Corporate Overview

March 2024

AnaptysBio

Safe harbor statement

This presentation and any accompanying oral presentation contains forward-looking statements within the meaning of the "safe harbor" provisions of the Private Securities Litigation Reform Act of 1995, including, but not limited to: the timing of the release of data from the Company's clinical trials, including rosnilimab's Phase 2b clinical trial in rheumatoid arthritis and Phase 2 clinical trial in ulcerative colitis and ANB032's Phase 2b clinical trial in atopic dermatitis; the timing of IND filings for ANB033 and ANB101; whether any of the Company's product candidates will be best in class or optimized; the potential to receive any additional 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.

Certain information contained in this presentation may be derived from information provided by industry sources. The Company believes such information is accurate and that the sources from which it has been obtained are reliable. However, the Company cannot guarantee the accuracy of, and has not independently verified, such information.

The trademarks included herein are the property of the owners thereof and are used for reference purposes only. Such use should not be construed as an endorsement of such products.

Best-in-class immune cell modulating antibodies

Immune Cell Modulators

Rosnilimab
(PD-1 agonist)

P2b in
Rheumatoid Arthritis

P2 in
Ulcerative Colitis

ANB032
(BTLA agonist)

P2b in
Atopic Dermatitis

ANB033
(CD122 antagonist)

ANB101
(BDCA2 modulator)

IND-enabling

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

Etokimab
(IL-33)

P2b/3-ready in
epithelial driven diseases

Research-driven

Preclinical pipeline of immunology targets

Strong capital position

Cash runway to YE 2026
YE 2023 cash of ~$417MM

GSK immuno-oncology financial collaboration

Significant royalty potential

Immune cell modulator development

Three P2 trials ongoing across three therapeutic areas; Top-line AD data expected by YE 24

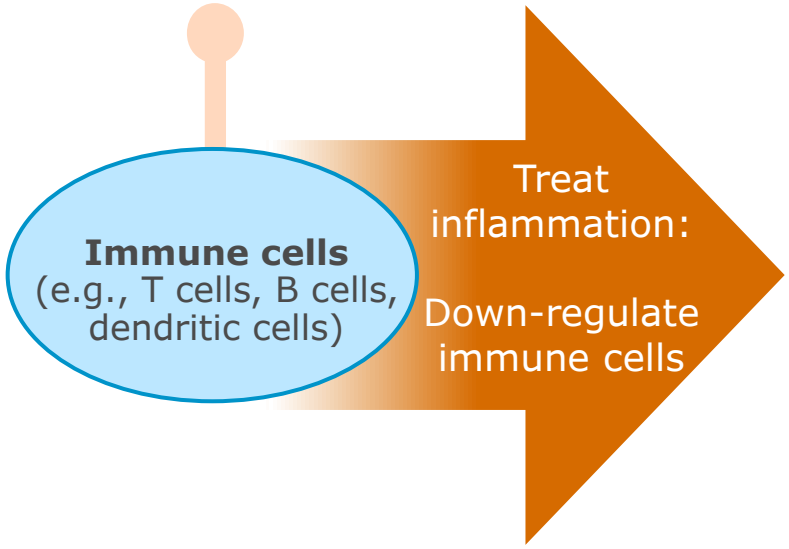
Development Stage and Anticipated Milestones

	Antibody Program	Therapeutic Indication	Lead Optimization / IND Enabling / Phase 1	Phase 2	Phase 3
Immune Cell Modulators	**Rosnilimab** (PD-1 agonist)	Rheumatoid Arthritis		P2b initiated Q3 2023; Top-line data mid 2025	
		Ulcerative Colitis		P2 initiated Q4 2023; Top-line data H1 2026	
	ANB032 (BTLA agonist)	Atopic Dermatitis		P2b initiated Q2 2023; Top-line data YE 2024	
	ANB033 (CD122 antagonist)	Inflammatory Diseases	IND submission Q2 2024		
	ANB101 (BDCA2 modulator)	Inflammatory Diseases	IND submission H2 2024		

Legacy Programs Available for Out-licensing

Cytokine Antagonists	**Imsidolimab** (IL-36R antagonist)	Generalized Pustular Psoriasis (GPP)	*Out-license in 2024*		Submit data abstract to med meeting in H2 2024; GEMINI-1 BLA Q3 2024
	Etokimab (IL-33 antagonist)	Epithelial Driven Diseases	*No further internal investment*	P2b/3 ready	

Checkpoint agonists “hit the brakes” to restore immune balance and deliver differentiated outcomes



Rosnilimab (PD-1 agonist) targets PD-1+ T cells through 3 MOAs:

1. Deplete PD-1^{high} Teff cells
2. Deplete PD-1^{high} Tfh/Tph cells
3. Agonize PD-1^{int} Teff cells

ANB032 (BTLA agonist) modulates activated immune cells:

1. Agonize T cells (Th1, Th2, Th17, Th22)
2. Modulate dendritic cells
3. Agonize B cells

Membrane-proximal binding epitope and optimized Fc receptor binding affinity enables tight immune synapse and best-in-class potency

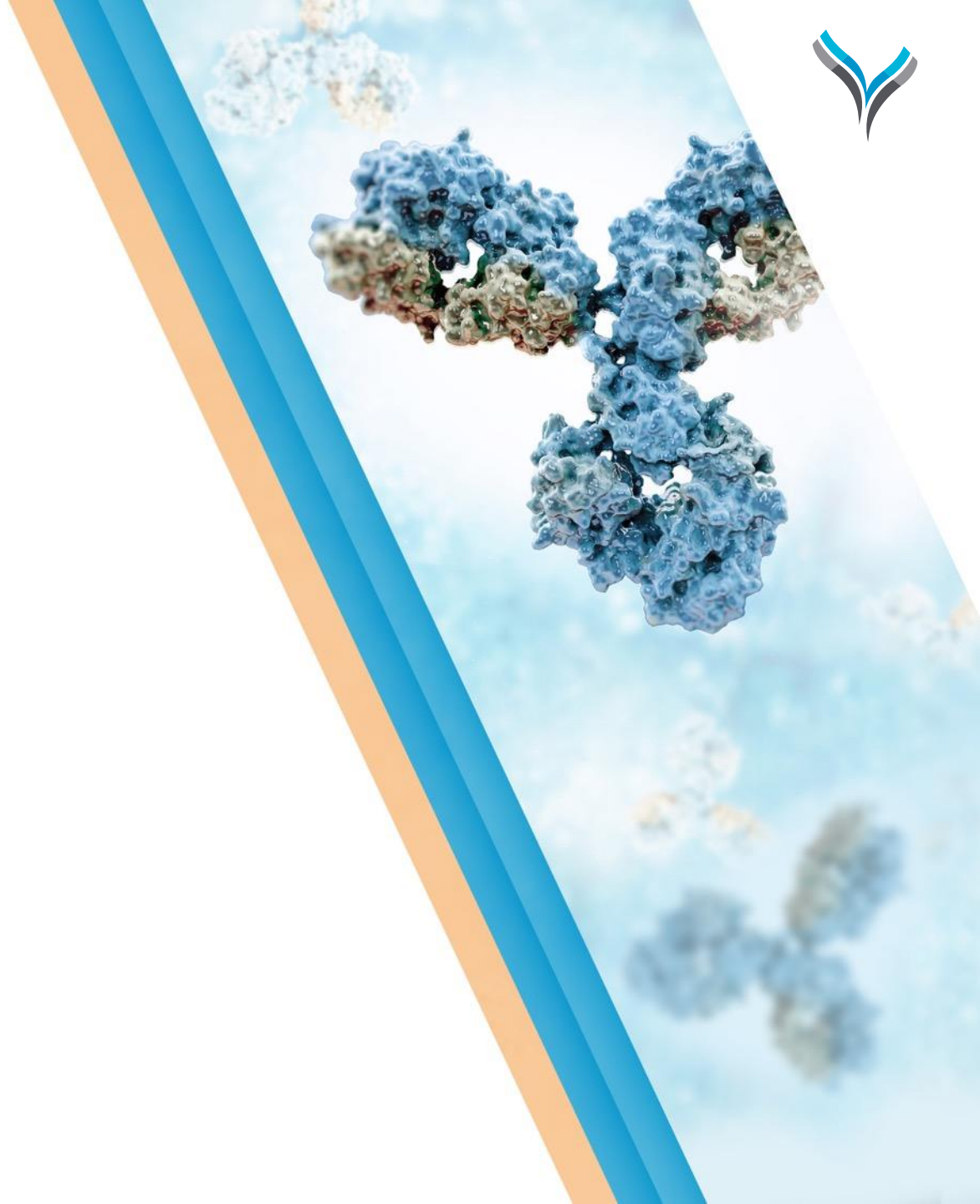
Teff=T effector cells; Tph cells=Peripheral helper cells; Tfh cells=Follicular helper cells.

Anaptys' checkpoint agonists combined attributes contribute to best-in-class potency

		PD-1 Agonist		BTLA Agonist	
		Rosnilimab (IgG1k)	**Lilly's Peresolimab** (IgG1k)	**ANB032** (IgG4)	**Lilly's LY-3361237** (IgG4 PAA[1])
Structural characteristics	Membrane-proximal epitope	✓	✗	✓	✗
	Fc receptor binding affinity	✓	✓	✓	✗
	Binding properties	✓	✓	✓	✓
Functional outputs	Agonism	✓	✗ **Significantly Decreased**[2]	✓	✗ **Significantly Decreased**[2]
	Depletion	✓	✓ **Decreased**[2]	**None**[3]	**None**[3]

1. IgG4 PAA (S228P/F234A/L235A) is a variation of IgG4 specifically engineered to eliminate FcγR affinity
2. Membrane-distal binding epitope results in wider immune synapse, contributing to significantly weaker agonism and less potent depletion
3. Depletion of PD-1^{high} Teff and Tfh/Tph cells is expected to contribute to PD-1 agonist clinical efficacy. However, given the broader expression profile of BTLA on T cells, B cells and DCs, a non-depleting antibody is preferred

Rosnilimab
(PD-1 agonist mAb)



Rosnilimab's impact in inflamed tissue and periphery has potential to deliver differentiated efficacy and safety

Rosnilimab's 3 MOAs impact on T cells	1. Reduce PD-1^{high} Teff cells	2. Reduce PD-1+ T cell activation	3. Reduce PD-1^{high} Tfh/Tph cells
Immunologic outcomes	Reduce T cell migration, proliferation & cytokine secretion		Reduce plasma cell generation & autoantibody levels

Relevant Approved Treatments[1]

HUMIRA adalimumab (TNF)

ACTEMRA tocilizumab (IL-6)

ORENCIA (abatacept) (CD-80)

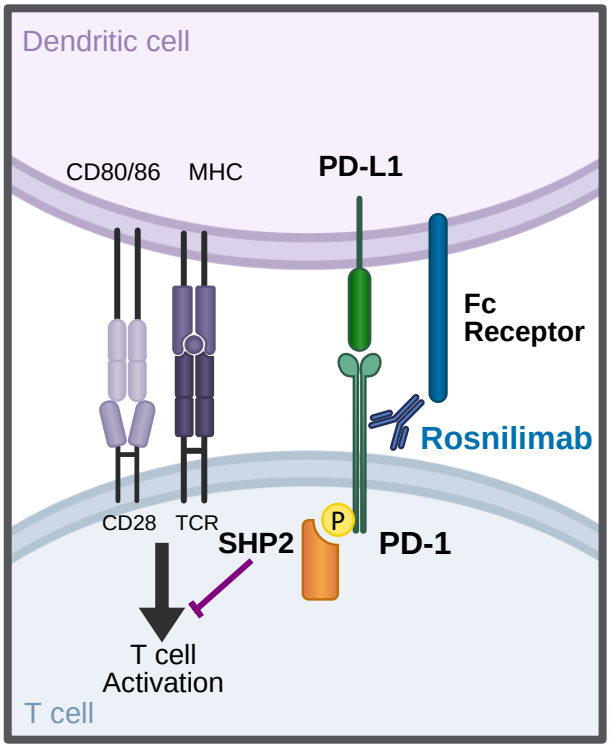
Entyvio vedolizumab (α4β7)

ZEPOSIA (ozanimod) (S1P)

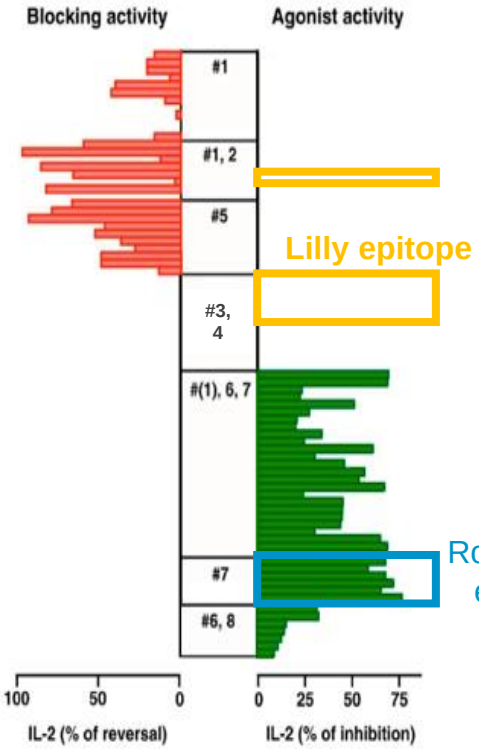
These treatments only target a subset of the immunologic outcomes driven by rosnilimab

[1]Other efficacious treatments have less specific MOAs causing safety liabilities, including Rinvoq (JAKs), Rituxan (broad B cell depleter), Lemtrada (broad lymphocyte depleter)

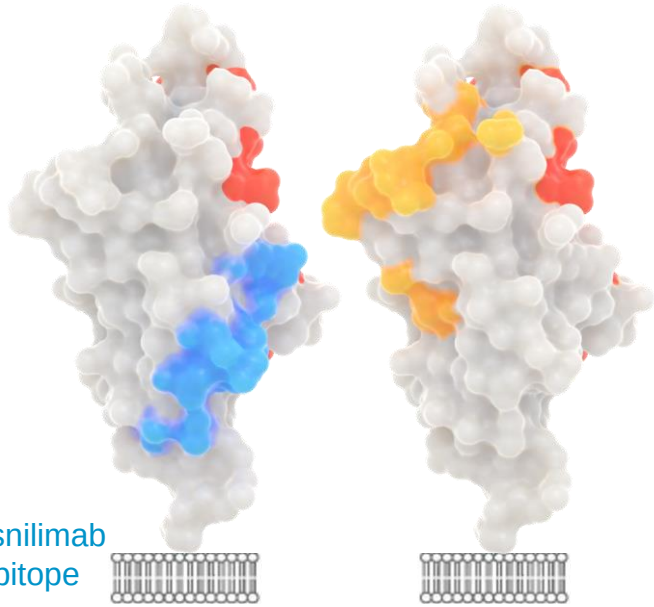
Rosnilimab optimizes PD-1+ T cell inhibitory signaling by enabling tight immune synapse formation

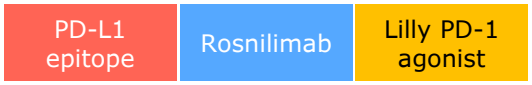


Functional assay of antagonism or agonism[1]



Rosnilimab | **Lilly PD-1 agonist**





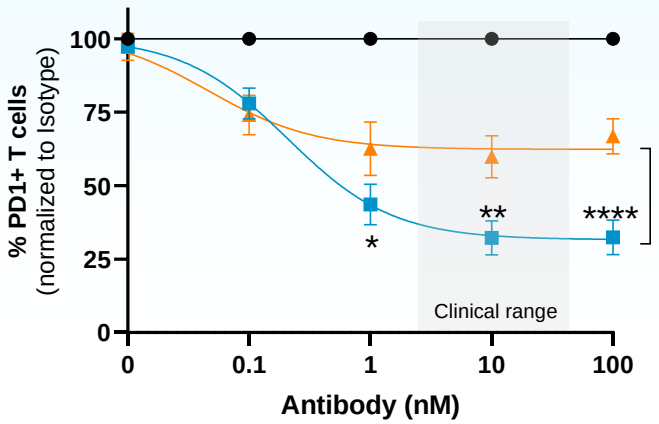
"A shared feature of agonist mAbs is recognition of the membrane-proximal extracellular region…" and "…activity depends on Fc receptor–supported crosslinking"

Suzuki, et al. 2023

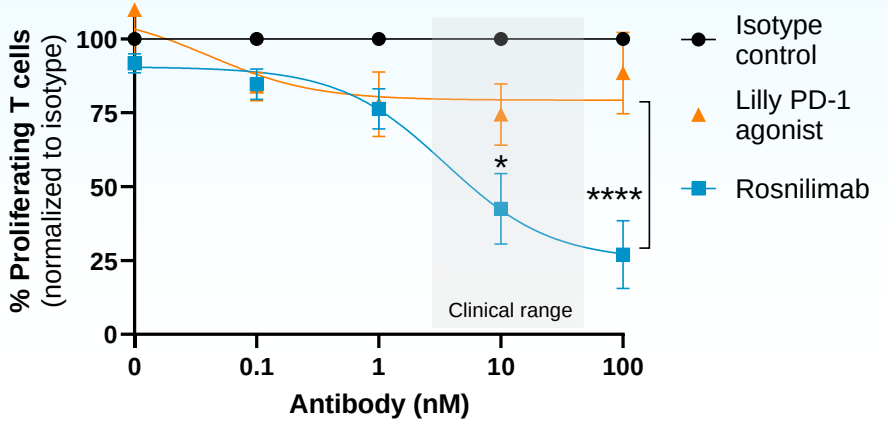
1. Adapted from Suzuki et al., Sci. Immunol. 8, eadd4947 (2023).

Rosnilimab demonstrates potent depletion and agonism at clinically relevant concentrations

Depletion (NK cell-mediated ADCC)[1]



Agonism (DCs + total T cells; no ADCC)[2]



1. Healthy donor T cells + NK cells (1:5 ratio) + antibody in in-vitro ADCC assay, representative data from N=5 donors.
2. Healthy donor purified DCs + autologous total T cells stimulated with anti-CD3, cultured for 3 days for assessment of T cell proliferation
   Two-way ANOVA. Tukey's multiple comparison test. ****P<0.0001, ***p<0.001, **p<0.01, *p<0.05.

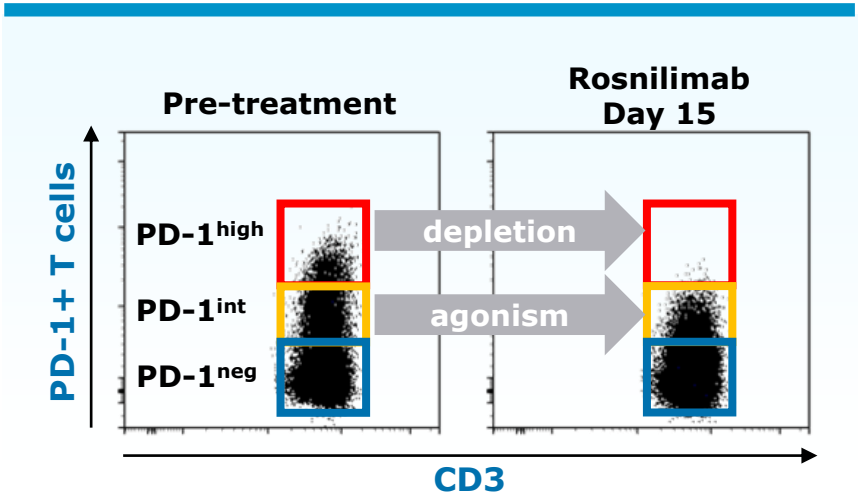
Rosnilimab restores immune balance bringing T cell composition to a less activated state

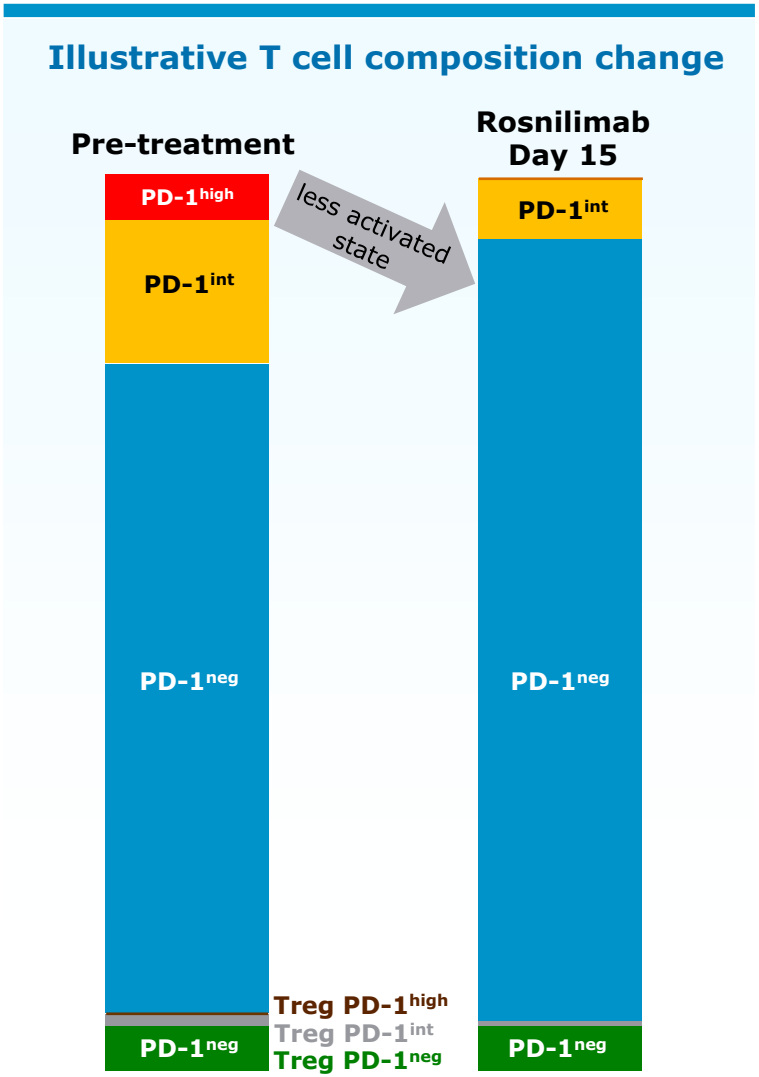
PD-1 expression on both CD4 and CD8 T cells correlates with activation state

Rosnilimab targets only a small proportion of T cells

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

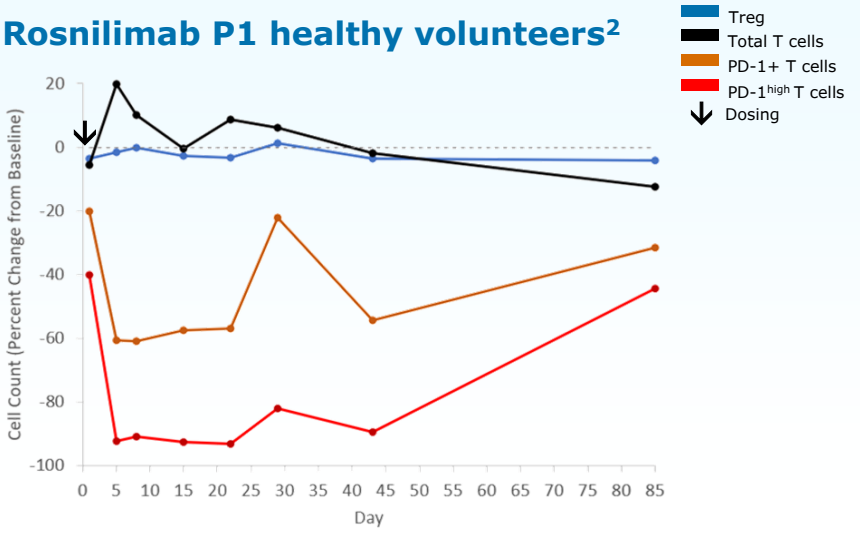




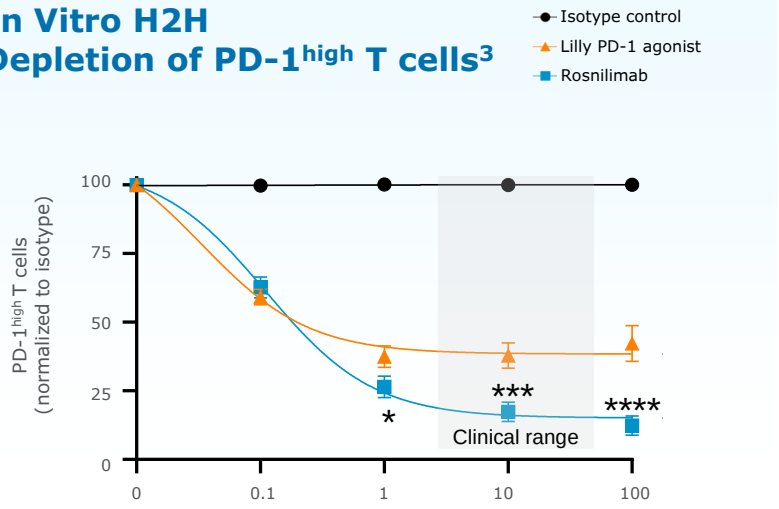
Data illustrative, based on Phase 1 data from healthy volunteer study. Data on file with Anaptys.

Comparative data of rosnilimab demonstrates consistently higher reduction of peripheral PD-1^{high} and PD-1+ T cells

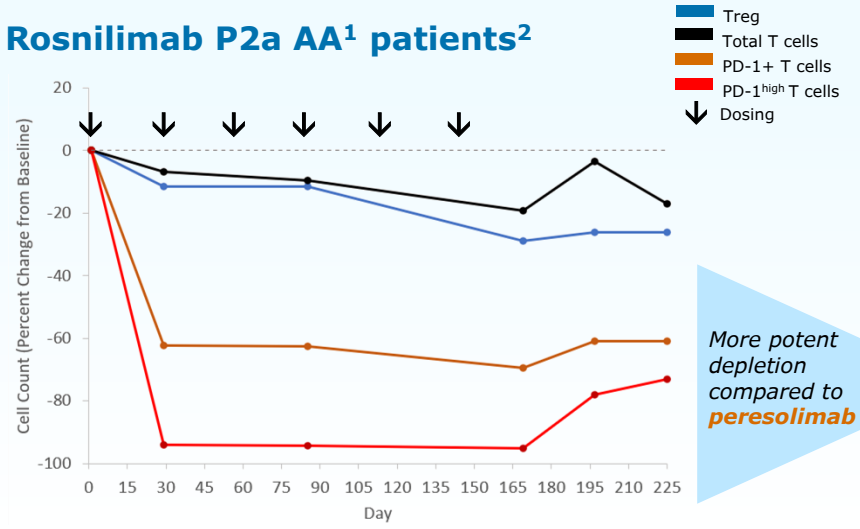
Rosnilimab P1 healthy volunteers[2]



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

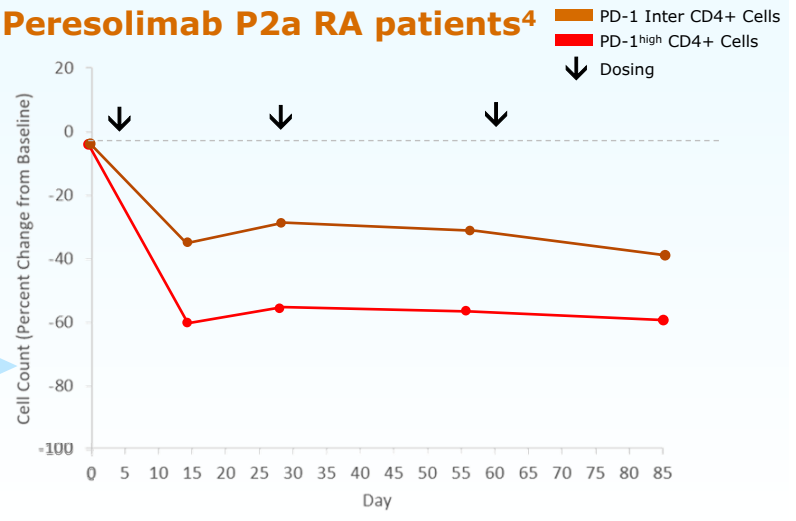


Rosnilimab P2a AA[1] patients[2]



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

Peresolimab P2a RA patients[4]



1. AA=Alopecia Areata; 2. Rosnilimab clinical results shown from 400mg subcutaneous dose (single dose in healthy and monthly dose in AA); 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, and overall PD-1 agonist class, well-tolerated with no dose limiting tox observed to date

Rosnilimab Phase 1: 144 healthy volunteers (HV) in SAD and MAD cohorts

- Supports monthly SC dosing
- Favorable safety and tolerability
  - No SAEs related to rosnilimab[1]
  - No carcinogenic events observed
  - No infection risk signal

Rosnilimab P2a in alopecia areata (AA) for Q4W SC dosing for 6 months was well tolerated with no significant safety signals

PD-1 agonist class: consistent tolerability profile to date

- Competitor PD-1 programs no carcinogenic events or infection risk signal
- >100+ RA patients treated with Lilly PD-1 agonist (highest dose of 700 mg IV over 6 months) showed tolerable profile[2]

Abatacept, targeting all T cells, has not shown clinically relevant carcinogenic increases in decades of commercial use

SAD=Single ascending dose; MAD=Multiple ascending doses; RO=Receptor occupancy; PK=Pharmakokinetics, SC=subcutaneous.

1. MAD cohort no SAEs; SAD cohorts 2 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).

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

Rheumatoid arthritis:

~500,000 patients
>$10bn U.S. sales in "post TNF" market[1]

20-25% cycle
all treatment classes
not achieving low disease activity[2]

Ulcerative colitis:

~100,000 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

Standard of care is insufficient and fragmented

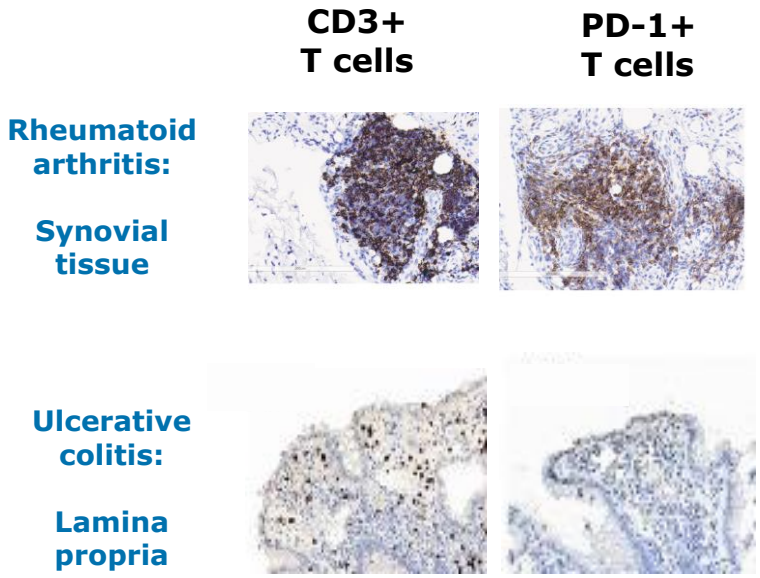
- RA (Post TNF): ~20–35% ACR50
- UC: ~25-30% clinical remission induction

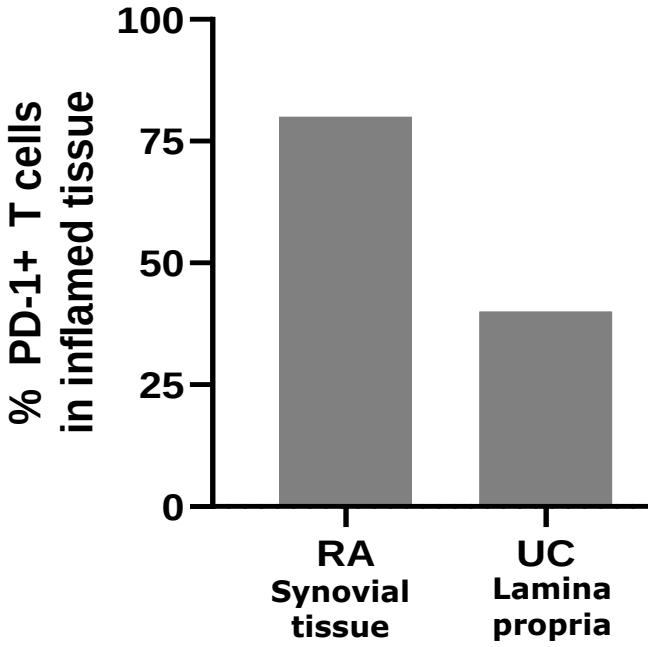
Significant room to differentiate

- Drive deeper responses across broader patient population
- Restore immune balance

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.

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





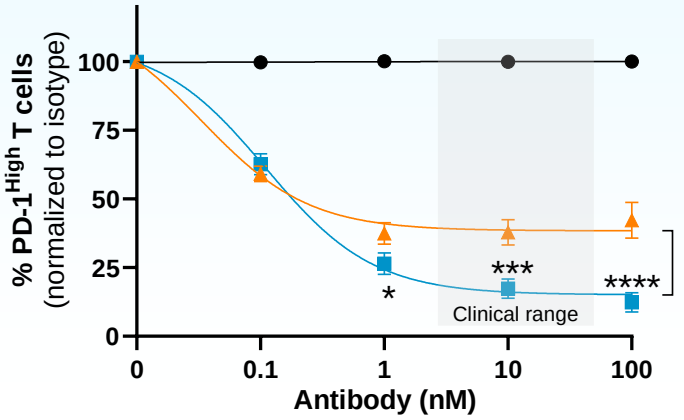
In systemic inflammatory diseases, a multiple fold increase of PD-1+ T cells is observed in periphery compared to healthy controls[1]

- **~1.5x in RA**
- **~2x in UC**

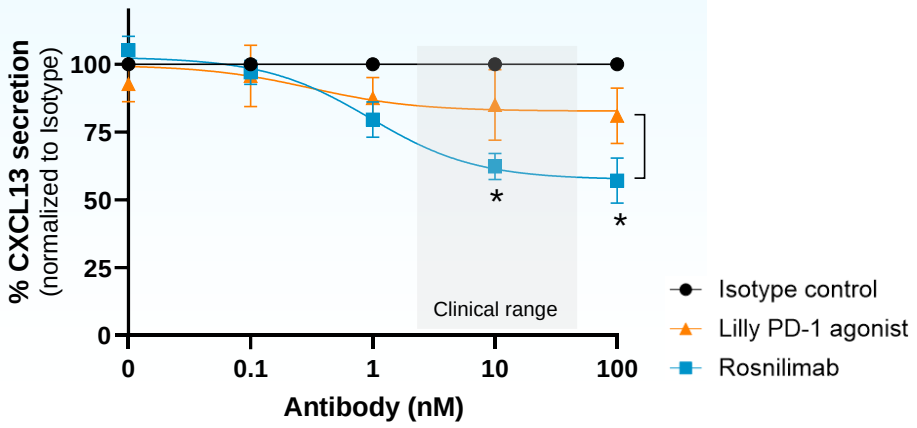
Adapted from Nguyen et al, Human Pathology (2022) 126, 19e27; Guo et al, PLoS One 2018; 13(2). Roosenboom et al, Scand J of Gastro. 2021; 56(6):671-679. 1. Murray-Brown et al, RMC Open, 2022. Shi et al. (2023), PeerJ, DOI 10.7717/peerj.15481.

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

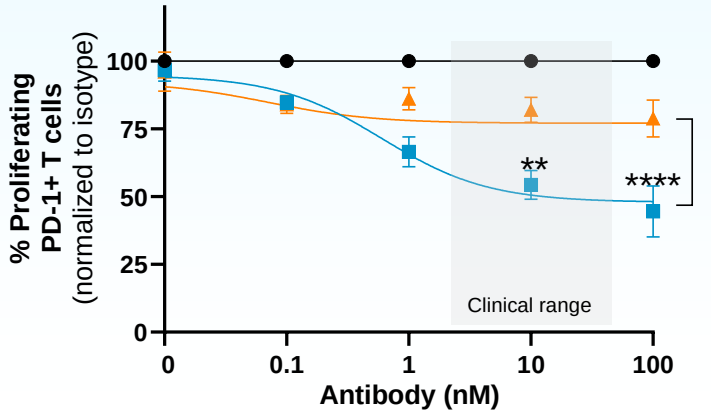
Depletion of PD-1^{high} T cells



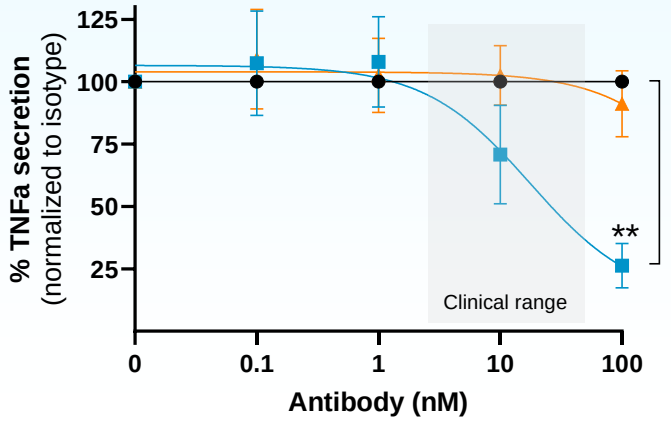
Inhibition of Tfh/Tph chemokine



Inhibition of T cell proliferation

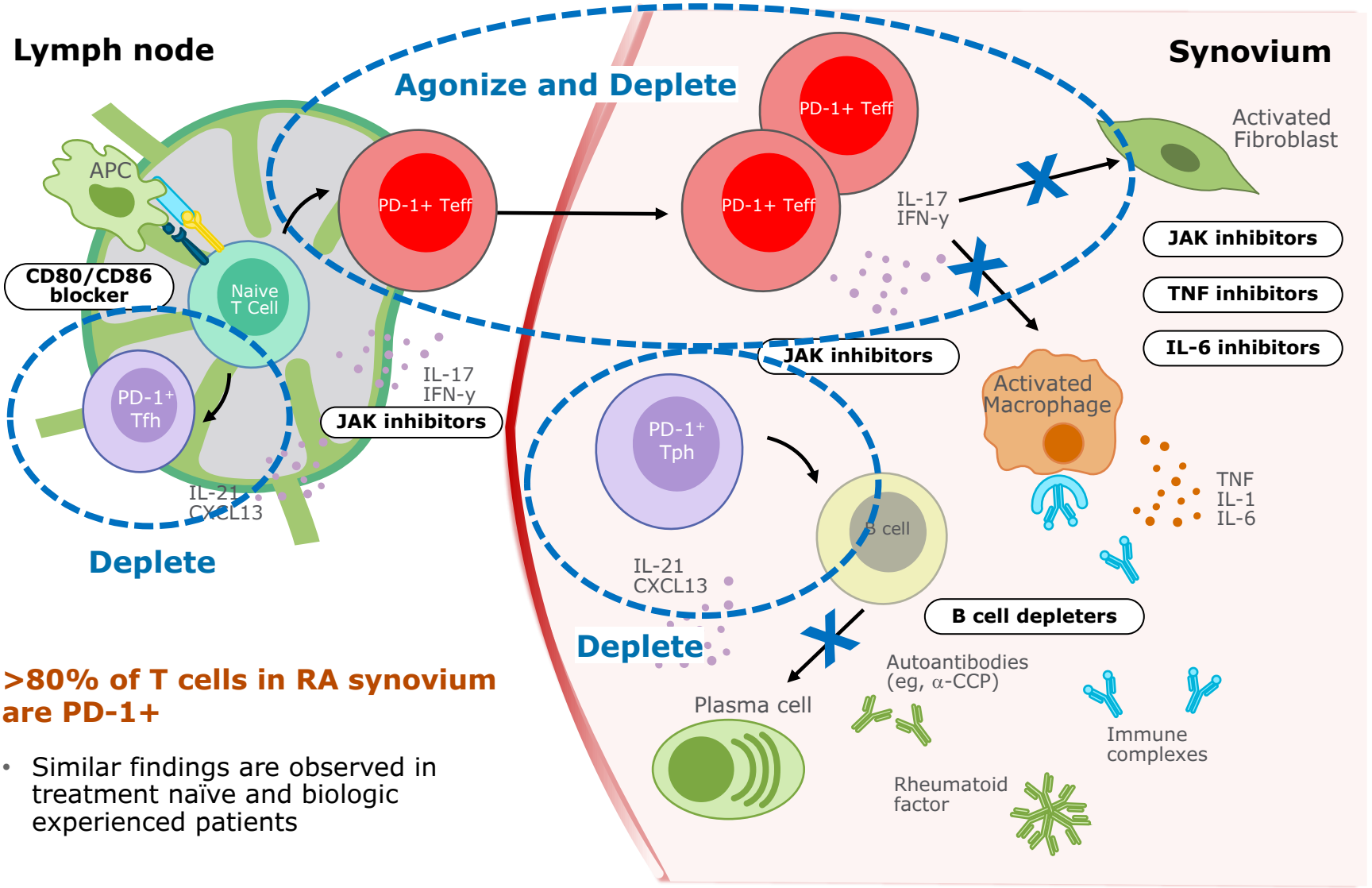


Inhibition of inflammatory cytokine[1]



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.

PD-1+ T cells broadly impact multiple clinically validated drivers of RA pathogenesis

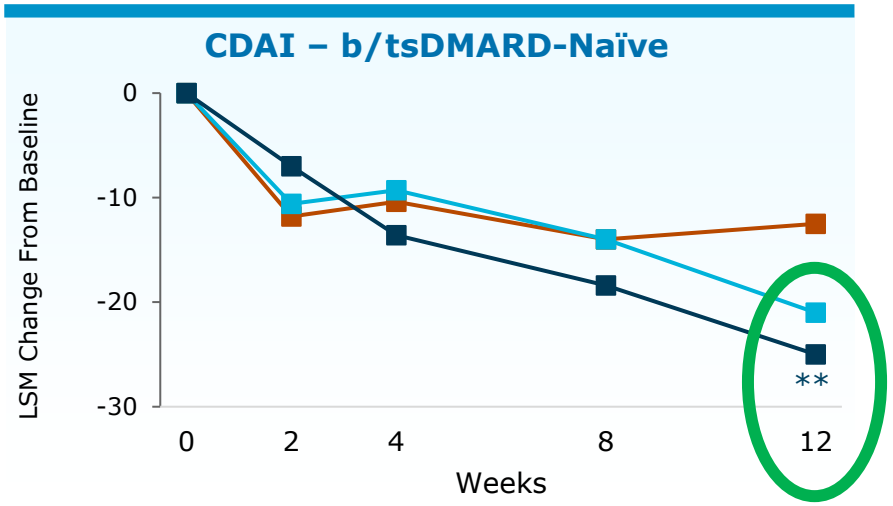


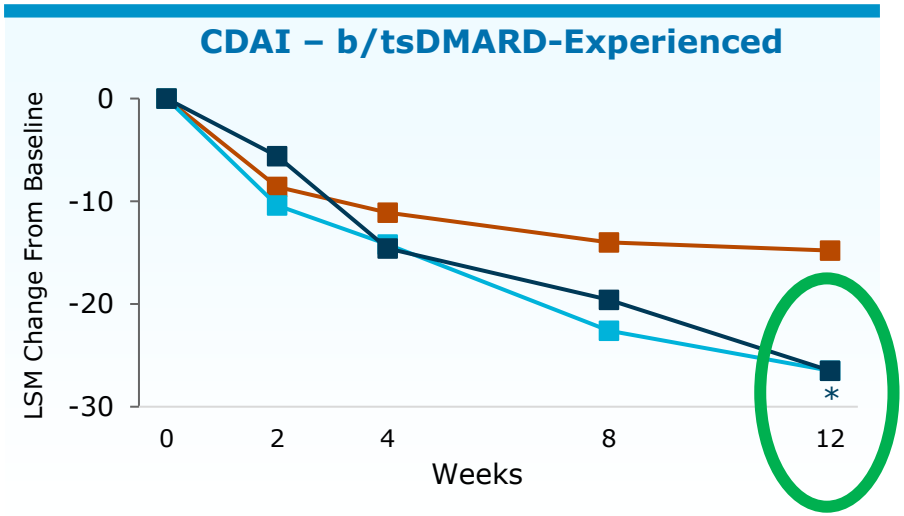
>80% of T cells in RA synovium are PD-1+

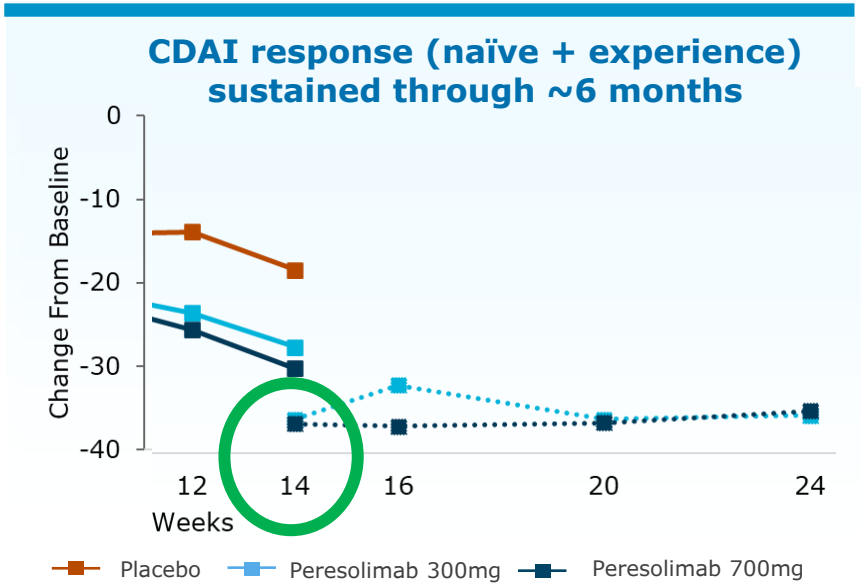
- Similar findings are observed in treatment naïve and biologic experienced patients

Adapted from Aletaha and Smolen, JAMA, 2018.

PD-1 agonist class is clinically validated in RA with compelling proof of mechanism





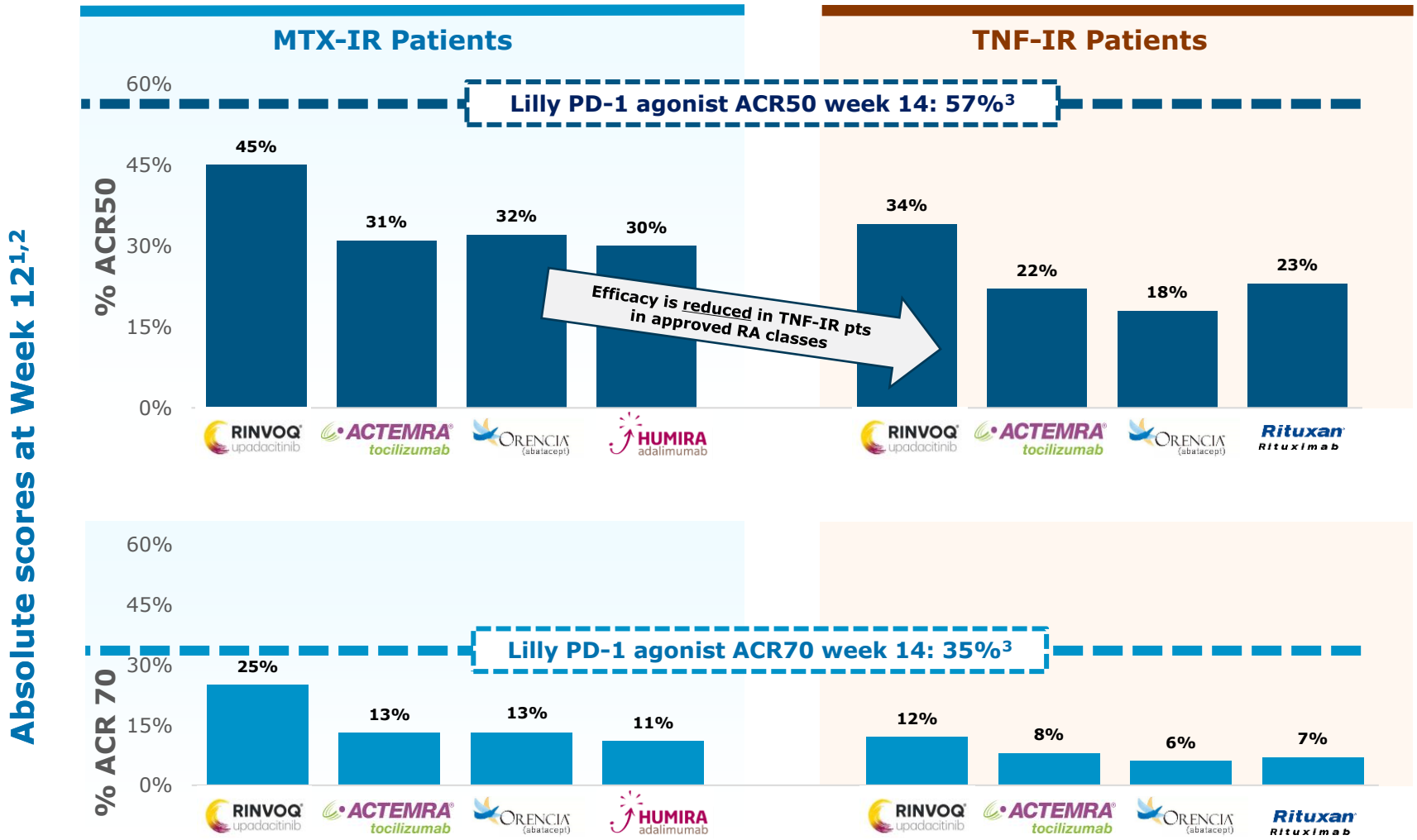


PD-1 agonist emerging profile: Peresolimab 98 patient placebo-controlled P2a study

- No safety signal
- Consistent efficacy across biologic-naïve and biologic-experienced patients
- Week 14 efficacy sustained through 6 months

Tuttle, J. EULAR 2023, Week 14-Week 24 data estimated from peresolimab (PD-1 agonist) 2022 ACR presentation; CDAI=Clinical disease activity index. Sample size for top two charts: placebo n=24; peresolimab 300mg n=25 and peresolimab 700mg n=49. In bottom graph, placebo n=11, peresolimab 300mg n=12 and peresolimab 700mg n=18, **p<0.01, *p<0.05. Green circles indicate separation at key timepoints.

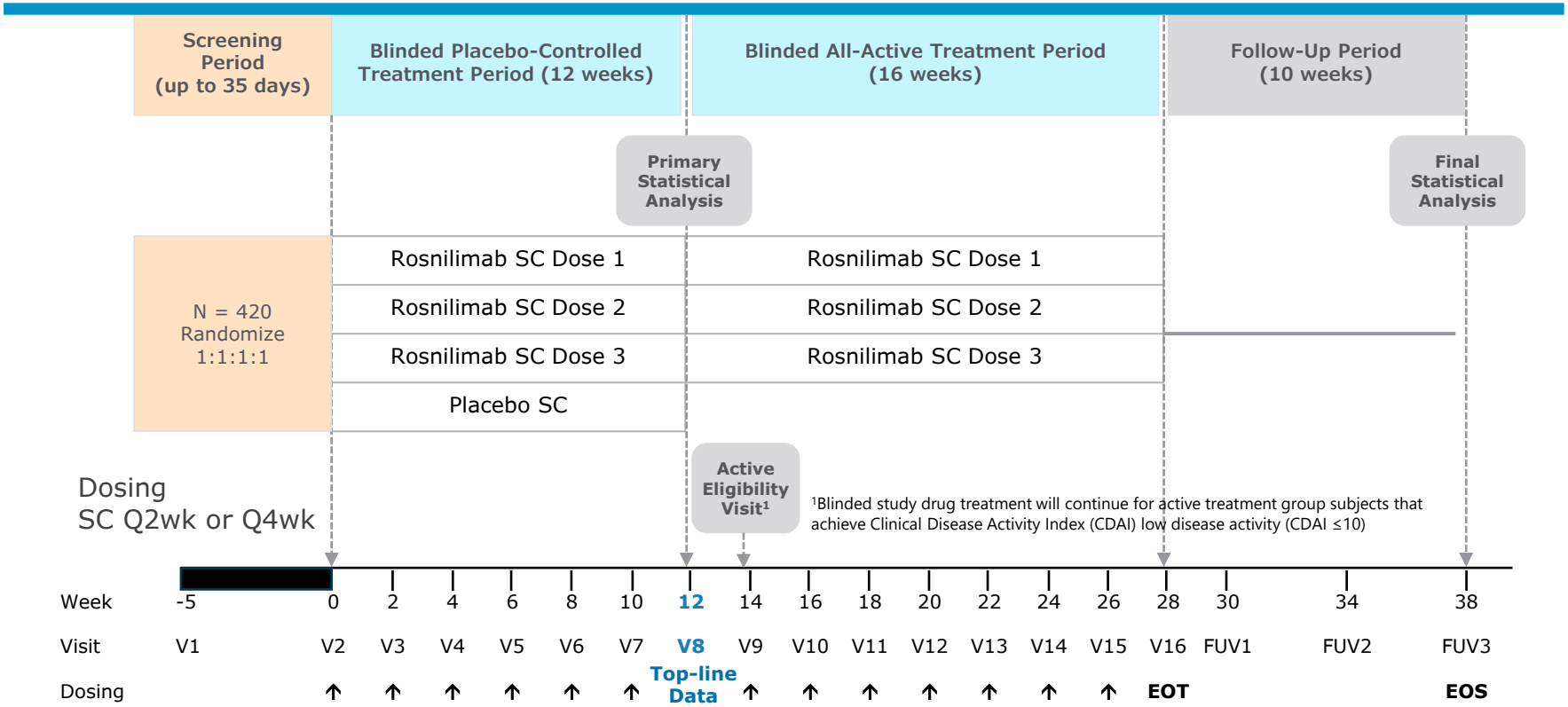
PD-1 agonist class has shown commercially meaningful outcomes (ACR50 and ACR70) regardless of prior treatment



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.

Rosnilimab Phase 2b in moderate-to-severe RA

Initiated Q3 2023; Top-line data mid-2025

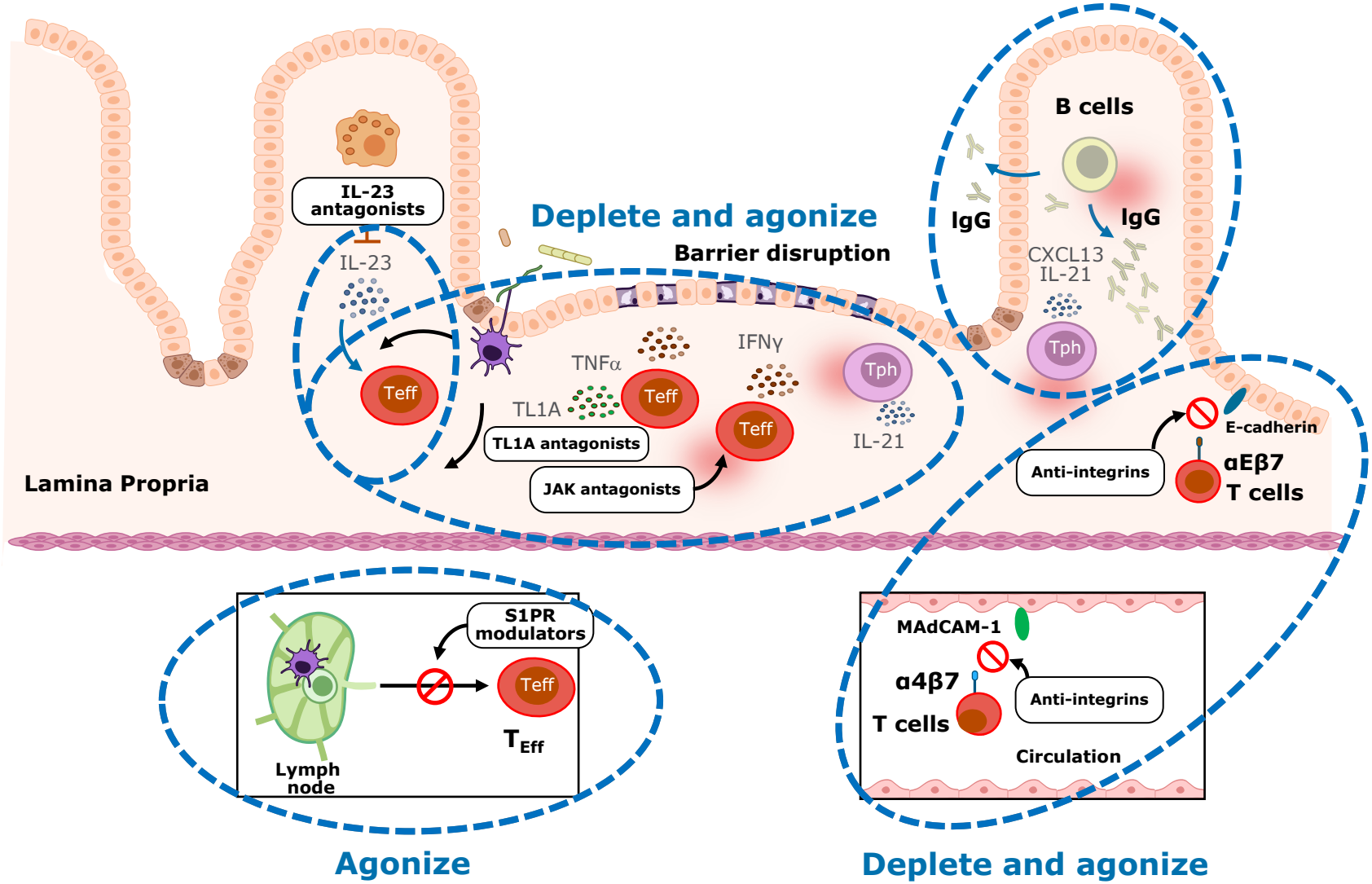


Patient population		• Adults with moderate-to-severe rheumatoid arthritis, >=6 TJC and SJC • Positive RF or CCP • Includes both MTX-IR and b/tsDMARD experienced patients (~50% b/tsDMARD experienced) • IR or intolerance to < 3 classes of b/tsDMARDs
Endpoints	Primary	• Mean change from Baseline in DAS28-CRP 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

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

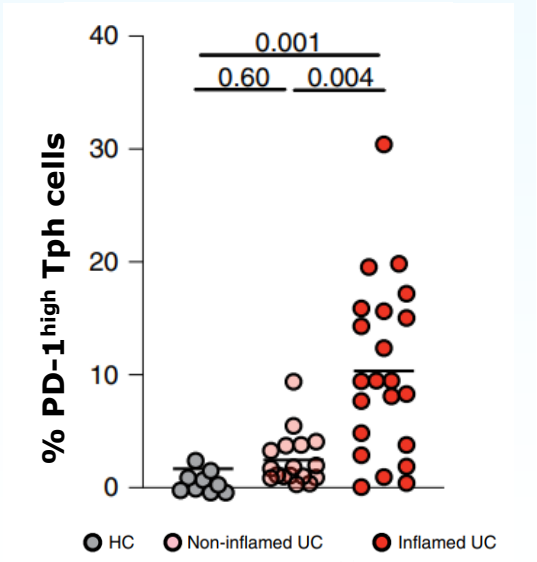
>40% of T cells in lamina propria in UC are PD-1+



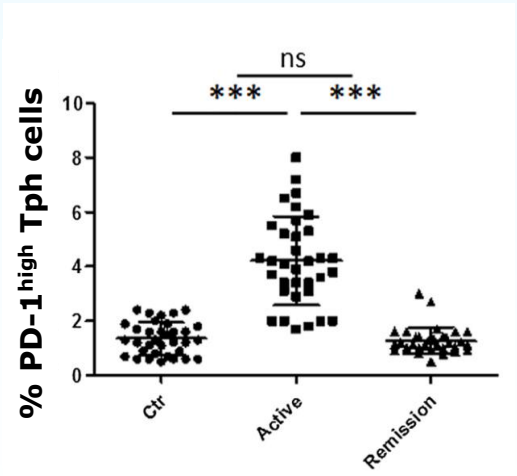
Adapted from Gastroenterology & Hepatology Volume 18, Issue 8 August 2022.

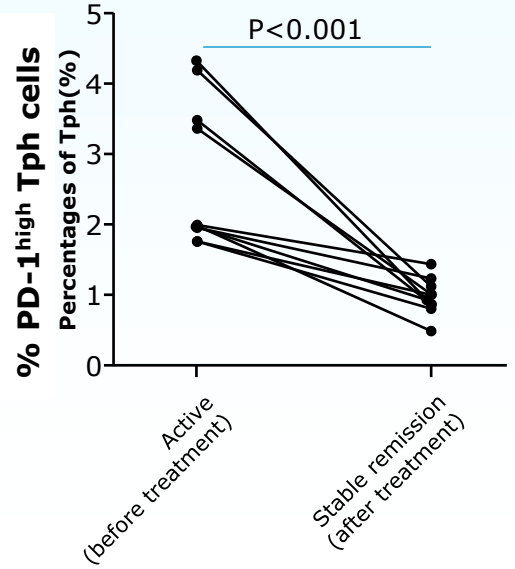
Reduction of elevated PD-1^{high} Tph cells in both UC colon and periphery correlates with remission

PD-1^{high} Tph cells are elevated



PD-1^{high} Tph cells are reduced with remission



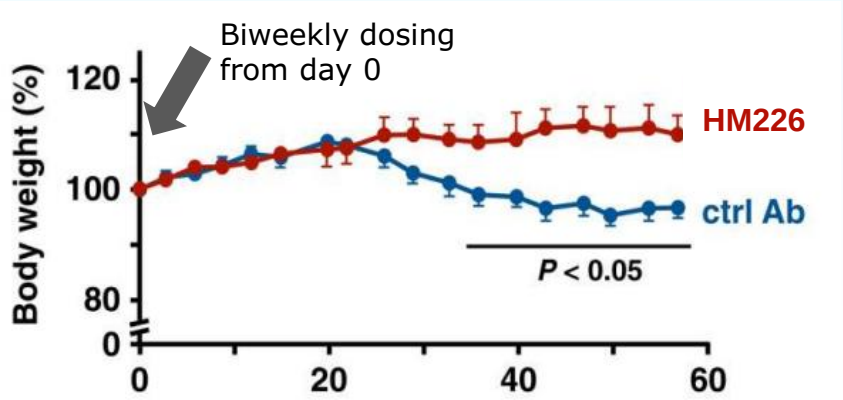


<u>Reduction</u> of plasma cell generation & autoantibody levels, including anti-microbial IgG antibodies contributing to colonic inflammation and barrier disruption

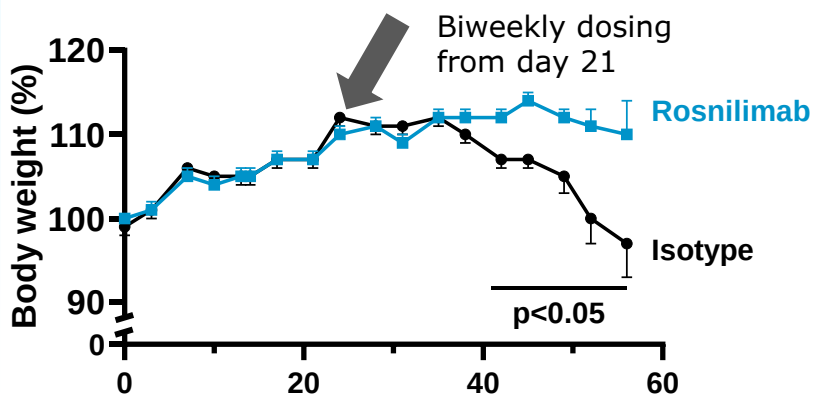
PD-1^{high} Tph cells defined by CD3+CD4+CD45RA-PD-1+TIGIT+ICOS+CXCR5-). Long et al, Immunology Letters 233 (2021) 2-10., Rao et al, Nature, 2017. *** p<0.001.

Prophylactic or therapeutic dosing of PD-1 agonists induce and maintain remission in murine models

Prophylactic dosing of HM226 PD-1 agonist induce and maintain remission from colitis



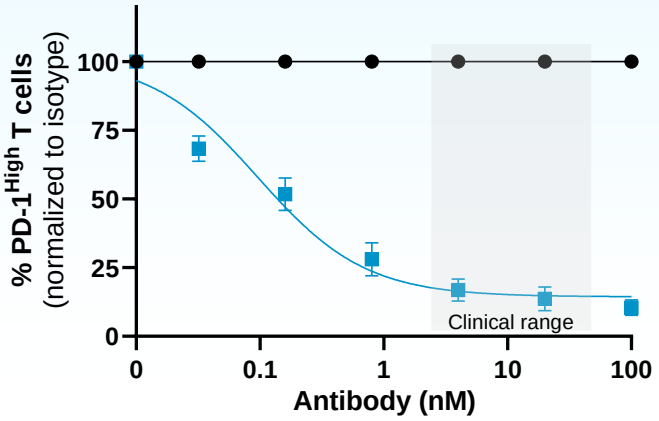
Therapeutic dosing of rosnilimab induce and maintain remission from colitis



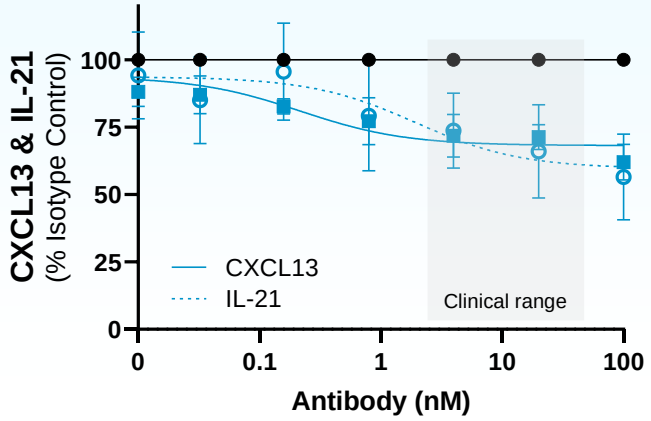
Rosnilimab and HM226 bind to the membrane proximal “epitope 7” of PD-1 that contributes to maximal PD-1 agonism. Rosnilimab formatted to mIgG2a to mediate effector function in mice. Suzuki et al., Sci. Immunol. 8, eadd4947 (2023).

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

Depletion of PD-1^{high} T cells

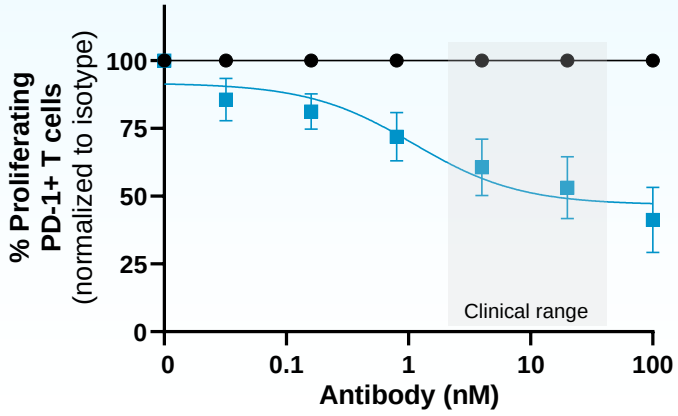


Inhibition of Tfh/Tph cytokine

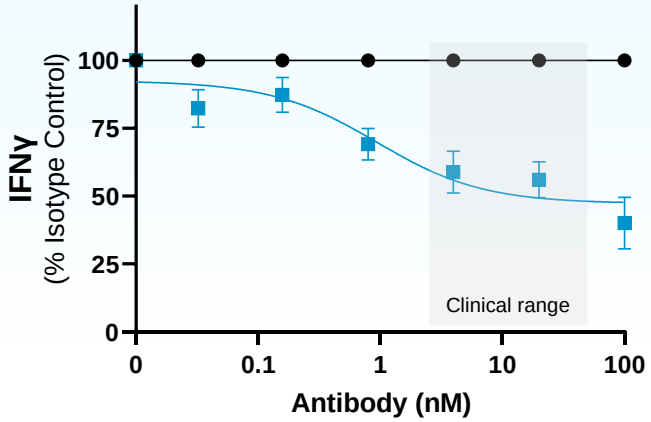


Isotype control
Rosnilimab
(N=6 UC patients)

Inhibition of T cell proliferation



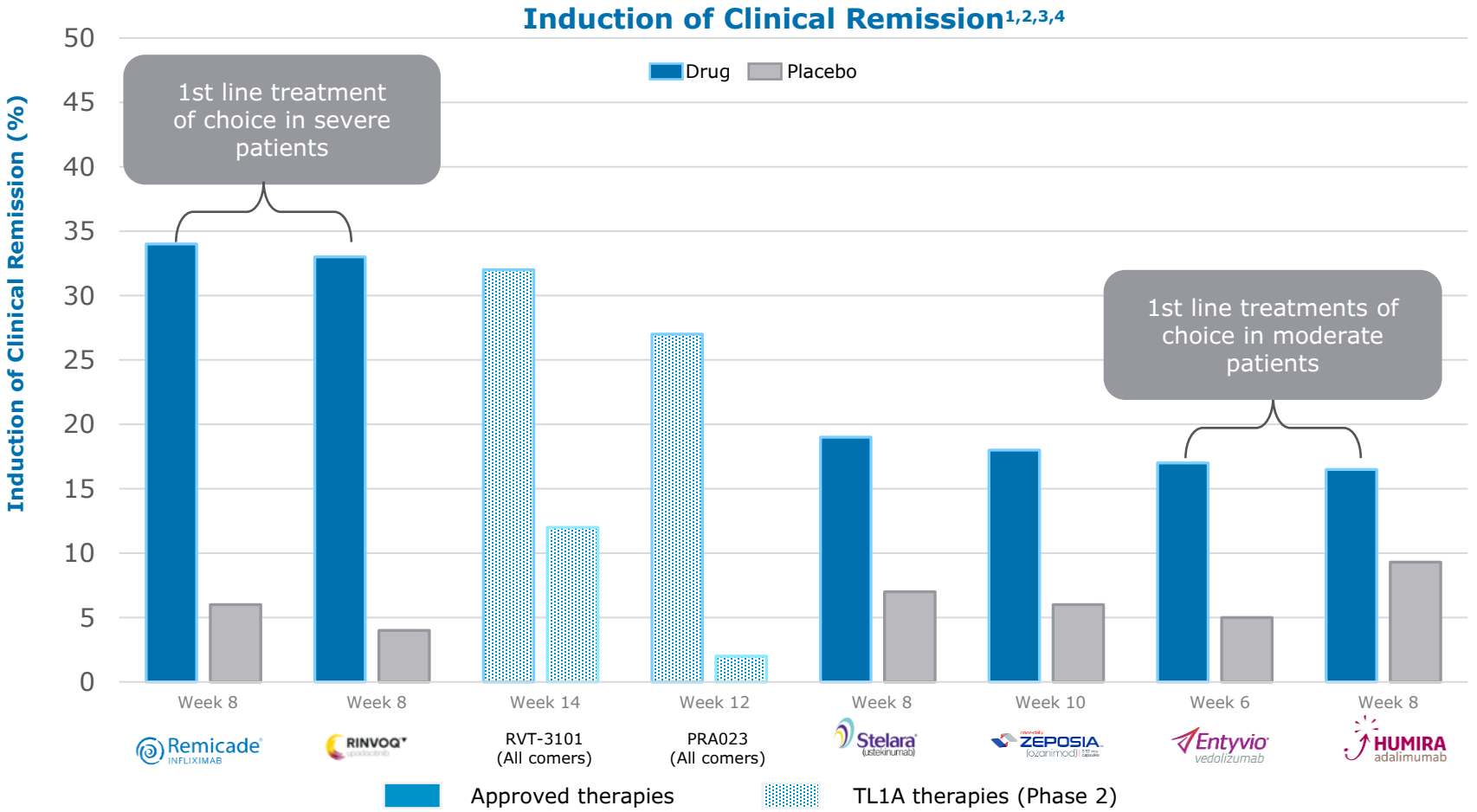
Inhibition of inflammatory cytokine



Anti-CD3+ anti-CD28 stimulation of UC patient PBMCs for assessment of depletion and agonism MOA, representative data from N=6 donors.

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

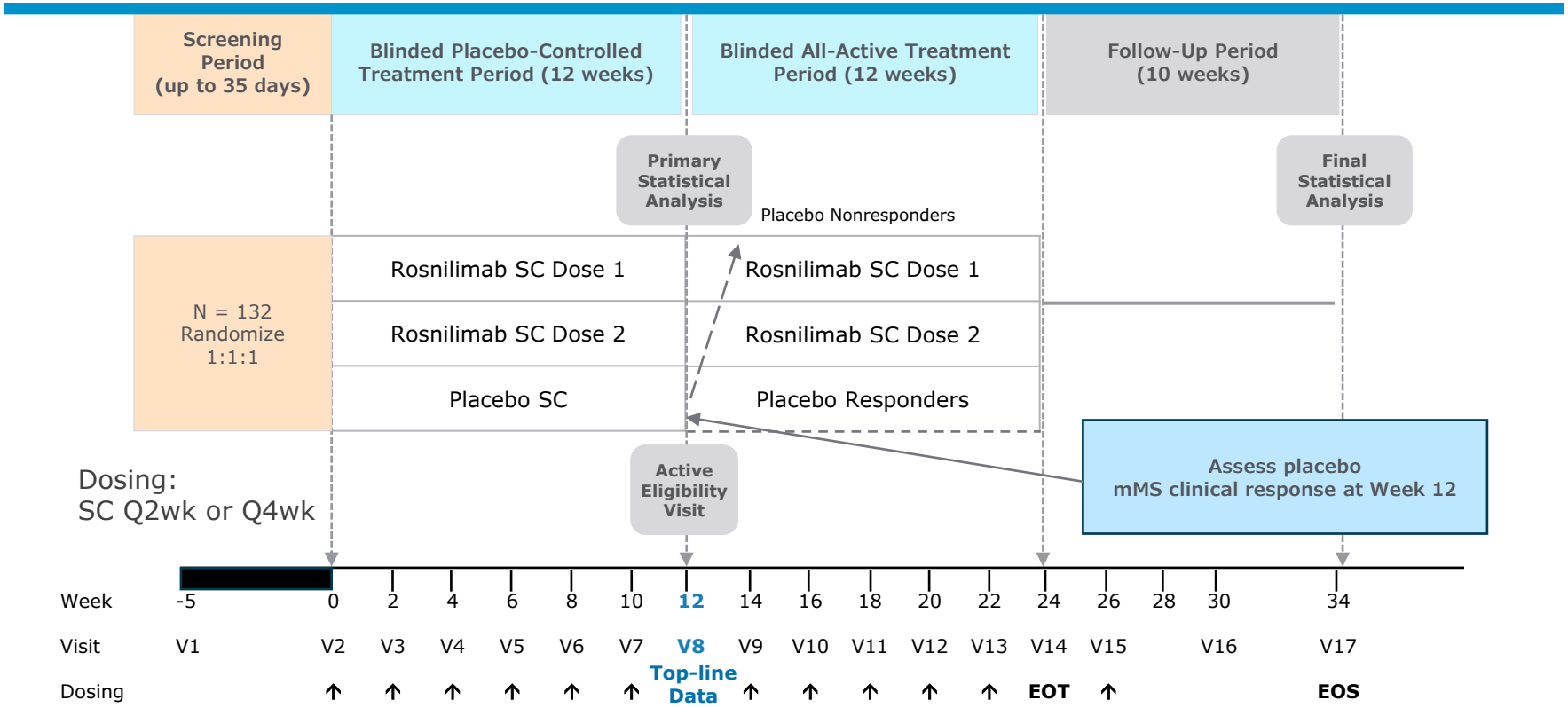
Following remission on induction therapy, one third to one half of patients relapse within 1 year



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

Rosnilimab Phase 2b in moderate-to-severe UC

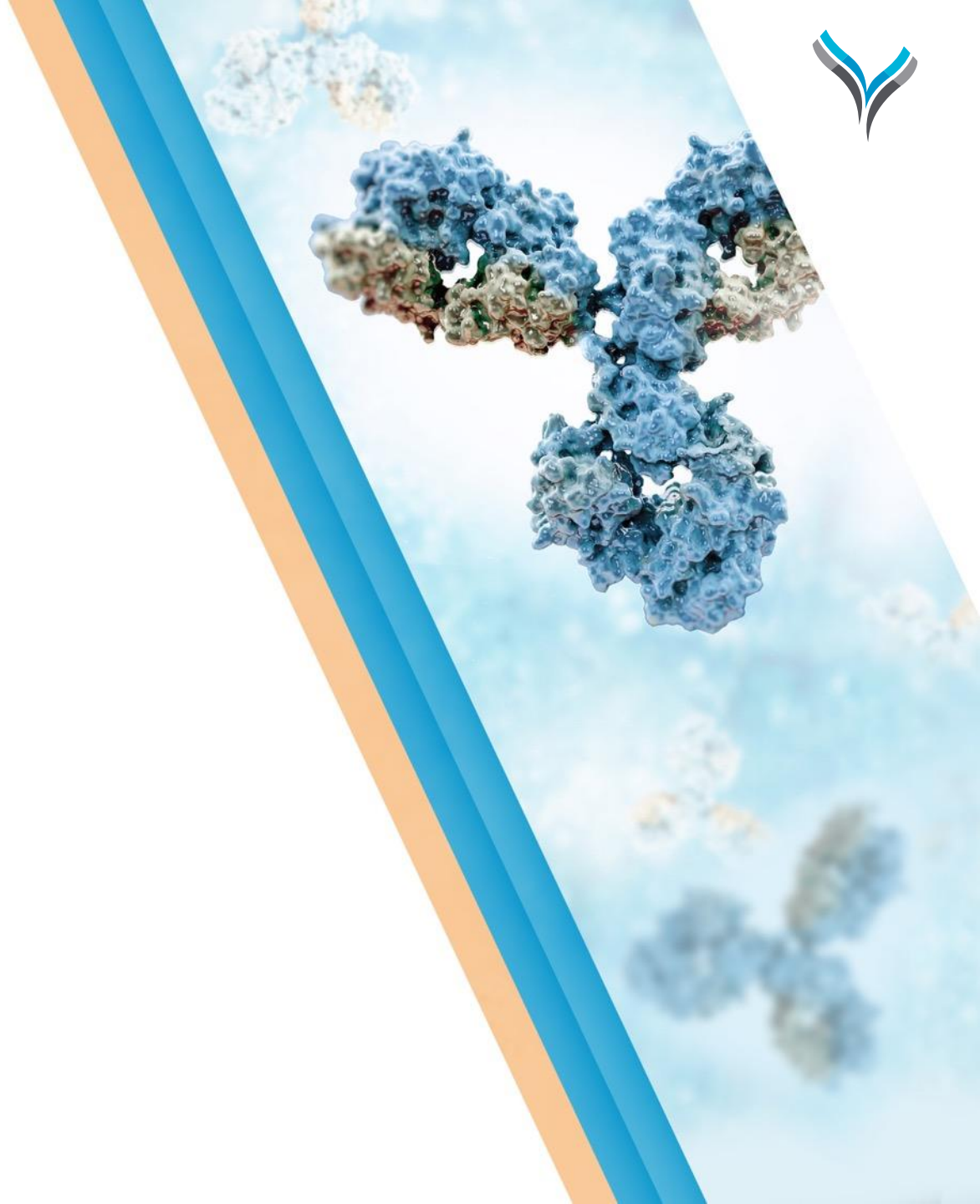
Initiated Q4 2023; Top-line data H1 2026



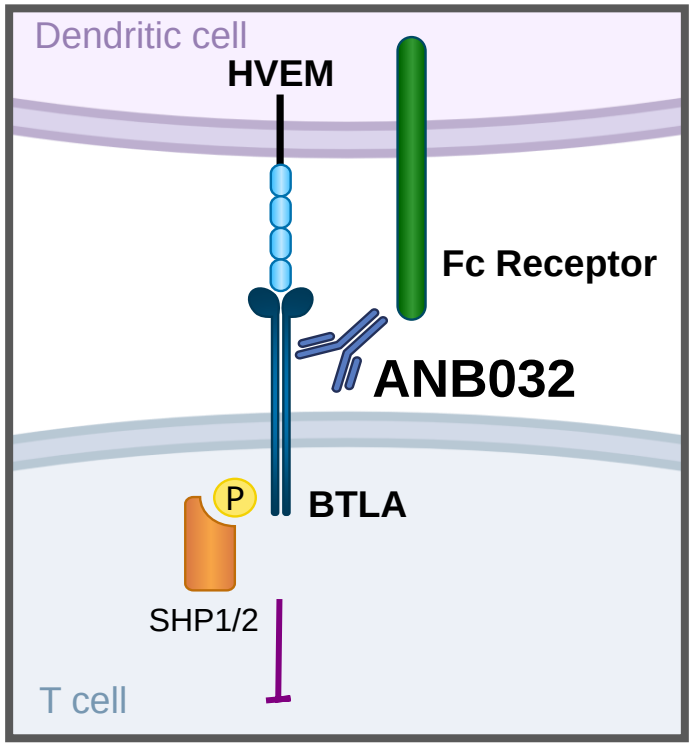
Patient population		• Adults with moderate-to-severe ulcerative colitis • Inadequate response to, loss of response to, or intolerance to as least 1 conventional or advanced UC therapy (~50% advanced UC therapy experienced)
Endpoints	Primary	• Mean change from Baseline in modified Mayo Score (mMS) at 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

ANB032
(BTLA agonist mAb)



ANB032 has potential to treat wide range of systemic inflammatory diseases[1]



BTLA is key node of immune regulation

- B and T lymphocyte attenuator (BTLA) is a potent checkpoint receptor
- Expressed only on immune cells and preferentially on activated immune cells
- Dysregulation of BTLA pathway accelerates onset and exacerbates disease

ANB032: IgG4 antibody (non-depleting)

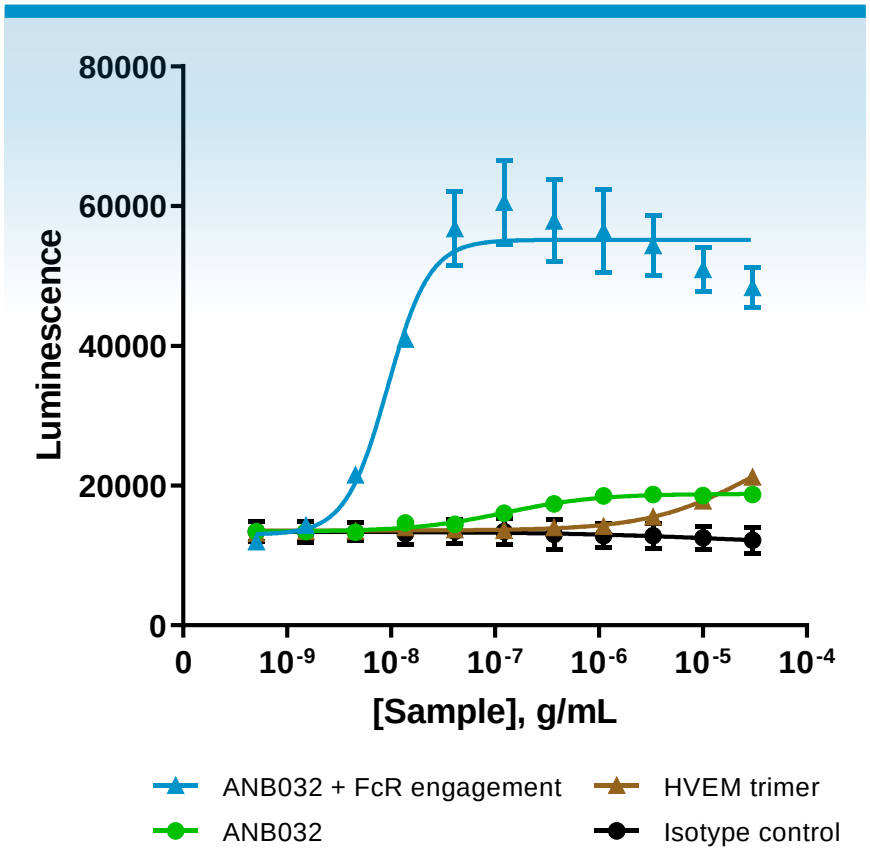
- Binds BTLA proximal to immune cell
- Fc receptor binding contributes to differentiated potency
- Non-blocking of HVEM engagement

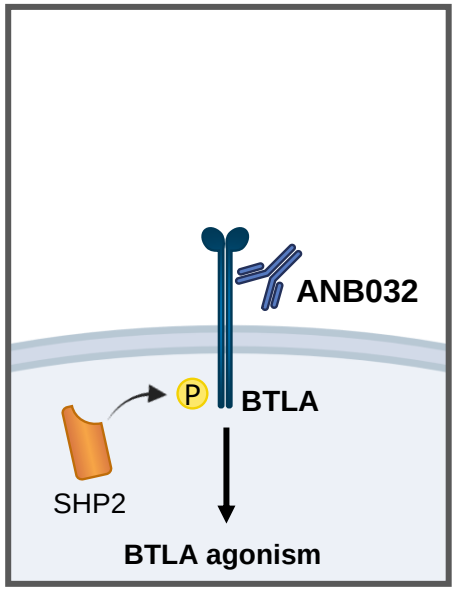
ANB032 modulates immune cells:

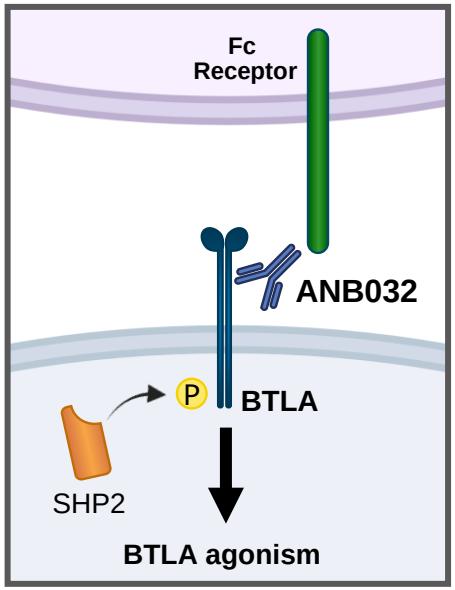
inhibit activated T cell proliferation, reduce inflammatory cytokine secretion and modulate DC function including inducing Tregs

1. Therapeutic area classes include dermatology, rheumatology, gastroenterology, metabolic, neurology and respiratory.

ANB032 is best-in-class with optimized Fc receptor engagement significantly enhances BTLA agonism



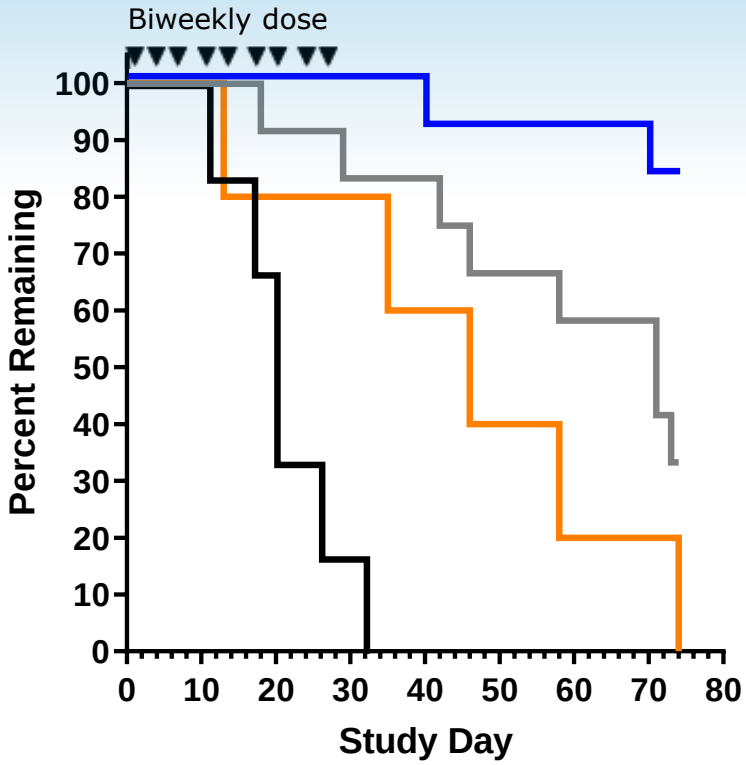




Jurkat BTLA SHP2 Recruitment Assay methodology: BTLA and SHP2 are fused with complementary enzyme fragments, when SHP2 is recruited to activated phosphorylated BTLA, the enzyme donor and enzyme acceptor form active β-gal that is detected by chemiluminescence.

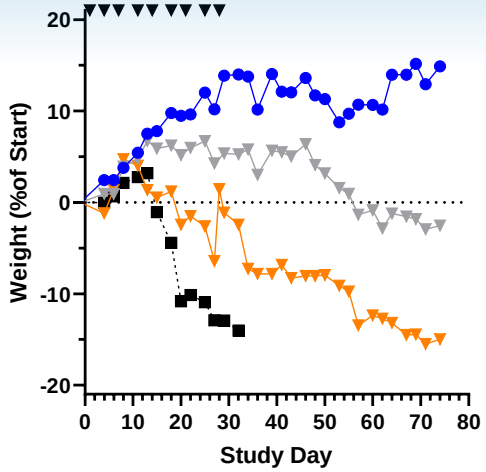
ANB032 demonstrates best-in-class in-vivo efficacy in humanized murine model of GvHD

Survival

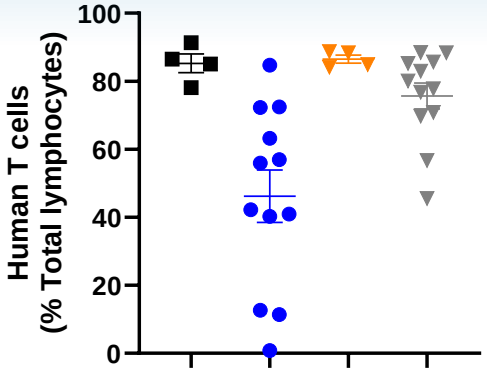


- Isotype
- ANB032
- Lilly BTLA agonist (Clinical candidate, LY-3361237)
- Lilly BTLA agonist (Alternative clone 23C8)

Body weight



Human T cell expansion (Day 18)



Note: All treatment groups were run together in the same study. Isotype, ANB032 and Lilly clone 23C8 were dosed at 3 mg/kg biweekly. LY-3361237 was dosed at 10 mg/kg biweekly.

ANB032 demonstrated favorable safety and tolerability with rapid and sustained PK/PD activity

96 healthy volunteers in SAD and MAD cohorts in Phase 1 study

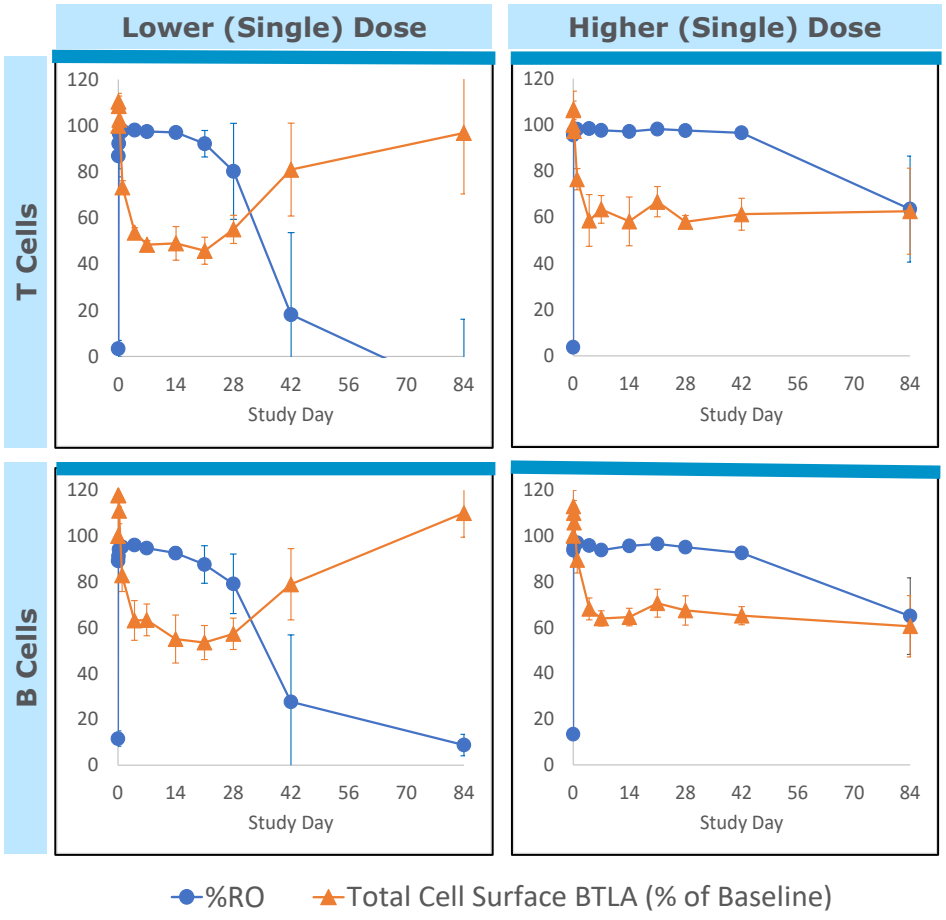
- Favorable ~2-week half-life with IV and SQ dosing
- Full receptor occupancy (RO) within hours and maintained for >30 days

Rapid and sustained target engagement on both T and B cells

- Duration of reduced BTLA expression persisted in dose-dependent manner

Well-tolerated with no dose limiting tox

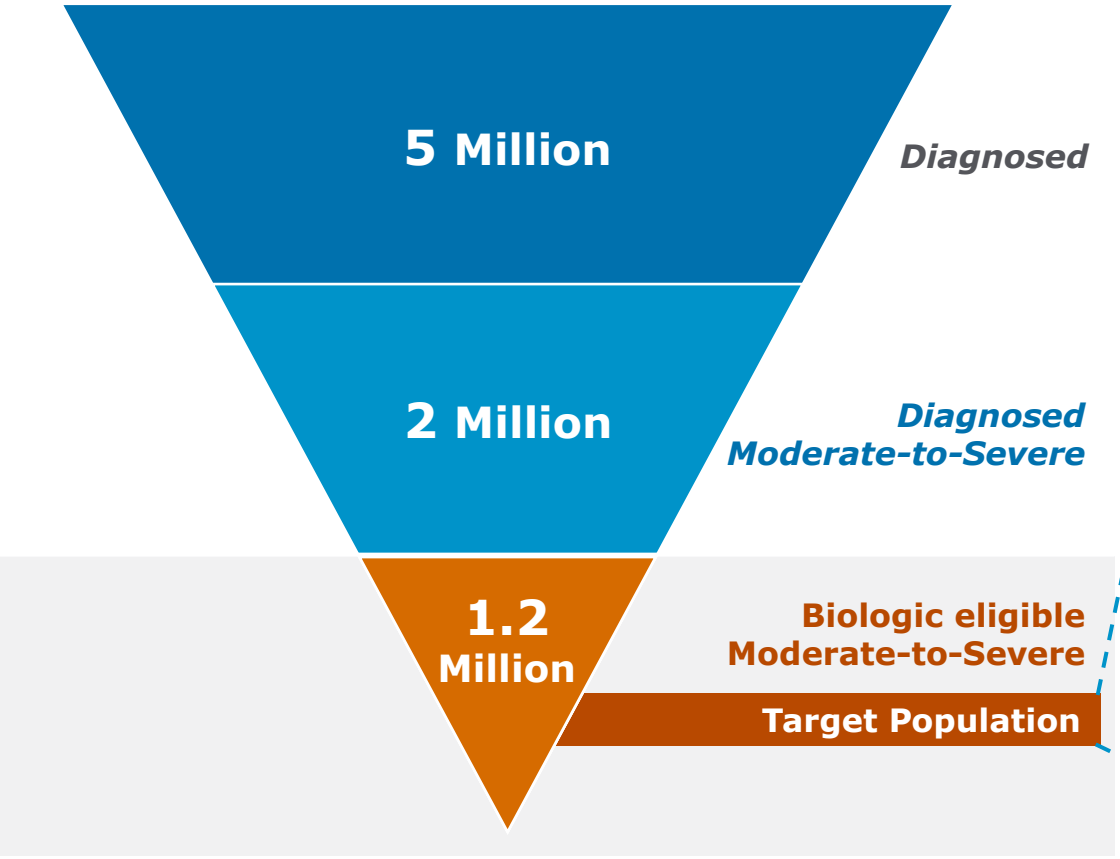
- No SAEs
- Most AEs mild-to-moderate, short duration, dose independent and resolved without sequelae
- No evidence of infection risk or cancer risk to date



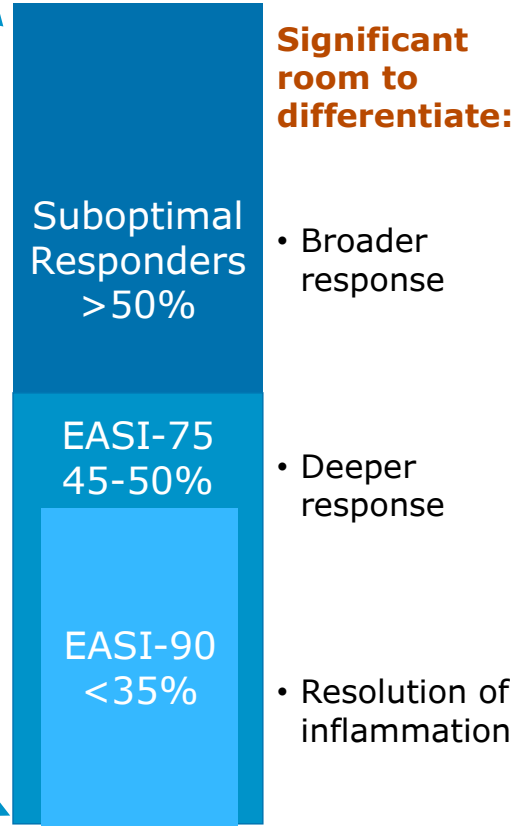
Opportunity for new biologic class with differentiated outcomes in AD regardless of prior treatment

AD $16 billion global sales by 2030[1]

US prevalence[2]



e.g., dupilumab response profile

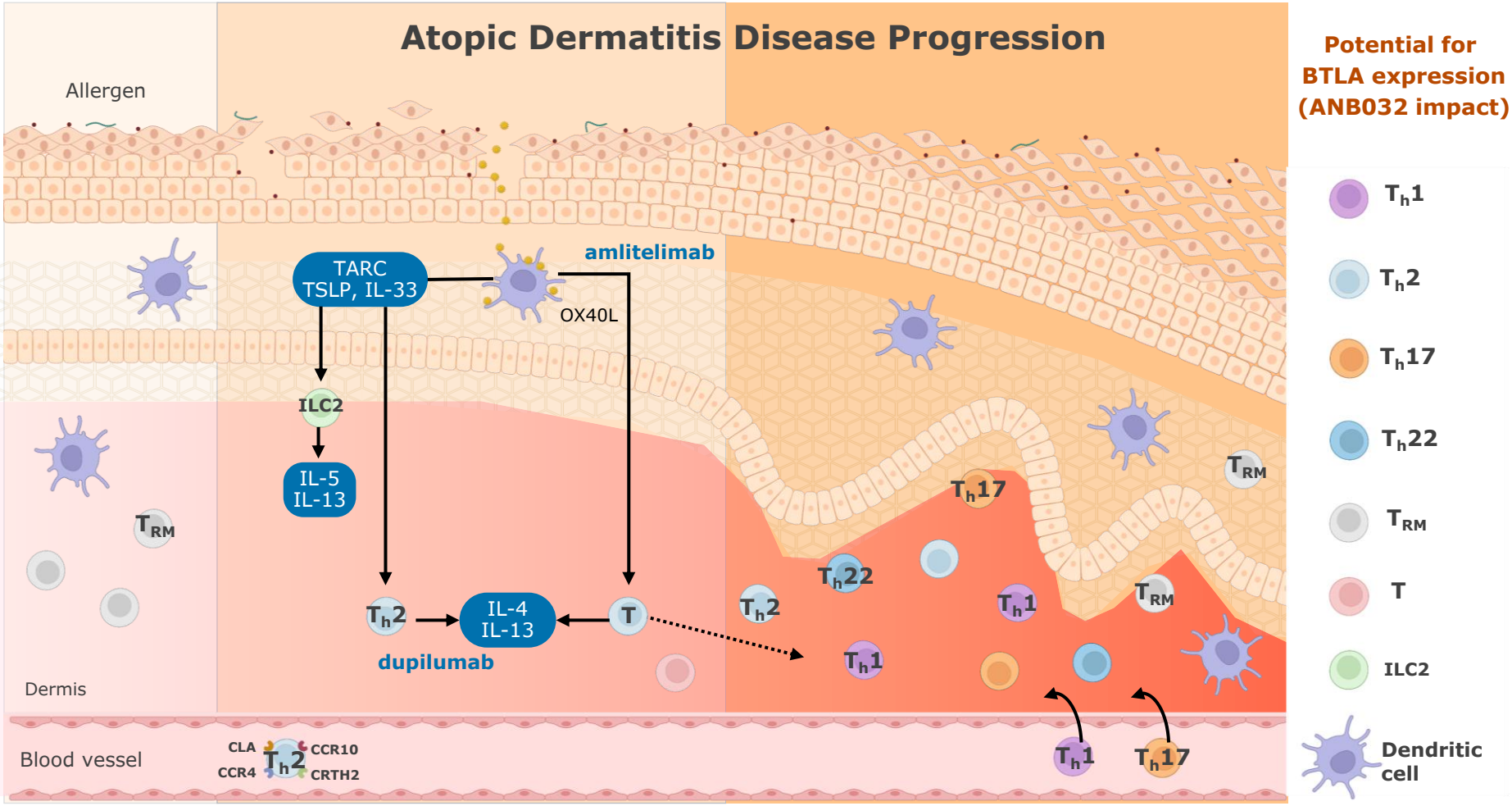


Significant room to differentiate:

- Broader response
- Deeper response
- Resolution of inflammation

1. GlobalData, AD Global Drug Forecast and Analysis, 2030; 2. Claims analysis to determine market size based on 5 years of claims history; dupilumab responsive profile per prescribing information (label (fda.gov).

Th1, Th2, Th17, Th22 and dendritic cells in tissue and periphery drive atopic dermatitis pathogenesis

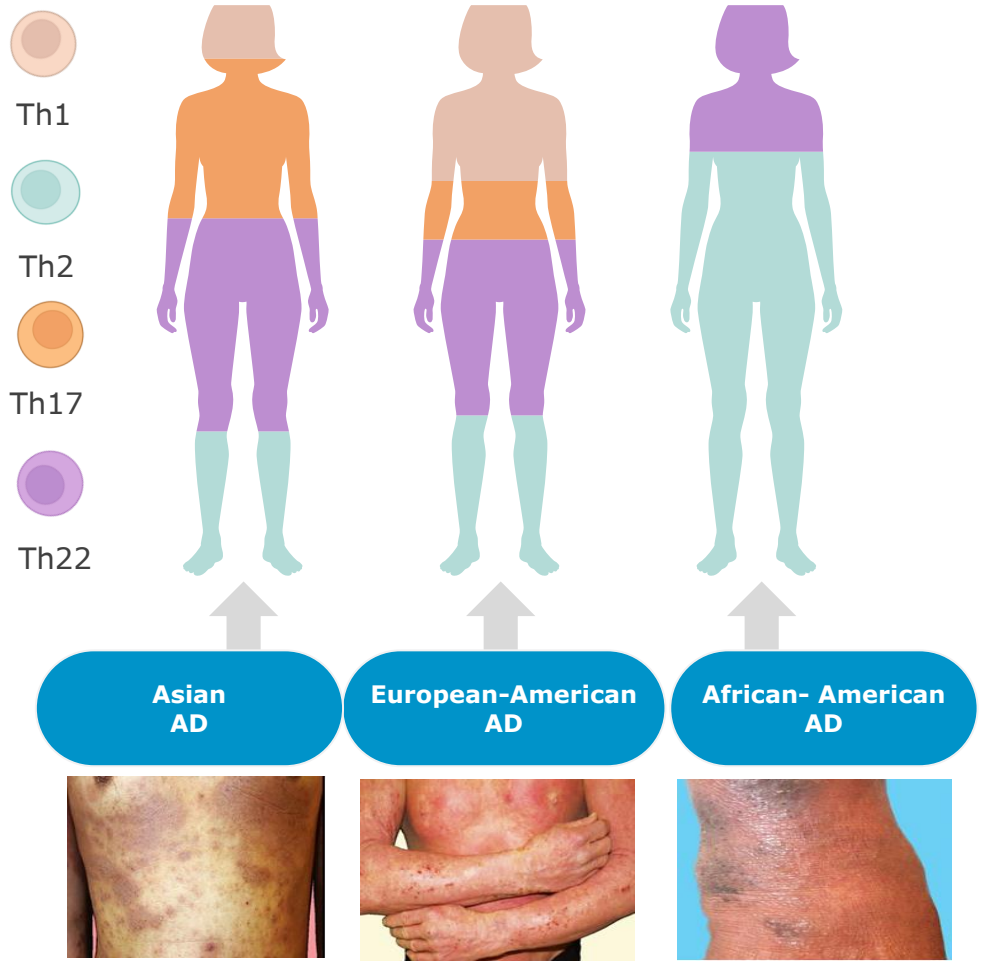


Adapted from Nature Reviews Disease Primers volume 4, Article number: 1 (2018).

Immune pathway skewing in atopic dermatitis patient populations highlights the need for new therapies

SOC only directly targets Th2 pathway

Immune Pathway Skewing in AD Patient Populations



AD is highly heterogeneous involving multiple immune cytokines

- Immune activation can vary by ethnicity resulting in a highly heterogeneous presentation
- Substantial unmet need across all patient populations

Expect ANB032 to drive deeper responses across broader patient population

- Restore immune balance

Adapted from Renert-Yuval Y, et al. Ann Allergy Asthma Immunol 2020;124:28–35; Czarnowicki T, et al. J Allergy Clin Immunol 2019;143:1–11.

Anaptys is leading where the AD landscape is moving: Treating AD beyond only Th2 inflammation

Initial AD clinical efficacy

"Th2 only" pathway approaches

Current AD disease understanding

Broader immune cell modulator approaches

AnaptysBio
(ANB032, BTLA agonist)
Broadly reduces T cell (Th1, Th2, Th17, Th22) cytokine secretion and modulates dendritic cells inducing Tregs
Only company with a BTLA agonist program in AD

SANOFI
(Dupixent, IL-4/13)

SANOFI
(Amlitelimab, OX-40L)

Lilly
(Lebrikizumab, IL-13)

Lilly
(Ucenprubart, CD200R agonist)

LEO
(IL-13 & IL-22)

ASLAN PHARMACEUTICALS
(IL-13R)

APOGEE THERAPEUTICS
(IL-13)

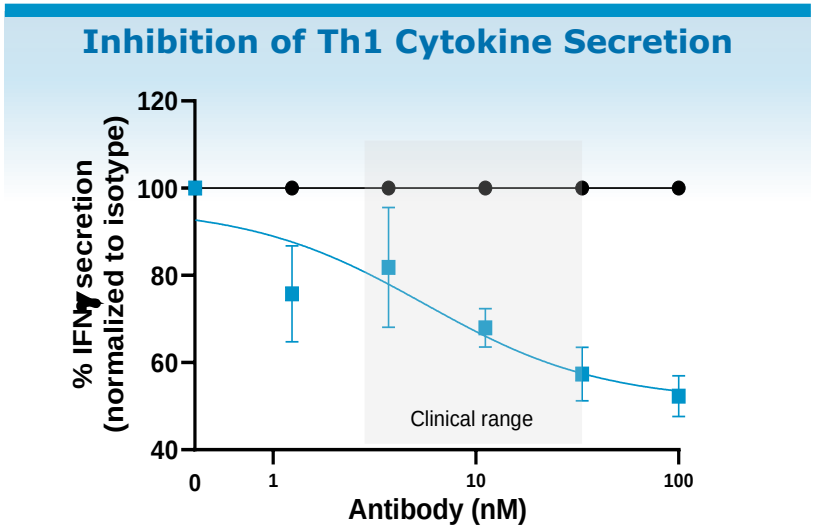
Johnson&Johnson
(JNJ-67484703, PD-1 agonist)

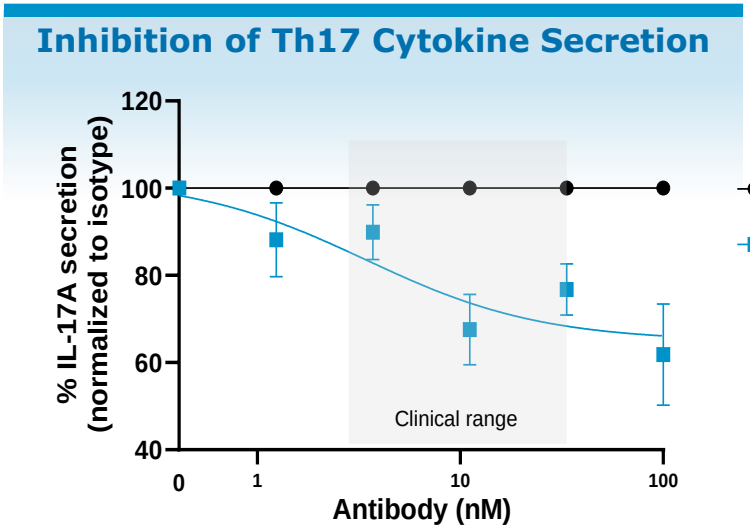
GALDERMA
(IL-31)

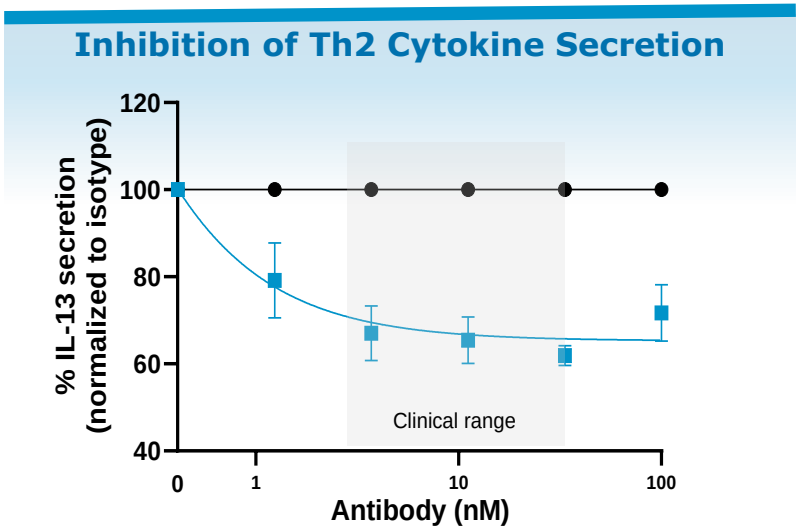
RAPT THERAPEUTICS
(CCR4)

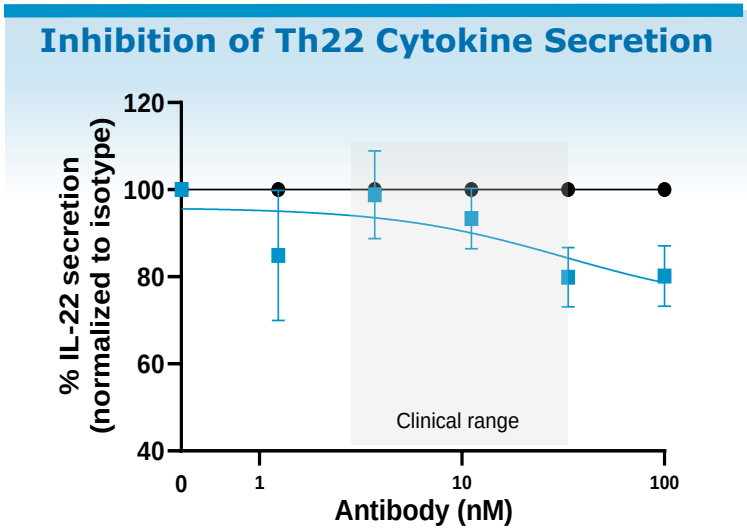
AMGEN
(Rocatinlimab, OX-40)

ANB032 inhibits Th1, Th2, Th17 and Th22 cytokine secretion more broadly than anti-OX40L in AD patient-derived PBMCs



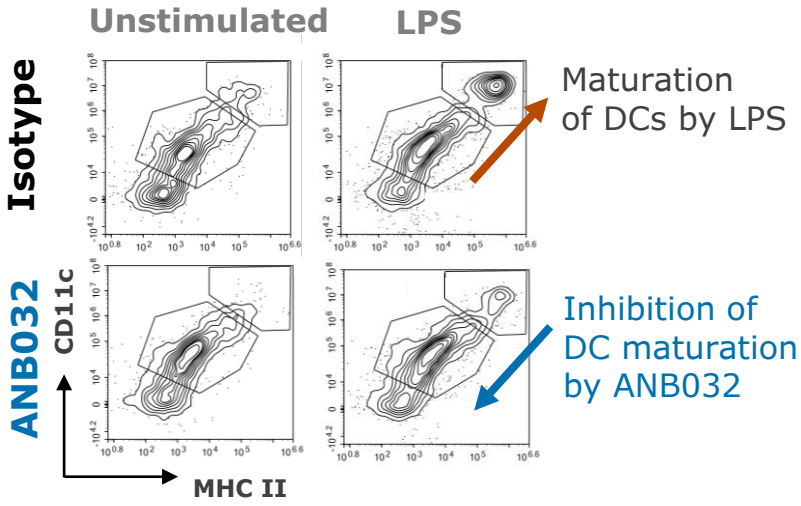




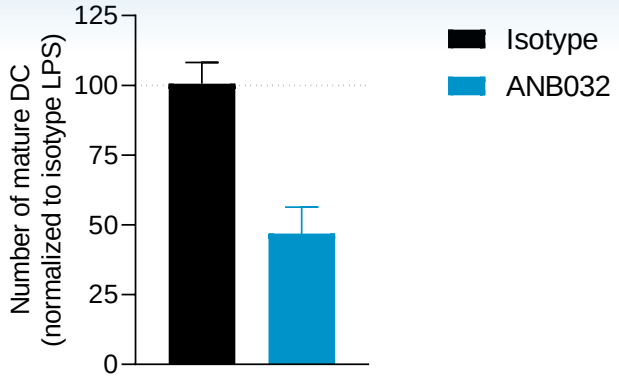


Sanofi's OX40L P2b AD data demonstrates impact on disease pathology beyond Th2; While ANB032 more broadly inhibits T cell cytokine secretion, it additionally modulates dendritic cells inducing Tregs

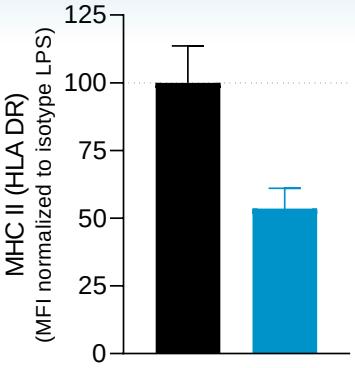
ANB032 inhibits DC maturation and reduces antigen presentation and co-stimulatory molecules



ANB032 inhibits DC maturation

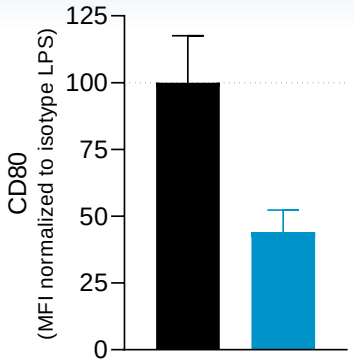


Antigen presentation
(Gated on total CD11c+ DCs)
MHC II

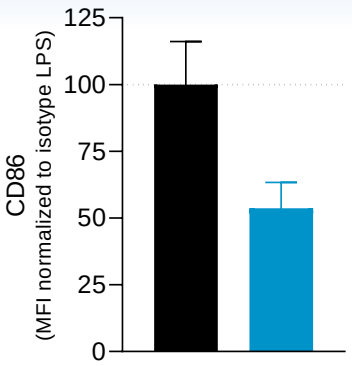


Co-stimulatory molecules on DCs
(Gated on total CD11c+ DCs)

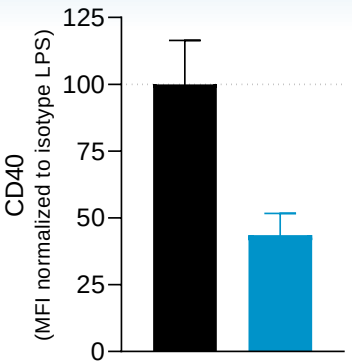
CD80



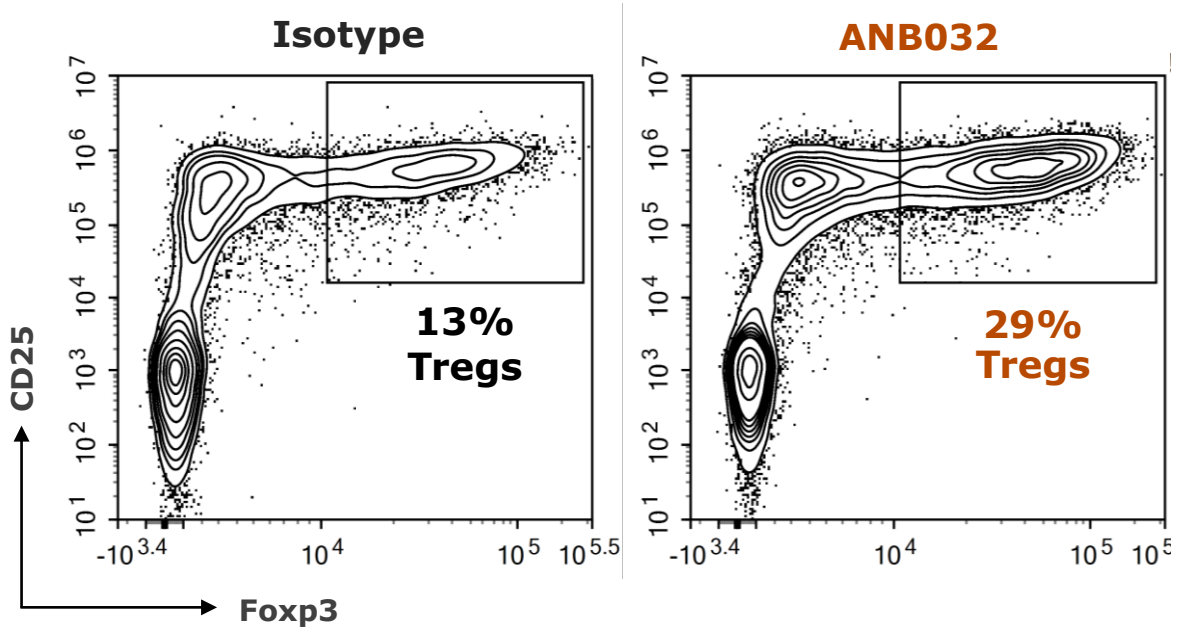
CD86



CD40

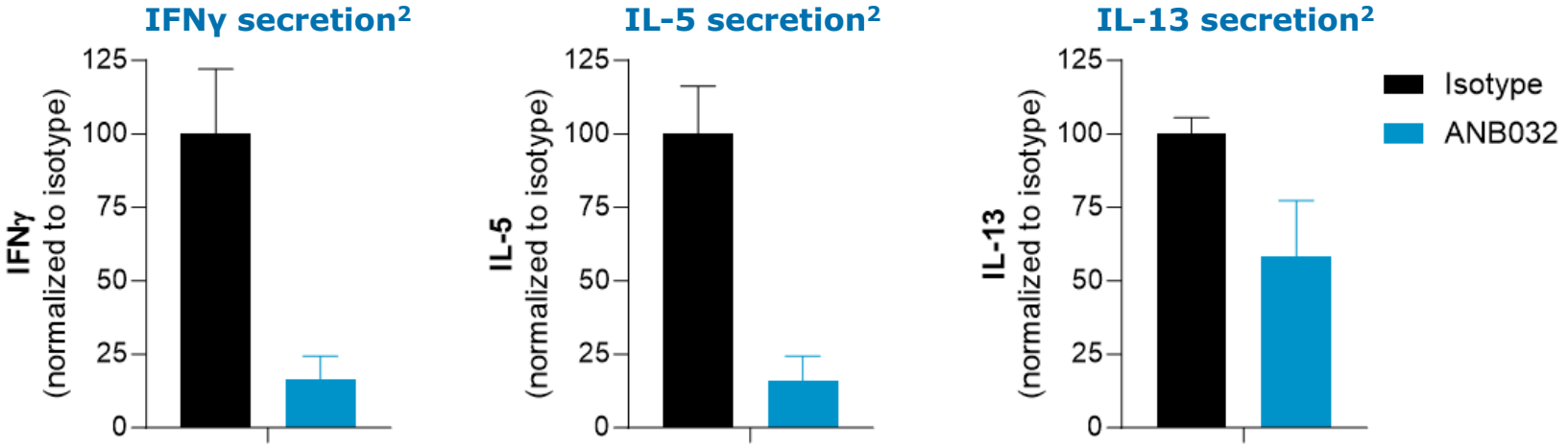


ANB032-treated DCs induce functional Tregs offering potential to restore immune balance



Significantly more DCs in AD patients' skin:

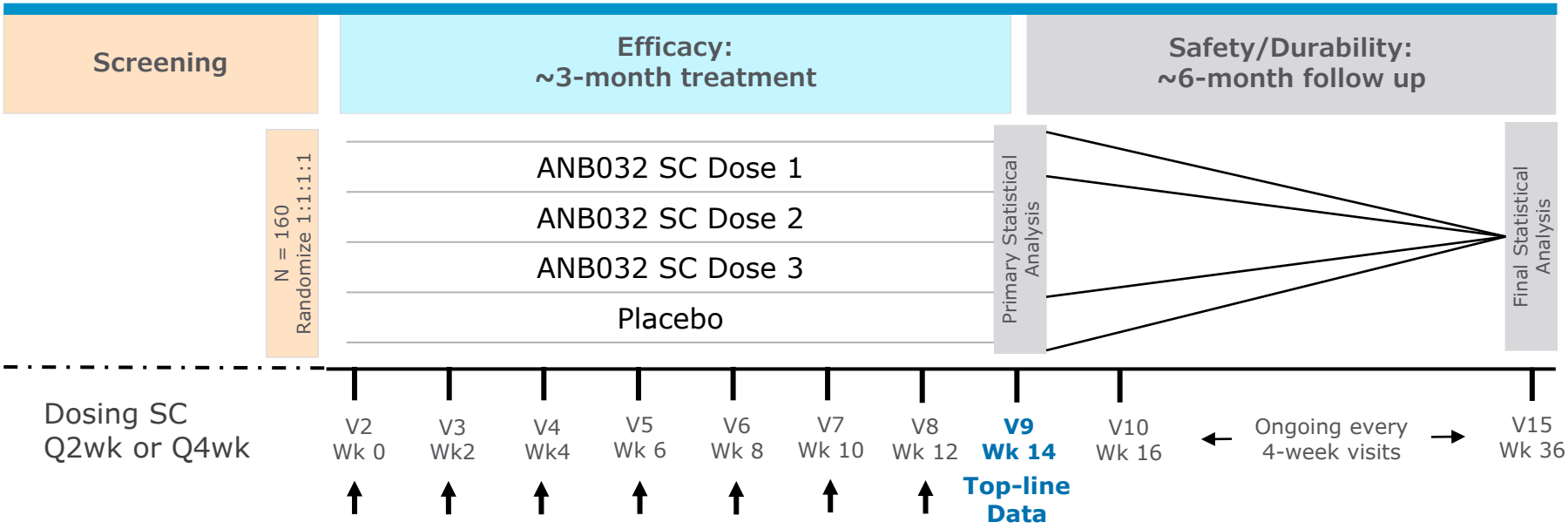
- **~10-fold increase in epidermis**
- **Up to 3.5-fold increase in dermis[3]**



1. DCs were treated with either ANB032 or isotype and then co-cultured with allogenic naïve CD4 T cells to allow T cell differentiation. T cells were stained for CD4, CD25 and intracellular Foxp3 to identify inducible Tregs 2. Effect of ANB032-treated DCs on functional Tregs and inflammatory cytokine secretion in an MLR assay 3. Guttman-Yassky, et al. *J Allergy Clin Immnol* 2007;119:1210-7.

ANB032 Phase 2b in IL-13 mAb naïve and experienced AD patients

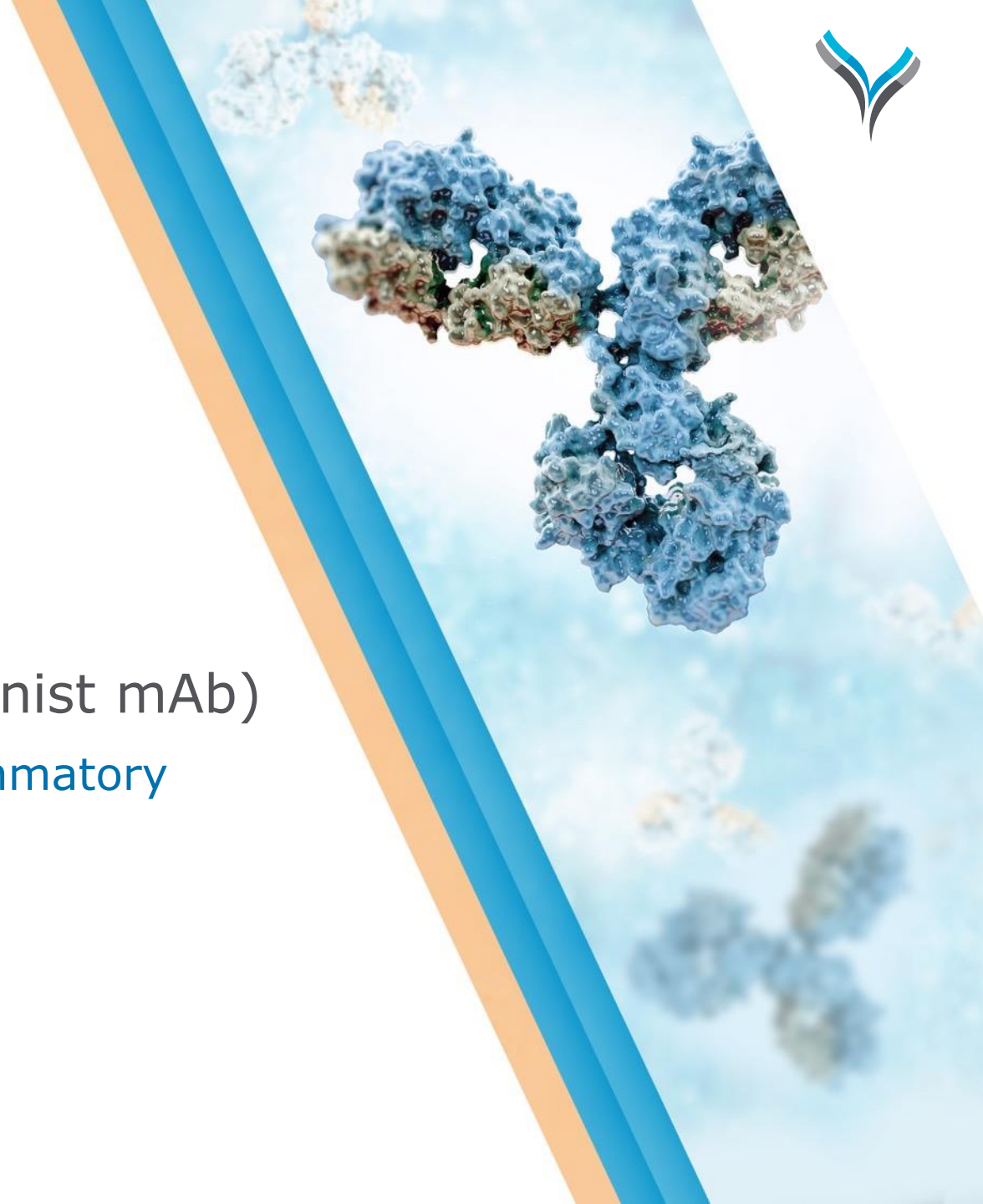
Initiated Q2 2023; Top-line data YE 2024



Patient population		• Adults with moderate-to-severe atopic dermatitis[1] • Include both dupilumab/IL-13 naïve and experienced patients
Endpoints	Primary	• Mean change from Baseline in EASI at Week 14
	Secondary	• EASI-75 • vIGA-AD 0 (clear) or 1 (almost clear) and a ≥ 2-point reduction (improvement) • PNRS (itch), DLQI, SCORAD • Safety
Exploratory endpoints		• Th1/Th2/Th17 cytokines and other soluble biomarkers, tissue samples (tape strips, biopsies)

ClinicalTrials.gov: NCT05935085

1. Moderate-to-severe atopic dermatitis: at least 10% of their total body surface area (BSA), an Eczema Area and Severity Index (EASI) score ≥ 16, and a validated Investigator Global Assessment for Atopic Dermatitis (vIGA) score ≥ 3.



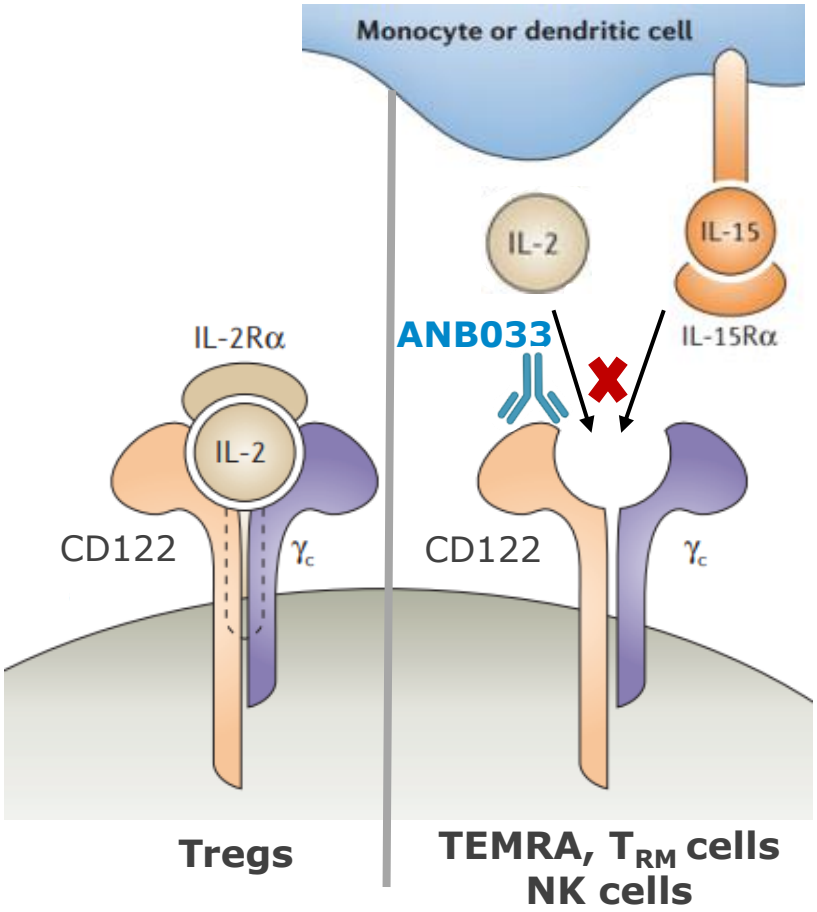
ANB033
(anti-CD122 antagonist mAb)

Autoimmune and Inflammatory Diseases

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

IND filing targeted Q2 2024

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

Reduces pathogenic T cells by preferentially inhibiting the lower affinity dimeric IL-2 receptor complex

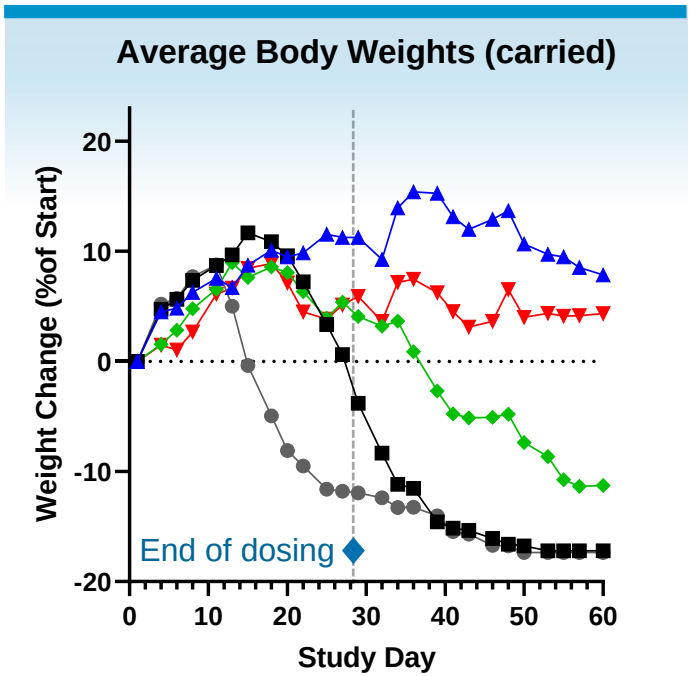
- Spares Tregs which express higher affinity trimeric IL-2 receptor complex

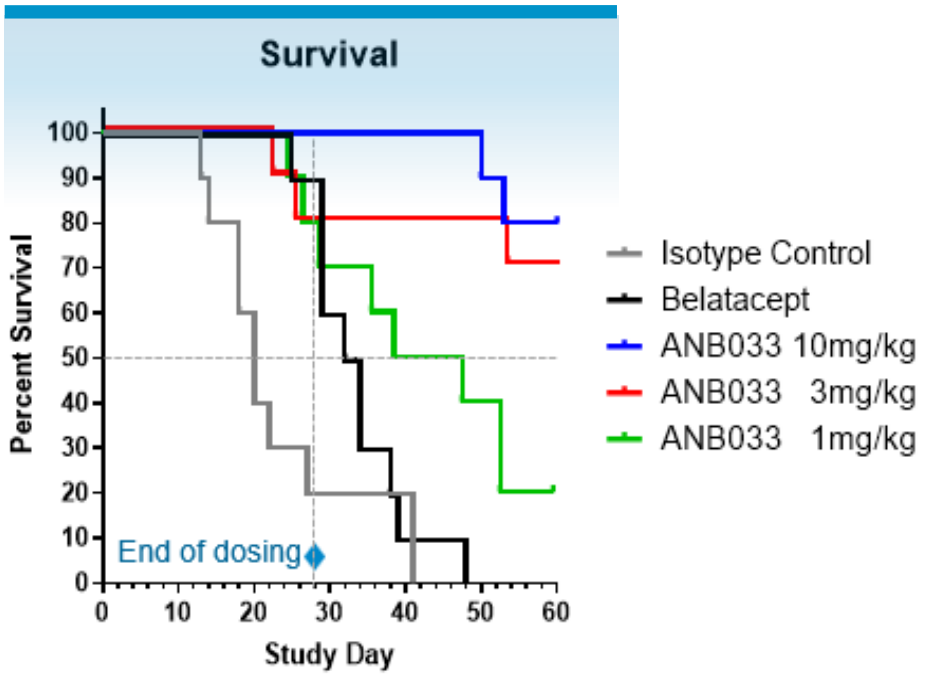
Targeted reduction of CD122 expressing T_{RM} cells, which require IL-15 for survival, may potentially drive durable response

ANB033: Durable survival in GVHD model

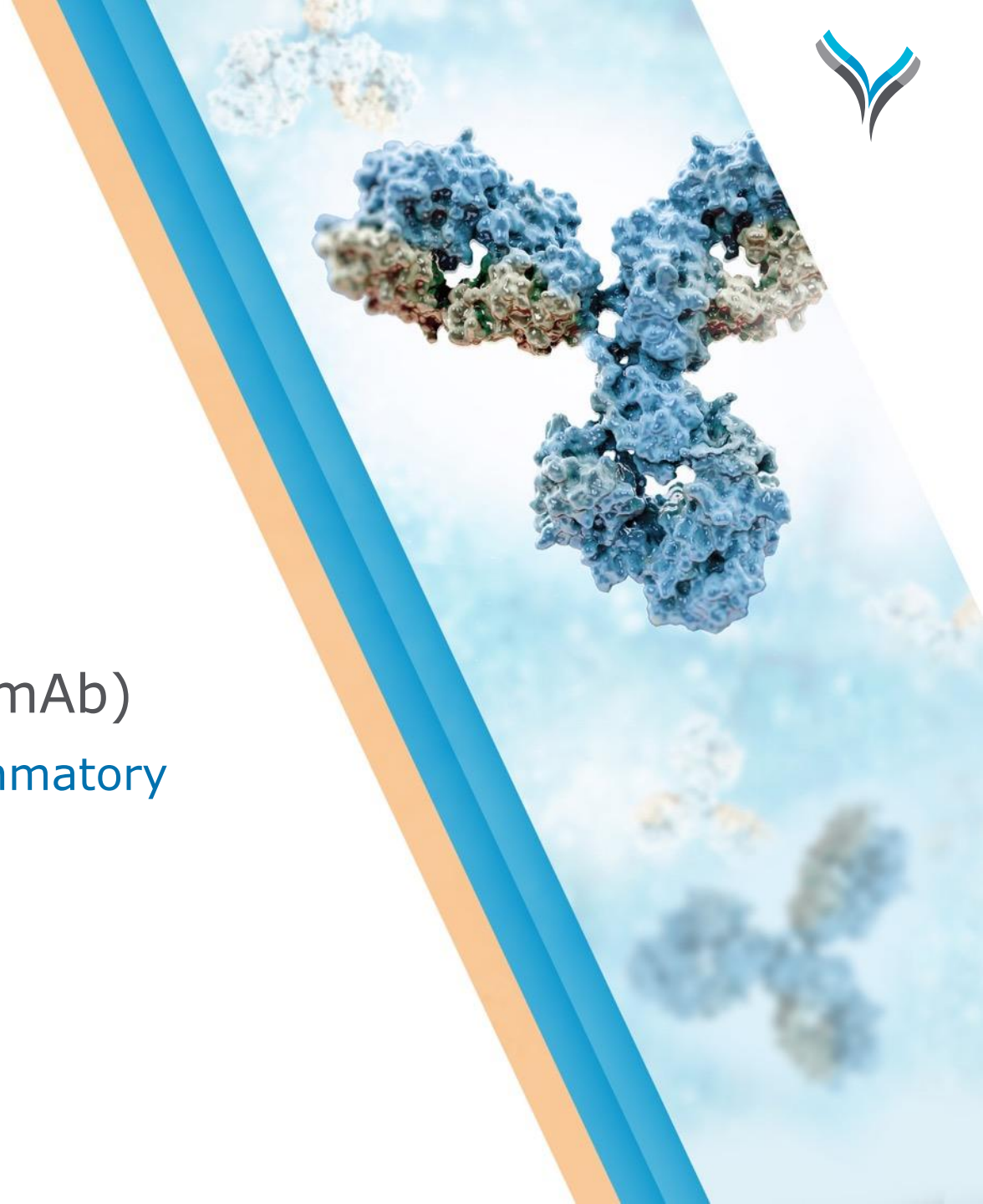
All mice treated at high-dose survived well beyond end of dosing

- 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



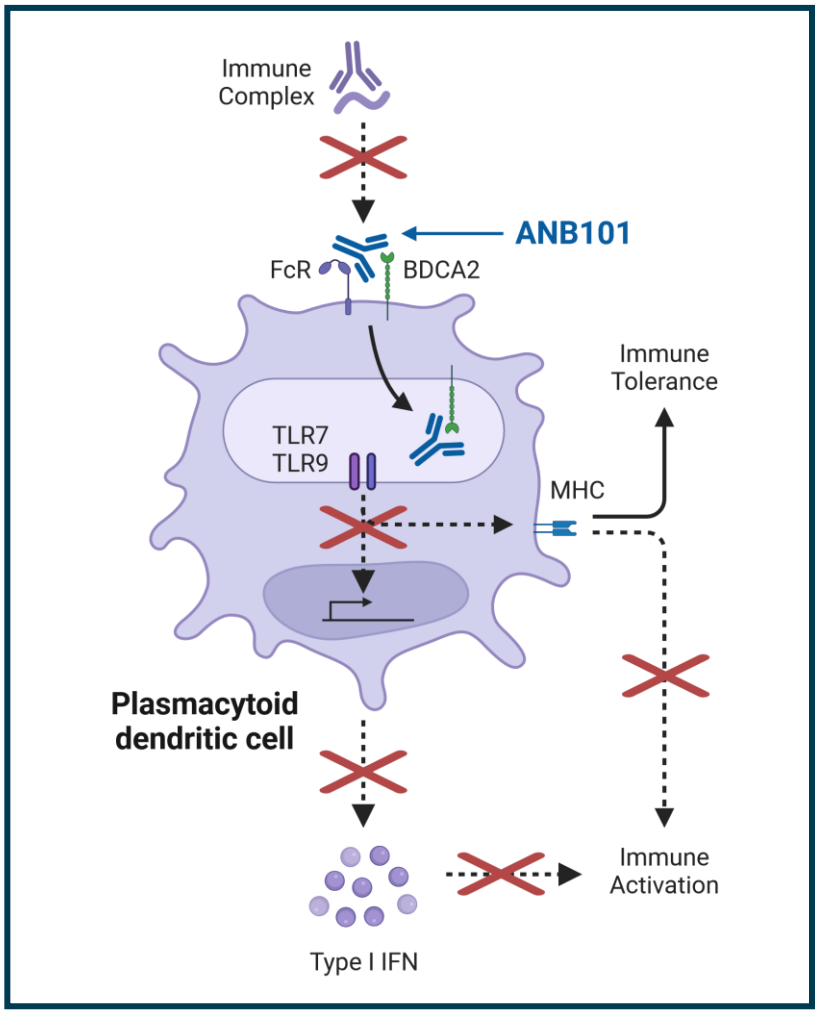
ANB101
(BDCA2 modulator mAb)

Autoimmune and Inflammatory Diseases

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

IND filing targeted H2 2024

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

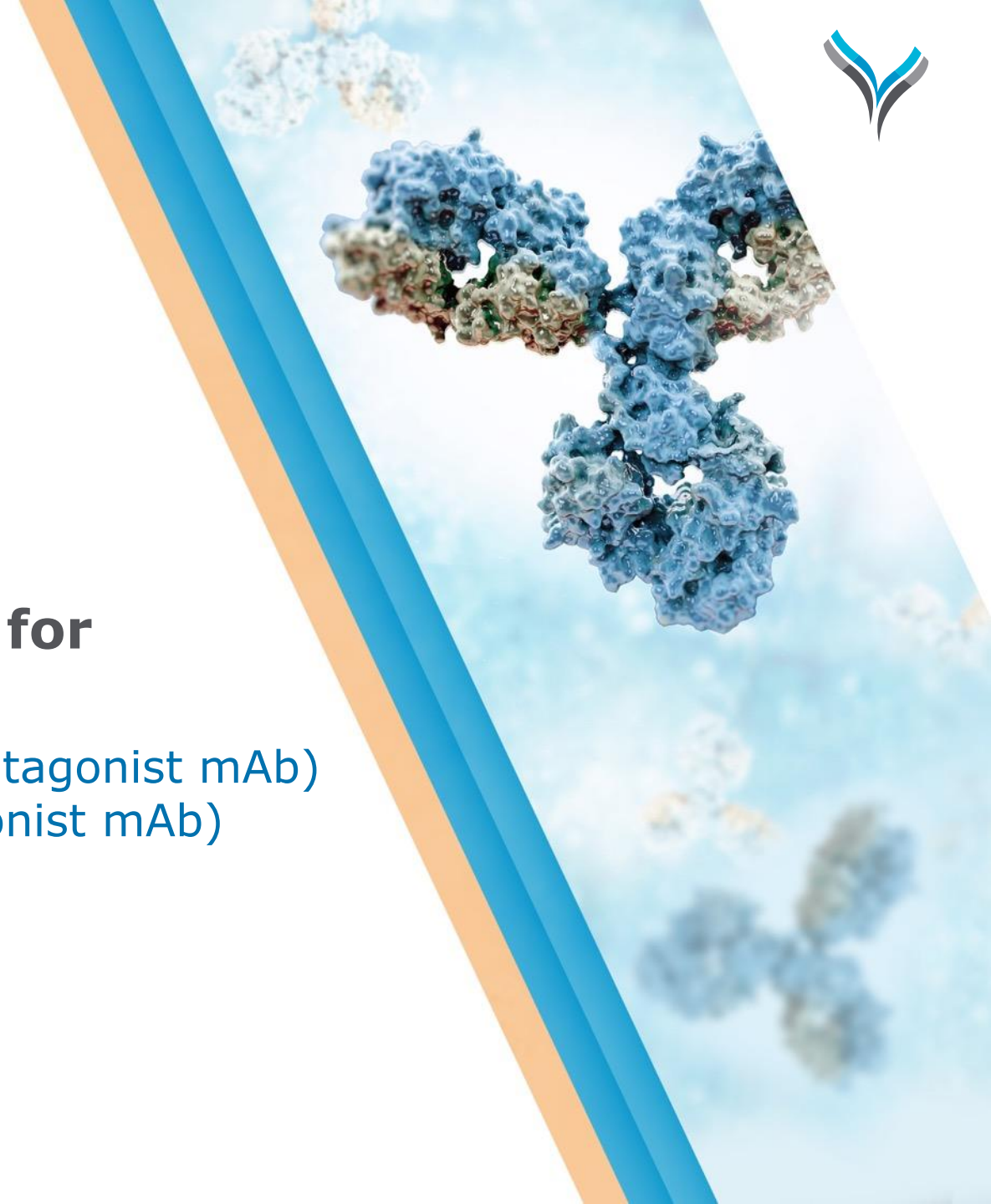
pDCs enriched in tissue in rheumatology and other inflammatory diseases

- 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.



Legacy Programs for Out-license

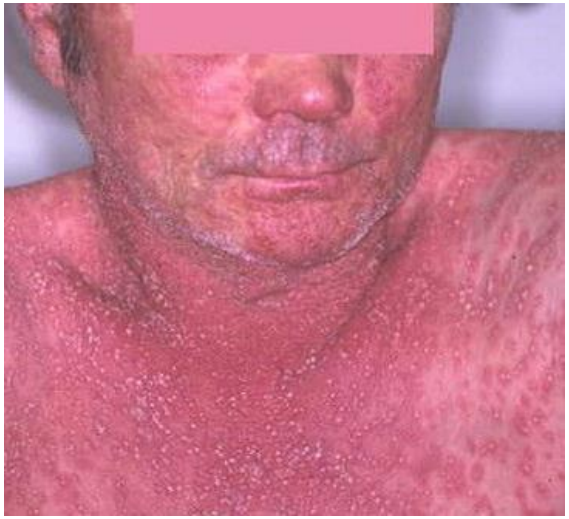
Imsidolimab (IL-36R antagonist mAb)
Etokimab (IL-33 antagonist mAb)

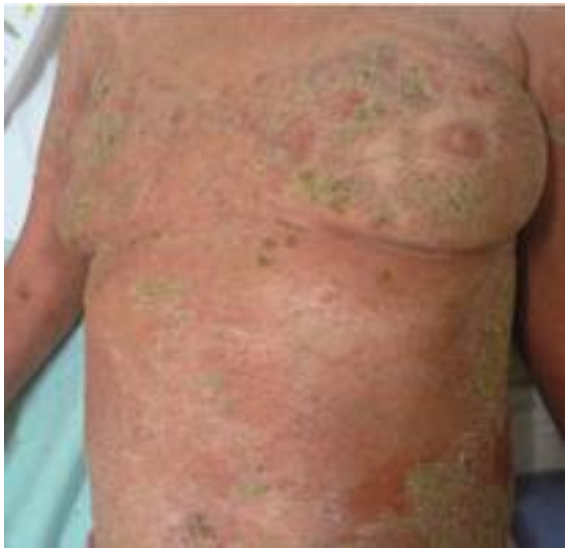
Generalized Pustular Psoriasis (GPP)

Systemic inflammatory disease where IL-36 pathway plays key role in pathology

GPP is a systemic, life-threatening inflammatory disease characterized by widespread pustules

- Associated with unregulated IL-36 signaling
- Patients have a high fever and elevated levels of serum CRP and inflammatory cytokines (e.g., IL-8)
- Severe GPP patients can die from cardio-pulmonary failure, exhaustion, toxicity and infection





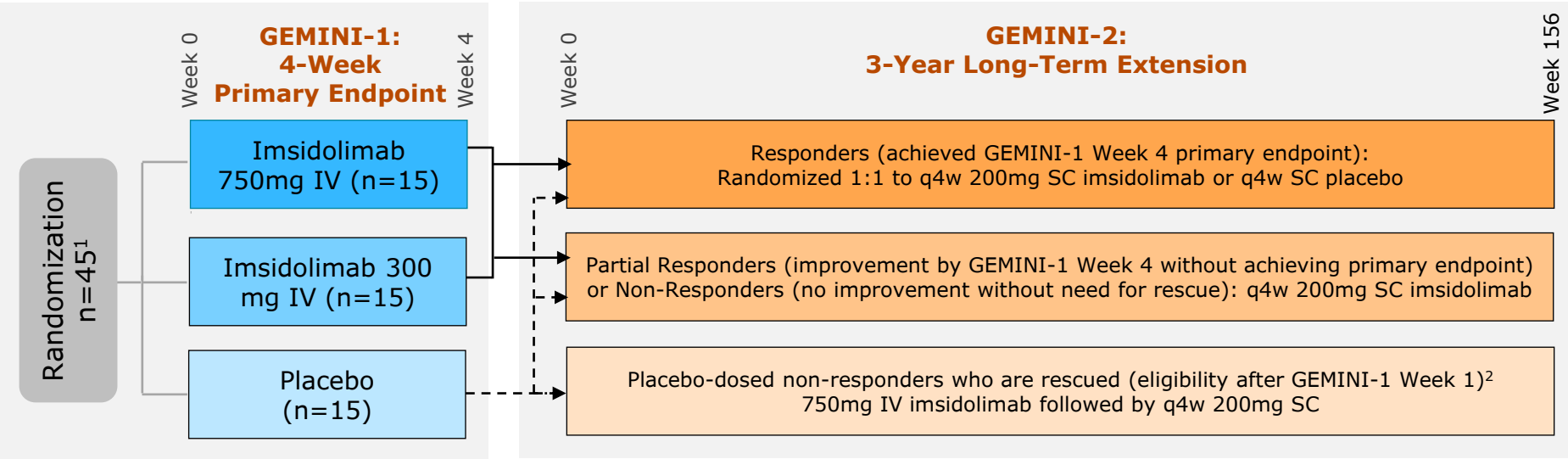
GPP ICD-10 diagnostic code analysis by IQVIA assessed US prevalence during 2017-2019 timeframe

- ~37,000 unique patients diagnosed at least once
- ~15,000 unique patients diagnosed two or more times

FDA has granted ODD for treatment of GPP

GEMINI-1 & 2: Imsidolimab GPP Phase 3 trials

Positive GEMINI-1 top-line data announced October 2023



Patient Population	• Male and female subjects 18 to 80 years of age • Clinically confirmed diagnosis of GPP as per ERASPEN definition • Baseline Generalized Pustular Psoriasis Physician Global Assessment (GPPPGA) score of at least moderate severity (3 and higher) • Active flare with pustules and erythema accounting for at least 5% of body surface area at baseline
Key Endpoints	• Primary: GPPPGA score of clear (0) or almost clear (1) at GEMINI-1 Week 4 • Key Secondary: Pustulation Rating Scale (PRS) of 0 or 1 at GEMINI-1 Week 1 • Other: Time to flare recurrence, proportion of subjects in remission, DLQI, safety

ClinicalTrials.gov: NCT05352893, NCT05366855

1. 80% power calculated for GEMINI-1 using two-sized test alpha of 0.05 assuming ~40% effect size with 45 patient sample size
2. Starting at week 1 in GEMINI-1, placebo patients who have not improved or are worsening are eligible to be rescued with imsidolimab.

Imsidolimab Positive GEMINI-1 Top-Line Phase 3 Data

Intend to out-license imsidolimab in 2024

- GEMINI-1 trial: registration-enabling trial enrolled 45 patients
- First randomized, double-blind, placebo-controlled trial to use the composite endpoint of Generalized Pustular Psoriasis Physician Global Assessment (GPPPGA) at Week 4 as primary assessment
- 53.3% of patients who received a single dose of 750mg IV imsidolimab achieved GPPPGA 0/1 (clear or almost clear) at Week 4 (primary endpoint), compared to 13.3% of patients on placebo (p=0.0131)
- Demonstrated favorable safety and tolerability with no SAEs, low incidence and no increase of infections vs. placebo and no cases of DRESS or Guillain-Barre in imsidolimab-treated patients
- Only one of 30 (3.3%) imsidolimab-treated patients had detectable ADA, which were non-neutralizing

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

IL-33 biology applicable to epithelial driven diseases

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

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

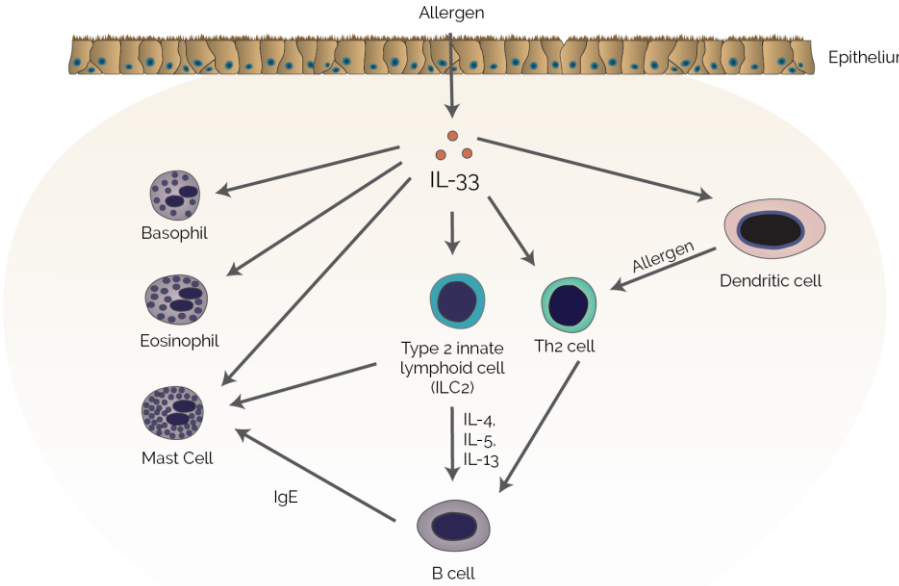
IL-33 is genetically associated with asthma

- 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/SA)

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, with AZ POC data in diabetic nephropathy expected this year)

GSK Immuno-Oncology Financial Collaboration

Jemperli™ (dostarlimab, anti-PD-1 Antagonist)
Cobolimab (GSK4069889, anti-TIM-3 Antagonist)

Significant potential royalties from GSK immuno-oncology financial collaboration

	Jemperli (dostarlimab-gxly) Injection 500 mg *(Anti-PD-1 antagonist)*	**Cobolimab** *(Anti-TIM-3 antagonist)*	Sagard
Royalties	**8% royalties on annual net sales <$1B**** **12-25% royalties on annual net sales ≥ $1B**	*Royalty rates shown reflect economics for potential combination use with dostarlimab as in COSTAR* **4-8% royalties on annual net sales (cobolimab portion)** **8-25% royalties on annual net sales (*Jemperli* portion)**	**$250mm *Jemperli* Capped Non-Recourse Monetization** 2021 transaction in exchange for **selected** (in orange text) *Jemperli* receivables until** Sagard paid back one of the following capped returns: • $312.5MM (125%) by end 2026 -or- • $337.5MM (135%) by end 2027 -or- • $412.5MM (165%) if after 2027
Remaining Milestones	**$15mm regulatory** **$90MM commercial on annual net sales <$1B**** **$75mm commercial on annual net sales ≥ $1B**	**$5MM clinical development** **$90MM regulatory** **$165MM commercial**	

Note: Sale of *Zejula* (niraparib) royalty interest in September 2022 to wholly-owned subsidiary of 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.

GSK immuno-oncology financial collaboration

Jemperli (dostarlimab-gxly) Injection 500 mg

(anti-PD-1 antagonist)

Women's cancers

- **1L endometrial cancer:** Approved in US and EU for dMMR/MSI-H primary advanced or recurrent endometrial cancer and dMMR/MSI-H recurrent or advanced endometrial cancer after progressing on a platinum-containing regimen
  - P3 RUBY Part 2 trial (dostarlimab + niraparib) demonstrated significant improvement in PFS in MMRp/MSS patients and may expand use
  - 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
  - P3 data (interim analysis) H1 2024
  - Significant U.S. market opportunity with ~20,000 eligible diagnoses/year[1]

Colorectal cancer

- **Rectal cancer:** P2 AZUR-1 trial in dMMR/MSI-H
- **Colon cancer:** P3 AZUR-2 trial in perioperative dMMR/MSI-H colon cancer

Lung cancer

- **1L NSCLC:** P2 PERLA trial[2]: 46% cORR for dostarlimab + chemo vs 37% cORR for pembrolizumab + chemotherapy

Additional dostarlimab royalty opportunities

- P2: 1L NSCLC and 1L H&NSCC, in combination with anti-TIGIT (belrestotug)
- P1/2 combinations with anti-CD96 and PVRIG across multiple solid tumors

---

Cobolimab

(anti-TIM-3 antagonist)

Lung cancer

- **2L NSCLC:** P3 COSTAR trial (docetaxel *vs* dostarlimab + docetaxel *vs* docetaxel + dostarlimab + cobolimab)
  - Top-line data expected in H2 2024
  - Significant U.S. market opportunity with 237,000 new NSCLC diagnoses/year[1]

1. NCI SEER data
2. Phase 2 GSK-sponsored PERLA study in 1L NSCLC. Peters S, et al. Annals of Oncology (2023) 34 (suppl_2): S1254-S1335. 10.1016/annonc/annonc1358

NB: Treatment-emergent adverse events (TEAEs) for dostarlimab in the PERLA phase II trial were consistent with previous trials of similar regimens.