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: June 3, 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 June 03, 2025, AnaptysBio, Inc. ("***AnaptysBio***" or the "***Company***") issued a press release announcing positive rosnilimab data updated through six months in robust Phase 2b clinical trial in rheumatoid arthritis, a copy of which is attached hereto as Exhibit 99.1.

On June 03, 2025, AnaptysBio presented a slide presentation regarding the rosnilimab data, a copy of which is attached hereto as Exhibit 99.2.

On June 03, 2025, AnaptysBio updated its corporate investor presentation, a full copy of which is attached hereto as Exhibit 99.3.

The information in this Item 7.01, including Exhibits 99.1, 99.2 and 99.3, 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.

Item 8.01. Other Events.

On June 03, 2025, AnaptysBio announced positive rosnilimab data updated through six months in robust Phase 2b RENOIR clinical trial in rheumatoid arthritis.

AnaptysBio announced that investigational rosnilimab, a depleter and agonist targeting PD-1+ T cells, demonstrated a best-in-disease profile in patients with moderate-to-severe rheumatoid arthritis (RA). In the robust, global 424-patient Phase 2b trial, rosnilimab achieved JAK-like efficacy on multiple clinically meaningful measures, including low disease activity (LDA) and remission on the Clinical Disease Activity Index (CDAI), as well as ACR70 response, over a six-month period. Furthermore, responses were then durable for at least two months off drug. Rosnilimab was safe and well tolerated.

Exhibit Number	Exhibit Title or Description
99.1	Press release issued by AnaptysBio, Inc. regarding the positive rosnilimab data, dated June 03, 2025.
99.2	Presentation regarding updated rosnilimab data, presented June 03, 2025.
99.3	Anaptys Corporate Presentation June 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: June 03, 2025

By: /s/ Dennis Mulroy

Name: Dennis Mulroy

Title: Chief Financial Officer

Anaptys Announces Positive Rosnilimab Data Updated Through Six Months in Robust Phase 2b Trial in RA

- Best-in-disease profile with JAK-like efficacy and monthly (Q4W) dosing in both three-month placebo-controlled and six-month blinded treatment period
- Favorable safety and tolerability, particularly when compared to standard of care biologics or JAKs
- Max response rates have not yet been observed; strict continuation criteria at three months in this Phase 2b trial excluded many patients who either achieved or were trending toward LDA and ACR50
- Durable responses for at least two months off drug, with potential for extended dosing intervals (e.g. Q8W) in the maintenance setting
- Conference call and webcast today at 4:15pm ET/1:15pm PT

SAN DIEGO, June 3, 2025 — AnaptysBio, Inc. (Nasdaq: ANAB), a clinical-stage biotechnology company focused on delivering innovative immunology therapeutics, today announced that investigational rosnilimab, a depleter and agonist targeting PD-1+ T cells, demonstrated a best-in-disease profile in patients with moderate-to-severe rheumatoid arthritis (RA). In the robust, global 424-patient Phase 2b trial, rosnilimab achieved JAK-like efficacy on multiple clinically meaningful measures, including low disease activity (LDA) and remission on the Clinical Disease Activity Index (CDAI), as well as ACR70 response, over a six-month period. Furthermore, responses were then durable for at least two months off drug. Rosnilimab was safe and well tolerated, particularly when compared to standard of care biologics or JAKs.

“This is exciting news for patients living with RA, who cycle through numerous treatment options without achieving a low level of disease activity shown to be correlated with slowing of disease progression. The updated Phase 2b data for rosnilimab confirm a best-in-disease profile through six months that is safe and well tolerated, with JAK-like efficacy and the potential to be administered in a monthly, subcutaneous dose. Additionally, responses after six months of treatment are durable for at least two months off drug, suggesting the potential for extended, every eight-week dosing intervals during maintenance treatment,” said Daniel Faga, president and chief executive officer of Anaptys. “These findings, consistent with compelling and objective translational data, surpass our target product profile for rosnilimab in the ~$20 billion U.S. RA market. Our next priority is to complete the ongoing Phase 2 study for rosnilimab in UC, which is on track to report initial data in Q4 2025.”

Most rosnilimab patients showed symptomatic and clinical improvement by three months. As previously reported, all three doses of rosnilimab achieved statistically significant reductions in mean change from baseline in DAS-28 CRP, the study’s primary endpoint, as well as for ACR20, at Week 12 versus placebo. During the three-month placebo-controlled period in both b/tsDMARD-naïve and b/tsDMARD-experienced patients, rosnilimab demonstrated a rapid onset of ACR20 response, an accepted Phase 3 registrational endpoint, as well as a substantial decrease in objective measures such as C-reactive protein (CRP), that were both comparable to the Phase 2b trial results of upadacitinib.

Rosnilimab demonstrated JAK-like efficacy with deepening of responses through six months on CDAI LDA, CDAI remission and ACR70. Importantly, this is particularly observed in the b/tsDMARD-experienced patients

for the 400mg Q4W and 600mg Q2W doses, showing a dose response relative to the 100mg Q4W dose. At baseline, patients had high disease activity with a mean CDAI of 38 and median CDAI of 36. Of the 318 rosnilimab patients in the intent to treat (ITT) population, CDAI of ≤10 (LDA) was achieved by 45% of patients by Week 12 and increased to 69% of patients (220 responders) across all three doses at Week 14, the timepoint required in the trial to be eligible to remain in the all-active treatment period on rosnilimab through Week 28. This sets a ceiling on the number of CDAI LDA responders at Week 28 when using a non-responder imputation (NRI) analysis of the ITT population to calculate rosnilimab response rates.

Max response rates for rosnilimab have not yet been observed as strict criteria at Week 14 prevented patients with meaningful improvement from continuing treatment in the trial. For example, 12 patients achieved CDAI LDA four or six weeks after their last rosnilimab dose at their first follow-up visit (Week 18), but were ineligible to continue in the all-active treatment period and imputed as non-responders at Week 28 in the below NRI analysis. If these 12 responding patients had been eligible to continue treatment, CDAI LDA would have been up to 232 responders (73%) of 318 rosnilimab patients in the ITT across all three doses. Of the remaining patients ineligible to enter the all-active treatment period, ~50% had achieved ACR20 at Week 14 and were trending toward CDAI LDA.

The following data table reports the results at Week 28 (six months) using a conservative NRI analysis of the ITT population of rosnilimab patients randomized in the trial, which counts as responses only patients who entered the all-active treatment period at Week 14 and were in response at Week 28:

Efficacy Measures	**Rosnilimab** 100mg Q4W	**Rosnilimab** 400mg Q4W	**Rosnilimab** 600mg Q2W
b/tsDMARD-Naïve *(n=188 rosnilimab patients)*	*n=62*	*n=62*	*n=64*
	Week 28 *(Δ from Week 12)*		
CDAI ≤ 10 (LDA)	66% *(+18%)*	53% *(+5%)*	72% *(+29%)*
CDAI ≤ 2.8 (Remission)	21% *(+13%)*	18% *(+10%)*	17% *(+12%)*
ACR50	58% *(+10%)*	52% *(+10%)*	69% *(+17%)*
ACR70	53% *(+27%)*	37% *(+9%)*	55% *(+32%)*
b/tsDMARD-Experienced *(n=130 rosnilimab patients)*	*n=44*	*n=45*	*n=41*
	Week 28 *(Δ from Week 12)*		
CDAI ≤ 10 (LDA)	34% *(-9%)*	56% *(+5%)*	49% *(+20%)*
CDAI ≤ 2.8 (Remission)	14% *(+7%)*	16% *(+7%)*	15% *(+3%)*
ACR50	27% *(-12%)*	44% *(+15%)*	42% *(+3%)*
ACR70	23% *(+7%)*	36% *(+20%)*	29% *(+9%)*

NRI analysis on ITT population (n =318 total rosnilimab patients; 188 b/tsDMARD-naïve, 130 b/tsDMARD-experienced); Values rounded to the nearest whole number

CDAI LDA responders at Week 28 had durable responses for at least two months off drug, supporting the potential for maintenance dosing with extended dosing intervals (e.g. Q8W). As of the March 11, 2025 data cutoff date, 83% were still in LDA at Week 34 across all doses. Of the remaining 17% of patients who did not sustain CDAI LDA at Week 34, most remained near the cutoff of CDAI=10, with a median CDAI of 13.

Importantly, rosnilimab demonstrated clinically meaningful improvements that deepened through Week 28 across multiple validated patient-reported outcomes, including the pain visual analog scale (VAS) and the health assessment questionnaire, or HAQ-Disability Index (DI), a self-reporting tool to measure function and disability. The Pain VAS scale ranges from zero to 100, where scores of 20 indicate mild pain and above 60 indicate severe pain. The rosnilimab patients who entered the all-active treatment period improved on the Pain VAS from a mean baseline of ~65 to ~15, a ~50-point change where the minimal clinically important difference (MCID) is a ~10-point change from baseline. HAQ-DI scores range from zero to three, where scores of one to two indicate moderate to severe disability. The rosnilimab patients who entered the all-active treatment period with a score of ~1.6 reported a 0.9-point reduction in the HAQ-DI to ~0.7, where the MCID is a 0.22-point change from baseline.

Clinical outcomes were further substantiated by compelling and objective translational data. An ~50% reduction in the mean CRP from baseline, an objective measure of inflammation, was observed through Week 28 in rosnilimab patients who entered the all-active period. Additionally, translational blood and synovial biopsy biomarker data showed differentiated and consistent immunological impact with robust, on-target pharmacological activity in rosnilimab patients that was not observed on placebo. In blood, rosnilimab demonstrated rapid, deep and sustained reductions of ~90% in PD-1^{high} T cells and ~50% in PD-1+ T cells, and an increase in total Tregs. Together, this resulted in stable total T cell counts and favorable T cell composition reflective of healthy immune homeostasis. Additionally, synovial biopsies of the most impacted joint taken at baseline and after six weeks showed a deep reduction of ~90% in PD-1+ T cells at the 400mg Q4W and 600mg Q2W doses, showing a dose response relative to the 100mg Q4W dose. Gene expression studies of the synovium demonstrated significant decreases of T cell activation and B cell activation pathways in rosnilimab patients. Similarly, highly significant decreases in additional downstream pathways including those relevant to TNF and IL-6 within the synovium were observed.

“RA is a chronic disease that often begins in early adulthood, making it critical to effectively control disease activity over a patient's entire lifetime and prevent damage to joints and other organs, reduce pain and improve quality of life. Witnessing rosnilimab, with its novel mode of action, dramatically reduce RA disease activity through six months in most patients, whether having failed multiple classes of b/tsDMARD therapies or b/tsDMARD-naïve, is truly exciting for patients living with this disease and the field of RA treatment,” said Jonathan Graf, M.D., professor of Medicine, Division of Rheumatology at the University of California, San Francisco and RENOIR investigator. “Additionally, impressive translational data provide further evidence that by targeting specific PD-1 expressing cells, rosnilimab has a substantial impact downstream on multiple known pathways that drive RA pathogenesis, with the potential to restore immune homeostasis necessary to achieve meaningful, long-lasting disease remission. Developing innovative and safe treatment options with novel modes of action for long-term use is crucial to meet the urgent needs of today's patients with lifelong disease.”

Rosnilimab Was Well Tolerated with No Safety Signals

Consistent with prior studies, a favorable safety and tolerability profile across all rosnilimab doses was observed with no treatment-related serious adverse events (SAEs), malignancies, anaphylaxis or systemic hypersensitivity, and a low incidence of injection site reactions. Most adverse events (AEs) were mild to moderate in severity. Less than 2% of patients in the entire trial discontinued rosnilimab due to an AE, including only one patient after three months for a moderate headache treated with over-the-counter pain medication. Non-treatment related SAEs observed were consistent with known RA patient history and comorbidities.

"This was a robust and well-controlled Phase 2b study with more than 300 patients treated with rosnilimab for up to six months. To date, rosnilimab has shown a safe and well tolerated profile with almost all patients choosing to stay on therapy through the end of the study. Rosnilimab has not demonstrated any concerning safety trends or signals, such as those seen with the JAK inhibitors and most other biologics," said Paul Emery, M.D., Versus Arthritis professor of rheumatology at the University of Leeds and Leeds Biomedical Research Centre, UK. "This is remarkable, given these patients have a two-to-threefold increased risk of comorbidities such as infections, cardiac events and malignancies, before accounting for the impact of background DMARDs, mostly methotrexate."

The table below shows safety data for trial participants on an exposure adjusted incidence rate (per 100 patient years) from Week 0 through Week 28. Placebo data provided for 12 weeks of treatment, only.

Participants with Adverse Events	**Placebo** (n=106)	**Rosnilimab** 100mg Q4W (n=105)	**Rosnilimab** 400mg Q4W (n=107)	**Rosnilimab** 600mg Q2W (n=105)
Any AE, n (per 100 PY)	**39 (125.6)**	**73 (260.9)**	**66 (206.5)**	**52 (149.1)**
Any SAE[1]	1 (2.4)	2 (3.8)	2 (3.7)	4 (7.7)
Any Drug-Related SAE	1 (2.4)	0 (0)	0 (0)	0 (0)
Severe AE[2]	2 (4.8)	4 (7.5)	1 (1.9)	4 (7.8)
Drug-Related AE	18 (48.8)	17 (36.1)	28 (62.0)	19 (41.7)
AE Leading to Treatment Discontinuation	1 (2.4)	1 (1.9)	3 (5.6)	2 (3.8)
Infections	16 (41.5)	41 (98.7)	39 (89.4)	31 (67.6)
Serious	1 (2.4)	1 (1.9)	1 (1.9)	1 (1.9)
Opportunistic[3]	2 (4.8)	1 (1.9)	1 (1.8)	1 (1.9)
Patients with any AE > 5%, n (per 100 PY)				
Headache	4 (9.7)	10 (19.9)	10 (19.4)	5 (9.8)
Upper respiratory tract infection	2 (4.8)	14 (27.8)	7 (13.4)	10 (19.6)
Nasopharyngitis	4 (9.6)	9 (17.5)	8 (15.4)	1 (1.9)
Elevated ALT (alanine aminotransferase)[4]	1 (2.4)	8 (15.5)	5 (9.5)	4 (7.8)

1. SAEs (severe unless otherwise noted): RSV – moderate (600mg Q2W); anaphylaxis from wasp sting (600mg Q2W); ureter stone (600mg Q2W); cholecystitis / pericardial effusion (600mg Q2W); meniscus tear – moderate (400mg Q4W); diverticulitis – moderate (400mg Q4W); embolic ischemic stroke (100mg Q4W); pneumonia – mild (100mg Q4W); cellulitis/diarrhea (placebo)
2. Severe AEs (excluding SAEs): RA flare (600mg Q2W); blood creatine phosphokinase increase (400mg Q4W); endometriosis (100mg Q4W); alanine aminotransferase increased/aspartate aminotransferase increase (100mg Q4W); flu/headache (100mg Q4W); macular degeneration/retinal hemorrhage (placebo)
3. Values shown are for herpes zoster, the only opportunistic infection reported, and were all moderate
4. No patient met the predefined protocol liver function test stopping criteria. Only one ALT elevation was severe, which resolved without interruption of therapy, none were serious, all had an outcome of recovered/resolved or recovering/resolving

"At Anaptys, we are dedicated to developing novel treatments for patients living with inflammatory diseases. Beyond achieving necessary symptomatic improvements and reductions, we strive to advance treatment toward the clinical resolution of disease by restoring immune homeostasis. Today's updated positive data reinforce our targeted goals with the added potential for convenient monthly dosing through six months and beyond, on top of maintaining sustainable and durable outcomes," said Paul Lizzul, M.D., Ph.D., M.P.H., MBA, chief medical officer of Anaptys. "We extend our sincere gratitude to all the patients and investigators who participated in this trial and contributed to these clinically meaningful and significant findings that will help advance the field for all patients living with RA, as well as patients with other autoimmune or inflammatory diseases."

Anaptys Investor Call

Anaptys management will host an investor call and live webcast, with an accompanying slide presentation, to review results of the data from the Phase 2b RA trial, today, June 3, 2025, at 4:15pm ET/1:15pm PT. A live webcast of the call will be available on the Anaptys website at: https://ir.anaptysbio.com/presentations-and-events. A replay of the webcast will be available for at least 30 days following the event.

About the Phase P2b RENOIR Trial and the Primary and Secondary Endpoints

The Phase 2b RENOIR trial evaluated the efficacy, safety, tolerability, pharmacokinetics and pharmacodynamics of rosnilimab in patients with moderate-to-severe RA on background conventional disease-modifying antirheumatic drugs (cDMARDs) (e.g., methotrexate). The trial enrolled 424 patients across the U.S., Canada and Europe, who were either biologic or targeted synthetic DMARD (b/tsDMARD) naïve (n=250; 59%) or experienced (n=174; 41%). Patients classified as b/tsDMARD-experienced reported prior utilization of at least one biologic or targeted synthetic therapy, such as TNFα inhibitors, B cell inhibitors, selective costimulatory modulators or JAK inhibitors. Approximately 29% (n=50) of the b/tsDMARD-experienced patients were treated with prior JAK inhibitors.

Patients were randomized to receive either 100mg of subcutaneous rosnilimab Q4W, 400mg Q4W, 600mg Q2W, or placebo. The primary and secondary endpoints were assessed at Week 12. Following completion of the Week 14 visit, 220 of the 318 rosnilimab patients (71% of b/tsDMARD-naïve and 66% of b/tsDMARD-experienced) across all doses who achieved a high threshold of CDAI LDA continued with their assigned treatments through Week 28 in a blinded, all-active treatment period. At that time, participants moved into an off-drug observation period that assessed safety and efficacy for 10-12 weeks, or ~3 months, depending on their treatment assignment and completed the trial by Week 38 (Month 9).

The primary endpoint of mean change in DAS28-CRP at Week 12 is calculated based on differential weighting of individual measures, including the patient's general health, CRP and a count of 28 swollen and tender joints, with a score ranging from 0 to 9.4.

Secondary endpoints include the CDAI score, a composite assessment 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 defined as the threshold for LDA. CDAI remission score is ≤2.8. Additionally, ACR20/50/70 responses are used to measure change in RA disease activity. For example, an ACR70 response requires a patient to have a 70% reduction in the number of swollen and tender joints, and a reduction of 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 HAQ score.

Further details about the trial are available on: Trial Details | ClinicalTrials.gov.

About Rosnilimab

Rosnilimab is a novel therapeutic antibody that directly targets PD-1, a co-inhibitory receptor preferentially expressed on the surface of activated T cells, which broadly impacts the pathogenic drivers of inflammatory diseases such as RA and UC. Rosnilimab is a targeted therapy designed to deplete PD-1^{high} T cells and agonize the remaining PD-1+ T cells to restore the immune system back to a state of homeostasis. This is anticipated to result in specific immunological outcomes in both inflamed tissue and the periphery, such as reduction in T cell proliferation, migration and cytokine secretion, and reduction of plasma cell generation and autoantibody levels.

Rosnilimab is currently under clinical investigation, and its safety and efficacy have not been evaluated by any regulatory authority.

About Anaptys

Anaptys is a clinical-stage biotechnology company focused on delivering innovative immunology therapeutics for autoimmune and inflammatory diseases. Its lead program, rosnilimab, a depleter and agonist targeting PD-1+ T cells, is in a Phase 2b trial for the treatment of rheumatoid arthritis and in a Phase 2 trial for the treatment of ulcerative colitis. The company's pipeline also includes ANB033, a CD122 antagonist, and ANB101, a BDCA2 modulator, in Phase 1 trials. Anaptys has also discovered multiple therapeutic antibodies licensed to GSK in a financial collaboration for immuno-oncology, including a PD-1 antagonist (*Jemperli* (dostarlimab-gxly)) and a TIM-3 antagonist (cobolimab, GSK4069889). To learn more, visit www.AnaptysBio.com or follow us on LinkedIn.

Forward-Looking Statements

This press release 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 38 and Phase 2 clinical trial in ulcerative colitis; and whether current trends will be maintained once complete Week 38 data becomes available. 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 press release, and the company undertakes no obligation to revise or update any forward-looking statements to reflect events or circumstances after the date hereof.

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AnaptysBio
Rosnilimab:
Updated Phase 2b Data in RA
June 3, 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 38 and top-line data for rosnilimab's Phase 2 clinical trial in ulcerative colitis; whether current trends in rosnilimab's data in the rheumatoid arthritis Phase 2b clinical trial will be maintained once complete Week 38 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; whether rosnilimab 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 partner for rosnilimab 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.

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Participants on today's call

Dan Faga
CEO and president
Anaptys

Paul Lizzul, M.D., Ph.D.
CMO
Anaptys

Paul Emery, M.D.
Professor, Rheumatology, University of Leeds; Leeds Biomedical Research Centre, UK

Jonathan Graf, M.D.
Professor of Medicine, Rheumatology, University of California, San Francisco; RENOIR investigator

Previously reported highly differentiated RA data from RENIOR 424-patient placebo-controlled Phase 2b study

Recap:

n=318 rosnilimab patients

- Within 3 months, most had symptomatic improvement (>70% ACR20) with statistical significance at all doses vs. placebo
- Impressive achievement of CDAI LDA at 3 months with signal of stable outcomes through 6 months
- Robust translational and pharmacological activity supporting restoration of immune homeostasis
- Safe and well tolerated: similar AE rate versus placebo
- Monthly (Q4W) dose during initial “induction” period

Rosnilimab demonstrates best-in-disease profile in RA

1

Best-in-disease profile through 6 months

- JAK-like efficacy in both 3-month placebo-controlled portion and through 6 months
- Favorable safety and tolerability, particularly when compared to standard of care
- Monthly (Q4W) dosing

2

Max response rates have not yet been observed

- Strict continuation criteria prevented patients with improvement at 3 months from continuing in this P2b trial
- Many patients beyond 3 months achieved, or were trending toward, CDAI LDA and ACR50

3

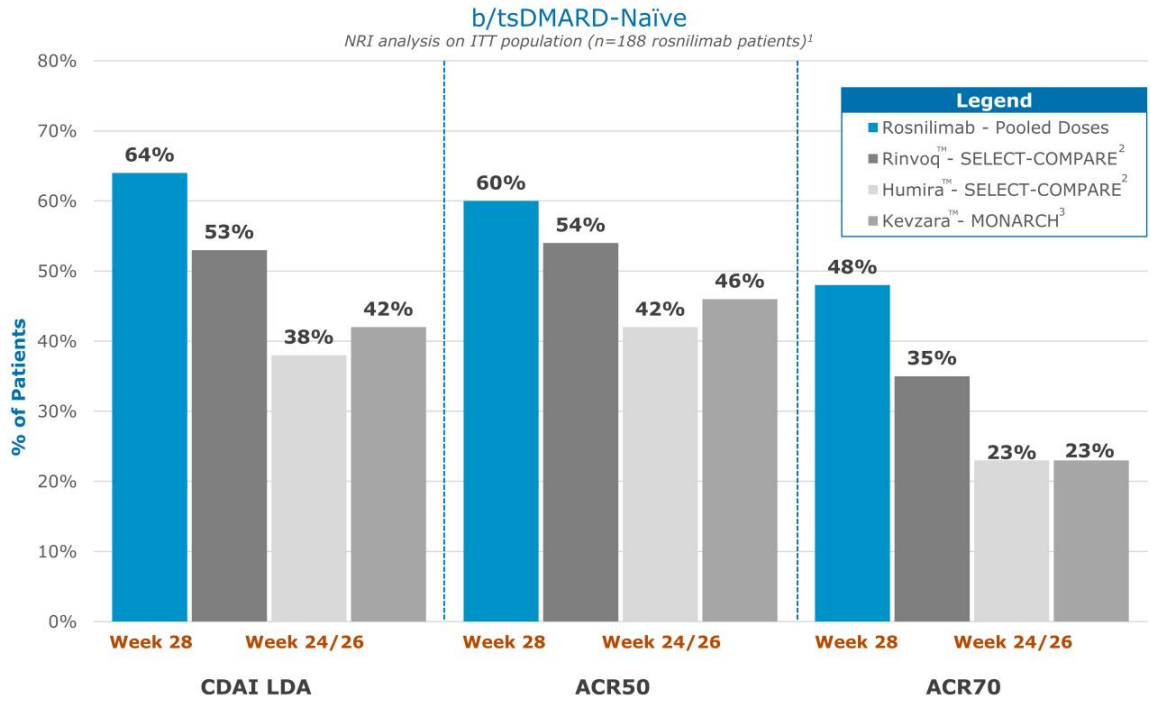
Responses durable after 6 months

- Potential for maintenance dosing with extended dosing intervals (e.g. Q8W)

Rosnilimab, a best-in-class depleter and agonist targeting PD-1+ T cells, is well-positioned for the ~$20 billion U.S. RA market which hasn't had a new mechanism approved since 2012

Rosnilimab shows JAK-like efficacy in naïve patients

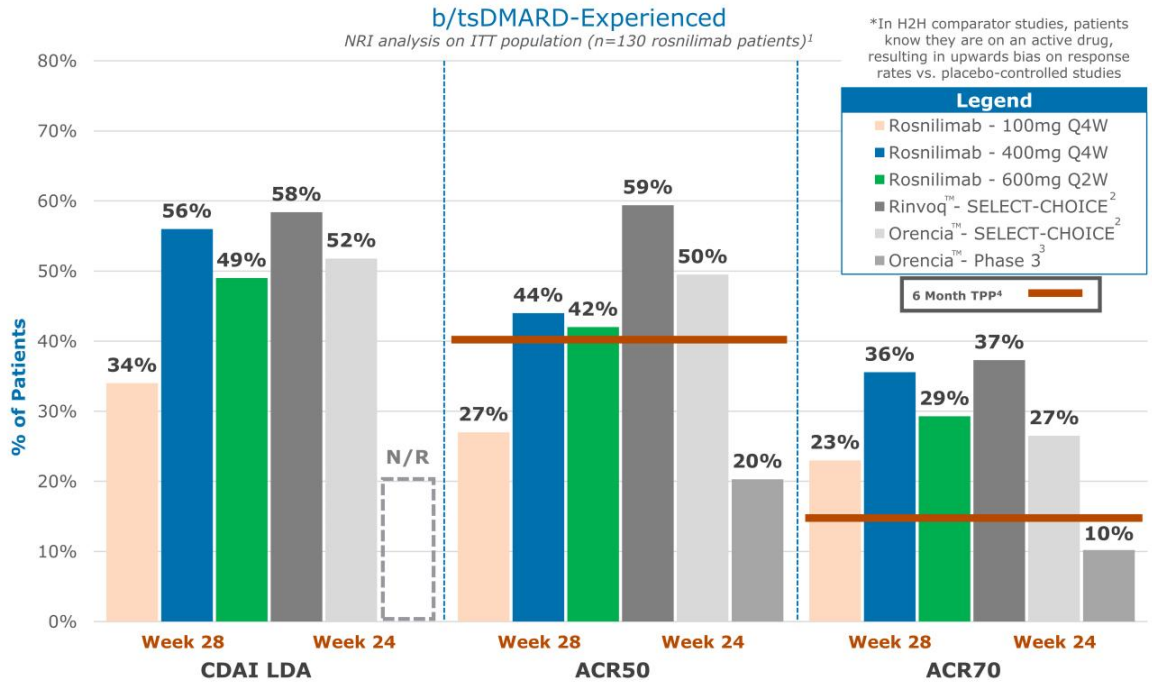
Compares favorably despite most conservative analysis and capped trial design



1. Non-responder imputated (NRI) analysis on intent-to-treat (ITT) of all b/tsDMARD-naïve patients randomized; b/tsDMARD-naïve population (n=62 100mg Q4W, n=62 400mg Q4W, n=64 600mg Q2W; n=188 total rosnilimab b/tsDMARD-naïve patients); 2. SELECT-COMPARE Phase 3 study; 3. Kevzara Phase 3 study; NRI data; CDAI = Clinical Diseases Activity Index; LDA = Low Disease Activity; N/R = Not Reported

Rosnilimab surpassed TPP in experienced patients and comparable at mid/high dose to JAKs in all-active H2H study*

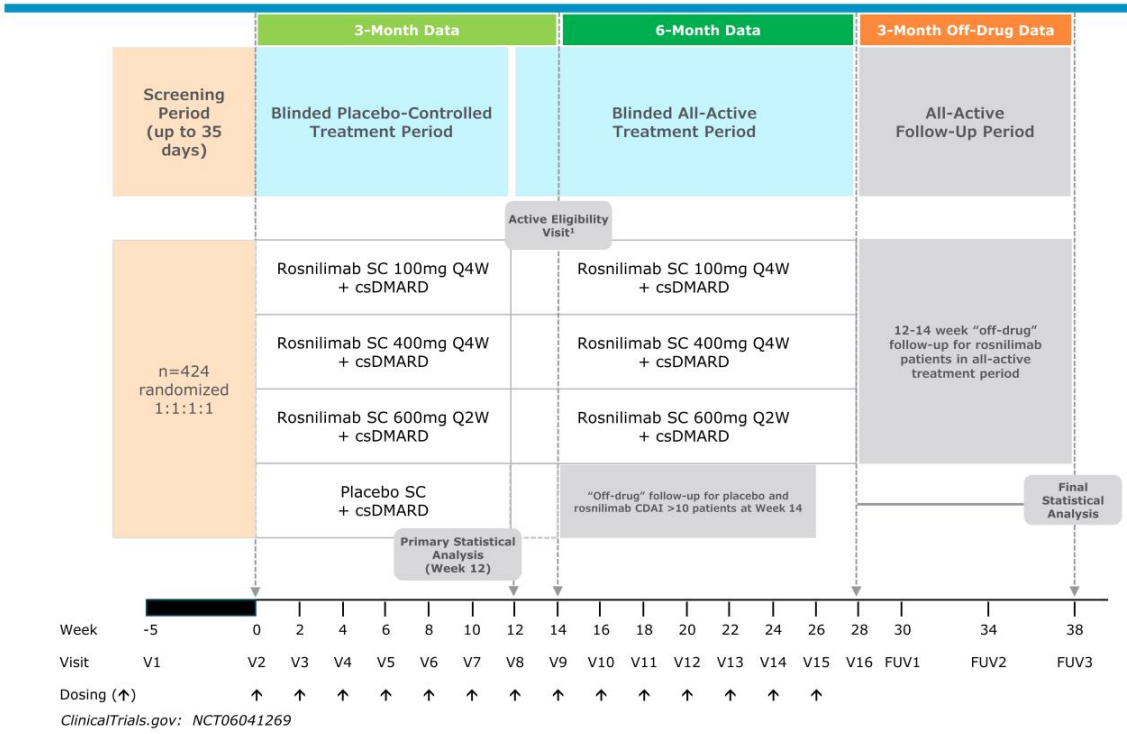
Includes 29% with prior JAK experience



1. Non-responder imputated (NRI) analysis on intent-to-treat (ITT) of all b/tsDMARD-experienced patients randomized; b/tsDMARD-experienced population (n=44 100mg Q4W, n=45 400mg Q4W, n=41 600mg Q2W; n=130 total rosnilimab b/tsDMARD-experienced patients); 2. SELECT-CHOICE Phase 3 study; 3. Orencia Phase 3 study; NRI data; 4. Anaptys Jan. 2025 Target Product Profile (TPP);

CDAI = Clinical Diseases Activity Index; LDA = Low Disease Activity; N/R = Not Reported

Rosnilimab Phase 2b trial



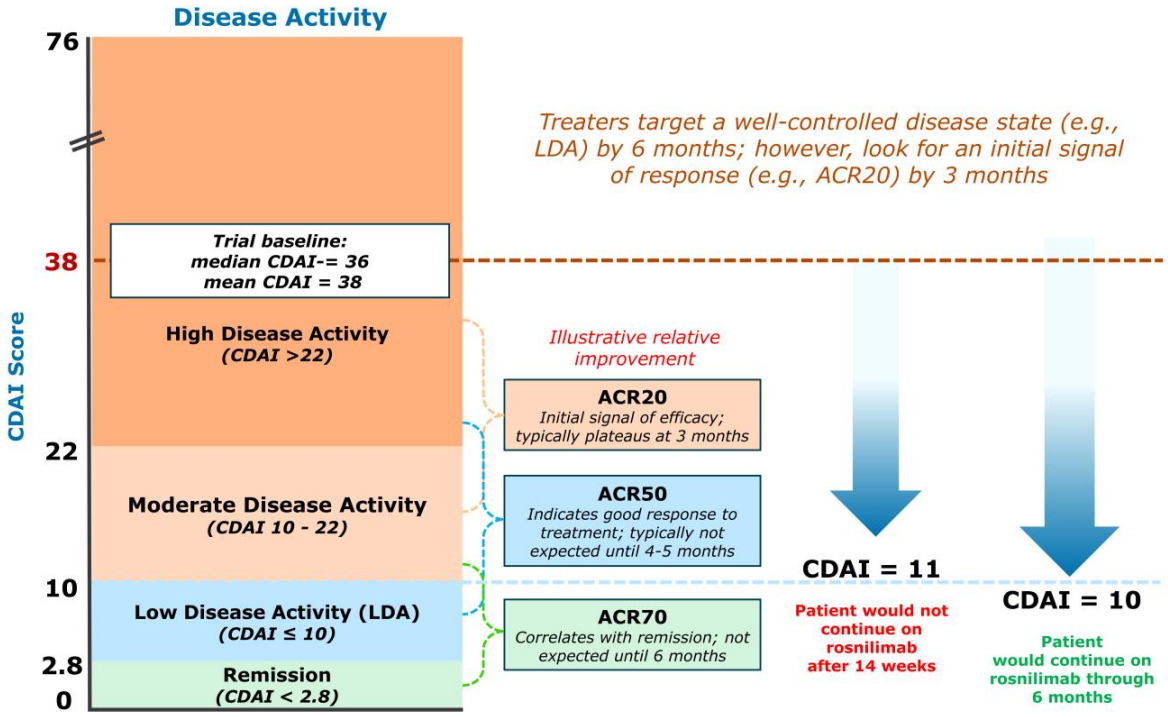
ClinicalTrials.gov: NCT06041269

Note: All patients in trial (rosnilimab and placebo arms) are required to be on stable background csDMARD

1. Blinded study drug treatment continued for active treatment group subjects that achieved Clinical Disease Activity Index (CDAI) low disease activity (≤10)

LDA requirement at 14 weeks to continue on rosnilimab was a high bar for patients with baseline high disease activity

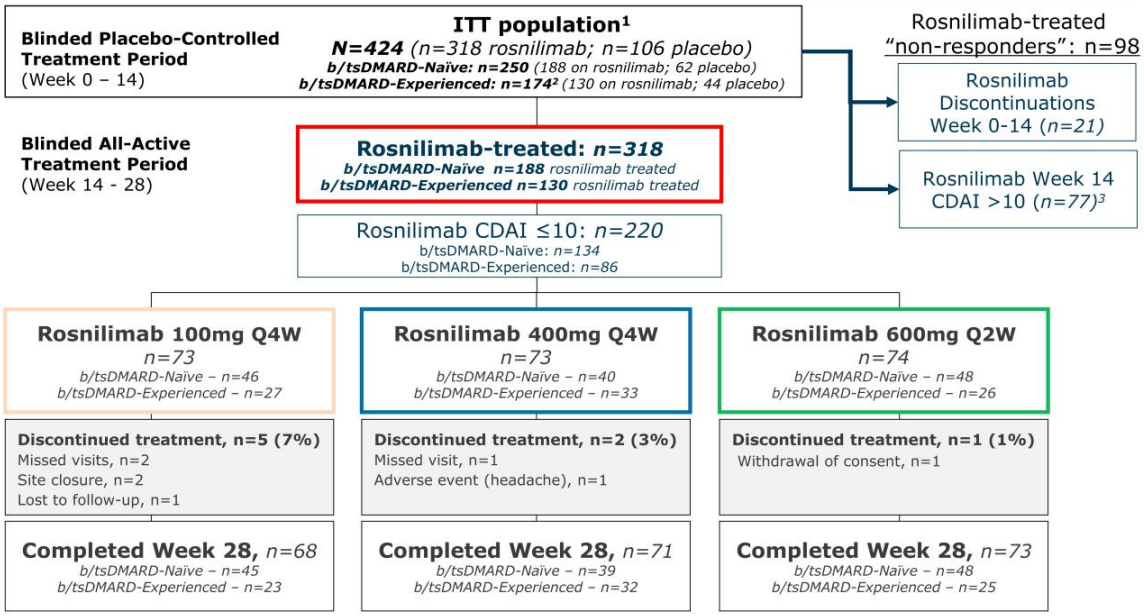
95% of trial participants had high disease activity (CDAI > 22) at baseline



95% completed 6-month all-active treatment period supporting rosnilimab's favorable efficacy and tolerability profile

Discontinuations
All-active treatment period (Week 14 - 28)

- ✓ 7 (of 8 total) discontinuations in all active treatment period were still in CDAI LDA at time of discontinuation
- ✓ No discontinuations due to disease progression
- ✓ Only 1 discontinuation due to AE (headache - moderate)



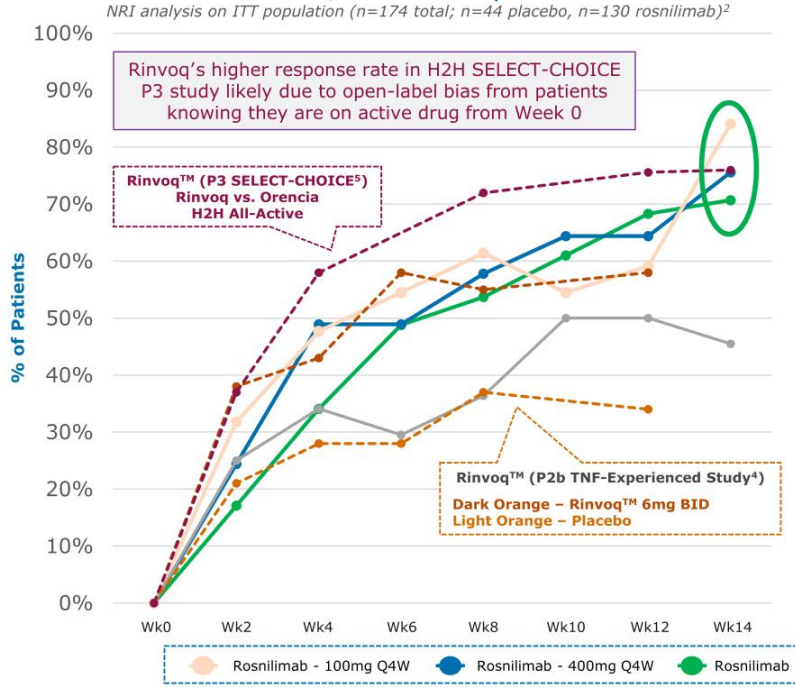
1. Non-responder imputated (NRI) analysis on intent-to-treat (ITT) population; b/tsDMARD-naïve population (n=62 placebo, n=62 100mg Q4W, n=62 400mg Q4W, n=64 600mg Q2W); b/tsDMARD-experienced population (n=44 placebo, n=44 100mg Q4W, n=45 400mg Q4W, n=41 600mg Q2W); 2. b/tsDMARD-experienced population included 50 patients (29% of n=174 total b/tsDMARD-experienced patients) with prior JAK experience; 3. Patients assessed at Week 14, dosed, and returned for follow-up visit at Week 18.

ACR20 response rates are comparable to Rinvoq™

Most patients had symptomatic and clinical improvement by 3 months

ACR20: b/tsDMARD-Experienced[1]

NRI analysis on ITT population (n=174 total; n=44 placebo, n=130 rosnilimab)[2]



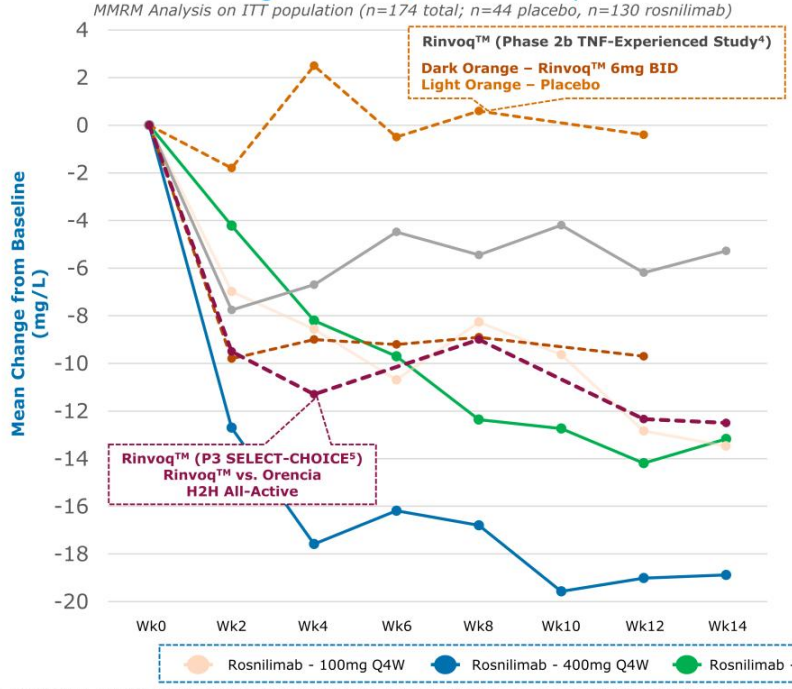
ACR20 at Week 12		
Arm	**Absolute**	**PBO Adjusted**
b/tsDMARD-Experienced Population *(as graphed)*		
100mg	59%	*9%*
400mg	64%	*14%*
600mg	68%	*18%*
Rinvoq[4]	58%	*24%*
Rinvoq[5]	76%	*N/A*
b/tsDMARD-Naïve Population *(for reference)*		
100mg	76%	*21%*
400mg	74%	*19%*
600mg	80%	*25%*
Rinvoq[3]	68%	*22%*

1. b/tsDMARD-experienced population included 29% (n=50 of n=174 total experienced patients) with prior JAK experience; 2. Non-responder imputated (NRI) analysis on intent-to-treat (ITT) of all b/tsDMARD-experienced patients randomized; b/tsDMARD-experienced population (n=44 placebo, n=44 100mg Q4W, n=45 400mg Q4W, n=41 600mg Q2W; n=130 total rosnilimab b/tsDMARD-experienced patients); 3. Rinvoq™ Phase 2b MTX-IR study; 4. Rinvoq™ Phase 2b TNF-experienced study; 6mg BID (equivalent to 15mg QD); 5. SELECT-CHOICE Phase 3 study

CRP reductions are comparable to Rinvoq™

Mean Change in CRP: b/tsDMARD-Experienced[1]

MMRM Analysis on ITT population (n=174 total; n=44 placebo, n=130 rosnilimab)



Change in CRP at Week 12		
Arm	**Baseline Mean CRP**	**PBO Adjusted**
b/tsDMARD-Experienced Population *(as graphed)*		
100mg	20.0	*-6.7*
400mg	29.4	*-12.8*
600mg	23.3	*-8.0*
Rinvoq[4]	16.0	*-9.3*
Rinvoq[5]	19.0	*N/A*
b/tsDMARD-Naïve Population *(for reference)*		
100mg	14.9	*-10.6*
400mg	14.3	*-7.0*
600mg	15.7	*-6.7*
Rinvoq[3]	17.0	*-8.4*

1. b/tsDMARD-experienced population included 29% (n=50 of n=174 total experienced patients) with prior JAK experience; 2. Mixed Model for Repeated Measures (MMRM) analysis on intent-to-treat (ITT) of all b/tsDMARD-experienced patients randomized; b/tsDMARD-experienced population (n=44 placebo, n=44 100mg Q4W, n=45 400mg Q4W, n=41 600mg Q2W); 3. Rinvoq™ Phase 2b MTX-IR study; 4. Rinvoq™ Phase 2b TNF-experienced study; 6mg BID (equivalent to 15mg QD) 5. SELECT-CHOICE Phase 3 study

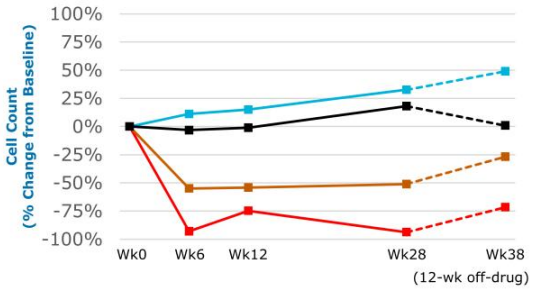
KOL Point of View

Paul Emery, M.D.
Professor, Rheumatology, University of Leeds; Leeds Biomedical Research Centre, UK

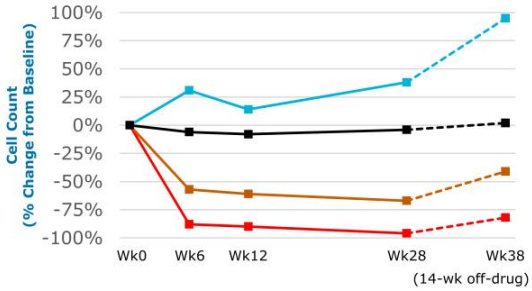
Jonathan Graf, M.D.
Professor of Medicine, Rheumatology, University of California, San Francisco; RENOIR investigator

Deep, sustained reduction of PD-1+ T cells led to favorable T cell composition reflective of immune homeostasis and durable response

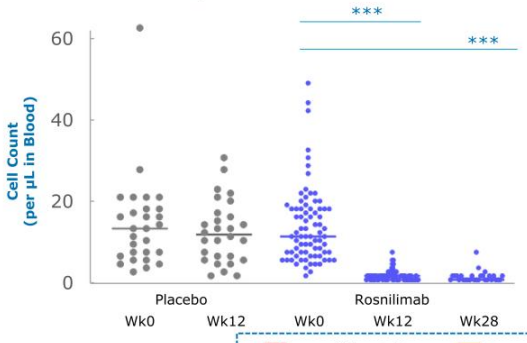
Rosnilimab 400mg Q4W T Cell Impact



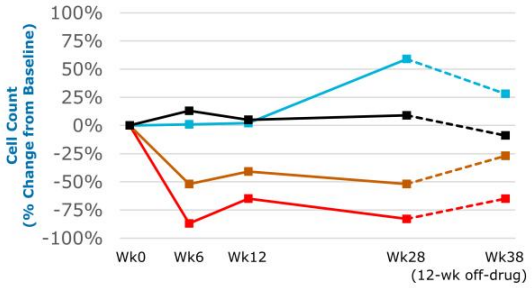
Rosnilimab 600mg Q2W T Cell Impact



Rosnilimab T_{ph} Impact – Pooled Doses

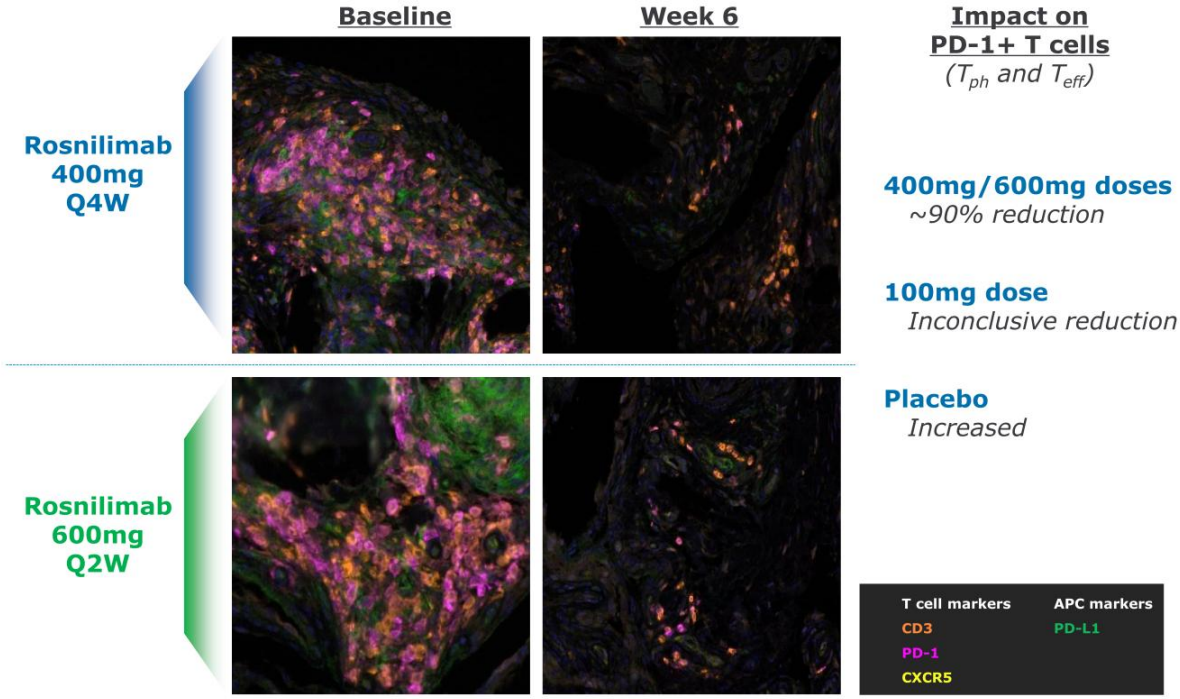


Rosnilimab 100mg Q4W T Cell Impact



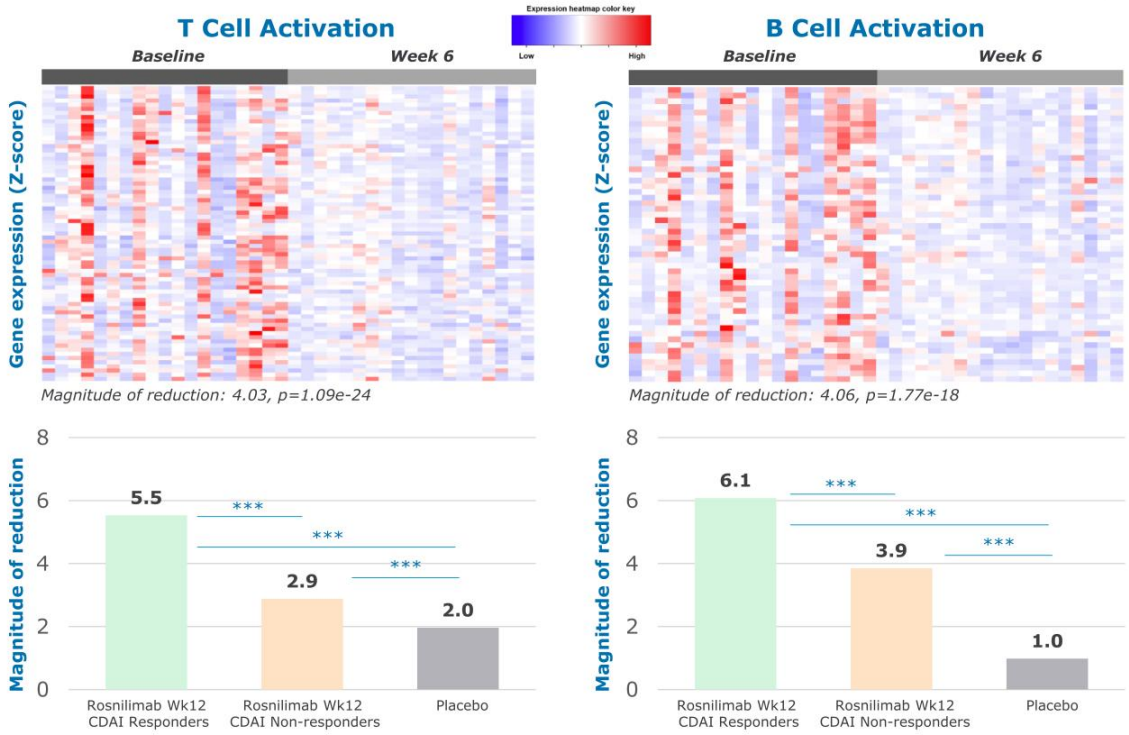
Note: data representative sample of ~50% of ITT population; T_{ph} – T peripheral helper cell defined as CD3+ CD4+ CD45RA- PD-1^{high} CXCR5-, ***p<0.001

Synovial biopsies show ~90% reduction of PD-1+ T cells in the target issue



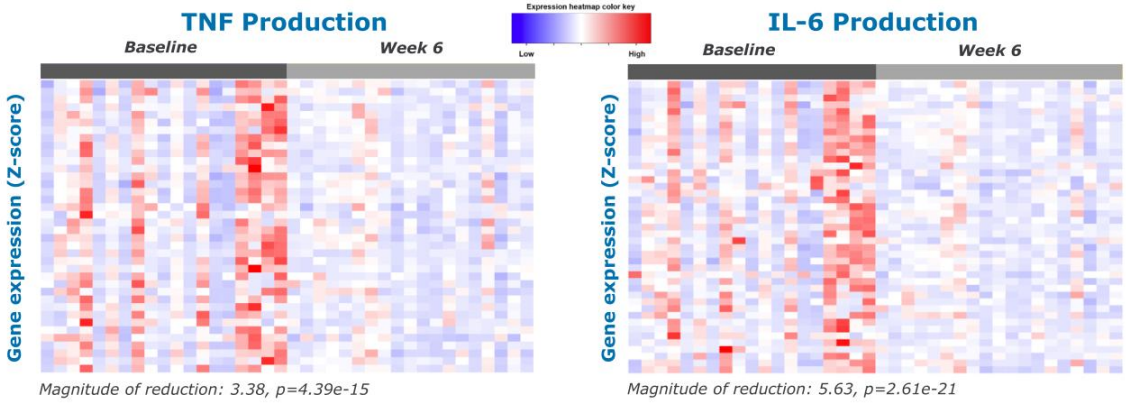
Note: Synovial biopsies of the most impacted joint taken at baseline and 6 weeks on study. Immunofluorescence performed to identify PD-1 positive cells. T_{ph} cells (PD-1+CD3+CD4+CXCR5-)

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



Note: Gene ontology (GO) pathway analysis performed on samples with evidence of inflammation at baseline (all rosnilimab doses pooled, n=19 paired biopsies) and with myosin normalization. Rows reflect genes with p<0.05 between Weeks 6 and 0. Magnitude of reduction defined as fold enrichment score. Rosnilimab responders achieved CDAI LDA in 3 months. ***p<0.001 for difference in fold change between baseline and Week 6 between groups.

Significant reduction of additional downstream pathways including TNF and IL-6 within the synovium



Magnitude of reduction: 3.38, p=4.39e-15

Magnitude of reduction: 5.63, p=2.61e-21

Pathway changes reflect rosnilimab's broad MOA

Significantly downregulated (p<0.05) genes of interest in RA:	
	T cell activation: IL2RA, TNFSF14 (LIGHT), CD28, CD69, CD40L, ICOS, CD226, ZAP70, TCF7, IRF1
	B cell activation: IL7R, CD27, CD79A, BTK, SYK, IL21R
	TNF and IL-6 production: MYD88, PTPN22, LILRB1, LILRB2, NOD2, CCR2, NLRC3, IRAK3, IL1RAP, IL6R, IL17RA
	Mediators of RA structural damage: MMP1, MMP3, and RANK-L
	IBD-related genes: NOD2, TREM1, IL12RB, IFNGR1, S100A8

Note: Gene ontology (GO) pathway analysis performed on samples with evidence of inflammation at baseline (all rosnilimab doses pooled, n=19 paired biopsies) and with myosin normalization. Rows reflect genes with p<0.05 between Weeks 6 and 0. Magnitude of reduction defined as fold enrichment score.

KOL Point of View

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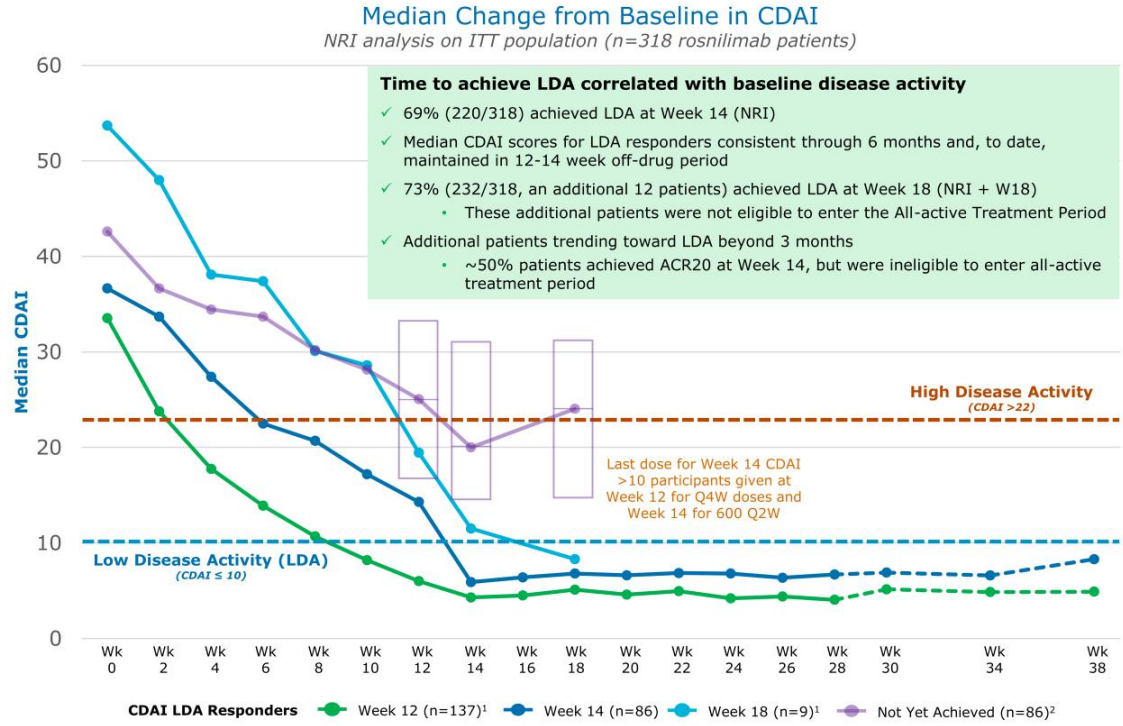
Professor of Medicine, Rheumatology, University of California, San Francisco; RENOIR investigator

Max response was not achieved in this Phase 2b trial

On average, patients with higher disease activity take longer to achieve CDAI LDA

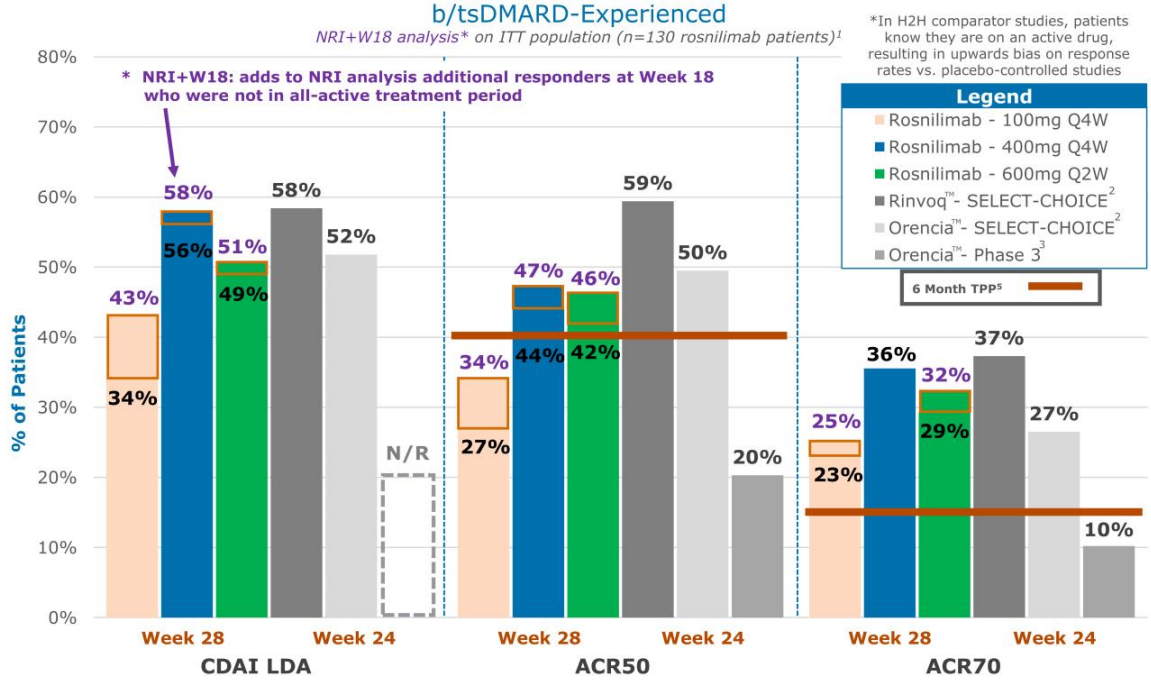


1. Green line includes 3 patients that achieved LDA at Week 12, were not CDAI LDA at Week 14, but returned to CDAI LDA at Week 18. These same 3 patients were excluded from the Light Blue line. In total 12 patients achieved CDAI LDA at Week 18. 2. Purple line includes rosnilimab patients that discontinued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

Rosnilimab surpassed TPP in experienced patients and comparable at mid/high dose to JAKs in all-active H2H study*

Includes 29% with prior JAK experience

Excludes 7 patients who discontinued in the all-active treatment period while in CDAI LDA



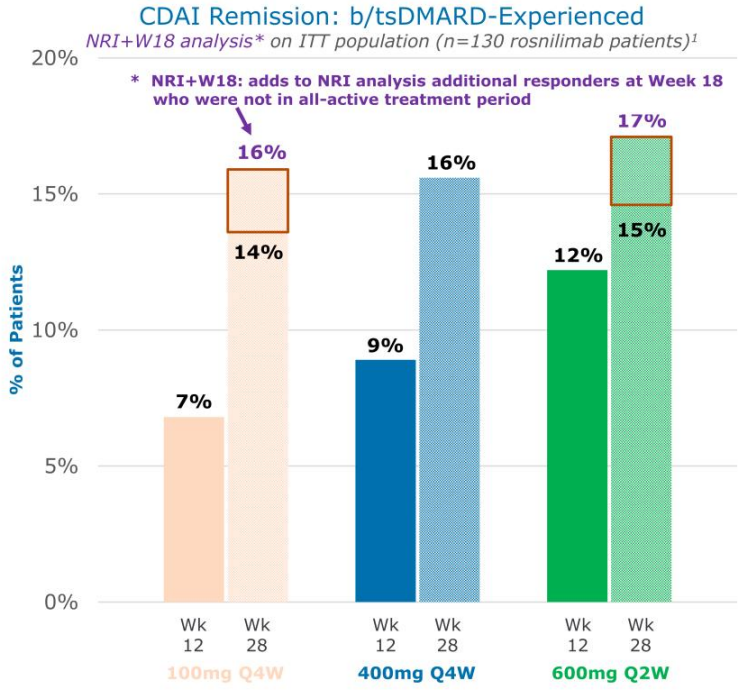
1. Non-responder imputated (NRI) analysis on intent-to-treat (ITT) of all b/tsDMARD-experienced patients randomized; b/tsDMARD-experienced population (n=44 100mg Q4W, n=45 400mg Q4W, n=41 600mg Q2W; n=130 total rosnilimab b/tsDMARD-experienced patients); 2. SELECT-CHOICE Phase 3 study; 3. Orencia Phase 3 study; NRI data; 4. Anaptys Jan. 2025 Target Product Profile (TPP);

CDAI = Clinical Diseases Activity Index; LDA = Low Disease Activity; N/R = Not Reported

JAK-like CDAI remission rates which deepened into six months

Includes 29% with prior JAK experience

Excludes 2 patients who discontinued in the all-active treatment period while in CDAI remission



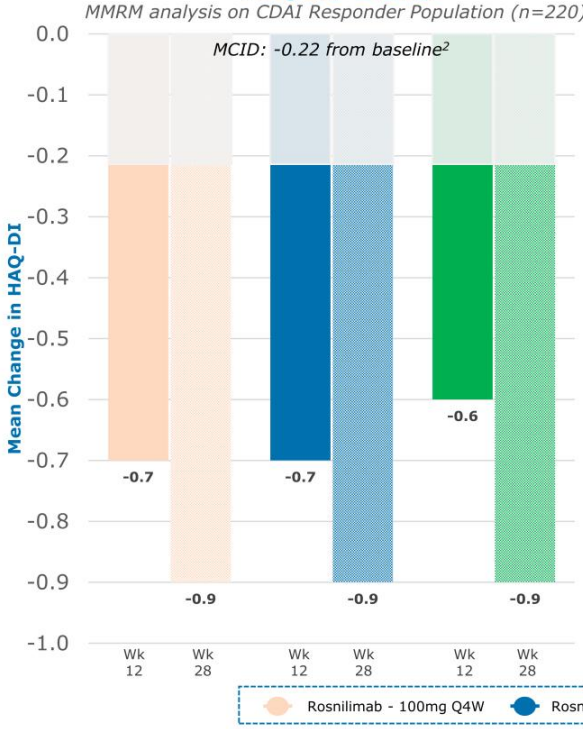
CDAI Remission at Week 28		
Arm	NRI	NRI+W18
b/tsDMARD-Experienced Population *(as graphed)*		
100mg	14%	16%
400mg	16%	16%
600mg	15%	17%
b/tsDMARD-Naïve Population		
100mg	21%	21%
400mg	18%	18%
600mg	17%	19%

1. Non-responder imputated (NRI) analysis on intent-to-treat (ITT) of all b/tsDMARD-experienced patients randomized; b/tsDMARD-experienced population (n=44 100mg Q4W, n=45 400mg Q4W, n=41 600mg Q2W; n=130 total rosnilimab b/tsDMARD-experienced patients)

Highly meaningful clinically and symptomatic improvement across multiple PROs and CRP

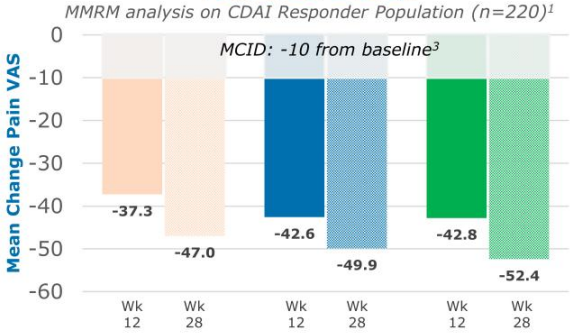
HAQ-DI: Overall

MMRM analysis on CDAI Responder Population (n=220)[1]



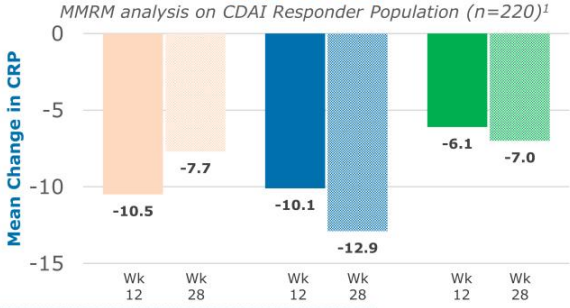
Pain VAS: Overall

MMRM analysis on CDAI Responder Population (n=220)[1]



CRP: Overall

MMRM analysis on CDAI Responder Population (n=220)[1]

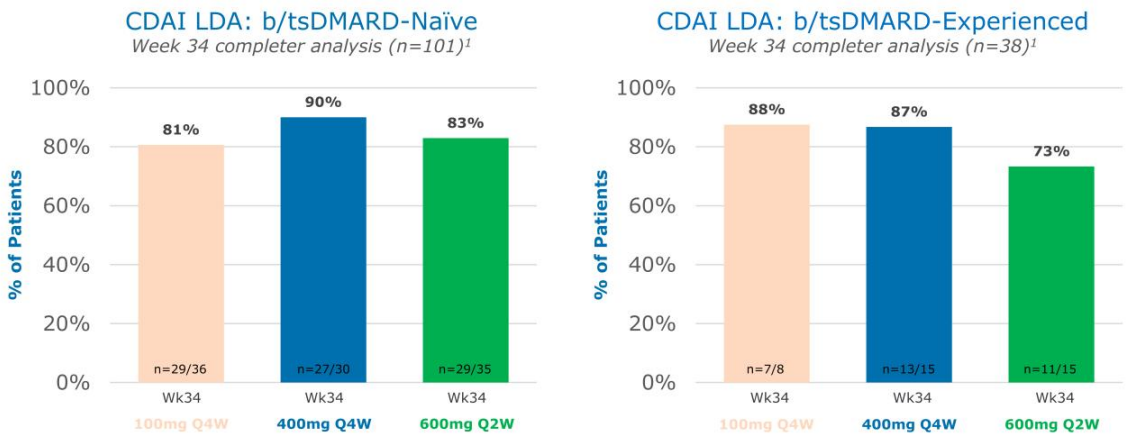


Rosnilimab - 100mg Q4W Rosnilimab - 400mg Q4W Rosnilimab - 600mg Q2W

1. Mixed Model for Repeated Measures (MMRM) analysis on rosnilimab CDAI LDA responder at Week 14 population (n=220) includes naïve population (n=46 100mg Q4W, n=40 400mg Q4W, n=48 600mg Q2W; n=134 total rosnilimab patients) and experienced population (n=27 100mg Q4W, n=33 400mg Q4W, n=26 600mg Q2W; n=86 total rosnilimab patients); 2. Behrens et. al, BMC Rheumatology, Dec. 2019; 3. Strand et. al, Journal of Rheumatology, Aug. 2011

Durable responses for at least 2-months off-drug

83% of Week 28 CDAI LDA responders were still in response at Week 34



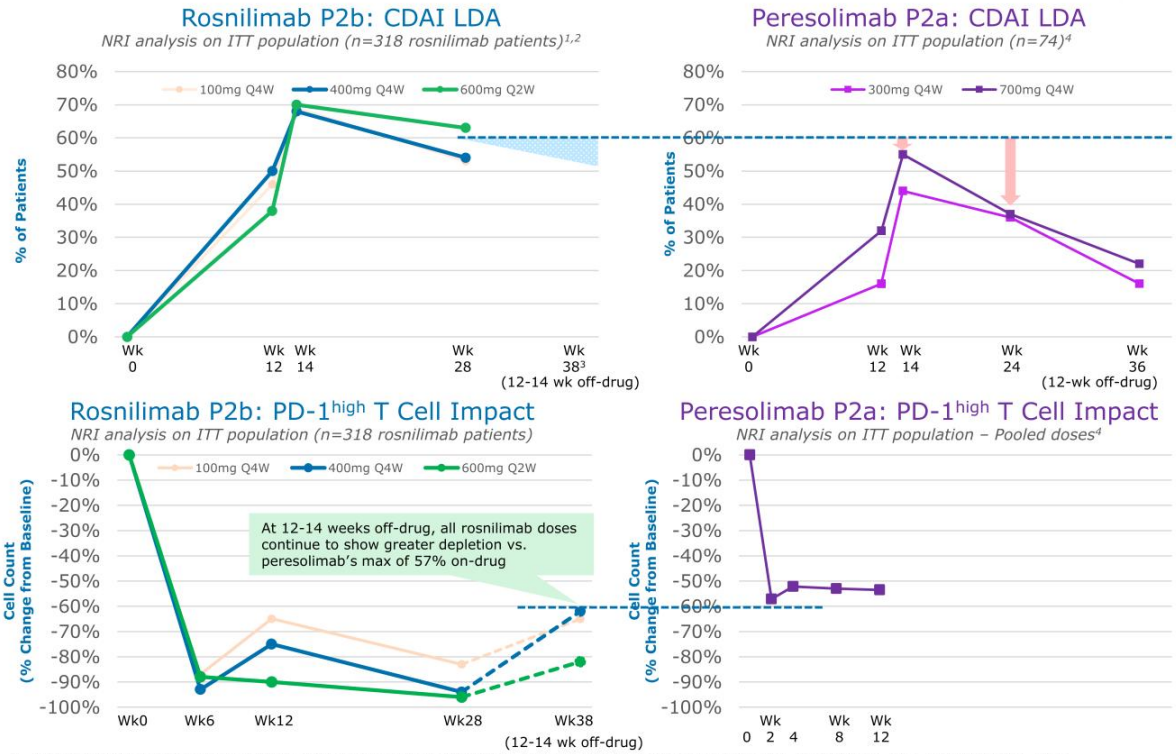
Most patients who did not sustain CDAI LDA remained near the cutoff of CDAI=10

Only 23 of 139 (17%) patients of Week 28 CDAI LDA responders were not CDAI LDA (≤10) at Week 34:

- 25% (6/23) were CDAI <11
- Median CDAI = 13
- 91% (21/23) were CDAI <22 (e.g. remained CDAI moderate disease activity)

1. ~75% of patients who completed Week 28 (n=212) had reached Week 34 as of the March 11, 2025 data cutoff; this responder analysis represents patients who were in CDAI LDA, as of Week 28, relative to their CDAI status as of Week 34

LDA response rates and durability for rosnilimab are differentiated from Lilly's peresolimab



1. Non-responder imputated (NRI) analysis on intent-to-treat (ITT) of all 318 rosnilimab patients randomized; 2. At Week 28, 53% (100mg Q4W), 54% (400mg Q4W), and 63% (600mg Q2W) rosnilimab patients were in CDAI LDA (57% pooled); 3. Off-drug follow-up period ongoing; 4. Tuttle et. al, NEJM, May 2023, Supplemental Appendix, At Week 28, 36% (300mg Q4W) and 37% (700mg Q4W) peresolimab patients were in CDAI LDA

KOL Point of View

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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 well tolerated with no safety signals

<2% dropout rate overall due to AEs through 6 months,
with only 1 dropout due to AE (headache-moderate) after 3 months

Study Period	Week 0 through Week 12 (N=424)				Week 0 through Week 28 (N=424)			
	Participants with Adverse Events, n (%)				Participants with Adverse Events, n (per 100 PY)*			
	Placebo (n=106)	100mg Q4W (n=106)	400mg Q4W (n=107)	600mg Q2W (n=105)	Placebo (n=106)	100mg Q4W (n=106)	400mg Q4W (n=107)	600mg Q2W (n=105)
Any AE	**36 (34%)**	**51 (48%)**	**48 (45%)**	**38 (36%)**	**39 (125.6)**	**73 (260.9)**	**66 (206.5)**	**52 (149.1)**
Any SAE[1]	1 (1%)	1 (1%)	1 (1%)	3 (3%)	1 (2.4)	2 (3.8)	2 (3.7)	4 (7.7)
Any Drug-Related SAE	1 (1%)	0 (0%)	0 (0%)	0 (0%)	1 (2.4)	0 (0)	0 (0)	0 (0)
Severe AE[2]	2 (2%)	1 (1%)	0 (0%)	4 (4%)	2 (4.8)	4 (7.5)	1 (1.9)	4 (7.8)
Drug-Related AE	18 (17%)	13 (12%)	18 (17%)	17 (16%)	18 (48.8)	17 (36.1)	28 (62.0)	19 (41.7)
AE Leading to Treatment Discontinuation	1 (1%)	1 (1%)	2 (2%)	2 (2%)	1 (2.4)	1 (1.9)	3 (5.6)	2 (3.8)
Infections	14 (13%)	24 (23%)	21 (20%)	12 (11%)	16 (41.5)	41 (98.7)	39 (89.4)	31 (67.6)
Serious	1 (1%)	1 (1%)	0	0	1 (2.4)	1 (1.9)	1 (1.9)	1 (1.9)
Opportunistic[3]	2 (1.9%)	0 (0%)	0 (0%)	0 (0%)	2 (4.8)	1 (1.9)	1 (1.8)	1 (1.9)
Participants with any AEs > 5%								
Headache	4 (4%)	7 (7%)	6 (6%)	4 (4%)	4 (9.7)	10 (19.9)	10 (19.4)	5 (9.8)
Upper respiratory tract infection	1 (1%)	7 (7%)	2 (2%)	3 (3%)	2 (4.8)	14 (27.8)	7 (13.4)	10 (19.6)
Nasopharyngitis	4 (4%)	5 (5%)	5 (5%)	0	4 (9.6)	9 (17.5)	8 (15.4)	1 (1.9)
Elevated ALT (alanine aminotransferase)[4]	1 (1%)	4 (4%)	3 (3%)	3 (3%)	1 (2.4)	8 (15.5)	5 (9.5)	4 (7.8)

** Measured as an exposure adjusted incidence rate (per 100 patient years)*

1. SAEs (severe unless otherwise noted): RSV – moderate (600mg Q2W); anaphylaxis from wasp sting (600mg Q2W); ureter stone (600mg Q2W); cholecystitis / pericardial effusion (600mg Q2W); meniscus tear – moderate (400mg Q4W); diverticulitis – moderate (400mg Q4W); embolic ischemic stroke (100mg Q4W); pneumonia – mild (100mg Q4W); cellulitis/diarrhea (placebo)

2. Severe AEs (excluding SAEs): RA flare (600mg Q2W); blood creatine phosphokinase increase (400mg Q4W); endometriosis (100mg Q4W); alanine aminotransferase increased/aspartate aminotransferase increase (100mg Q4W); flu/headache (100mg Q4W); macular degeneration/retinal hemorrhage (placebo)

3. Values shown are for herpes zoster, none were severe and are the only opportunistic infection reported.

4. No patient met the predefined protocol liver function test stopping criteria. Only one ALT elevation was severe, which resolved without interruption of therapy, none were serious, all had an outcome of recovered/resolved or recovering/resolving

KOL Point of View

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Professor of Medicine, Rheumatology, University of California, San Francisco; RENOIR investigator

Next steps for rosnilimab

- **Plan to present RA Phase 2b data at a future medical congress**

- **Assessing two, alternative, strategic paths forward**
  - Secure a global partnership, to help advance in all indications, including P3 for both RA and UC
  - Independently advance UC into P3 (assuming P2 data meets TPP)

- **2026+ activities**
  - P3 enablement: drug supply scale-up and regulatory interactions
  - Initiate P2 studies in additional indications

Best-in-class immune cell modulating antibodies

Immune Cell Modulators

Rosnilimab (PD-1 depleter and agonist)	ANB033 (CD122 antagonist)	ANB101 (BDCA2 modulator)
P2b in Rheumatoid Arthritis P2 in Ulcerative Colitis	P1 in Healthy Volunteers	P1 in Healthy Volunteers

Autoimmune and inflammatory diseases including dermatology, gastroenterology and rheumatology

Research and Capital

Research-driven
- Preclinical pipeline of immunology targets

Strong capital position
- Q1 2025 cash: ~$383MM
  - Expected cash runway: YE 2027

Royalty income
- Excludes significant royalty potential:
  - GSK royalty and milestone potential for *Jemperli* and cobolimab
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Rosnilimab demonstrates best-in-disease profile in RA

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Best-in-disease profile through 6 months

- JAK-like efficacy in both 3-month placebo-controlled portion and through 6 months
- Favorable safety and tolerability, particularly when compared to standard of care
- Monthly (Q4W) dosing

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- Strict continuation criteria prevented patients with improvement at 3 months from continuing in this P2b trial
- Many patients beyond 3 months achieved, or were trending toward, CDAI LDA and ACR50

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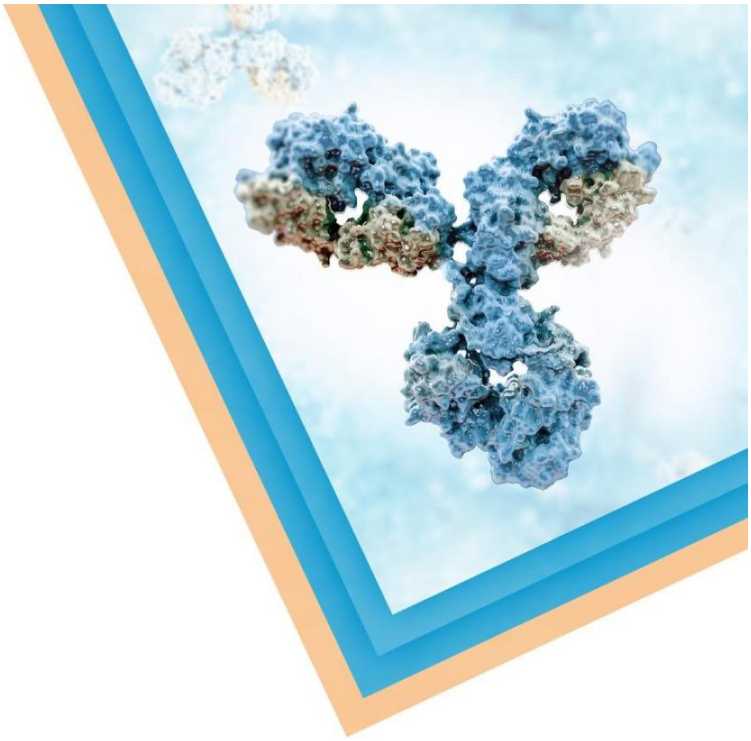
Responses durable after 6 months

- Potential for maintenance dosing with extended dosing intervals (e.g. Q8W)

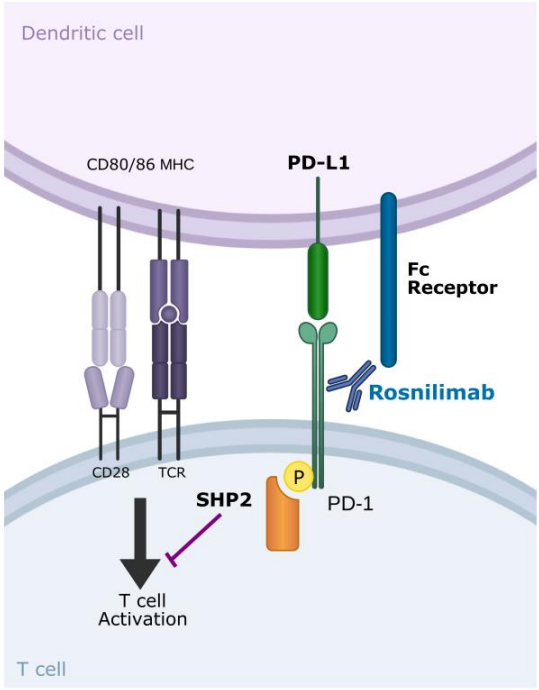
Rosnilimab, a best-in-class depleter and agonist targeting PD-1+ T cells, is well-positioned for the ~$20 billion U.S. RA market which hasn't had a new mechanism approved since 2012

Appendix



AnaptysBio

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

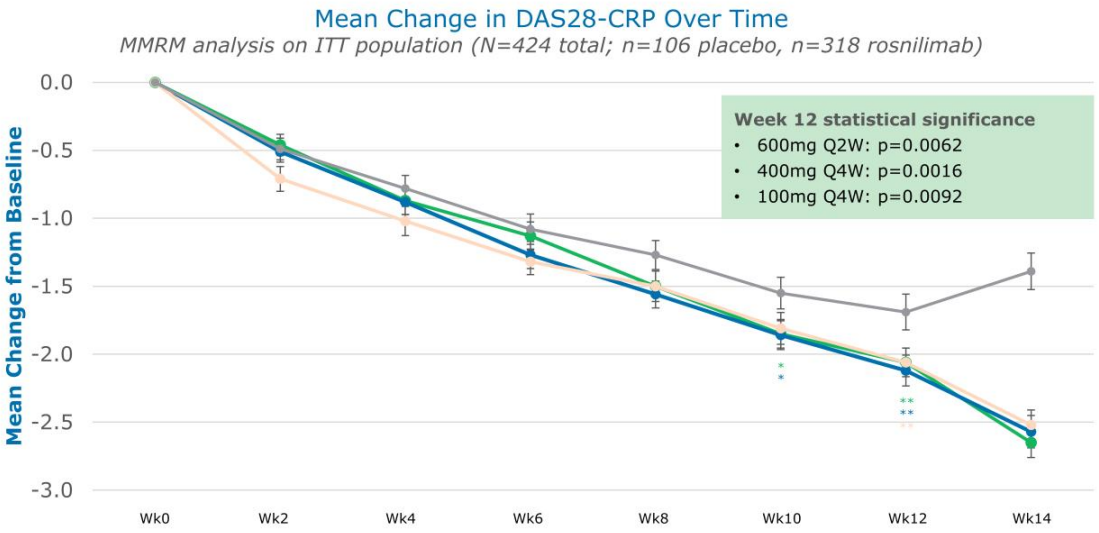
Effector T cells (T_{eff}): activated T cells (cytotoxic, helper, Treg); Follicular/Peripheral Helper T cells (T_{fh}, T_{ph}): support B cell differentiation and maturation.

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

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

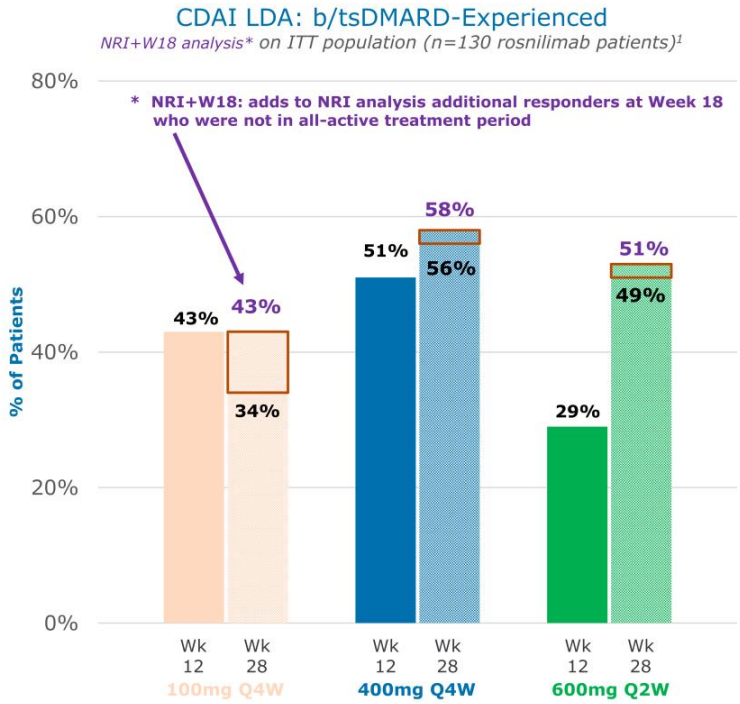


- All rosnilimab doses statistically significant at Week 12
- All rosnilimab doses continue to improve into Week 14 with no evidence of flattening
- Following Week 14 visit, placebo patients proceeded to post treatment follow-up

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

1. Mixed Model for Repeated Measures (MMRM) analysis on intent-to-treat (ITT) population; b/tsDMARD-naïve population (n=62 placebo, n=62 100mg Q4W, n=62 400mg Q4W, n=64 600mg Q2W); b/tsDMARD-experienced population (n=44 placebo, n=44 100mg Q4W, n=45 400mg Q4W, n=41 600mg Q2W); 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<0.01, *p<0.05, Standard error (SE) used to present figures of least squares mean changes from baseline.

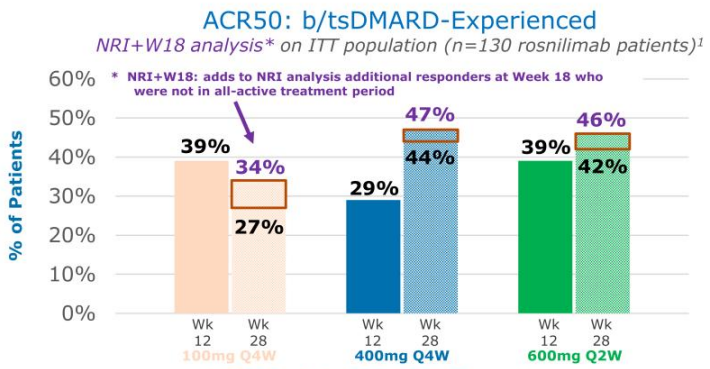
Demonstrated JAK-like CDAI LDA rates by 6 months

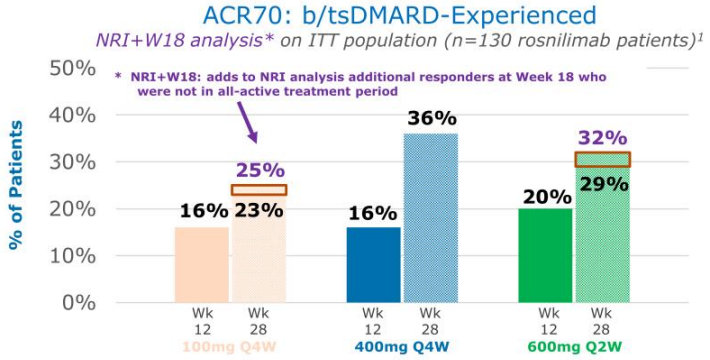


CDAI LDA at Week 28		
Arm	NRI	NRI+W18
b/tsDMARD-Experienced Population *(as graphed)*		
100mg	34%	43%
400mg	56%	58%
600mg	49%	51%
b/tsDMARD-Naïve Population		
100mg	66%	71%
400mg	53%	55%
600mg	72%	75%

1. Non-responder imputated (NRI) analysis on intent-to-treat (ITT) of all b/tsDMARD-naïve patients randomized; b/tsDMARD-experienced population (n=44 placebo, n=44 100mg Q4W, n=45 400mg Q4W, n=41 600mg Q2W; n=130 total rosnilimab b/tsDMARD-experienced patients)

Demonstrated JAK-like ACR70 rates which deepened into 6 months





ACR50 at Week 28		
Arm	NRI	NRI+W18
b/tsDMARD-Experienced Population *(as graphed)*		
100mg	27%	34%
400mg	44%	47%
600mg	42%	46%
b/tsDMARD-Naïve Population		
100mg	58%	61%
400mg	52%	53%
600mg	69%	75%

ACR70 at Week 28		
Arm	NRI	NRI+W18
b/tsDMARD-Experienced Population *(as graphed)*		
100mg	23%	25%
400mg	36%	36%
600mg	29%	32%
b/tsDMARD-Naïve Population		
100mg	53%	55%
400mg	37%	37%
600mg	55%	58%

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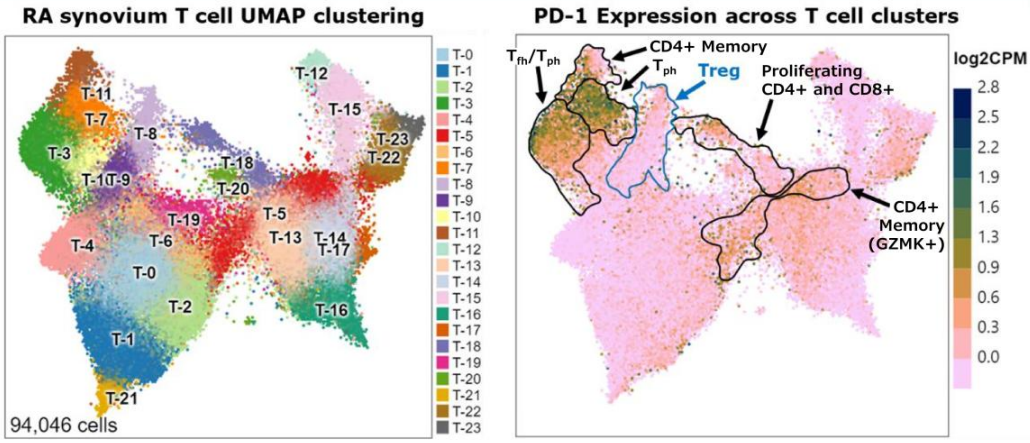

Very low % Tregs (<20%) are PD-1+ in RA synovium, even fewer are PD-1^{high1}

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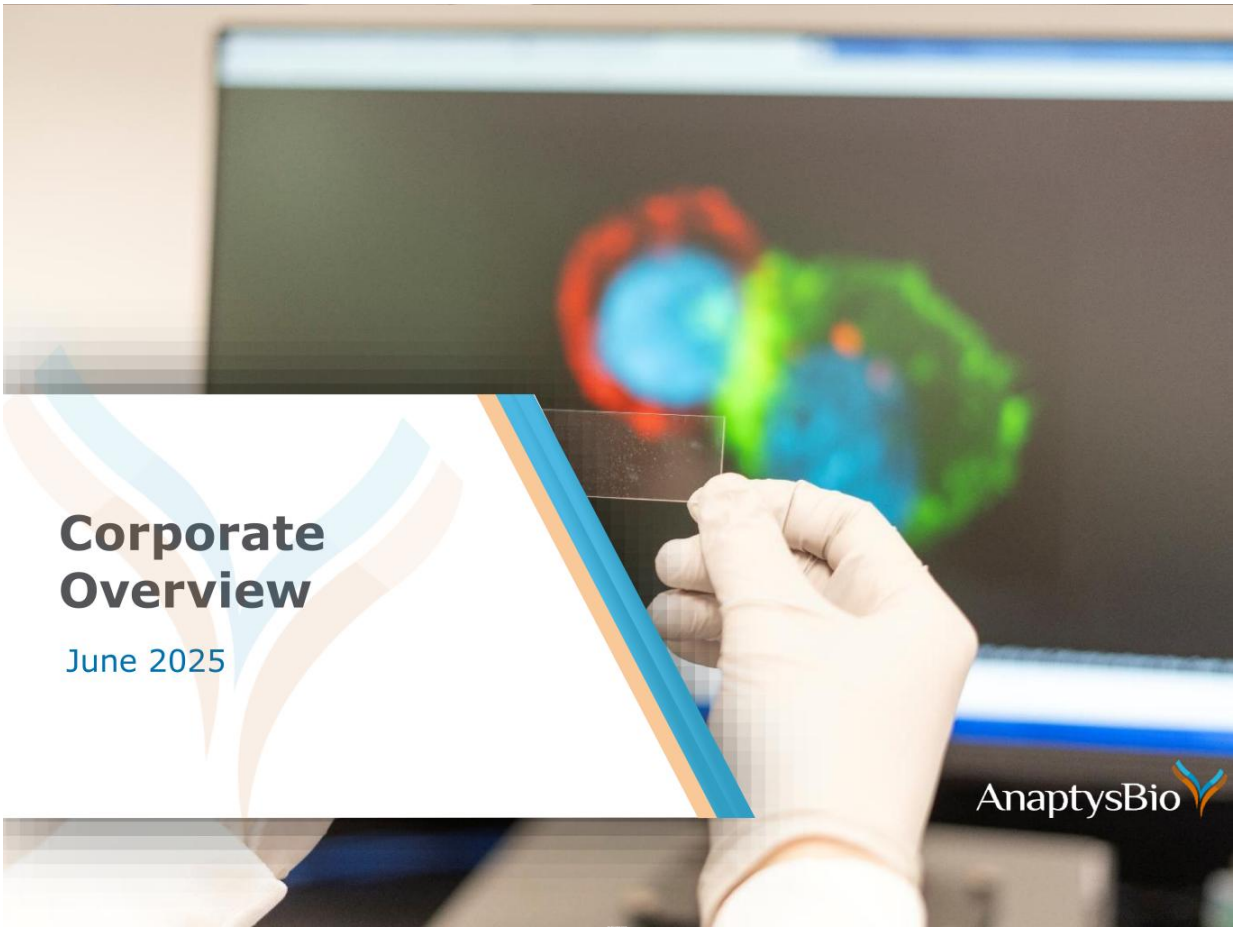
1. Uniform manifold approximation and projection (UMAP) clusters of T cells from RA patient synovium with arrows identifying T_{ph} and T_{fh}/T_{ph} cells, T-7 and T-3, respectively and feature plot of PD-1 expression across T cell subtypes; Ren et. al. ACR 2024. November 2024.


Corporate
Overview
June 2025
AnaptysBio

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This presentation and any accompanying oral presentation contains forward-looking statements within the meaning of the "safe harbor" provisions of the Private Securities Litigation Reform Act of 1995, including, but not limited to: the timing of the release of data from the Company's clinical trials, including rosnilimab's Phase 2b clinical trial in rheumatoid arthritis at Week 38 and initial data for rosnilimab's Phase 2 clinical trial in ulcerative colitis; whether current trends in rosnilimab's data in the rheumatoid arthritis Phase 2b clinical trial will be maintained once complete Week 38 data become 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; 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.

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Best-in-class immune cell modulating antibodies

Immune Cell Modulators

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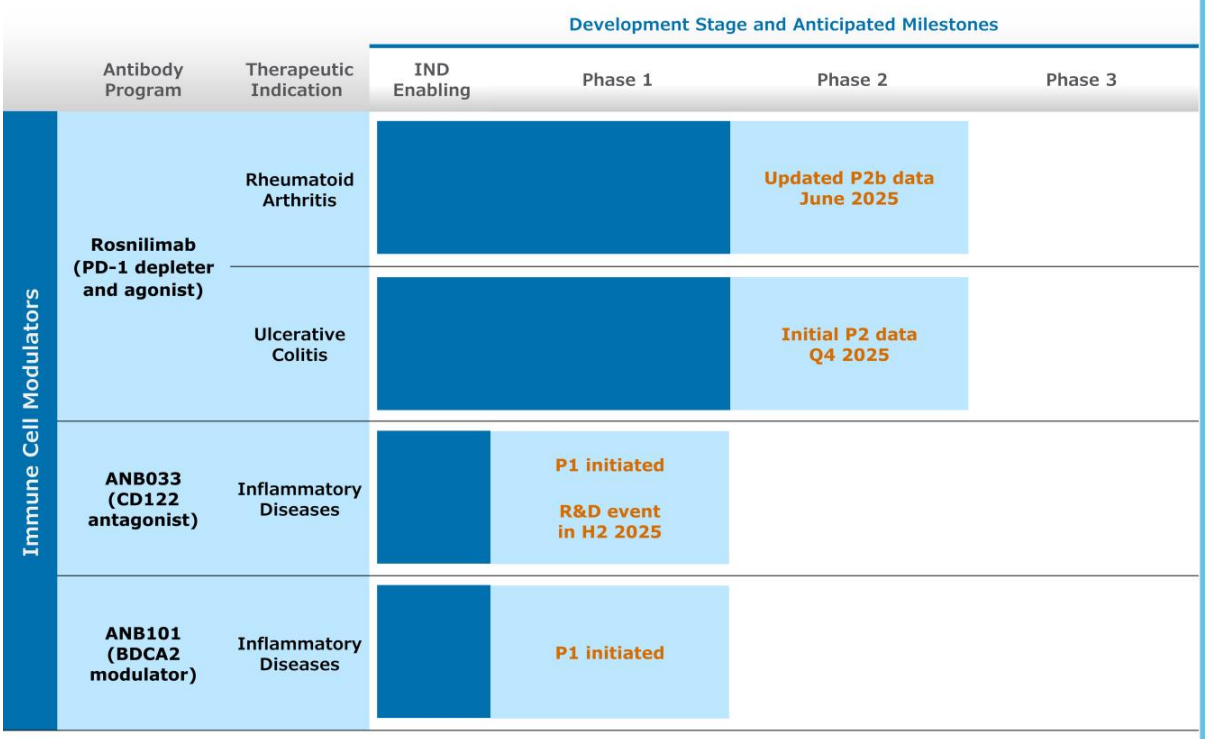
Leading pipeline of immune cell modulating antibodies with multiple 2025 catalysts



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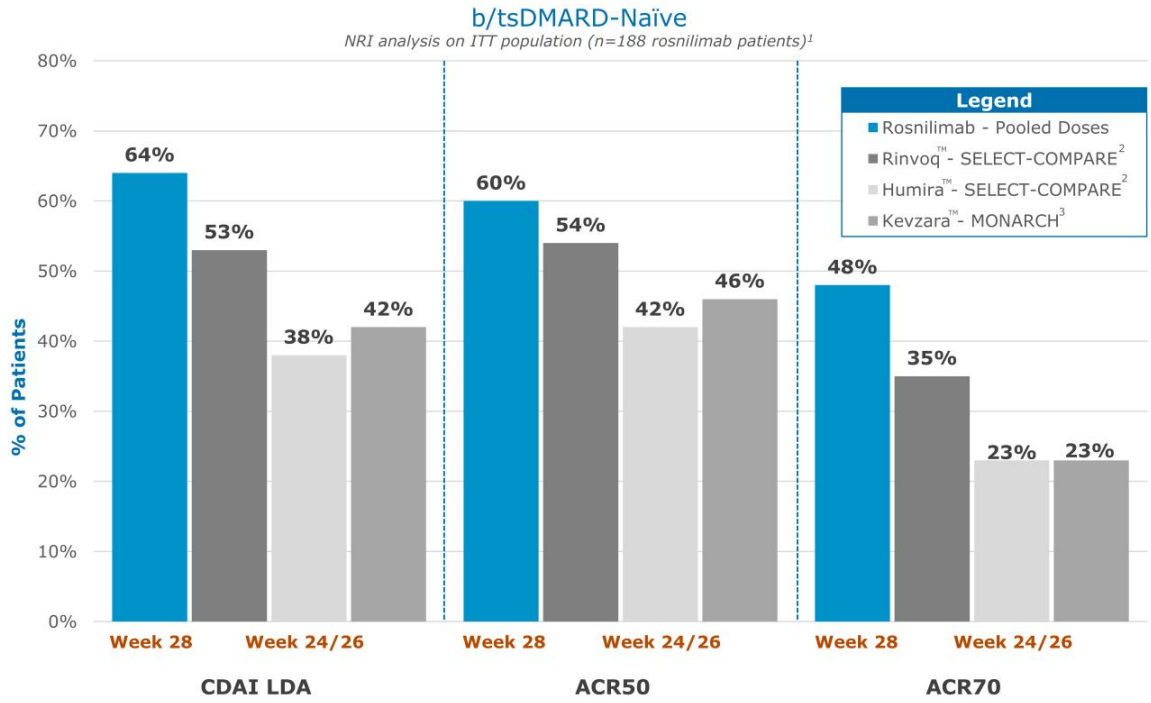
Rosnilimab shows JAK-like efficacy in naïve patients

Compares favorably despite most conservative analysis and capped trial design



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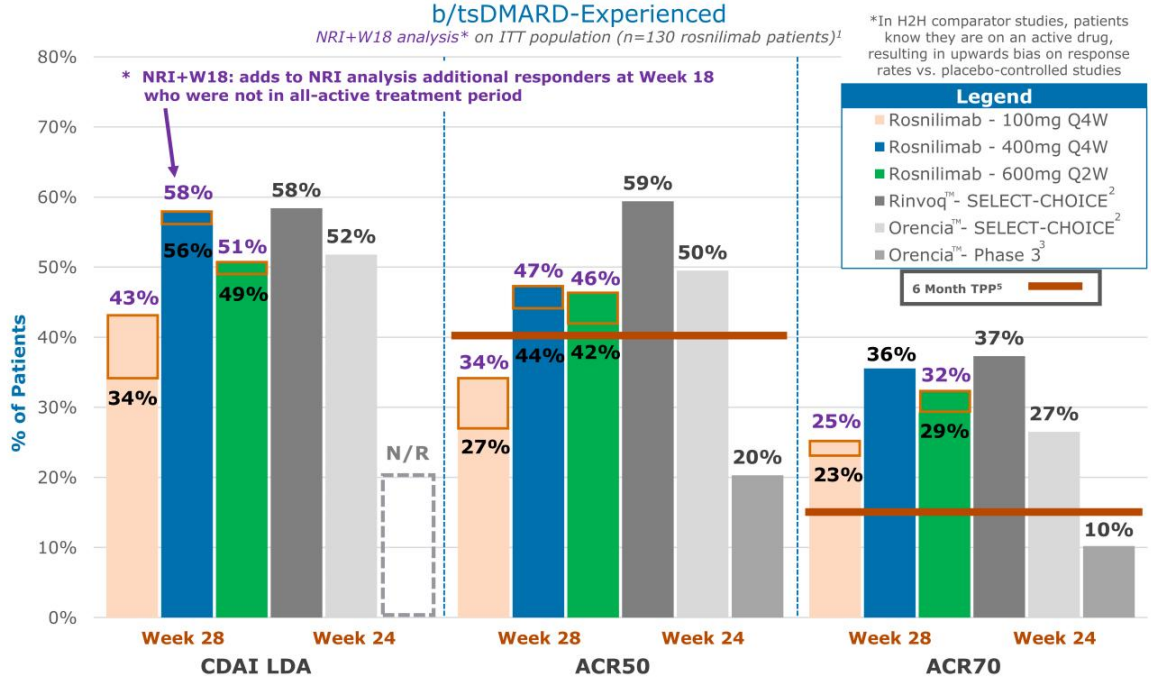
Rosnilimab surpassed TPP in experienced patients and comparable at mid/high dose to JAKs in all-active H2H study*

Includes 29% with prior JAK experience

Excludes 7 patients who discontinued in the all-active treatment period while in CDAI LDA



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CDAI = Clinical Diseases Activity Index; LDA = Low Disease Activity; N/R = Not Reported

Rosnilimab

(PD-1 Depleter and Agonist)

RA and UC in the U.S. are complementary, large commercial markets with similarly high unmet need

Treaters seek to maximize and sustain remission in maintenance phases

RA: U.S. Market

~$10bn
U.S. sales in b/tsDMARD-experienced market[1]

~500k
U.S. b/tsDMARD patients; 20-25% cycle through all classes of therapy[2]

UC: U.S. Market

$8bn ➔ >$10bn
U.S. sales in advanced therapy by 2030[3]

~300k and growing
U.S. advanced therapy patients; Cycling ramping up as advanced therapy market becomes established to avoid surgeries[3]

- Mature, non-growth market
- Established biosimilar 1L SoC (e.g. TNF)
- 2L access possible, but requires contracting
- No new MoA in the last decade

Similarities:

- Focus on achieving low disease activity or remissions long-term
- 1/3 to 1/2 relapse within 1 year following initial response at 3-6 months on induction therapy[4]
- 2L+ markets with blockbuster opportunity
- Minimal differentiation between biologics
- Assess combinations to enhance speed of induction and/or max remission rates
- **Substantial unmet need for new classes of safe, effective and durable therapies**

- Dynamic, growing market
- 1L SoC diversified with growth from branded products (IL-23p19, α4β7)
- Minimal payor management of 2L+ therapies

1. Expected by 2028 (Evaluate 29 Nov 2022); 2. Market research conducted by Ambit in 2022; 3. Market research conducted by ZS Associates in 2025; 4. Phase 3 registrational data from product labels

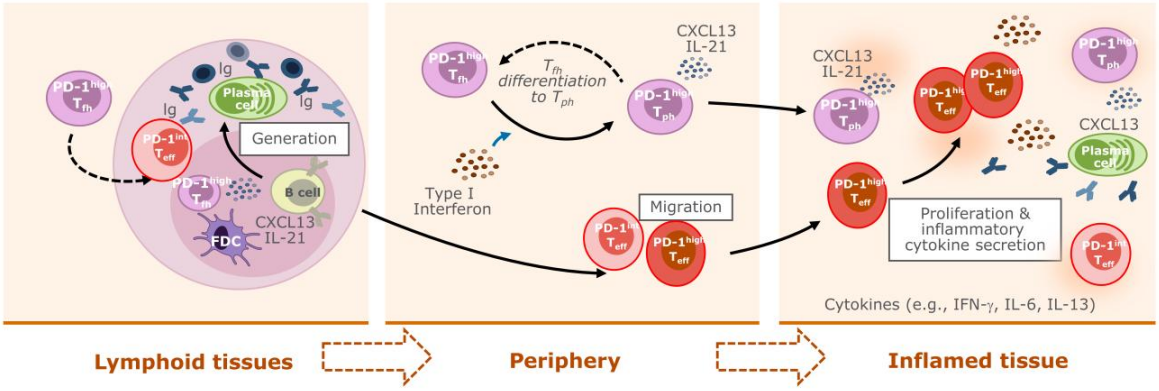
PD-1 is expressed preferentially on activated T_{eff} and T_{fh}/T_{ph} cells that mediate autoimmune pathology

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}

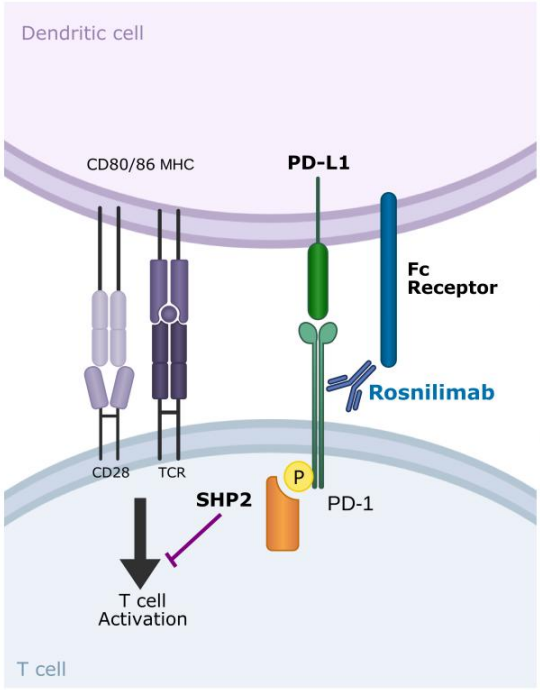
T_{eff} (effector)

- 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.

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



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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.

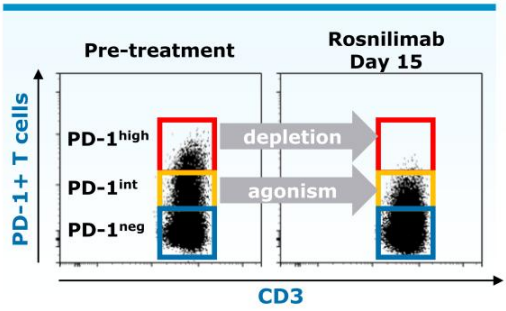
Rosnilimab is designed to bring the immune system back to homeostasis and modify disease

Rosnilimab preferentially targets activated T cells

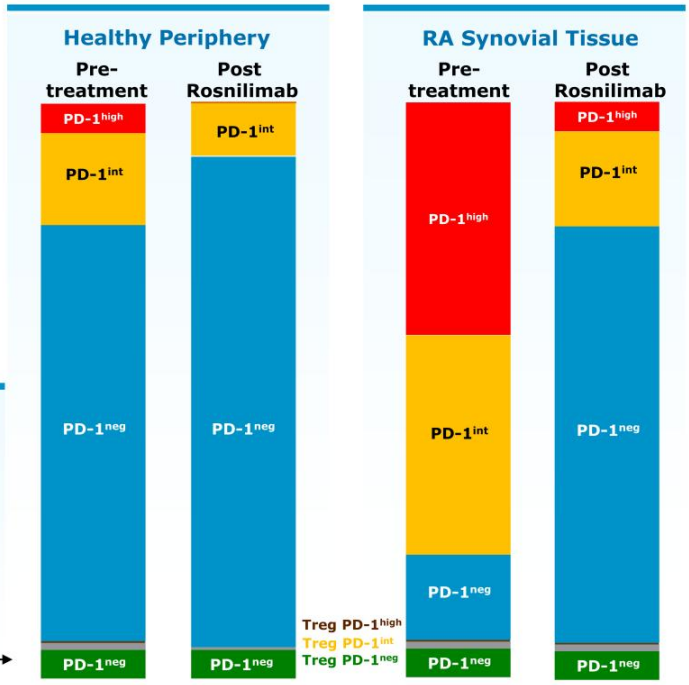
Leverages natural immune regulatory pathway to safely restore immune homeostasis

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

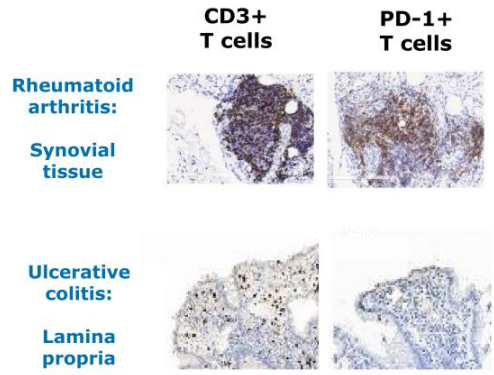


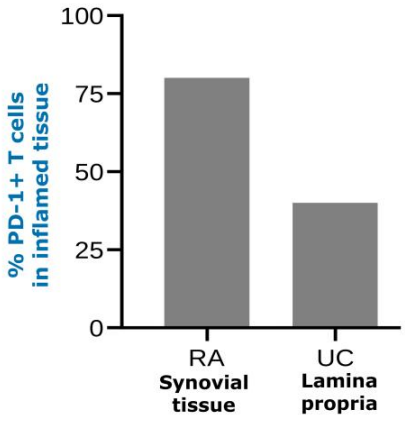
Illustrative T cell composition change



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

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



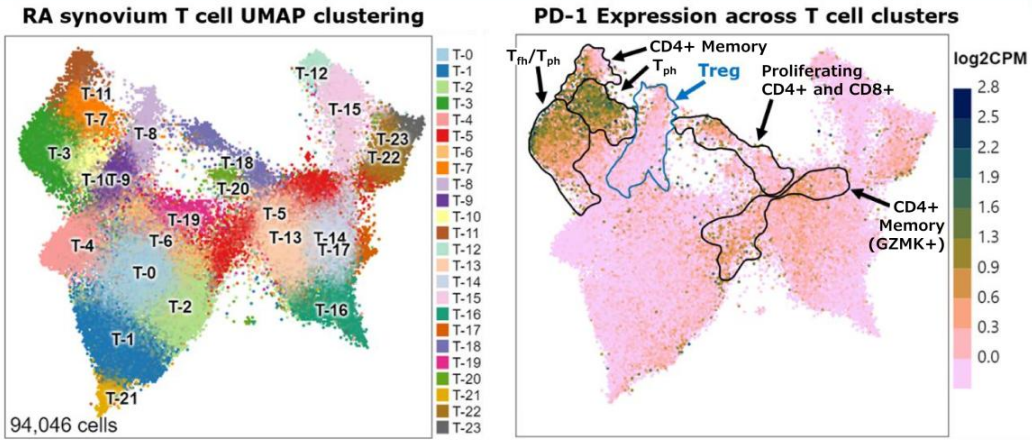


In systemic inflammatory diseases, a multiple fold increase of PD-1+ T cells is observed in periphery compared to healthy controls[1]

~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.

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



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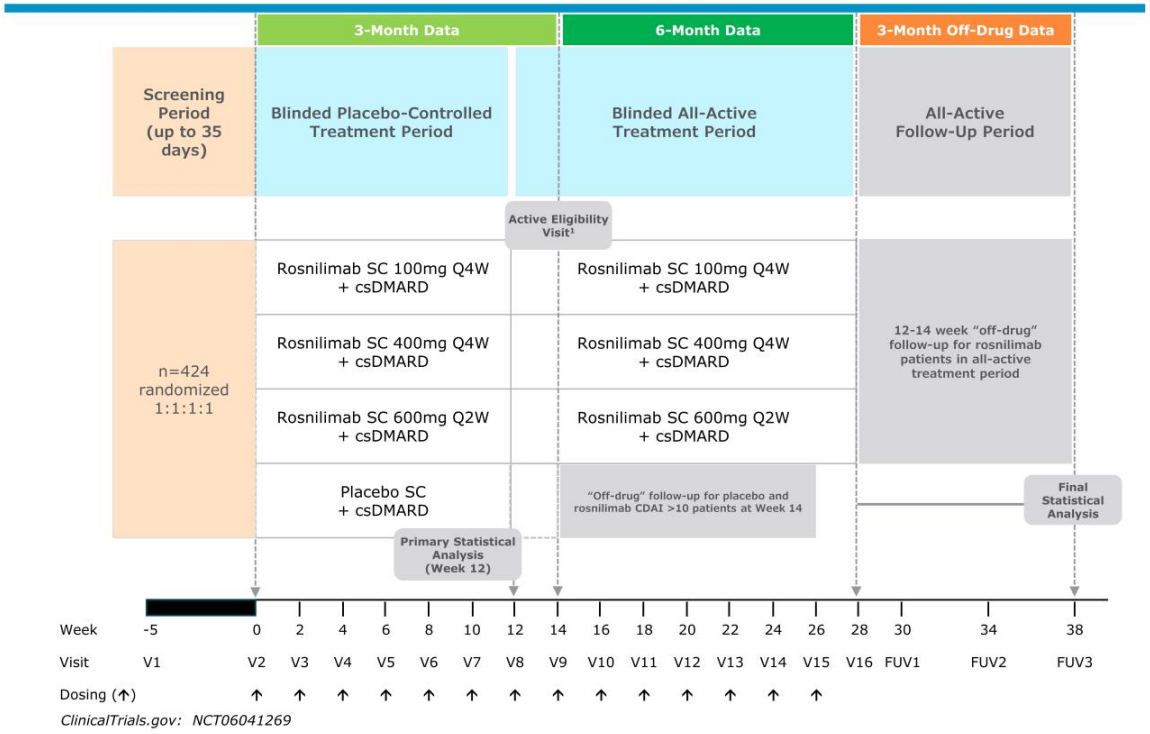
Rosnilimab Phase 2b trial in RA



ClinicalTrials.gov: NCT06041269

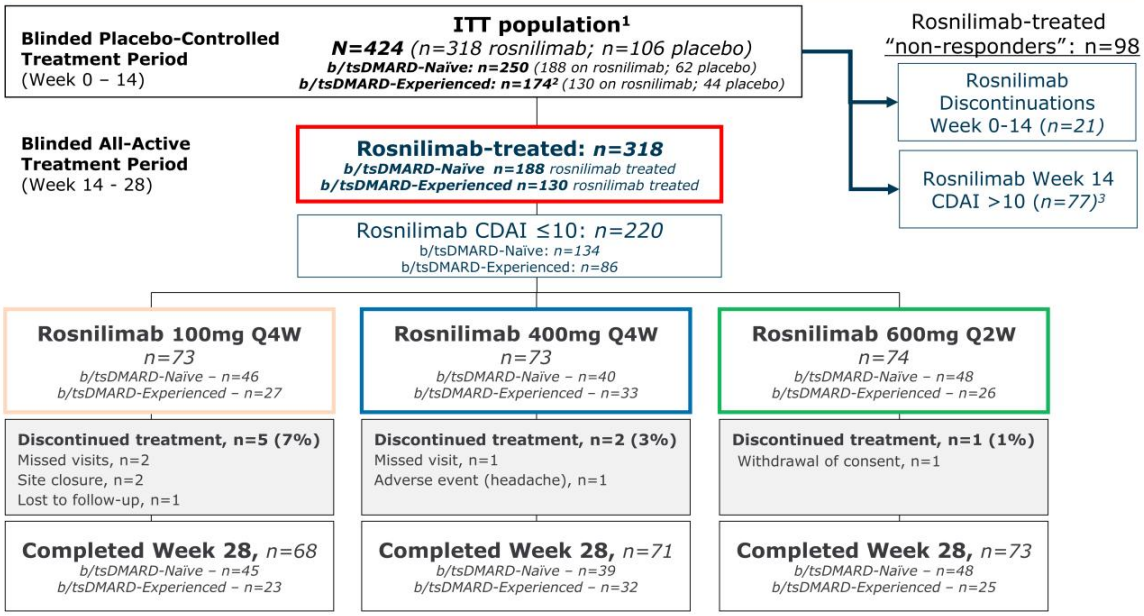
Note: All patients in trial (rosnilimab and placebo arms) are required to be on stable background csDMARD

1. Blinded study drug treatment continued for active treatment group subjects that achieved Clinical Disease Activity Index (CDAI) low disease activity (≤10)

95% completed 6-month all-active treatment period supporting rosnilimab's favorable efficacy and tolerability profile

Discontinuations
All-active treatment period (Week 14 - 28)

- ✓ 7 (of 8 total) discontinuations in all active treatment period were still in CDAI LDA at time of discontinuation
- ✓ No discontinuations due to disease progression
- ✓ Only 1 discontinuation due to AE (headache - moderate)



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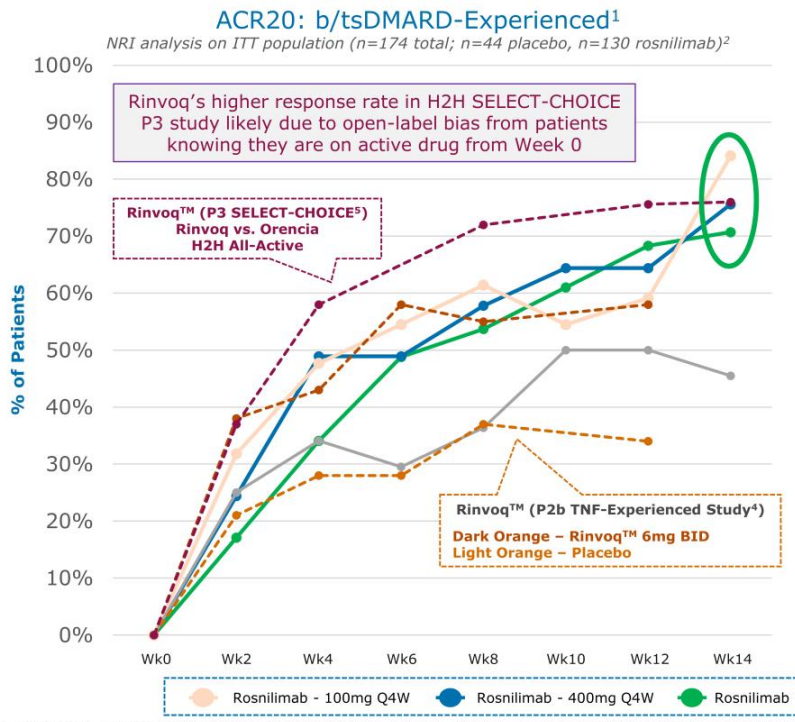
ACR20 response rates are comparable to Rinvoq™

Most patients had symptomatic and clinical improvement by 3 months



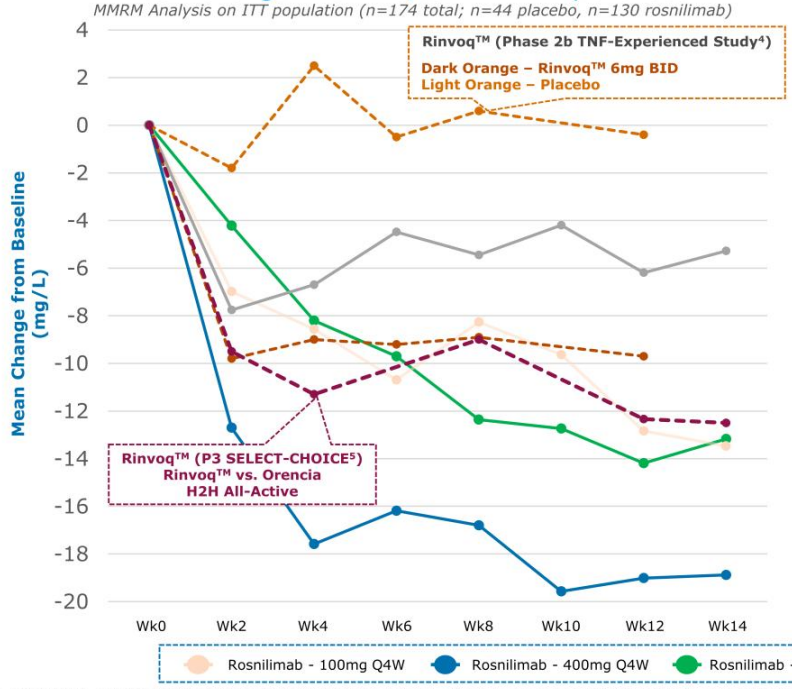
ACR20 at Week 12		
Arm	**Absolute**	**PBO Adjusted**
b/tsDMARD-Experienced Population *(as graphed)*		
100mg	59%	*9%*
400mg	64%	*14%*
600mg	68%	*18%*
Rinvoq[4]	58%	*24%*
Rinvoq[5]	76%	*N/A*
b/tsDMARD-Naïve Population *(for reference)*		
100mg	76%	*21%*
400mg	74%	*19%*
600mg	80%	*25%*
Rinvoq[3]	68%	*22%*

1. b/tsDMARD-experienced population included 29% (n=50 of n=174 total experienced patients) with prior JAK experience; 2. Non-responder imputated (NRI) analysis on intent-to-treat (ITT) of all b/tsDMARD-experienced patients randomized; b/tsDMARD-experienced population (n=44 placebo, n=44 100mg Q4W, n=45 400mg Q4W, n=41 600mg Q2W; n=130 total rosnilimab b/tsDMARD-experienced patients); 3. Rinvoq™ Phase 2b MTX-IR study; 4. Rinvoq™ Phase 2b TNF-experienced study; 6mg BID (equivalent to 15mg QD); 5. SELECT-CHOICE Phase 3 study

CRP reductions are comparable to Rinvoq™

Mean Change in CRP: b/tsDMARD-Experienced[1]

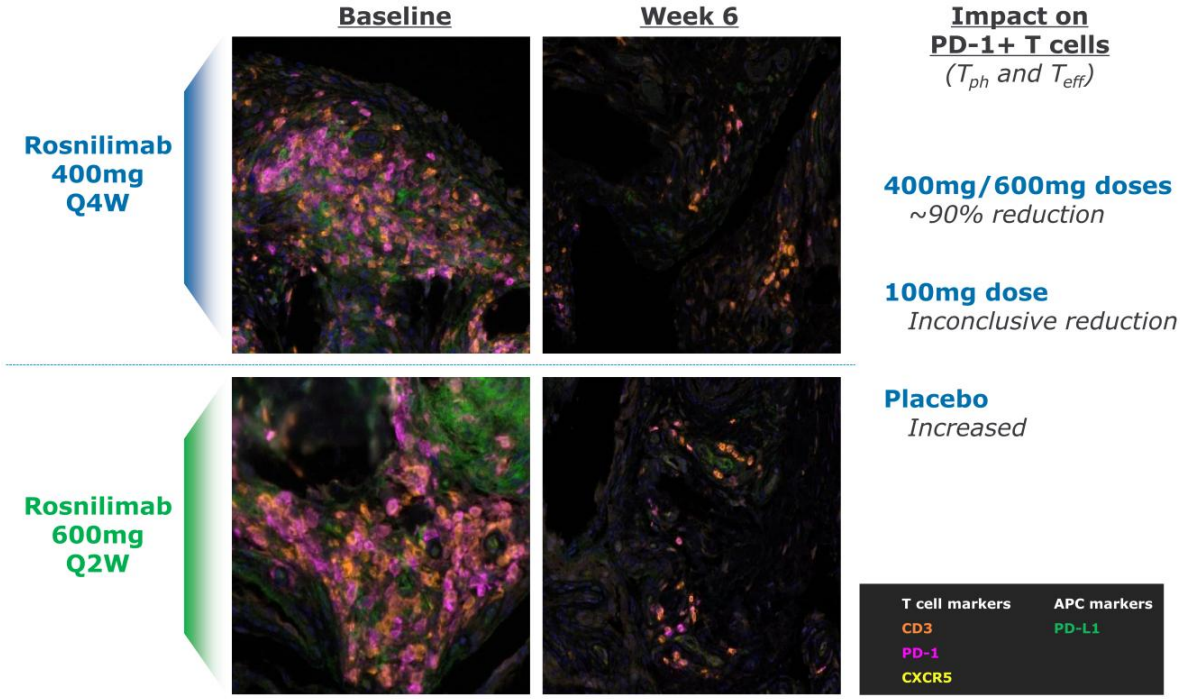
MMRM Analysis on ITT population (n=174 total; n=44 placebo, n=130 rosnilimab)



Change in CRP at Week 12		
Arm	**Baseline Mean CRP**	**PBO Adjusted**
b/tsDMARD-Experienced Population *(as graphed)*		
100mg	20.0	*-6.7*
400mg	29.4	*-12.8*
600mg	23.3	*-8.0*
Rinvoq[4]	16.0	*-9.3*
Rinvoq[5]	19.0	*N/A*
b/tsDMARD-Naïve Population *(for reference)*		
100mg	14.9	*-10.6*
400mg	14.3	*-7.0*
600mg	15.7	*-6.7*
Rinvoq[3]	17.0	*-8.4*

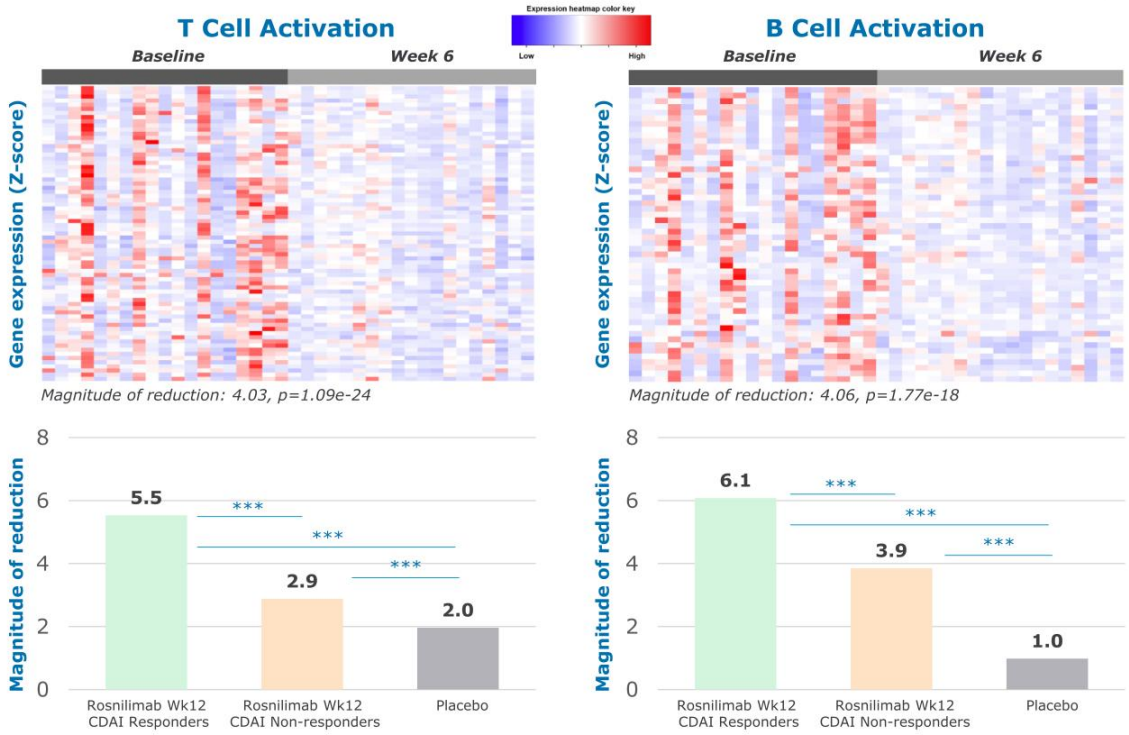
1. b/tsDMARD-experienced population included 29% (n=50 of n=174 total experienced patients) with prior JAK experience; 2. Mixed Model for Repeated Measures (MMRM) analysis on intent-to-treat (ITT) of all b/tsDMARD-experienced patients randomized; b/tsDMARD-experienced population (n=44 placebo, n=44 100mg Q4W, n=45 400mg Q4W, n=41 600mg Q2W); 3. Rinvoq™ Phase 2b MTX-IR study; 4. Rinvoq™ Phase 2b TNF-experienced study; 6mg BID (equivalent to 15mg QD) 5. SELECT-CHOICE Phase 3 study

Synovial biopsies show ~90% reduction of PD-1+ T cells in the target issue



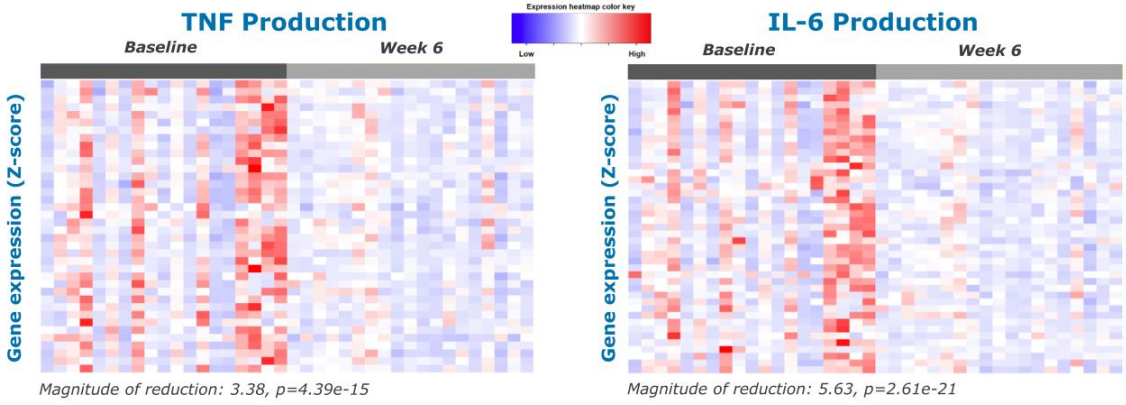
Note: Synovial biopsies of the most impacted joint taken at baseline and 6 weeks on study. Immunofluorescence performed to identify PD-1 positive cells. T_{ph} cells (PD-1+CD3+CD4+CXCR5-)

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



Note: Gene ontology (GO) pathway analysis performed on samples with evidence of inflammation at baseline (all rosnilimab doses pooled, n=19 paired biopsies) and with myosin normalization. Rows reflect genes with p<0.05 between Weeks 6 and 0. Magnitude of reduction defined as fold enrichment score. Rosnilimab responders achieved CDAI LDA in 3 months. ***p<0.001 for difference in fold change between baseline and Week 6 between groups.

Significant reduction of additional downstream pathways including TNF and IL-6 within the synovium



Magnitude of reduction: 3.38, p=4.39e-15

Magnitude of reduction: 5.63, p=2.61e-21

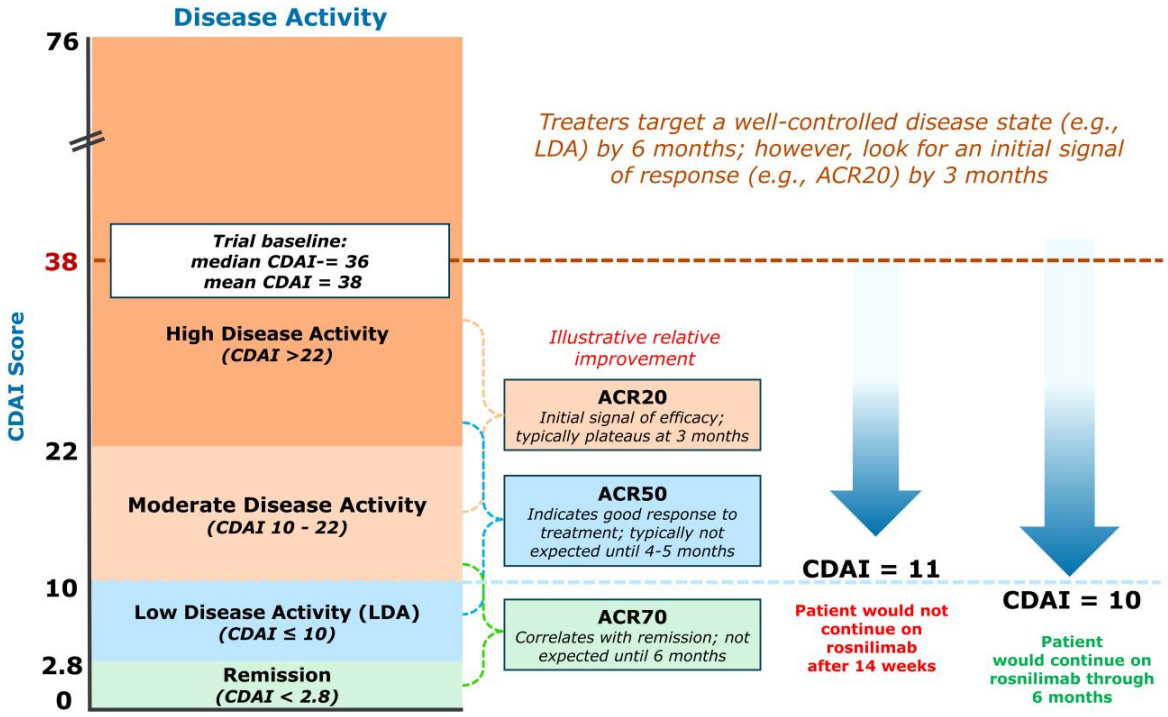
Pathway changes reflect rosnilimab's broad MOA

Significantly downregulated (p<0.05) genes of interest in RA:	
	T cell activation: IL2RA, TNFSF14 (LIGHT), CD28, CD69, CD40L, ICOS, CD226, ZAP70, TCF7, IRF1
	B cell activation: IL7R, CD27, CD79A, BTK, SYK, IL21R
	TNF and IL-6 production: MYD88, PTPN22, LILRB1, LILRB2, NOD2, CCR2, NLRC3, IRAK3, IL1RAP, IL6R, IL17RA
	Mediators of RA structural damage: MMP1, MMP3, and RANK-L
	IBD-related genes: NOD2, TREM1, IL12RB, IFNGR1, S100A8

Note: Gene ontology (GO) pathway analysis performed on samples with evidence of inflammation at baseline (all rosnilimab doses pooled, n=19 paired biopsies) and with myosin normalization. Rows reflect genes with p<0.05 between Weeks 6 and 0. Magnitude of reduction defined as fold enrichment score.

LDA requirement at 14 weeks to continue on rosnilimab was a high bar for patients with baseline high disease activity

95% of trial participants had high disease activity (CDAI > 22) at baseline

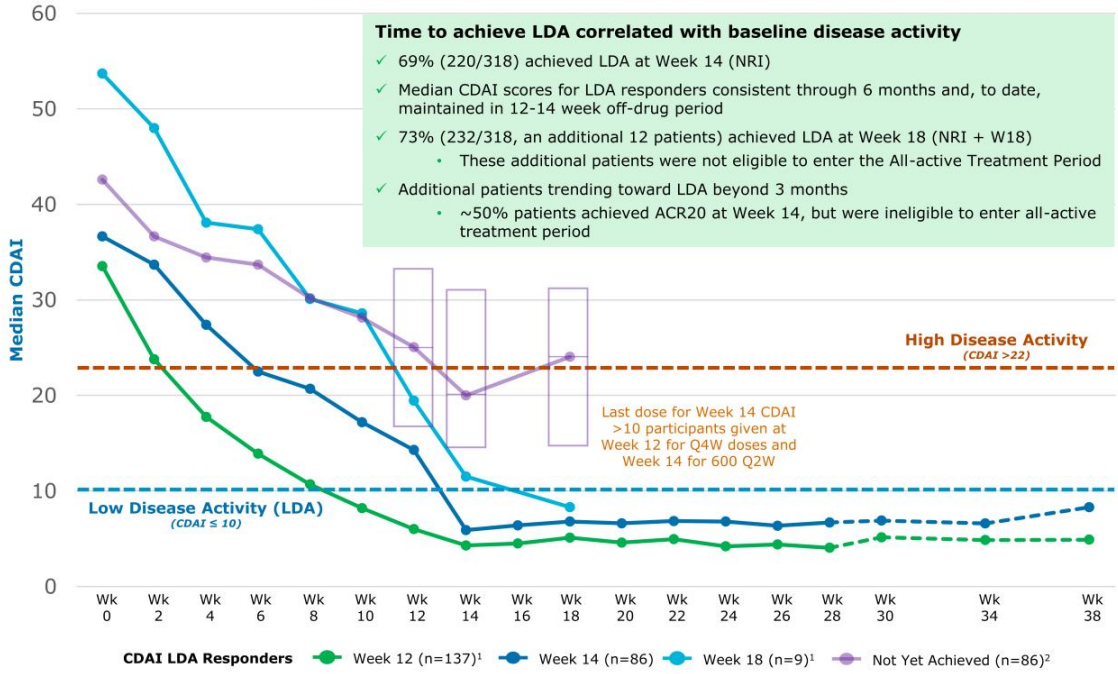


Max response was not achieved in this Phase 2b trial

On average, patients with higher disease activity take longer to achieve CDAI LDA

Median Change from Baseline in CDAI

NRI analysis on ITT population (n=318 rosnilimab patients)

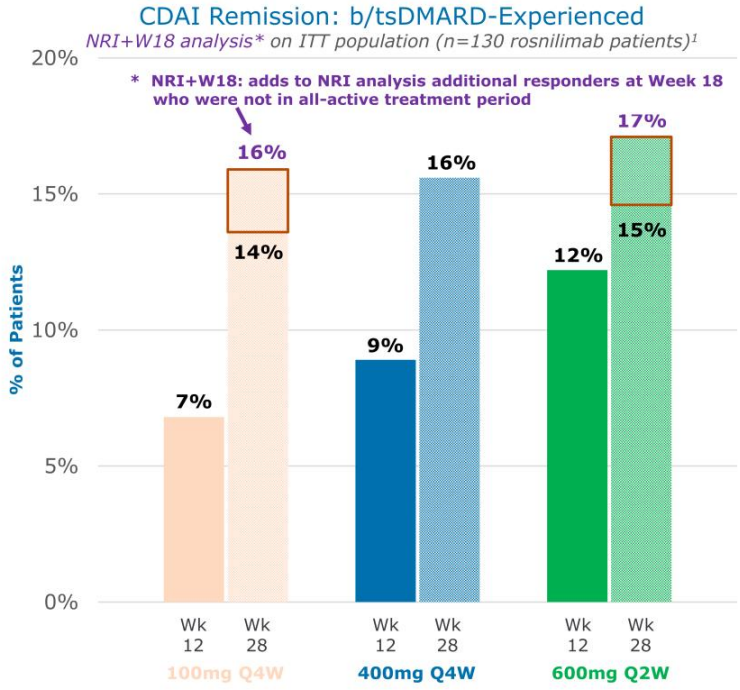


1. Green line includes 3 patients that achieved LDA at Week 12, were not CDAI LDA at Week 14, but returned to CDAI LDA at Week 18. These same 3 patients were excluded from the Light Blue line. In total 12 patients achieved CDAI LDA at Week 18. 2. Purple line includes rosnilimab patients that discontinued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

JAK-like CDAI remission rates which deepened into six months

Includes 29% with prior JAK experience

Excludes 2 patients who discontinued in the all-active treatment period while in CDAI remission



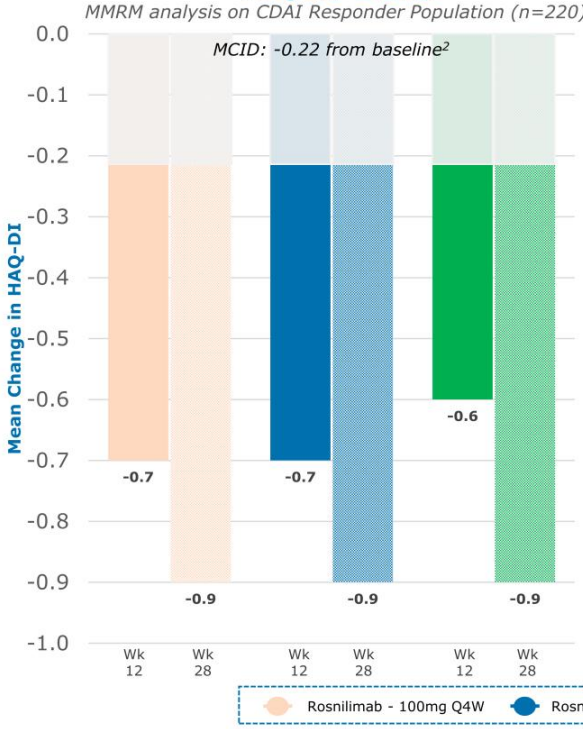
CDAI Remission at Week 28		
Arm	NRI	NRI+W18
b/tsDMARD-Experienced Population *(as graphed)*		
100mg	14%	16%
400mg	16%	16%
600mg	15%	17%
b/tsDMARD-Naïve Population		
100mg	21%	21%
400mg	18%	18%
600mg	17%	19%

1. Non-responder imputated (NRI) analysis on intent-to-treat (ITT) of all b/tsDMARD-experienced patients randomized; b/tsDMARD-experienced population (n=44 100mg Q4W, n=45 400mg Q4W, n=41 600mg Q2W; n=130 total rosnilimab b/tsDMARD-experienced patients)

Highly meaningful clinically and symptomatic improvement across multiple PROs and CRP

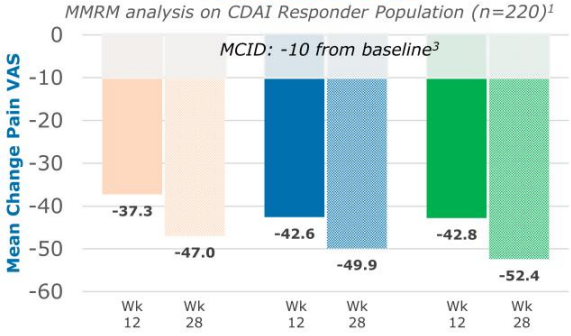
HAQ-DI: Overall

MMRM analysis on CDAI Responder Population (n=220)[1]



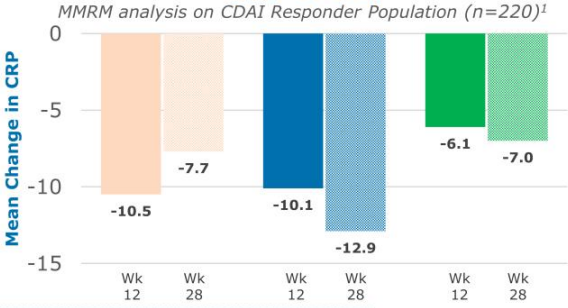
Pain VAS: Overall

MMRM analysis on CDAI Responder Population (n=220)[1]



CRP: Overall

MMRM analysis on CDAI Responder Population (n=220)[1]

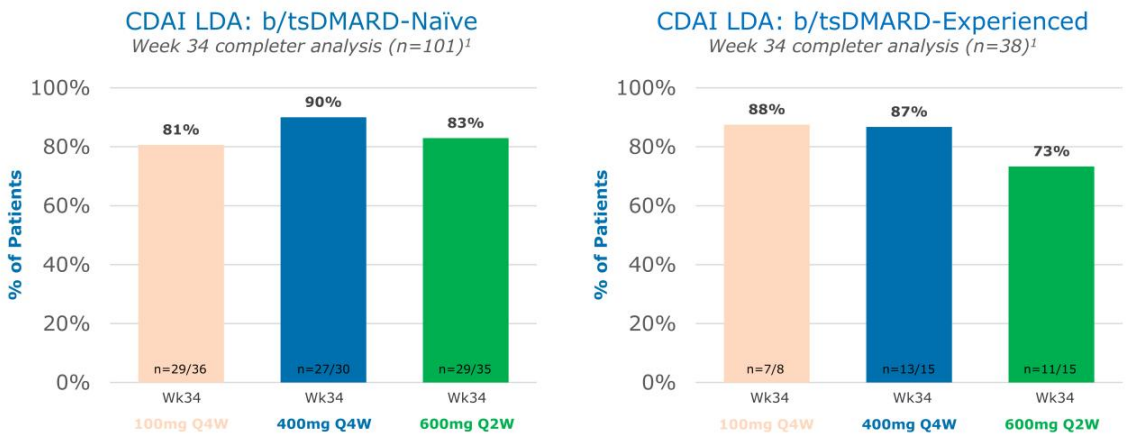


Rosnilimab - 100mg Q4W Rosnilimab - 400mg Q4W Rosnilimab - 600mg Q2W

1. Mixed Model for Repeated Measures (MMRM) analysis on rosnilimab CDAI LDA responder at Week 14 population (n=220) includes naïve population (n=46 100mg Q4W, n=40 400mg Q4W, n=48 600mg Q2W; n=134 total rosnilimab patients) and experienced population (n=27 100mg Q4W, n=33 400mg Q4W, n=26 600mg Q2W; n=86 total rosnilimab patients); 2. Behrens et. al, BMC Rheumatology, Dec. 2019; 3. Strand et. al, Journal of Rheumatology, Aug. 2011

Durable responses for at least 2-months off-drug

83% of Week 28 CDAI LDA responders were still in response at Week 34



Most patients who did not sustain CDAI LDA remained near the cutoff of CDAI=10

Only 23 of 139 (17%) patients of Week 28 CDAI LDA responders were not CDAI LDA (≤10) at Week 34:

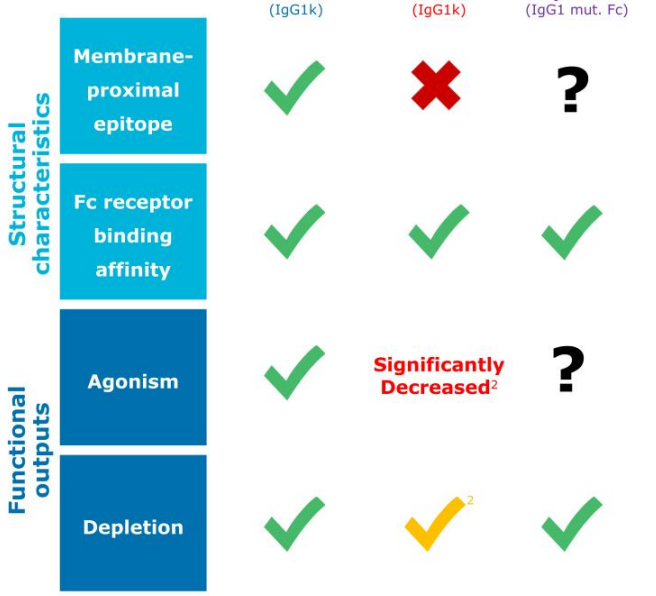
- 25% (6/23) were CDAI <11
- Median CDAI = 13
- 91% (21/23) were CDAI <22 (e.g. remained CDAI moderate disease activity)

1. ~75% of patients who completed Week 28 (n=212) had reached Week 34 as of the March 11, 2025 data cutoff; this responder analysis represents patients who were in CDAI LDA, as of Week 28, relative to their CDAI status as of Week 34

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"

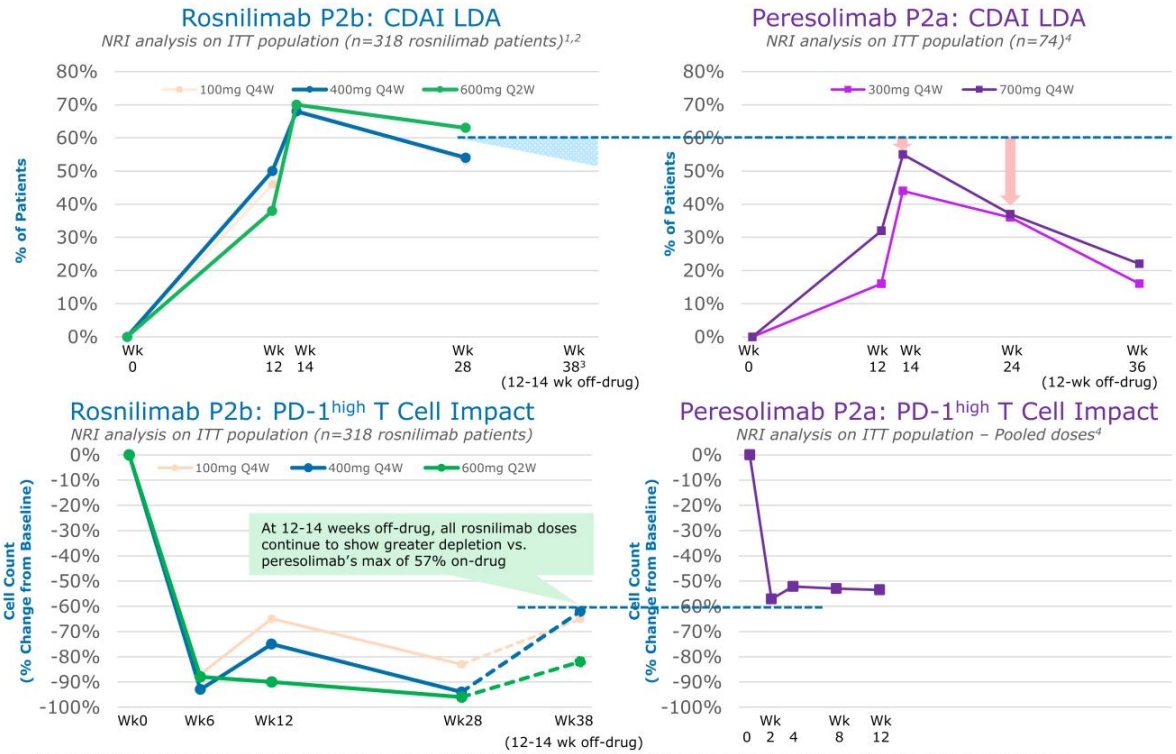
"FcgRs work as a hook in the IS [immune synapse] to allow PD-1 to relocate to a close proximity of the TCR to enable inhibition of TCR signalling"

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.

LDA response rates and durability for rosnilimab are differentiated from Lilly's peresolimab



1. Non-responder imputated (NRI) analysis on intent-to-treat (ITT) of all 318 rosnilimab patients randomized; 2. At Week 28, 53% (100mg Q4W), 54% (400mg Q4W), and 63% (600mg Q2W) rosnilimab patients were in CDAI LDA (57% pooled); 3. Off-drug follow-up period ongoing; 4. Tuttle et. al, NEJM, May 2023, Supplemental Appendix, At Week 28, 36% (300mg Q4W) and 37% (700mg Q4W) peresolimab patients were in CDAI LDA

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 well tolerated with no safety signals

<2% dropout rate overall due to AEs through 6 months,
with only 1 dropout due to AE (headache-moderate) after 3 months

Study Period	Week 0 through Week 12 (N=424)				Week 0 through Week 28 (N=424)			
	Participants with Adverse Events, n (%)				Participants with Adverse Events, n (per 100 PY)*			
	Placebo (n=106)	100mg Q4W (n=106)	400mg Q4W (n=107)	600mg Q2W (n=105)	Placebo (n=106)	100mg Q4W (n=106)	400mg Q4W (n=107)	600mg Q2W (n=105)
Any AE	**36 (34%)**	**51 (48%)**	**48 (45%)**	**38 (36%)**	**39 (125.6)**	**73 (260.9)**	**66 (206.5)**	**52 (149.1)**
Any SAE[1]	1 (1%)	1 (1%)	1 (1%)	3 (3%)	1 (2.4)	2 (3.8)	2 (3.7)	4 (7.7)
Any Drug-Related SAE	1 (1%)	0 (0%)	0 (0%)	0 (0%)	1 (2.4)	0 (0)	0 (0)	0 (0)
Severe AE[2]	2 (2%)	1 (1%)	0 (0%)	4 (4%)	2 (4.8)	4 (7.5)	1 (1.9)	4 (7.8)
Drug-Related AE	18 (17%)	13 (12%)	18 (17%)	17 (16%)	18 (48.8)	17 (36.1)	28 (62.0)	19 (41.7)
AE Leading to Treatment Discontinuation	1 (1%)	1 (1%)	2 (2%)	2 (2%)	1 (2.4)	1 (1.9)	3 (5.6)	2 (3.8)
Infections	14 (13%)	24 (23%)	21 (20%)	12 (11%)	16 (41.5)	41 (98.7)	39 (89.4)	31 (67.6)
Serious	1 (1%)	1 (1%)	0	0	1 (2.4)	1 (1.9)	1 (1.9)	1 (1.9)
Opportunistic[3]	2 (1.9%)	0 (0%)	0 (0%)	0 (0%)	2 (4.8)	1 (1.9)	1 (1.8)	1 (1.9)
Participants with any AEs > 5%								
Headache	4 (4%)	7 (7%)	6 (6%)	4 (4%)	4 (9.7)	10 (19.9)	10 (19.4)	5 (9.8)
Upper respiratory tract infection	1 (1%)	7 (7%)	2 (2%)	3 (3%)	2 (4.8)	14 (27.8)	7 (13.4)	10 (19.6)
Nasopharyngitis	4 (4%)	5 (5%)	5 (5%)	0	4 (9.6)	9 (17.5)	8 (15.4)	1 (1.9)
Elevated ALT (alanine aminotransferase)[4]	1 (1%)	4 (4%)	3 (3%)	3 (3%)	1 (2.4)	8 (15.5)	5 (9.5)	4 (7.8)

** Measured as an exposure adjusted incidence rate (per 100 patient years)*

1. SAEs (severe unless otherwise noted): RSV – moderate (600mg Q2W); anaphylaxis from wasp sting (600mg Q2W); ureter stone (600mg Q2W); cholecystitis / pericardial effusion (600mg Q2W); meniscus tear – moderate (400mg Q4W); diverticulitis – moderate (400mg Q4W); embolic ischemic stroke (100mg Q4W); pneumonia – mild (100mg Q4W); cellulitis/diarrhea (placebo)

2. Severe AEs (excluding SAEs): RA flare (600mg Q2W); blood creatine phosphokinase increase (400mg Q4W); endometriosis (100mg Q4W); alanine aminotransferase increased/aspartate aminotransferase increase (100mg Q4W); flu/headache (100mg Q4W); macular degeneration/retinal hemorrhage (placebo)

3. Values shown are for herpes zoster, none were severe and are the only opportunistic infection reported.

4. No patient met the predefined protocol liver function test stopping criteria. Only one ALT elevation was severe, which resolved without interruption of therapy, none were serious, all had an outcome of recovered/resolved or recovering/resolving

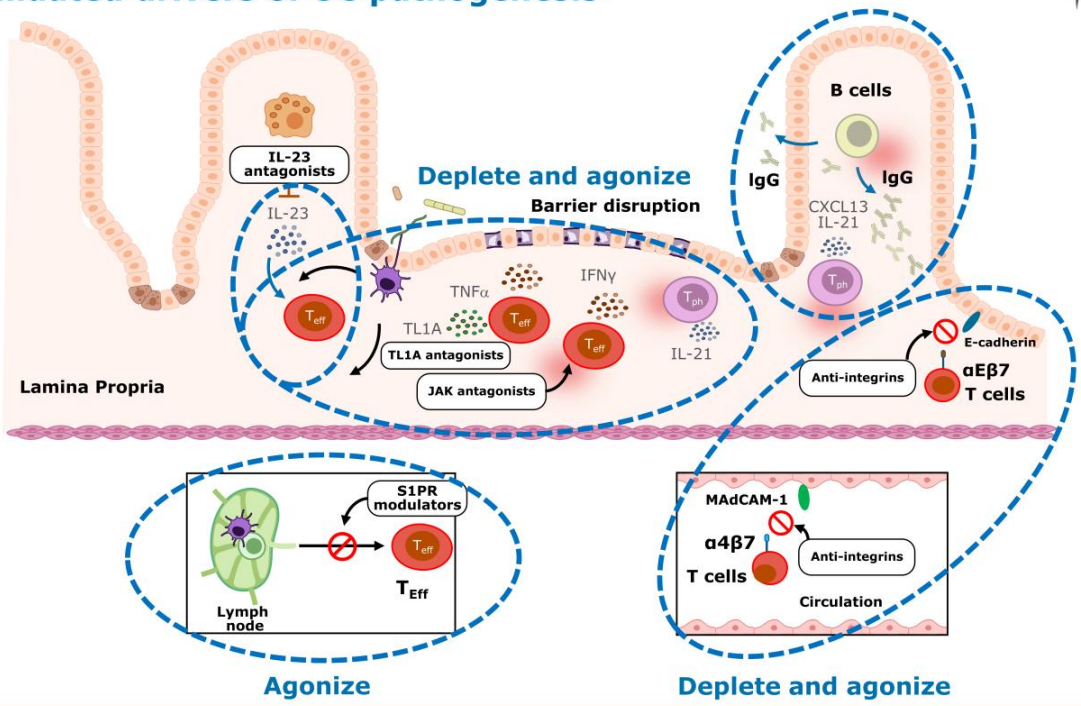
Next steps for rosnilimab

- **Plan to present RA Phase 2b data at a future medical congress**

- **Assessing two, alternative, strategic paths forward**
  - Secure a global partnership, to help advance in all indications, including P3 for both RA and UC
  - Independently advance UC into P3 (assuming P2 data meets TPP)

- **2026+ activities**
  - P3 enablement: drug supply scale-up and regulatory interactions
  - Initiate P2 studies in additional indications

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

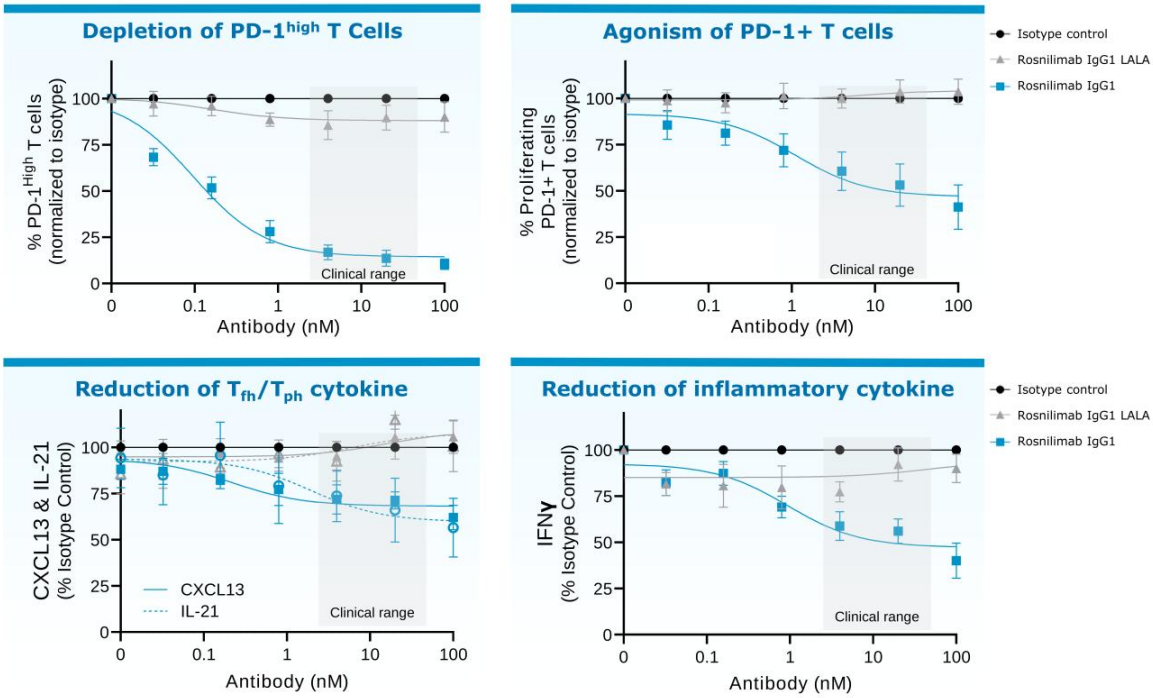


Agonize **Deplete and agonize**

>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.

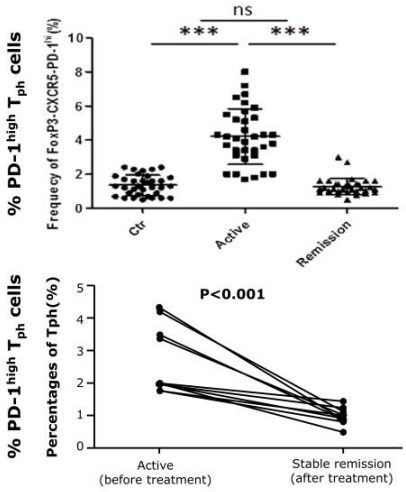
Rosnilimab's potent depletion and agonism reduces T cell proliferation and inflammatory cytokines that disrupt barrier function



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.

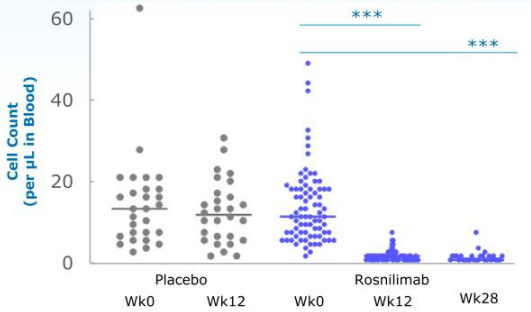
T_{ph} impact seen in RA Phase 2b trial relevant to UC biology and correlates to reduction of remission

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

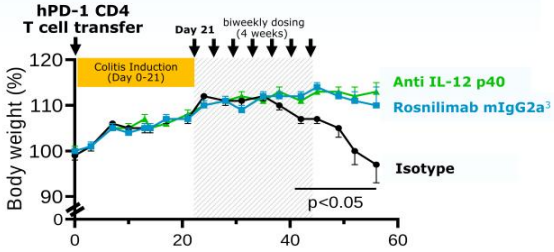


Reduction of T_{fh}/T_{ph} cells should impact plasma cell generation and autoantibody levels, including anti-microbial IgG antibodies that are contributing to colonic inflammation and barrier disruption[4]

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

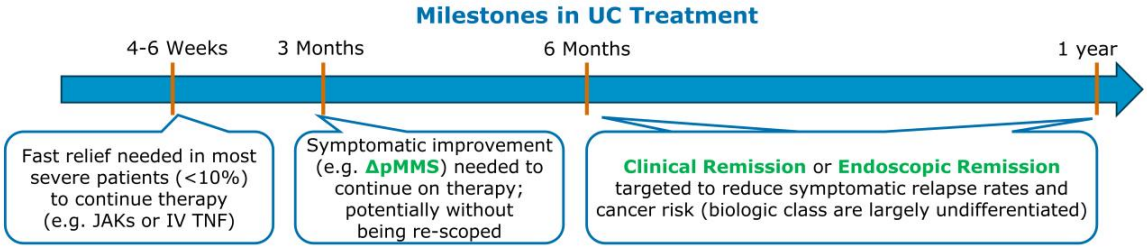
1. PD-1^{high} T_{ph} cells defined by CD3+CD4+CD45RA-PD-1+TIGIT+ICOS+CXCR5-. Long et al, Immunology Letters 233 (2021) 2-10.; 2. Rao et al, Nature, 2017. *** p<0.001, * p<0.05; 3. Rosnilimab formatted to mIgG2a to mediate effector function in mice. Suzuki et al., Sci. Immunol. 8, eadd4947 (2023); 4.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

Treaters target a well controlled disease state (e.g. endoscopic remission) in 6-12 month maintenance phase

However, treaters look for a signal of response (e.g., improved clinical symptoms) by 3+ month induction phase

UC TPP guidance

- 3 months: ΔMMS vs. placebo (primary endpoint) is statistically significant
  - Adequate symptomatic improvement to stay on drug to potential remission (by 6+ months)
- 6 months: "IL-23-like" clinical remission and endoscopic remission as measured by imputed ITT (not maintenance responder analysis)
- 6 – 12 months: Better durability than biologics, where 1/3 to 1/2 relapse within 1 year

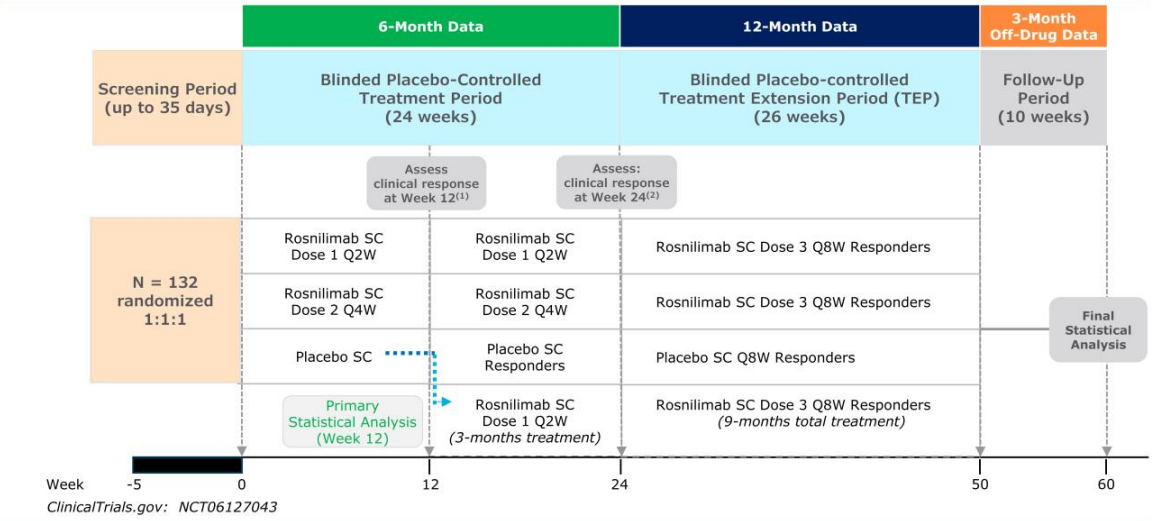


Increasingly, differentiation to be driven by 6+ month objective endpoints

AbbVie: focus on "extended induction" (~6 months) to maximize clinical response on IL-23p19's
J&J: primary endpoint of DUET TNF + IL-23p19 combo study is clinical remission at Week 48

Rosnilimab Phase 2 in moderate-to-severe UC

Enrollment ongoing; On track for interim 6-month data in Q4 2025



ClinicalTrials.gov: NCT06127043

- **Primary endpoint: Change in mMS[3] (stool frequency + rectal bleeding + endoscopy score) vs. placebo at Week 12**
  - Assesses symptomatic and objective clinical improvement at 3 months
- **"Treat-through" design aligned with clinical practice to give a real-world assessment of remission at 6 months**
  - Distinctly different from trials with an enriched responder analysis in 6 or 12-month maintenance studies

1. Week 12 visit: All patients, regardless of study arm, treat-through to Week 24 and remain blinded to treatment arm. Placebo-treated patients who achieved partial modified Mayo score (pmMS) clinical response or at Week 12 remain on placebo, while placebo-treated patients who are non-responders on pmMS are crossed over to the subcutaneous high-dose rosnilimab treatment arm; 2. Week 24 visit: All patients, regardless of study arm, can opt into the TEP to Week 50 if achieved pmMS clinical response by Week 24. All non-responders proceed to study follow-up; 3. mMS = modified Mayo score

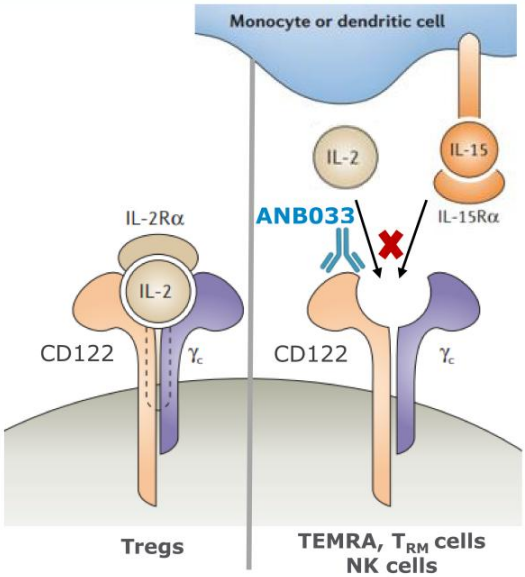
ANB033 (CD122 antagonist)

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

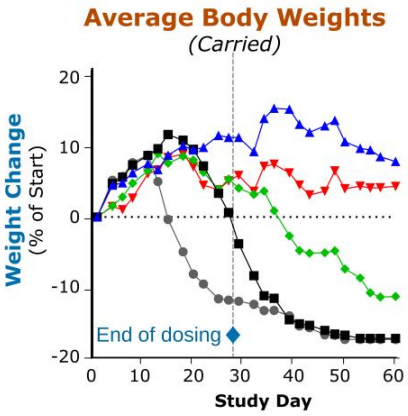
- T_{RM} cells require IL-15 for survival
- May potentially drive durable response

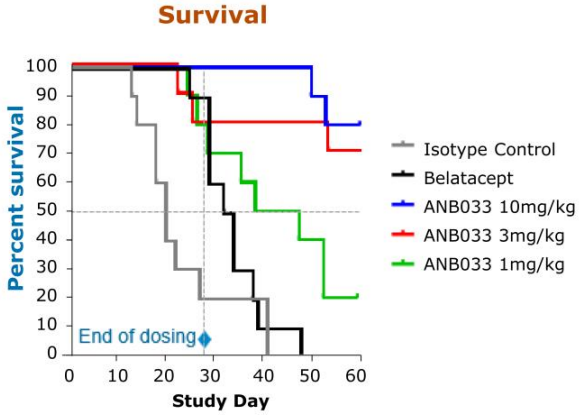
ANB033: Durable survival in GVHD model

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.

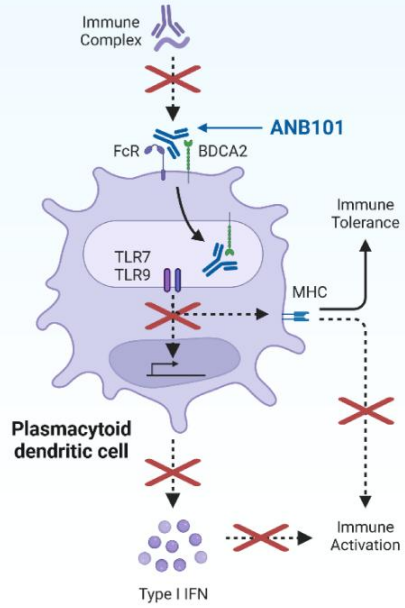
ANB101
(BDCA2 modulator)

Autoimmune and Inflammatory Diseases

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

Phase 1 trial ongoing in healthy volunteers

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)

Strong capital position with ~$383MM Q1 2025 cash and significant GSK royalty upside potential

Anaptys' strong capital position

- $383MM Q1 2025 cash
- Cash runway: YE 2027
  - Excludes GSK royalty and milestone potential for *Jemperli* and cobolimab
  - Excludes GSK $75MM milestone anticipated in 2025/2026 for *Jemperli* $1B annual WW sales

Potential GSK future growth drivers

- 1L "all-comers" endometrial: Approved in US mid-'24; in EU Jan. '25
- 2L+ NSCLC: Phase 3 COSTAR (*Jemperli* + cobolimab) OS data in mid-2025[1]
- Locally advanced dMMR/MSI-H rectal cancer: granted FDA Breakthrough Therapy Designation
- Substantial investment in additional *Jemperli* indications and combinations ongoing

Jemperli Quarterly Performance[1]

Jemperli Wall Street Consensus[1,2]

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).

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]
- ~$104MM paid to Sagard as of March 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 1/27/2025 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.

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 and EU for primary advanced or recurrent EC in combination with chemotherapy.
  - **2L endometrial cancer:** Monotherapy approved in US and EU for dMMR/MSI-H recurrent or advanced EC after progressing on a platinum-containing regimen
  - Significant U.S. market opportunity with 23,000 eligible diagnoses/year[1]

Colorectal cancer

- **Rectal cancer:** P2 AZUR-1 trial (dostarlimab monotherapy in dMMR/MSI-H in locally advanced [LA] rectal cancer)
  - Registrational, fully enrolled, with top-line data in 2026
- **Colon cancer:**
  - P3 AZUR-2 trial (perioperative dostarlimab monotherapy vs SoC adjuvant chemotherapy in patients with high-risk early-stage dMMR/MSI-H cancer)
  - P2 AZUR-4 trial (dostarlimab combination with chemo in neoadjuvant MMRp/MSS cancer)

Additional potential dostarlimab royalty opportunities

- P3: LA unHNSCC monotherapy sequentially after chemoradiation (JADE study)
- P1/2 combinations with anti-CD96 and PVRIG across multiple solid tumors, including HNSCC

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

Lung cancer

- **2L NSCLC:** P3 COSTAR trial (docetaxel *vs* dostarlimab + docetaxel *vs* docetaxel + dostarlimab + cobolimab)
  - Top-line data expected in mid-2025
  - Significant U.S. market opportunity with 237,000 new NSCLC diagnoses/year[1]
- **Head-to-Head vs. Keytruda:** P2 PERLA trial (46% cORR for dostarlimab + chemotherapy vs. 37% cORR for pembrolizumab + chemotherapy, HR 0.70)
  - *Not for registration*; data reported in December 2022

Liver cancer

- **1L HCC:** P1 AMBER Cohort F trial (dostarlimab + cobolimab)

1. NCI SEER data

Vanda Pharmaceuticals Financial Collaboration

Imsidolimab (IL-36R antagonist)

Imsidolimab (IL-36R antagonist) out-licensed to Vanda

Key financial terms to Anaptys

VANDA PHARMACEUTICALS INC.

Exclusive global license to Vanda
announced February 2025

$15 million upfront payment
$10 million upfront and $5 million for existing drug supply

$35 million future milestones
$5 million – FDA approval in GPP
$5 million – EMA approval in GPP
$25 million – Achievement of $100 million WW annual net sales

10% royalties on global net sales

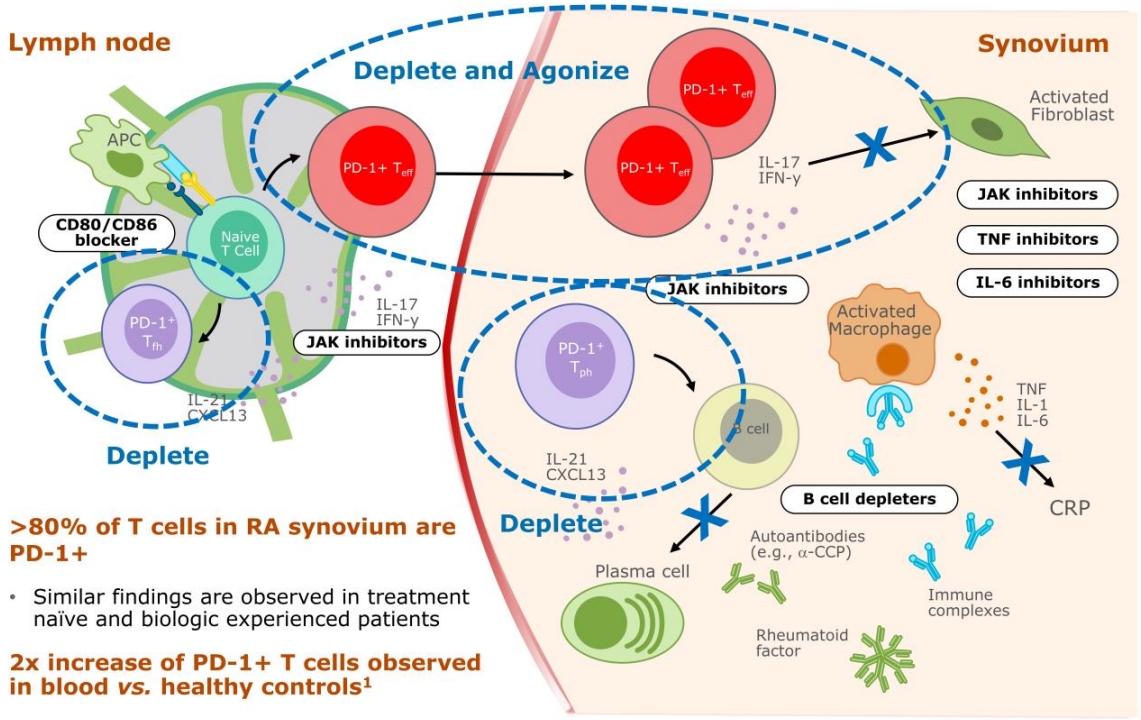
FDA BLA submission for generalized pustular psoriasis (GPP) expected in 2025

Imsidolimab completed two positive global Phase 3 studies in GPP

Vanda plans to expand development into additional indications

Appendix

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



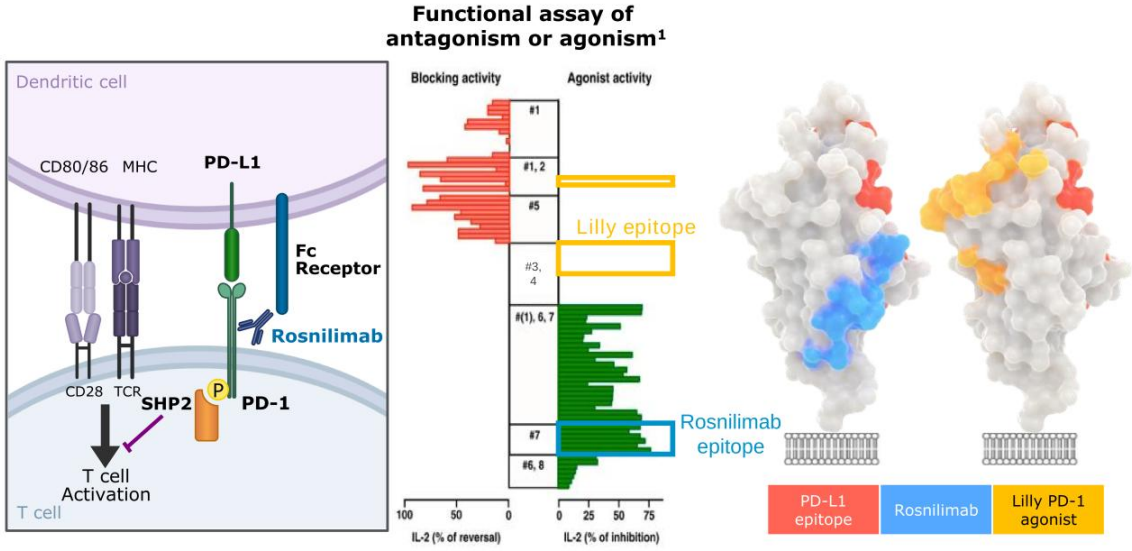
>80% of T cells in RA synovium are PD-1+

- 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"

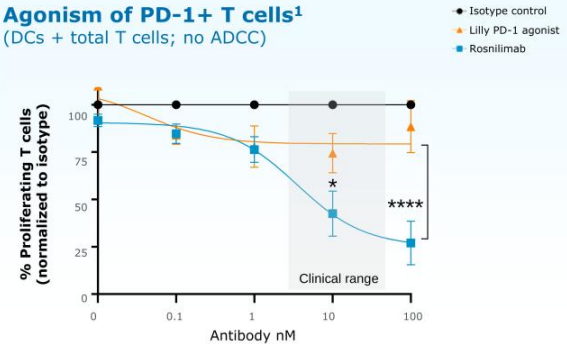
Suzuki, et al. 2023

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

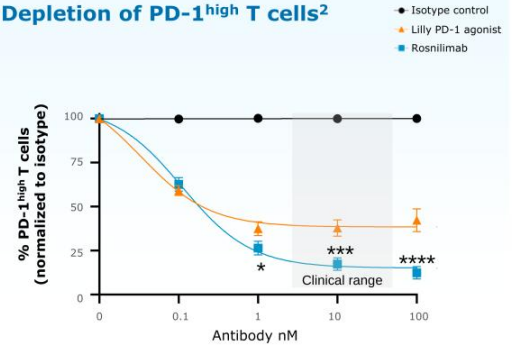
1. Adapted from Suzuki et al., Sci. Immunol. 8, eadd4947 (2023).

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

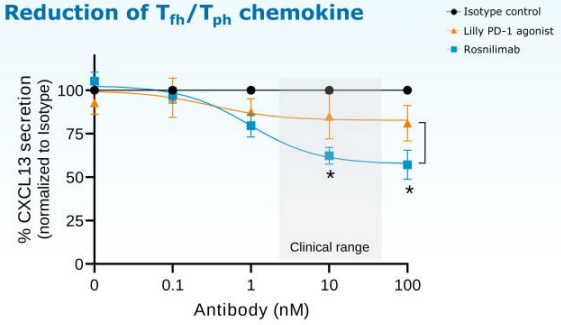
Agonism of PD-1+ T cells[1]
(DCs + total T cells; no ADCC)



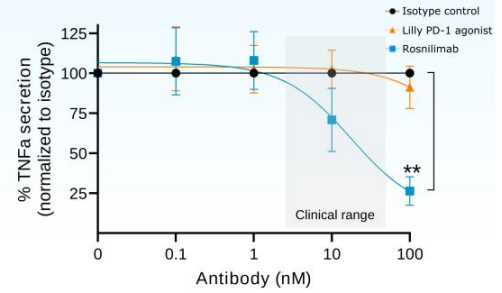
Depletion of PD-1^{high} T cells[2]



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



Reduction of inflammatory cytokine[3]



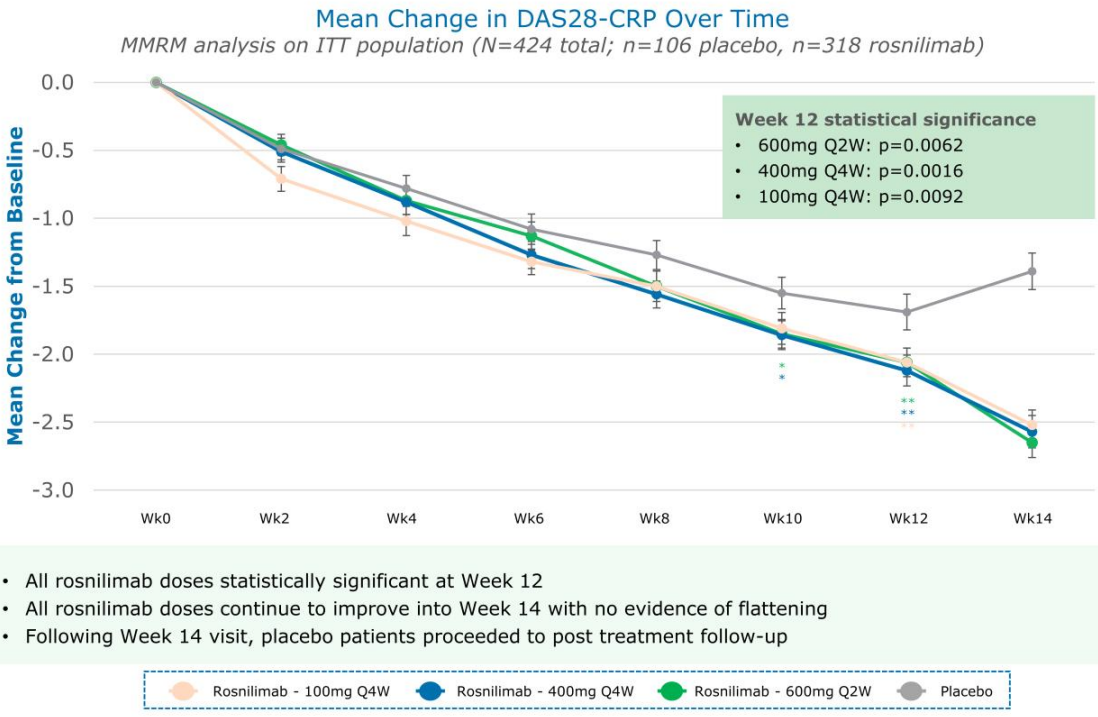
1. Healthy donor purified DCs + autologous total T cells stimulated with anti-CD3, cultured for 3 days for assessment of T cell proliferation
2. Anti-CD3+ anti-CD28 stimulation of RA patient PBMCs for assessment of depletion and agonism MOA, representative data from N=8 donors. Two-way ANOVA, Tukey's 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.

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

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

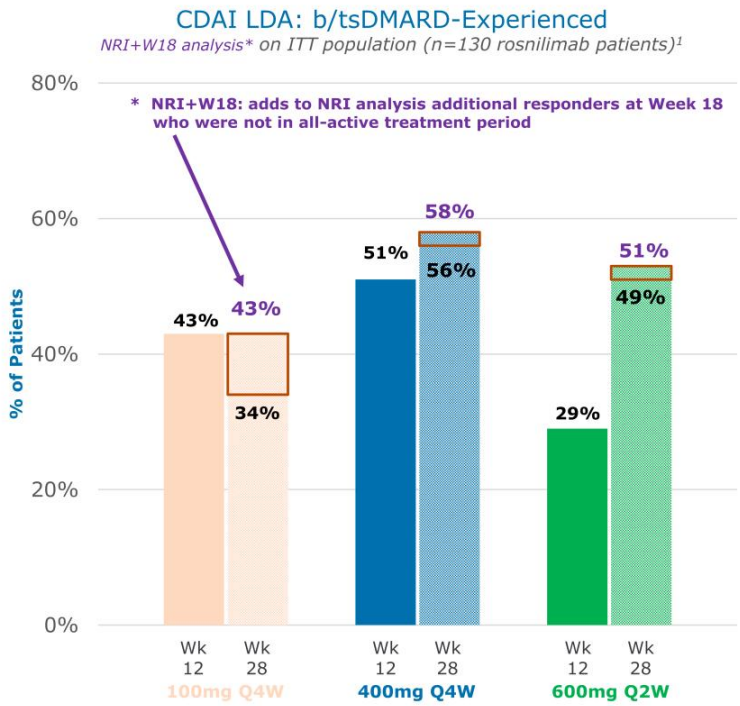


- All rosnilimab doses statistically significant at Week 12
- All rosnilimab doses continue to improve into Week 14 with no evidence of flattening
- Following Week 14 visit, placebo patients proceeded to post treatment follow-up

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

1. Mixed Model for Repeated Measures (MMRM) analysis on intent-to-treat (ITT) population; b/tsDMARD-naïve population (n=62 placebo, n=62 100mg Q4W, n=62 400mg Q4W, n=64 600mg Q2W); b/tsDMARD-experienced population (n=44 placebo, n=44 100mg Q4W, n=45 400mg Q4W, n=41 600mg Q2W); 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<0.01, *p<0.05, Standard error (SE) used to present figures of least squares mean changes from baseline.

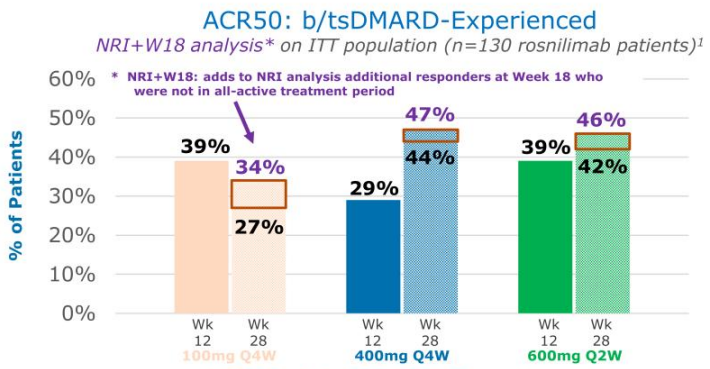
Demonstrated JAK-like CDAI LDA rates by 6 months

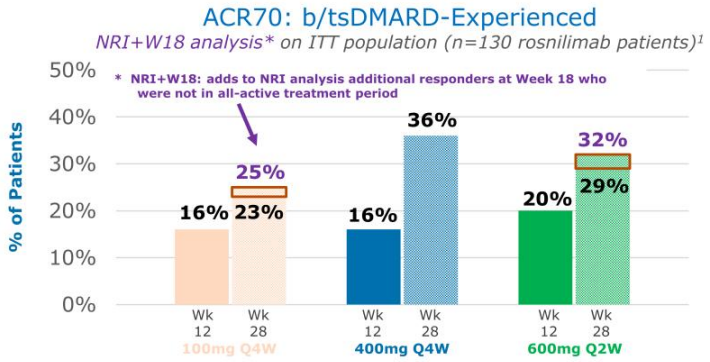


CDAI LDA at Week 28		
Arm	NRI	NRI+W18
b/tsDMARD-Experienced Population *(as graphed)*		
100mg	34%	43%
400mg	56%	58%
600mg	49%	51%
b/tsDMARD-Naïve Population		
100mg	66%	71%
400mg	53%	55%
600mg	72%	75%

1. Non-responder imputated (NRI) analysis on intent-to-treat (ITT) of all b/tsDMARD-naïve patients randomized; b/tsDMARD-experienced population (n=44 placebo, n=44 100mg Q4W, n=45 400mg Q4W, n=41 600mg Q2W; n=130 total rosnilimab b/tsDMARD-experienced patients)

Demonstrated JAK-like ACR70 rates which deepened into 6 months





ACR50 at Week 28		
Arm	NRI	NRI+W18
b/tsDMARD-Experienced Population *(as graphed)*		
100mg	27%	34%
400mg	44%	47%
600mg	42%	46%
b/tsDMARD-Naïve Population		
100mg	58%	61%
400mg	52%	53%
600mg	69%	75%

ACR70 at Week 28		
Arm	NRI	NRI+W18
b/tsDMARD-Experienced Population *(as graphed)*		
100mg	23%	25%
400mg	36%	36%
600mg	29%	32%
b/tsDMARD-Naïve Population		
100mg	53%	55%
400mg	37%	37%
600mg	55%	58%

1. Non-responder imputated (NRI) analysis on intent-to-treat (ITT) of all b/tsDMARD-naïve patients randomized; b/tsDMARD-experienced population (n=44 placebo, n=44 100mg Q4W, n=45 400mg Q4W, n=41 600mg Q2W; n=130 total rosnilimab b/tsDMARD-experienced patients)