Corporate Overview

January 2025

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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 rosnilimab's Phase 2b clinical trial in rheumatoid arthritis and Phase 2 clinical trial in ulcerative colitis; the timing of initiation of ANB101's Phase 1 clinical trial; 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; the Company's ability to find a licensing partner for imsidolimab or etokimab and the timing of any such transaction; and the Company's projected cash runway. Statements including words such as "plan," "continue," "expect," or "ongoing" and statements in the future tense are forward-looking statements. These forward-looking statements involve risks and uncertainties, as well as assumptions, which, if they do not fully materialize or prove incorrect, could cause its results to differ materially from those expressed or implied by such forward-looking statements. Forward-looking statements are subject to risks and uncertainties that may cause the company's actual activities or results to differ significantly from those expressed in any forward-looking statement, including risks and uncertainties related to the company's ability to advance its product candidates, obtain regulatory approval of and ultimately commercialize its product candidates, the timing and results of preclinical and clinical trials, the company's ability to fund development activities and achieve development goals, the company's ability to protect intellectual property and other risks and uncertainties described under the heading "Risk Factors" in documents the company files from time to time with the Securities and Exchange Commission. These forward-looking statements speak only as of the date of this presentation, and the company undertakes no obligation to revise or update any forward-looking statements to reflect events or circumstances after the date hereof.

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Best-in-class immune cell modulating antibodies

Immune Cell Modulators

Rosnilimab
(PD-1 depleter and agonist)

P2b in Rheumatoid Arthritis

P2 in Ulcerative Colitis

ANB033
(CD122 antagonist)

P1 in Healthy Volunteers

ANB101
(BDCA2 modulator)

IND Submitted

Autoimmune and inflammatory diseases including dermatology, gastroenterology and rheumatology

Cytokine Antagonists
(legacy programs for out-licensing)

Imsidolimab
(IL-36R)

Positive P3 data reported in GPP[1]

Etokimab
(IL-33)

P2b/3-ready in epithelial driven diseases

Research and Capital

Research-driven

- Preclinical pipeline of immunology targets

Strong capital position

- YE 2024 cash: ~$420MM
- Expected cash runway: YE 2027
  - Excludes GSK royalty and milestone potential for *Jemperli* and cobolimab
  - Excludes GSK $75MM milestone for *Jemperli* $1B annual WW sales

1. GPP – Generalized pustular psoriasis

Multiple clinical-stage data events

Rosnilimab RA P2b Week 12 data expected in February 2025

	Antibody Program	Therapeutic Indication	Development Stage and Anticipated Milestones: IND Enabling / Phase 1 / Phase 2 / Phase 3
Immune Cell Modulators	Rosnilimab (PD-1 depleter and agonist)	Rheumatoid Arthritis	Top-line P2b Week 12 data – February 2025; Top-line P2b Week 28 data – Q2 2025
		Ulcerative Colitis	Top-line P2 data Q1 2026
	ANB033 (CD122 antagonist)	Inflammatory Diseases	P1 initiated; R&D event in 2025
	ANB101 (BDCA2 modulator)	Inflammatory Diseases	P1 initiation Q1 2025

Rosnilimab

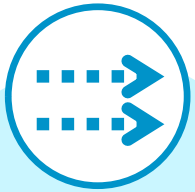
(PD-1 Depleter and Agonist)

Rosnilimab: PD-1+ T cell depleter and agonist

PD-1
Validated target

- PoC in RA via PD-1+ T cell depletion MOA
  - —LLY's peresolimab in Phase 2a has "modest ADCC activity"[1]
- PD-1 polymorphisms associated with increased risk of developing RA[2]
- Inflammatory arthritis common AE of PD-1 antagonist treatment[3]

Rosnilimab
Best-in-class antibody



- Potent depleter and agonist
  - —Deplete 90% of PD-1^{high} T cells
- Safety/Tolerability
  - —Clean tox profile; no DLT reached
  - —Benign AE profile in both Phase 1 (HV) and Phase 2 (Alopecia 6-months of dosing)

RA Phase 2b trial
Robust and well-controlled

- ~420 patient US + EU study (~40% b/tsDMARD-experience)
- Patients have high disease activity; RF or α-CCP sero-positive
- >80% power for ACR50 composite at Week 12
- CDAI LDA responders treated through Week 28
- CRO with extensive RA experience; no rescue tx

Rosnilimab RA top-line Phase 2b data:
Week 12 – February 2025; Week 28 – Q2 2025

1. Eli Lilly patents; WO2024196694A2 and WO2024040206A2 2. Liu et al., Int J Genomics, 2024 3. Canavan et al., BMC Rheumatology, 2021

Rosnilimab has potential to treat wide range of systemic inflammatory diseases, including RA and UC

Rheumatoid arthritis:

- **~500,000 U.S. patients**
  >$10bn U.S. sales in "bio-experienced" market[1]
- **20-25% cycle** through all treatment classes and do not achieve low disease activity[2]

Ulcerative colitis:

- **~100,000 U.S. patients**
  >$6.5bn U.S. sales, excluding TNF, market[3]
- **1/3 to 1/2 relapse**
  within 1 year following remission on induction therapy[4]

Large commercial markets

- Biologic experienced patients

SOC is insufficient and fragmented

- RA (bio-experienced): ~20–30% ACR50
- UC: ~25-30% induction of clinical remission

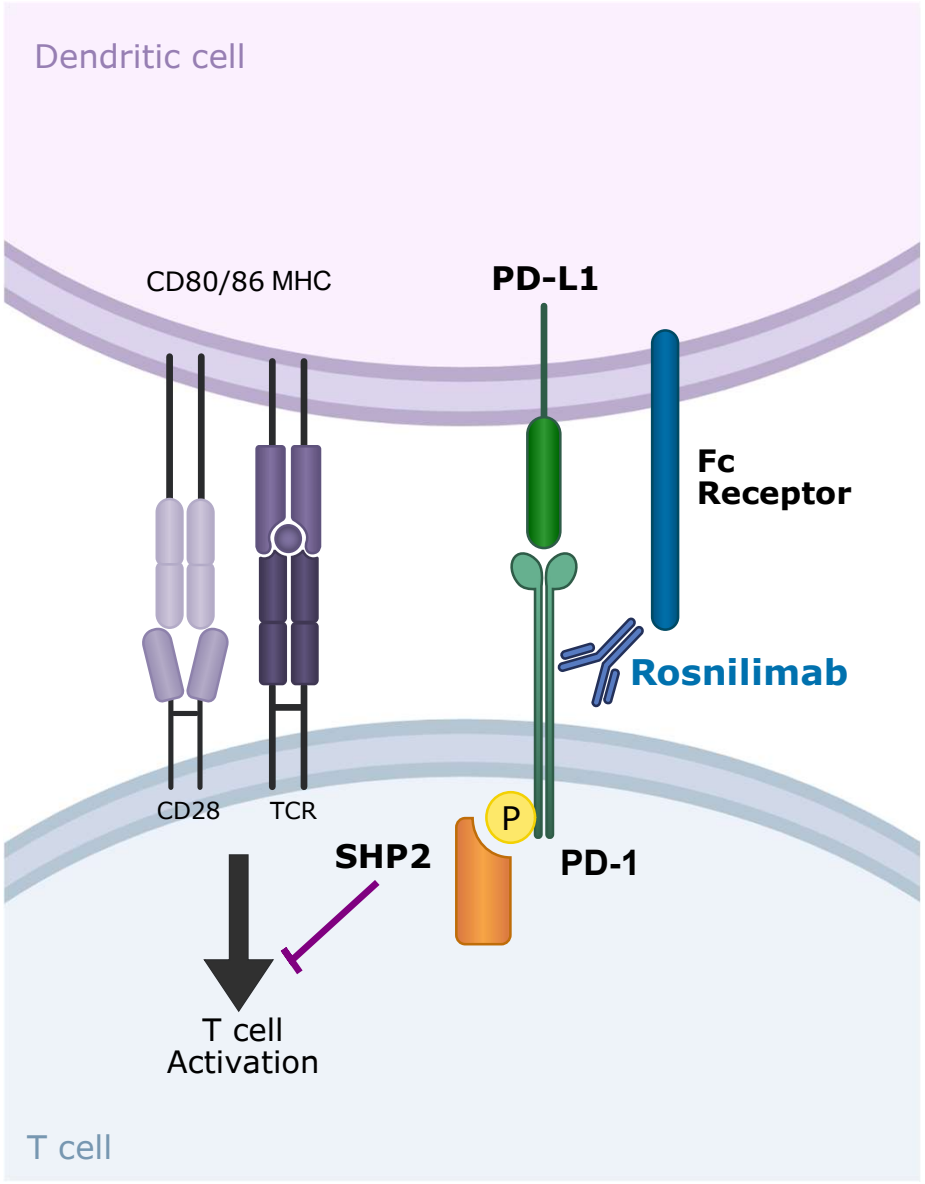
Significant room to differentiate

Drive deeper responses across broader patient population

Restore immune homeostasis

1. Expected by 2028 (Evaluate 29 Nov 2022); 2. Market research conducted by Ambit in 2022; 3. Expected by 2028 (Evaluate 21 Aug 2023); 4. Phase 3 registrational data from product labels.

Rosnilimab selectively targets activated PD-1+ T cells in the periphery and inflamed tissue



Rosnilimab aims to:

1. Rapidly engage homeostatic mechanisms to induce clinical response
2. Achieve durable remission through histologic normalization

Immune Cells Impacted	Mechanism	Immunologic Outcome
PD-1^{high} T_{fh}/T_{ph}	depletes ↓	*downstream effect on B cells* Plasma cell generation Autoantibody levels
PD-1^{high} T_{eff}	depletes ↓	Cytokine secretion T cell migration T cell proliferation
PD-1+ T_{eff}	agonizes ↓	Cytokine secretion T cell migration T cell proliferation

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

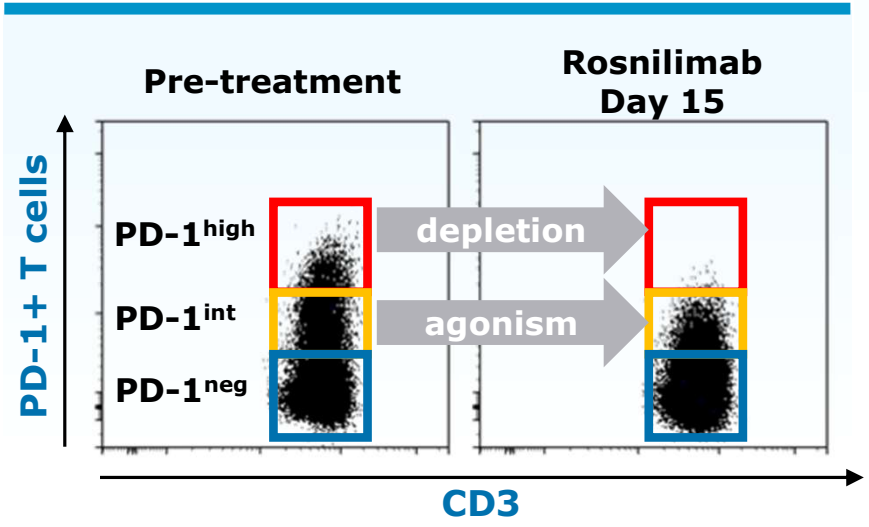
Rosnilimab is designed to bring the immune system back to homeostasis and modify disease

Rosnilimab preferentially targets activated T cells

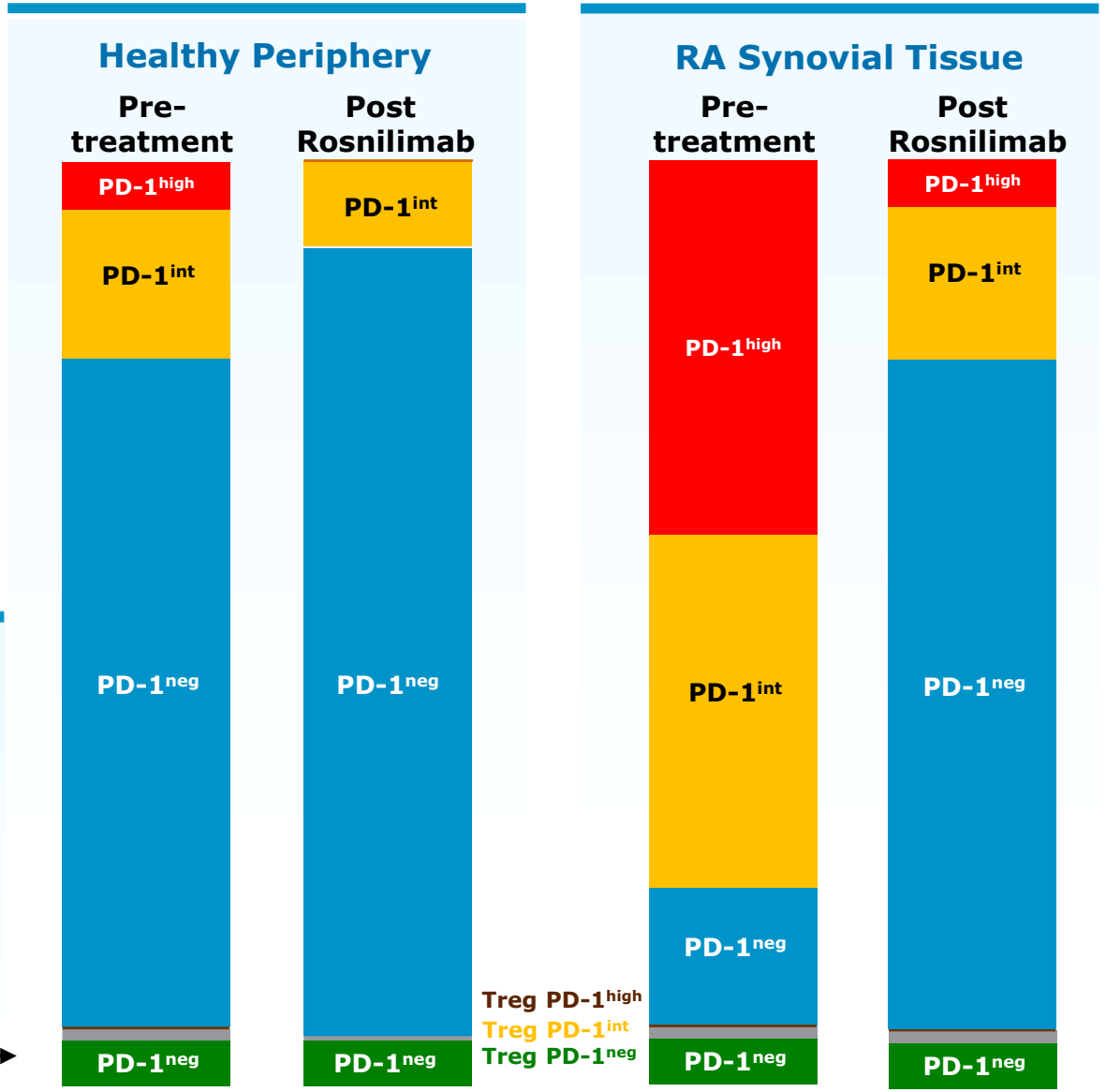
Leverages natural immune regulatory pathway to safely restore immune homeostasis

In healthy volunteers:

- Deplete PD-1^{high} T cells: ~5-8% of total T cells
- Agonize remaining PD-1^{int} T cells: ~15% of total T cells



Illustrative T cell composition change



Data illustrative; Luu K, et al. ACR 2023. November 2023

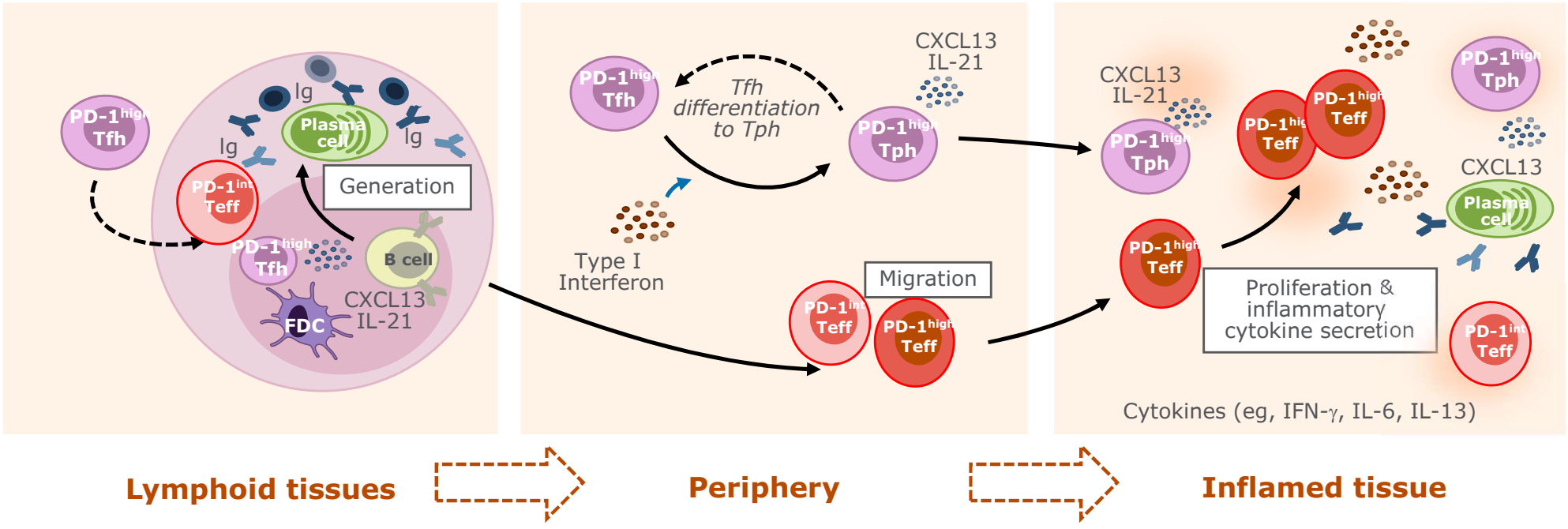
PD-1 is expressed preferentially on activated Teff and Tfh/Tph cells that mediate autoimmune pathology

Tfh (follicular helper)
Tph (peripheral helper)

- Secrete CXCL13 and IL-21 which recruit and mature B cells into "autoantibody secreting" plasma cells
- Are PD-1^{high}

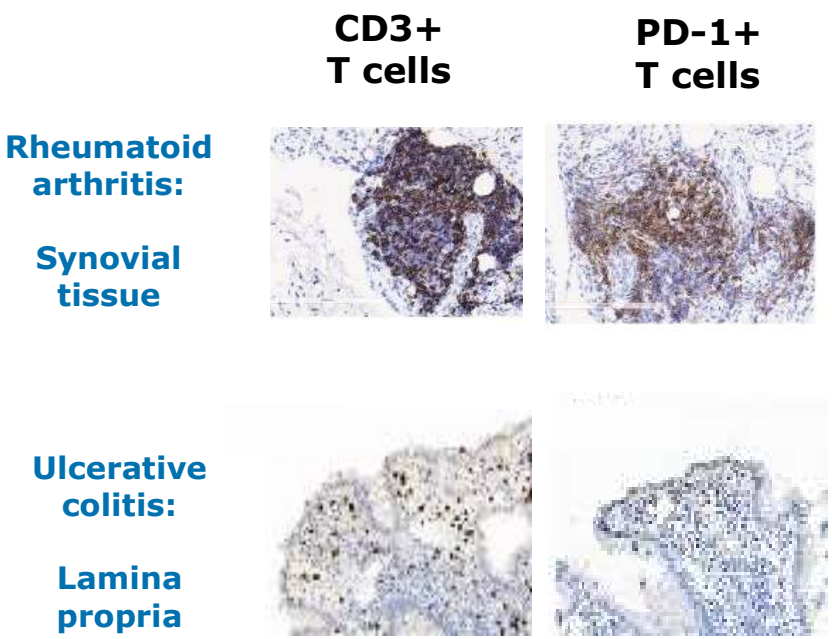
Teff (effector)

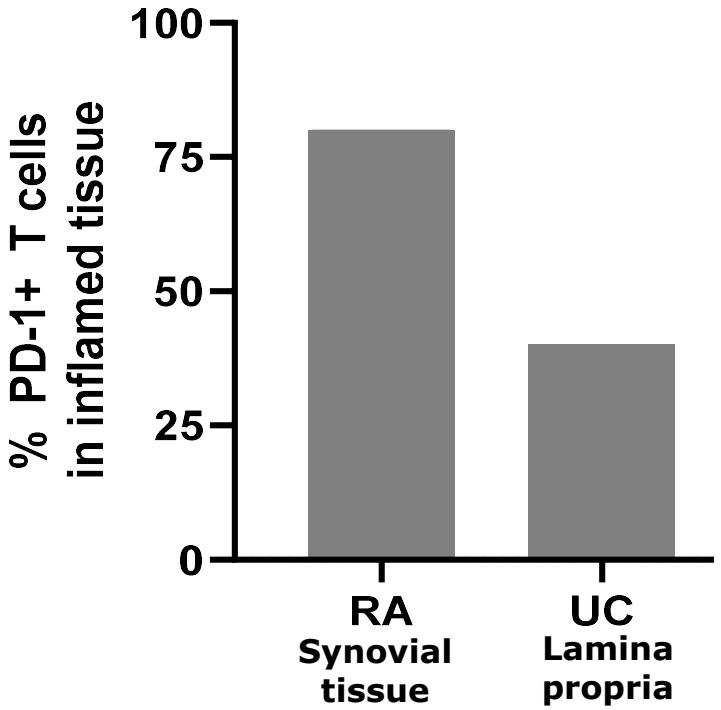
- In response to stimulation, become highly activated (PD-1^{high}) or moderately activated (PD-1^{int})
- Secrete inflammatory cytokines, cause tissue damage and perpetuate inflammatory cycle



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

PD-1+ T cells are prevalent in inflamed tissue and periphery in RA and UC



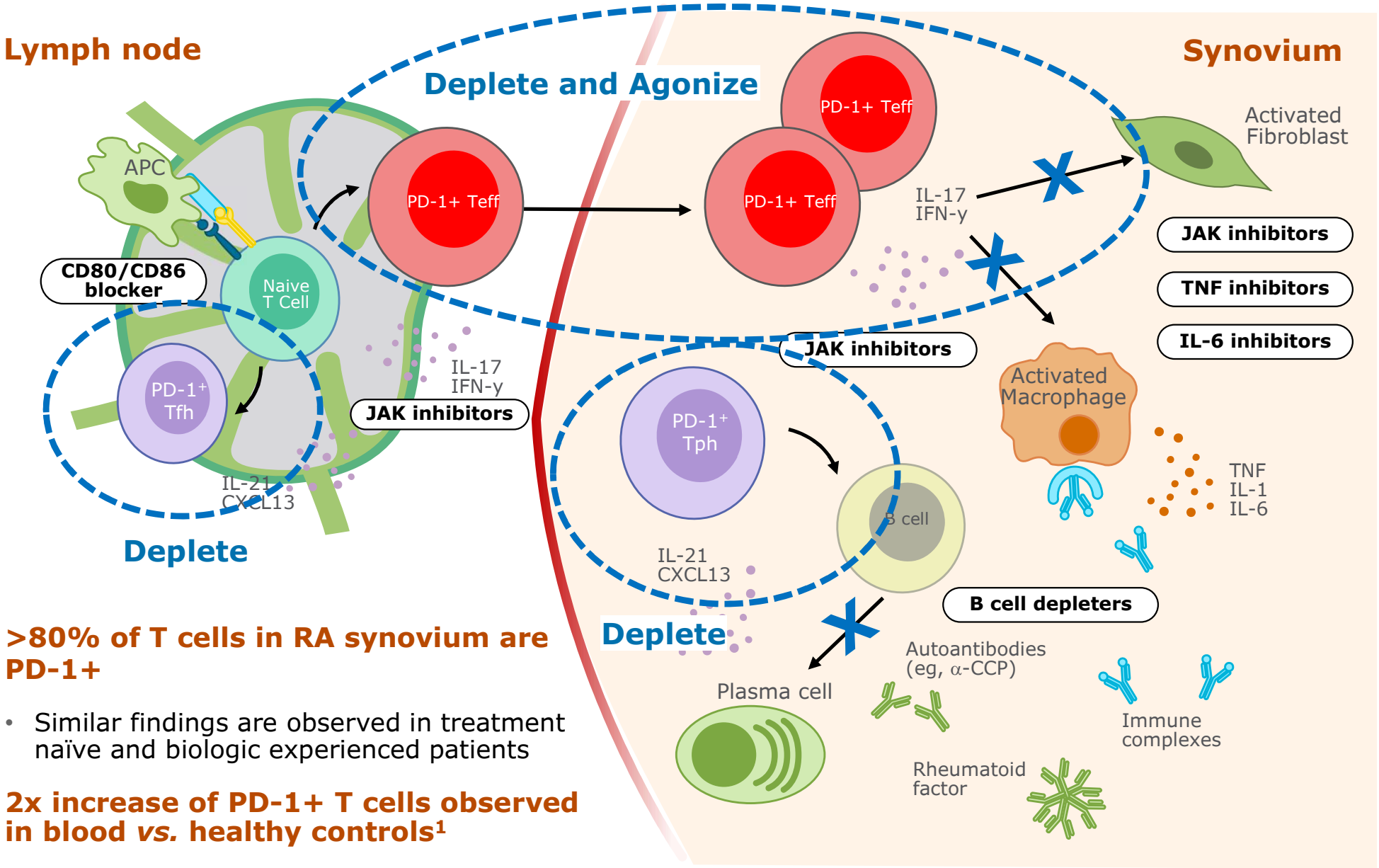


In systemic inflammatory diseases, a multiple fold increase of PD-1+ T cells is observed in periphery compared to healthy controls[1]

~2x in RA
~2x in UC

Adapted from Nguyen et al, Human Pathology (2022) 126, 19e27; Guo et al, PLoS One 2018; 13(2). Roosenboom et al, Scand J of Gastro. 2021; 56(6):671-679.
1. Chen et al, Clinical and Translational Immunology, 2024.

Reducing PD-1+ T cells broadly impacts multiple downstream, clinically validated drivers of RA pathogenesis



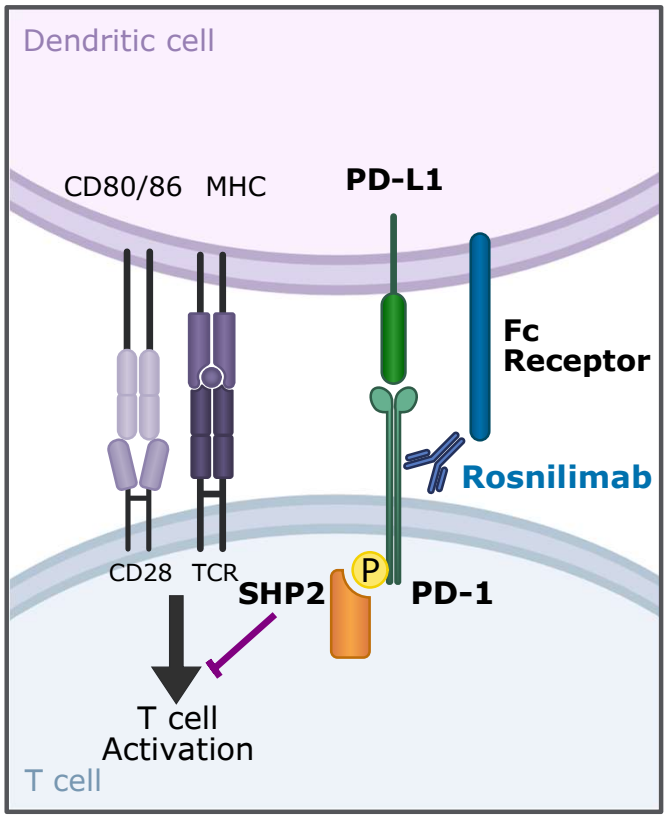
>80% of T cells in RA synovium are PD-1+

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

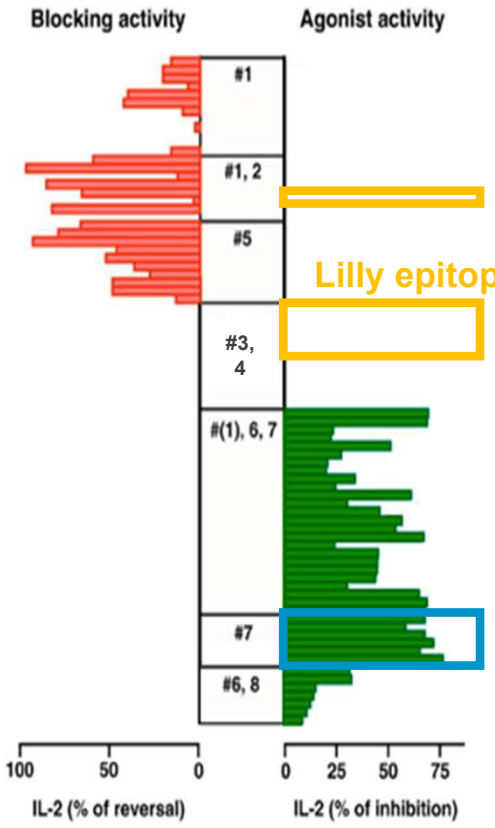
2x increase of PD-1+ T cells observed in blood *vs.* healthy controls[1]

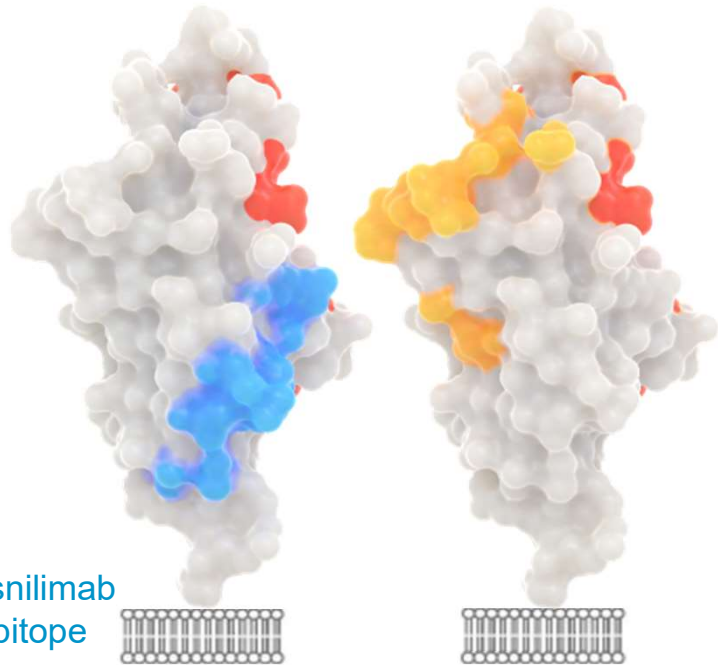
Adapted from Aletaha and Smolen, JAMA, 2018; 1. Chen et al, Clinical and Translational Immunology, 2024.

Rosnilimab optimizes PD-1+ T cell inhibitory signaling by enabling tight immune synapse formation



Functional assay of antagonism or agonism[1]





PD-L1 epitope | Rosnilimab | Lilly PD-1 agonist

"A shared feature of agonist mAbs is recognition of the membrane-proximal extracellular region…" and "…activity depends on Fc receptor–supported crosslinking"

Suzuki, et al. 2023

Parmley S, et al. ECCO 2024. February 2024.

1. Adapted from Suzuki et al., Sci. Immunol. 8, eadd4947 (2023).

Rosnilimab is a best-in-class PD-1 depleter and agonist

Lilly's patent notes peresolimab's "modest" activity and disclosed more potent PD-1 candidates closer to rosnilimab's profile

PD-1 Agonist Landscape

		Anaptys Rosnilimab (IgG1k)	Lilly Peresolimab (IgG1k)	Lilly Improved[1] (IgG1 mut. Fc)	Gilead GS-0151 (IgG1 mut. FC[3])
Structural characteristics	Membrane-proximal epitope	✓	✗	?	✓
	Fc receptor binding affinity	✓	✓	✓	✓[3]
Functional outputs	Agonism	✓	Significantly Decreased[2]	?	✓[3]
	Depletion	✓	✓[2]	✓	✗[3]

Insights from Lilly Patent:[1]

"[peresolimab] demonstrated modest ADCC activity"

"FcgRIIB binding alone is not a sufficient driver of IgG1 mediated bioactivity"

"Multiple FcgR interactions contribute to PD-1 agonist activity…[and] increased FcgR binding increased inhibition of T cell proliferation"

"FcgRs work as a hook in the IS [immune synapse] to allow PD-1 to relocate to a close proximity of the TCR to enable inhibition of TCR signalling"

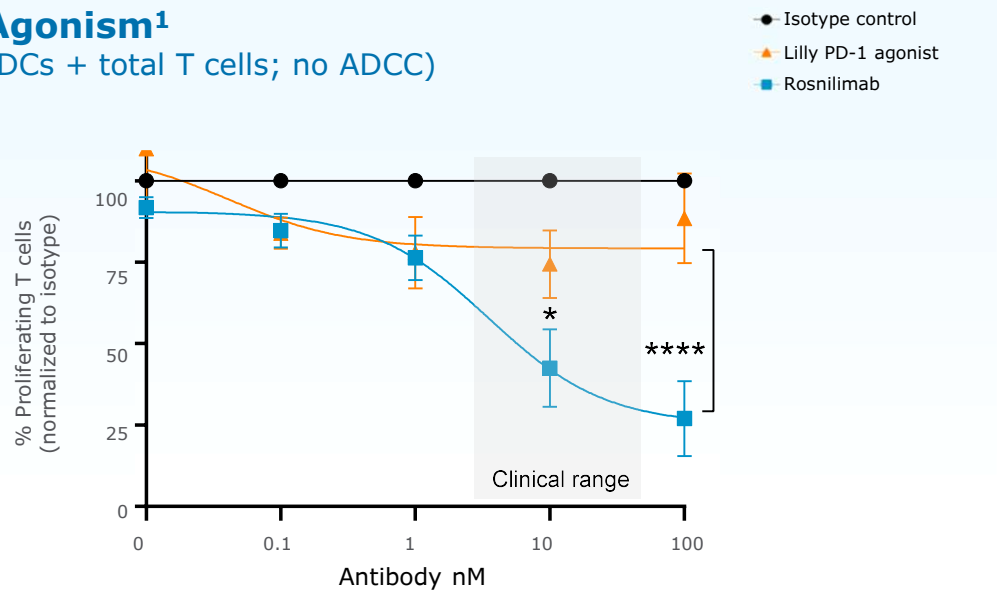
Additional LLY PD-1 variants engineered to have greater potency than peresolimab[1]

Binding Domain	Human Fc	% Mean T cell Proliferation Inhibition
G2	IgG1 (Peresolimab)	56
F1	IgG1	40
F1	IgG1 S239D + I332E	93
	Abatacept	14 – 16

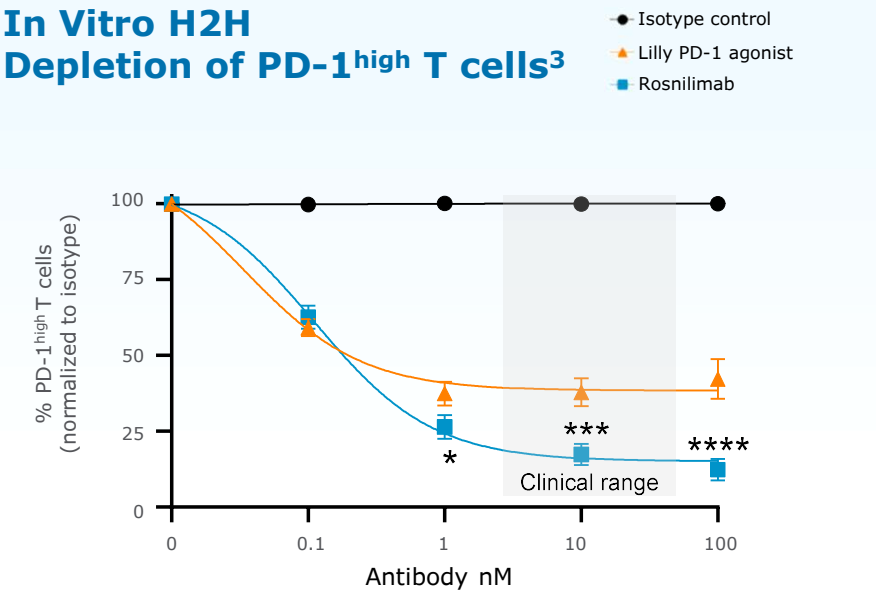
1. Eli Lilly patents; WO2024196694A2 and WO2024040206A2
2. Less potent depletion and significantly weaker agonism from membrane-distal binding epitope results in wider immune synapse and lower clustering of PD-1
3. Fc binding to FcγRIIb only

Comparative data of rosnilimab consistently demonstrates potency of impact on PD-1+ T cells

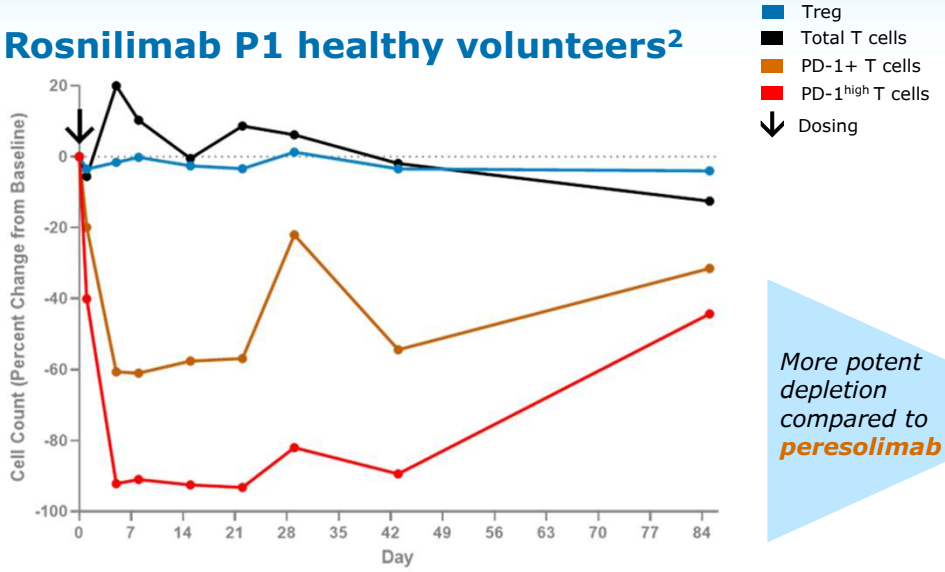
Agonism[1]
(DCs + total T cells; no ADCC)



In Vitro H2H Depletion of PD-1^{high} T cells[3]

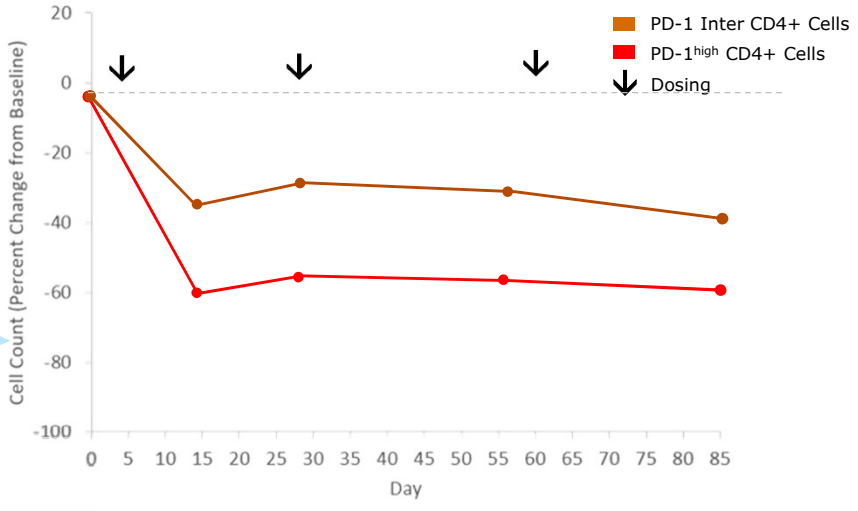


Rosnilimab P1 healthy volunteers[2]



More potent depletion compared to ***peresolimab***

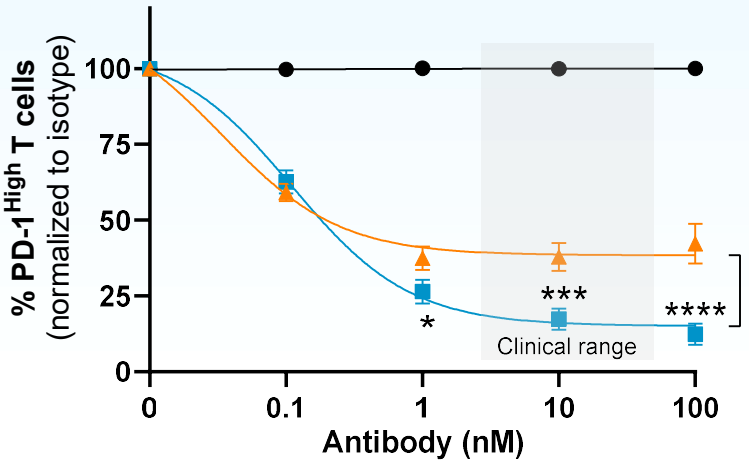
Lilly's Peresolimab P2a RA patients[4]



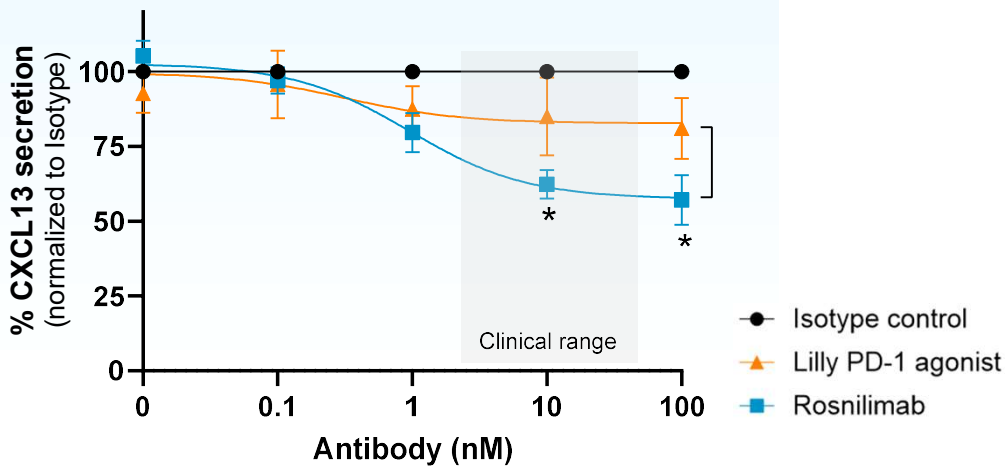
1. Healthy donor purified DCs + autologous total T cells stimulated with anti-CD3, cultured for 3 days for assessment of T cell proliferation
2. Luu K, et al. ACR 2023. November 2023; 3. Anti-CD3+ anti-CD28 stimulation of RA patient PBMCs for assessment of depletion and agonism MOA, representative data from N=8 donors. Two-way ANOVA, Tukey's multiple comparison test. ****P<0.0001, ***p<0.001, **p<0.01, *p<0.05.
4. Benschop, R. ACR 2023, Eli Lilly peresolimab Phase 2a data.

Rosnilimab's potent depletion and agonism reduces T cell proliferation and inflammatory cytokines that cause joint damage

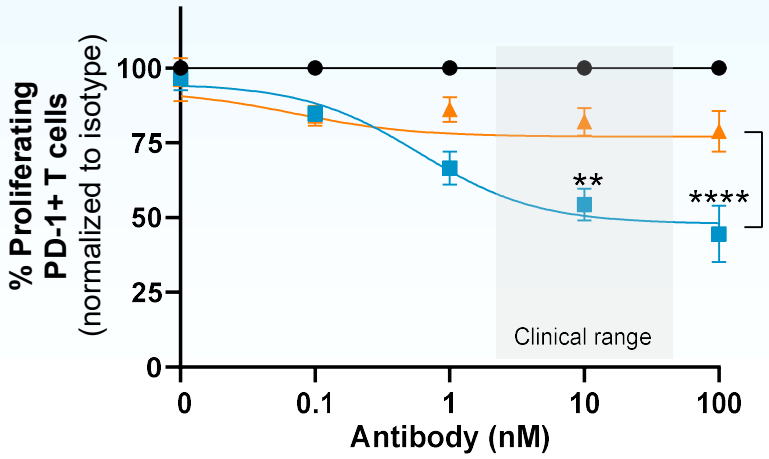
Depletion of PD-1^{high} T cells



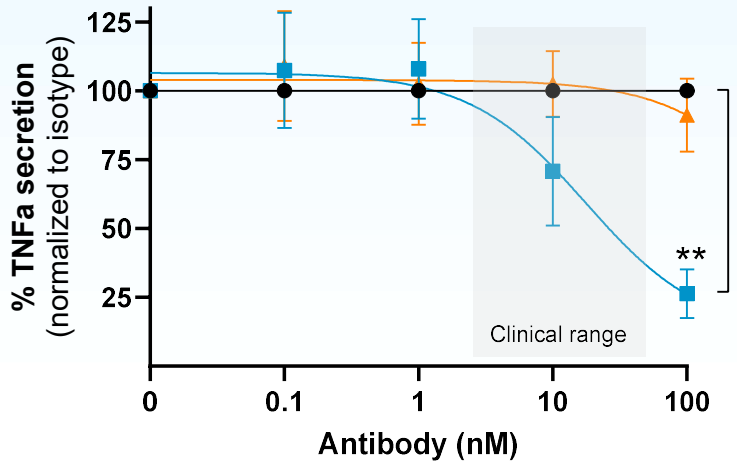
Reduction of Tfh/Tph chemokine



Reduction of T cell proliferation

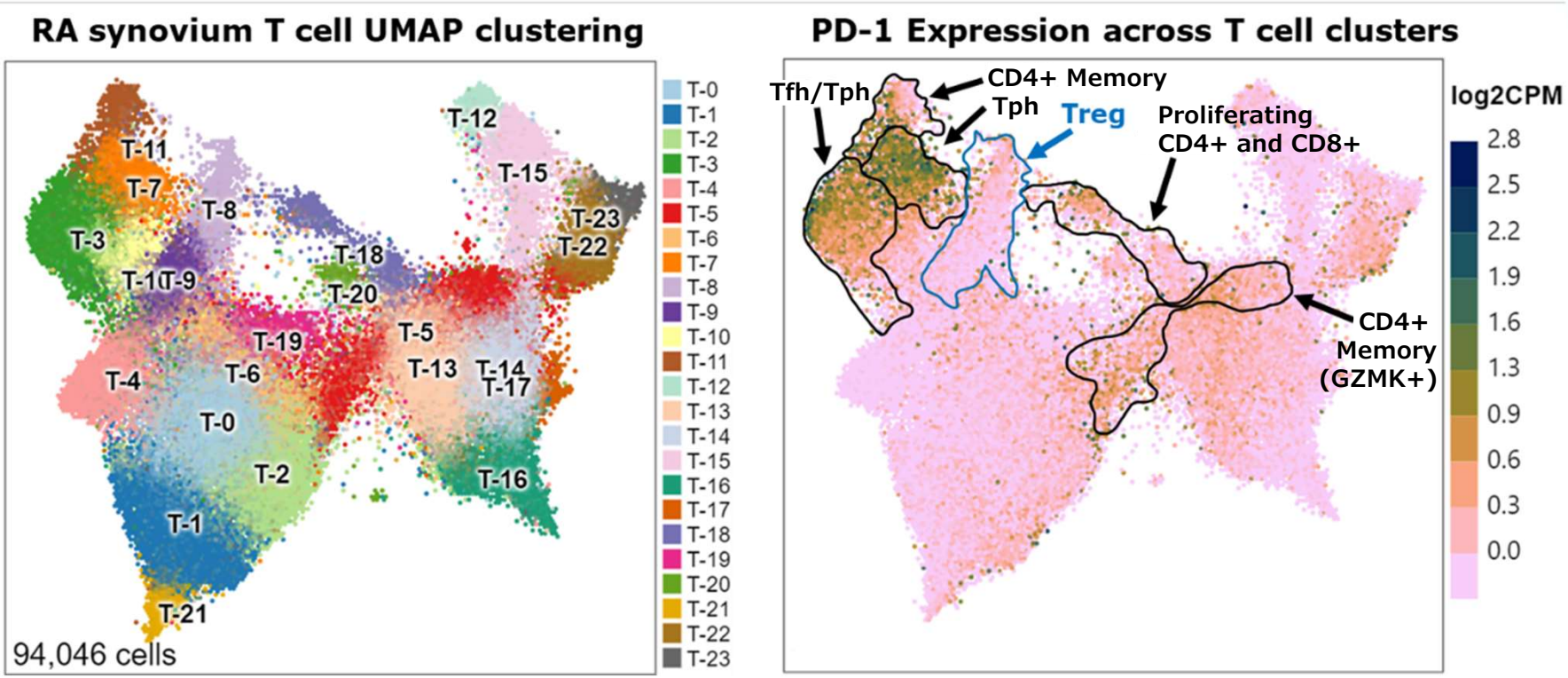


Reduction of inflammatory cytokine[1]



Anti-CD3+ anti-CD28 stimulation of RA patient PBMCs for assessment of depletion and agonism MOA, representative data from N=8 donors
Two-way ANOVA, Tukey's multiple comparison test. ****P<0.0001, ***p<0.001, **p<0.01, *p<0.05.
1. TNFa secretion measured in anti-CD3+ anti-CD28 stimulation of purified DC+T cells from N=4 healthy donors.

In disease, PD-1+ Tregs exhibit a dysregulated phenotype, which induce proinflammatory cytokines



Very low % Tregs (<20%) are PD-1+ in RA synovium, even fewer are PD-1^{high1}

PD-1+ Tregs may be pro-inflammatory and induce IFNγ, IL-17A, TNFα

Rosnilimab likely depletes PD-1^{high} Tregs and reduces PD-1+ Tregs, resulting in favorable Treg/Teff cell ratio

1. Uniform manifold approximation and projection (UMAP) clusters of T cells from RA patient synovium with arrows identifying Tph and Tfh/Tph cells, T-7 and T-3, respectively and feature plot of PD-1 expression across T cell subtypes; Ren et. al. ACR 2024. November 2024

Rosnilimab, and overall PD-1 agonist class, well-tolerated with no significant safety signals

Rosnilimab: Favorable safety and tolerability in P1a and P2a studies

- Phase 1a individuals and Phase 2a alopecia areata patients for up to 6 months (400mg Q4W SC)
- No SAEs related to rosnilimab[1]
- No malignancies observed
- No infection risk signal

Competitor PD-1 agonist programs

- >100+ RA patients in P2a treated with Lilly PD-1 agonist (highest dose of 700 mg IV over 6 months)[2]
- No public disclosure of any PD-1 agonist to show a malignancy or infection risk signal

Rosnilimab: Ongoing RA and UC studies

- ~420-patient 6 month RA study
- ~132-patient 1 year UC study
- Blinded surveillance: no safety signal to date

Abatacept (competing T Cell Modulator)

- Broadly impacts all T cells including all Tregs
- Decades of commercial use
- Have not shown clinically relevant carcinogenic increases

1. SAEs unrelated to rosnilimab as follows: Obstructive pancreatitis occurred in a placebo subject and Coronavirus infection occurred in drug 400 mg SC cohort on Day 24 until Day 31; participant recovered and discontinued from the study, and AE was deemed unrelated to rosnilimab. 2. Lilly peresolimab Phase 2 data in RA, published in NEJM (A Phase 2 Trial of Peresolimab for Adults with Rheumatoid Arthritis | NEJM).

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]

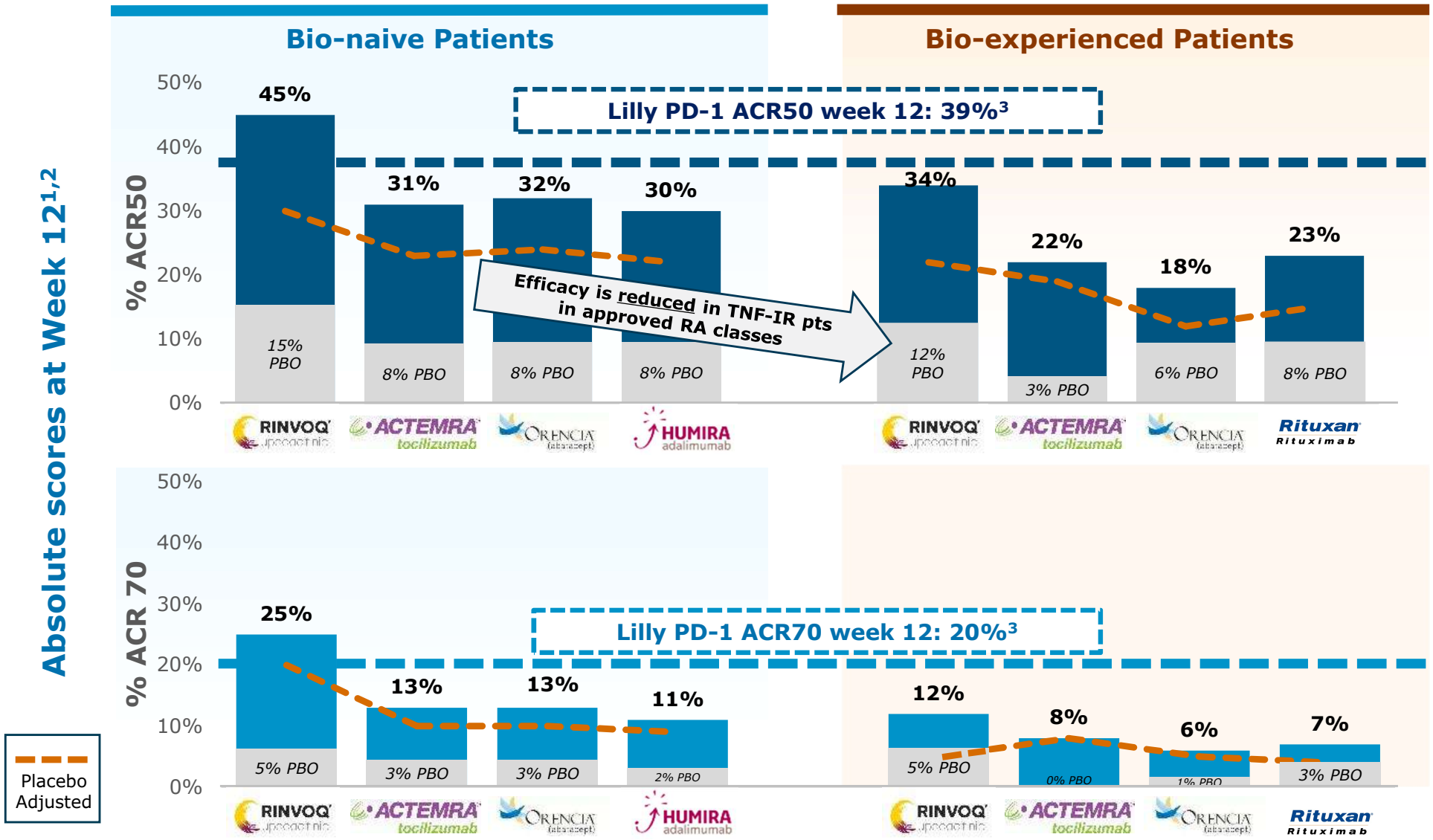
2x
Malignancy Rate[3]

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

Targeting PD-1+ T cells is a clinically validated approach in RA with proof of mechanism



1. Phase 3 registrational data from product labels; 15mg dose for upadacitinib in STUDY V 2. Tocilizumab (8mg/kg dose); Smolen J (2008) The Lancet Vol 371: 987-997; Emery, P. (2008) ARD 67(11): 1516-1523; Adalimumab; Keystone E (2004) Arthritis & Rheumatism Vol 50 #5:1400-1411; Rituximab; Cohen S (2006) Arthritis & Rheumatism Vol 54 #9: 2793-2806 3. Tuttle, J. (2023) NEJM;388:1853-62. Note patient population is 63% MTX-IR, 37% b/tsDMARD-IR; Similar efficacy was observed regardless of prior b/tsDMARD use.

Treat-to-target practice in RA results in the importance of multiple efficacy endpoints across both Week 12 and 24

Rheumatologists seek disease modification

— CDAI LDA correlates with slowed radiographic progression

Week 12: Broad response

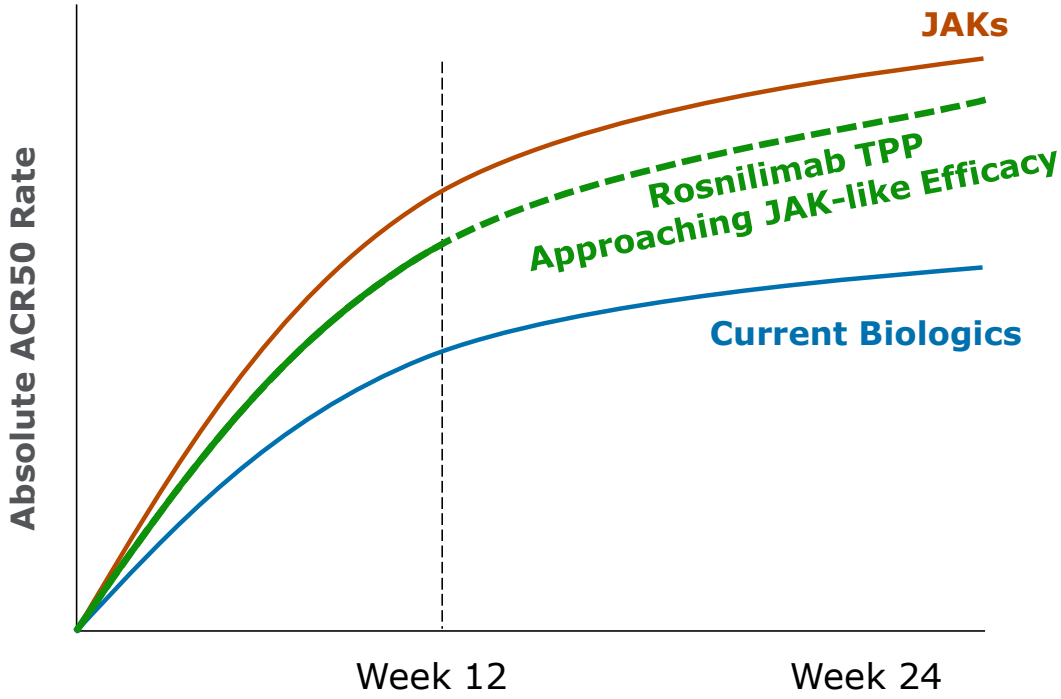
— Switch patients if not improving (i.e. ACR20)

Week 24: Stable or deepening response

— ACR50/70 in as many patients as possible

— Only modest deepening observed for approved drugs from Week 12 to 24

Illustrative Response Curves



Phase 2 Target Product Profile

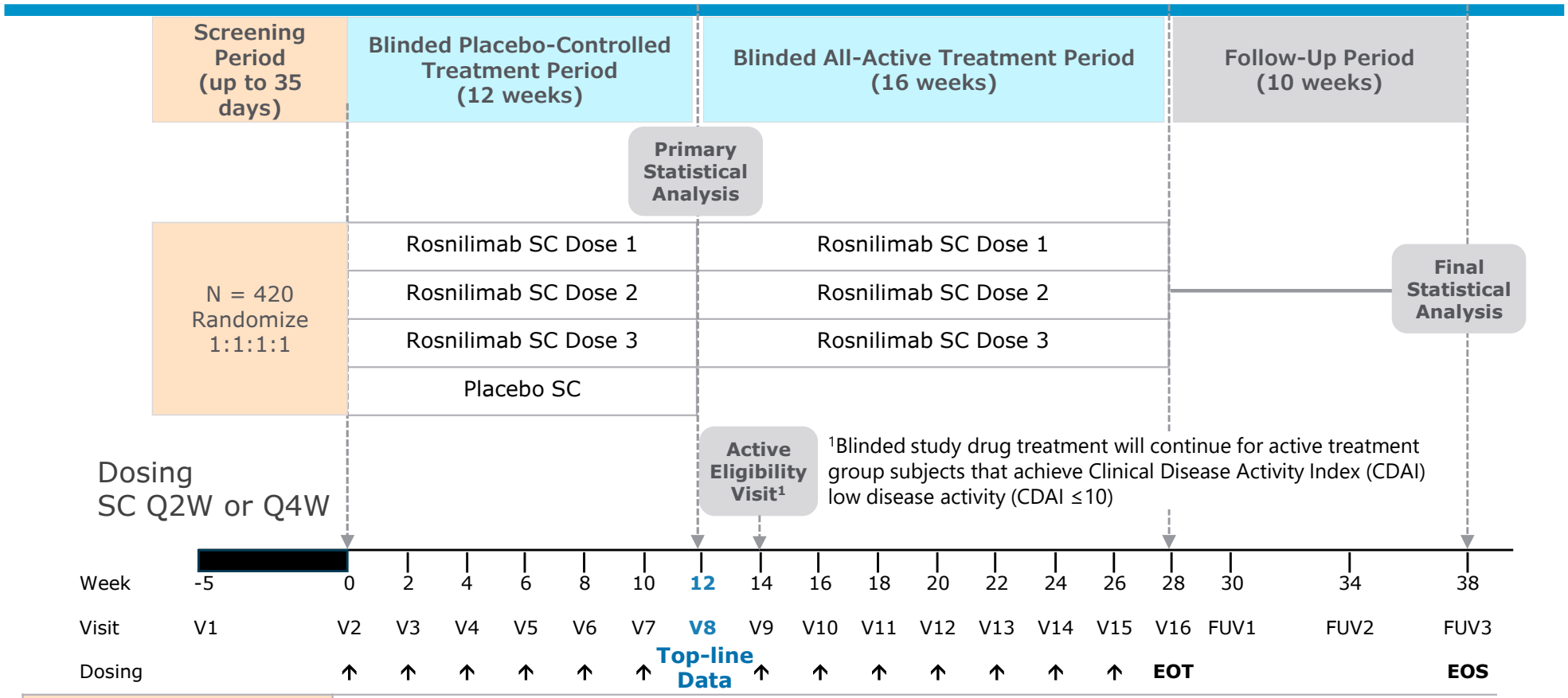
Phase 2 trial design only allows for sustained response rates, not improved response rates, between Week 16 and 28

Population	Week 12 (Placebo-adjusted)	Week 12 (Absolute)	Week 24 (Absolute)
Bio-experienced	CDAI LDA: ~5-15% ACR50: ~15-20% ACR70: ~5-10%	CDAI LDA: ~30% ACR50: ~30% ACR70: ~10%	ACR50: ~40% ACR70: ~15%
Bio-naïve	CDAI LDA: ~10-20% ACR50: ~20-30% ACR70: ~5-15%	CDAI LDA: ~35% ACR50: ~40% ACR70: ~20%	ACR50: ~50% ACR70: ~25%

CDAI = Clinical Diseases Activity Index; LDA = Low Disease Activity.

Rosnilimab Phase 2b in moderate-to-severe RA

Anticipate top-line Week 12 data February 2025; Week 28 data Q2 2025



Patient population		• Adults with moderate-to-severe rheumatoid arthritis, ≥ 6 TJC and SJC • Positive RF or CCP • Includes both MTX-IR and b/tsDMARD experienced patients (~40% b/tsDMARD experienced) • IR or intolerance to < 3 classes of b/tsDMARDs
Endpoints	Primary	• Mean change from Baseline in DAS28-CRP at Week 12
	Secondary	• ACR20/50/70 • CDAI ≤ 10 (low disease) and ≤ 2.8 (remission) • DAS28-CRP ≤ 3.2 (low disease); DAS28-CRP ≤ 2.6 (remission)
Exploratory endpoints		• Mean change from Baseline in synovial and peripheral biomarkers

ClinicalTrials.gov: NCT06041269

Robust and well-controlled Phase 2b RA trial

Trial design and endpoints

Large (~420 patient) study

- 3-active SC arms (Q2W / Q4W) vs. PBO
- 60% b/tsDMARD naïve
- ~40% b/tsDMARD experienced (up to 2 prior classes)

Standardized composite endpoints

- >80% power for ACR50 composite (secondary endpoint) at Week 12
- CDAI LDA (≤ 10) responders at Week 14 treated through Week 28

Well-established inclusion/ exclusion criteria

High disease activity

- ≥ 6 tender and ≥ 6 swollen joints
- Seropositive RA - Rheumatoid factor or α-CCP positive
- CRP > 3mg/L
- Majority CDAI>22 (e.g. severe)

Stable background medications

- Stable dose of cDMARDs >8 weeks prior to baseline
- No changes in prednisone (≤10mg)
- No changes in background DMARDs
- No rescue medications

CRO and monitoring

CRO has extensive RA experience

- US and EU countries only
- Excluded countries with historically high PBO rates (e.g. Mexico, LatAm)

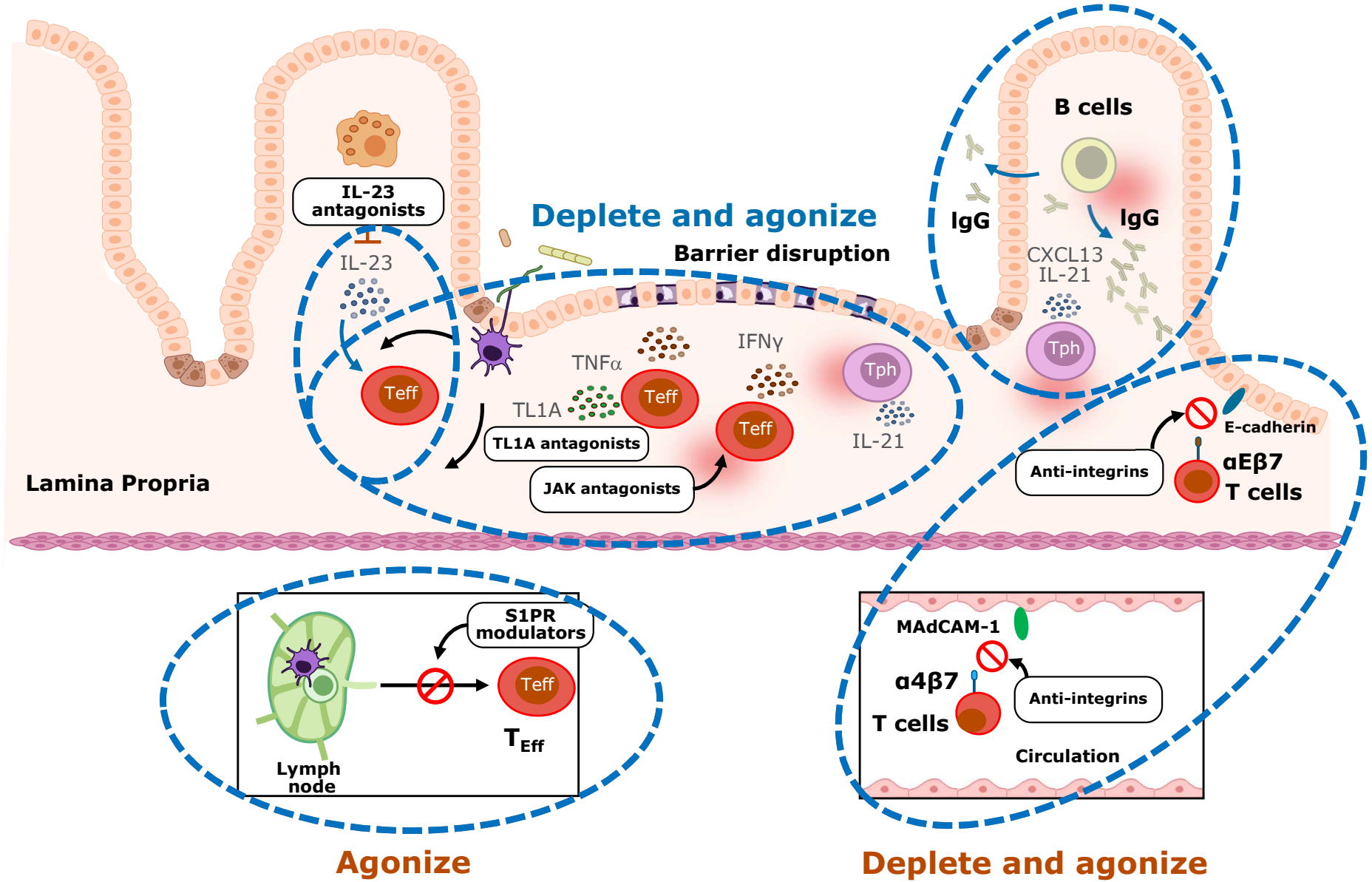
All sites, PIs experienced in RA

- Blinded independent joint assessors
- Participant eligibility review

CCP=cyclic citrullinated peptide; CRP=C-reactive protein; SIV=site-initiation visit.

PD-1+ T cell activation broadly impacts multiple clinically validated drivers of UC pathogenesis

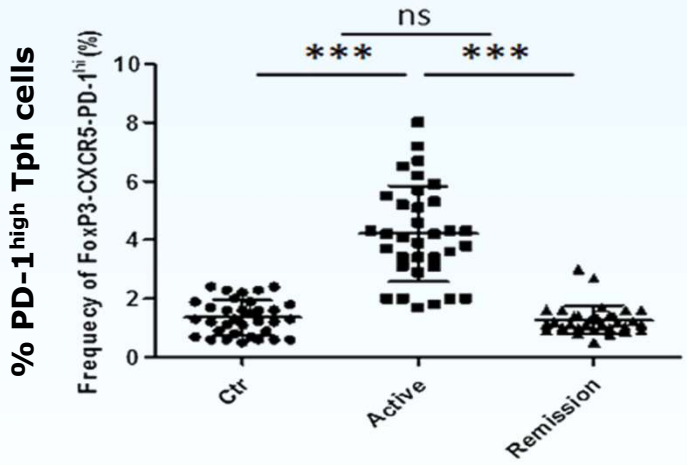
- >40% of T cells in lamina propria in UC are PD-1+
- 2x increase of PD-1+ T cells observed in blood *vs.* healthy controls[1]

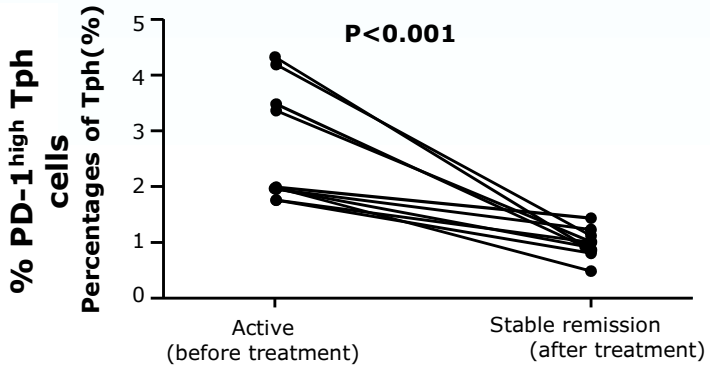


Adapted from Gastroenterology & Hepatology Volume 18, Issue 8 August 2022. 1. Chen et al, Clinical and Translational Immunology, 2024.

Reduction of elevated PD-1^{high} Tph cells in both UC colon and periphery correlates with remission

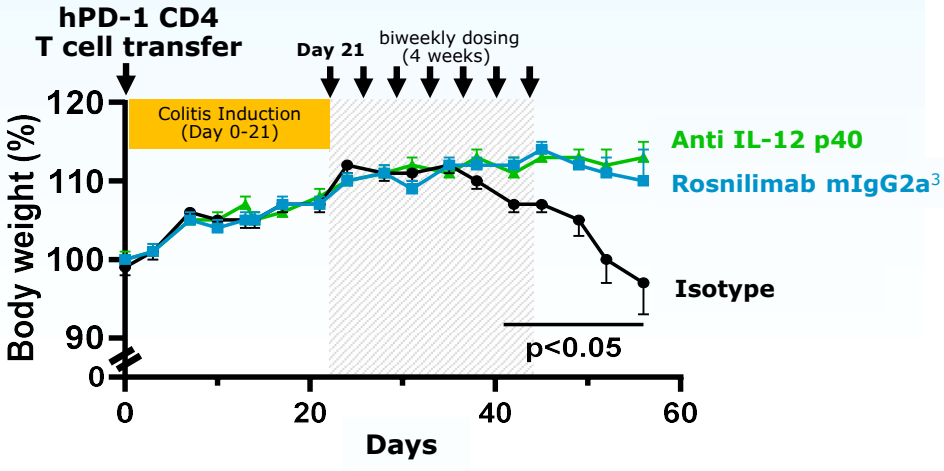
PD-1^{high} Tph cells are reduced with remission[1,2]



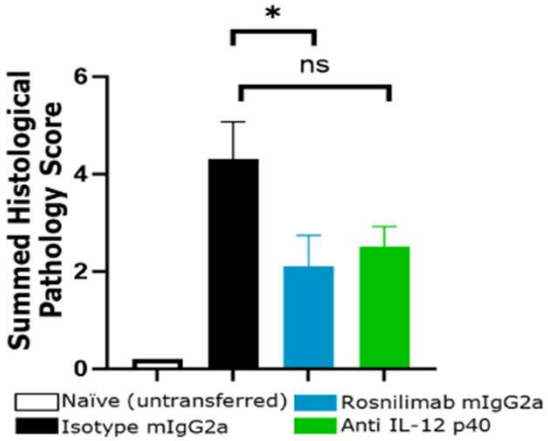


Reduction of Tfh/Tph cells should impact plasma cell generation and autoantibody levels, including anti-microbial IgG antibodies that are contributing to colonic inflammation and barrier disruption[4]

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



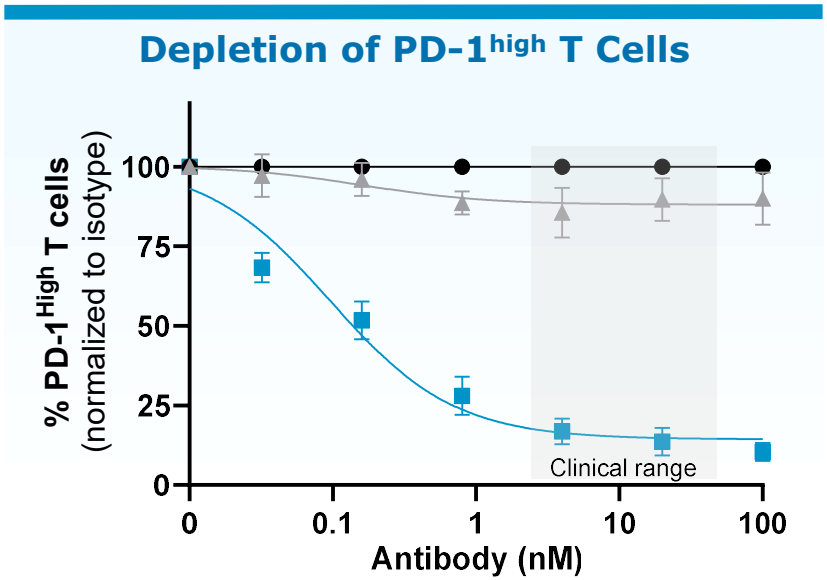
Distal colon histology and scoring

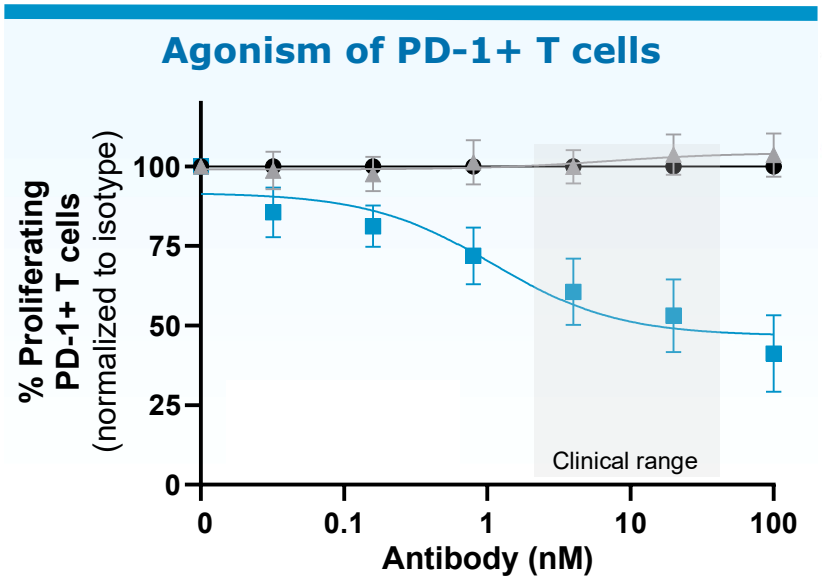


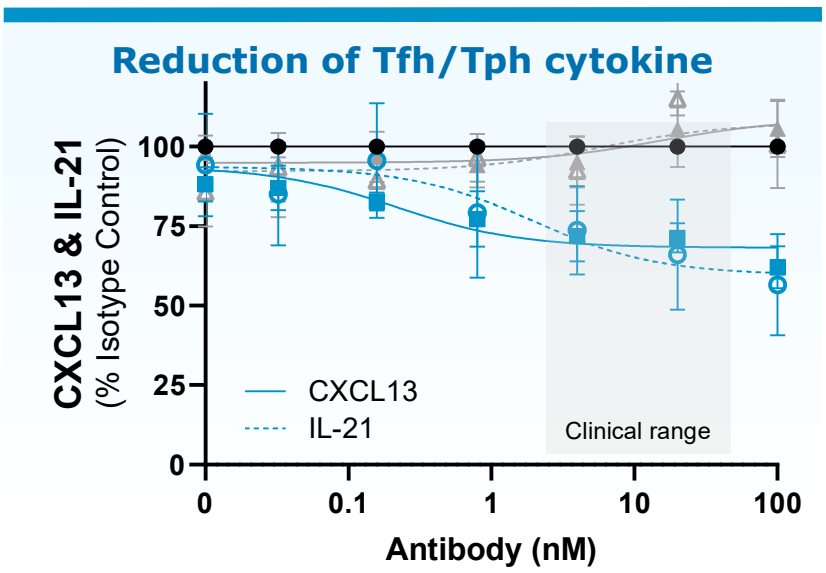
Parmley et. al. UEGW 2024. October 2024

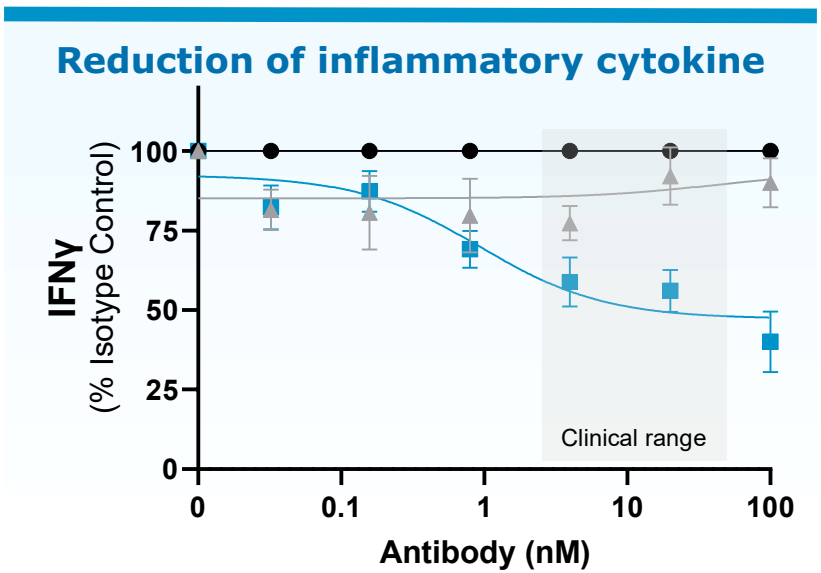
1. PD-1^{high} Tph cells defined by CD3+CD4+CD45RA-PD-1+TIGIT+ICOS+CXCR5-. Long et al, Immunology Letters 233 (2021) 2-10.
2. Rao et al, Nature, 2017. *** p<0.001, * p<0.05
3. Rosnilimab formatted to mIgG2a to mediate effector function in mice. Suzuki et al., Sci. Immunol. 8, eadd4947 (2023).
4. Uzzan et al, Nature, 2022

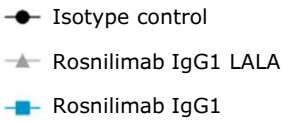
Rosnilimab's potent depletion and agonism reduces T cell proliferation and inflammatory cytokines that disrupt barrier function







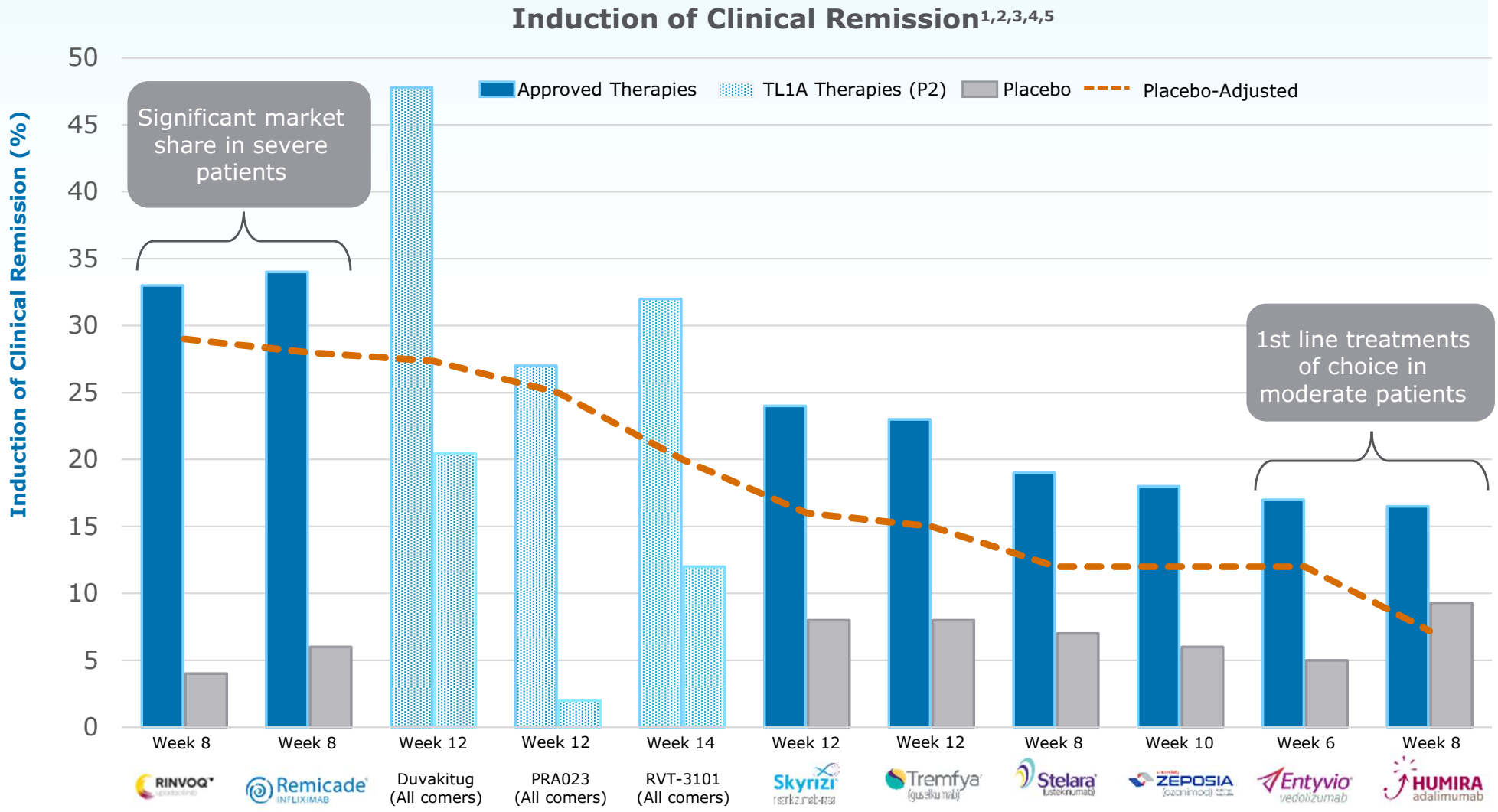




Parmley et. al. UEGW 2024. October 2024
Anti-CD3+ anti-CD28 stimulation of UC patient PBMCs for assessment of depletion and agonism MOA, representative data from N=6 donors.
Rosnilimab IgG1 LALA included to demonstrate importance of Fc effector function

UC lacks highly effective treatment options to induce and maintain clinical remission

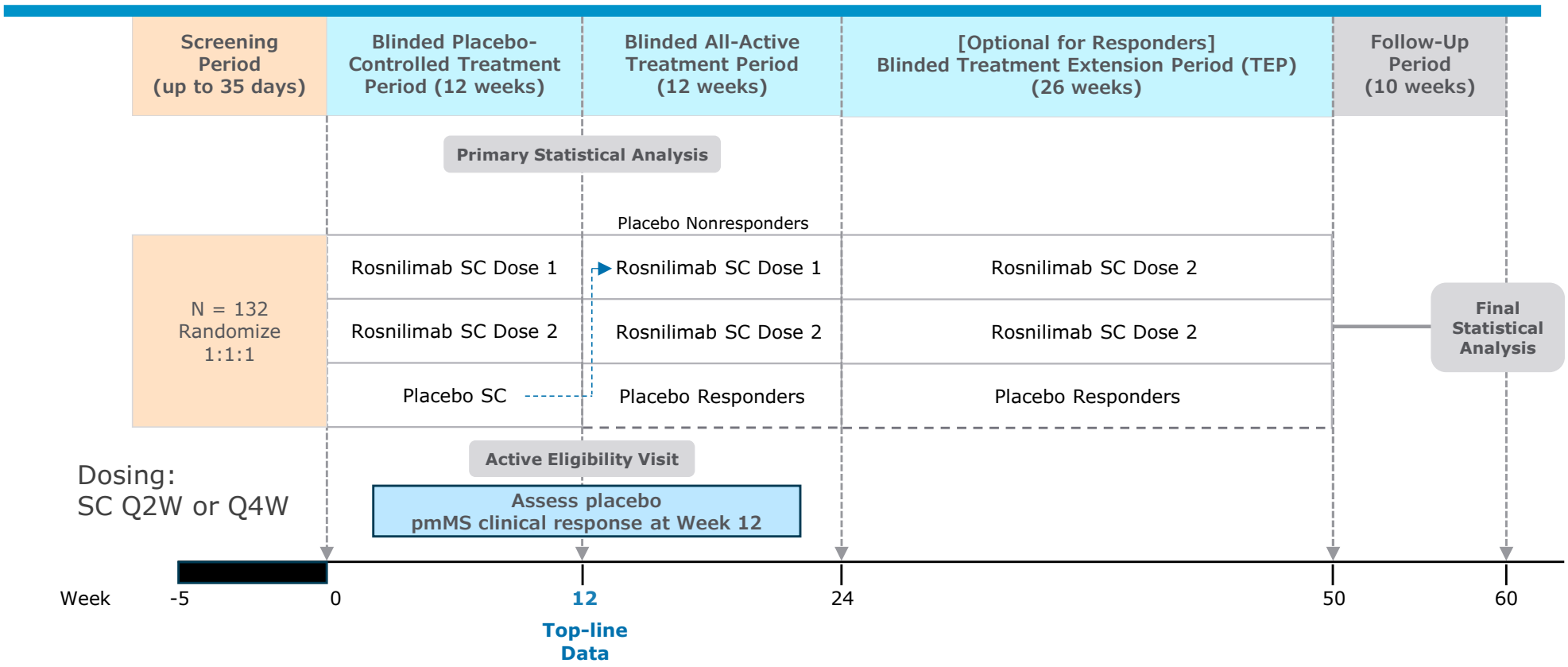
Following remission on induction therapy, one third to one half of patients relapse within 1 year[1]



1. Phase 3 registrational data from product labels; 2 Prometheus Bioscience corp. presentation Mar 2023; 3. Roivant corp presentation Jan 2023; 4. Teva corp presentation Dec 2024; 5. Remission measured using modified Mayo Score, except for Remicade, Humira and Entyvio which used full Mayo Score.

Rosnilimab Phase 2 in moderate-to-severe UC

Top-line data anticipated Q1 2026



Patient population		• Adults with moderate-to-severe ulcerative colitis • Inadequate response to, loss of response to, or intolerance to as least 1 conventional or advanced UC therapy (~30-40% advanced UC therapy experienced)
Endpoints	Primary	• Mean change from Baseline in modified Mayo Score (mMS) at Week 12
	Secondary	• Clinical remission on mMS • Clinical response on mMS • Endoscopic remission • Mucosal healing
Exploratory endpoints		• Mean change from Baseline in colonic tissue and peripheral biomarkers

ClinicalTrials.gov: NCT06127043

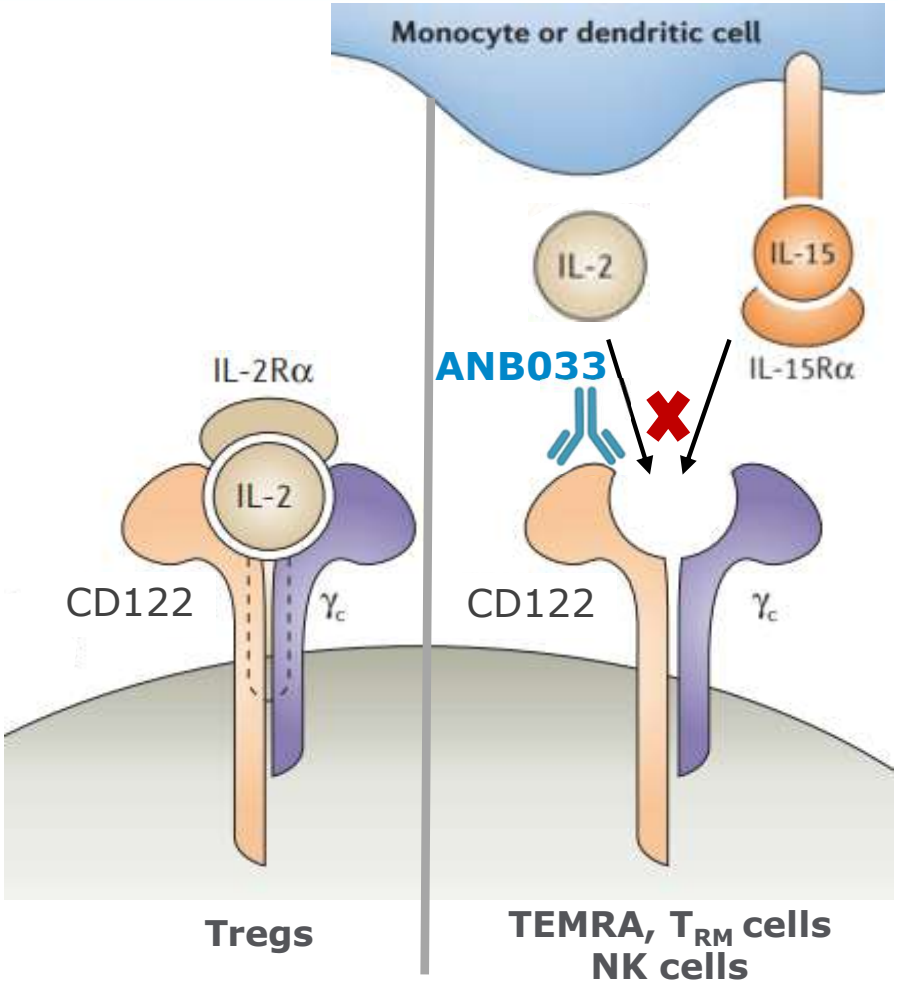
ANB033 (CD122 antagonist)

Autoimmune and Inflammatory Diseases

ANB033: CD122 high affinity antagonist reduces pathogenic T cells and NK Cells

Phase 1 trial initiated in healthy volunteers

CD122 is a shared beta subunit of the receptors for IL-15 and IL-2



CD122 antagonist mAb will potently inhibit IL-15 and IL-2 biology

Both IL-15 and IL-2 mediate:

- Proliferation and survival of T cell subsets, particularly CD8+ TEMRA, and NK cells
- Inflammatory cytokine secretion (IFNγ) during T cell activation

ANB033 reduces pathogenic T cells

- Preferentially inhibits lower affinity dimeric IL-2 receptor complex
- Spare Tregs which express higher affinity trimeric IL-2 receptor complex

ANB033 has targeted reduction of CD122 expressing T_{RM} cells

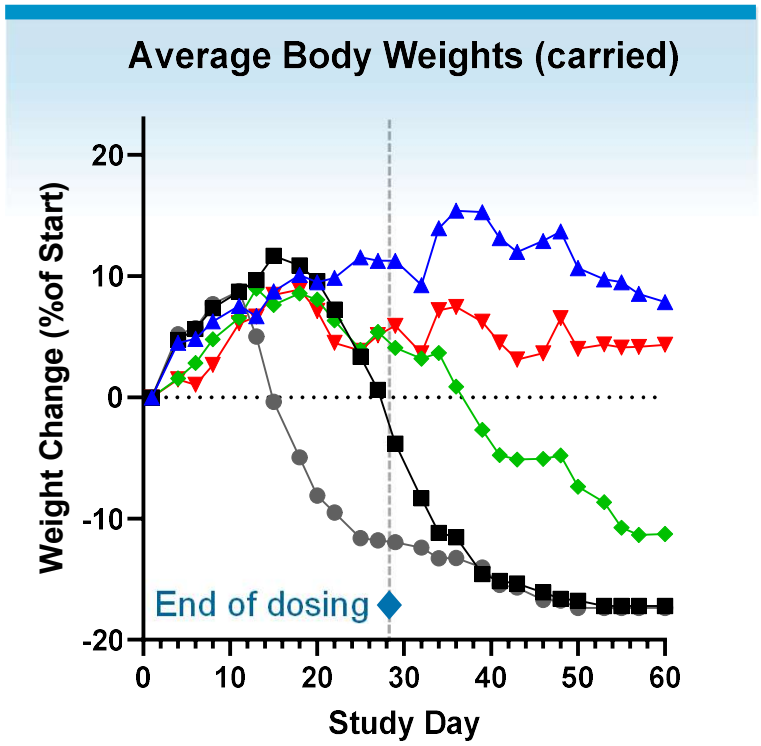
- T_{RM} cells require IL-15 for survival
- May potentially drive durable response

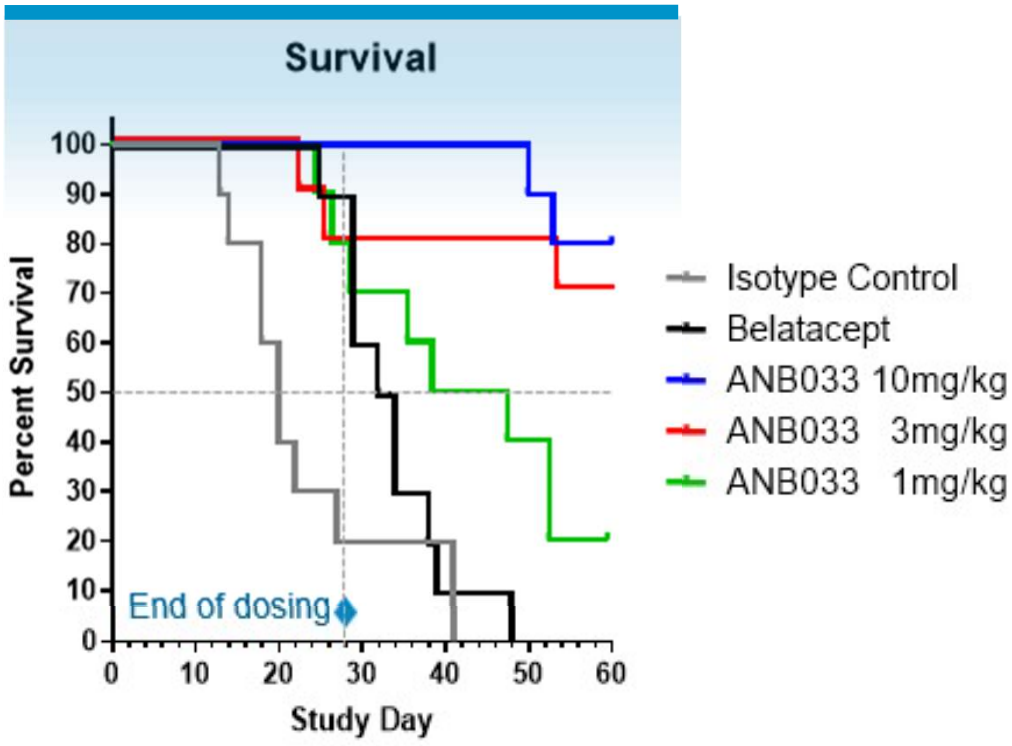
ANB033: Durable survival in GVHD model

All mice treated at high-dose survived well beyond end of dosing

GVHD (severe phenotype) model in human IL-15 transgenic mouse supports T cell and NK cell survival

- ANB033 preclinical data suggests targeted elimination of pathogenic T cells and reduction of tissue infiltrating T cells leading to a more potent and durable response than belatacept
- Belatacept (GVHD SOC which only impedes T cell activation) shows minimal benefit over control





GVHD model is biologically relevant to CD122 antagonist MoA with translation to inflammatory diseases driven by pathogenic T_{RM} and Treg imbalance including rheumatology, dermatology, gastroenterology and respiratory

Hare E, et al. FOCIS 2023. June 2023.

Note: ANB033 treated mice dosed twice per week through Day 28.

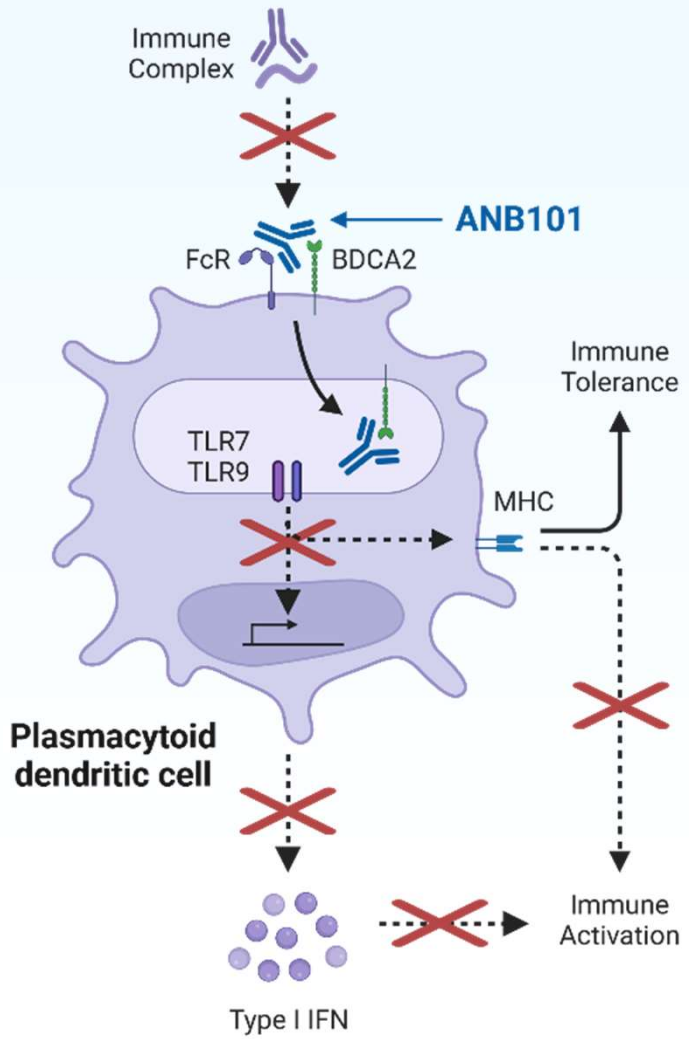
ANB101 (BDCA2 modulator)

Autoimmune and Inflammatory Diseases

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

IND submitted; Phase 1 initiation anticipated Q1 2025

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
- Preserves pDCs for potential tolerogenic effects

Note: ANB101 (formerly known as CBS004) was in-licensed from Centessa Pharmaceuticals. Has completed NHP tox studies and P1 clinical material available.

GSK Immuno-Oncology Financial Collaboration

Jemperli™
(dostarlimab, PD-1 Antagonist)

Cobolimab
(TIM-3 Antagonist)

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

	Jemperli (dostarlimab-gxly) Injection 500 mg *(PD-1 antagonist)*	Cobolimab *(TIM-3 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	4% - $0 to $250 million 5% - $250 to $500 million 6% - $500 to $750 million 7% - >$750 to $1.0 billion 8% - >$1.0 billion *Royalty rate on cobolimab includes potential cobolimab-portion of combination use with dostarlimab*
Remaining retained milestones	$75MM when annual net sales ≥ $1 billion[1]	$5MM clinical development $90MM regulatory $165MM commercial

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

- *Jemperli* receivables payable to Sagard until cumulative $600MM paydown by Mar. 31, 2031[1,2]
- ~$90MM paid to Sagard as of early January 2025
- Projected cumulative $600MM paydown by 2029 based on Wall Street Consensus[3]

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

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

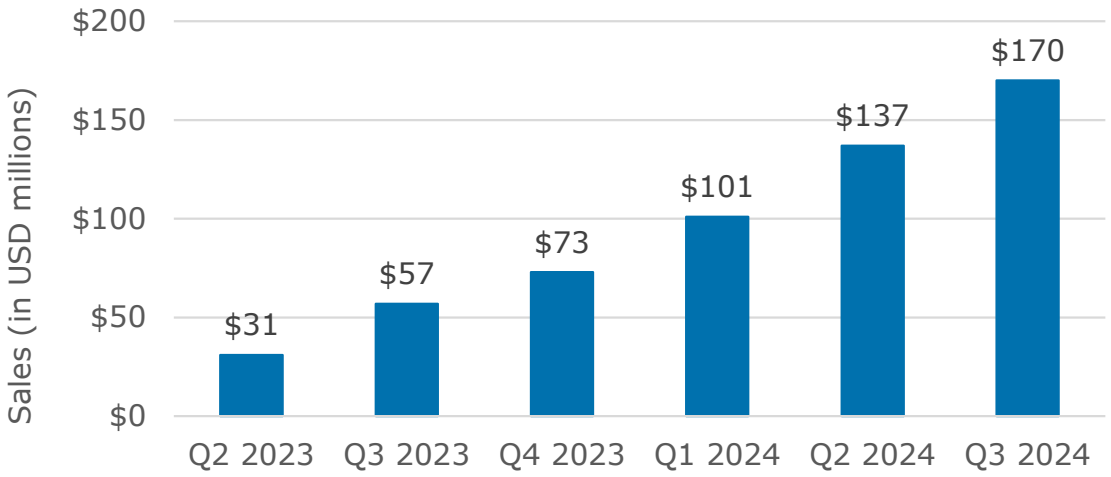
3. GSK analyst consensus as of 11/14/2024 converted to USD (1.25 conversion rate), GSK website - https://www.gsk.com/en-gb/investors/analyst-consensus/

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

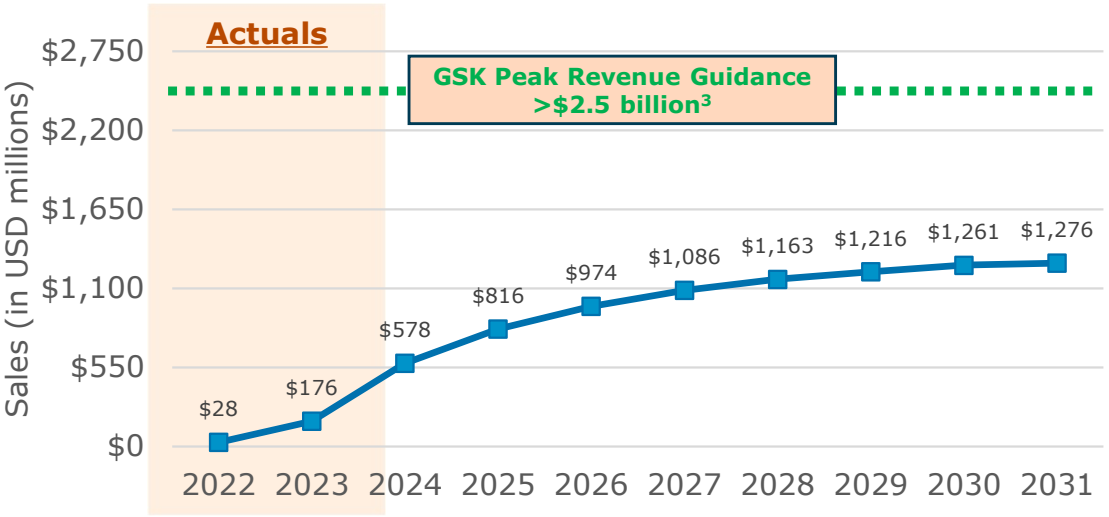
Note: Separate sale of Anaptys' *Zejula* (niraparib) royalty interest occurred in September 2022 to DRI Healthcare Trust for $35MM upfront + $10MM potential milestone upon FDA approval of *Zejula* for the treatment of endometrial cancer, to the extent that such approval occurs on or before 12/31/25.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

Consensus projections of *Jemperli* imply significant royalty upside to Anaptys post-Sagard paydown

Jemperli Quarterly Performance[1]



Jemperli Wall Street Consensus[1,2]



Current commercial performance

- $170MM Q3 2024 Sales (>100% YoY growth)[1]
- Driven from US all-comers launch and higher new patients starts in 1L dMMR endometrial
- Continued growth of EU 2L endometrial sales
- Substantial investment in additional indications ongoing

Potential future growth drivers

- 1L "all-comers" endometrial: EU approval expected Q1 2025[1]
- 1L ovarian: Positive P3 PFS data reported in Dec. 2024 to be shared with regulators
- 2L+ NSCLC: Phase 3 COSTAR (*Jemperli* + TIM-3) data anticipated H1 2025[1]
- Locally advanced dMMR/MSI-H rectal cancer: granted FDA Breakthrough Therapy Designation

1. GSK earnings presentation, US dollar conversion 2. GSK analyst consensus as of 11/14/2024 converted to USD (1.25 conversion rate), GSK website - https://www.gsk.com/en-gb/investors/analyst-consensus/ 3. GSK June 2024 Oncology Management IR event converted to USD (1.25x conversion rate)

GSK immuno-oncology financial collaboration

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

Women's cancers

- **Endometrial Cancer:**
  - **1L endometrial cancer:** Approved in US for primary advanced or recurrent EC; GSK has received a positive CHMP opinion for this same indication in the EU
  - **2L endometrial cancer:** Approved in US and EU for dMMR/MSI-H recurrent or advanced EC after progressing on a platinum-containing regimen
  - **P3 RUBY Part 2:** Addition of niraparib to dostarlimab in maintenance setting (dostarlimab + niraparib compared to placebo plus chemotherapy followed by placebo) demonstrated significant improvement in PFS in MMRp/MSS
  - Significant U.S. market opportunity with 23,000 eligible diagnoses/year[1]
- **Ovarian cancer:** P3 (FIRST) trial (combination of dostarlimab + niraparib) in 1L ovarian cancer
  - Demonstrated significant improvement in PFS
  - Significant U.S. market opportunity with ~20,000 eligible diagnoses/year[1]

Colorectal cancer

- **Rectal cancer:** P2 AZUR-1 trial (dostarlimab monotherapy in dMMR/MSI-H in locally advanced [LA] rectal cancer)
- **Colon cancer:** P3 AZUR-2 trial (perioperative dostarlimab monotherapy vs SoC adjuvant chemotherapy in patients with high-risk early-stage dMMR/MSI-H cancer)

Additional dostarlimab royalty opportunities

- P3: LA unHNSCC monotherapy sequentially after chemoradiation (JADE study)
- P3: 1L NSCLC in combination with anti-TIGIT (belrestotug) (GALAXIES Lung-301)
- P1/2 combinations with anti-CD96 and PVRIG across multiple solid tumors

Jemperli (dostarlimab-gxly) Injection 500 mg
+
Cobolimab
(TIM-3 antagonist)

Lung cancer[2]

- **2L NSCLC:** P3 COSTAR trial (docetaxel *vs* dostarlimab + docetaxel *vs* docetaxel + dostarlimab + cobolimab)
  - Top-line data expected in H1 2025
  - Significant U.S. market opportunity with 237,000 new NSCLC diagnoses/year[1]

1. NCI SEER data 2. In 1L NSCLC, Phase 2 PERLA trial demonstrated 46% cORR for dostarlimab + chemo vs. 37% cORR for pembrolizumab + chemotherapy (not for registration)

Legacy Programs for Out-Licensing

Imsidolimab (IL-36R antagonist)

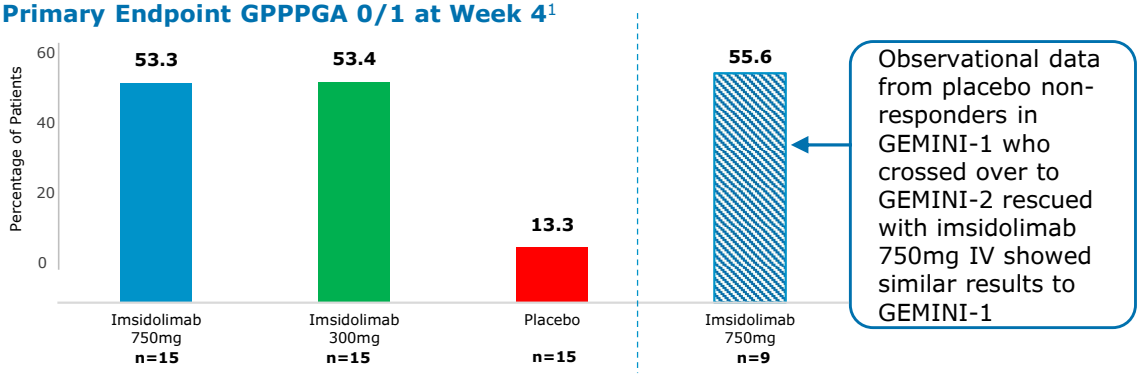
Etokimab (IL-33 antagonist)

GEMINI-1 and -2 Imsidolimab positive Phase 3 data

Data presented at EADV 2024

GEMINI-1: Imsidolimab (750mg and 300mg IV) Effective in Treatment of GPP Flare in GEMINI-1 & in Crossover Placebo Patients in GEMINI-2 (750mg IV)

Primary Endpoint GPPPGA 0/1 at Week 4[1]



Single doses of imsidolimab were highly effective at inducing Generalized Pustular Psoriasis Physician Global Assessment (GPPPGA) response vs. placebo

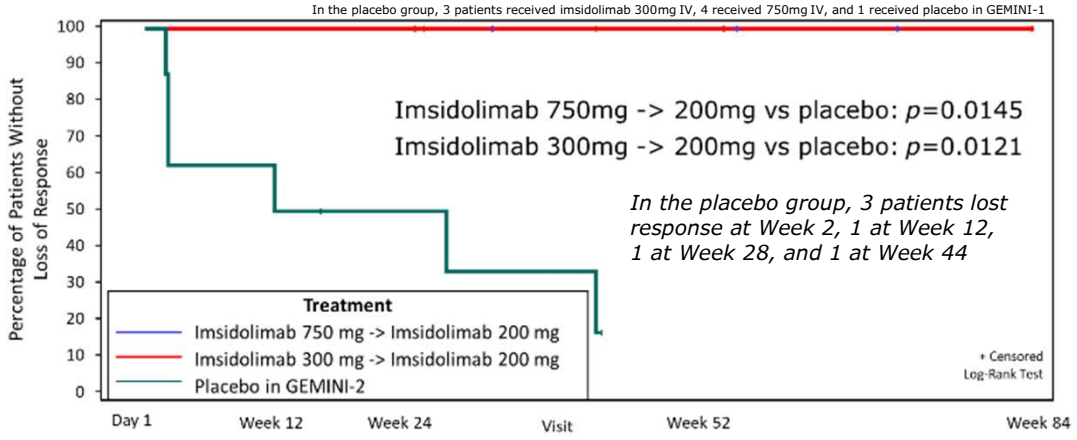
Safety and Tolerability

- Treatment-emergent adverse events (TEAE) similar across treatment groups
- No SAEs or severe AEs in imsidolimab-treated patients
- No cases of DRESS or GBS*
- Low incidence and no elevation of infections vs. placebo
- 1 patient treated with 750 mg (n=30, 3%) had detectable non-neutralizing anti-drug antibodies (ADA)
- Similar safety across both GEMINI-1 and -2

**Drug Reaction with Eosinophilia and Systemic Symptoms, Guillain-Barre Syndrome*

GEMINI-2: Imsidolimab (200mg SC) Q4W Maintained Response & Prevented GPP Re-flaring Regardless of GEMINI-1 Imsidolimab Dose

Time to Loss of GPPPGA 0/1 Response[2]



- Imsidolimab (n=8) 0% flared vs. placebo (n=8) 62.5% flared
- Imsidolimab maintained GPPPGA 0/1 response regardless of GEMINI-1 dose
- Placebo crossover patients who received imsidolimab 750mg IV/200mg SC in GEMINI-2 (n=9): 77.8% maintained remission for at least 24 weeks (observational data)

Full EADV 2024 poster and oral presentations are available on Anaptys website here

Reich A., et al. EADV 2024. September 2024

1. % of patients achieving GPPPGA 0/1 at Week 4 and PRS 0/1 at Week 1 in GEMINI-1 after a single IV dose of imsidolimab 750mg, 300mg, or placebo
2. Kaplan-Meier curve of time to loss of response with imsidolimab 200mg SC (shown by dose of imsidolimab received in GEMINI-1) and placebo every 4 weeks

Etokimab: Ph 2b/3-ready IL-33 antagonist antibody

IL-33 biology applicable to epithelial driven diseases

Etokimab: IgG1 antibody that inhibits the active form of IL-33

- Binding affinity of etokimab is <1 pM; best-in-class based on competitor affinities published in patents and literature
- Targeting IL-33 cytokine rather than IL-33 receptor (ST2) has potential to not only modify disease, but also drive epithelial remodeling

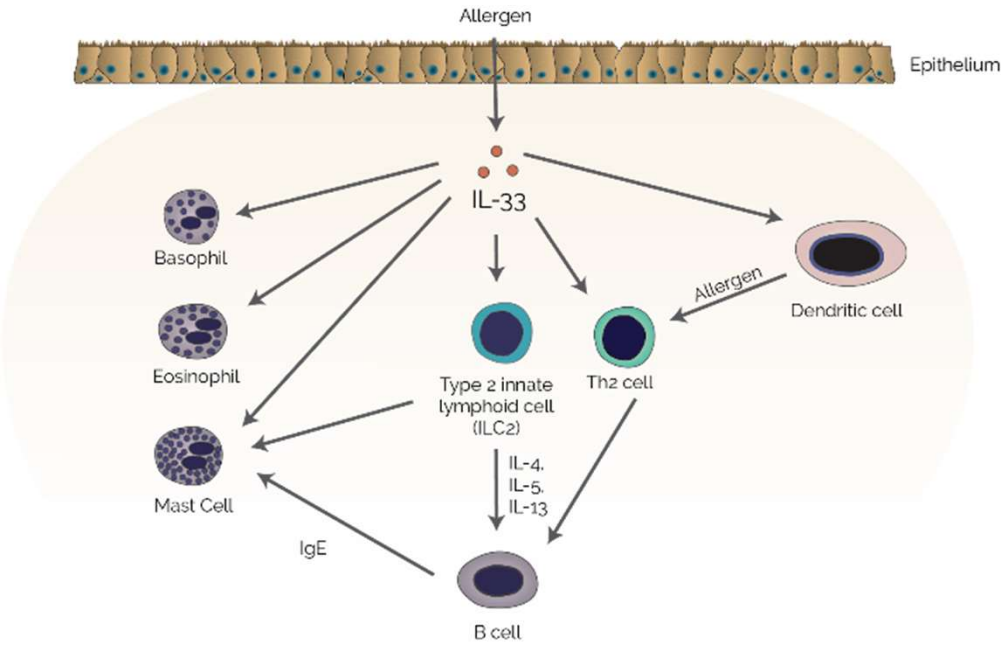
IL-33 is genetically associated with asthma

- IL-33 loss-of-function mutations protect against asthma, while gain-of-function mutations increase asthma incidence
- Translational studies have demonstrated IL-33's role as a pro-inflammatory cytokine released upon allergen contact with epithelium

IL-33 pathway derisked in COPD

(positive Phase 2 data via AZ and REGN/SNY)

Broad commercial opportunity in additional non-respiratory diseases: allergy, epithelial driven diseases in GI and nephrology TAs



- IL-33 is active in its reduced form and is quickly oxidized into an inactive form as a mechanism to limit its local activity
- The majority of IL-33 in the body is the inactive oxidized form

Given etokimab's MOA, it specifically inhibits only the IL-33 molecules that are driving activity and not "wasted" by binding to non-active oxidized IL-33

Etokimab is Phase 2b/3 Ready

(drug supply on hand, preclinical toxicology, P2 data, and competitor POC data across respiratory diseases)