OP0202

Rosnilimab, a Selective and Potent Depleter of Pathogenic T Cells, Demonstrates Efficacy, Safety and Translational Proof of Mechanism in RENOIR, a Phase 2B Trial in Moderate-to-Severe Rheumatoid Arthritis

Jonathan Graf,[1] Sergii Kovalenko,[2] Katarzyna Kolossa,[3] John Serpa,[4] Tamta Kobakhidze,[5] Daniela Cepoi,[6] Andrea Everding,[7] Costantino Pitzalis,[8] Martin Dahl,[9] May Hafez,[9] Paul Lizzul,[9] Priya Raina,[9] Bruce Randazzo,[9] Khalil Saikali,[9] Cailin Sibley,[9] Gerd R. Burmester,[10] Jacques-Eric Gottenberg,[11] Iain McInnes,[12] Eduardo Mysler,[13] Lee S. Simon,[14] Josef S. Smolen,[15] Jeffrey A. Sparks,[16] Ronald van Vollenhoven,[17] Michael Weinblatt,[18] **Paul Emery[19]**

[1]UCSF, San Francisco, CA, USA; [2]Arensia Exploratory Medicine, Kyiv, Ukraine; [3]MICS Centrum Medycze, Bydgoszcz, Poland; [4]Allied Biomedical Research Institute, Miami, FL, USA; [5]Research Institute of Clinical Medicine Todua Clinic, Tbilisi, Georgia; [6]Nicolae Testemitanu State University of Medicine & Pharmacy, Chisinau, Moldova; [7]MVZ Rheumatologie & Autoimmunmedzin Hamburg GmbH, Hamburg, Germany; [8]QMUL, London, UK; [9]First Tracks Bio, San Diego, CA, USA; [10]Charité University Hospital, Berlin, Germany; [11]Hautepierre Hospital, Strasbourg, France; [12]University of Glasgow, Glasgow, Scotland; [13]Organizacion Medica de Investigatcion, Buenos Aires, Argentina; [14]SDG LLC, West Newton, MA, USA; [15]Medical University of Vienna, Vienna, Austria; [16]Brigham and Women's Hospital, Boston, MA, USA; [17]Amsterdam UMC, Amsterdam, Netherlands; [18]Harvard Medical School, Boston, MA, USA; [19]Leeds NIHR Biomedical Research Centre, LTHT Leeds Institute of Rheumatic and Musculoskeletal Medicine, University of Leeds, Leeds, UK

Sponsored by First Tracks Biotherapeutics, Inc., the biopharma operations business spun out of AnaptysBio, Inc. on April 20, 2026

Disclosures

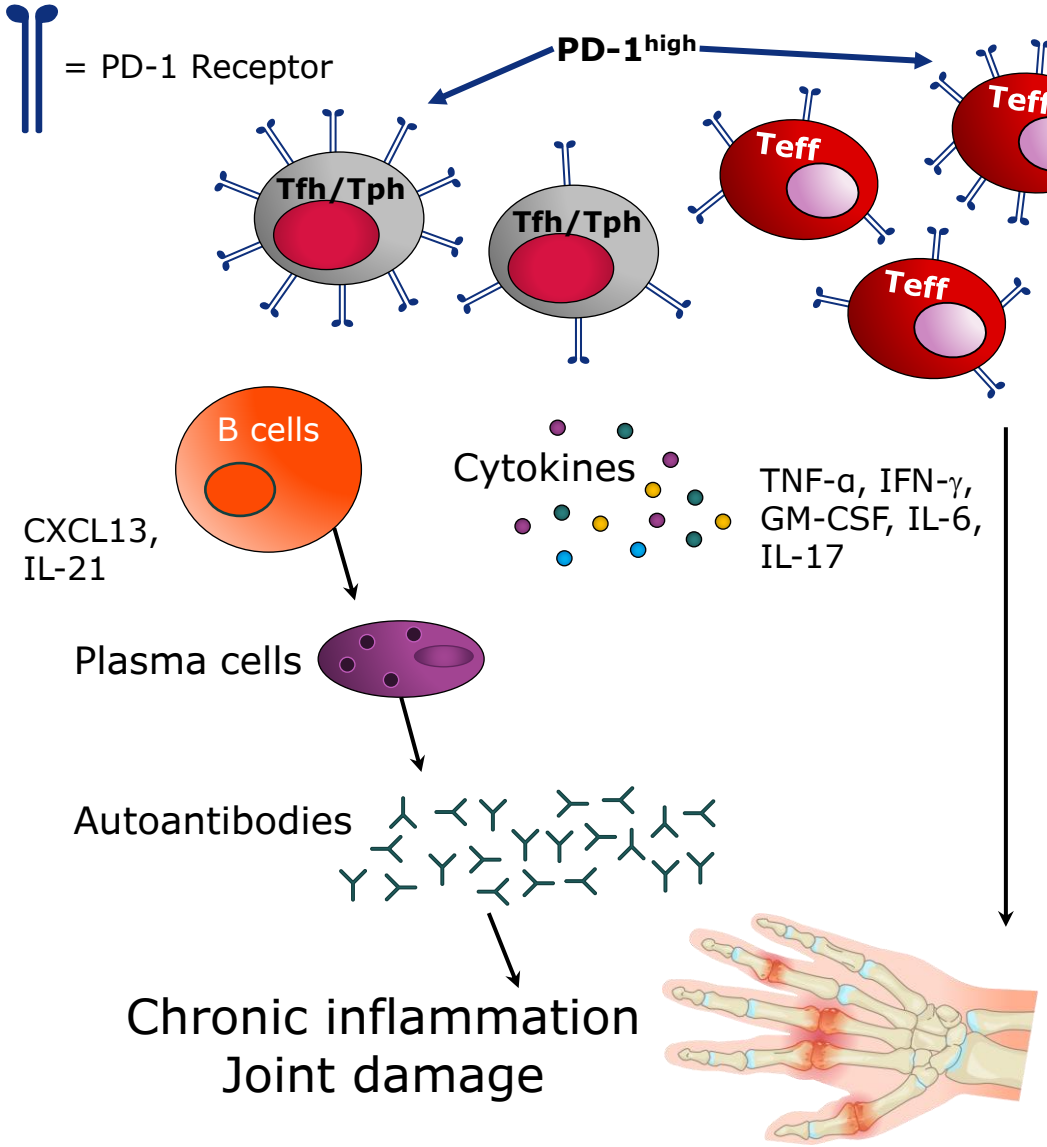
J Graf: grant/research support from Amgen, Fate Therapeutics, First Tracks Bio, Sonoma Biotherapeutics; consultant for First Tracks Bio, ImmPACT Bio. **C Pitzalis:** consultant for AbbVie/Abbott, First Tracks Bio, Novartis; grant/research support from AbbVie/Abbott, Novartis, Pfizer, Roche, Sanofi; independent contractor for AbbVie/Abbott, First Tracks Bio, Novartis, Roche, Sanofi; speaker for AbbVie/Abbott, Novartis, Roche, Sanofi, and UCB. **GR Burmester**: honoraria for lectures and consulting from: AbbVie, Alfasigma, Amgen, First Tracks Bio, Biogen, Gilead, Lilly, MSA, MSD, Pfizer, Sandoz, Sanofi, UCB. **JE Gottenberg:** speaker for AbbVie/Abbott, Amgen, BMS, Eli Lilly, Galapagos, Gilead, Johnson & Johnson, Novartis, Pfizer, and Sanofi; consultant to First Tracks Bio. **I McInnes:** advisor/review panel member for AbbVie/Abbott, Absci, Amgen, AZ, Boehringer-Ingelheim, BMS, Cabaletta, Causeway Therapeutics, Compugen, Dextera, Elasmogen, Eli Lilly, Janssen, Montai, Moonlake, Morningside, Novartis, Pfizer, Pioneering Medicines, Recludix, Roche, Sanofi, SciRhom, Sixtry, UCB; consultant to First Tracks Bio; grant/research support from AZ, BMS, Compugen, Dextera, Eli Lilly, Novartis, SciRhom, UCB; stock options or bond holdings in a for-profit corporation or self-directed pension plan for Cabaletta, Causeway, Compugen, Dextera, and Recludix. **E Mysler** is advisor/review panel member and receives grant/research support from AbbVie/Abbott, AZ, GSK, Novartis, and Pfizer; consultant to AbbVie/Abbott, AstraZeneca, First Tracks Bio, GSK, Novartis, Pfizer; speaker for AbbVie/Abbott, AZ, GSK, Novartis, and Pfizer. **LS Simon**: consultant, independent contractor for Asahi, AZ, Avenue, BioViva, BMS, Eupraxia, First Tracks Bio, Galvani, GSK, Immunovant, Inmedix, Kiniksa. **JS Smolen**: received grant/research support for his institution from: AbbVie, Astra-Zeneca, Eli Lilly and Company, Galapagos and Novartis; consultancy fees from: AbbVie, Ananda, BMS, Celltrion, Chugai, Eli Lilly, First Tracks Bio, Immunovant, Janssen, MSD, Novartis-Sandoz, Pfizer, Roche, R-Pharma, Samsung, Sanofi, and UCB. **JA Sparks**: consultant for Boehringer-Ingelheim, BMS, First Tracks Bio, Fresenius Kabi, Gilead, GSK, Inova Diagnostics, Invivyd, Johnson & Johnson, Merck, MustangBio, Novartis, Optum, Pfizer, ReCor, Sana, Sobi, UCB; research grant/support from Boehringer-Ingelheim, Johnson & Johnson, Sonoma. **R van Vollenhoven**: advisor/review panel member for AbbVie, AZ, Biogen, BMS, Cabaletta, Galapagos, GSK, Janssen, Kyowakirin, Merck, Pfizer, RemeGen, Sanofi, UCB; consultant for AbbVie, AZ, Biogen, BMS, Cabaletta, First Tracks Bio, Galapagos, GSK, Janssen, Kyowakirin, Merck, Pfizer, RemeGen, Sanofi, UCB; Grant/research support from Alfasigma, AZ, BMS, Caletta, Galapagos, GSK, Merck, Novartis, Pfizer, RemeGen, Roche, Sanofi, UCB; speaker for AbbVie, AZ, Biogen BMS, Cabaletta, GSK, Janssen, Kyowakirin, Merck, Pfizer, Sanofi, UCB. **M Weinblatt**: consultant for Aclaris, Amgen, Artiva Bio, First Tracks Bio, Biohaven, BMS, Curie Bio, Deep Cure, Forward Therapeutics, Eli Lilly, Gilead, Ignite, Janux Therapeutics, Johnson & Johnson, Lifordi, Marvel Bio, Matchpoint, Merck, Neutrolis, Novartis, Roche, Santa Ana, Sana, Sci Rhom, Set Point, Surf Therapeutics, Thymmune, Xencorp, ZuraBio; grant/research support from BMS; stock options Canfite, Inmedix, Scipher. **P Emery**: Advisor/review panel member for Abbvie, Activa, Artiva, AstraZeneca, BMS, Boehringer-Ingelheim, Eli Lilly, First Tracks Bio, Galapagos, Gilead, BMS, Immunovant, Janssen, Novartis; Clinical trials for Abbvie, BMS, Eli Lilly, Novartis, Pfizer, Samsung

S Kovalenko, K Kolossa, T Kobakhidze, D Cepoi, A Everding, J Serpa have nothing to disclose

M Dahl, M Hafez, P Lizzul, P Raina, B Randazzo, K Saikali, C Sibley are employees and shareholders of First Tracks Bio

Sponsored by First Tracks Biotherapeutics, Inc., the biopharma operations business spun out of AnaptysBio, Inc. on April 20, 2026

Pathogenic T Cells Drive Inflammation and Joint Damage in RA

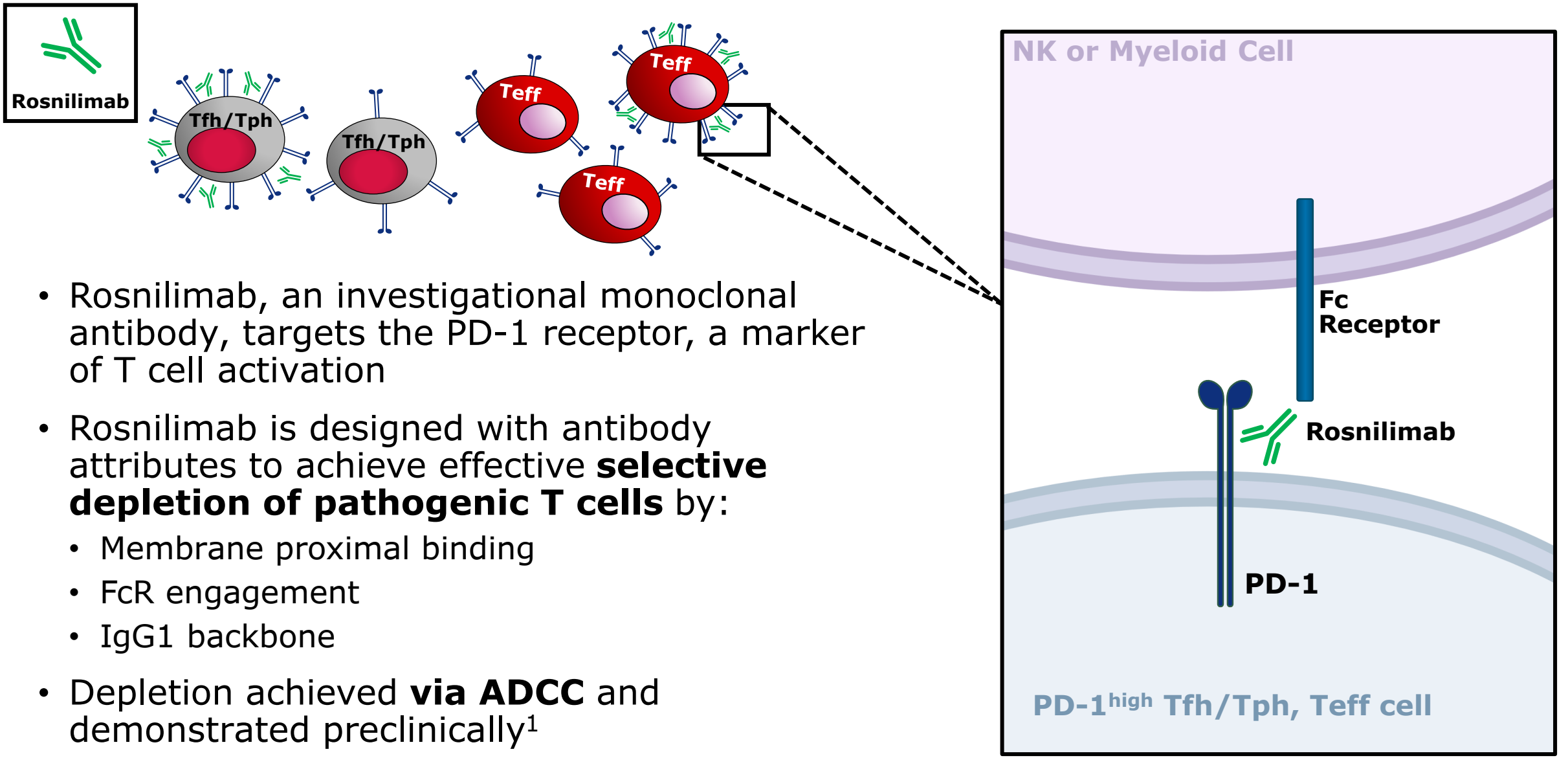


- Pathogenic T cells (PD-1^{high} Tfh/Tph, Teff):
  - Play a role in inflammation
  - Are upstream to a broad range of clinically validated targets in RA including TNF-α, IL-6, and B cells[1,2]
  - Are enriched in patients with RA (>80% of synovial T cells and 3x higher in peripheral blood) and low levels in healthy individuals[2,3]
- Tfh/Tph cells drive B cell activation and maturation, including autoantibody producing cells[1]
- Activated Teff cells proliferate and secrete proinflammatory cytokines

PD-1 Receptor = Programmed Cell Death Protein 1; Teff = T effector cells; Tfh = T follicular helper cells; Tph = T peripheral helper cells

1. Rao D, et al. *Nature* 2017;542:7639. 2. Guo Y, et al. *PLOS ONE* 2018;1:e0192704. 3. Chen Y-j, et al. *Clin Translational Immunol* 2024;13:e70006.

Rosnilimab Characteristics & Mechanism of Action



- Rosnilimab, an investigational monoclonal antibody, targets the PD-1 receptor, a marker of T cell activation
- Rosnilimab is designed with antibody attributes to achieve effective **selective depletion of pathogenic T cells** by:
  - Membrane proximal binding
  - FcR engagement
  - IgG1 backbone
- Depletion achieved **via ADCC** and demonstrated preclinically[1]

1. Parmley S, et al. *Arthritis Rheumatol* 2023;75 (Suppl 9):Abstract 0086.

Global, Randomized, Placebo-controlled Phase 2B Trial with Rosnilimab in Moderate-to-Severe Rheumatoid Arthritis

Key Inclusion Criteria

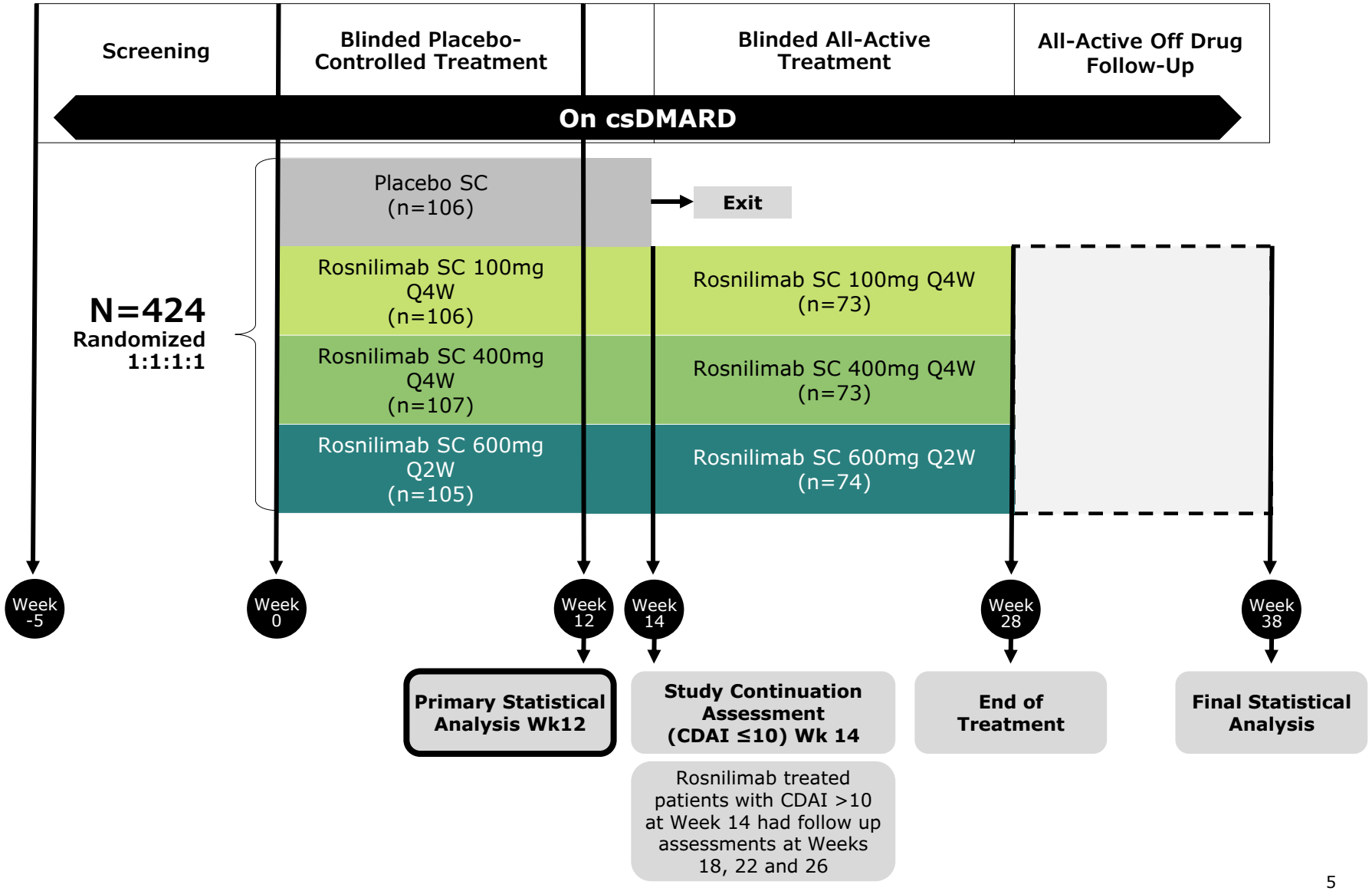
- Seropositive RA
- ≥6 swollen and ≥6 tender joints
- hs-CRP ≥ 3mg/L during Screening
- Concurrent use of 1 or 2 csDMARDs that were initiated at least 3 months before screening

Key Exclusion Criteria

- Inadequate response, loss of response, or intolerance to any combination of ≥ 3 b/tsDMARD classes

Primary Endpoint

- Mean change from baseline at Week 12 for DAS28-CRP



ClinicalTrials.gov: NCT06041269

Baseline Patient and Disease Characteristics Reflect Moderate-to-Severe Patient Population

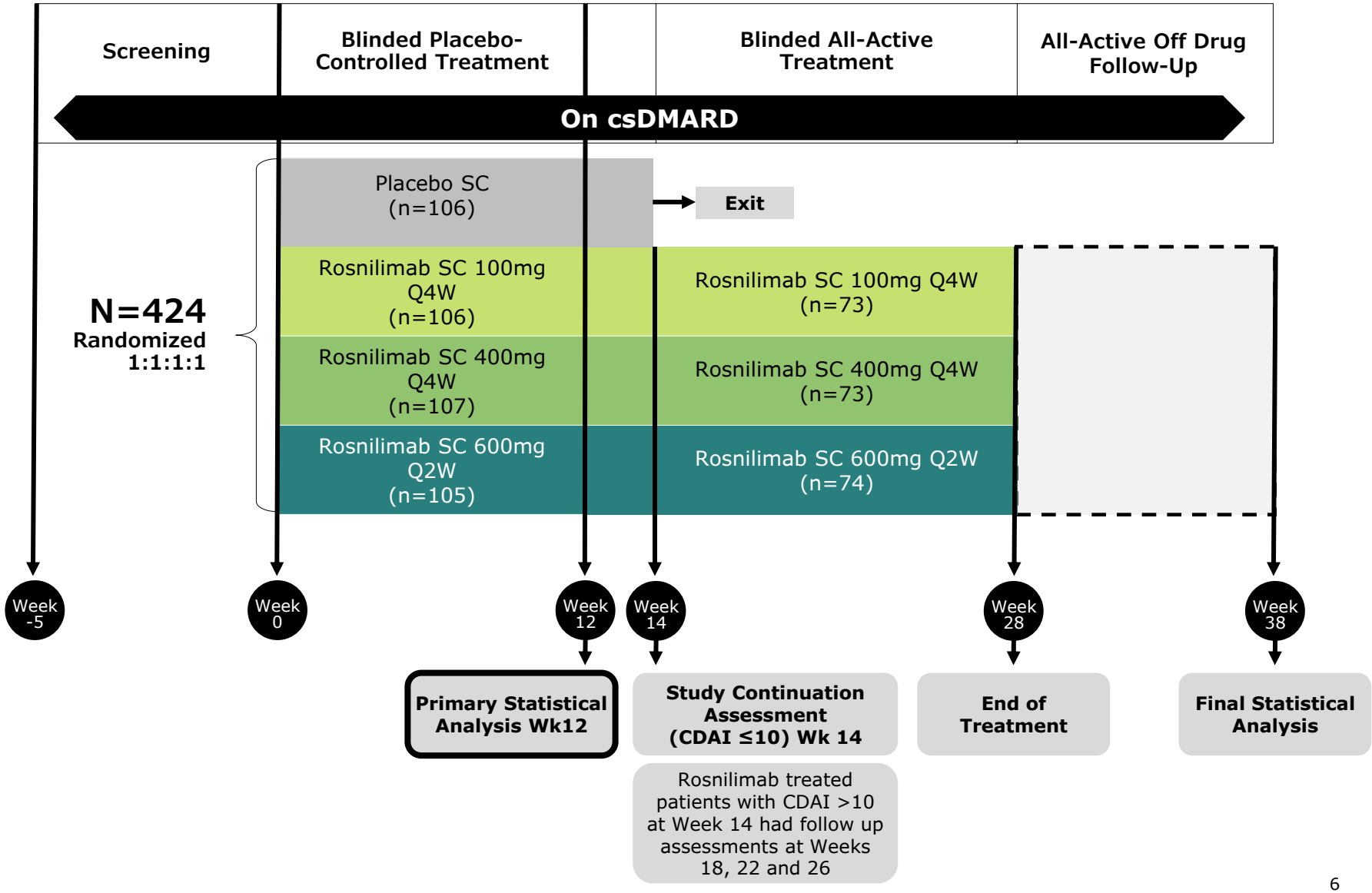
Key Baseline Characteristics

- Mean age 57 (11)* years, 76% female, mean body weight 78 (18)* kg

Key Baseline Disease Characteristics

- 41% received previous b/tsDMARDs
- 14% received >1 b/tsDMARDs
- 95% RF+
- 87% CCP+
- Mean DAS28-CRP 5.6 (0.8)*
- Mean CDAI 37.7 (10.6)*; 95% >22

*Mean (SD)

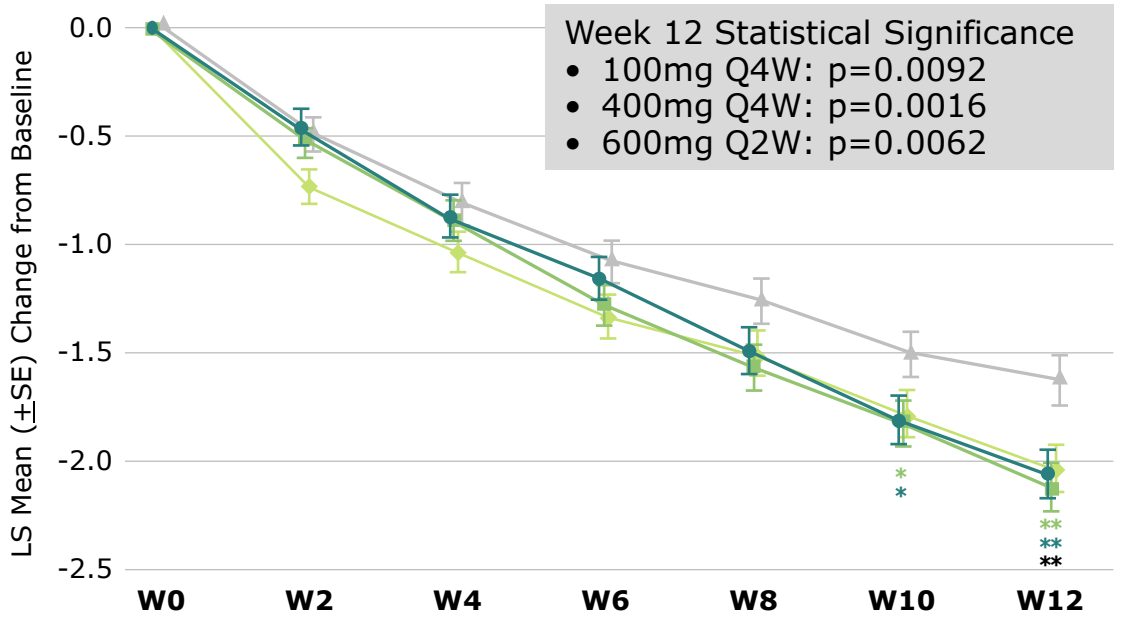


ClinicalTrials.gov: NCT06041269

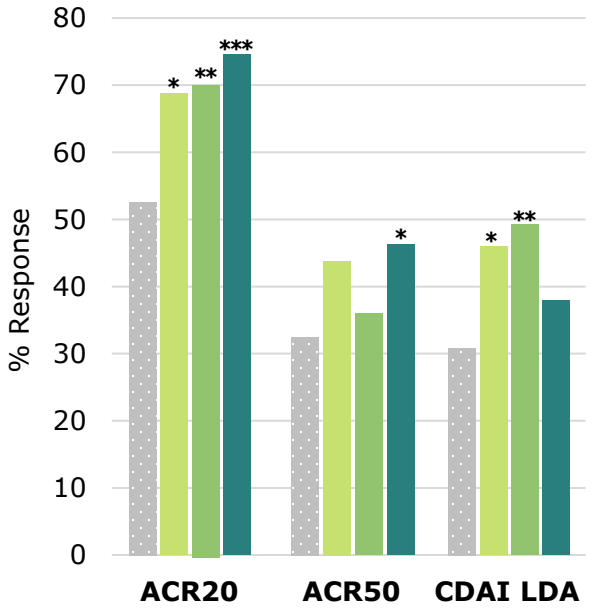
Primary and Select Secondary and Exploratory Endpoints Show Statistical Significance at Week 12

ITT population, MMRM and NRI analysis

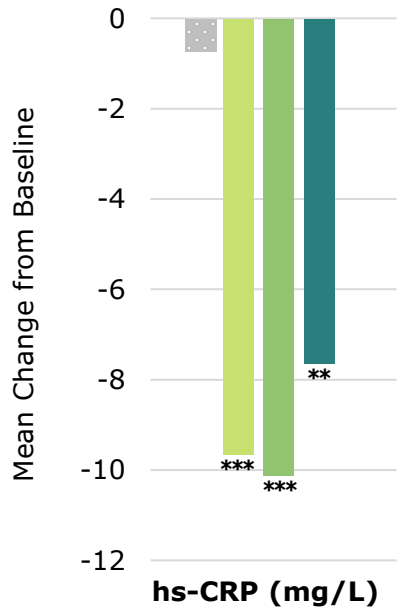
Primary Endpoint: Mean Change from Baseline in DAS28-CRP at Week 12



Secondary Endpoints at Week 12



Exploratory Endpoint at Week 12



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

All dose arms demonstrated statistically significant changes in DAS28-CRP

All dose arms showed statistically significant changes for traditional FDA regulatory endpoint (ACR20)

More stringent endpoints showed numerical, if not statistically significant, responses versus placebo at Week 12

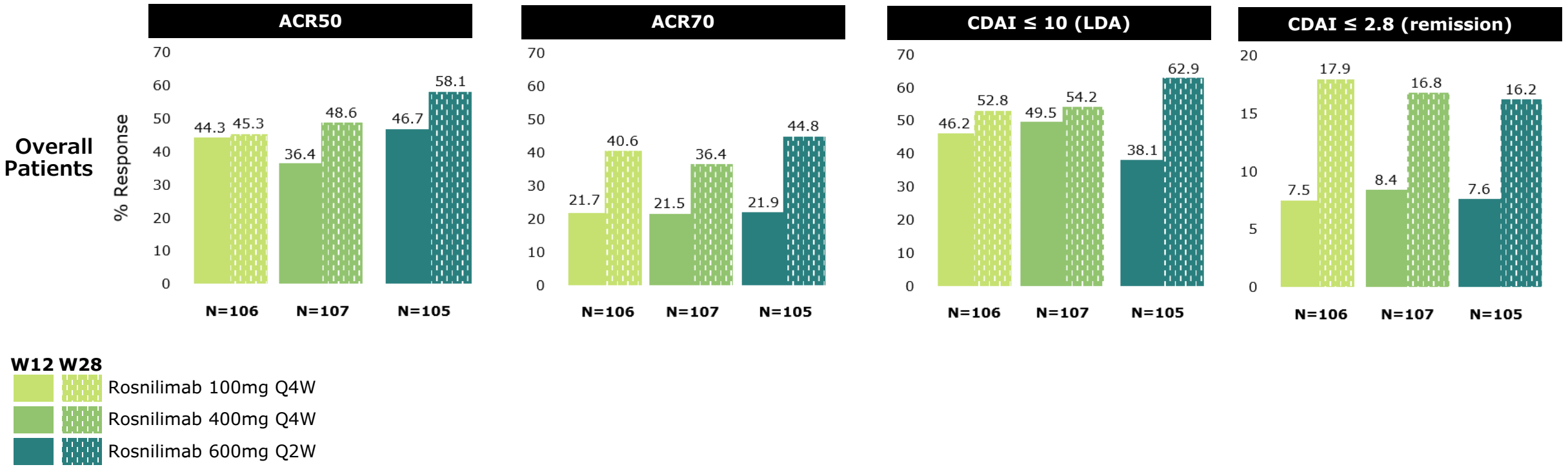
All dose arms had statistically significant changes for hs-CRP

Statistically significant differentiation of hs-CRP occurred as early as Week 2

*p < 0.05; **p < 0.01; ***p < 0.001; ITT population; MMRM for continuous endpoints, NRI for dichotomous endpoints

Responses Increase from Week 12 to Week 28 in Overall Population

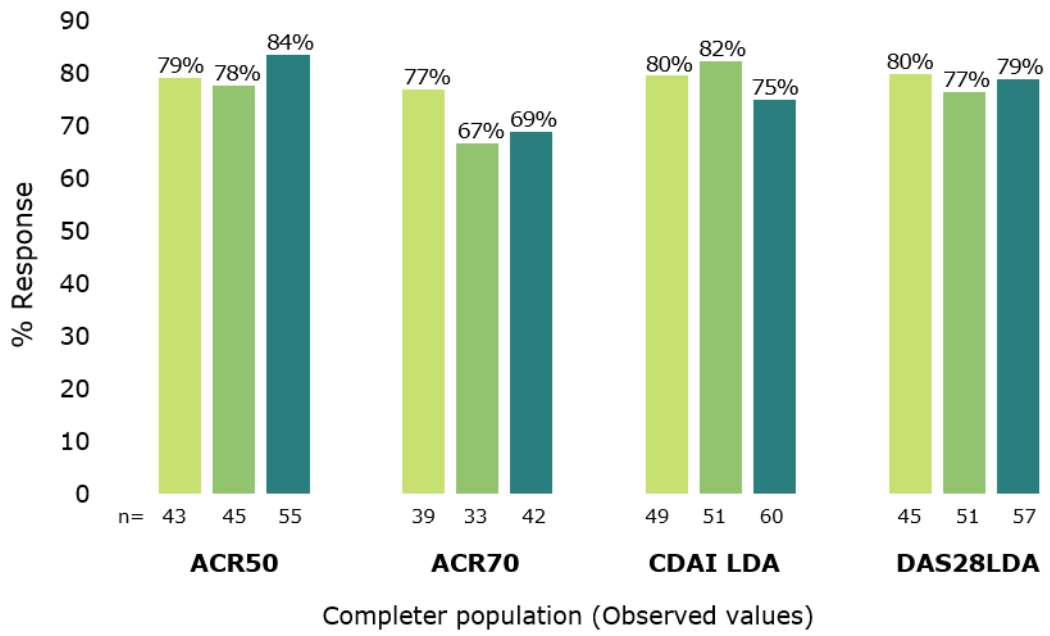
ITT population, NRI analysis



- Consistent improvement across various stringent endpoints
- In the ≥2 b/tsDMARD experienced population (data shown at ACR2025):
  - Consistent improvements observed in the 400mg and 600mg dose arms but not in the 100mg dose arm (supporting a dose response)
  - ACR50, ACR70, CDAI LDA, and CDAI remission observed regardless of prior therapy type (e.g., Anti-TNFα, Anti-IL6R, and JAK inhibitors)

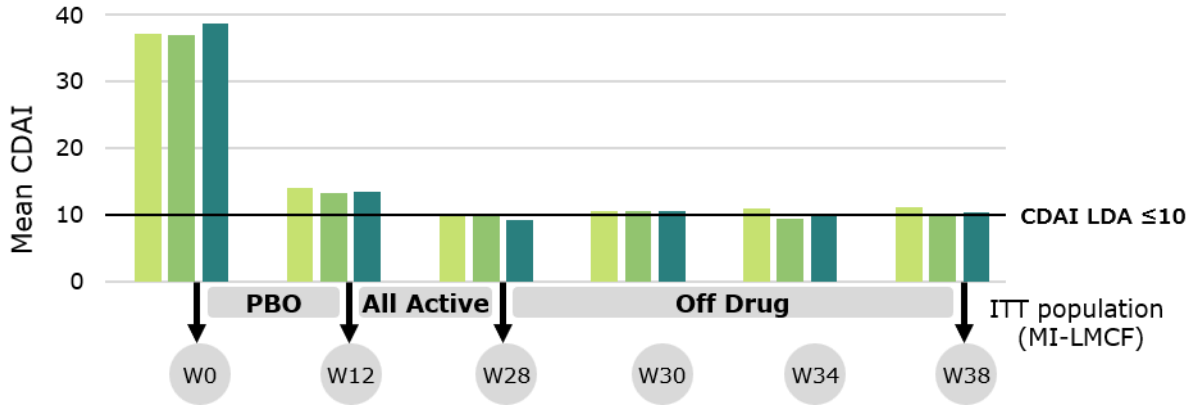
Responses Were Durable Off Rosnilimab for 3 Months

Week 28 Responders Maintaining Response Off-Drug (Week 38)

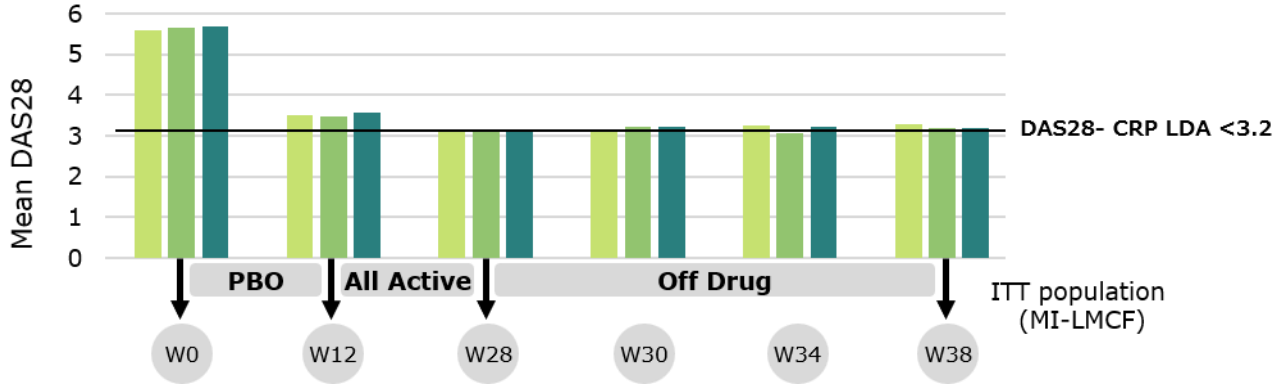


- Rosnilimab responders at Week 28 were likely to maintain their response during 3 months off drug
- Most rosnilimab participants achieved low disease activity by CDAI and DAS28-CRP which was maintained off rosnilimab for 3 months

Mean CDAI Over Time



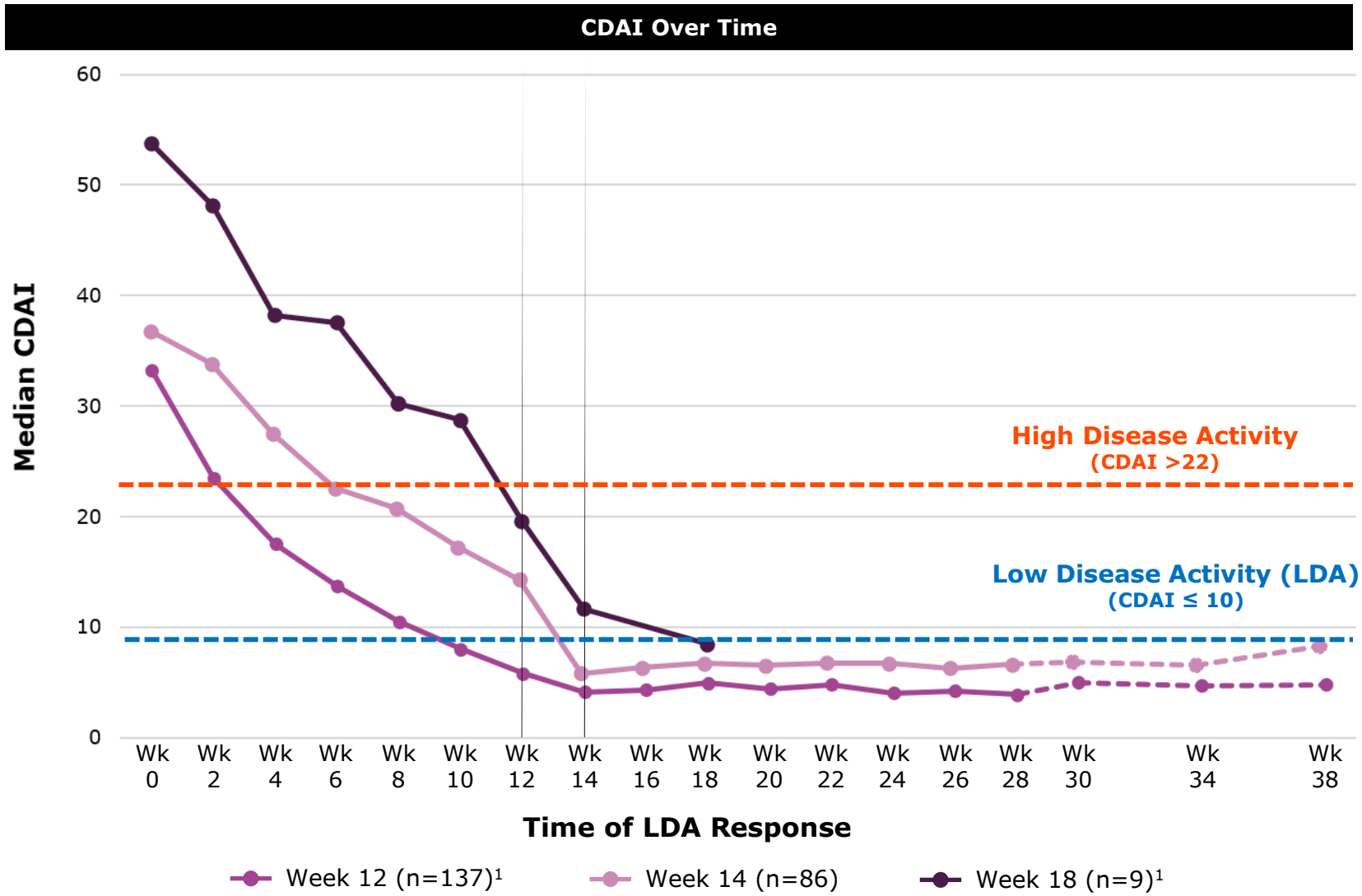
Mean DAS28-CRP Over Time



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

CDAI Over Time for Rosnilimab-treated Participants Achieving LDA

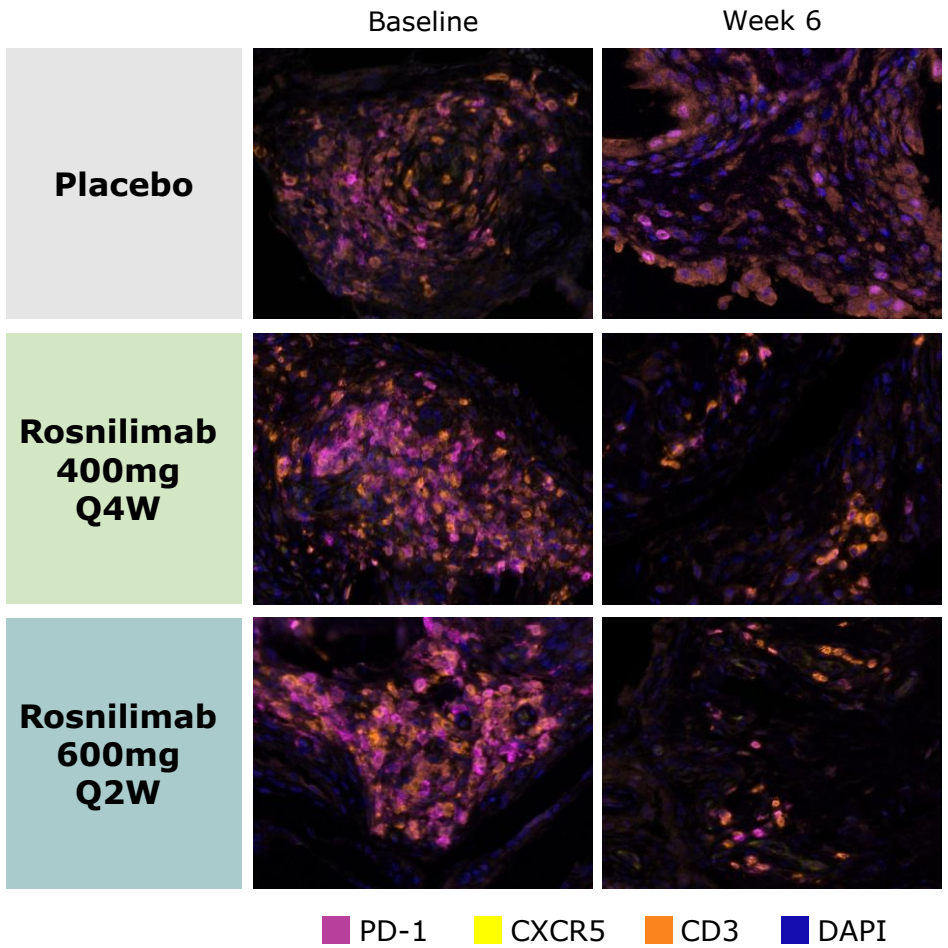
ITT population; NRI Analysis



- Participants with higher disease activity at baseline take longer to achieve CDAI LDA
- Of Week 14 CDAI LDA nonresponders
  - 60% achieved ACR20
  - 18% achieved ACR50
  - Median CDAI improvement of 22.6
- Of CDAI LDA responders at Week 18 but not Week 14:
  - Median baseline CDAI was 53.7
  - Median CDAI improvement of 44

Rosnilimab Demonstrated Pathogenic T cell Depletion and Broad Downregulation of Inflammatory Pathways in Synovium

Synovial Immunohistochemistry
n= 39 paired samples



Summary of Findings

~90% reduction of pathogenic T cells observed in the synovium for the 400mg Q4W and 600mg Q2W doses

- Inconsistent changes observed in the 100mg Q4W dose, and no changes observed in placebo

Similarly, >90% reduction of pathogenic T cells (including Tph cells) was observed at all doses in the periphery (data not shown here)

- No changes to overall T cells
- Treg numbers unchanged to increased

This was associated with broad reductions in downstream inflammatory pathways including T cell activation, B cell activation, and myeloid activation (data not shown here)

- Responders showed greater reductions in T cell and B cell activation

Data presented tomorrow morning by Professor Pitzalis, abstract #OP0278

Representative IHC samples from synovial biopsies. % change calculated for synovial samples with ≥10cells/mm^2 in baseline biopsy.
T_{ph}: PD-1+CD3+CD4+CXCR5-

Safety and Tolerability: Treatment Initiation Through Week 38 (End of Follow Up)

	Placebo** N=106 n (Rate)	Rosnilimab 100mg Q4W N=106 n (Rate)	Rosnilimab 400mg Q4W N=107 n (Rate)	Rosnilimab 600mg Q2W N=105 n (Rate)	All Rosnilimab N=318 n (Rate)
Participants with at least one AE	47 (152.72)	75 (238.29)	69 (190.35)	57 (140.09)	201 (185.41)
Participants with at least one AE related to study treatment	19 (51.15)	17 (29.07)	28 (49.50)	20 (35.38)	65 (37.89)
Participants with at least one severe AE	3 (7.12)	4 (6.00)	3 (4.38)	4 (6.13)	11 (5.49)
Participants with at least one severe study treatment-related AE	1 (2.35)	0	1 (1.45)	0	1 (0.49)
Participants with at least one SAE	1 (2.35)	3 (4.46)	5 (7.34)	4 (6.14)	12 (5.98)
Participants with at least one SAE related to study treatment	1 (2.35)	0	0	0	0
Participants with at least one AE leading to study treatment discontinuation	1 (2.35)	1 (1.48)	3 (4.36)	2 (3.01)	6 (2.95)
Participants with at least one SAE leading to study treatment discontinuation	1 (2.35)	0	0	0	0
Infections and infestations	23 (60.22)	43 (87.34)	43 (83.83)	35 (64.74)	121 (78.27)
Serious (SAE) infections	1 (2.35)	1 (1.48)	1 (1.45)	1 (1.50)	3 (1.48)
Opportunistic infections*	2 (4.75)	1 (1.48)	1 (1.45)	1 (1.50)	3 (1.47)
MACE	0	1 (1.47)	0	0	1 (0.49)
Malignancies	0	0	0	0	0
Deaths	0	0	0	0	0

- Rosnilimab was well tolerated with no safety dose effect
  - Low rates of treatment discontinuation on account of TEAEs
- Serious infections and opportunistic infections (herpes zoster) were balanced with no dose response
- 1 MACE in 100 mg group was ischemic stroke in participant with stenosis in common carotid artery
- There were no malignancies or deaths

*Herpes zoster is the only opportunistic infection. **Placebo participants received treatment through week 12.

Exposure adjusted incidence rate per 100 person-year = 100 x (Number of subjects with AE in the given period / Total years of exposure in the given period across all subjects at risk for the treatment). All adverse events (AEs) that are summarized above are treatment emergent adverse events. SAE=serious adverse event. N – total number of subjects in analysis set, n – number of subjects in specific category

Summary & Conclusions for Rosnilimab in RA

- Rosnilimab's novel MoA of selective and targeted pathogenic T cell depletion demonstrated the following:
  - Clinical proof of concept in RA
  - Durable disease control on and off drug
  - Broad downstream impact on Teff, Tfh/Tph, cytokine and B cells that drive the pathogenesis of RA and reflective of a return to immune homeostasis

  Data to be presented tomorrow, 5 June 2026 at 8:15 am, by Professor Pitzalis, abstract #OP0278

- Rosnilimab demonstrated a favorable safety profile
  - No deaths, no malignancies, balanced infection risk, no increase in opportunistic infections, no cytokine release, and no hypogammaglobulinemia

The **efficacy, durability, mechanistic validation, and safety/tolerability** supports continued development in RA

Thank You!

to

Patients
Caregivers
Investigators
Study Staff
& Everyone

involved in this study!

Medical writing assistance and graphics support was provided by Susan Sutch, PharmD of *Get-it-Write* Med Coms, Ellen Zigmont, PharmD, BCOP, and Cynthia Alexander, PhD of First Tracks Bio, and Lori Chollar and team of TLC Creative Services