Rosnilimab: Updated Phase 2b Data in RA

June 3, 2025

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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

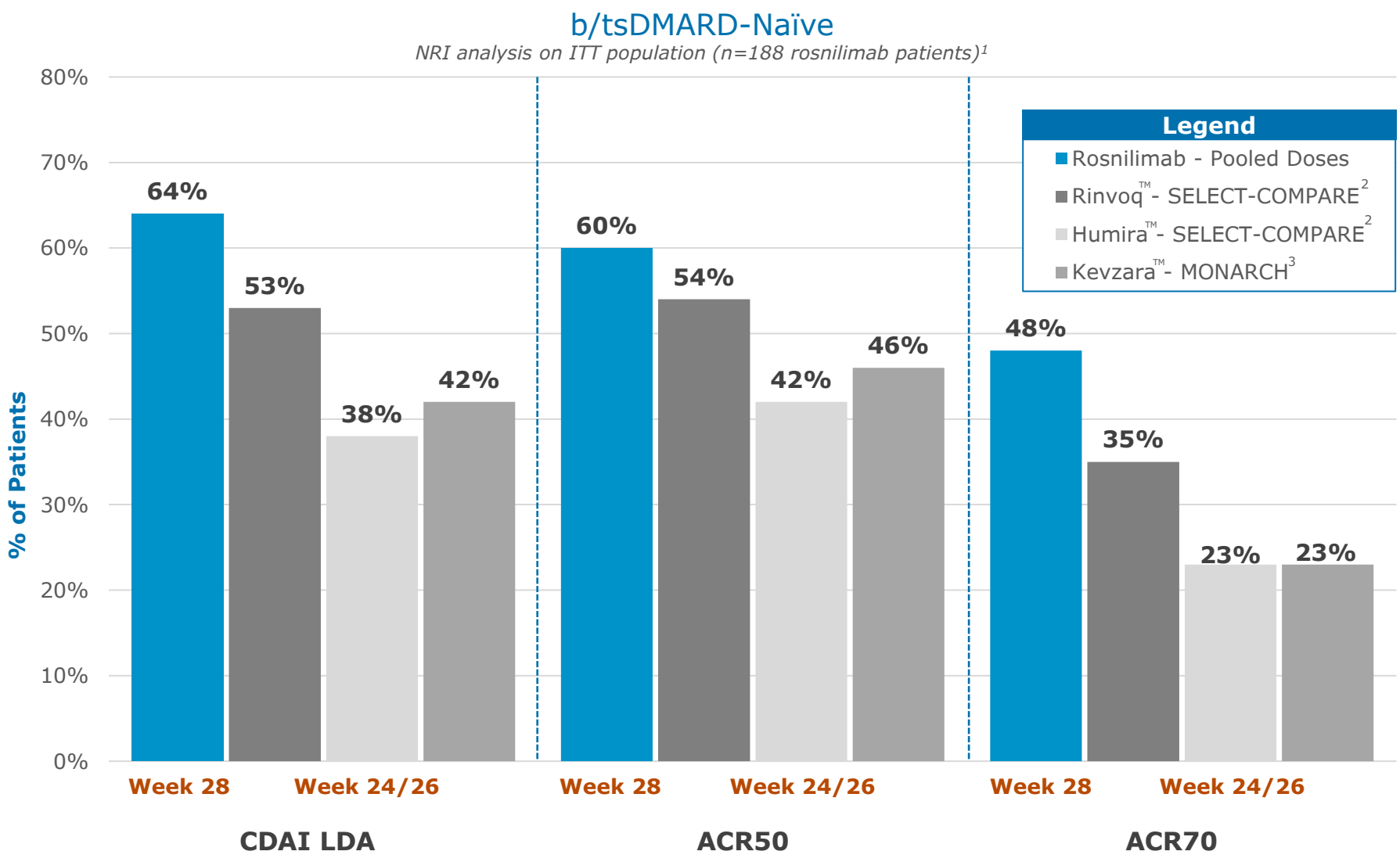
Responses durable after 6 months

- 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

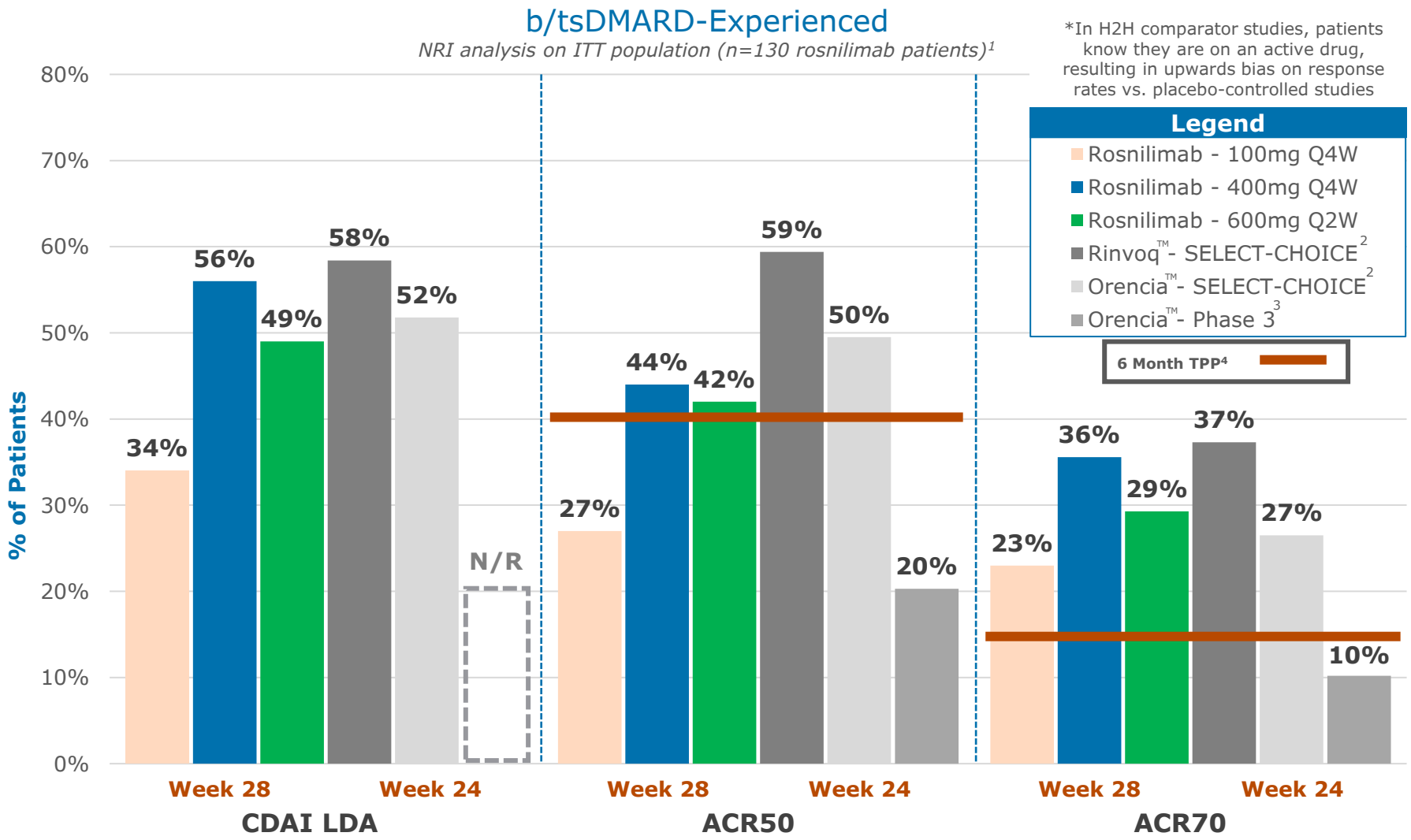
Compares favorably despite most conservative analysis and capped trial design



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*

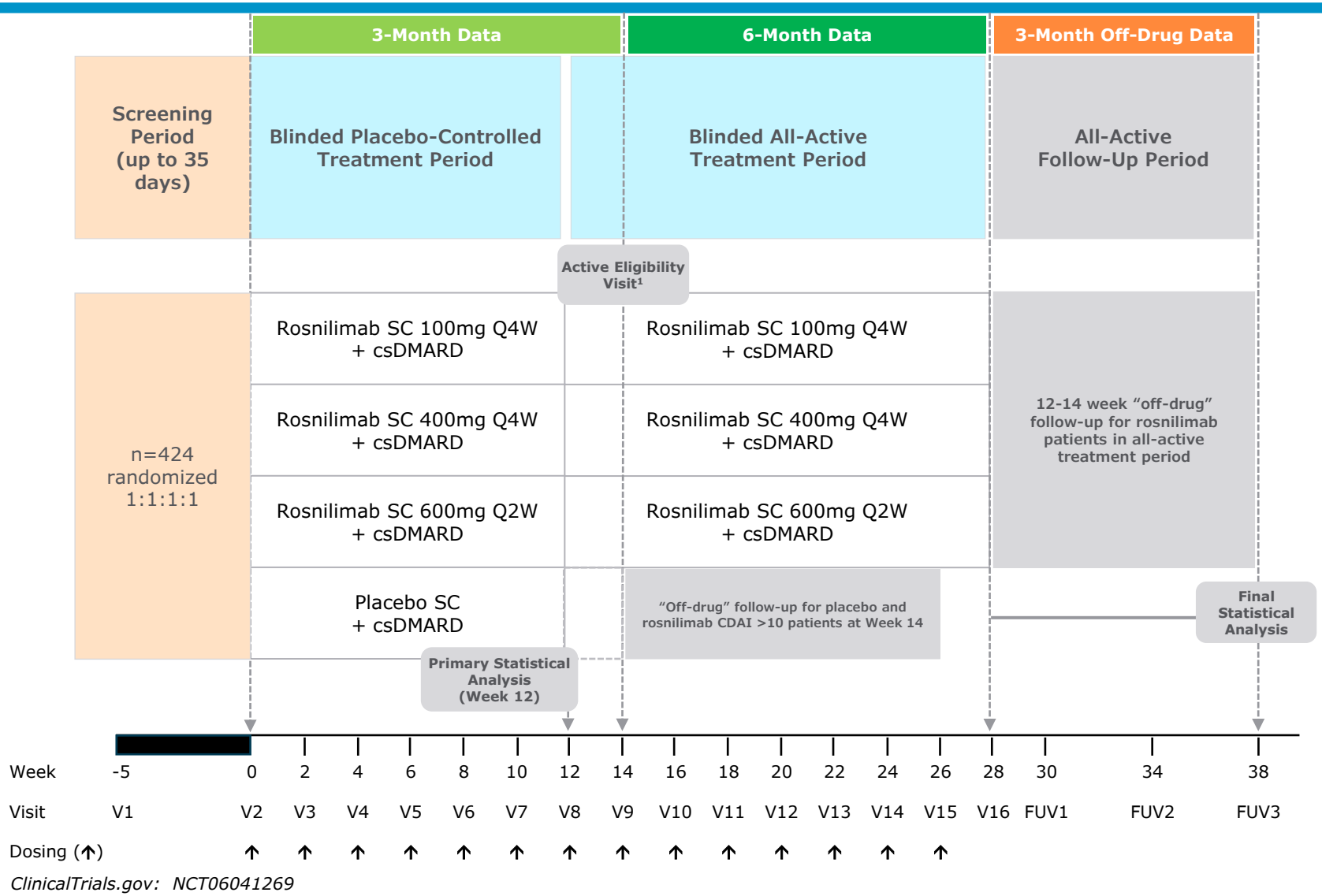
Includes 29% with prior JAK experience



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



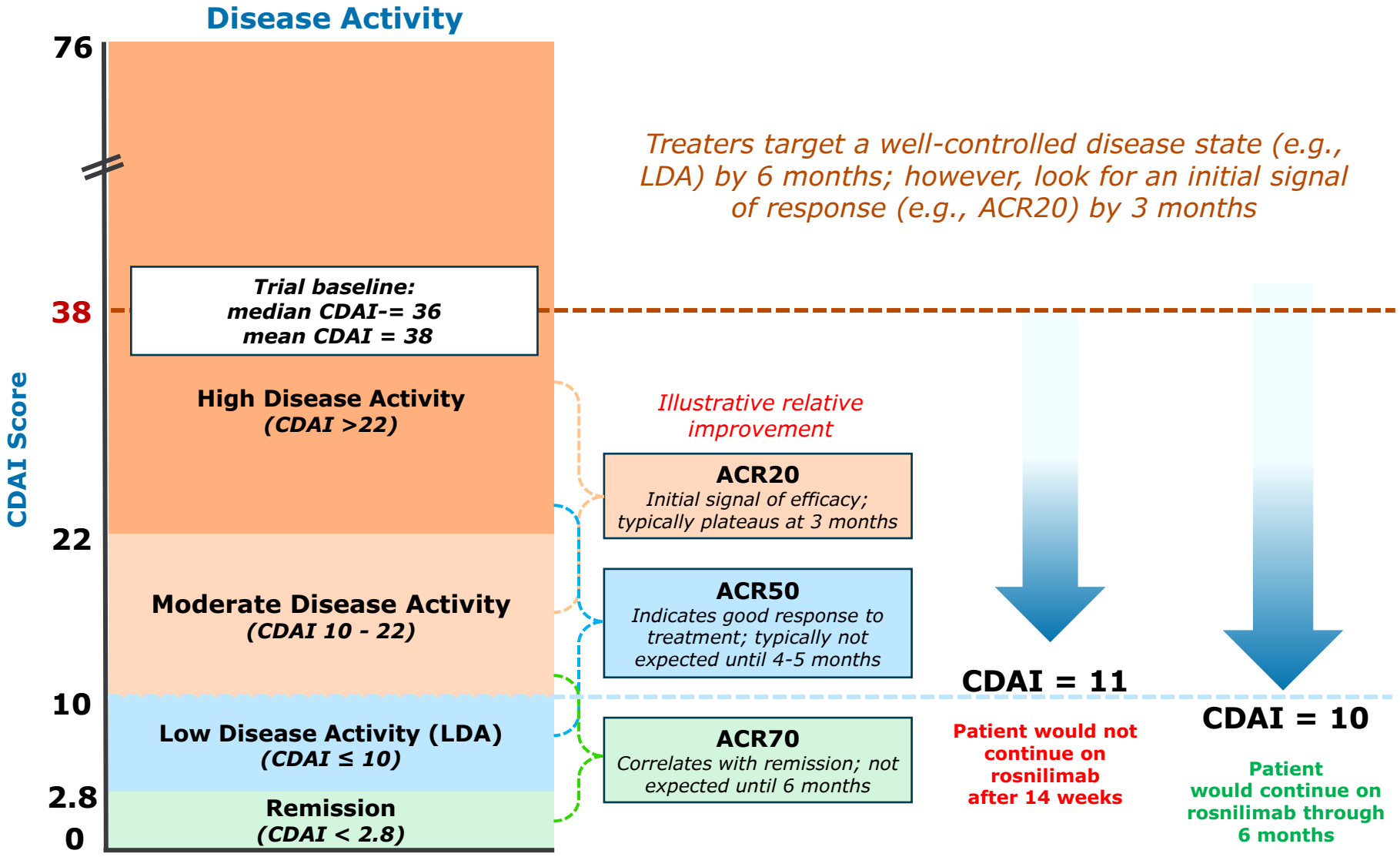
ClinicalTrials.gov: NCT06041269

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

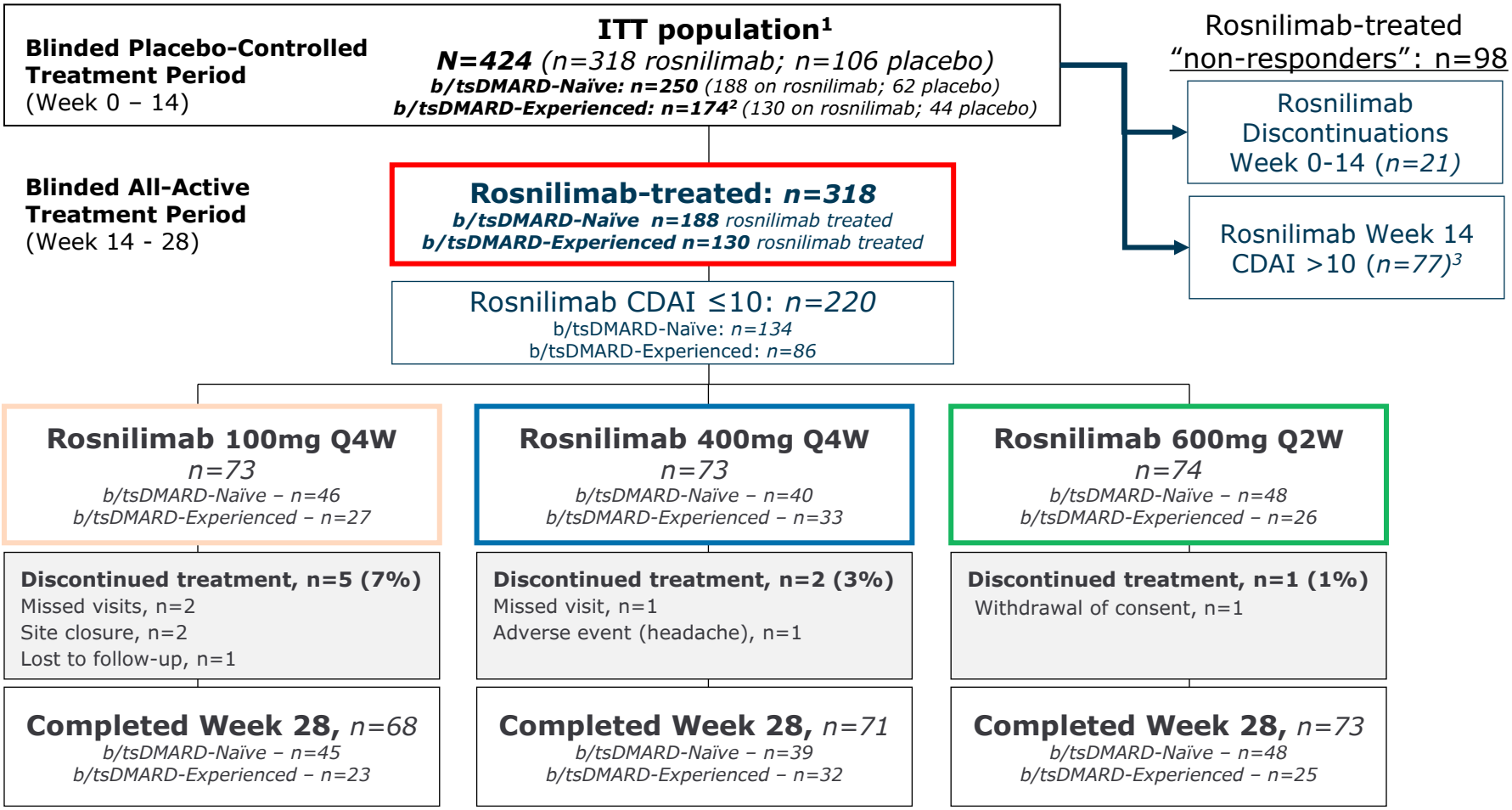
95% of trial participants had high disease activity (CDAI > 22) at baseline



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)



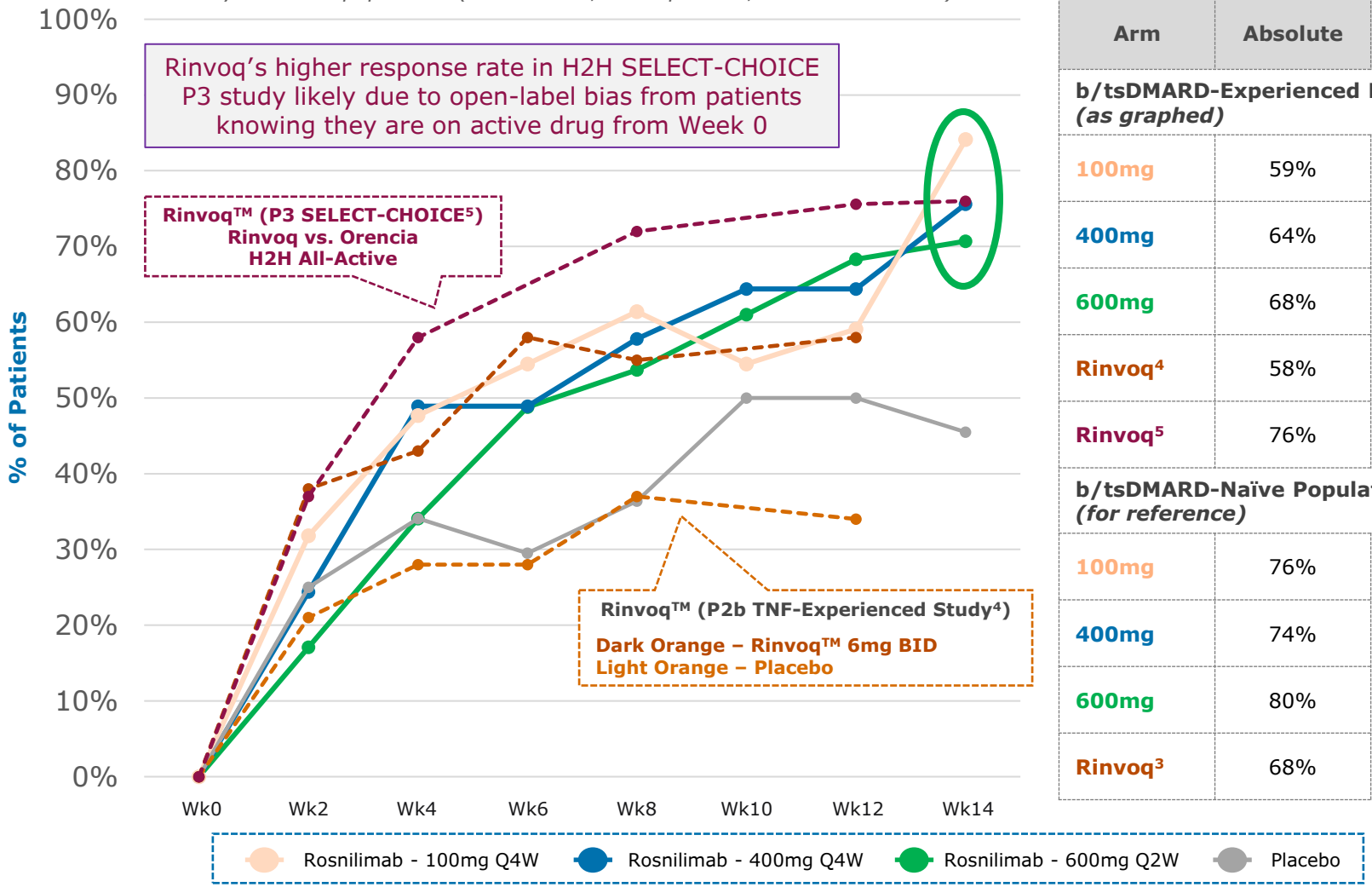
1. Non-responder imputated (NRI) analysis on intent-to-treat (ITT) population; b/tsDMARD-naïve population (n=62 placebo, n=62 100mg Q4W, n=62 400mg Q4W, n=64 600mg Q2W); b/tsDMARD-experienced population (n=44 placebo, n=44 100mg Q4W, n=45 400mg Q4W, n=41 600mg Q2W); 2. b/tsDMARD-experienced population 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]



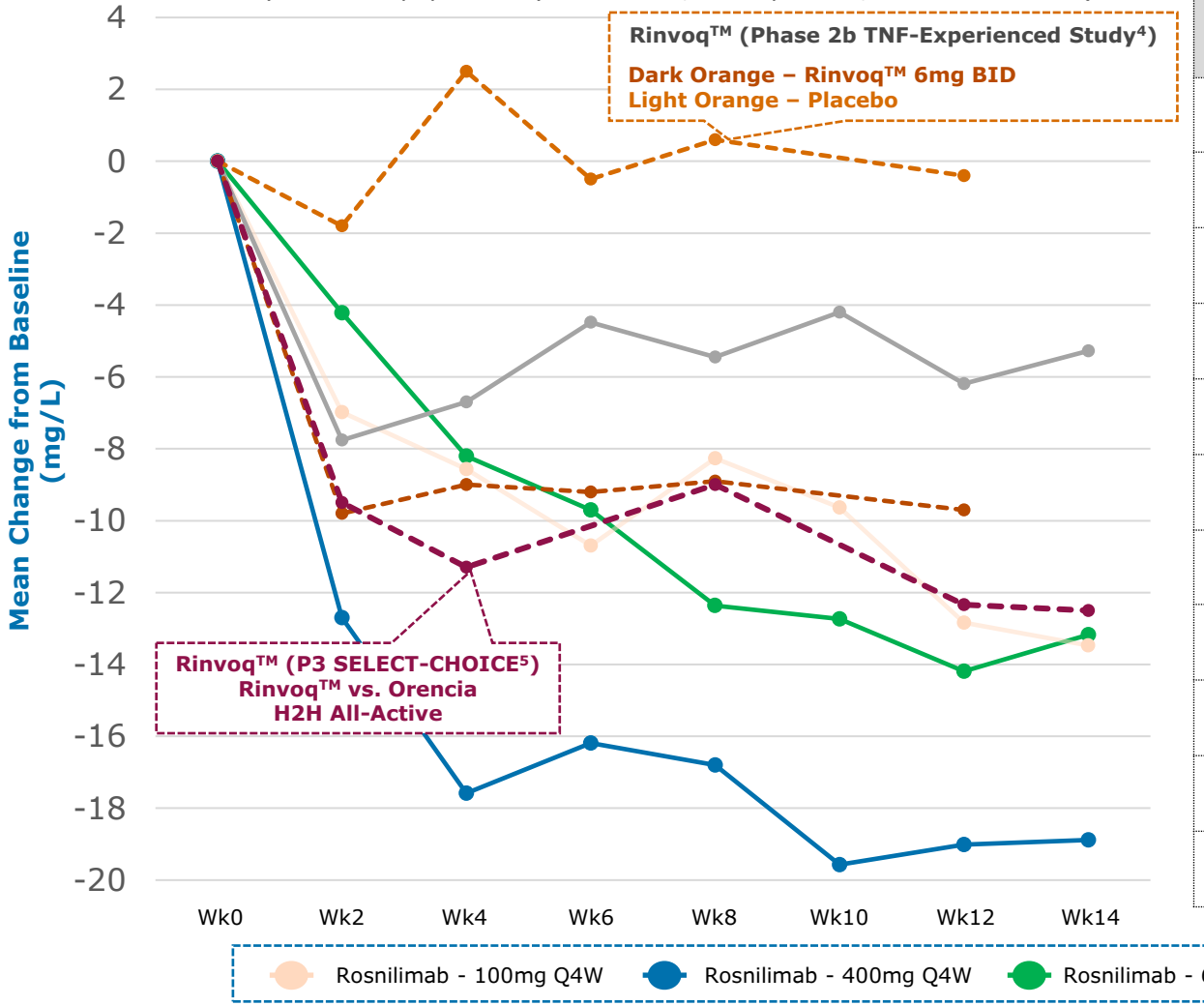
ACR20 at Week 12		
Arm	**Absolute**	**PBO Adjusted**
b/tsDMARD-Experienced Population *(as graphed)*		
100mg	59%	*9%*
400mg	64%	*14%*
600mg	68%	*18%*
Rinvoq[4]	58%	*24%*
Rinvoq[5]	76%	*N/A*
b/tsDMARD-Naïve Population *(for reference)*		
100mg	76%	*21%*
400mg	74%	*19%*
600mg	80%	*25%*
Rinvoq[3]	68%	*22%*

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

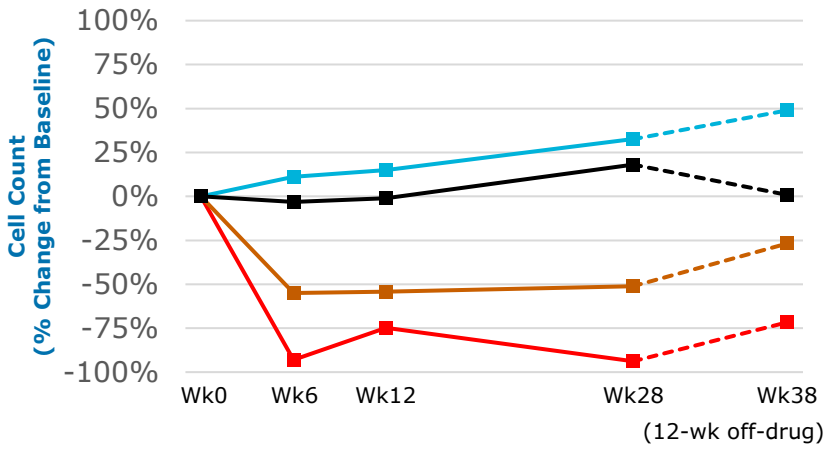
KOL Point of View

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

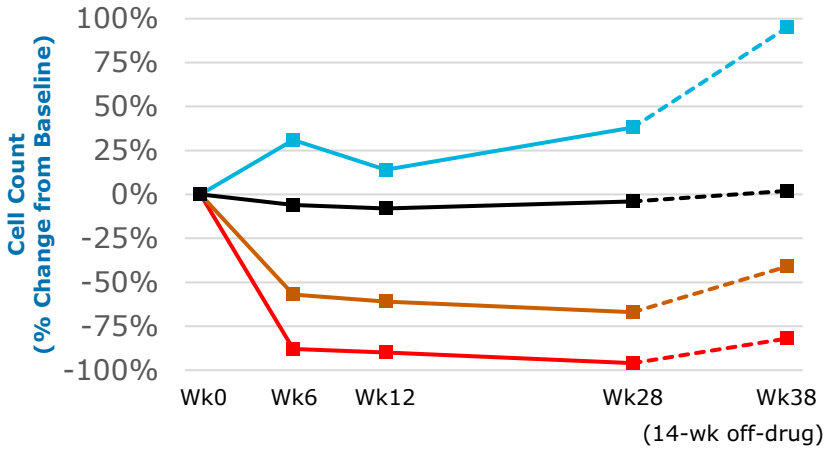
Jonathan Graf, M.D.
Professor of Medicine, Rheumatology, University of California, San Francisco; RENOIR investigator

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

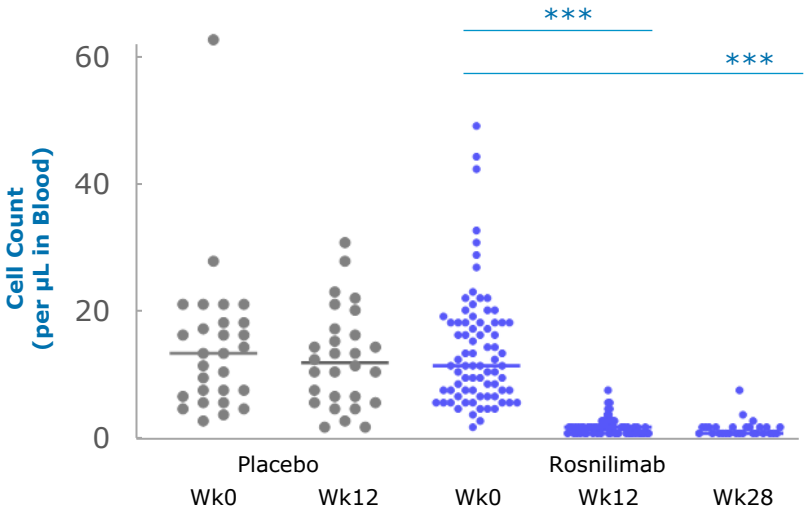
Rosnilimab 400mg Q4W T Cell Impact



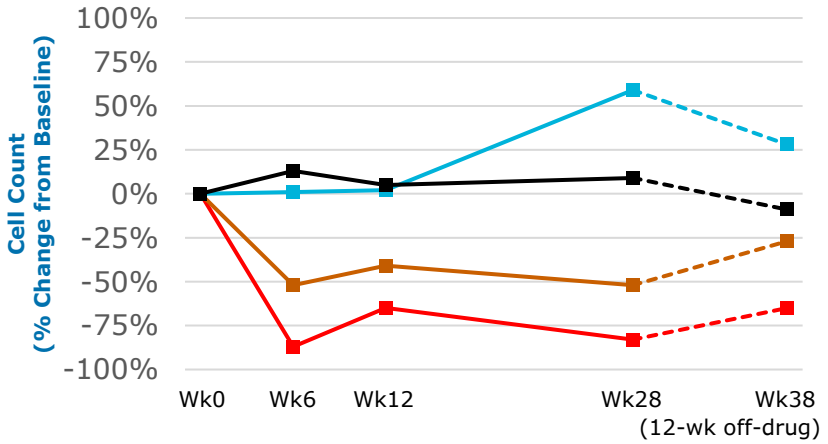
Rosnilimab 600mg Q2W T Cell Impact



Rosnilimab T_{ph} Impact – Pooled Doses

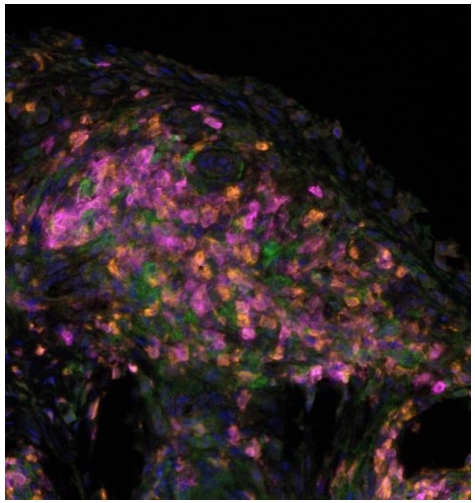
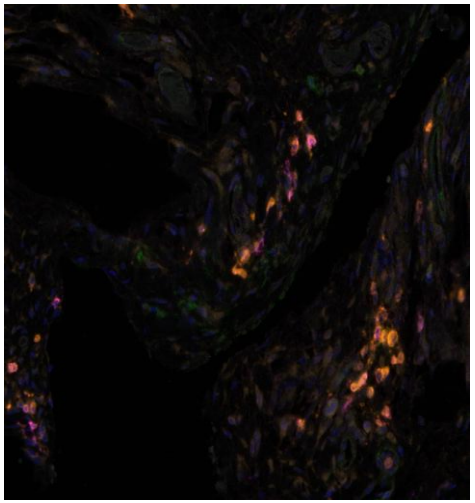
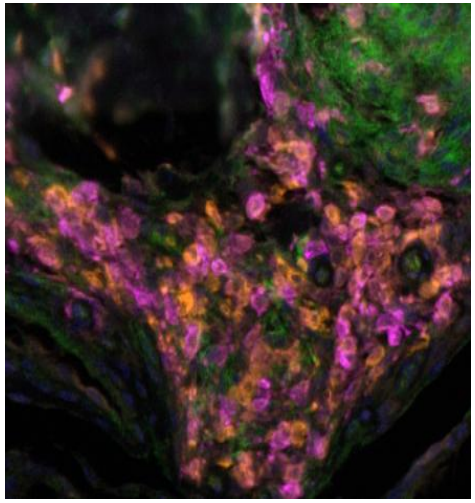
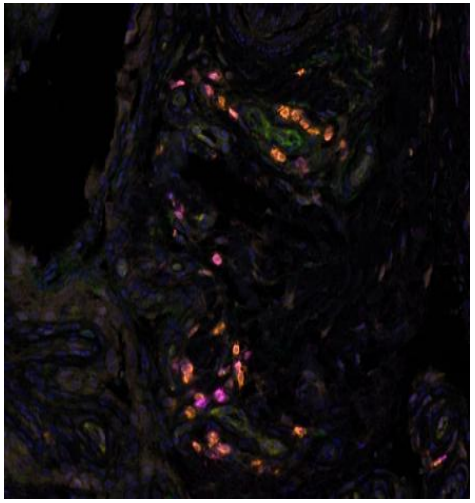


Rosnilimab 100mg Q4W T Cell Impact



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

	Baseline	Week 6
Rosnilimab 400mg Q4W		
Rosnilimab 600mg Q2W		

Impact on PD-1+ T cells
(T_{ph} and T_{eff})

400mg/600mg doses
~90% reduction

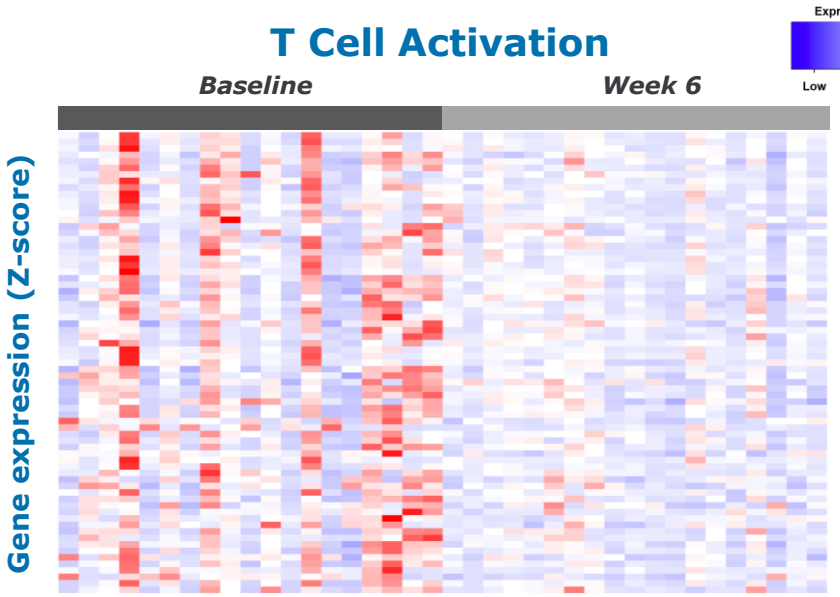
100mg dose
Inconclusive reduction

Placebo
Increased

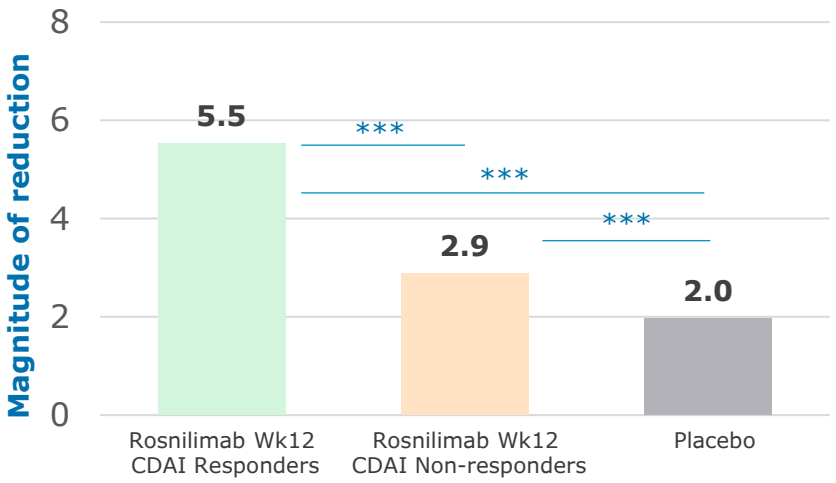
T cell markers	APC markers
CD3	PD-L1
PD-1	
CXCR5	

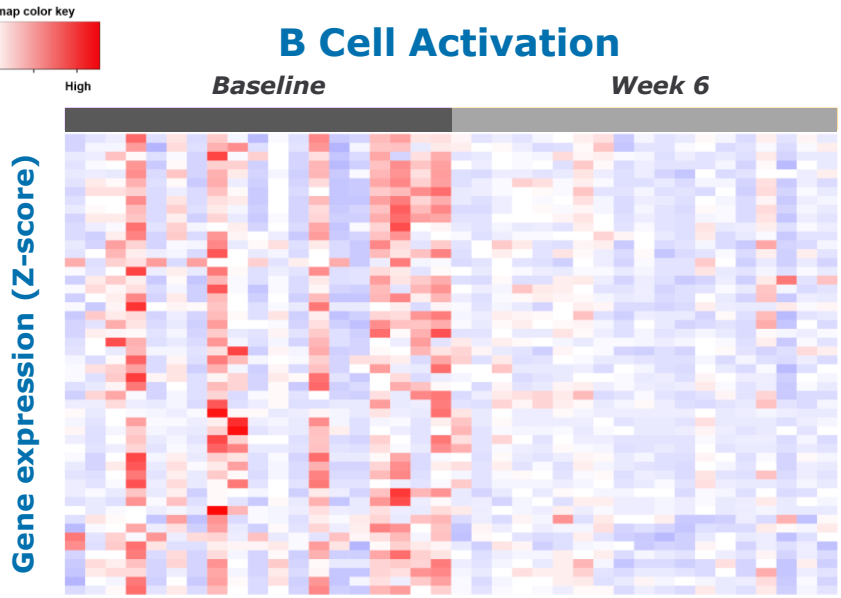
Note: Synovial biopsies of the most impacted joint taken at baseline and 6 weeks on study. Immunofluorescence performed to identify PD-1 positive cells. T_{ph} cells (PD-1+CD3+CD4+CXCR5-)

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

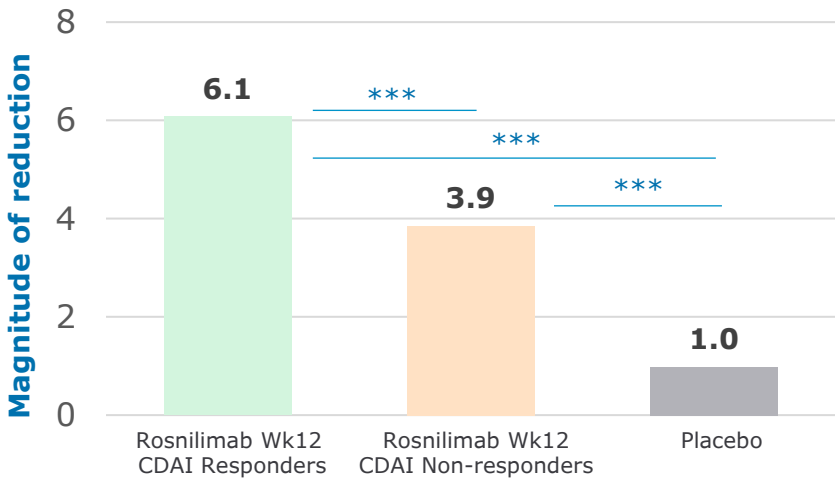


Magnitude of reduction: 4.03, p=1.09e-24



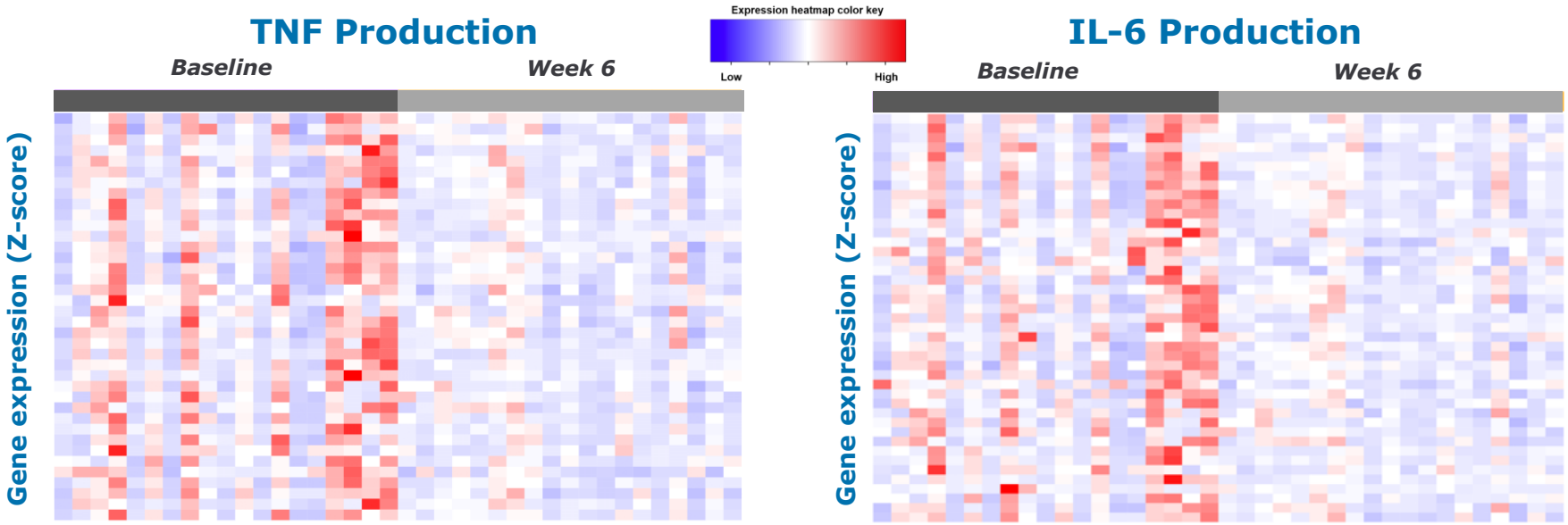


Magnitude of reduction: 4.06, p=1.77e-18



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.

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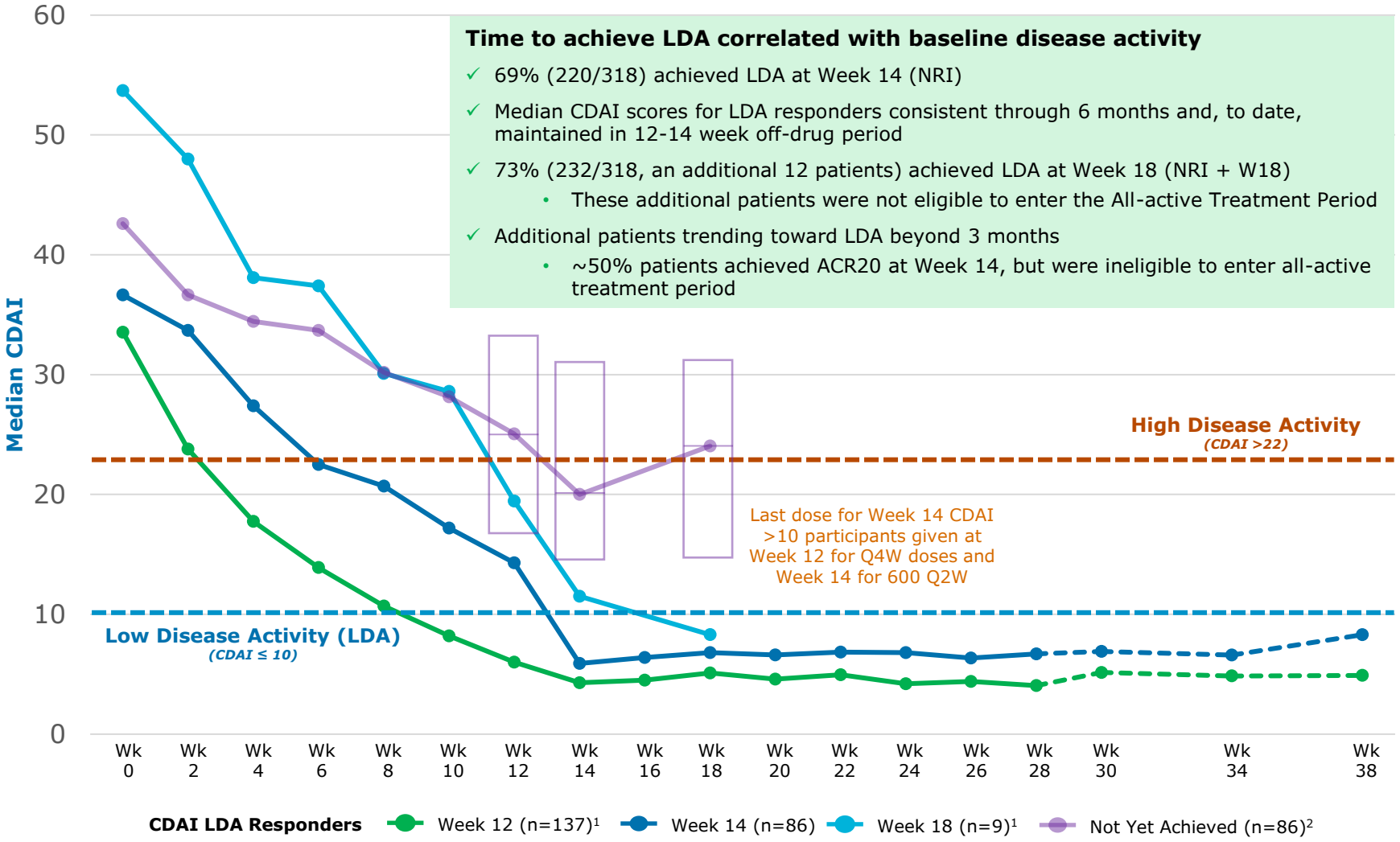
Max response was not achieved in this Phase 2b trial

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)

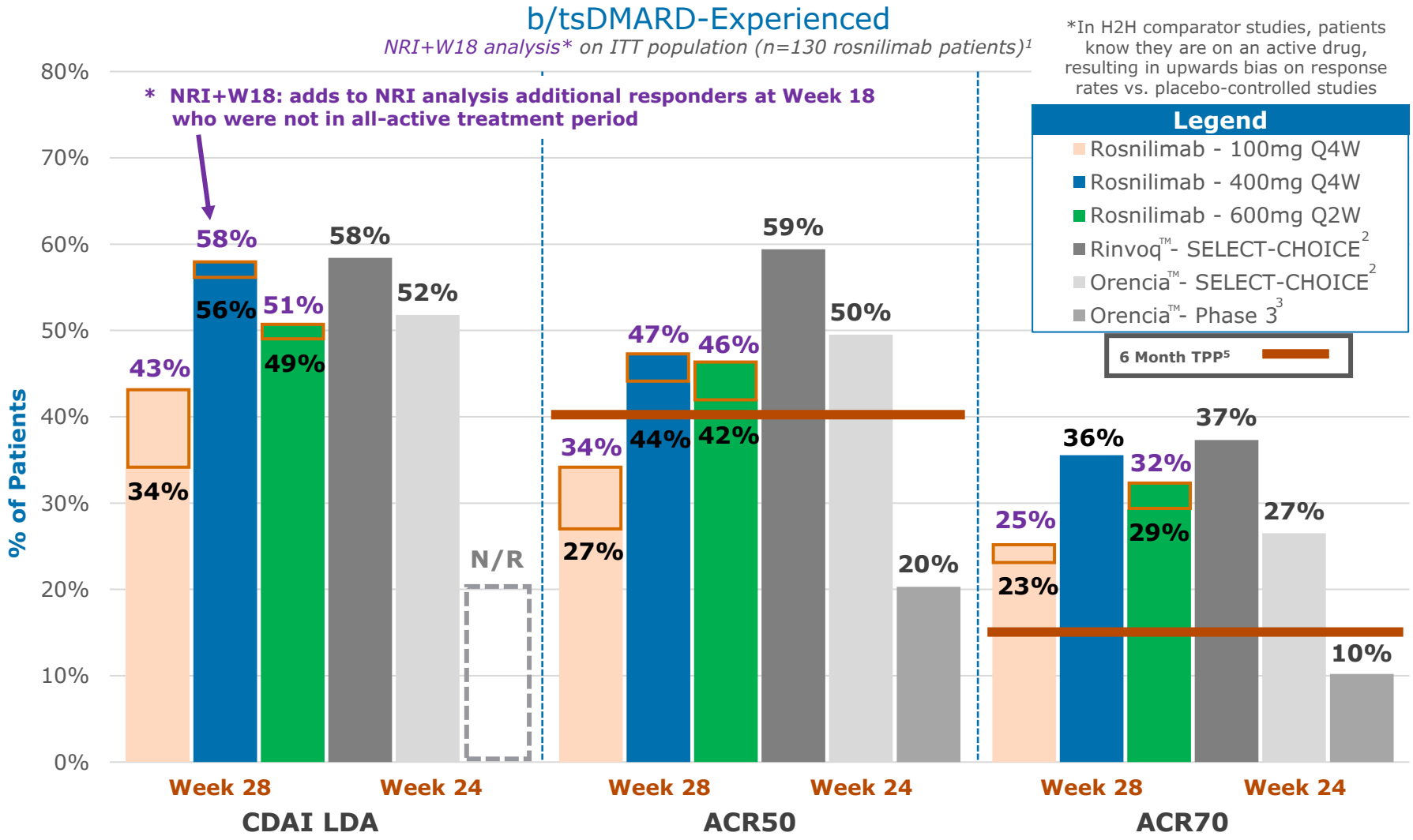


1. Green line includes 3 patients that achieved LDA at Week 12, were not CDAI LDA at Week 14, but returned to CDAI LDA at Week 18. These same 3 patients were excluded from the Light Blue line. In total 12 patients achieved CDAI LDA at Week 18. 2. Purple line includes rosnilimab patients that discontinued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

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

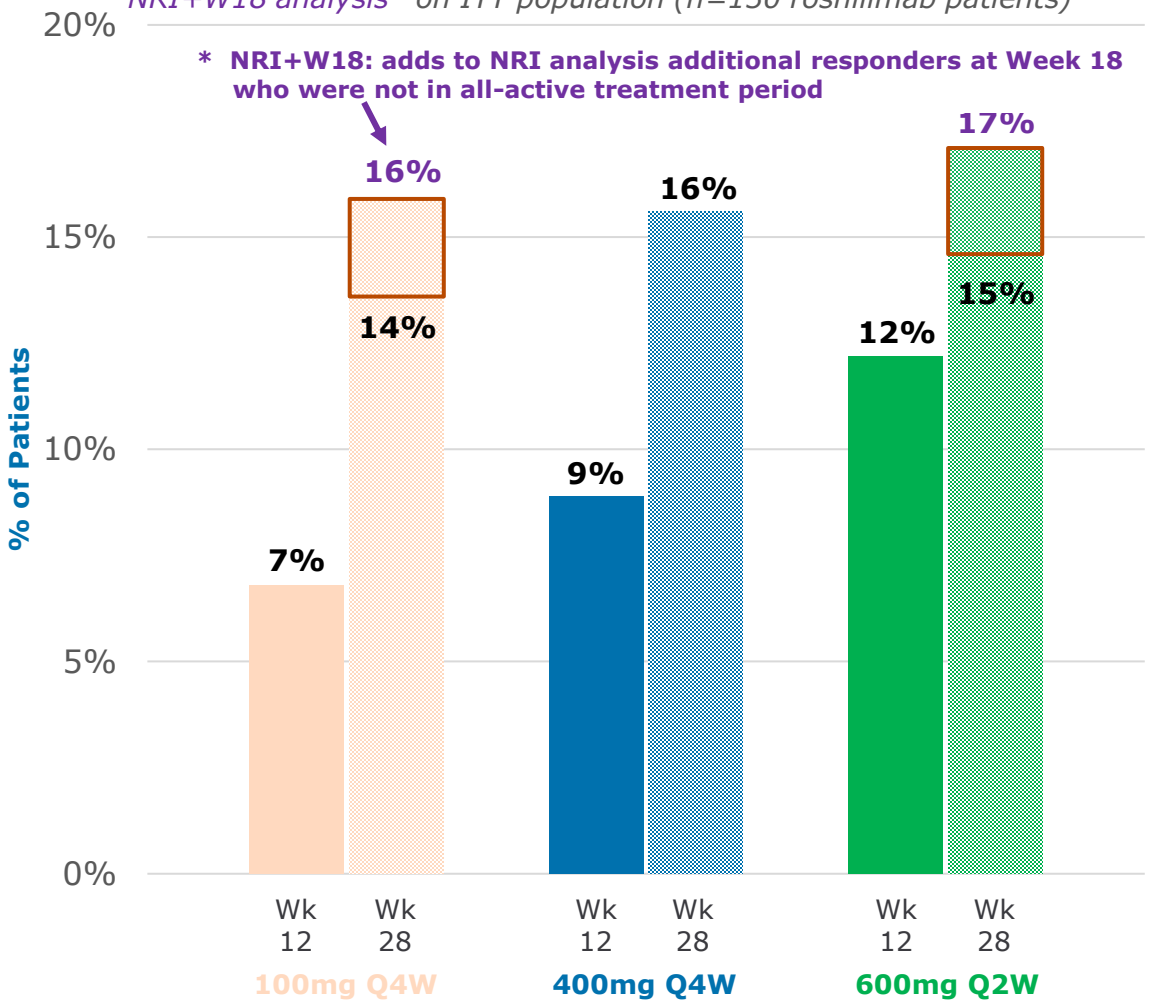
JAK-like CDAI remission rates which deepened into six months

Includes 29% with prior JAK experience

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

CDAI Remission: b/tsDMARD-Experienced

NRI+W18 analysis on ITT population (n=130 rosnilimab patients)*[1]



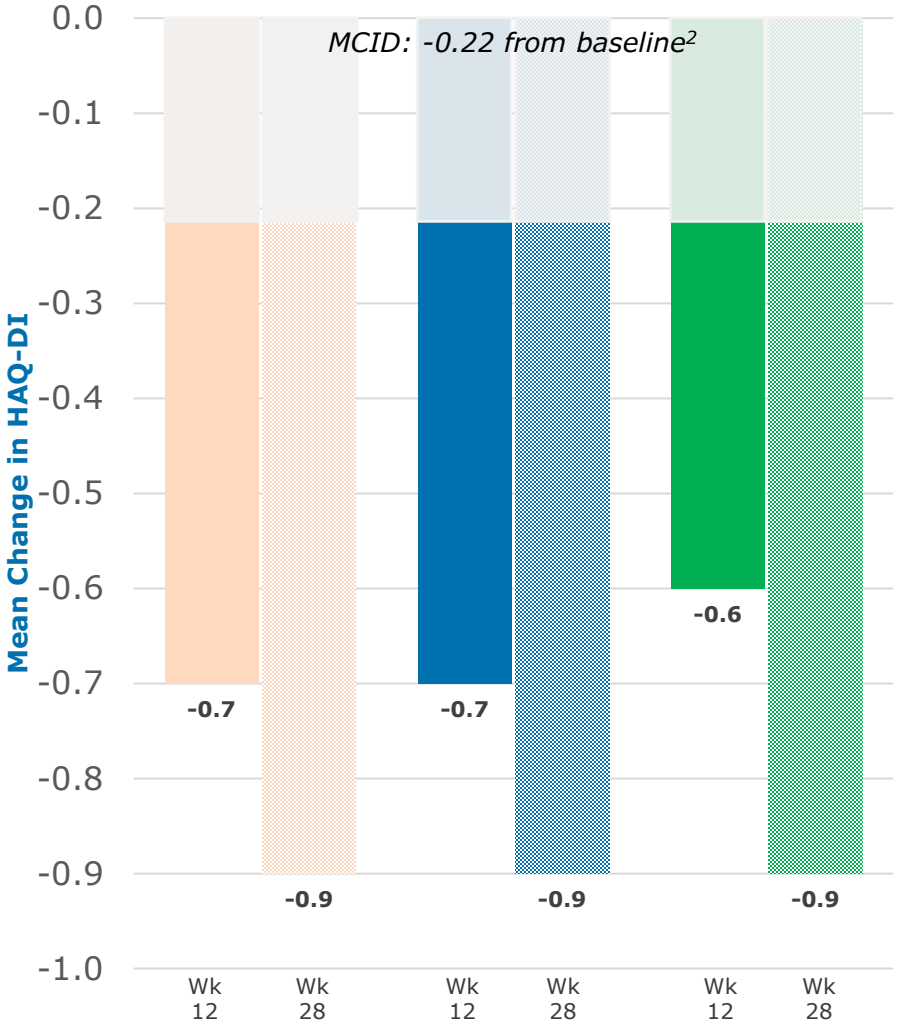
CDAI Remission at Week 28		
Arm	**NRI**	**NRI+W18**
b/tsDMARD-Experienced Population *(as graphed)*		
100mg	14%	16%
400mg	16%	16%
600mg	15%	17%
b/tsDMARD-Naïve Population		
100mg	21%	21%
400mg	18%	18%
600mg	17%	19%

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

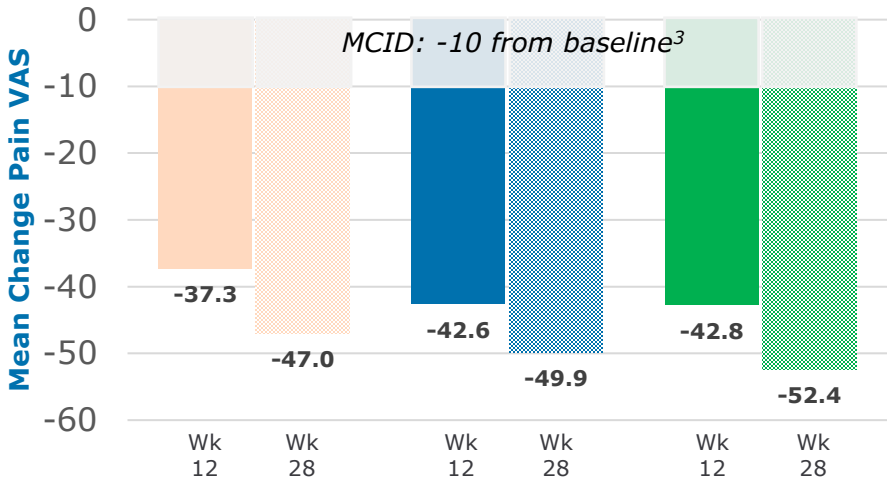
HAQ-DI: Overall

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



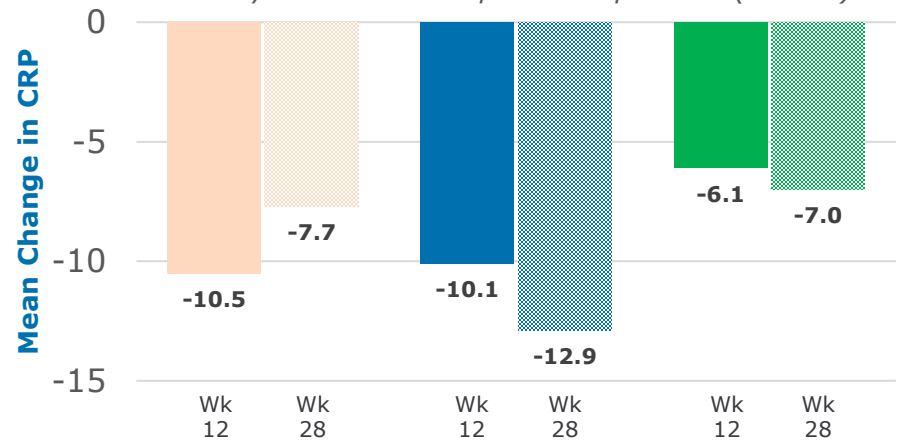
Pain VAS: Overall

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

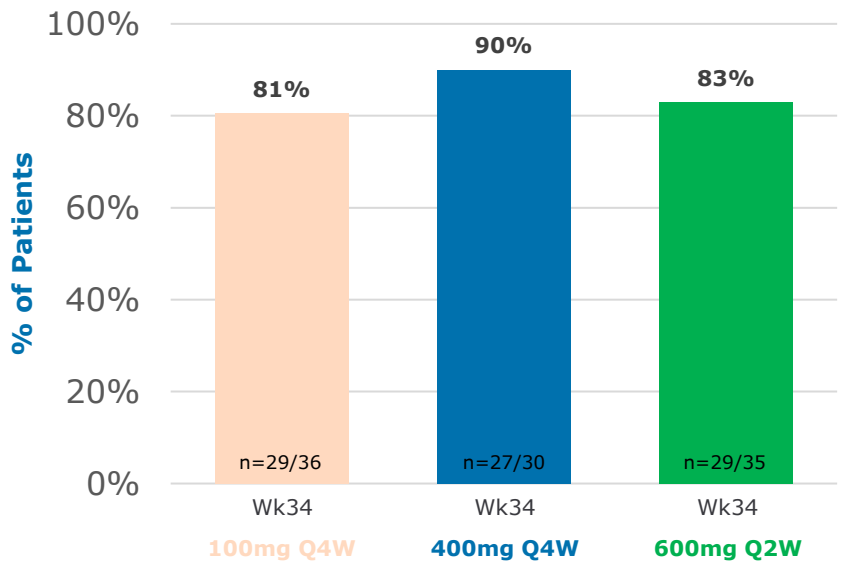
1. Mixed Model for Repeated Measures (MMRM) analysis on rosnilimab CDAI LDA responder at Week 14 population (n=220) includes naïve population (n=46 100mg Q4W, n=40 400mg Q4W, n=48 600mg Q2W; n=134 total rosnilimab patients) and experienced population (n=27 100mg Q4W, n=33 400mg Q4W, n=26 600mg Q2W; n=86 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

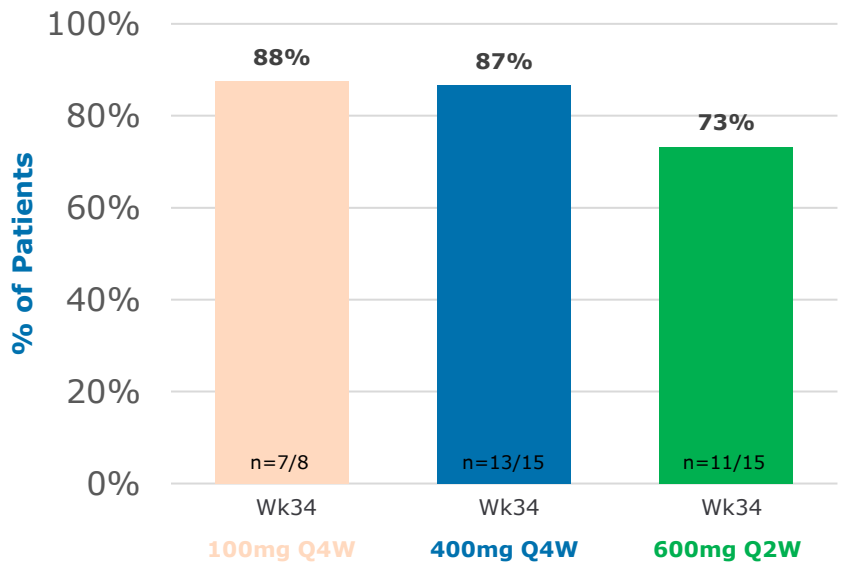
CDAI LDA: b/tsDMARD-Naïve

Week 34 completer analysis (n=101)[1]



CDAI LDA: b/tsDMARD-Experienced

Week 34 completer analysis (n=38)[1]



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:

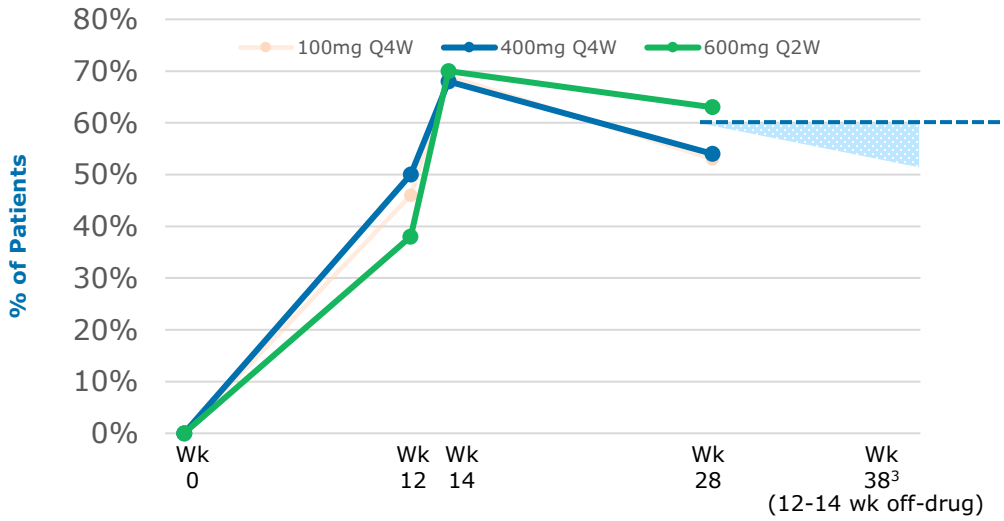
- 25% (6/23) were CDAI <11
- Median CDAI = 13
- 91% (21/23) were CDAI <22 (e.g. remained CDAI moderate disease activity)

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

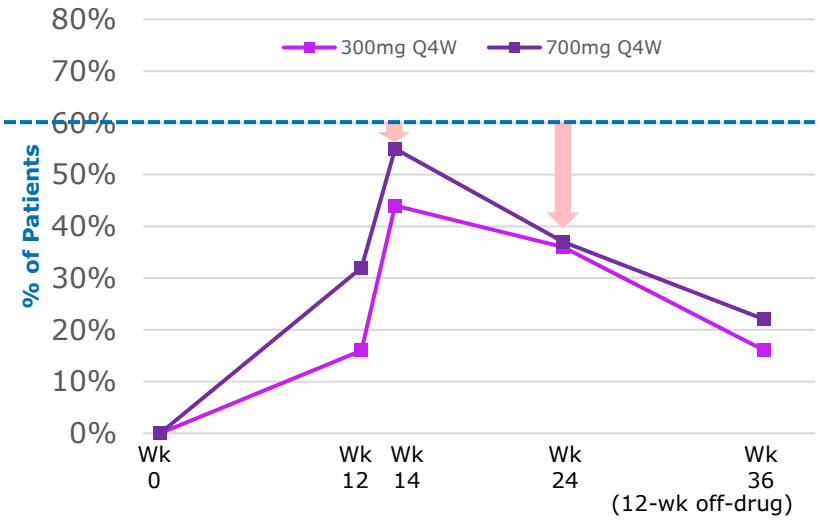
Rosnilimab P2b: CDAI LDA

NRI analysis on ITT population (n=318 rosnilimab patients)[1,2]



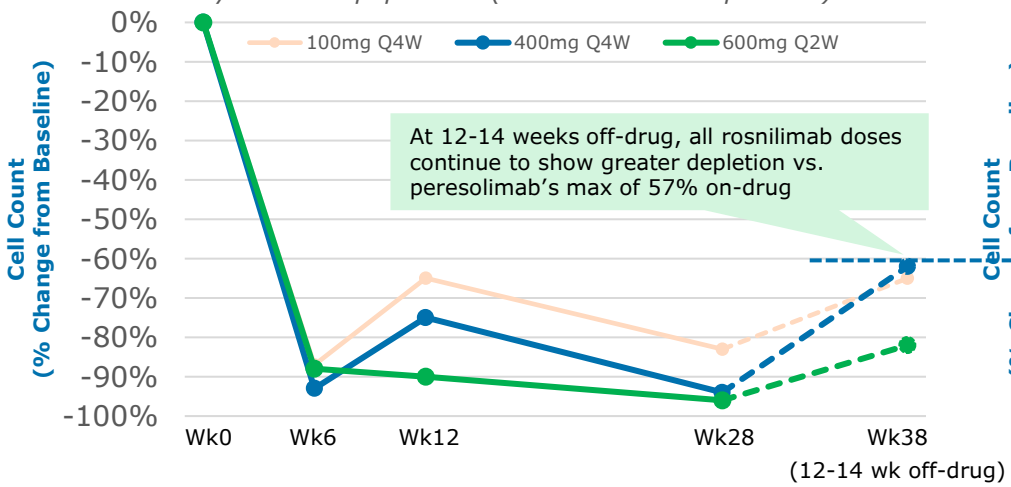
Peresolimab P2a: CDAI LDA

NRI analysis on ITT population (n=74)[4]



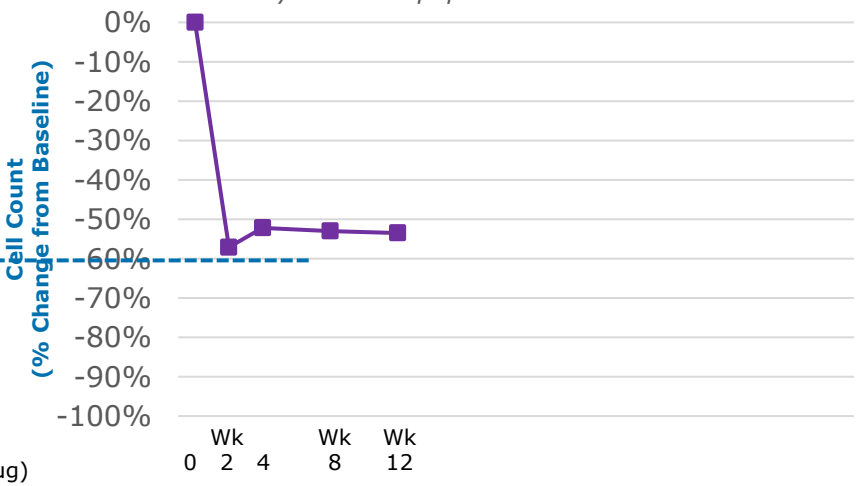
Rosnilimab P2b: PD-1^{high} T Cell Impact

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



Peresolimab P2a: PD-1^{high} T Cell Impact

NRI analysis on ITT population – Pooled doses[4]



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

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

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

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

2x
Infection Rate[1]

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

2x
Malignancy Rate[3]

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

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

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

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

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

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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)

P2b in Rheumatoid Arthritis

P2 in Ulcerative Colitis

ANB033
(CD122 antagonist)

P1 in Healthy Volunteers

ANB101
(BDCA2 modulator)

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
  - GSK $75MM milestone for *Jemperli* $1B annual WW sales
  - Vanda royalty and milestone potential for imsidolimab

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

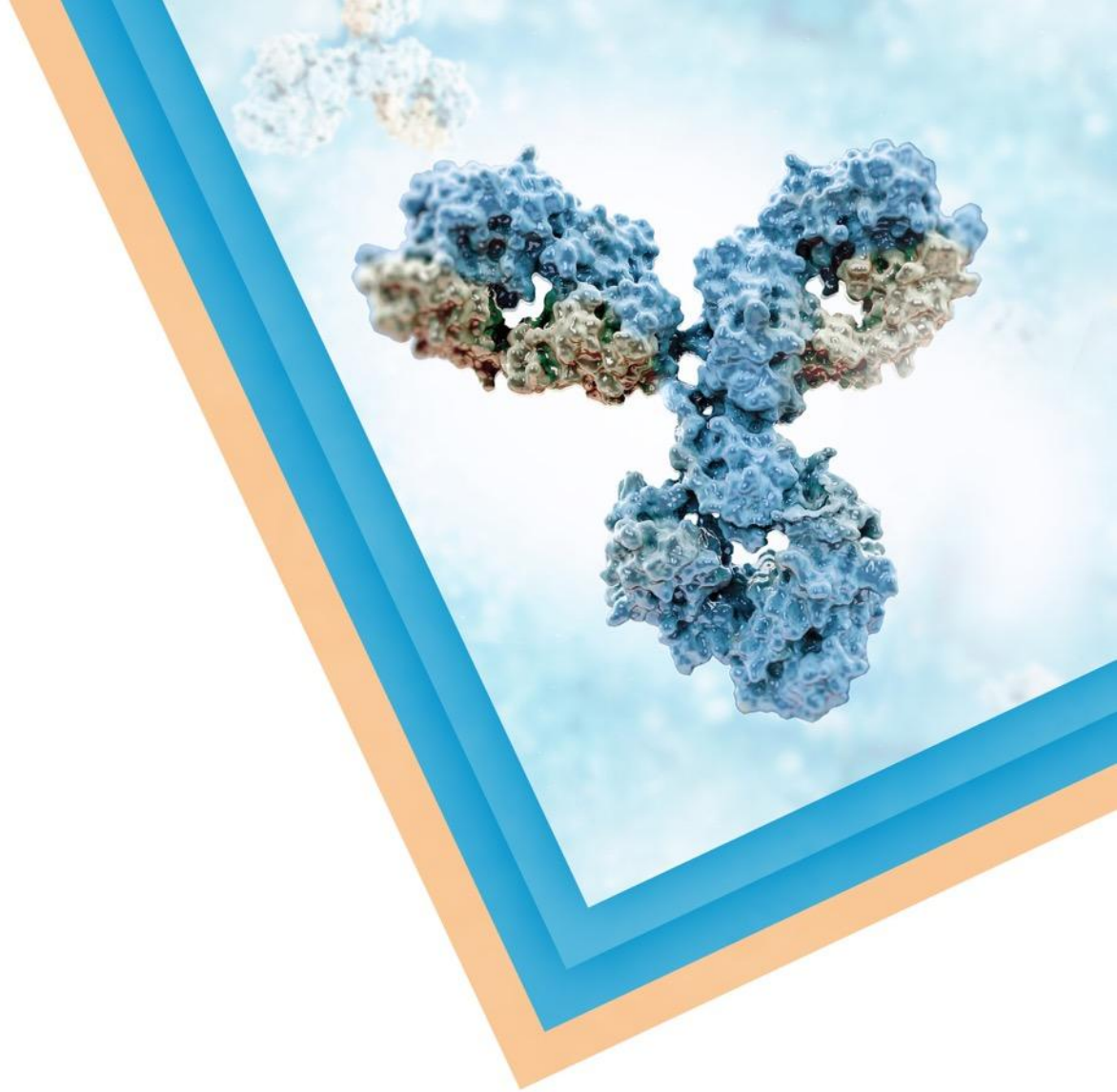
3

Responses durable after 6 months

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

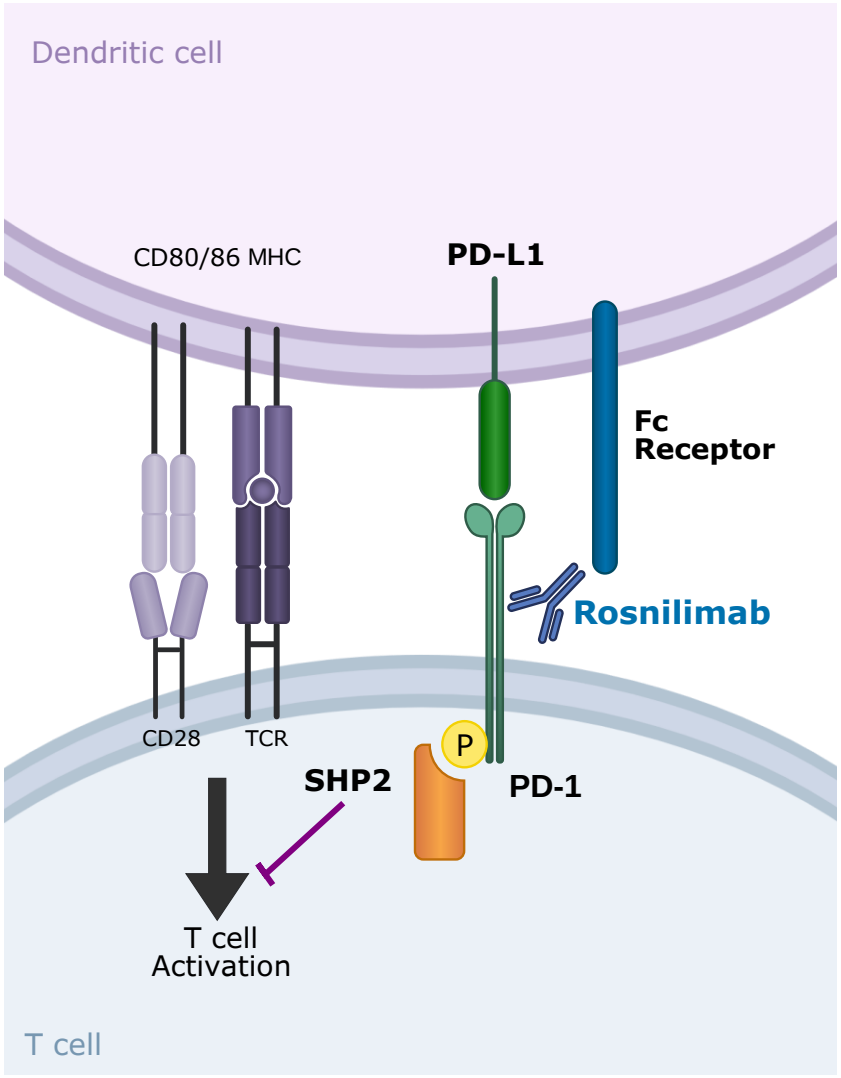
Rosnilimab, a best-in-class depleter and agonist targeting PD-1+ T cells, is well-positioned for the ~$20 billion U.S. RA market which hasn't had a new mechanism approved since 2012

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

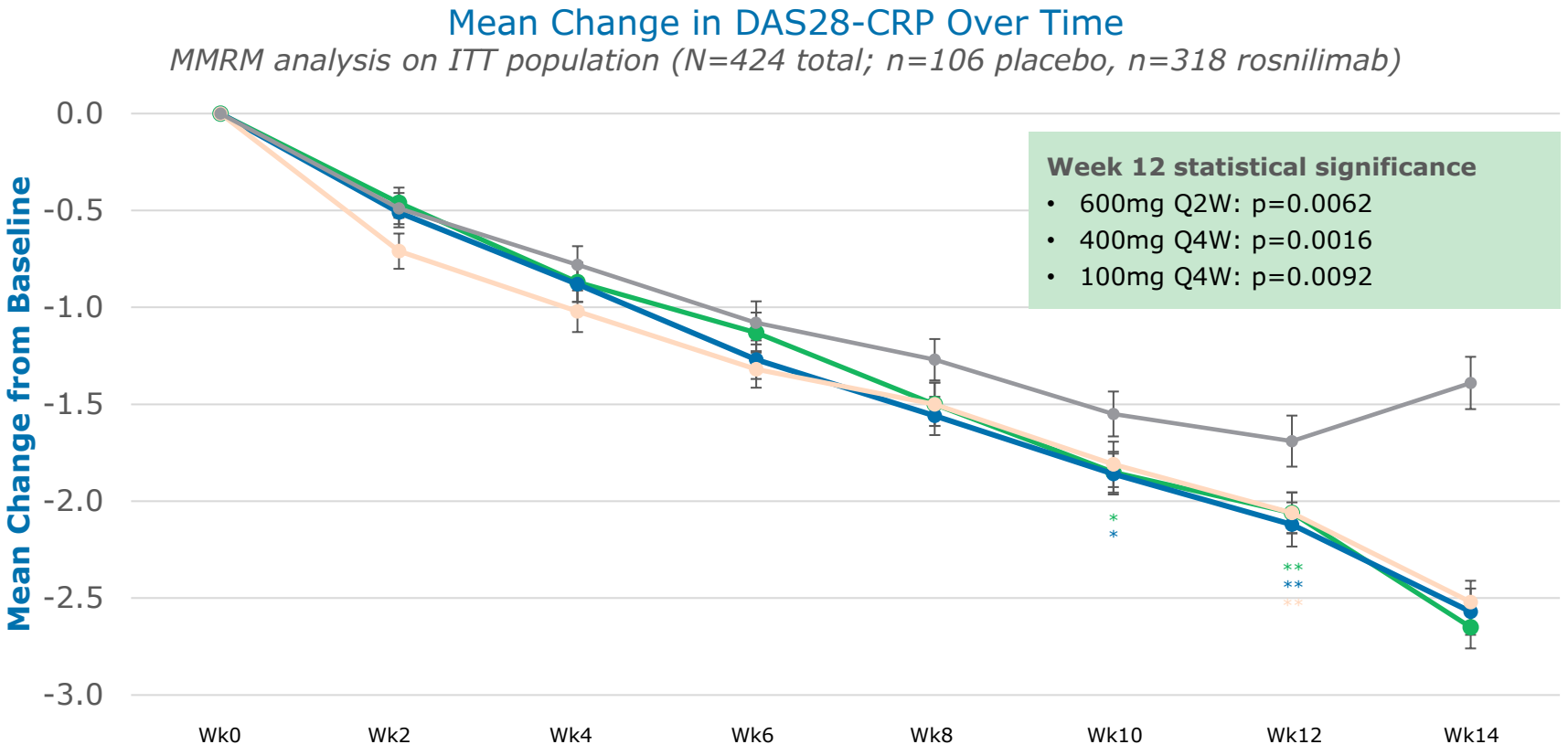
Effector T cells (T_{eff}): activated T cells (cytotoxic, helper, Treg); Follicular/Peripheral Helper T cells (T_{fh}, T_{ph}): support B cell differentiation and maturation.

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



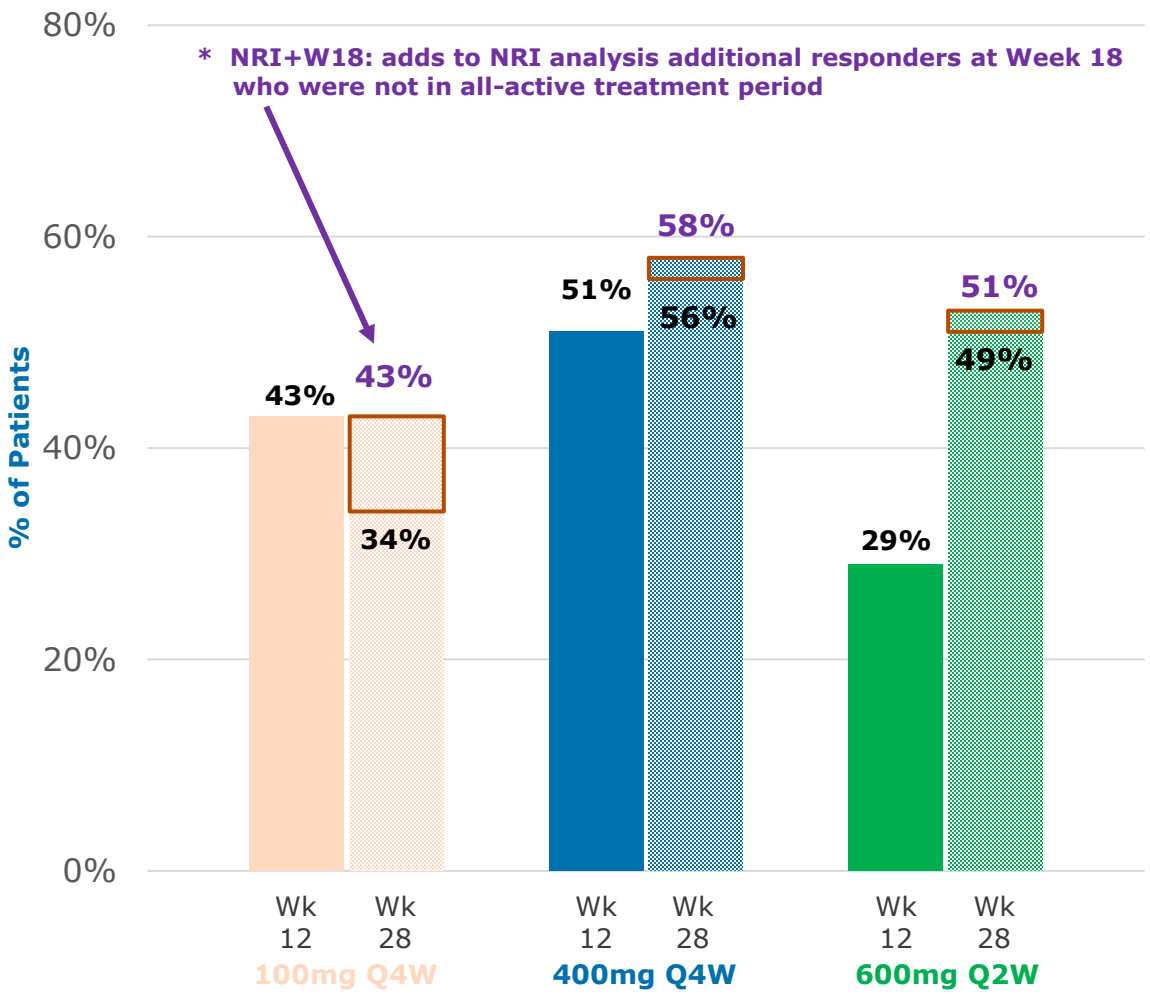
- All rosnilimab doses statistically significant at Week 12
- All rosnilimab doses continue to improve into Week 14 with no evidence of flattening
- Following Week 14 visit, placebo patients proceeded to post treatment follow-up

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: b/tsDMARD-Experienced

NRI+W18 analysis on ITT population (n=130 rosnilimab patients)*[1]



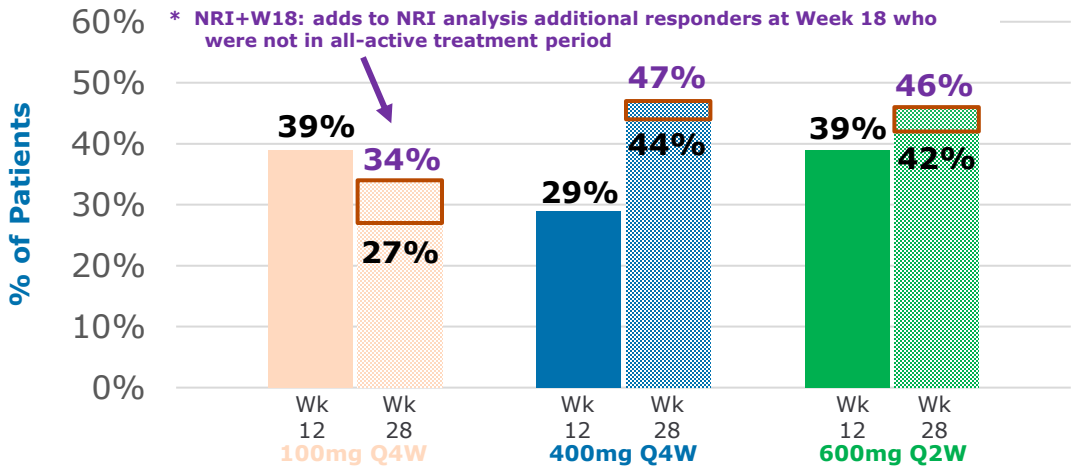
CDAI LDA at Week 28		
Arm	**NRI**	**NRI+W18**
b/tsDMARD-Experienced Population *(as graphed)*		
100mg	34%	43%
400mg	56%	58%
600mg	49%	51%
b/tsDMARD-Naïve Population		
100mg	66%	71%
400mg	53%	55%
600mg	72%	75%

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

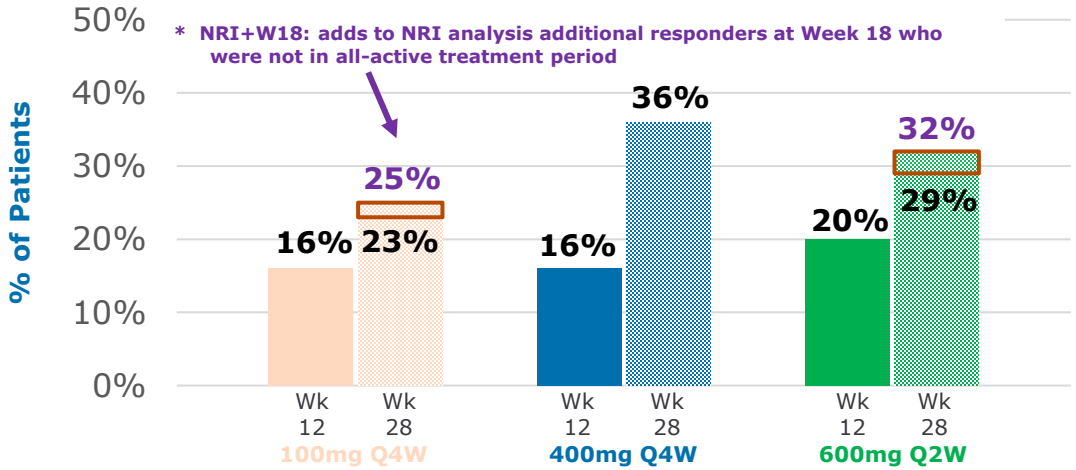
ACR50: b/tsDMARD-Experienced

NRI+W18 analysis on ITT population (n=130 rosnilimab patients)*[1]



ACR70: b/tsDMARD-Experienced

NRI+W18 analysis on ITT population (n=130 rosnilimab patients)*[1]

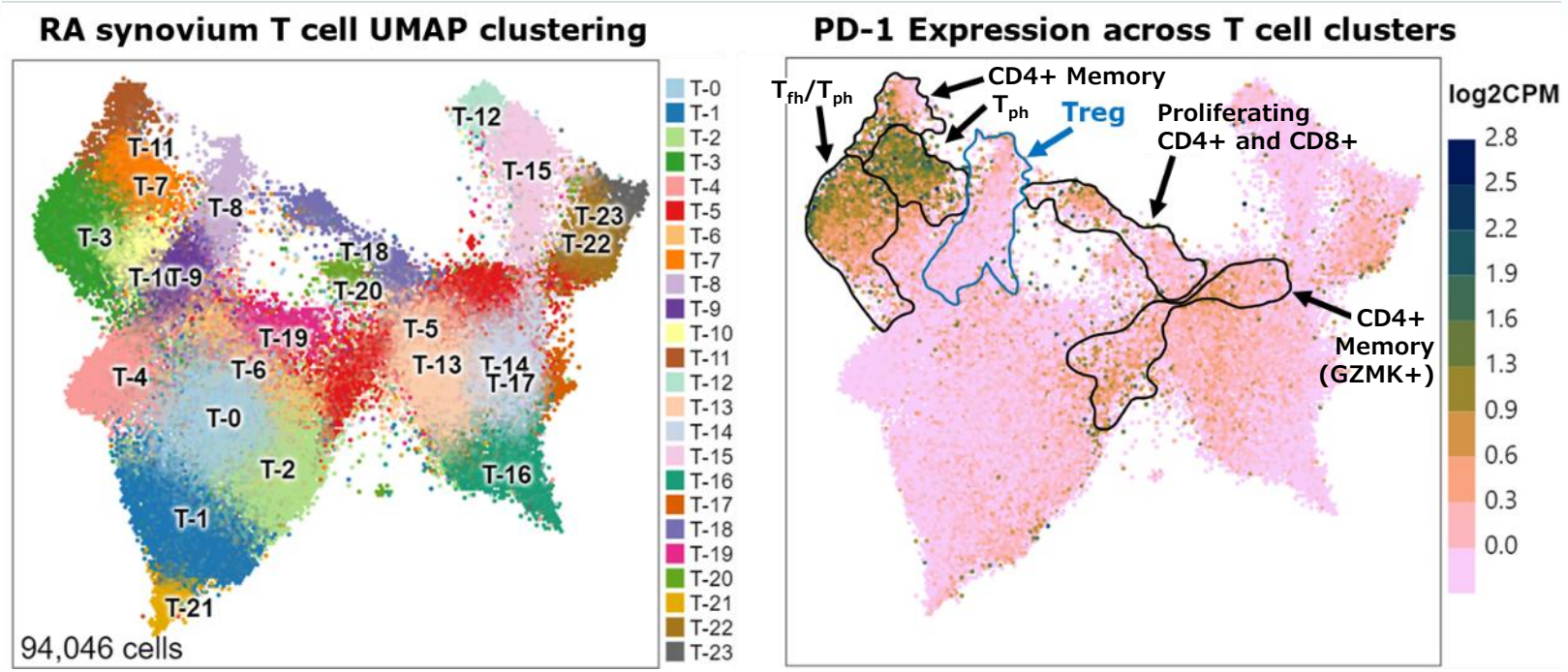


ACR50 at Week 28		
Arm	**NRI**	**NRI+W18**
b/tsDMARD-Experienced Population *(as graphed)*		
100mg	27%	34%
400mg	44%	47%
600mg	42%	46%
b/tsDMARD-Naïve Population		
100mg	58%	61%
400mg	52%	53%
600mg	69%	75%

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)

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



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

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

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

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

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