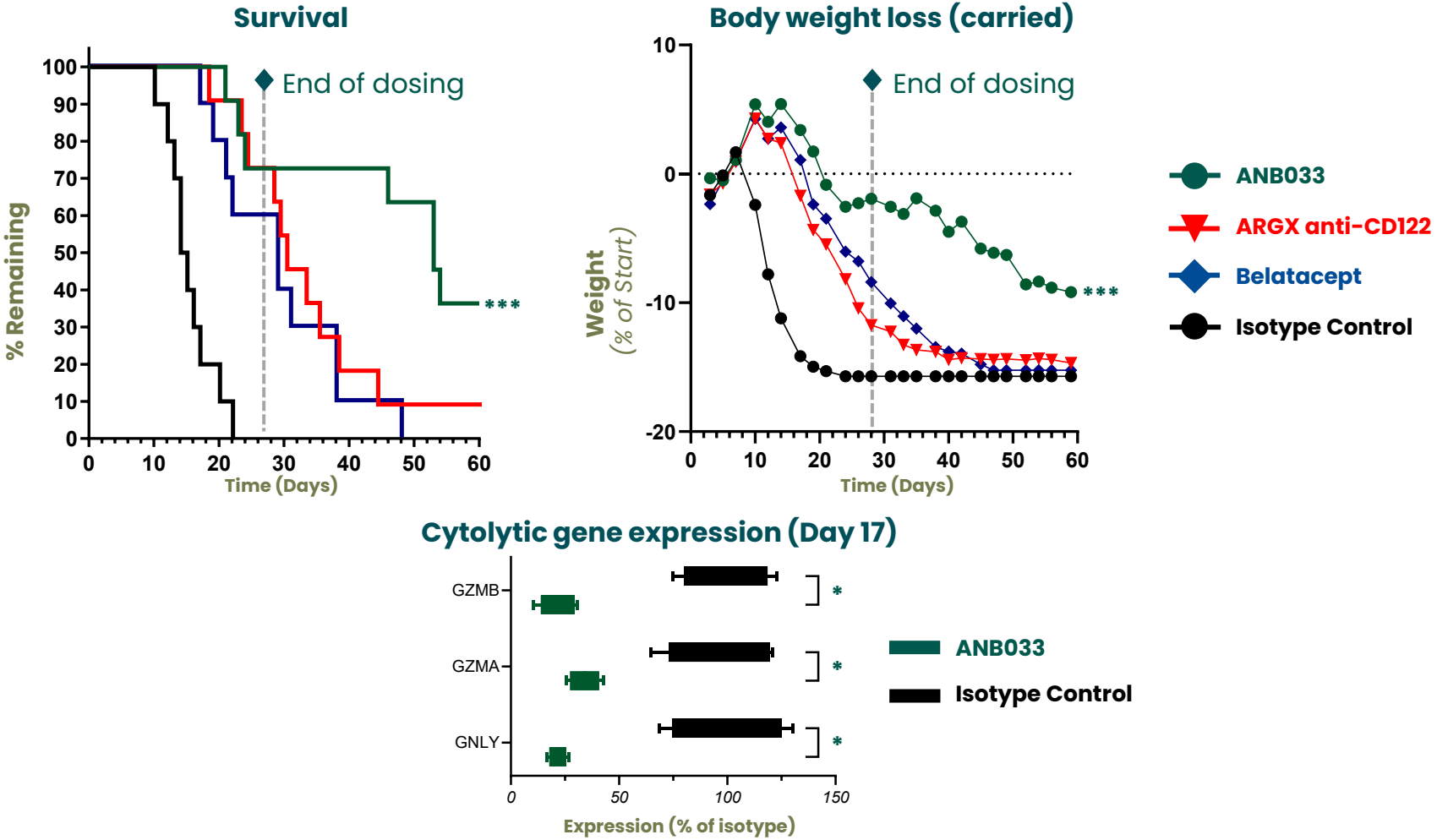
Broad therapeutic potential across autoimmune and inflammatory diseases

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	• PoC in CeD and EoE • Atopic dermatitis, vitiligo, and cutaneous lichen planus
teva	IL-15	• CeD and vitiligo • *Assessing atopic dermatitis, alopecia areata and EoE*
argenx	CD122	• CeD, vitiligo, alopecia areata

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), Argenx 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), Argenx 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.

Phase 1a demonstrated favorable safety, tolerability and PK profile with clear impact on immune cells driving CeD

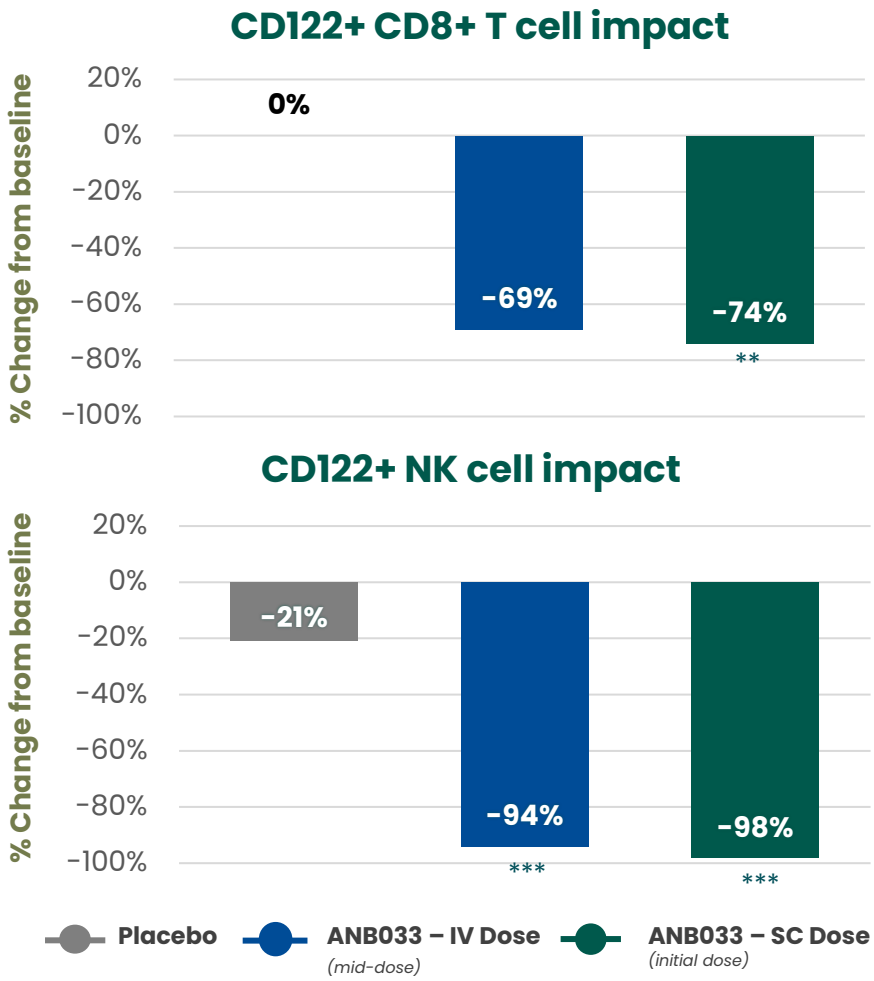
ANB033 Phase 1a results[1]

Rapid and sustained PK profile

- Up 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

Favorable safety and tolerability at all doses

- No viral infections or unexpected lab abnormalities
- Overall, no impact on peripheral total Treg counts

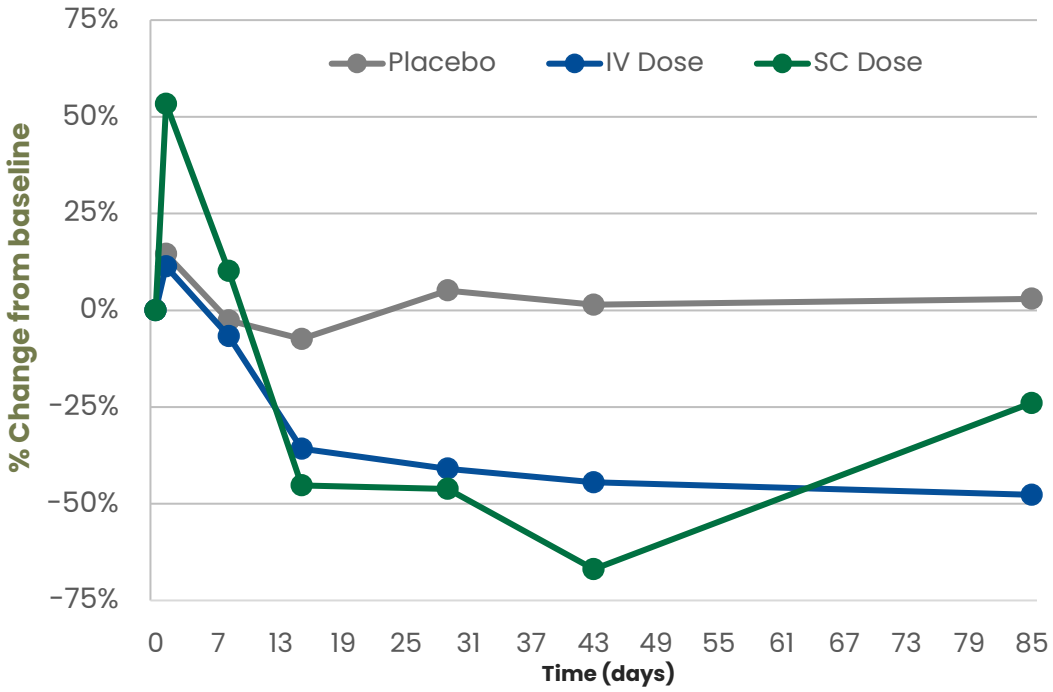


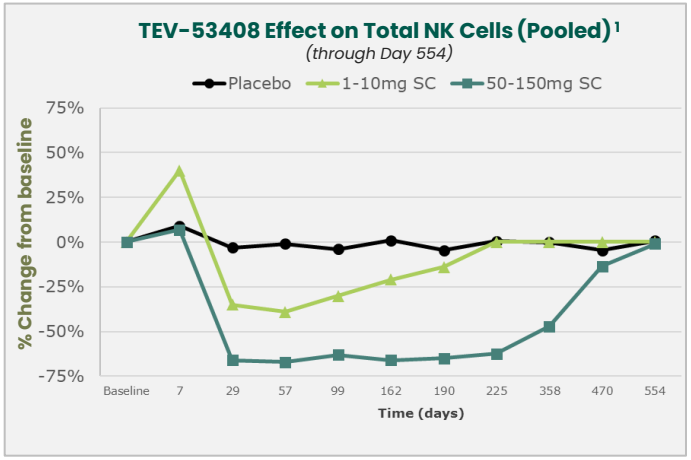
First Tracks BIOTHERAPEUTICS

1. N=80 (n=60 ANB033, n=20 placebo) Phase 1a trial, administered both IV and SC dosing, 10 cohorts (four SAD IV, three SAD SC, and three MAD SC, follow-up to >7 months; 2. 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

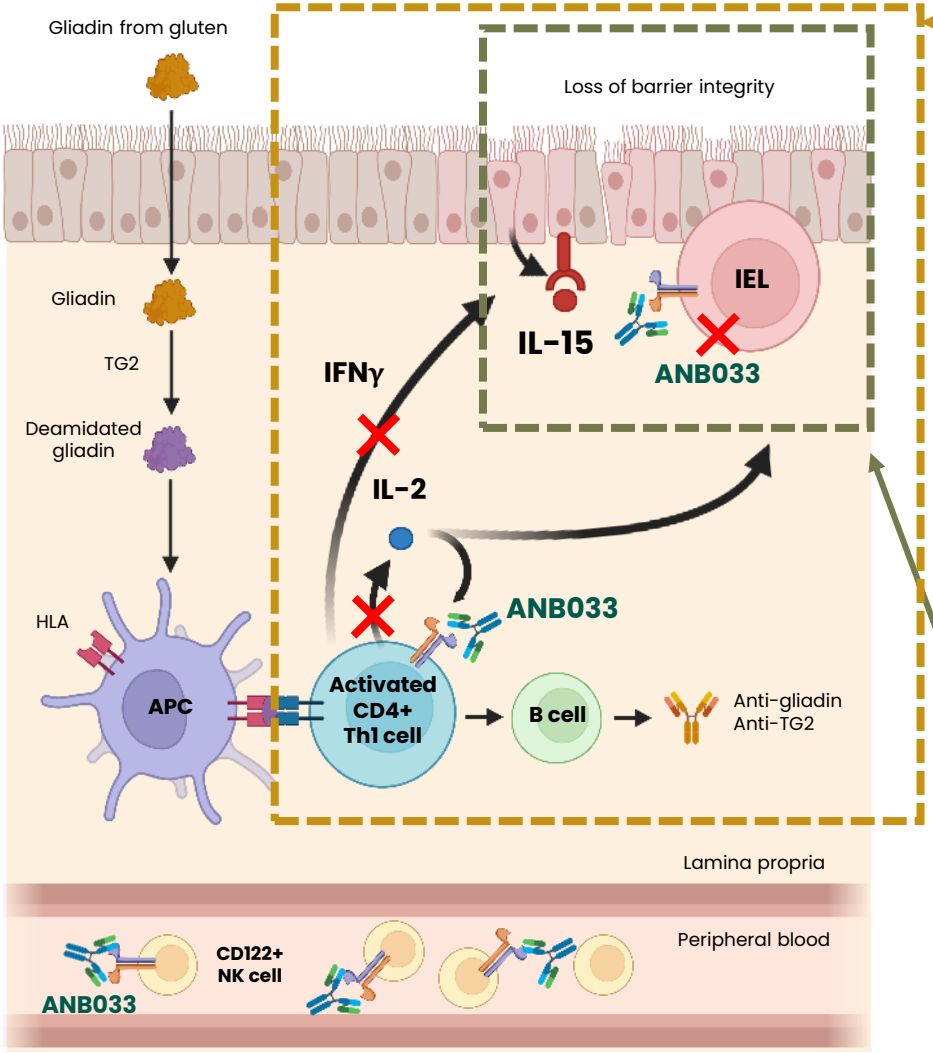
- ✓ ANB033 >50% peak total NK cell reduction with return towards baseline within 3 months
- ✓ TEV-53408: >50% sustained total NK cell reduction for 1 year with return to baseline over 18 months

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-2 and IL-15 production which perpetuates disease



Inhibition of IL-2 signaling

- IL-2 stimulates
  - CD4 effector memory T cell activation and proliferation
  - IFNγ production leading to IL-15 secretion
  - Induction closely linked to acute symptom development
- Inhibiting IL-2 signaling reduces
  - Gluten-responsive CD4 T cell expansion
  - Inflammatory cytokine secretion

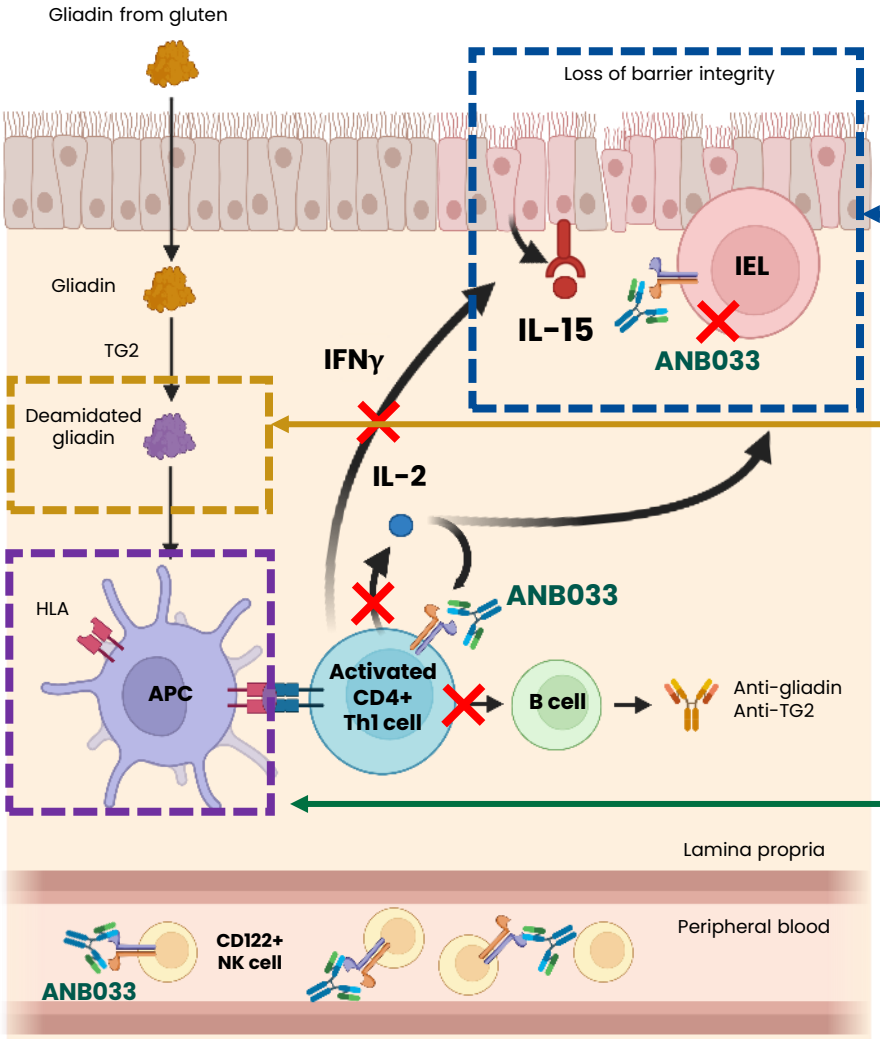
Inhibition of IL-15 signaling

- 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

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 — *P2a PoC*
- NOVARTIS — *P1b PoC*
- AMGEN — *Lacked potency*

Non-immune cell targeting

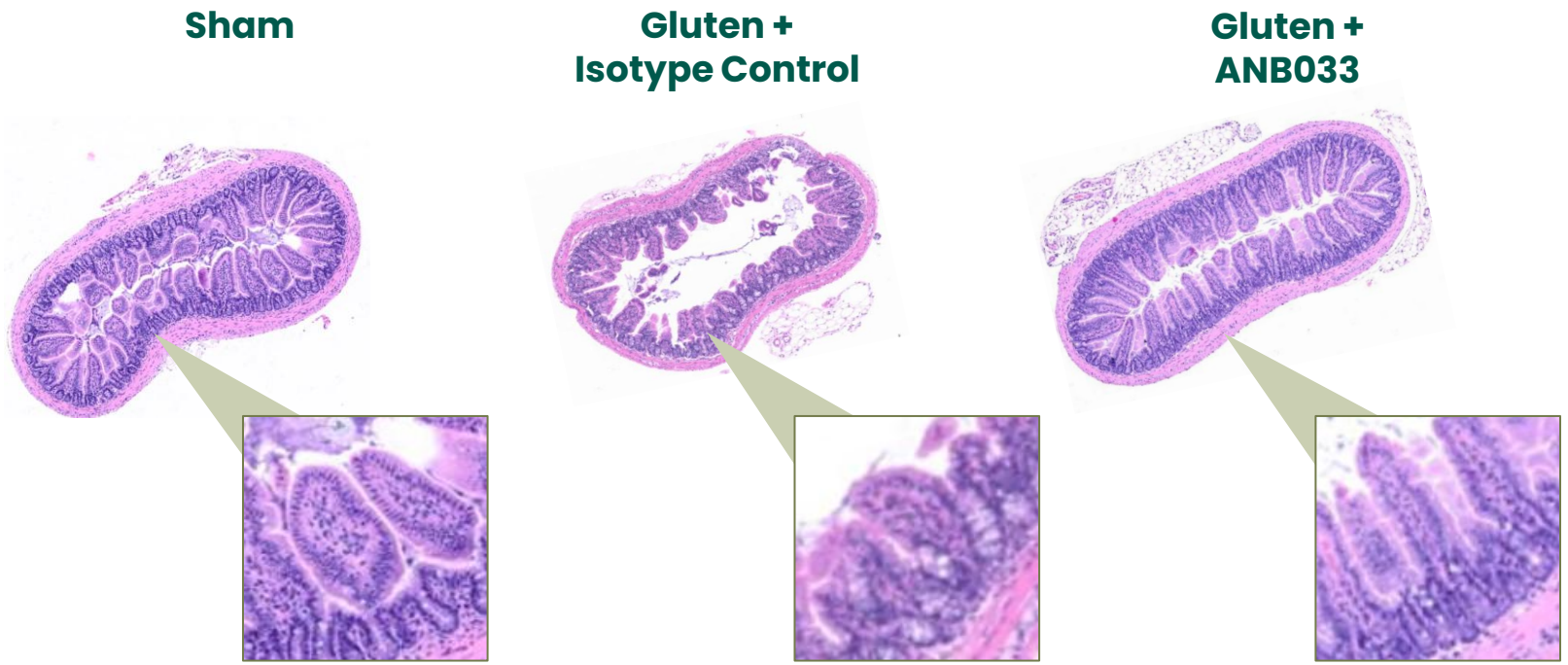
- Takeda — *P2 ongoing* — *Gluten tolerance*
- astellas — *Discontinued* — *Gluten tolerance*
- Roche — *P1 ongoing* — *HLA-DQ2.5 gluten peptide complex*
- Immunic THERAPEUTICS — *P1 ongoing* — *SIRT6 modulator*

OX-40L antagonist

- sanofi — *P2 data readout in H2 2026*

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

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



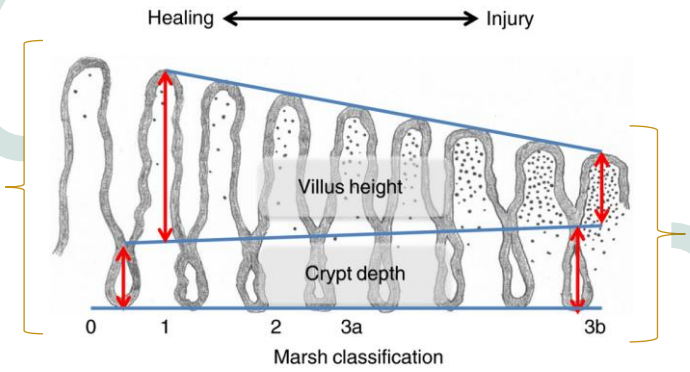
ANB033 treatment shows improved histology:
preserves villus height and crypt depth (Vh:Cd) in CeD mouse model

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

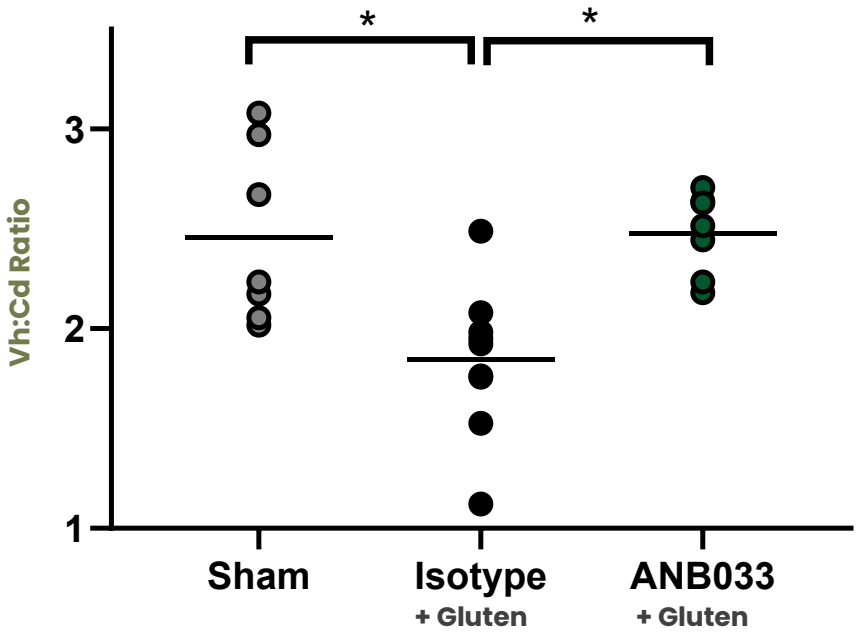
Vh:Cd ratio

Healthy tissue:
High villus height, lower crypt depth



CeD tissue:
Reduced villus height, extended crypt depth

ANB033 impact on Vh:Cd ratio



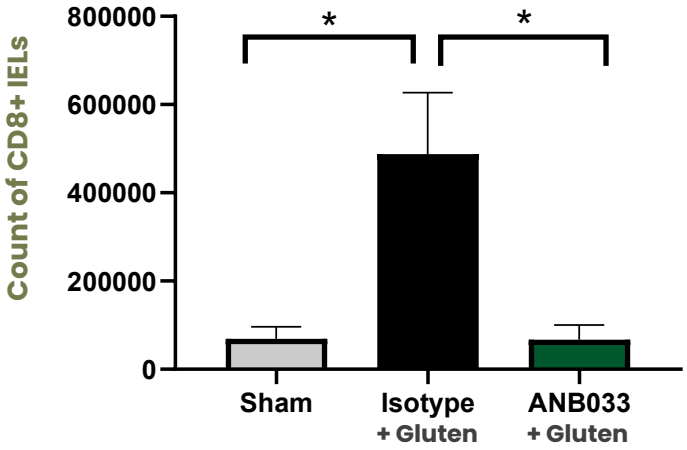
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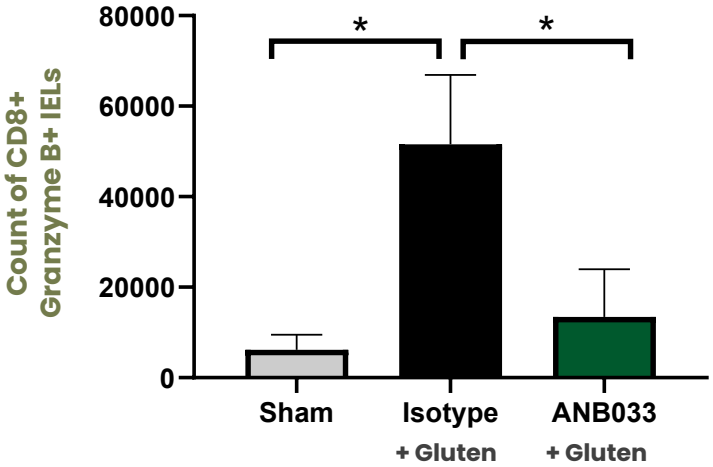
ANB033 prevents gluten-induced intestinal inflammation

Epithelial layer of small intestine

No increase in CD8+ IELs

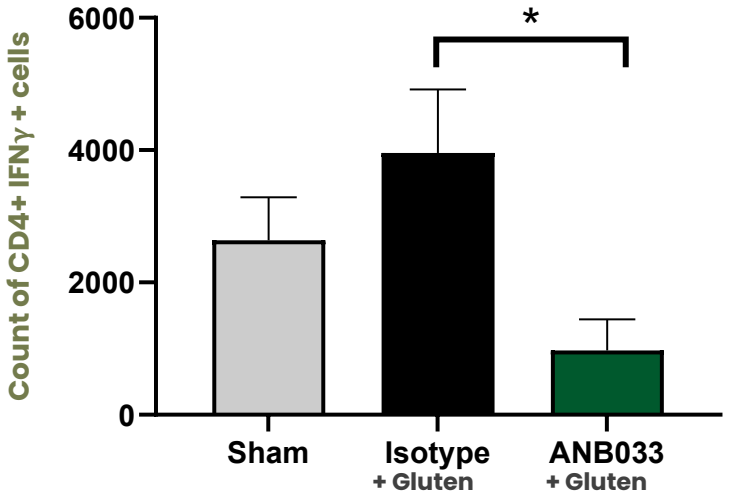


No increase in Granzyme B+ IELs



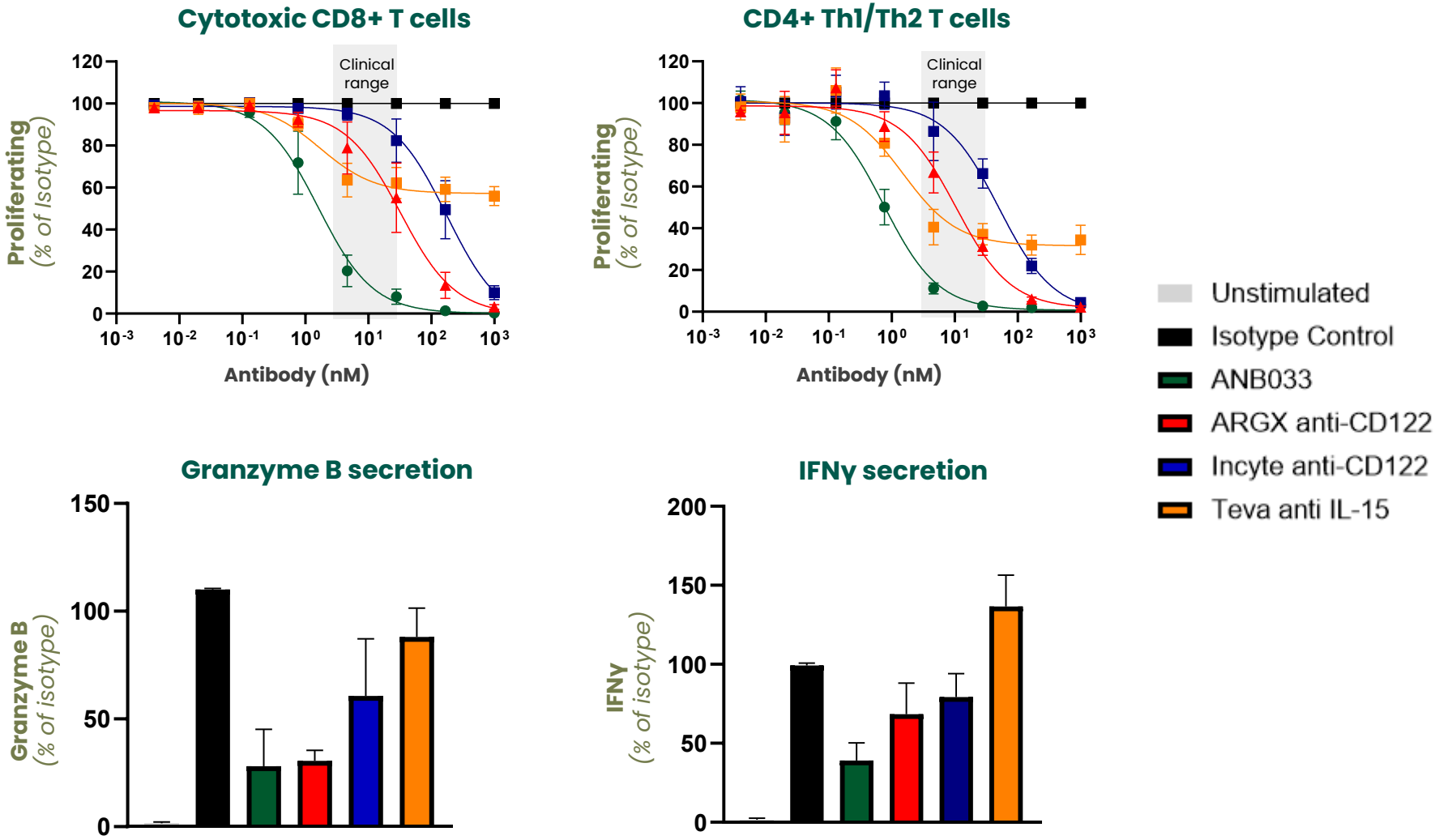
Lamina propria (LP)

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.

CeD patients present with range of histologic activity and symptoms that are often not directly correlated

Histology *(Vh:Cd ratio)*



Symptoms are intermittent and can vary from none to mild to moderate to severe across histological damage

First Tracks BIOTHERAPEUTICS *(Phase 1b)*

Cohort 1 (Vh:Cd ≥ 2.0)
Gluten challenge to assess the prevention of intestinal damage

Cohort 2 (Vh:Cd ≤ 2.5)
Added additional cohort to P1b **to inform on potential to heal mucosa** in patients with existing histologic intestinal damage and further derisk future development

Competitive Landscape

teva *(Phase 2a)*

NOVARTIS Calypso biotech *(Phase 1b)*

argenx FORTE *(Phase 1b/2a)*

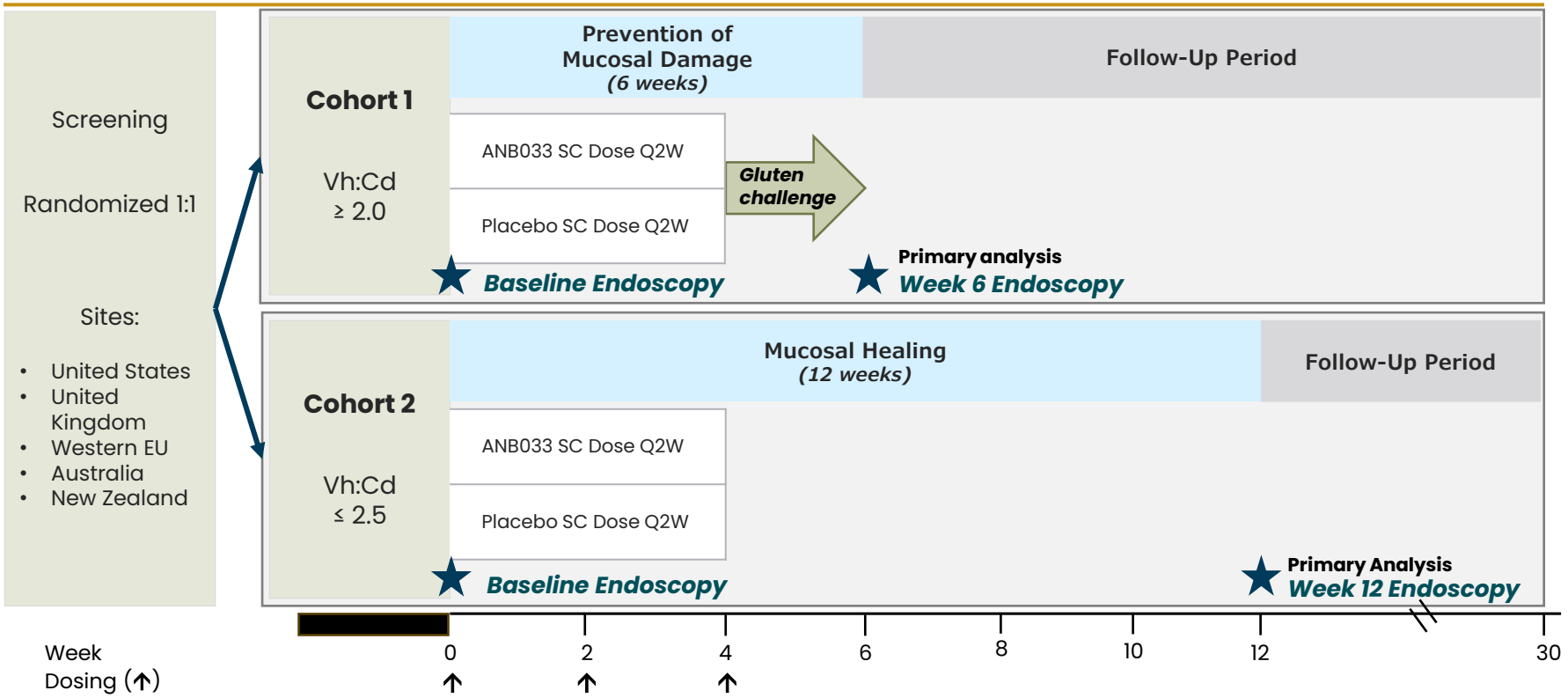
Gluten challenge: patients with higher Vh:Cd ratios (>2.5 or >2.0)

sanofi
(P2 data readout in H2 2026)

Phase 2b study to assess if drug can heal damaged mucosa and restore normal symptomatology (Vh:Cd ≤ 2.5)

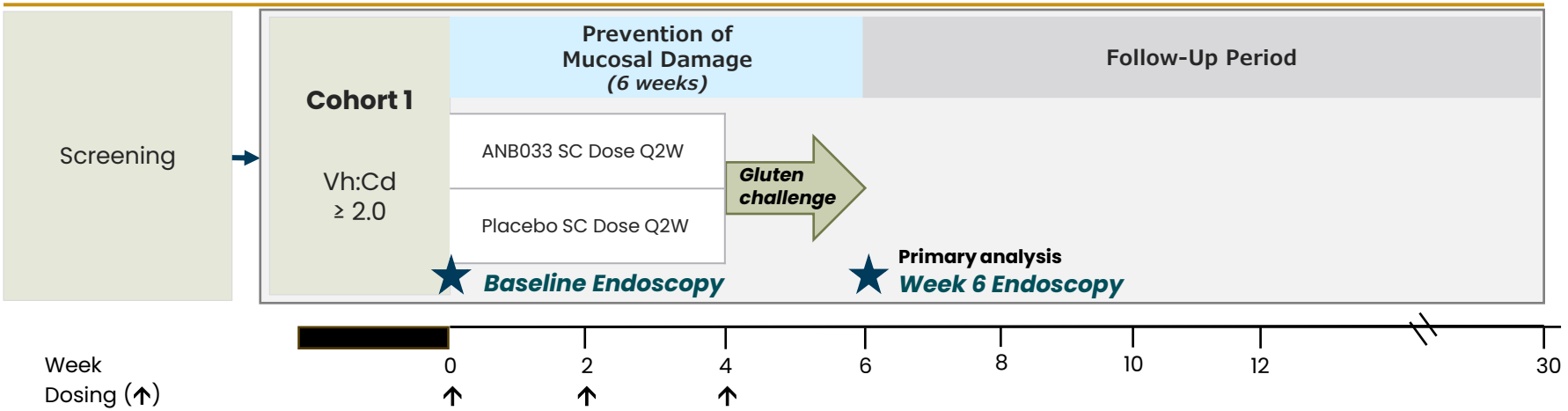
ANB033 Phase 1b trial in CeD

Anticipate top-line Cohort 2 data in Q1 2027



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

Cohort 1 (gluten challenge) to assess prevention of mucosal damage and symptoms



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

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

- ANB033 subcutaneous loading dose at Week 0, 2, 4 (pre-gluten challenge)
- Gluten challenge allowed for controlled induction of mucosal damage
  - Beginning Week 4, 6g gluten dose daily (two 3g study supplied cookies) for 14 days through Week 6
- Endoscopy at Week 6
  - Assessed prevention of gluten-induced mucosal damage and daily symptoms

Cohort 1 baseline characteristics

Baseline Characteristic	Placebo (n=15)	ANB033 (n=17)	Overall (n=32)
Age (years)	41.1	44.3	42.8
Sex (n, %) Male Female	 2 (13.3%) 13 (86.7%)	 5 (29.4%) 12 (70.6%)	 7 (21.9%) 25 (78.1%)
Baseline mean Vh:Cd ratio	2.77	2.74	2.75
Baseline mean IEL density/100 enterocytes	22.50	23.20	22.90
Baseline mean VCIEL	-0.004	-0.146	-0.080

Vh:Cd = Villous height to crypt depth ratio; IEL = intraepithelial lymphocyte; VCIEL = Vh:Cd and IEL unified scale.

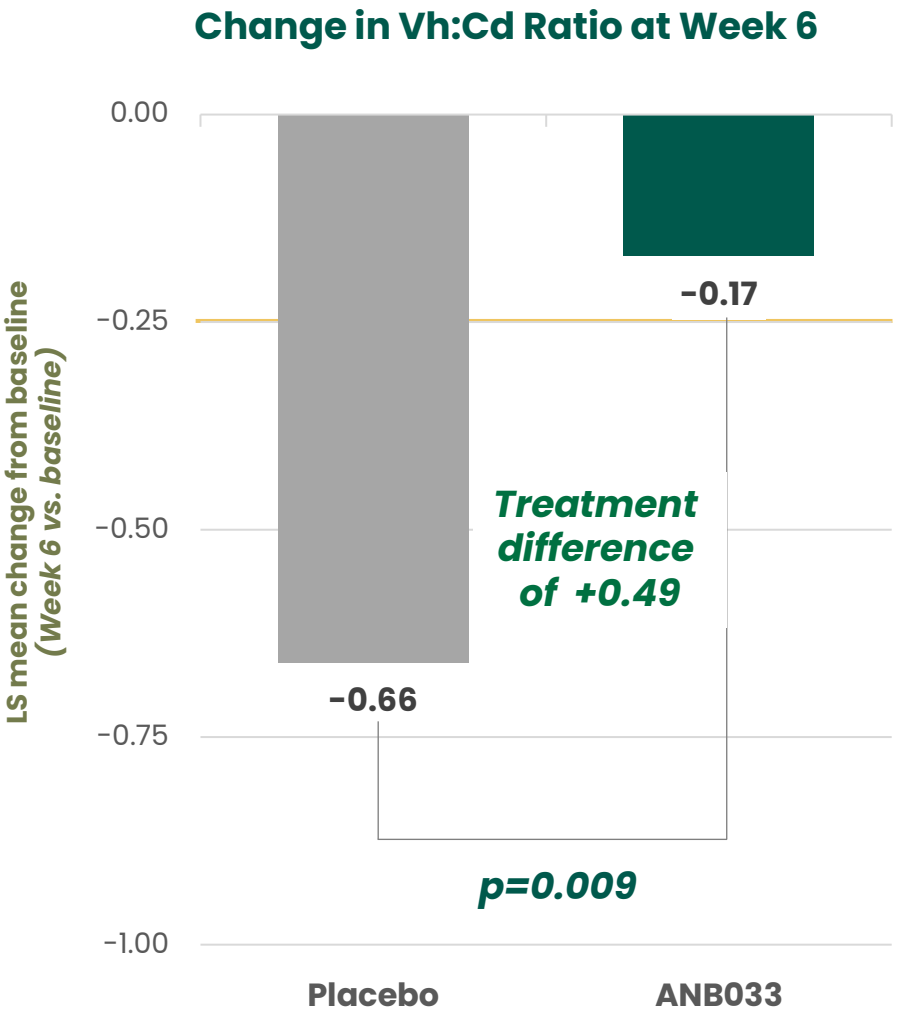
ANB033 achieved statistically significant and clinically meaningful protective effect on both gluten-induced damage and symptoms

Endpoint		Target	Result
Histology	Vh:Cd	Stat. sig. benefit	**0.49 Δ vs. placebo (p=0.009)**
		Protective effect (Vh:Cd <0.25)	**0.17 change Below clinically meaningful threshold**
	IEL density	Stat. sig. benefit	**20.03 Δ vs. placebo (p<0.001)**
		Protective effect (No increase)	**1.20 decrease (improvement)**
Symptoms	Total	Trend	**Prevented an increase in symptoms throughout entire gluten-challenge** **Lower CDSD score vs. placebo; Stat sig difference from first day**
	Moderate-to-severe	Trend	**Stat sig difference from first day**
Safety	Safety and tolerability	Well tolerated	**Well tolerated with no safety signals**

First Tracks BIOTHERAPEUTICS

Δ = Delta.

ANB033 demonstrated a statistically significant Vh:Cd histologic benefit vs. placebo



ANB033: Vh:Cd Histologic Protection

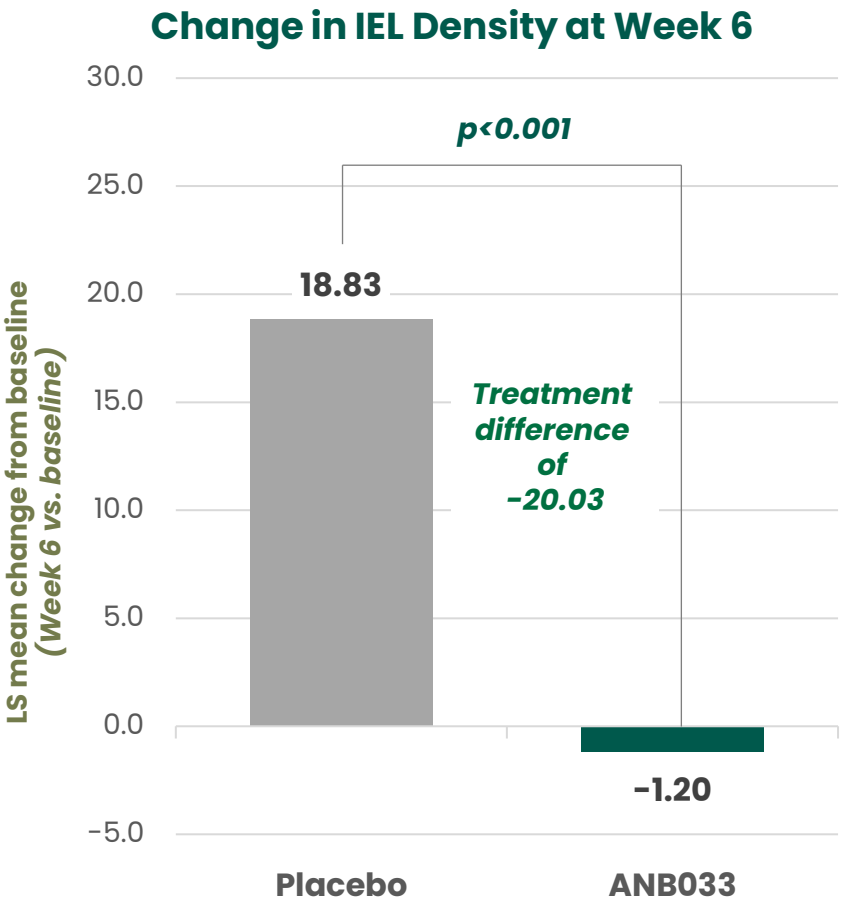
- Prevented a clinically meaningful decline below the ~0.25 threshold[1]
- Protected against gluten-induced intestinal damage

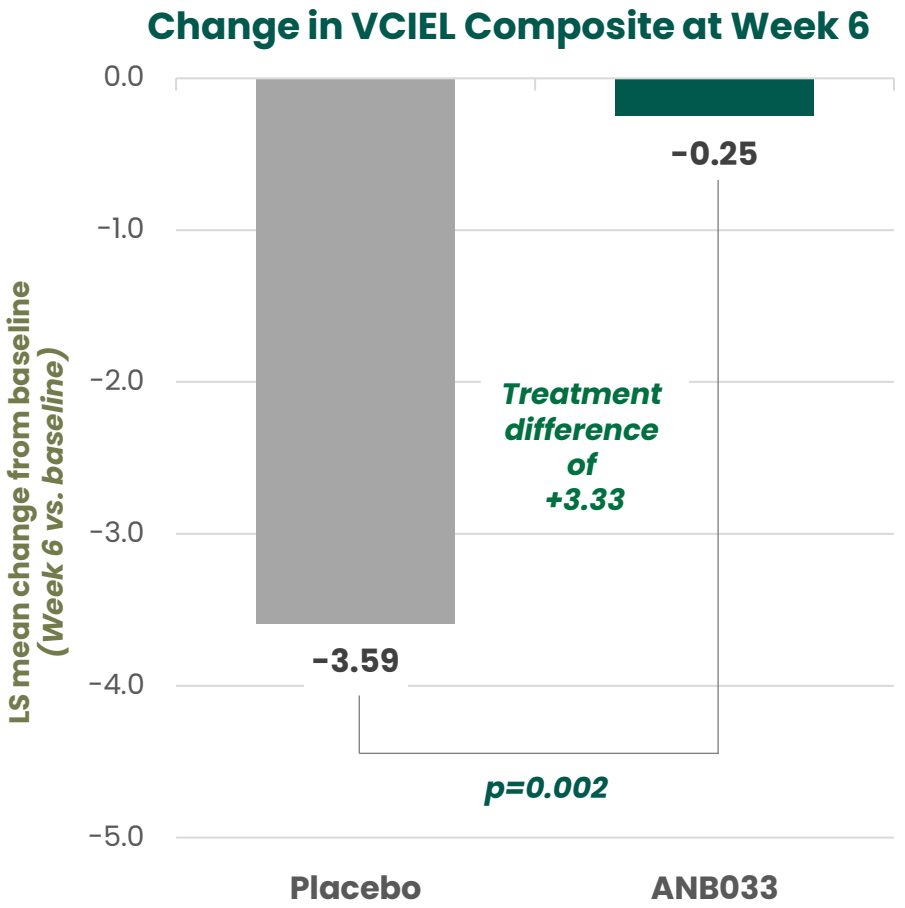
ANB033 vs. Placebo

- Reduction in Vh:Cd decline of ~75%

1. CeD KOL and gastroenterologist feedback on clinically meaningful Vh:Cd change; Vh:Cd = Villous height to crypt depth ratio; LS mean change = Least-squares mean change

ANB033 demonstrated statistically significant IEL density and composite VCIEL benefit vs. placebo





ANB033 not only prevented an increase, but reduced IEL density, despite an intense gluten challenge

IEL = Intraepithelial lymphocytes; IEL density = the number of IELs per 100 enterocytes; VCIEL = Vh:Cd and IEL unified scale.

ANB033 is the first therapy to report statistical significance on both histology and symptoms

Placebo patients experienced a marked increase in symptoms consistent with an intense gluten challenge

ANB033 prevented an increase in symptoms throughout the entire gluten challenge

47% of placebo patients and only 6% of ANB033 patients took permitted medications **to mitigate their GI symptoms**

Lower CDSD score on ANB033 vs. placebo, **a statistically significant difference from the first day through the seventh day**

Lower moderate-to-severe symptoms on ANB033 vs. placebo, **a statistically significant difference from the first day through the twelfth day**

First Tracks BIOTHERAPEUTICS

CDSD = Celiac Disease Symptom Diary; The CDSD is a validated, patient-reported outcome measure that assesses the frequency and severity of five key celiac disease symptoms which are abdominal pain, bloating, nausea, diarrhea, and tiredness.

ANB033 well tolerated with no safety signals observed to date

Treatment	Placebo (n=16)	ANB033 (n=18)	Overall (n=34)
Participants with any AE	**16 (100%)**	**16 (88.9%)**	**32 (94.1%)**
Any SAE	0 (0%)	0 (0%)	0 (0%)
Any severe AE	1 (6.3%)	0 (0%)	1 (2.9%)
Any moderate AE	12 (75.0%)	3 (16.7%)	15 (44.1%)
AE leading to treatment discontinuation	1 (6.3%)	0 (0%)	1 (2.9%)
AEs occurring in ≥ 3 participants			
Nausea	7 (43.8%)	1 (5.6%)	8 (23.5%)
Abdominal pain	6 (37.5%)	2 (11.1%)	8 (23.5%)
Injection site erythema	2 (12.5%)	5 (27.8%)	7 (20.6%)
Fatigue	5 (31.3%)	1 (5.6%)	6 (17.6%)
Diarrhea	4 (25.0%)	2 (11.1%)	6 (17.6%)
Vomiting	4 (25.0%)	2 (11.1%)	6 (17.6%)
Upper respiratory tract infection	3 (18.8%)	2 (11.1%)	5 (14.7%)
Injection site bruising	3 (18.8%)	1 (5.6%)	4 (11.8%)
Headache	3 (18.8%)	1 (5.6%)	4 (11.8%)
Abdominal distension	3 (18.8%)	0 (0%)	3 (8.8%)
Constipation	3 (18.8%)	0 (0%)	3 (8.8%)
Flatulence	3 (18.8%)	0 (0%)	3 (8.8%)

First Tracks BIOTHERAPEUTICS

Safety analysis set includes two additional participants who were randomized in Cohort 1 but discontinued the study prior to initiating the gluten challenge, were therefore unevaluable for gluten challenge efficacy analysis and did not undergo the second biopsy at Week 6. The one participant on ANB033 discontinued due to a decision not to undergo the gluten challenge.

ANB033 achieved statistically significant and clinically meaningful protective effect on both gluten-induced damage and symptoms

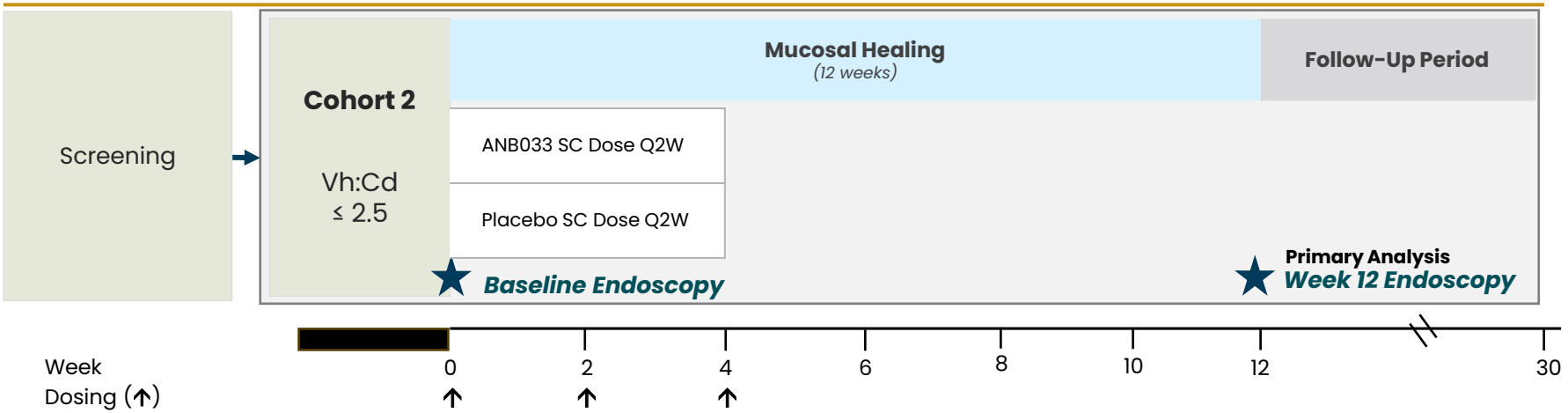
Program / Company	Gluten Challenge	Admin / MOA	Change in Vh:Cd	Change in IEL Density	Symptom Benefit
ANB033 *First Tracks*	**Phase 1b (n=32, 1:1) 14-day, 6g per day**	**SC (CD122)**	**Drug: -0.17 Placebo: -0.66** **0.49 PBO Δ** ***p=0.009***	**Drug: -1.20 Placebo: +18.83** **20.03 PBO Δ** ***p<0.001***	**Prevented an increase in symptoms throughout gluten challenge** **Lower CDSD scores vs. placebo; stat sig difference from first day**
TEV-53408[1] *Teva*	Phase 2a (n=50, 1:1) 6-week, 3g gluten/day	SC (IL-15)	Drug: -0.43 Placebo: -0.88 0.45 PBO Δ *p<0.05*	Drug: +0.37 Placebo: +27.60 27.2 PBO Δ No stats reported	*"Stable CDSD scores on drug; worse on placebo"* No stats reported
FB-102[2,3] *Forte / Argenx*	Phase 1b (n=32, 3:1) 16-day, 2g, 4g, to 8g/day	IV (CD122)	Drug: -0.046 Placebo: -0.173 0.13 PBO Δ p value not significant	Drug: -1.5 Placebo: +13.3 14.8 PBO Δ *P=0.0035*	4.0 drug vs. 6.9 placebo cumulative symptoms +AEs /subject No stats reported
	Phase 2a (n=126., 2:2:1) 54-day, 8g to 3g gluten/day	IV (CD122)	Drug (high dose): 0.41 PBO Δ; *p=0.0176* Drug (low dose): 0.29 PBO Δ; *p=0.0939*	Drug (high dose): 15.8 PBO Δ; *p=0.0001* Drug (low dose): 21.0 PBO Δ; *p<0.0001*	*"Pooled FB102 treatment significantly reduced selected symptoms through Week 1"* No stats reported

Note: Data reflect cross-trial comparisons and not head-to-head studies. Caution should be used when comparing data due to differences in trial designs, participant characteristics and endpoint definitions.

First Tracks BIOTHERAPEUTICS

1. Teva Phase 2a celiac results reported Sept. 2, 2026; 2. Forte Biosciences Phase 1b results reported June 23, 2025; 3. Argenx Phase 2a results reported Oct. 8, 2026; Δ = Delta

Cohort 2 (Vh:Cd ≤ 2.5) assesses ability to heal mucosal damage in patients with a broad range of histologic and symptom severity

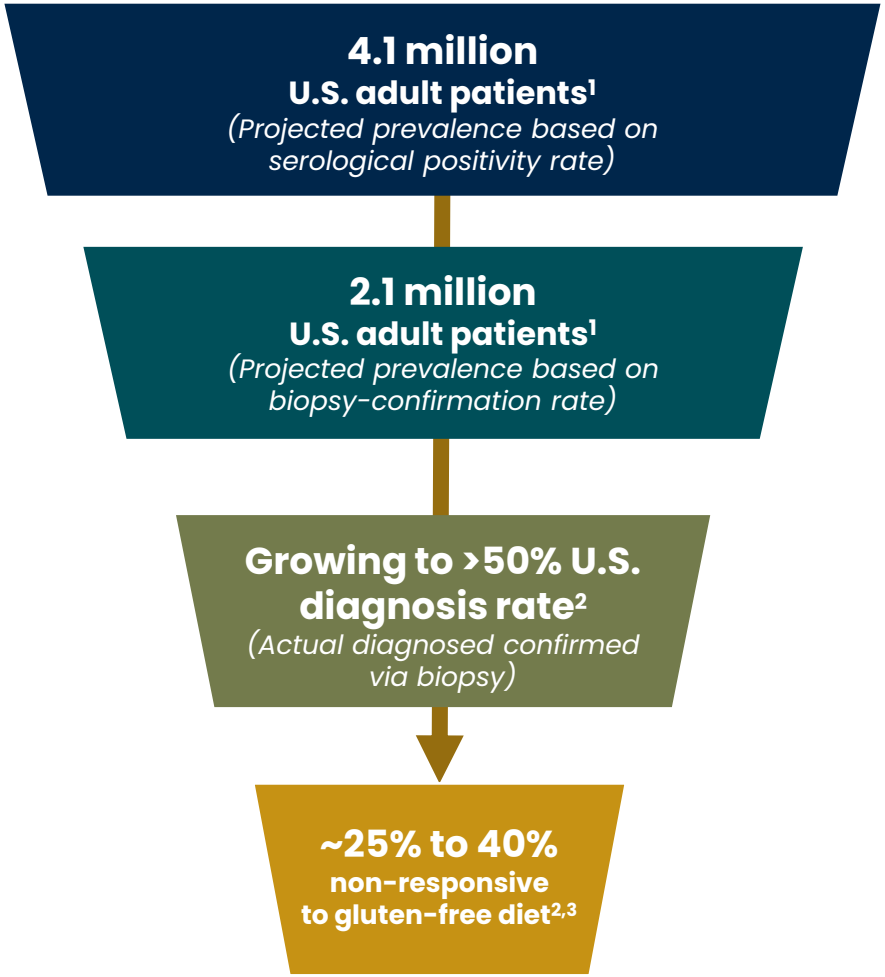


Persistent evidence of histologic CeD activity (Vh:Cd ≤ 2.5)

- Broad range of histologic (Vh:Cd ratio ≤ 2.5) and symptom severity
- 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

Potential blockbuster opportunity for ANB033 in non-responsive CeD



High disease burden

- Debilitating symptoms and QoL
- Increased risk of developing other autoimmune disorders
- Disease awareness driving growth
- No approved therapies

CD122s differentiated from other Tx in development

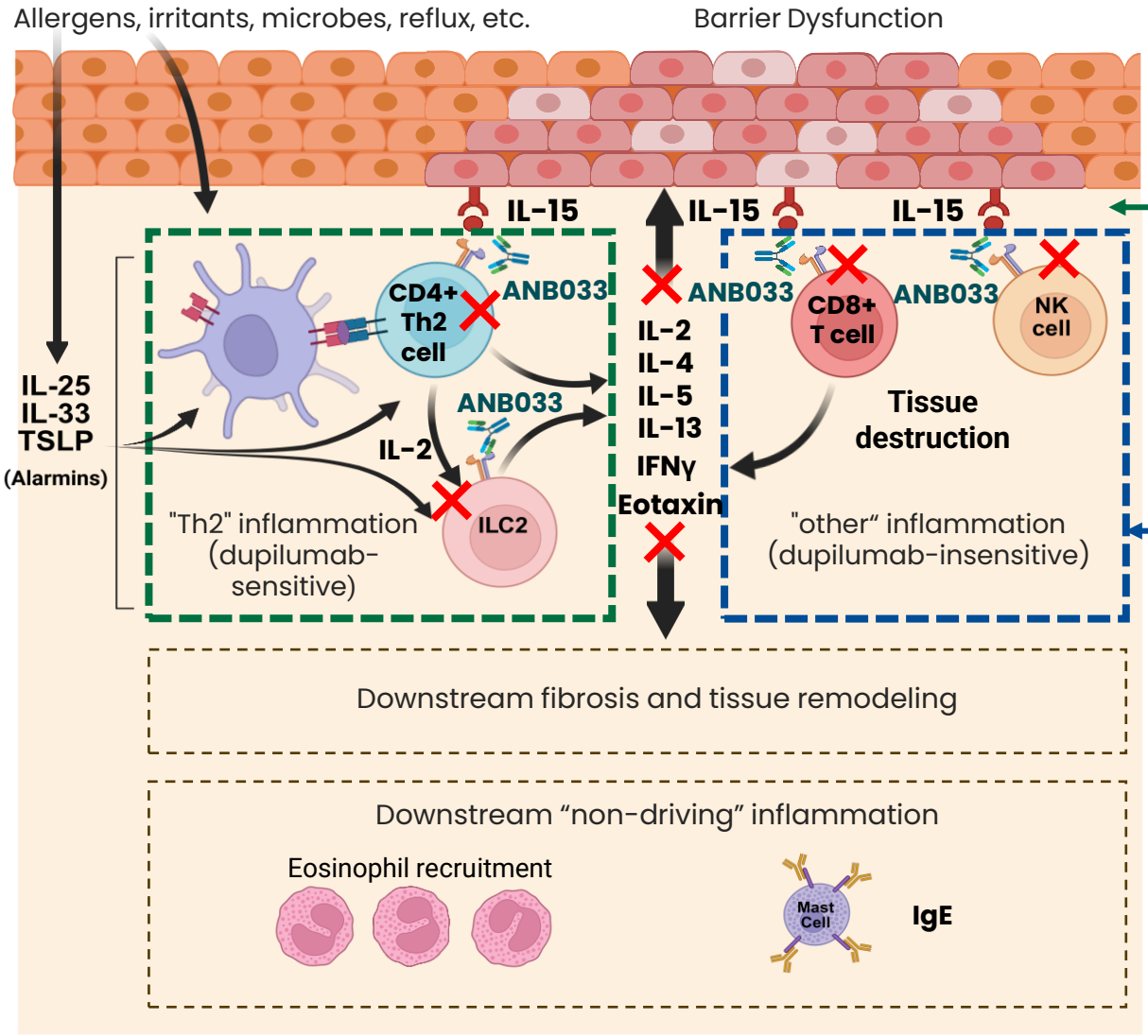
- HCPs favor MOA that targets both symptoms and histology

>$20B+ U.S. market in patients non-responsive to gluten-free diet

- Expect reimbursement with limited utilization management

1. King et al. (2020), 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). 3. Lebwohl et al. Presented at DDW (2026); Percent of CeD non-responders to gluten free diet whose symptoms are not well controlled with or without villous atrophy.

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



CD4+ and ILC2 (dupilumab-sensitive)

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

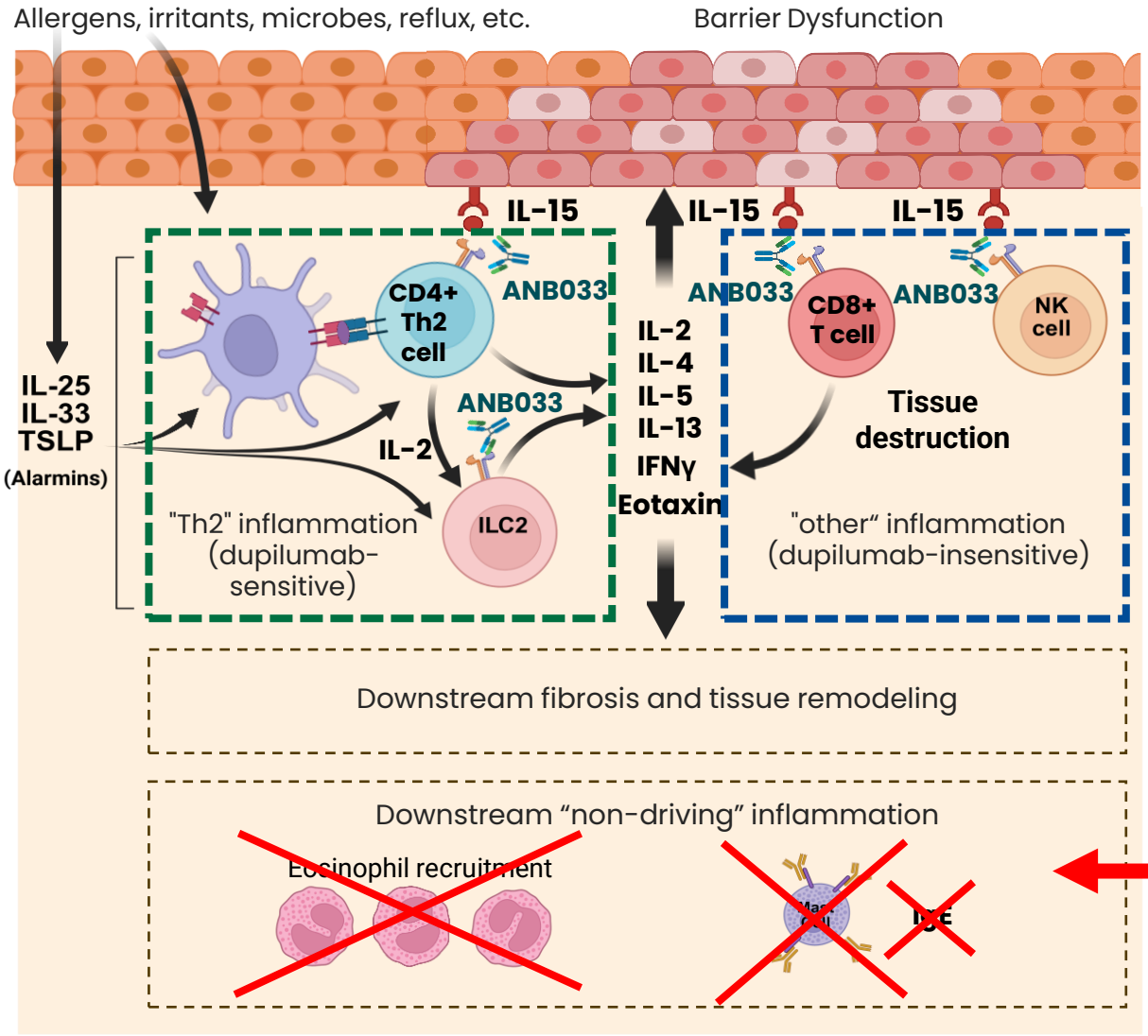
CD8+ and NK (dupilumab-insensitive)

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

Upstream pathways lead to downstream eosinophilic infiltration into tissue

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

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



Approved

sanofi

Dupilumab (anti-IL-4Ra)

Has PoC

NOVARTIS Calypso biotech

GIA-632 (anti-IL-15)

AMGEN AstraZeneca

tezepelumab (anti-TSLP)

Failed mechanisms

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

Celldex AstraZeneca

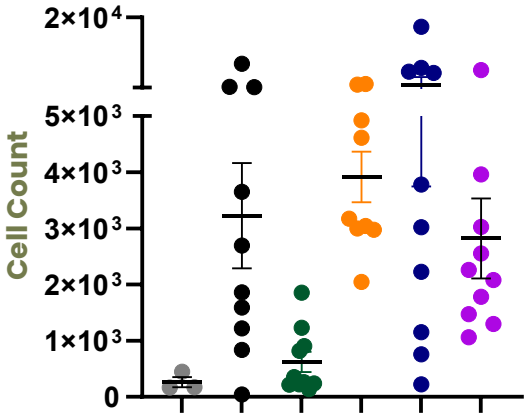
First Tracks BIOTHERAPEUTICS

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

ANB033 prevents eosinophilia by targeting upstream inflammation

Aspergillus-induced eosinophilia

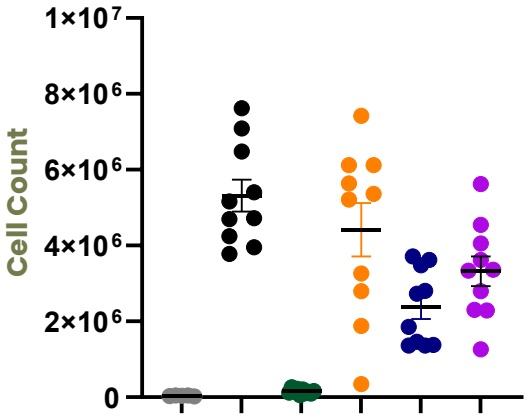
Esophageal eosinophils



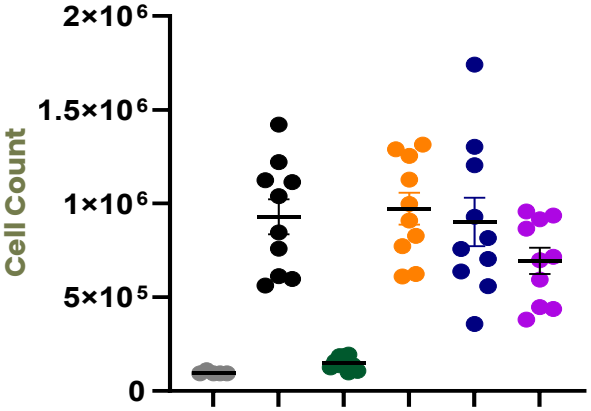
Treatment

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

Lung eosinophils

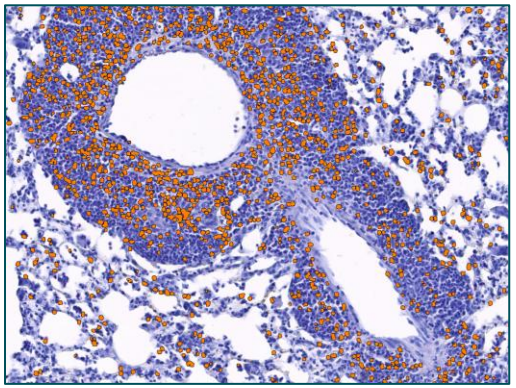


Lung ILC2s

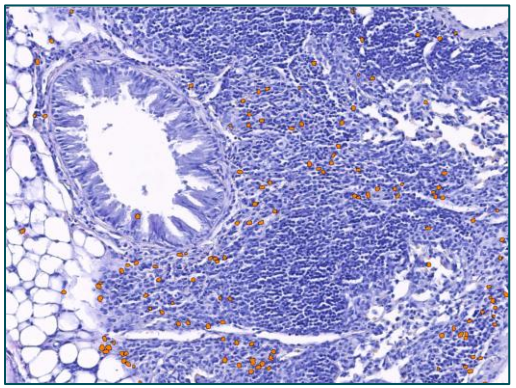


Eos observed in histology

Isotype Control



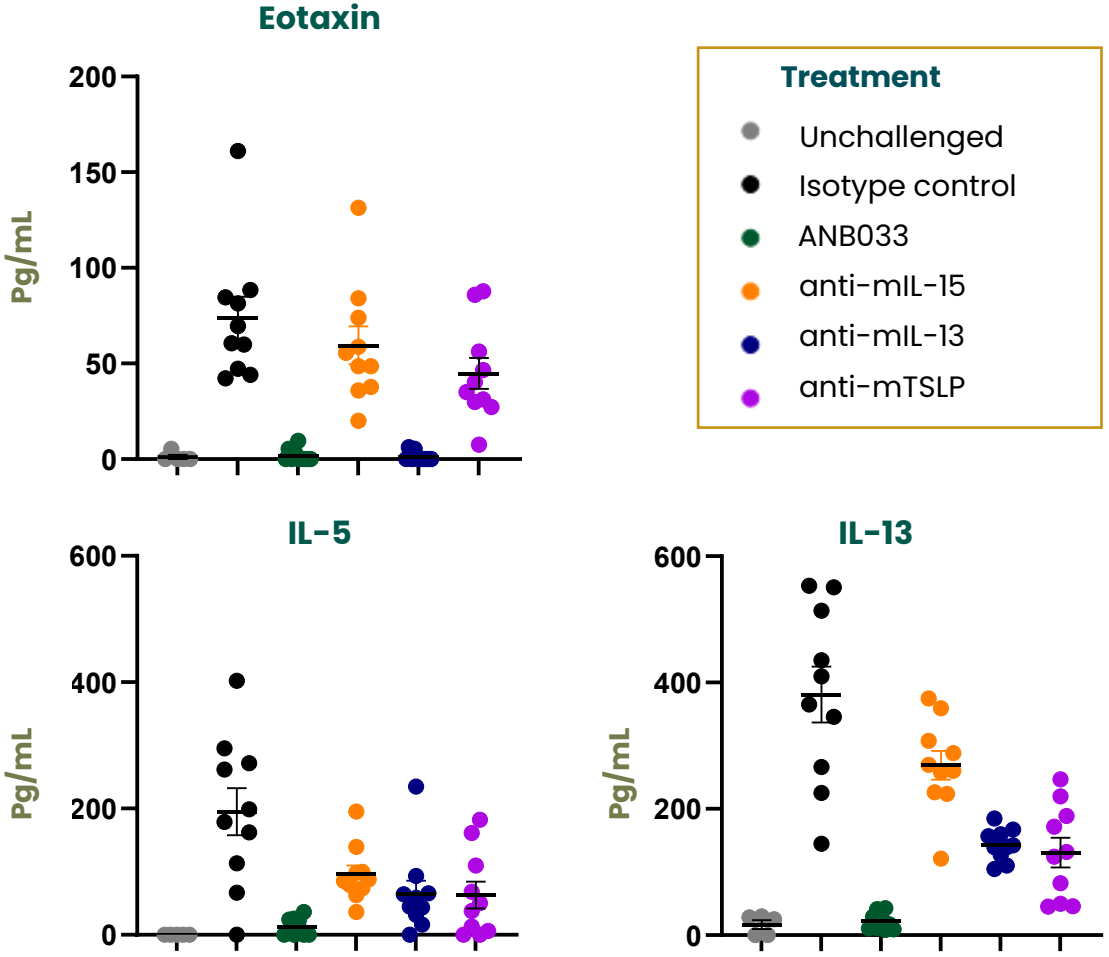
ANB033



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



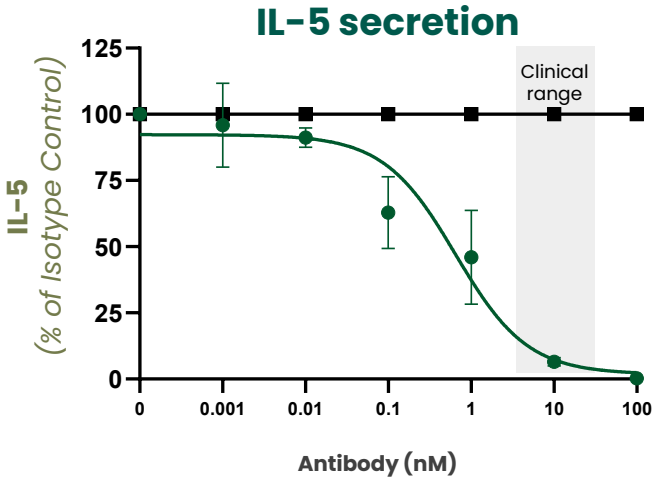
ANB033 prevents –

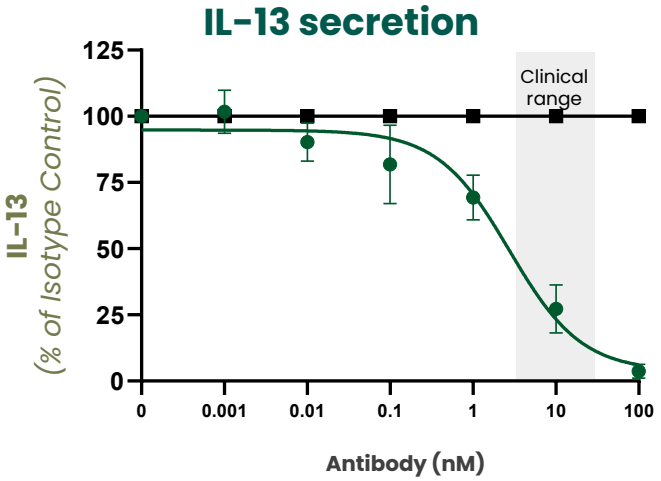
- Cytokine secretion (IL-5/IL-13) responsible for eos expansion
- Chemokine secretion (eotaxin) responsible for eos recruitment

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).

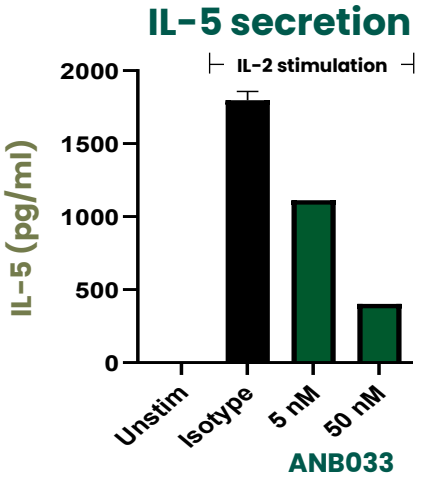
ANB033 reduces CD4+ T cell and ILC2 derived Th2 cytokines, proven drivers of EoE pathology

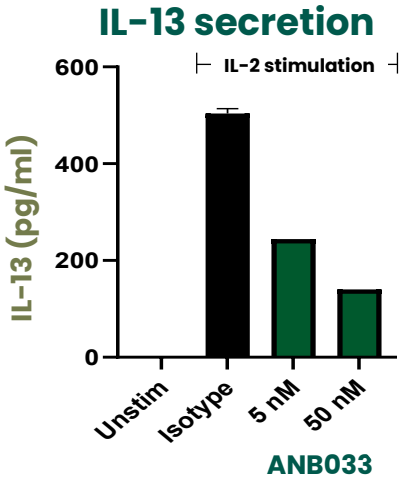
PBMC





ILC2



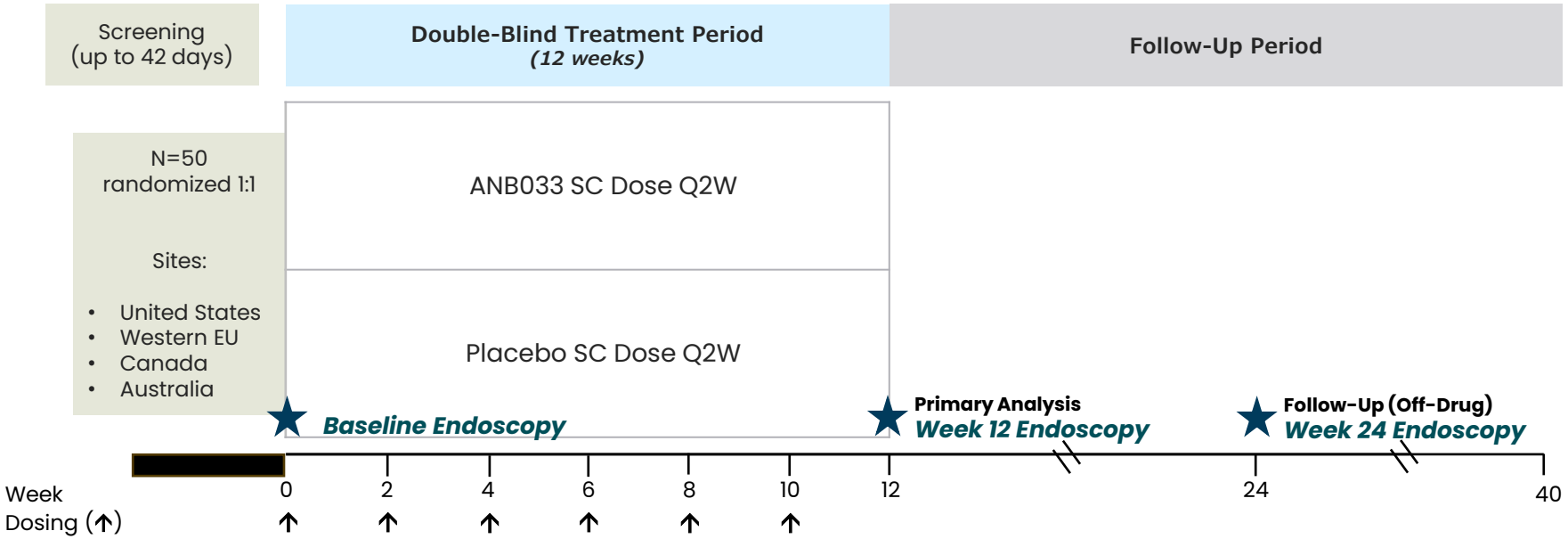


ANB033 Isotype Control

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.

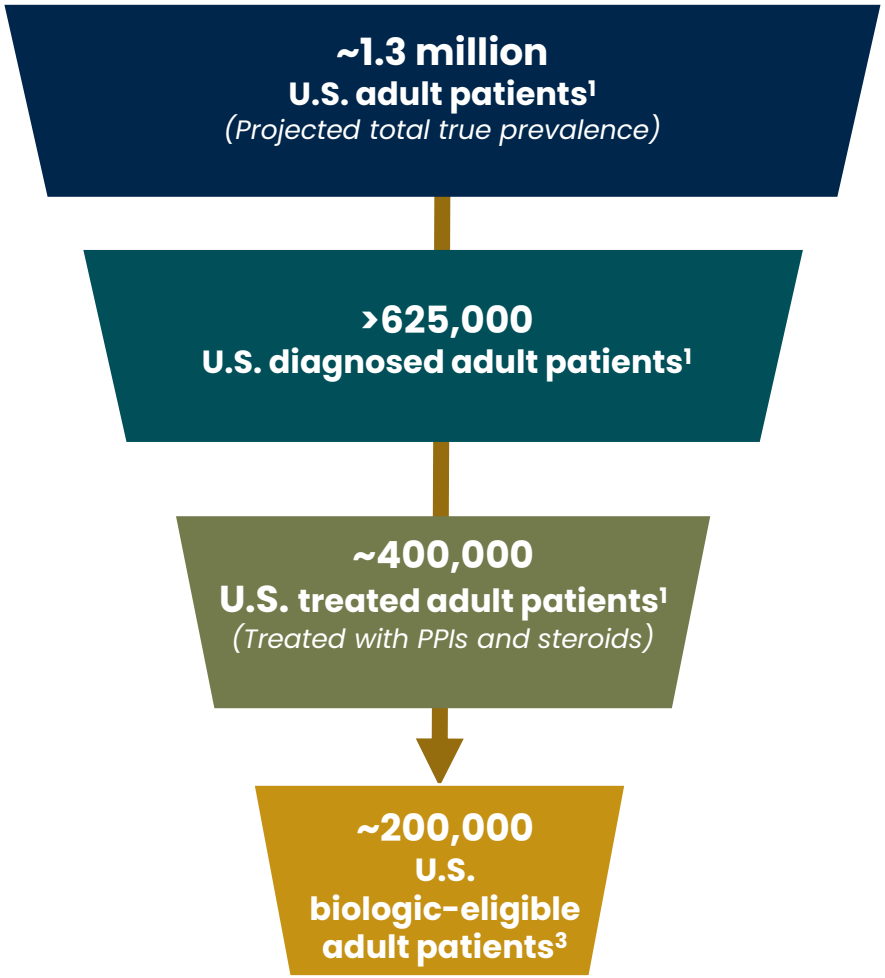
ANB033 Phase 1b trial in EoE ongoing

Anticipate top-line data in Q3 2027



Population	Adult participants with EoE, including dupilumab-experience (<25% of trial) Peak eosinophil count ≥15/hpf at the screening endoscopy and with symptomatic dysphagia
Safety	Safety and tolerability
Clinical PK	PK and immunogenicity
Efficacy	• Peak esophageal intraepithelial eosinophilic count (eos/hpf) • Change from baseline in dysphagia symptom questionnaire (DSQ)
Biomarkers	Characterize ANB033 effects on circulating biomarkers, including robust translational plan

EoE is a significant market with increasing prevalence and unmet need



Increasing disease recognition with >10% CAGR[1,2]

- Heightened rates of endoscopic procedures and biopsies

Significant unmet need with emerging therapies gaining approval

- ~50% PPI or steroid non-responsive or intolerant
- Dupixent QW: approved 2022 as first biologic
- ~30%+ Dupixent non-responsive and non-compliant

>$15B+ U.S. market

- Dupixent currently on ~$3B annualized run-rate in EoE (>60,000 patients on drug)[1]
- New MOAs expected to expand 1st line use and accelerate 2L+ market opportunity
- Potential for IBD biologic penetration given additional therapeutic options

First Tracks BIOTHERAPEUTICS

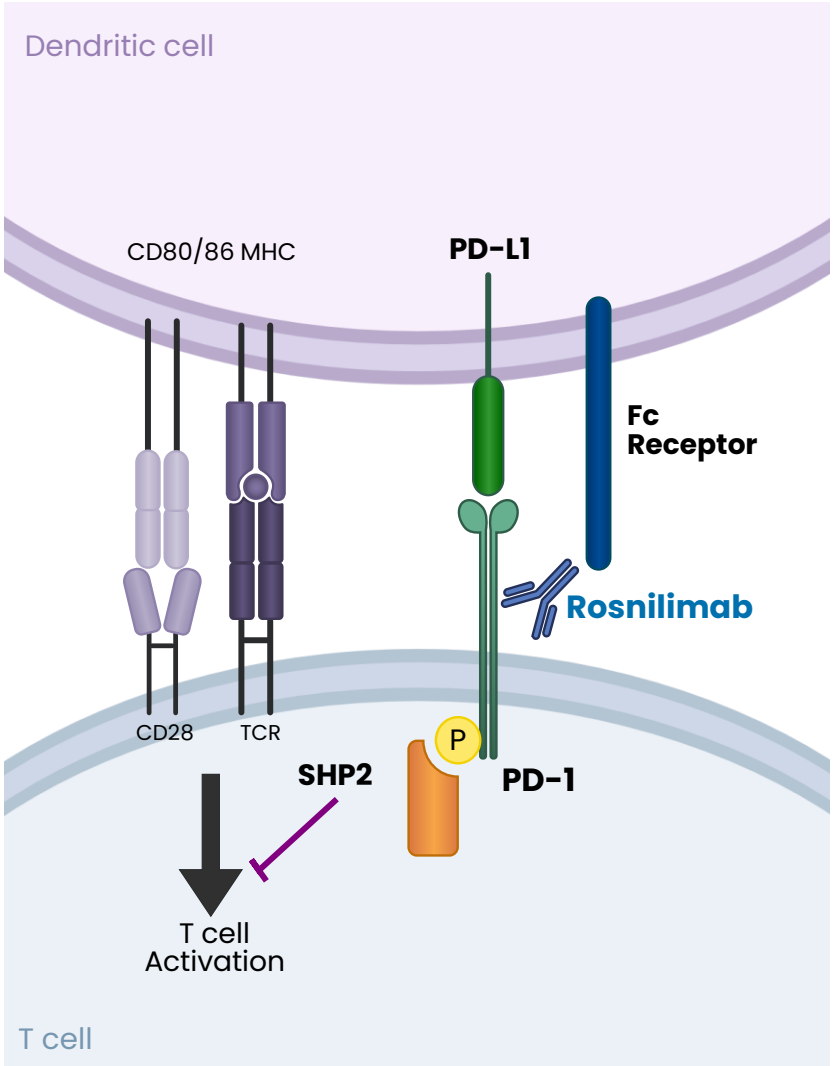
1. ZS Associates claims analysis Q1 2026; 2. "Prevalence and costs of eosinophilic esophagitis in the United States" Thel H et al., 2025 Clinical Gastroenterology and Hepatology). 14% CAGR from 2009-2022; 3. Suboptimally treated by PPI or steroids



Rosnilimab

Pathogenic T cell depleter

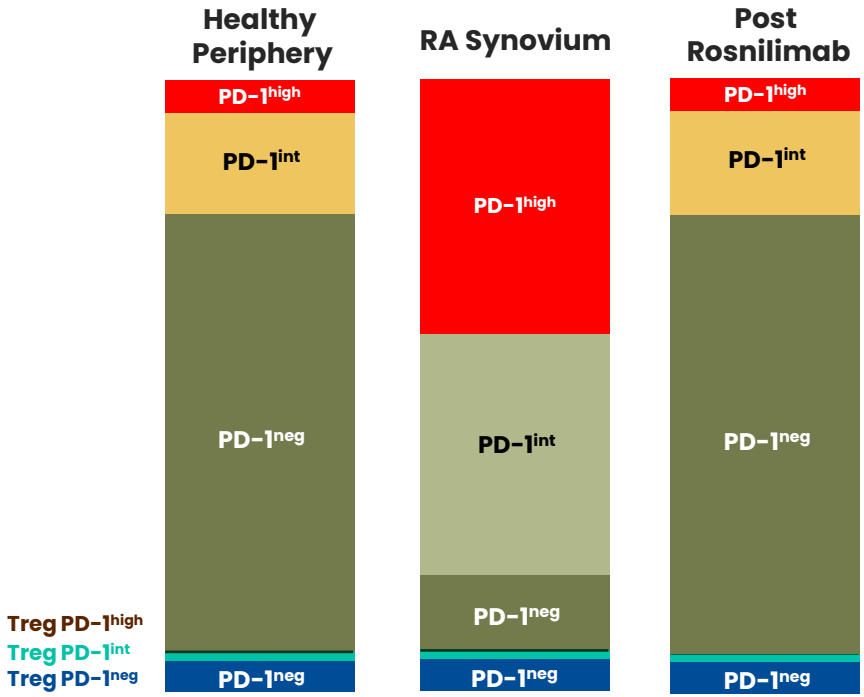
Rosnilimab selectively targets pathogenic T cells in periphery and inflamed tissue to restore immune homeostasis



Rosnilimab aims to:

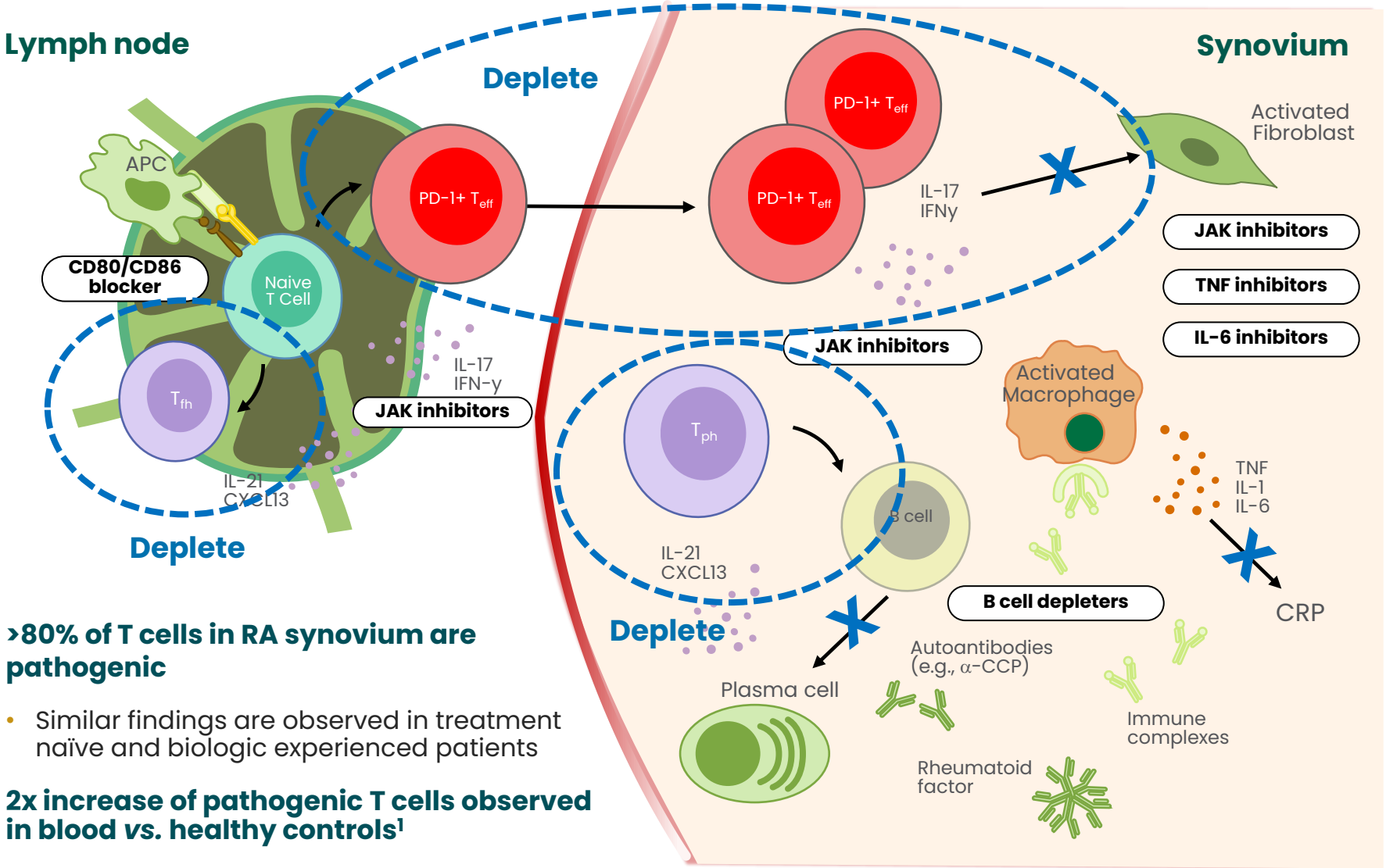
1. Leverage natural immune regulatory pathway to safely restore immune homeostasis
2. Achieve durable remission and modify disease

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.

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



>80% of T cells in RA synovium are pathogenic

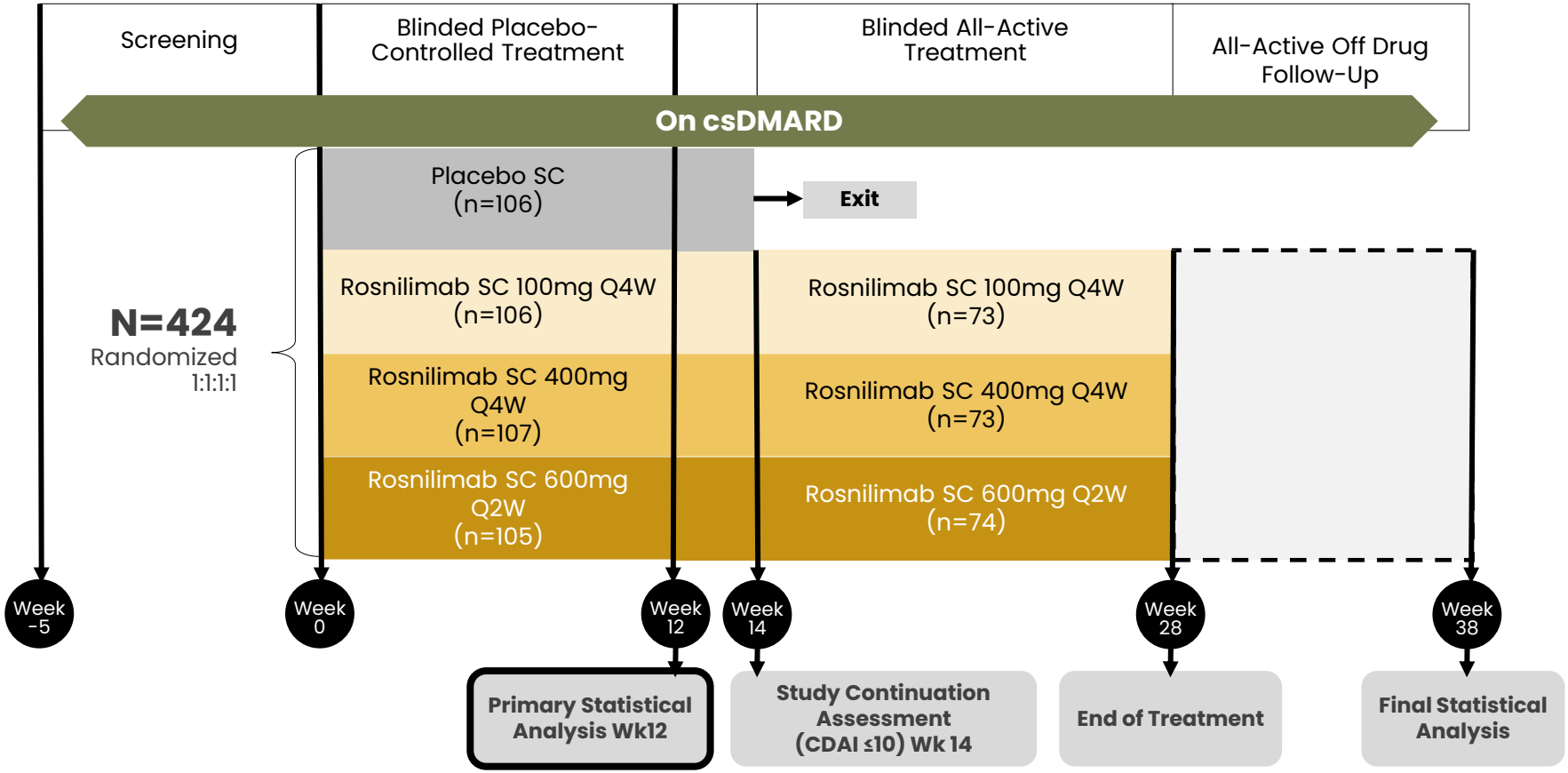
- Similar findings are observed in treatment naïve and biologic experienced patients

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



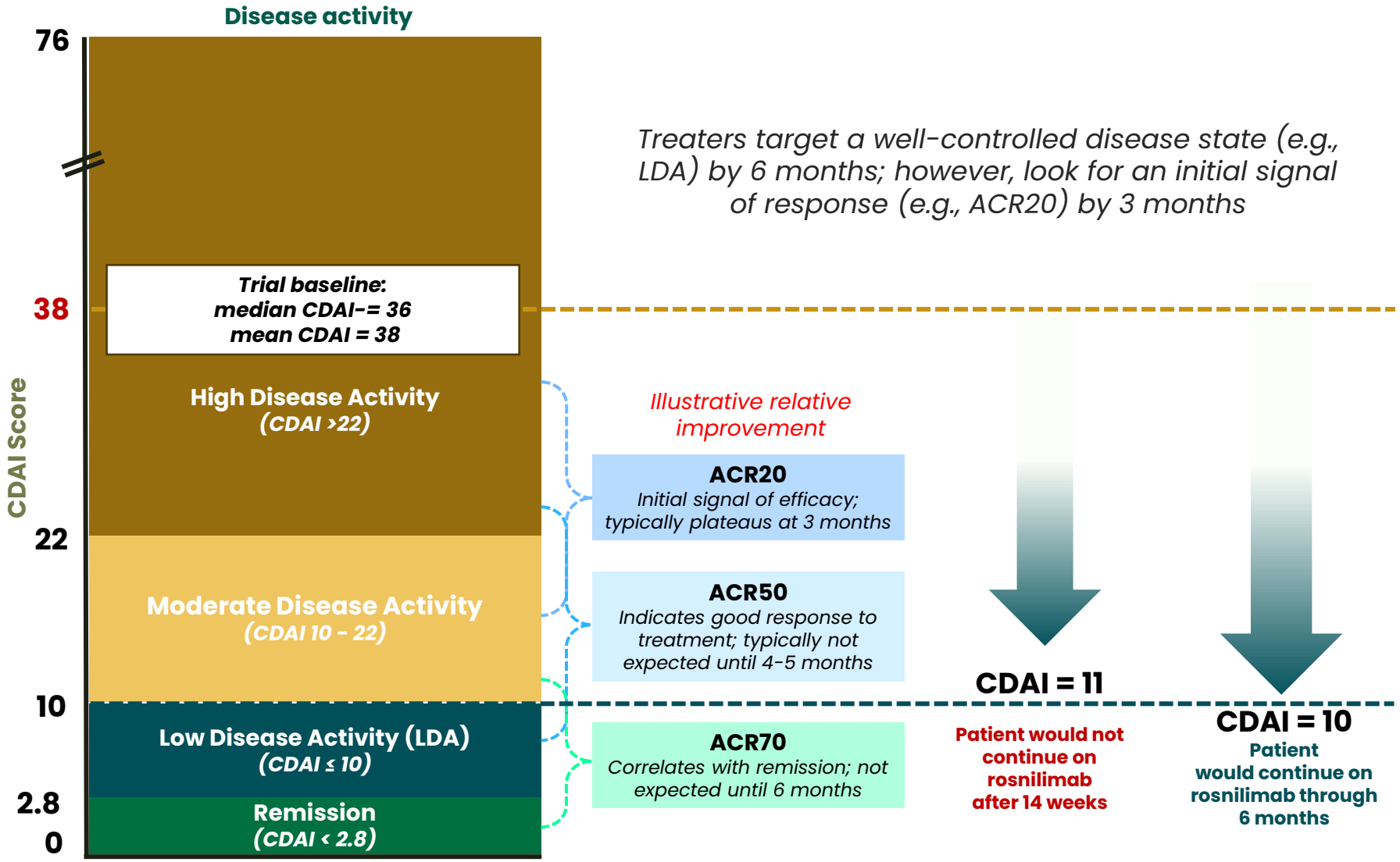
Key Inclusion Criteria – Seropositive RA, ≥6 swollen and ≥6 tender joints, hs-CRP ≥ 3mg/L during Screening, Concurrent use of 1 or 2 csDMARDs that were initiated at least 3 months before screening

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



First Tracks BIOTHERAPEUTICS

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)

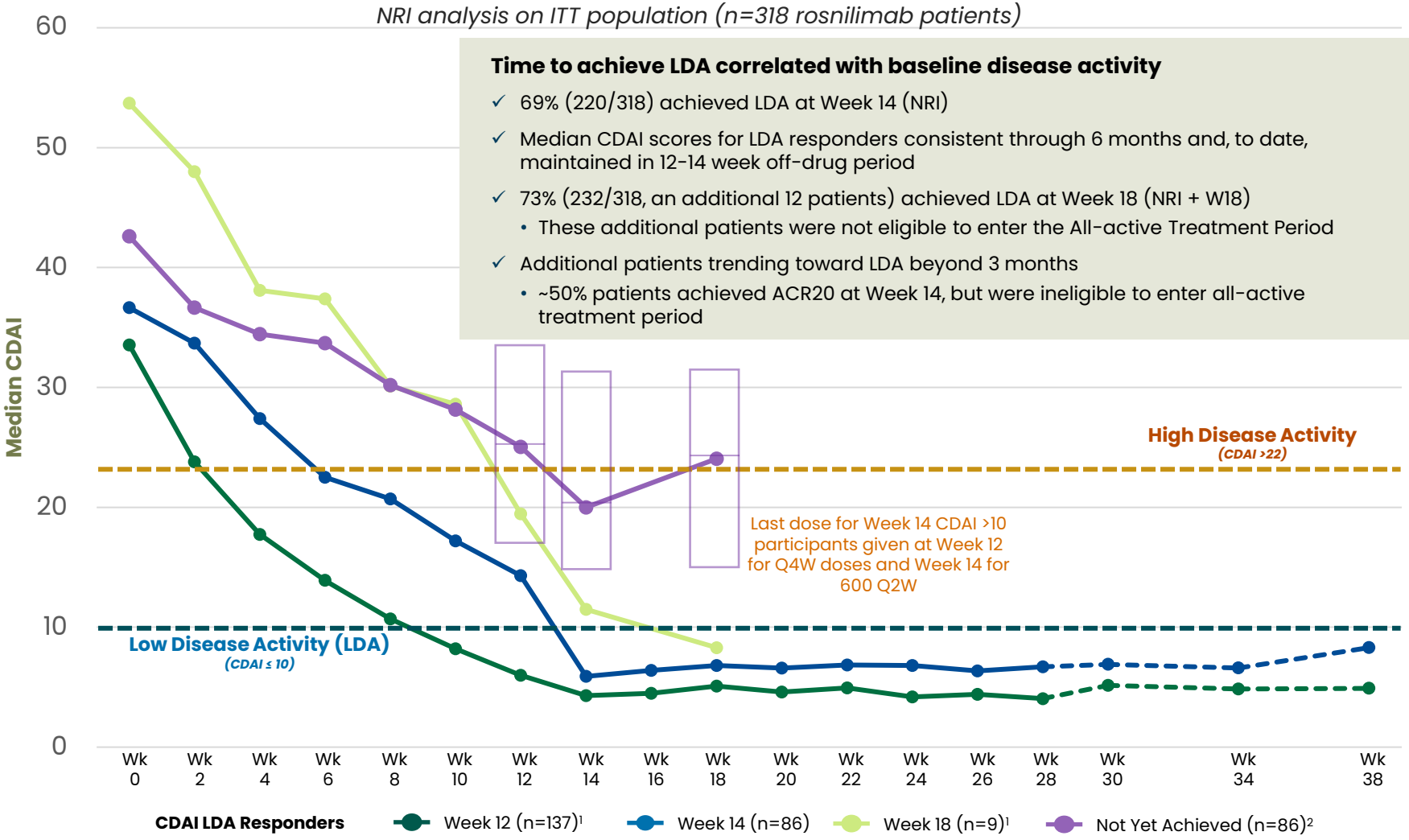
Rosnilimab, a pathogenic T cell depleter, is well-positioned for the ~$20 billion U.S. RA market which hasn't had a new mechanism approved since 2012

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)



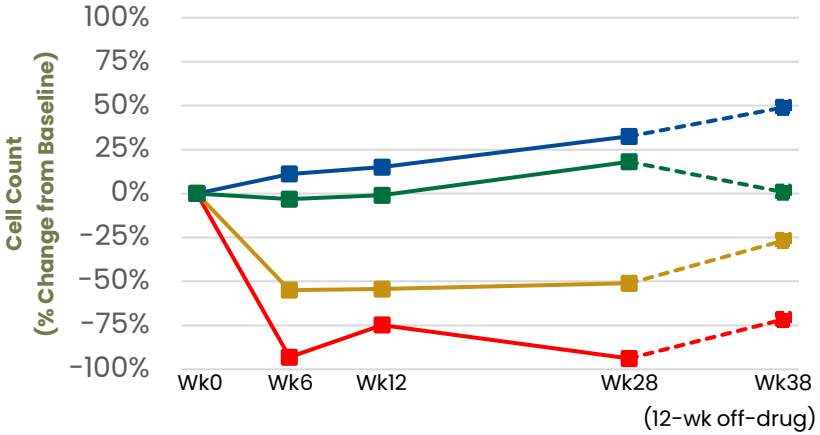
Time to achieve LDA correlated with baseline disease activity

- ✓ 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

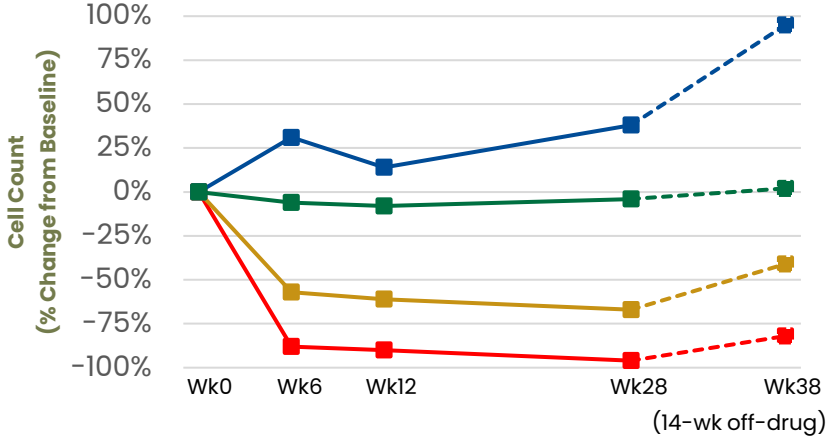
1. Green line includes 3 patients that achieved LDA at Week 12, were not CDAI LDA at Week 14, but returned to CDAI LDA at Week 18. These same 3 patients were excluded from the Light Blue line. In total 12 patients achieved CDAI LDA at Week 18. 2. Purple line includes rosnilimab patients that discontinued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

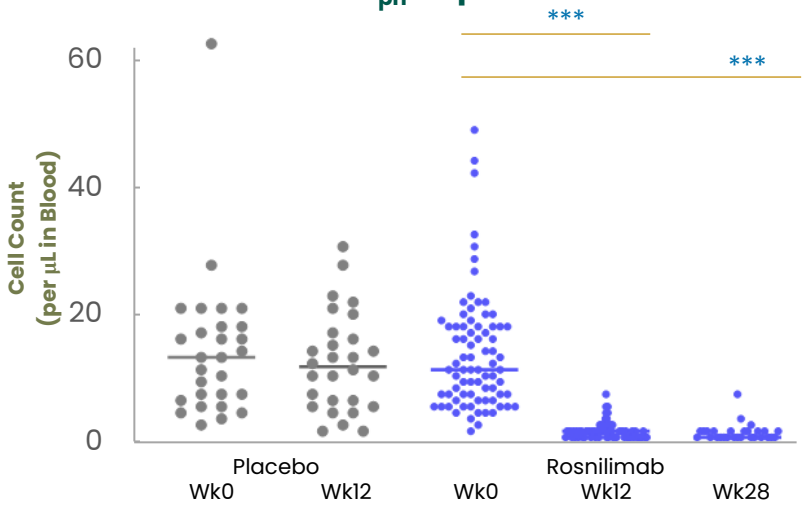
Rosnilimab 400mg Q4W T Cell Impact



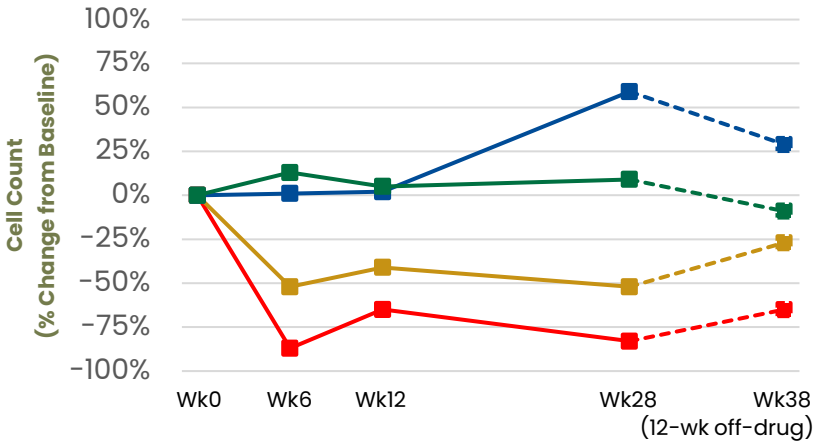
Rosnilimab 600mg Q2W T Cell Impact



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.

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
  - 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



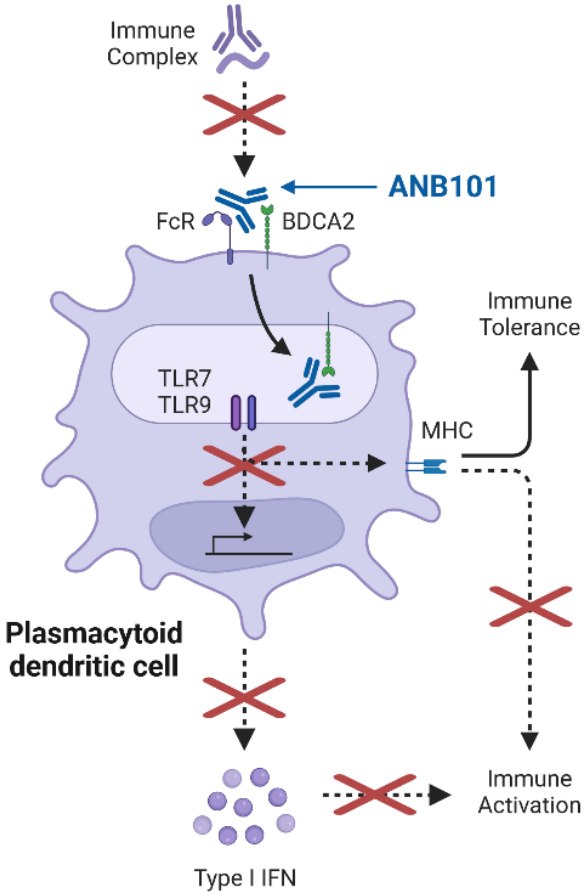
ANB101

BDCA2 modulator

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

Phase 1 trial completed 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

Currently evaluating potential development approach informed by clinical and translational evidence, as well as competitive registrational data in SLE expected in Q4 2026 and CLE expected in early 2027