UNITED STATES
SECURITIES AND EXCHANGE COMMISSION
WASHINGTON, D.C. 20549

FORM 8-K

CURRENT REPORT PURSUANT TO
SECTION 13 OR 15(d) OF THE SECURITIES EXCHANGE ACT OF 1934

Date of Report: February 19, 2025
(Date of earliest event reported)

ANAPTYSBIO, INC.
(Exact Name of Registrant as Specified in Charter)

Delaware	**001-37985**	**20-3828755**
(State or Other Jurisdiction of Incorporation)	(Commission File Number)	(IRS Employer Identification No.)

10770 Wateridge Circle, Suite 210,
San Diego, CA 92121
(Address of Principal Executive Offices, and Zip Code)

(858) 362-6295
(Registrant's Telephone Number, Including Area Code)

Not Applicable
(Former name or former address, if changed since last report.)

Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions (see General Instruction A.2. below):

☐Written communication pursuant to Rule 425 under the Securities Act (17 CFR 230.425)
☐Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)
☐Pre-commencement communication pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))
☐Pre-commencement communication pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))

Securities registered pursuant to Section 12(b) of the Act:

Title of each class	Trading Symbol(s)	Name of each exchange on which registered
Common Stock, par value $0.001 per share	ANAB	The Nasdaq Stock Market LLC

Indicate by check mark whether the registrant is an emerging growth company as defined in Rule 405 of the Securities Act of 1933 (17 CFR §230.405 of this chapter) or Rule 12b-2 of the Securities Exchange Act of 1934 (17 CFR §240.12b-2 of this chapter).

Emerging growth company ☐

If an emerging growth company, indicate by check mark if the registrant has elected not to use the extended transition period for complying with any new or revised financial accounting standards provided pursuant to Section 13(a) of the Exchange Act. ☐

Item 7.01. Regulation FD.

On February 19, 2025, AnaptysBio, Inc. updated its corporate investor presentation, a full copy of which is attached hereto as Exhibit 99.1.

The information in this Item 7.01, including Exhibit 99.1, shall not be deemed "filed" for purposes of Section 18 of the Securities Exchange Act of 1934, as amended (the "Exchange Act"), or otherwise subject to the liabilities of that section, nor shall it be deemed incorporated by reference into any other filing under the Exchange Act or the Securities Act of 1933, as amended, except as expressly set forth by specific reference in such a filing.

Exhibit Number	Exhibit Title or Description
99.1	Anaptys Corporate Presentation February 2025.
104	Cover Page Interactive Data File (the cover page XBRL tags are embedded within the inline XBRL document).

SIGNATURES

Pursuant to the requirements of the Securities Exchange Act of 1934, the Registrant has duly caused this report to be signed on its behalf by the undersigned thereunto duly authorized.

AnaptysBio, Inc.

Date: February 19, 2025

By: /s/ Dennis Mulroy

Name: Dennis Mulroy

Title: Chief Financial Officer


AnaptysBio
Corporate
Overview
February 2025

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 at Week 28 and Phase 2 clinical trial in ulcerative colitis; whether current trends in rosnilimab's partial Week 28 data in the rheumatoid arthritis Phase 2b clinical trial will be maintained once complete Week 28 data becomes available; whether positive clinical trial results in rosnilimab's Phase 2b clinical trial in rheumatoid arthritis increases the likelihood of getting positive results from rosnilimab's 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 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 depleter and agonist)

P2b in Rheumatoid Arthritis

P2 in Ulcerative Colitis

ANB033
(CD122 antagonist)

P1 in Healthy Volunteers

ANB101
(BDCA2 modulator)

P1 (Q1 2025) in Healthy Volunteers

Autoimmune and inflammatory diseases including dermatology, gastroenterology and rheumatology

Cytokine Antagonists

Imsidolimab (IL-36R)

Out-licensed to VANDA PHARMACEUTICALS INC

Etokimab (IL-33)

P2b/3-ready in epithelial-driven diseases

(available for out-license)

Research and Capital

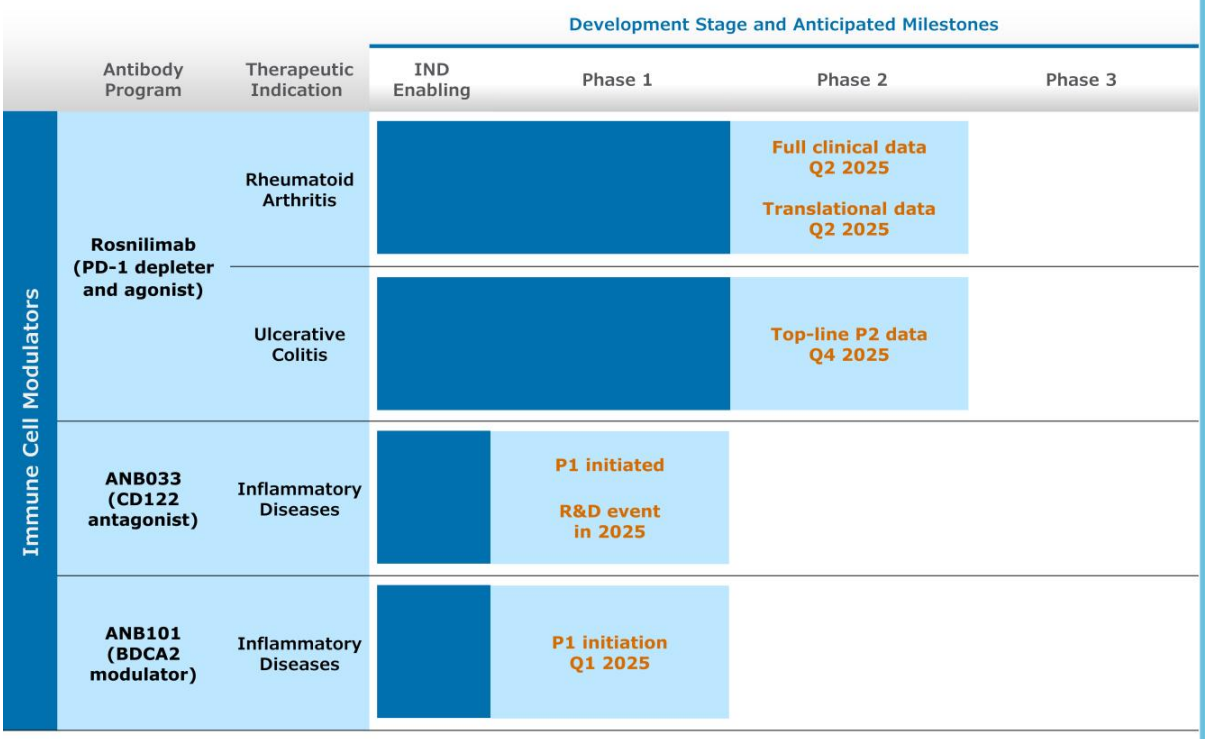
Research-driven

- 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

Leading pipeline of immune cell modulating antibodies with multiple 2025 catalysts



Rosnilimab

(PD-1 Depleter and Agonist)

RA and UC are large commercial markets with high unmet need

Rheumatoid arthritis:

- **~500,000 U.S. patients**
  >$10bn U.S. sales in "bio-experienced" market[1]
- **20-25% cycle** through <u>all</u> 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]

Potential first new differentiated class in RA in over a decade

RA trial increases confidence in UC trial outcome

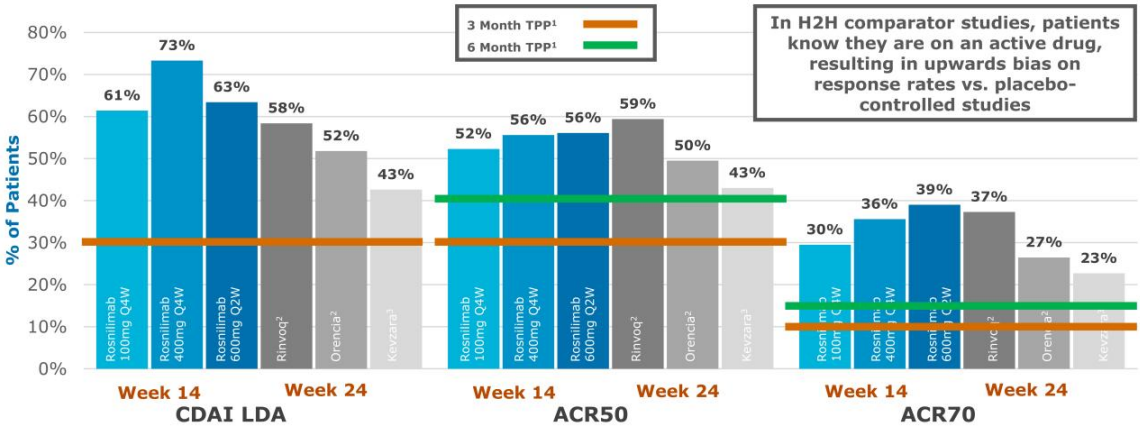
- Differentiated efficacy and safety data
- Translational data
  - Depletion of PD-1^{high} T_{ph} cells
  - Decrease of CRP demonstrates objective reduction of inflammation beyond directly targeting T cells

Rosnilimab has potential to treat wide range of systemic inflammatory diseases

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 Month 3 data highest ever reported, surpassing 6 Month TPP[1] and 6 Month competitor all-active H2H data

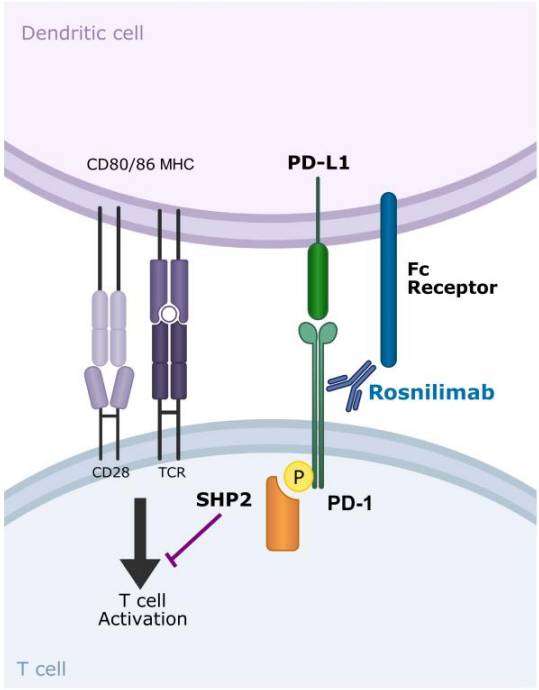
Bio-Experienced Patients: Absolute Rosnilimab Week 14 vs. Competitors H2H Comparator Week 24



- **By Month 3, treaters look for signal of response (i.e. ACR20) to treat through to Month 6**
  - 70%+ ACR20 rate at Month 3
- **However, treatment target is minimally LDA by Month 6[4]**
  - 69% (220/318) is highest ever rate of CDAI LDA reported already at Week 14
  - Some portion of the 31% LDA non-responders at Week 14 may not have hit peak response
- **Long-term sustained response is most important**
  - Sustained CDAI LDA and ACR50 and potentially deepening ACR70 responses at Week 28

1. Anaptys Jan. 2025 Target Product Profile (TPP) 2. SELECT-CHOICE Phase 3 study; NRI data 3. ContRAst-3 Phase 3 study; Missing data in ContRAst-3 handled with multiple imputation method 4. Only Week 14 CDAI LDA responders initially randomized to rosnilimab were eligible to stay on treatment from Week 14 to Week 28 (As of Dec. 10, 2024); CDAI = Clinical Diseases Activity Index; LDA = Low Disease Activity.

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

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.

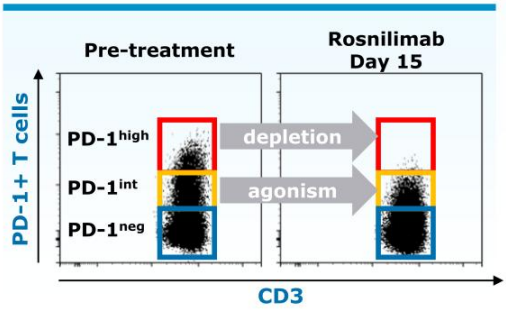
Rosnilimab is designed to bring the immune system back to homeostasis and modify disease

Rosnilimab preferentially targets activated T cells

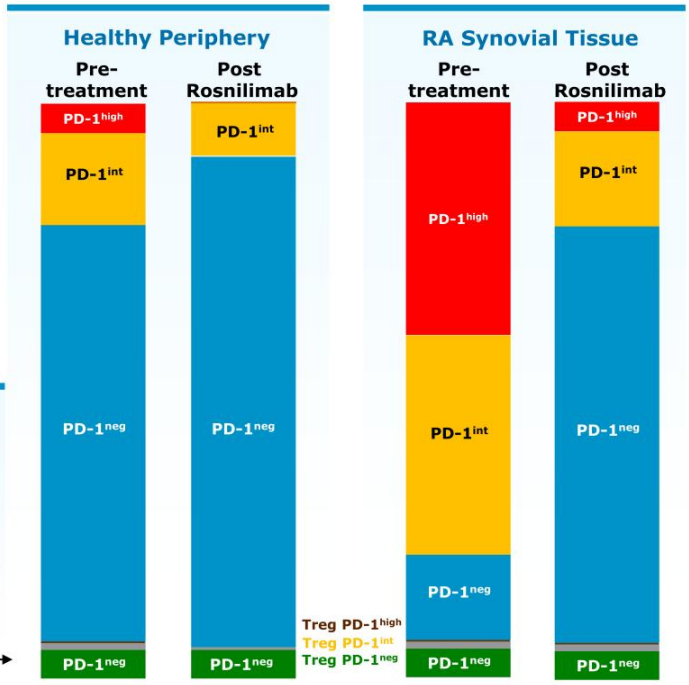
Leverages natural immune regulatory pathway to safely restore immune homeostasis

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.

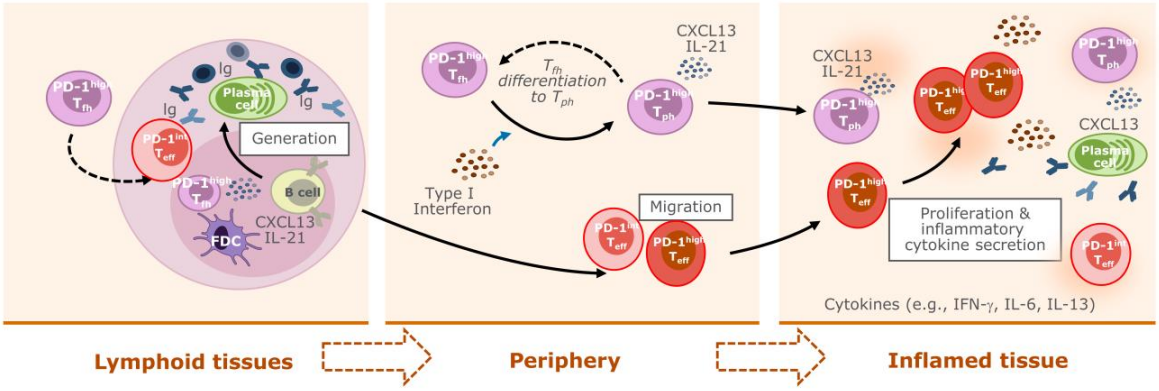
PD-1 is expressed preferentially on activated T_{eff} and T_{fh}/T_{ph} cells that mediate autoimmune pathology

T_{fh} (follicular helper)
T_{ph} (peripheral helper)

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

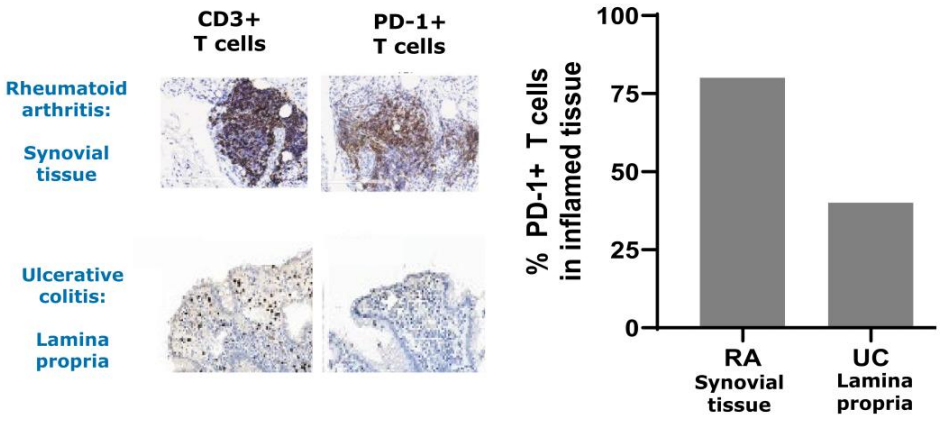
T_{eff} (effector)

- 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

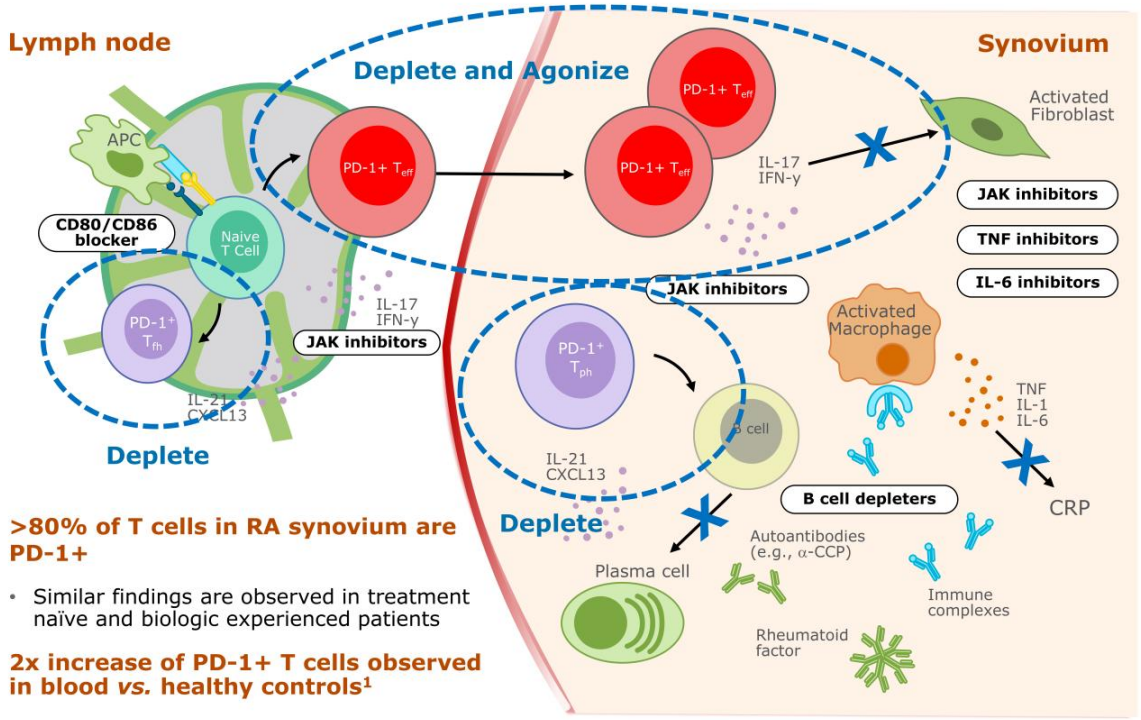


In systemic inflammatory diseases, a multiple fold increase of PD-1+ T cells is observed in periphery compared to healthy controls[1]

~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



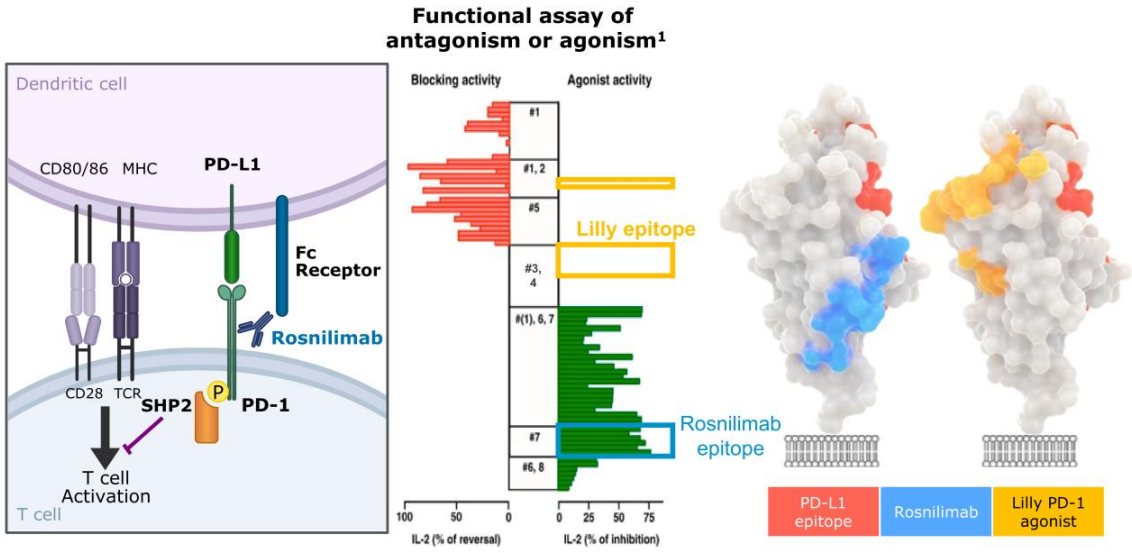
>80% of T cells in RA synovium are PD-1+

- 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



"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

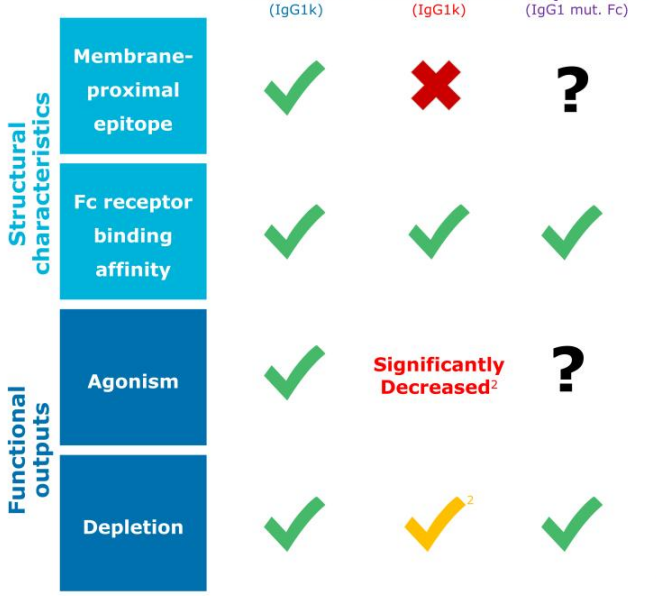
Parmley S, et al. ECCO 2024. February 2024.

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



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"

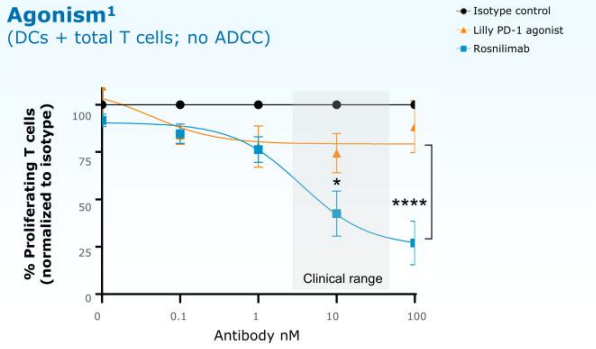
Additional LLY PD-1 variants engineered to have greater potency than peresolimab[1]

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

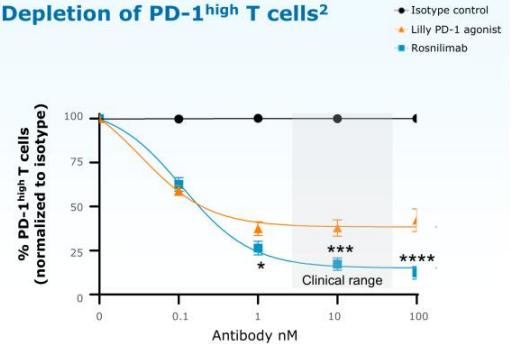
1. Eli Lilly patents; WO2024196694A2 and WO2024040206A; 2. Less potent depletion and significantly weaker agonism from membrane-distal binding epitope results in wider immune synapse and lower clustering of PD-1; 3. Fc binding to FcγRIIb only.

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

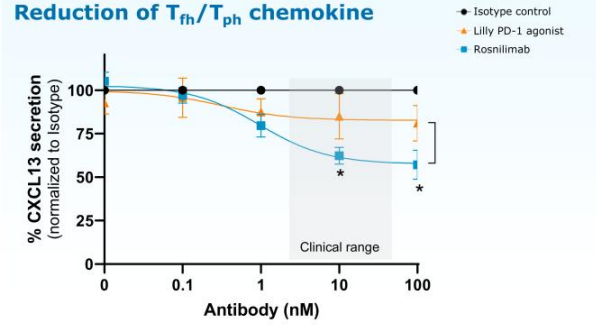
Agonism[1]
(DCs + total T cells; no ADCC)



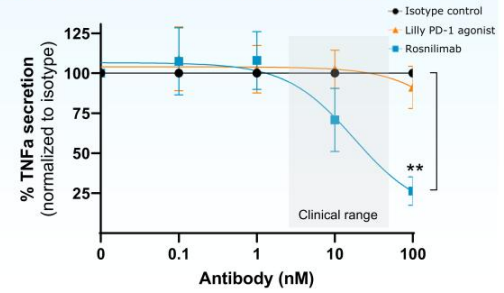
Depletion of PD-1^{high} T cells[2]



Reduction of T_{fh}/T_{ph} chemokine



Reduction of inflammatory cytokine[3]

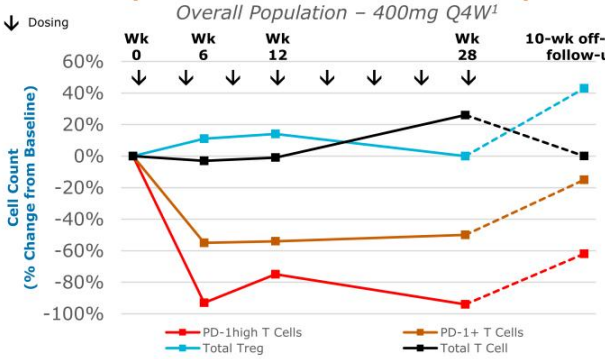


1. Healthy donor purified DCs + autologous total T cells stimulated with anti-CD3, cultured for 3 days for assessment of T cell proliferation
2. Anti-CD3+ anti-CD28 stimulation of RA patient PBMCs for assessment of depletion and agonism MOA, representative data from N=8 donors. Two-way ANOVA, Tukey's multiple comparison test.
3. TNFa secretion measured in anti-CD3+ anti-CD28 stimulation of purified DC+T cells from N=4 healthy donors, ****P<0.0001, ***p<0.001, **p<0.01, *p<0.05.

Rosnilimab's rapid and sustained ~90% reduction of PD-1^{high} T cells supports favorable T cell composition reflective of immune homeostasis

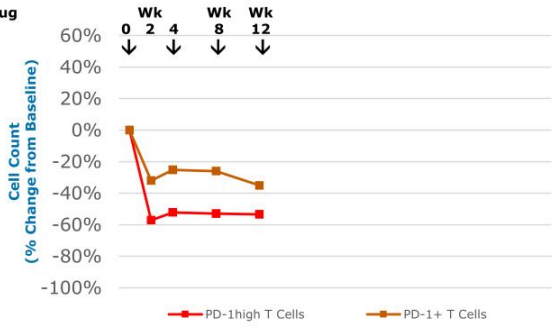
Representative Rosnilimab T Cell Impact

Overall Population – 400mg Q4W[1]



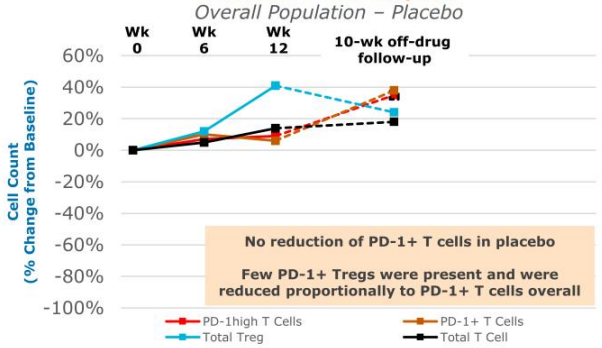
LLY Peresolimab T Cell Impact

Overall Population – Pooled Doses[2]



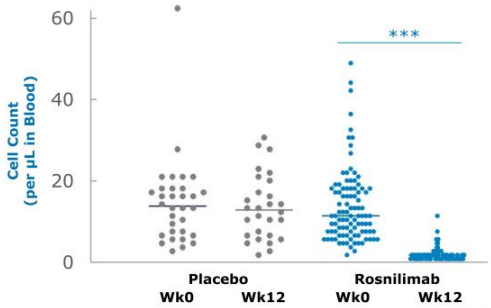
Placebo T Cell Impact

Overall Population – Placebo



Rosnilimab T_{ph} Impact

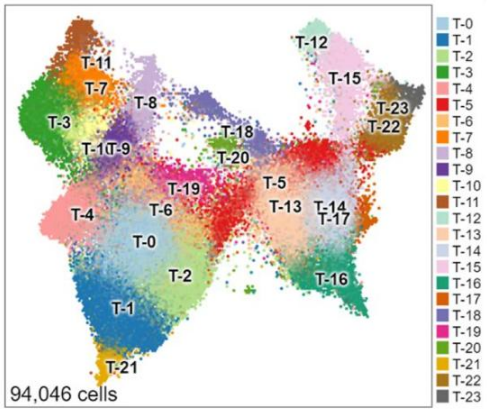
Overall Population[3]



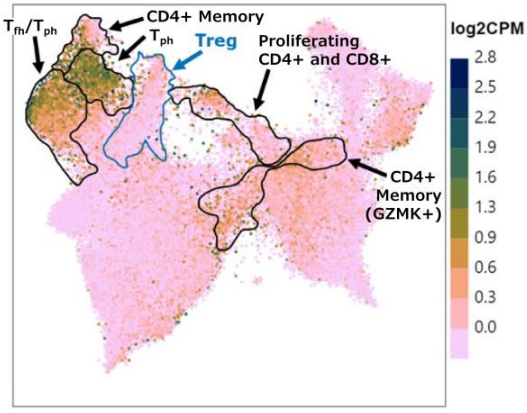
T_{ph} – T peripheral helper cell defined as CD3+ CD4+ CD45RA- PD-1^{high} CXCR5-, ***p<0.001 1. Rosnilimab 400mg Q4W dose is representative of all other doses 2. Benschop, R. ACR 2023, Eli Lilly peresolimab Phase 2a data 3. Pooled rosnilimab data

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

RA synovium T cell UMAP clustering



PD-1 Expression across T cell clusters



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

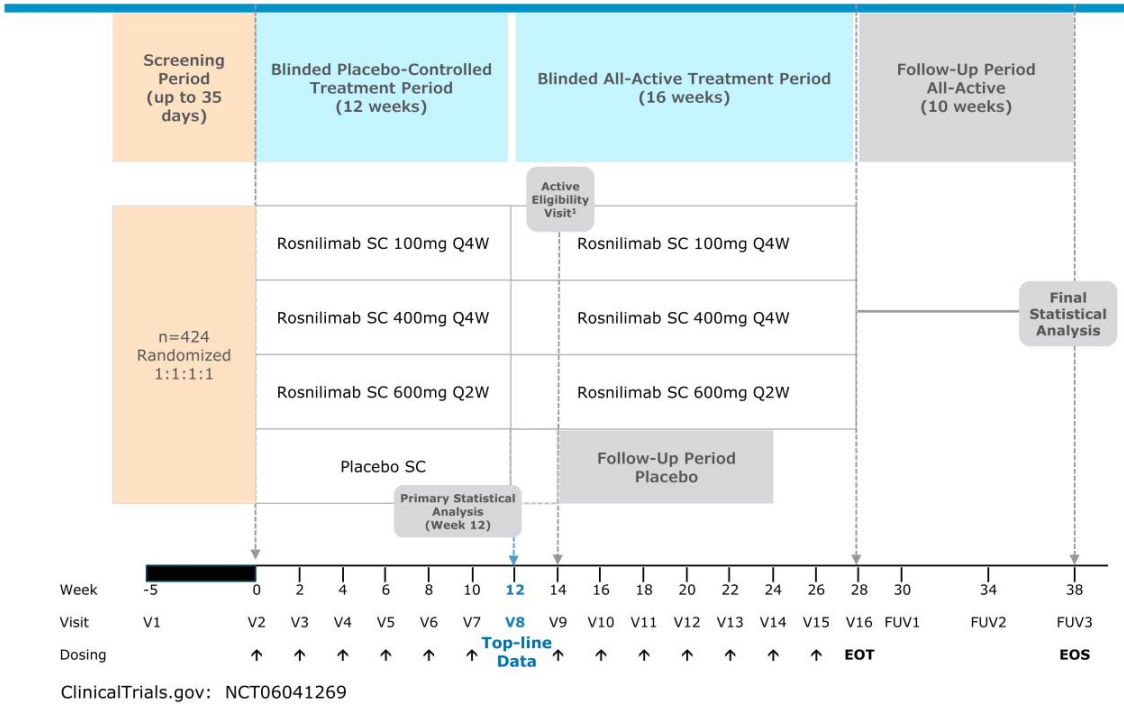
PD-1+ Tregs may be pro-inflammatory and induce IFNγ, IL-17A, TNFα

In Phase 2b RA trial, few PD-1+ Tregs were present in periphery and were reduced proportionally to PD-1+ T cells overall

Minimal impact on total T cells with an increase in total Tregs

1. Uniform manifold approximation and projection (UMAP) clusters of T cells from RA patient synovium with arrows identifying T_{ph} and T_{fh}/T_{ph} 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.

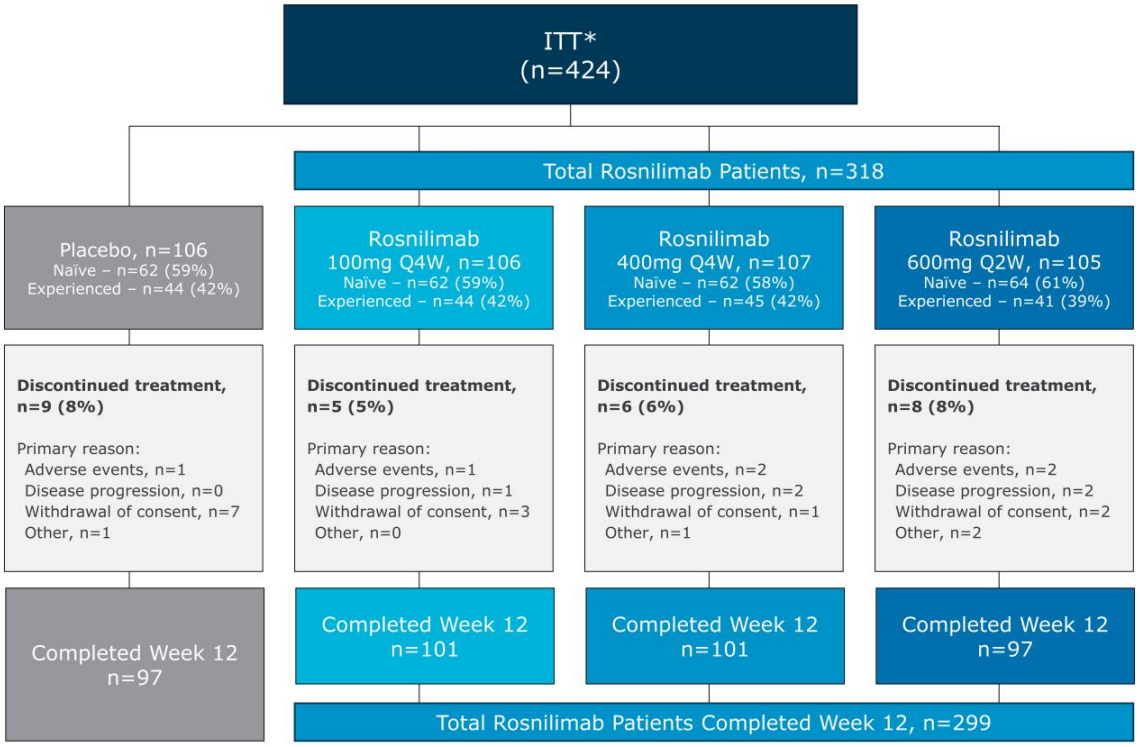
Rosnilimab RENOIR Phase 2b in moderate-to-severe RA



ClinicalTrials.gov: NCT06041269

1. Blinded study drug treatment will continue for active treatment group subjects that achieve Clinical Disease Activity Index (CDAI) low disease activity (CDAI ≤10)
Note: All patients in trial (rosnilimab and placebo arms) are required to be on background cDMARD.

Patient disposition through Week 12



*ITT – Intent-to-Treat (all randomized participants)

Baseline disease characteristics and demographics

Baseline Characteristic	Placebo (n=106)	100mg Q4W (n=106)	400mg Q4W (n=107)	600mg Q2W (n=105)	Overall (N=424)
Age, years, mean (SD)	58 (11)	57 (10)	57 (12)	56 (11)	57 (11)
Female, n (%)	83 (78%)	79 (75%)	79 (74%)	80 (76%)	321 (76%)
Weight (kg), mean (SD)	78 (17)	78 (19)	81 (19)	77 (16)	78 (18)
Geographic region, n (%) US Ex-US	 35 (33%) 71 (67%)	 34 (32%) 72 (68%)	 35 (33%) 72 (67%)	 26 (25%) 79 (75%)	 130 (31%) 294 (69%)
Race, n (%) White Black or African American Asian Other	 102 (96%) 3 (3%) 0 (0%) 1 (1%)	 102 (96%) 1 (<1%) 0 (0%) 3 (4%)	 103 (96%) 4 (4%) 0 (0%) 0 (0%)	 101 (96%) 4 (4%) 0 (0%) 0 (0%)	 408 (96%) 12 (3%) 0 (0%) 4 (1%)
Duration of disease, years, mean (SD)	11 (9)	11 (10)	9 (8)	10 (9)	10 (9)
DAS28-CRP, mean (SD)	5.7 (0.8)	5.6 (0.8)	5.7 (0.9)	5.7 (0.8)	5.6 (0.8)
CDAI, mean (SD) CDAI >22, n (%)	37.9 (10.2) 101 (95%)	37.2 (10.6) 101 (95%)	37.1 (10.6) 102 (95%)	38.6 (11) 100 (95%)	37.7 (10.6) 404 (95%)
TJC68, mean (SD)	23 (13)	22 (12)	22 (12)	23 (13)	22 (12)
SJC66, mean (SD)	14 (7)	15 (7)	14 (7)	16 (9)	15 (8)
CRP, mean (SD)	16 (22)	17 (20)	21 (26)	19 (28)	18 (24)

DAS28-CRP – Disease Activity Score 28-C-reactive protein; CDAI – Clinical Disease Activity Index; TJC68 – tender joint count, 68 joints; SJC66 – swollen joint count, 66 joints; CRP – high-sensitivity C-reactive protein

RA patients have significant co-morbidities which are further exacerbated with treatment

Increased co-morbidity rate in RA patients vs. general population

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

Black box warnings for increasing SAE incidence of commercial products have not impeded blockbuster sales

HUMIRA® adalimumab	ORENCIA® (abatacept)	RINVOQ® upadacitinib	Rituxan® Rituximab
$4.5B RA sales[4]	**$3.6B RA sales[4]**	**$2.3B RA sales[4]**	**~$1B RA sales**
Black box warning		**Black box warning**	**Black box warning**
~30% infection rate vs. 28% placebo[5]	~54% infection rate vs. 48% placebo[5]	~20% infection rate vs. 18% placebo[5]	~39% infection rate vs. 34% placebo[5]
~0.7% MACE rate vs. 0.4% placebo[5]	~0.2% MACE rate vs. 0.5% placebo[5]	~3.4% MACE rate vs. 2.5% placebo[5]	~1.7% MACE rate vs. 1.3% placebo[5]
		~4.2% malignancy rate vs. 2.9% placebo[5]	

1. Avina-Zubieta et al., A&R, 2008, 2. Fazal et al., BMC Rheumatology, 2024, 3. Smitten et al., ART, 2008, 4. Evaluate Pharma 2023 WW RA sales, 5. Phase 3 registrational data from product labels

Rosnilimab was safe and well tolerated with similar adverse event rates vs. placebo

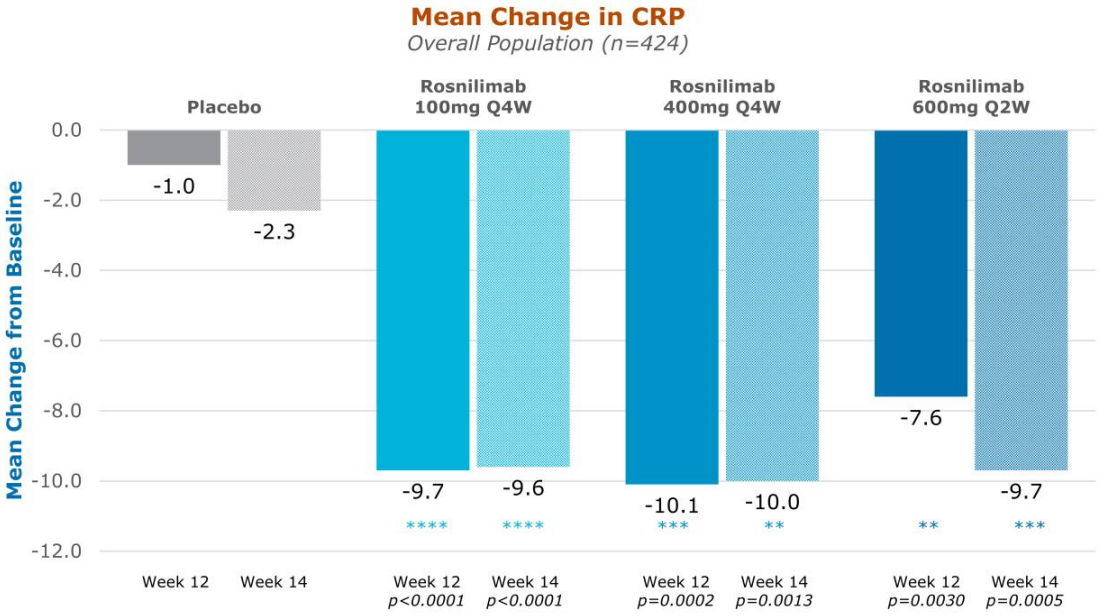
Adverse Events through Week 12, n (%)	Placebo (n=106)	100mg Q4W (n=106)	400mg Q4W (n=107)	600mg Q2W (n=105)
Participants with any AE, n (%)	**36 (34%)**	**51 (48%)**	**48 (45%)**	**38 (36%)**
Any SAE[1]	1 (1%)	1 (1%)	1 (1%)	3 (3%)
Any Drug-Related SAE	1 (1%)	0 (0%)	0 (0%)	0 (0%)
Severe AE[2]	2 (2%)	1 (1%)	0 (0%)	4 (4%)
Drug-Related AE	18 (17%)	13 (12%)	18 (17%)	17 (16%)
Infections	14 (13%)	24 (23%)	21 (20%)	12 (11%)
AE Leading to Treatment Discontinuation	1 (1%)	1 (1%)	2 (2%)	2 (2%)
Participants with any AEs ≥5%, n				
Headache	4 (4%)	7 (7%)	6 (6%)	4 (4%)
Upper respiratory tract infection	1 (1%)	7 (7%)	2 (2%)	3 (3%)

- No malignancies
- No MACE
- No elevation of serious infections vs. placebo
- No anaphylaxis or systemic hypersensitivity associated with rosnilimab
- Low incidence of injection site reactions and similar to placebo

Safety profile up to Week 28 remains consistent with reported profile through Week 12

1. SAEs (severe unless otherwise noted): pneumonia – mild (100 mg Q4W); meniscus tear – moderate (400 mg Q4W); anaphylaxis from wasp sting, ureter stone, and cholecystitis/pericardial effusion (600mg Q2W) and cellulitis/diarrhea (placebo);
2. Severe AE (excluding SAEs): flu (100mg Q4W); RA flare (600mg Q2W); macular degeneration/retinal hemorrhage (placebo).

Rosnilimab significantly decreased CRP demonstrating an objective reduction of inflammation



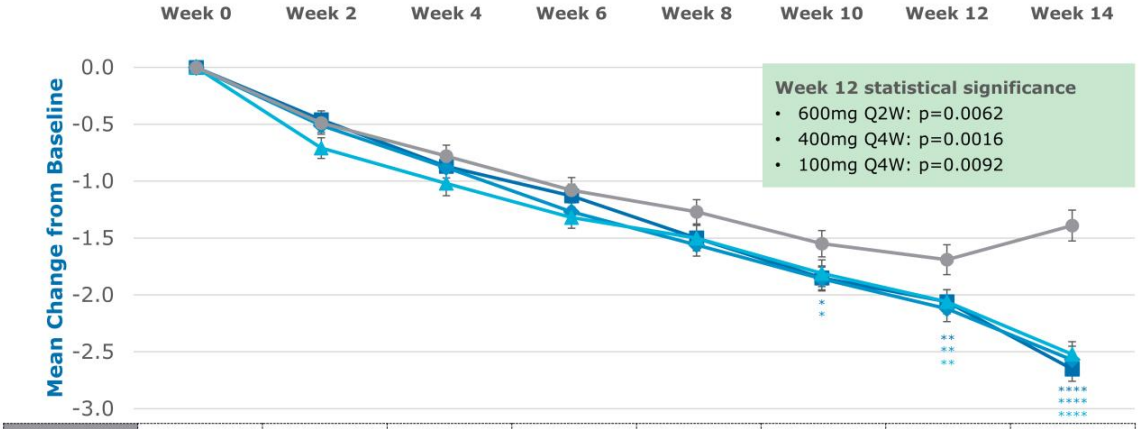
- Clear and consistent on target reduction at all rosnilimab doses
- No immunological change on placebo at both Week 12 or Week 14
- Decrease of CRP demonstrates objective reduction of inflammation beyond directly targeting T cells

CRP – high-sensitivity C-reactive protein, ****p<0.0001, ***p<0.001, **p<0.01, *p<0.05.

Rosnilimab met primary endpoint of mean change from baseline in DAS28-CRP at Week 12 for all active doses

Mean Change in DAS28-CRP Over Time

Overall Population (n=424)



Placebo	n=106	n=105	n=103	n=100	n=100	n=99	n=97	n=90
100mg Q4W	n=106	n=106	n=106	n=105	n=104	n=102	n=101	n=101
400mg Q4W	n=107	n=107	n=107	n=105	n=104	n=102	n=101	n=99
600mg Q2W	n=105	n=104	n=104	n=103	n=99	n=97	n=97	n=97
Rosnilimab Total	***n=318***	***n=317***	***n=317***	***n=313***	***n=307***	***n=301***	***n=299***	***n=297***

- All rosnilimab doses statistically significant at Week 12
- All rosnilimab doses continue to improve into Week 14 with no evidence of flattening
- Following Week 14 visit, placebo patients proceeded to post treatment follow-up

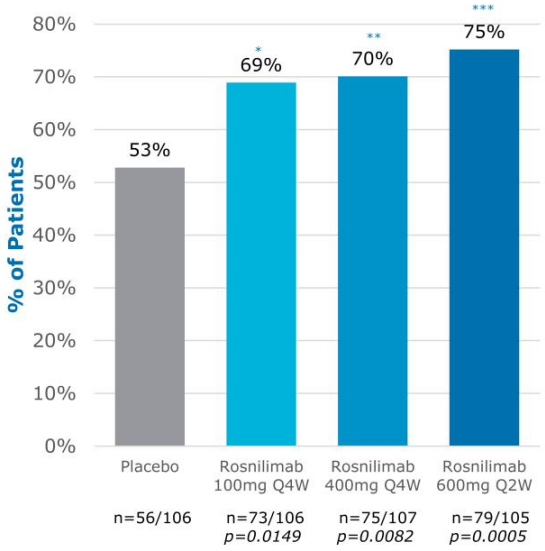
Placebo
Rosnilimab - 100mg Q4W
Rosnilimab - 400mg Q4W
Rosnilimab - 600mg Q2W

Data table includes n values for patients that completed visit; DAS28-CRP based on differential weighting of individual measures, including patient's general health, CRP and a count of 28 swollen and tender joints, with a score ranging from 0 to 9.4. ****p<0.0001, ***p<0.001, **p<0.01, *p<0.05, Standard error (SE) used to present figures of least squares mean changes from baseline.

Statistically significant clinically meaningful benefit was observed within 3-months for majority of rosnilimab patients

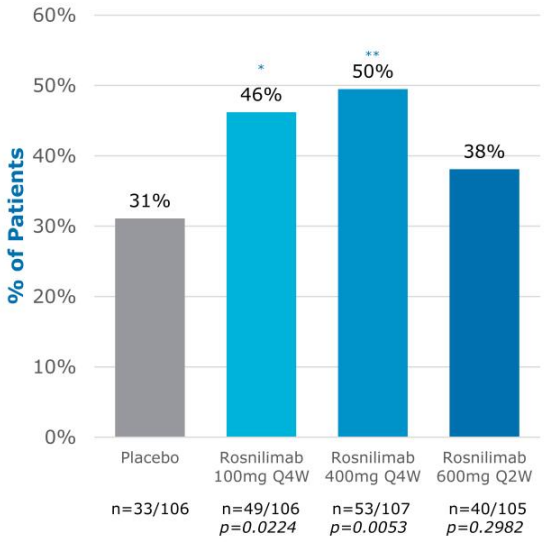
ACR20 at Week 12

Overall Population (n=424)



CDAI LDA of ≤ 10 at Week 12

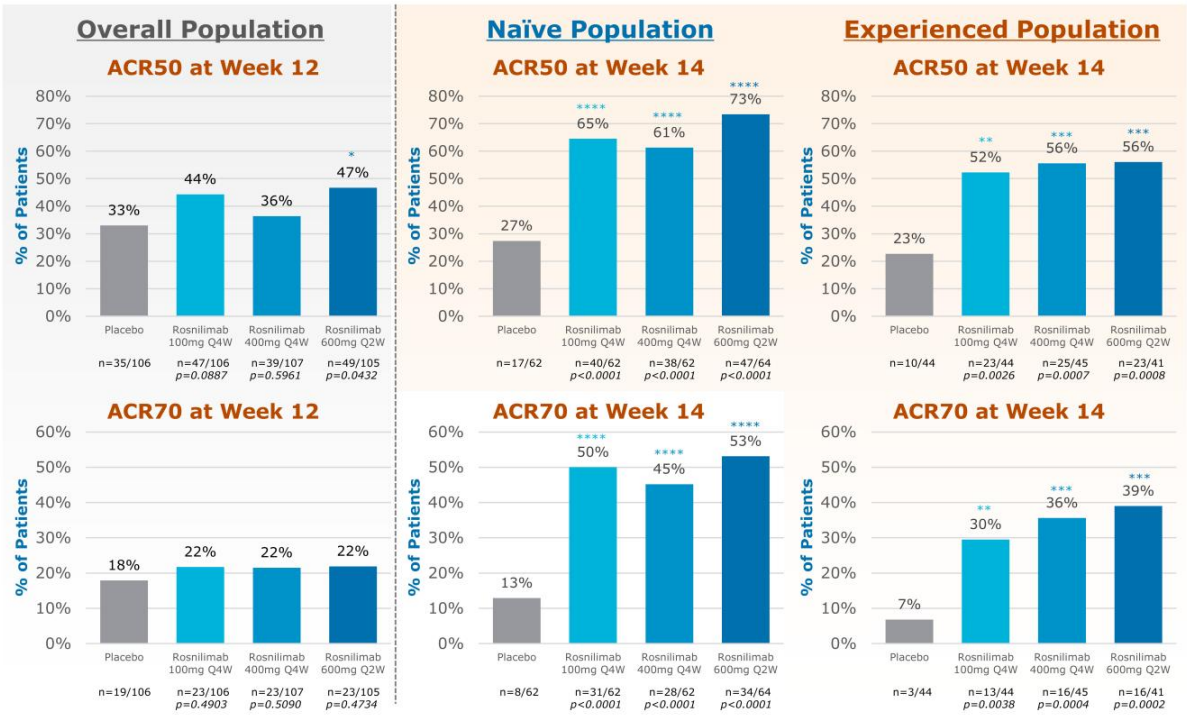
Overall Population (n=424)



- Physicians minimally seek to achieve LDA by 6-months
- Approximately half of rosnilimab-treated patients achieved LDA by Week 12

CDAI, a compositive assessment, is used to measure the severity of RA based on the sum of four assessment tools: the number of swollen and tender joints, the patient's global disease activity index, and the physician's global disease activity index. The score ranges from 0 to 76, with a score ≤ 10 is the threshold for LDA. ****p<0.0001, ***p<0.001, **p<0.01, *p<0.05

Consistent with DAS28 trends, ACR50/70 responses continue to materially deepen beyond Week 12 regardless of prior therapy

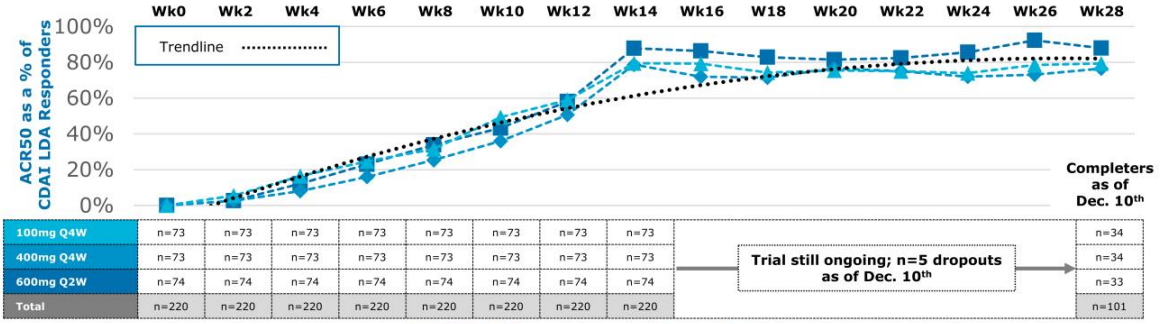


ACR50/70 - requires a patient to have a 50%/70% reduction in the number of swollen and tender joints, and a reduction of 50%/70% in three of the following five parameters: physician global assessment of disease, patient global assessment of disease, patient assessment of pain, CRP or erythrocyte sedimentation rate, and degree of disability in Health Assessment Questionnaire (HAQ) score. ****p<0.0001, ***p<0.001, **p<0.01, *p<0.05

69% (220/318) of patients achieved CDAI LDA at Week 14 and appear to show sustained ACR50 and potentially deepening ACR70 responses

ACR50 Responses Over Time

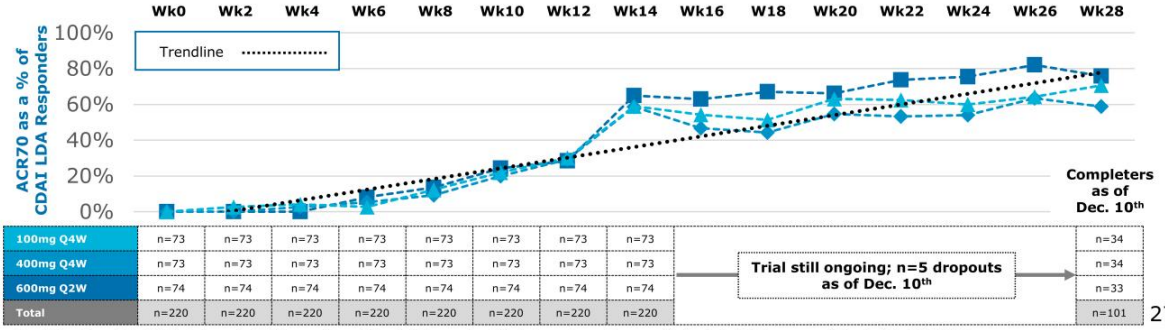
CDAI LDA Responders (Overall Population, n=220 responders at Week 14)



100mg Q4W	n=73	n=73	n=73	n=73	n=73	n=73	n=73	n=73	Trial still ongoing; n=5 dropouts as of Dec. 10th						n=34
400mg Q4W	n=73	n=73	n=73	n=73	n=73	n=73	n=73	n=73							n=34
600mg Q2W	n=74	n=74	n=74	n=74	n=74	n=74	n=74	n=74							n=33
Total	n=220	n=220	n=220	n=220	n=220	n=220	n=220	n=220							n=101

ACR70 Responses Over Time

CDAI LDA Responders (Overall Population, n=220 responders at Week 14)



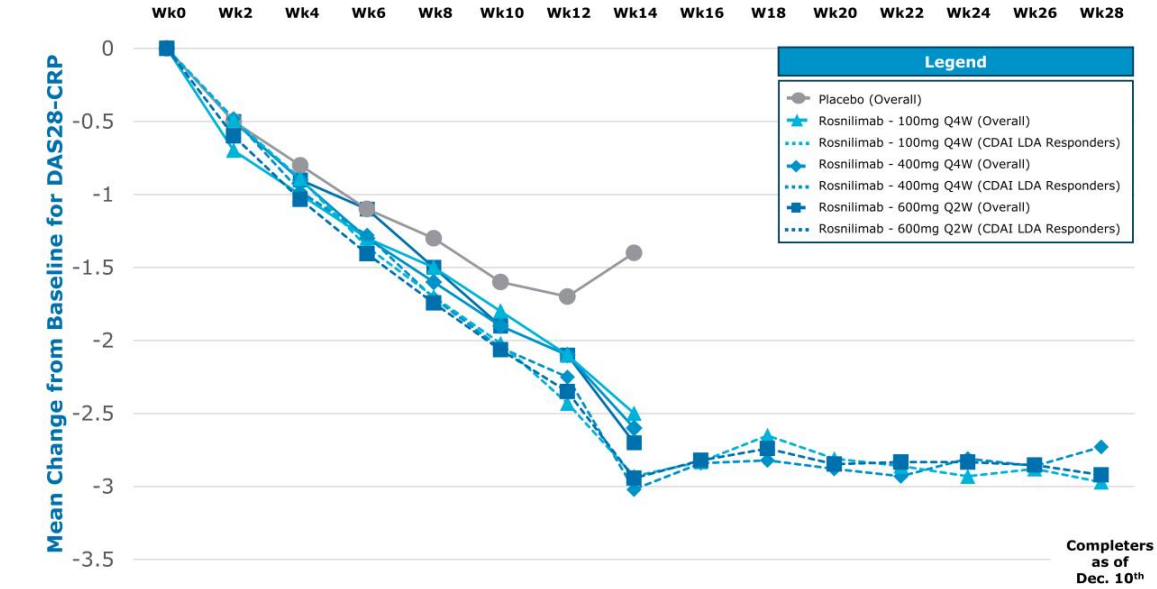
100mg Q4W	n=73	n=73	n=73	n=73	n=73	n=73	n=73	n=73	Trial still ongoing; n=5 dropouts as of Dec. 10th						n=34
400mg Q4W	n=73	n=73	n=73	n=73	n=73	n=73	n=73	n=73							n=34
600mg Q2W	n=74	n=74	n=74	n=74	n=74	n=74	n=74	n=74							n=33
Total	n=220	n=220	n=220	n=220	n=220	n=220	n=220	n=220							n=101

As of the Dec. 10, 2024 data cutoff, this portion of the trial remains blinded and ongoing, and full clinical data are anticipated in Q2 2025

69% (220/318) of patients achieved CDAI LDA at Week 14 and appear to show sustained DAS28-CRP responses

Mean Change in DAS28-CRP Over Time

CDAI LDA Responders vs. Overall Population

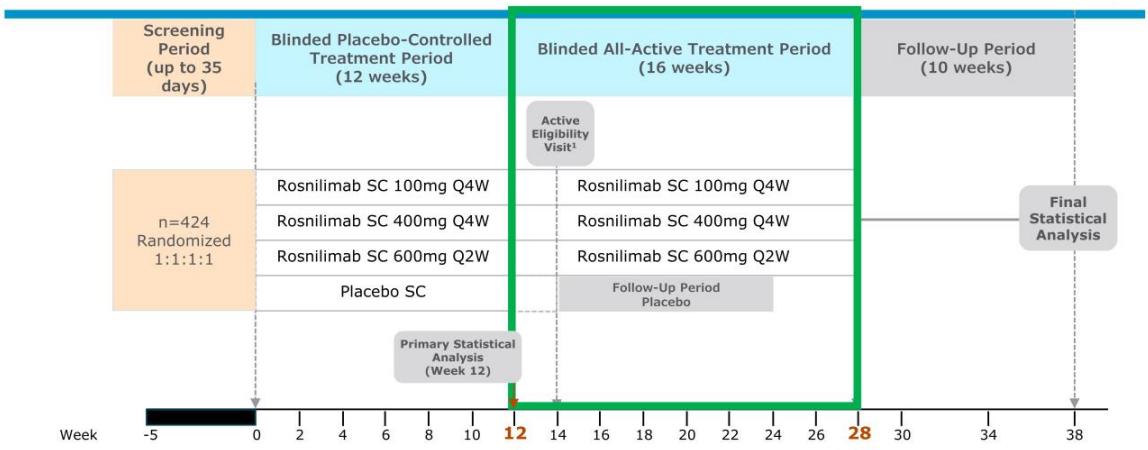


100mg Q4W	n=73	n=73	n=73	n=73	n=73	n=73	n=73	n=73	Trial still ongoing; n=5 dropouts as of Dec. 10th	n=34
400mg Q4W	n=73	n=73	n=73	n=73	n=73	n=73	n=73	n=73		n=34
600mg Q2W	n=74	n=74	n=74	n=74	n=74	n=74	n=74	n=74		n=33
Total	n=220	n=220	n=220	n=220	n=220	n=220	n=220	n=220		n=101

Completers as of Dec. 10th

As of the Dec. 10, 2024 data cutoff, this portion of the trial remains blinded and ongoing, and full clinical data are anticipated in Q2 2025

Next steps for rosnilimab in RA



At Week 14, 69% (71% of bio-naive and 66% of bio-experienced) or 220 of the 318 rosnilimab patients, across all doses, were CDAI LDA responders

CDAI LDA responders appear to show sustained CDAI LDA and ACR50 and potentially deepening ACR70 responses out to Week 28

Blinded all-active treatment period ongoing

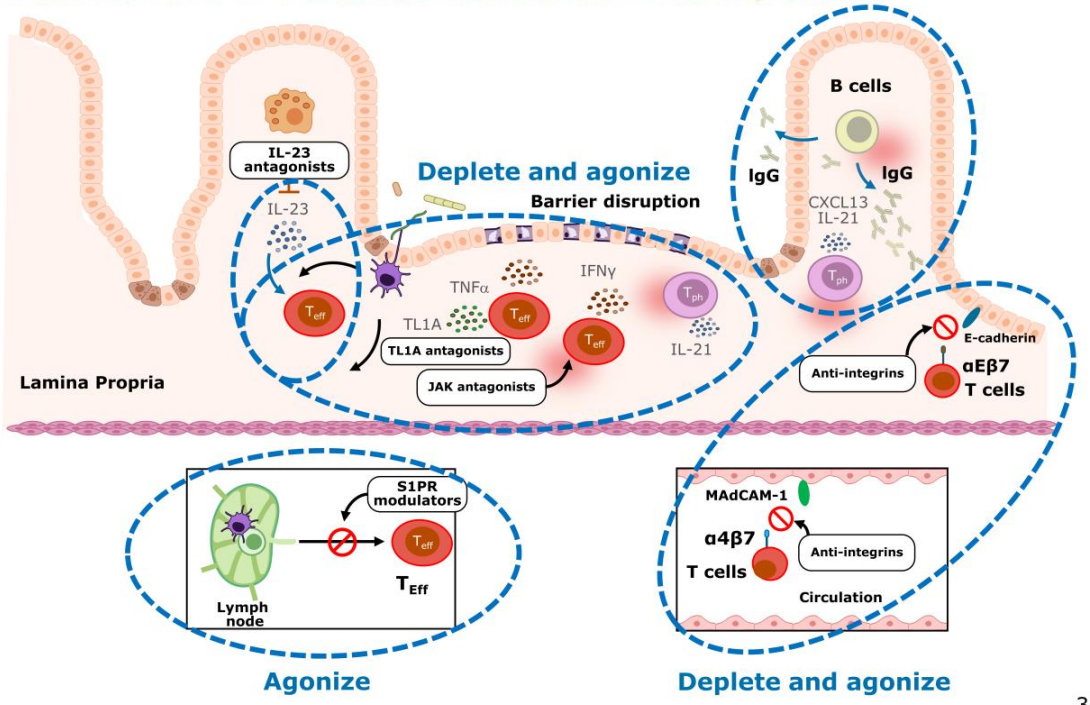
Later in 2025, report out

- Phase 2b full clinical data Q2 2025
- Additional translational data Q2 2025
- Present at a medical conference

1. Blinded study drug treatment will continue for active treatment group subjects that achieve Clinical Disease Activity Index (CDAI) low disease activity (CDAI ≤10)
Note: All patients in trial (rosnilimab and placebo arms) are required to be on background cDMARD

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

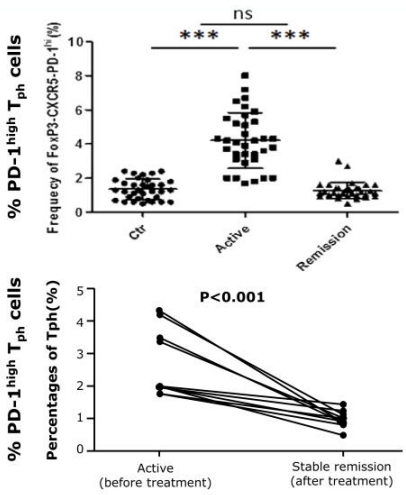
- >40% of T cells in lamina propria in UC are PD-1+
- 2x increase of PD-1+ T cells observed in blood *vs.* healthy controls[1]



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

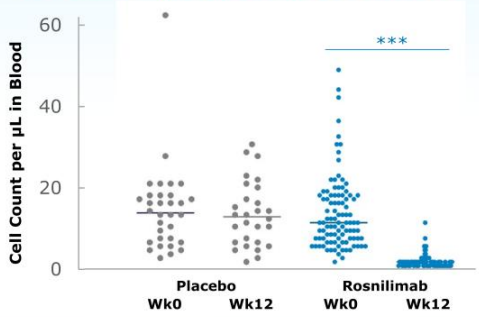
T_{ph} impact seen in RA Phase 2b trial relevant to UC biology and correlates to reduction of remission

PD-1^{high} T_{ph} cells are reduced with remission in UC[1,2]

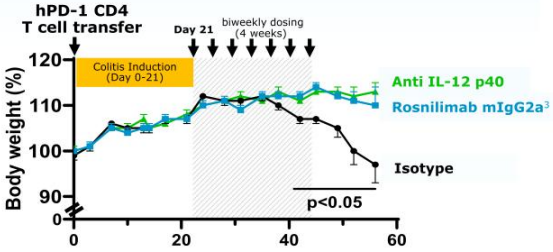


Reduction of T_{fh}/T_{ph} cells should impact plasma cell generation and autoantibody levels, including anti-microbial IgG antibodies that are contributing to colonic inflammation and barrier disruption[4]

Rosnilimab T_{ph} Impact in RA Phase 2b



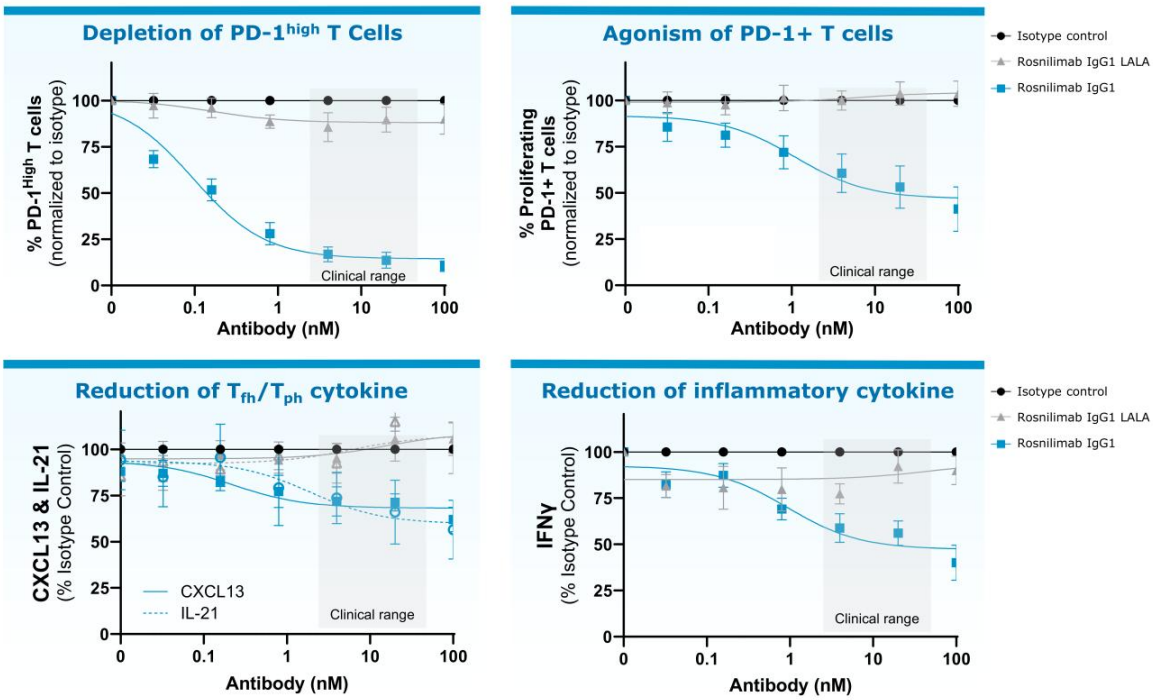
Therapeutic dosing of rosnilimab demonstrated efficacy in a murine model of colitis



Parmley et. al. UEGW 2024. October 2024

1. PD-1^{high} T_{ph} 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.

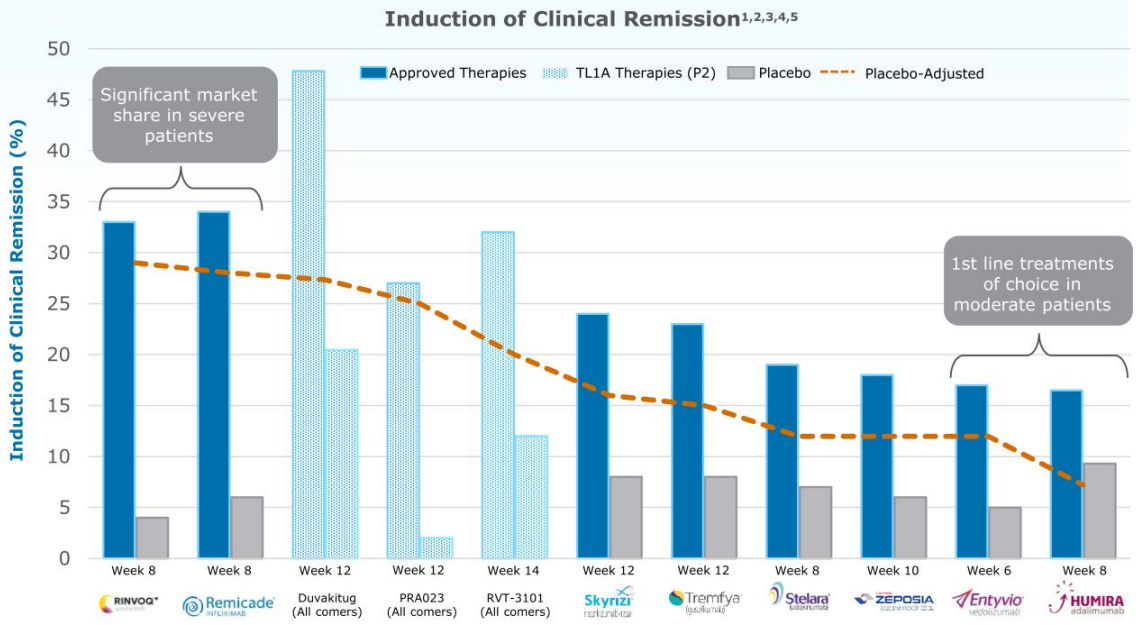
Rosnilimab's potent depletion and agonism reduces T cell proliferation and inflammatory cytokines that disrupt barrier function



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

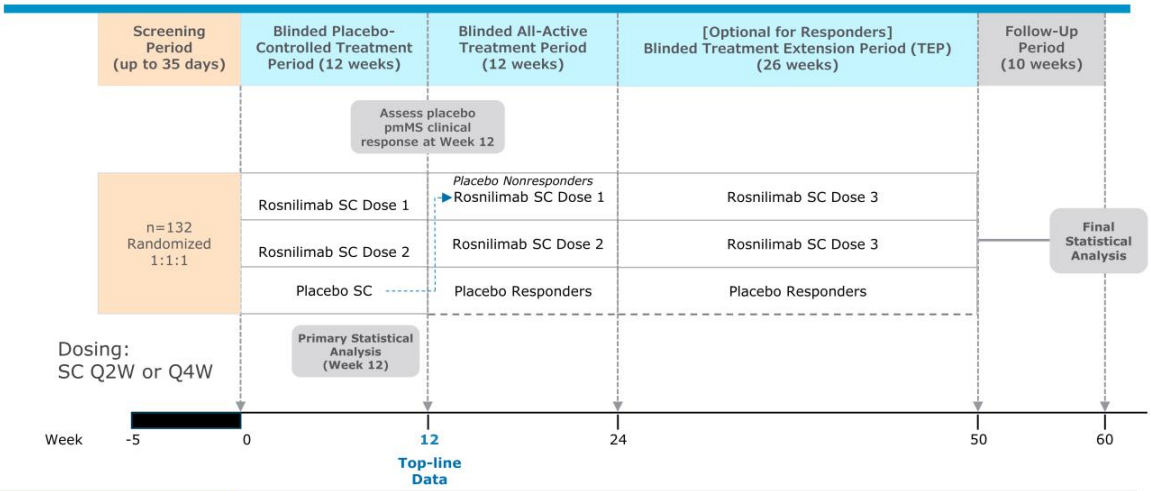
Following remission on induction therapy, one third to one half of patients relapse within 1 year[1]



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 Q4 2025



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

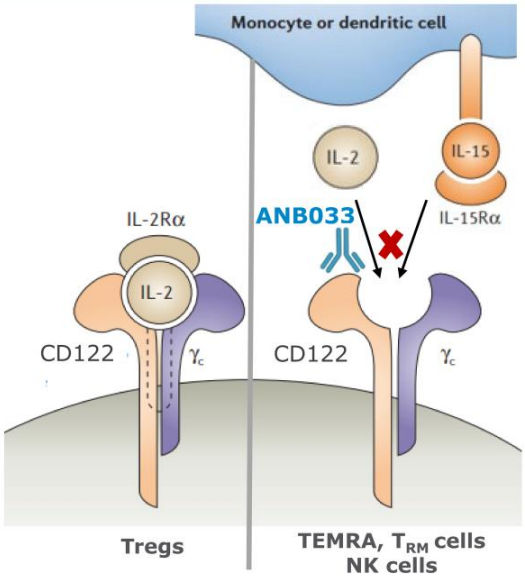
ANB033 (CD122 antagonist)

Autoimmune and Inflammatory Diseases

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

Phase 1 trial ongoing 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

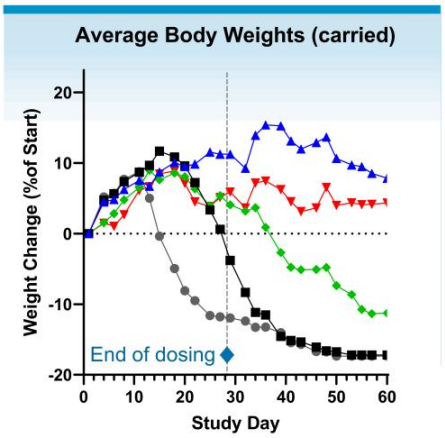
- T_{RM} cells require IL-15 for survival
- May potentially drive durable response

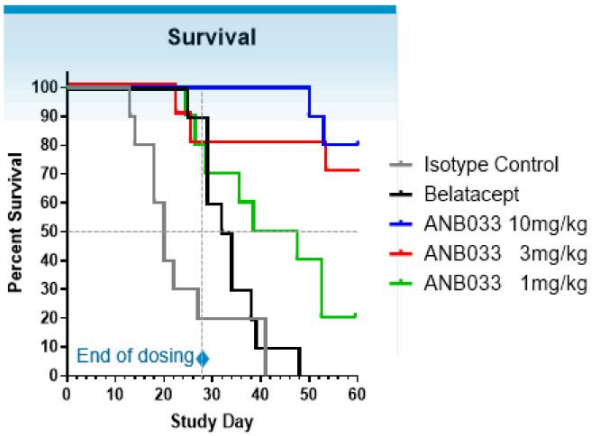
ANB033: Durable survival in GVHD model

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.

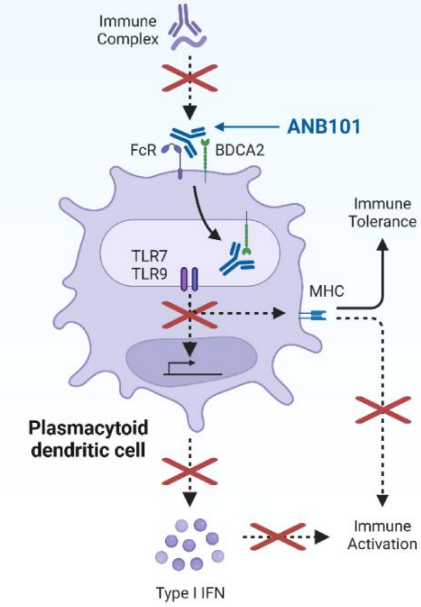
ANB101
(BDCA2 modulator)

Autoimmune and Inflammatory Diseases

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

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.

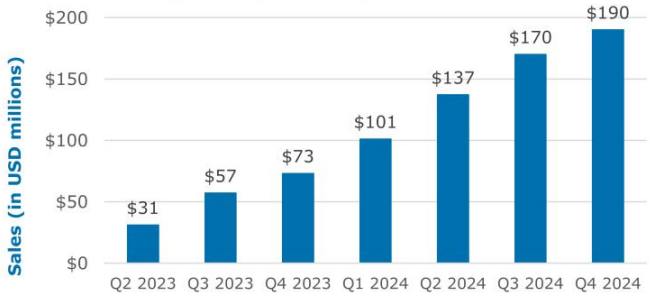
3. GSK analyst consensus as of 11/14/2024 converted to USD (1.25 conversion rate), GSK website - https://www.gsk.com/en-gb/investors/analyst-consensus/

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

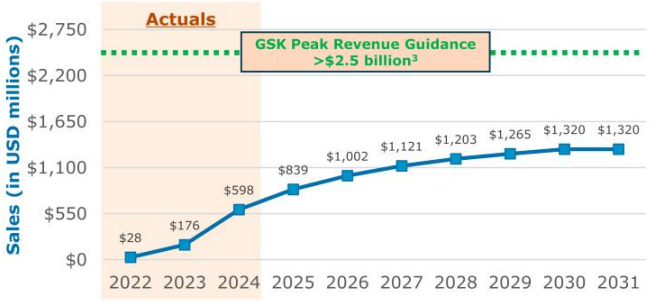
Note: Separate sale of Anaptys' *Zejula* (niraparib) royalty interest occurred in September 2022 to DRI Healthcare Trust for $35MM upfront + $10MM potential milestone upon FDA approval of *Zejula* for the treatment of endometrial cancer, to the extent that such approval occurs on or before 12/31/25. 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

- $190MM Q4 2024 Sales (>100% YoY growth)[1]
- $75MM milestone anticipated in 2025/2026 for *Jemperli* $1B annual WW sales
- 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: Approved in US mid-'24; in EU Jan. '25
- 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 1/27/2025 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 Program for Out-Licensing

Etokimab (IL-33 antagonist)

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

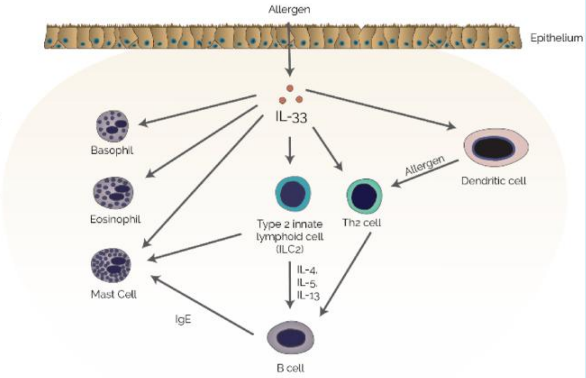
- Binding affinity of etokimab is <1 pM; best-in-class based on competitor affinities published in patents and literature
- Targeting IL-33 cytokine rather than IL-33 receptor (ST2) has potential to not only modify disease, but also drive epithelial remodeling

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)