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Nipocalimab in SLE: First-in-Class Efficacy and Safety Results Demonstrating Proof of Concept for FcRn Blockade From a Phase 2 Study

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Conflicts of Interest

RAF received personal fees from AstraZeneca, Biogen, Bristol Myers Squibb, GSK, Genentech/Roche, and Johnson & Johnson during the conduct of the study and outside the submitted work; and received grants from Genentech/Roche

RvV received consulting fees from AbbVie, AstraZeneca, Biogen, Bristol Myers Squibb, Galapagos, GSK, Instituto Científico Pfizer, Johnson & Johnson, Kyowa Kirin, RemeGen, Sanofi, UCB, and Viela Bio; and received financial support from Bristol Myers Squibb, F. Hoffmann-La Roche, and Merck

RW has no conflicts of interest to disclose

EM received grant/research support from AbbVie, Amgen, AstraZeneca, Biogen, Bristol Myers Squibb, Eli Lilly, EMD Serono, Genentech, GSK, Johnson & Johnson, and UCB; and received consulting fees from Amgen, AstraZeneca, Biogen, Bristol Myers Squibb, Eli Lilly, EMD Serono, Genentech, GSK, Johnson & Johnson, Servier, UCB, and Wolf Biotherapeutics; and received speaking fees and/or honoraria from AstraZeneca, Eli Lilly, GSK, and Novartis

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GAK participated in a speakers bureau for and received consulting fees from Bristol Myers Squibb, Johnson & Johnson, and Sanofi-Genzyme-Regeneron; and received grant/research support from Pfizer

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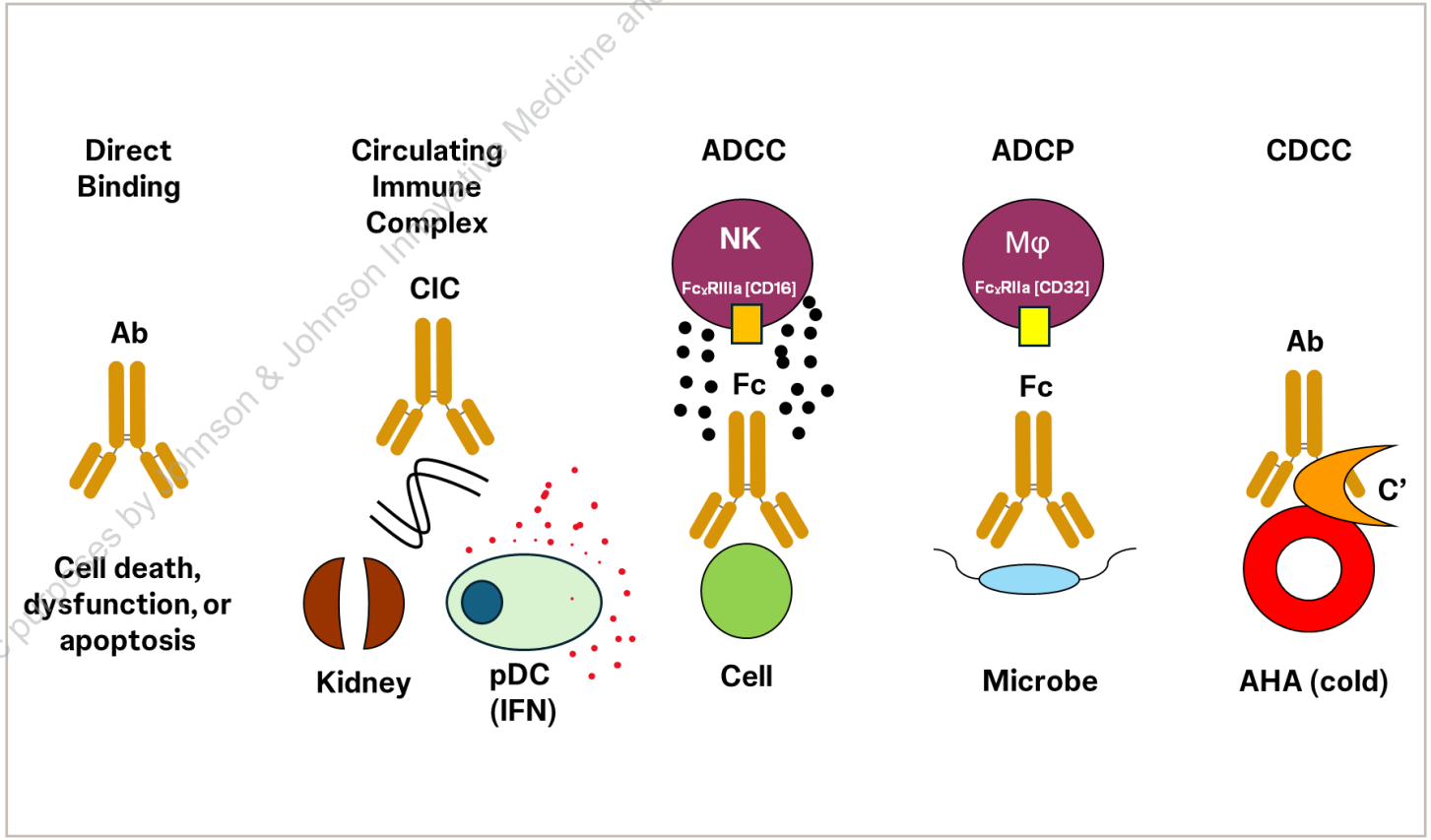
MP received grant/research support from AstraZeneca, Exagen, and GSK; and received consulting fees from Amgen, Annenberg Center for Health Sciences, AstraZeneca, Blackrock, Eli Lilly, Exagen, Glenmark, GSK, IQVIA, Johnson & Johnson, Merck EMD Serono, Novartis, Quintiles, Sanofi Japan, Thermo Fisher, and UCB

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Systemic Lupus Erythematosus (SLE)

IgG autoantibodies (aAbs) in SLE pathogenesis[1,2]

- IgG aAbs cause inflammation and tissue damage through:
  - Cytotoxicity by direct binding to cellular targets, ADCC, ADCP, and CDCC
  - Immune activation by tissue deposition of circulating immune complexes (CICs)

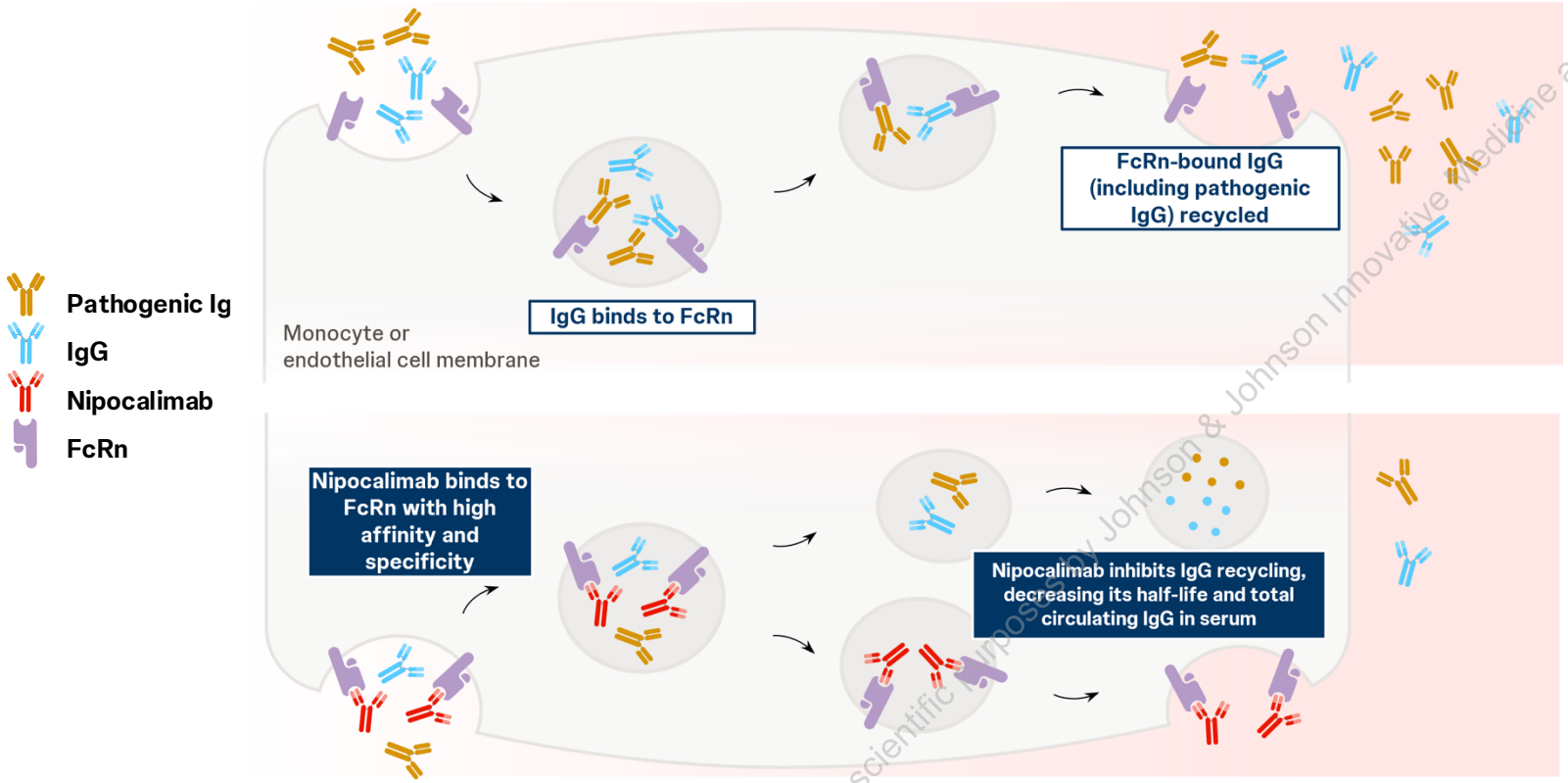


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aAb=autoantibody, **Ab**=antibody, **ADCC**=antibody-dependent cellular cytotoxicity, **ADCP**=antibody-dependent cellular phagocytosis, **AHA**=autoimmune hemolytic anemia, **C'**=complement, **CDCC**=complement-dependent cellular cytotoxicity, **CIC**=circulating immune complex, **Fc**=fragment crystallizable, **FcγR**=fragment crystallizable gamma receptor, **IFN**=interferon, **IgG**=immunoglobulin G, **Mφ**=macrophage, **NK**=natural killer, **pDC**=plasmacytoid dendritic cell, **SLE**=systemic lupus erythematosus.

1. Ameer MA, et al. *Cureus*. 2022;14(10):e30330. 2. Herrada AA, et al. *Front Immunol*. 2019;10:772.

Nipocalimab: FcRn Blocker Decreases Serum IgG, Including Pathogenic IgG and CICs



FcRn:

- A recycling or transcytosis receptor → maintains IgG levels in circulation[1-3]
- Expressed in multiple cell types, including monocytes and endothelial cells[1-3]

Nipocalimab:

- A high-affinity, fully human, IgG1 FcRn blocker → selectively blocks IgG recycling[4]
- Decreases serum IgG, including pathogenic IgG and CICs while preserving immune cell functions[4-6]
- Approved for wAIHA in the US, Brazil, and Panama[7]
- Approved for gMG in the US, Brazil, Panama, Japan, Europe, and China[7]

Objective: To evaluate the efficacy, safety, and pharmacodynamics of nipocalimab in adults with active SLE through a multicenter, randomized, double-blind, placebo-controlled, parallel-group study (JASMINE-SLE)

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CIC=circulating immune complex, **FcRn**=neonatal crystallizable fragment receptor, **gMG**=generalized myasthenia gravis, **IgG**=immunoglobulin G, **wAIHA**=warm autoimmune hemolytic anemia.

1. Blumberg LJ, et al. *Sci Adv*. 2019;5(12):eaax9586. 2. Roopenian DC, Akilesh S. *Nat Rev Immunol*. 2007;7(9):715-725. 3. Peter HH, et al. *J Allergy Clin Immunol*. 2020;146(3):479-491.e5. 4. Seth NP, et al. *MAbs*. 2025;17(1):2461191. 5. Ling LE, et al. *Clin Pharmacol Ther*. 2019;105(4):1031-1039. 6. Noaiseh G, et al. *Lancet*. 2025;406(10518):2435-2448. 7. IMAAVY™ (nipocalimab-aahu) injection, for intravenous use [prescribing information]. Janssen Biotech, Inc.; 2025.

Phase 2 JASMINE-SLE Study Design

Key eligibility criteria

- Adults with active SLE (SLICC 2012) for ≥6 months
- Received standard of care (antimalarials, oral GCs, and protocol-permitted immunomodulators)
- Positive for ANA, anti-dsDNA, and/or anti-Smith
- 1 BILAG A and/or 2 BILAG B scores
- SLEDAI-2K score ≥6

Primary endpoint[a]

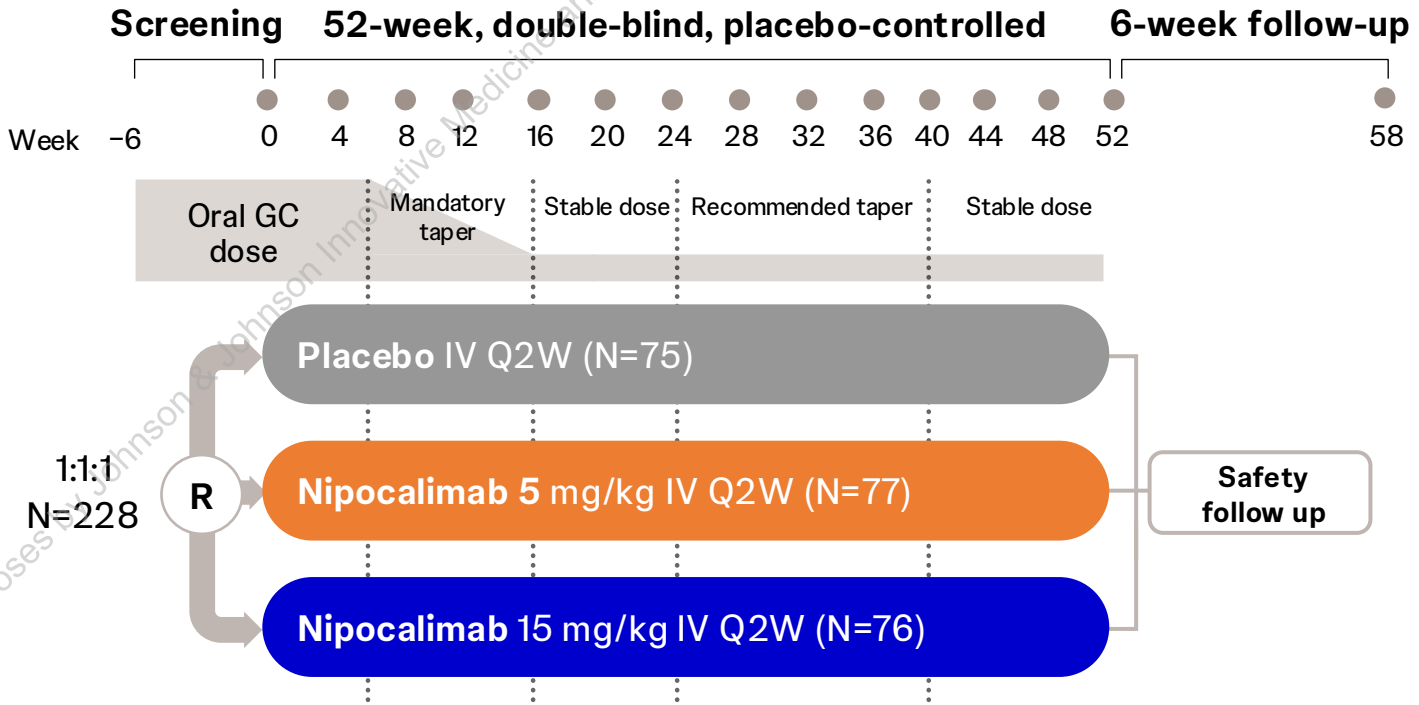
- % participants achieving SRI-4 composite response at Week 24

Key secondary/exploratory endpoints[b]

- % participants achieving SRI-4 composite response at Week 52
- % participants achieving LLDAS response at Week 52

Oral GC taper

- **Mandatory taper:** Weeks 6 to 16 to a target of 7.5 mg/day and maintained through Week 24[c]
- **Recommended taper:** Weeks 24 to 40 to a target of 7.5 mg/day and maintained through Week 52



[a]Only the primary endpoint was controlled for multiplicity, with a prespecified 2-sided alpha level of 0.10. [b]All other efficacy analyses were not adjusted for multiplicity, with nominal *P* values reported. [c]Participants on ≥10 mg/day oral GCs were required to taper; tapering could be paused/increased for disease worsening or safety concerns. FAS included all randomized participants who received ≥1 dose of any study intervention (for safety analyses and primary endpoint analysis). mFAS included all randomized participants who received ≥1 dose of any study intervention, excluding 7 participants from one study site due to noncompliance with Good Clinical Practice identified between Week 24 and Week 52, for Week 52 efficacy analyses.

ANA=antinuclear antibody, **BILAG**=British Isles Lupus Assessment Group Scale, **dsDNA**=double-stranded deoxyribonucleic acid, **GC**=glucocorticoid, **IV**=intravenous, **LLDAS**=Lupus Low Disease Activity State, **Q2W**=every 2 weeks, **R**=randomization, **SLE**=systemic lupus erythematous, **SLEDAI-2K**=Systemic Lupus Erythematosus Disease Activity Index 2000, **SLICC**=Systemic Lupus International Collaborating Clinics criteria, **SRI**=Systemic Lupus Erythematosus Responder Index, **FAS**=full analysis set, **mFAS**=modified full analysis set.

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Demographic and Baseline Characteristics

Demographic and baseline clinical characteristics were comparable across treatment groups

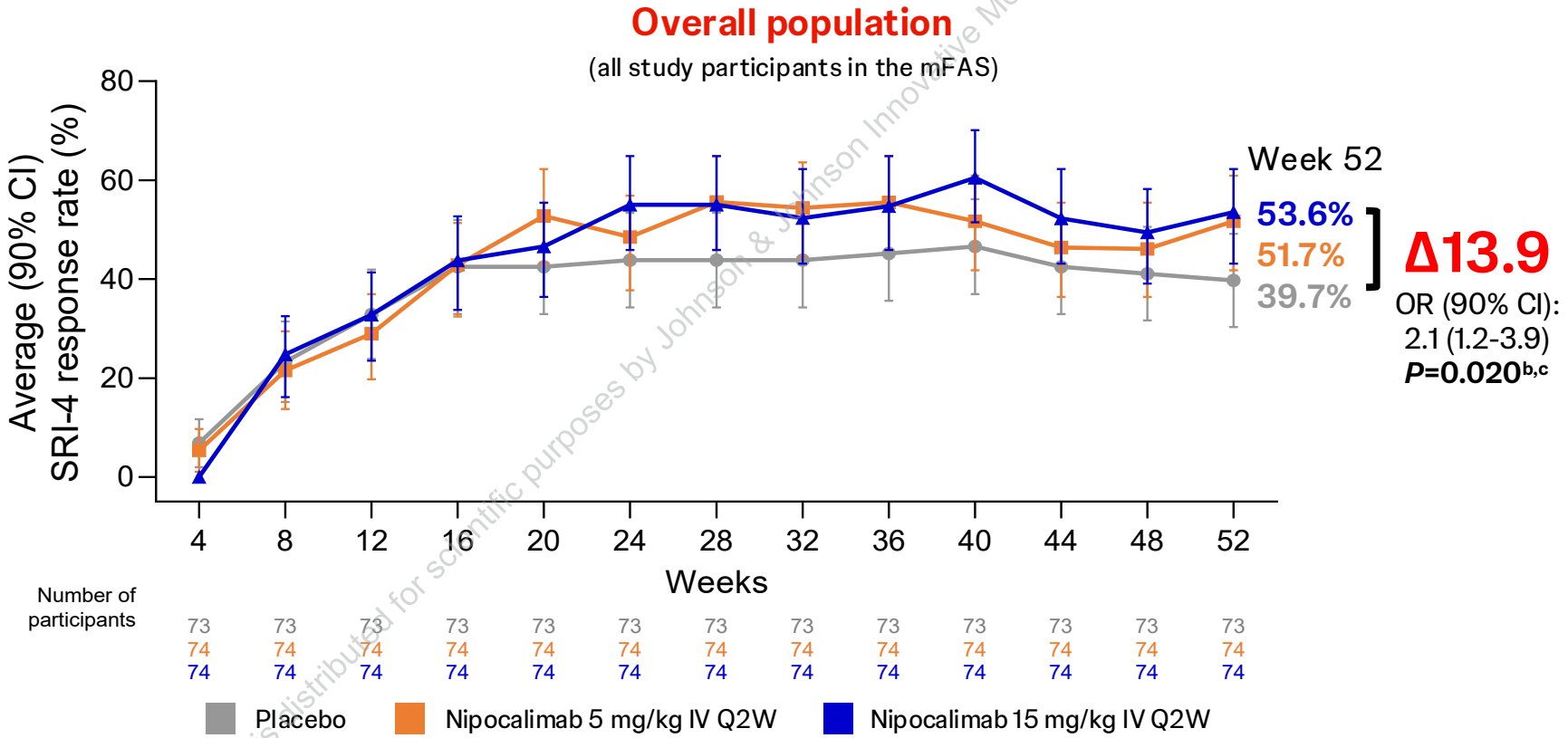
Baseline characteristics[a]	Placebo (N=73)	Nipocalimab 5 mg/kg IV Q2W (N=74)	Nipocalimab 15 mg/kg IV Q2W (N=74)	Total (N=221)
Demographics				
Age, years, mean (SD)	45.5 (11.3)	40.9 (11.9)	44.0 (11.9)	43.4 (11.8)
Female, n (%)	69 (94.5)	70 (94.6)	70 (94.6)	209 (94.6)
White, n (%)	50 (68.5)	50 (67.6)	44 (59.5)	144 (65.2)
Disease characteristics, mean (SD)				
SLE duration, years	9.3 (8.0)	8.1 (6.0)	10.8 (9.6)	9.4 (8.0)
SLEDAI-2K score (0-105)	**10.4 (3.0)**	**10.0 (3.2)**	**9.8 (2.5)**	**10.1 (2.9)**
PGA score (0-3)	2.0 (0.3)	1.9 (0.3)	2.0 (0.3)	2.0 (0.3)
BILAG, n (%)				
≥1 BILAG A	41 (56.2)	40 (54.1)	50 (67.6)	131 (59.3)
≥2 BILAG B	31 (42.5)	32 (43.2)	24 (32.4)	87 (39.4)
≥1 BILAG A or ≥2 BILAG B	72 (98.6)	71 (95.9)	72 (97.3)	215 (97.3)
CLASI activity score (0-70)	7.1 (4.5)	8.2 (7.2)	7.6 (6.7)	7.6 (6.2)
≥6 , n (%)	40 (54.8)	42 (56.8)	40 (54.1)	122 (55.2)
Number of active joints (0-62)[b]	8.0 (4.6)	8.6 (4.5)	10.1 (7.1)	8.9 (5.6)
≥4, n (%)	66 (90.4)	69 (93.2)	67 (90.5)	202 (91.4)
Total IgG, g/L	14.2 (4.8)	15.2 (6.8)	14.4 (5.4)	14.6 (5.7)
Anti-dsDNA (positive ≥75), IU/mL	**167.0 (467.2)**	**155.7 (354.1)**	**225.5 (663.2)**	**182.8 (509.8)**
C3 (0.9-1.8[c]), g/L	1.1 (0.3)	1.1 (0.3)	1.1 (0.3)	1.1 (0.3)
CIC (0-15.1[c]),[d] µg Eq/mL	11.4 (7.9)	12.7 (9.2)	11.9 (8.1)	12.0 (8.4)
Concomitant medications, n (%)				
Antimalarials	62 (84.9)	61 (82.4)	64 (86.5)	187 (84.6)
Oral GCs	**46 (63.0)**	**59 (79.7)**	**52 (70.3)**	**157 (71.0)**
Immunomodulators	**38 (52.1)**	**33 (44.6)**	**35 (47.3)**	**106 (48.0)**

[a]Based on the mFAS. Sample sizes for each parameter reflect nonmissing values. [b]Active joints are joints that are painful (as reported by participants) and tender and swollen (on physical exam as determined by the joint assessor). [c]Reflects a normal range. [d]Based on the mFAS with available baseline CIC values (placebo n=72, nipocalimab 5 mg/kg n=71, nipocalimab 15 mg/kg n=72).
BILAG=British Isles Lupus Assessment Group Scale, **C3**=complement component 3, **CIC**=circulating immune complex, **CLASI**=Cutaneous Lupus Erythematosus Disease Area and Severity Index, **dsDNA**=double-stranded deoxyribonucleic acid, **Eq**=equivalent, **GC**=glucocorticoid, **IgG**=immunoglobulin G, **IV**=intravenous, **mFAS**=modified full analysis set, **PGA**=Physician's Global Assessment of Disease Activity, **Q2W**=every 2 weeks, **SD**=standard deviation, **SLE**=systemic lupus erythematous, **SLEDAI-2K**=Systemic Lupus Erythematosus Disease Activity Index 2000.

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Primary Endpoint: SRI-4 at Week 24, Maintained Through Week 52

Primary endpoint: SRI-4 composite response at Week 24 was met for nipocalimab 15 mg/kg versus placebo (53.5% vs 46.7% *P*=0.081[a]) but not for nipocalimab 5 mg/kg versus placebo



[a]Only the primary endpoint was controlled for multiplicity, with a prespecified 2-sided alpha level of 0.10 across doses, appropriate for a phase 2, proof-of-concept study. **FAS** included all randomized participants who received ≥1 dose of any study intervention (for safety analyses and primary endpoint analysis). **mFAS** included all randomized participants who received ≥1 dose of any study intervention, excluding 7 participants from one study site due to noncompliance with Good Clinical Practice identified between Week 24 and Week 52, for Week 52 efficacy analyses. [b]Analysis based on average response rates with imputed data. [c]The *P* value was nominal.

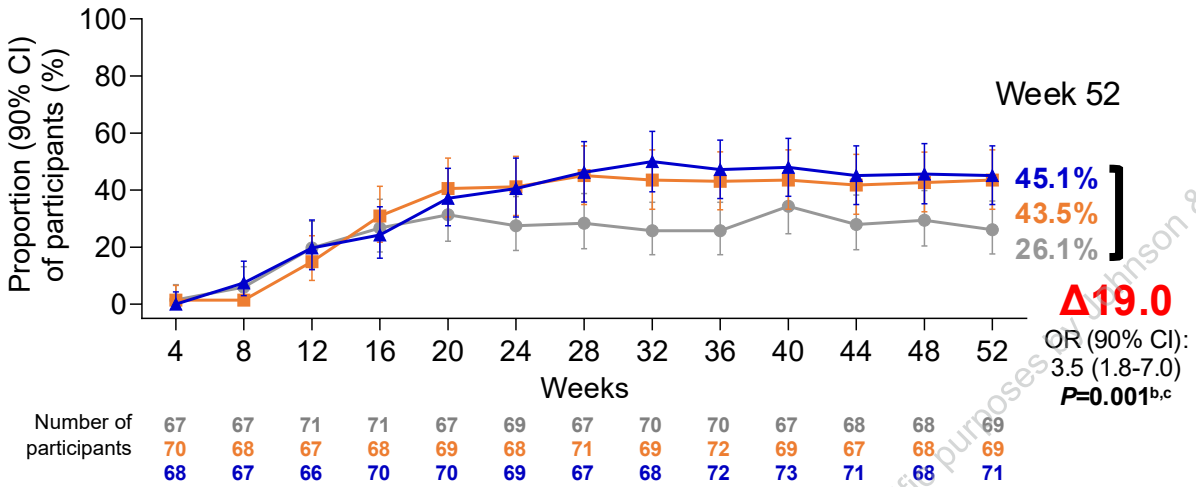
aAb=autoantibody, **CI**=confidence interval, **FAS**=full analysis set, **IV**=intravenous, **mFAS**=modified full analysis set, **OR**=odds ratio, **Q2W**=every 2 weeks, **SRI**=Systemic Lupus Erythematosus Responder Index.

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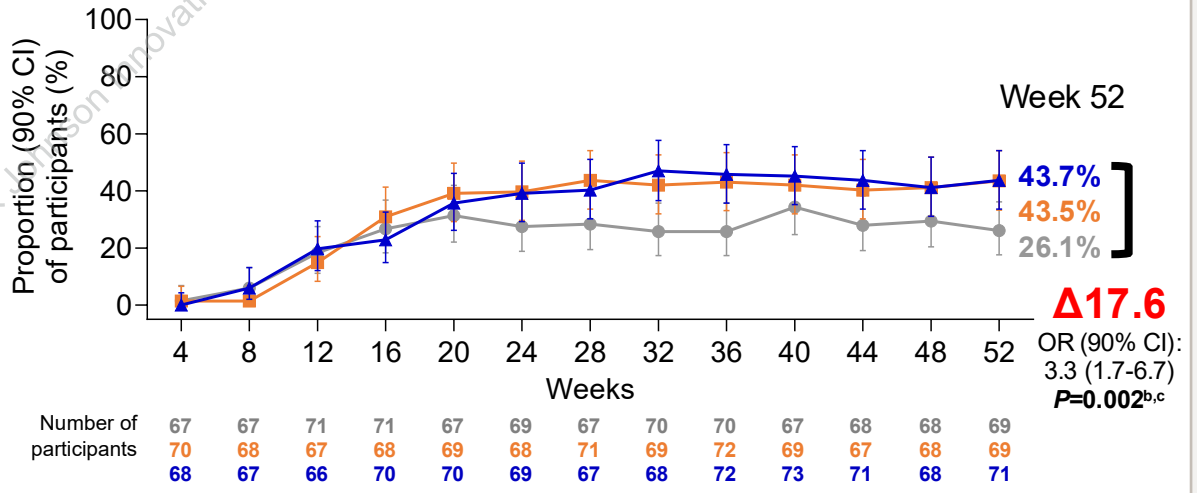
SRI-5 and SRI-6 Composite Response[a] Through Week 52

Higher proportions of participants receiving nipocalimab 15 mg/kg than placebo achieved SRI-5 or SRI-6 composite responses

SRI-5
(all study participants in the mFAS)



SRI-6
(all study participants in the mFAS)



Placebo Nipocalimab 5 mg/kg IV Q2W Nipocalimab 15 mg/kg IV Q2W

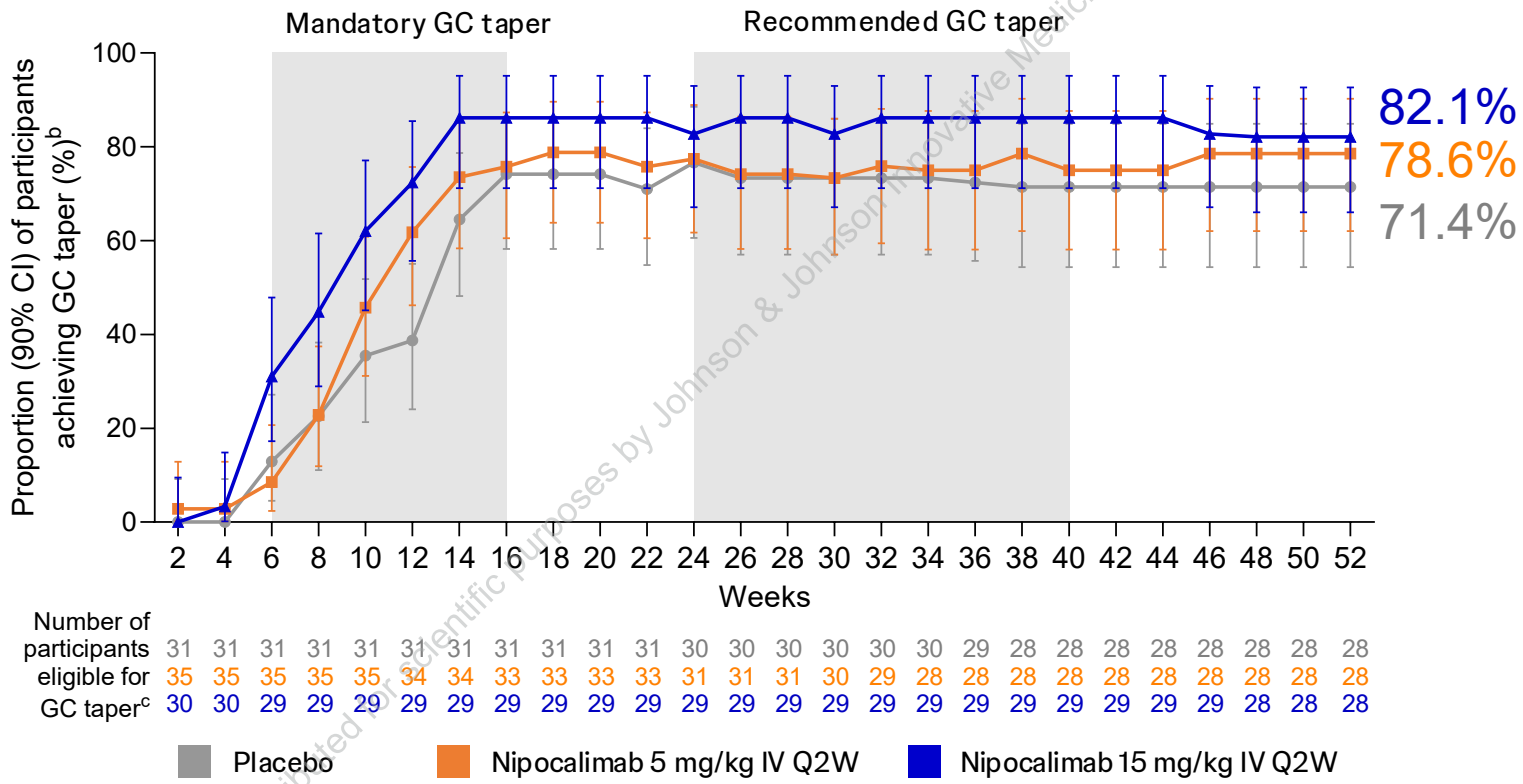
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Exploratory endpoints. Participants who initiated or adjusted concomitant medication that meets Treatment Failure criteria (ICE1) or discontinued study intervention for any reason other than COVID-19 related operational or logistical issues (ICE2) were considered as non-responders. [a]Defined as ≥5-point (SRI-5) or ≥6-point (SRI-6) reduction from baseline in SLEDAI-2K total score, no new BILAG A or >1 new BILAG B domain score, no worsening in PGA scores >10%, and not meeting any treatment failure criteria. [b]Analysis based on average response rates with imputed data. [c]*P* value was nominal.

aAb=autoantibody, **ANA**=antinuclear antibody, **BILAG**=British Isles Lupus Assessment Group Scale, **CI**=confidence interval, **dsDNA**=double-stranded deoxyribonucleic acid, **IV**=intravenous, **mFAS**=modified full analysis set, **OR**=odds ratio, **PGA**=Physician's Global Assessment of Disease Activity, **Q2W**=every 2 weeks, **RNP**=ribonucleoprotein, **SLEDAI-2K**=Systemic Lupus Erythematosus Disease Activity Index 2000, **SRI**=Systemic Lupus Erythematosus Responder Index.

Oral GC Taper

A higher proportion of participants in the nipocalimab 15 mg/kg group versus placebo achieved oral GC taper (≤7.5 mg/day) over time[a]

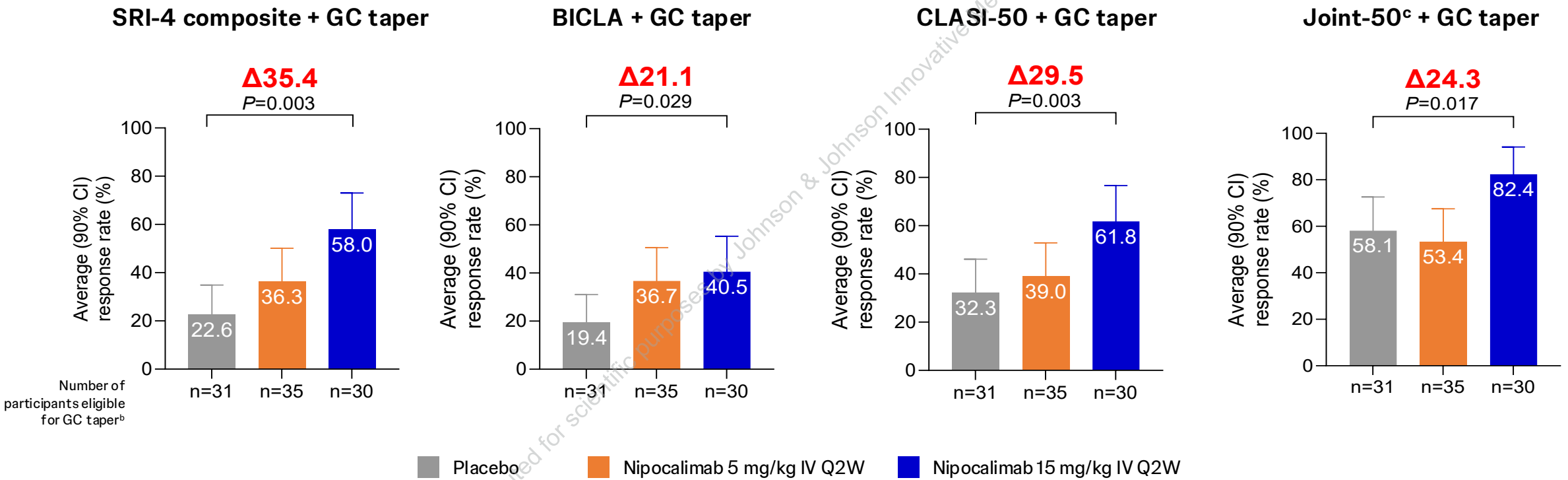


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Analyses were conducted post-hoc. [a]Based on the mFAS. [b]Proportion of participants eligible for GC taper, who achieved taper goal (≤7.5 mg/day prednisone or equivalent) through Week 52. [c]Participants who were receiving ≥10 mg/day prednisone or equivalent for SLE at baseline.
GC=glucocorticoid, **IV**=intravenous, **mFAS**=modified full analysis set, **Q2W**=every 2 weeks.

Oral GC Taper

Nipocalimab improved response rates for achieving oral GC taper goal and selected endpoints at Week 24[a]



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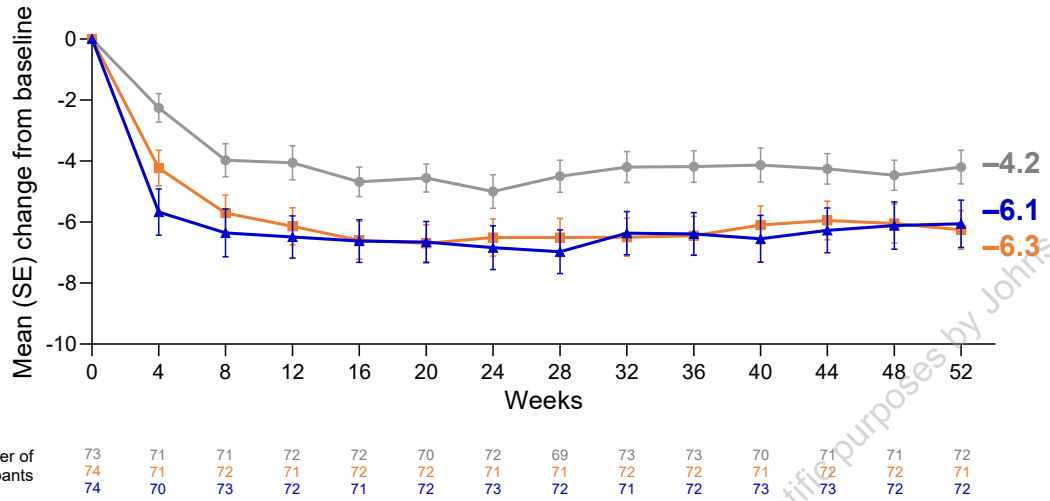
Analyses were conducted post-hoc. [a]Proportion of participants eligible for GC taper, who achieved Week 6–16 taper goal (≤7.5 mg/day prednisone or equivalent), maintained that reduction through Week 24, and achieved SRI-4 composite response, BICLA response, CLASI-50 (≥50% reduction in CLASI activity score) or Joint-50 (≥50% reduction in active joints) response at Week 24. Based on the mFAS with imputed data. [b]Participants who were receiving ≥10 mg/day prednisone or equivalent for SLE at baseline. [c]An active joint was reported as painful by the participant and demonstrated tenderness and at least one additional sign of inflammation (eg, observed swelling such as edema or effusion) on physical examination as determined by the joint assessor.

BICLA=British Isles Lupus Assessment Group–Based Composite Lupus Assessment, **CLASI-50**=≥50% reduction in Cutaneous Lupus Erythematosus Disease Area and Severity Index, **GC**=glucocorticoid, **IV**=intravenous, **Joint-50**=≥50% reduction in active joints, **mFAS**=modified full analysis set, **Q2W**=every 2 weeks, **SRI-4**=Systemic Lupus Erythematosus Responder Index.

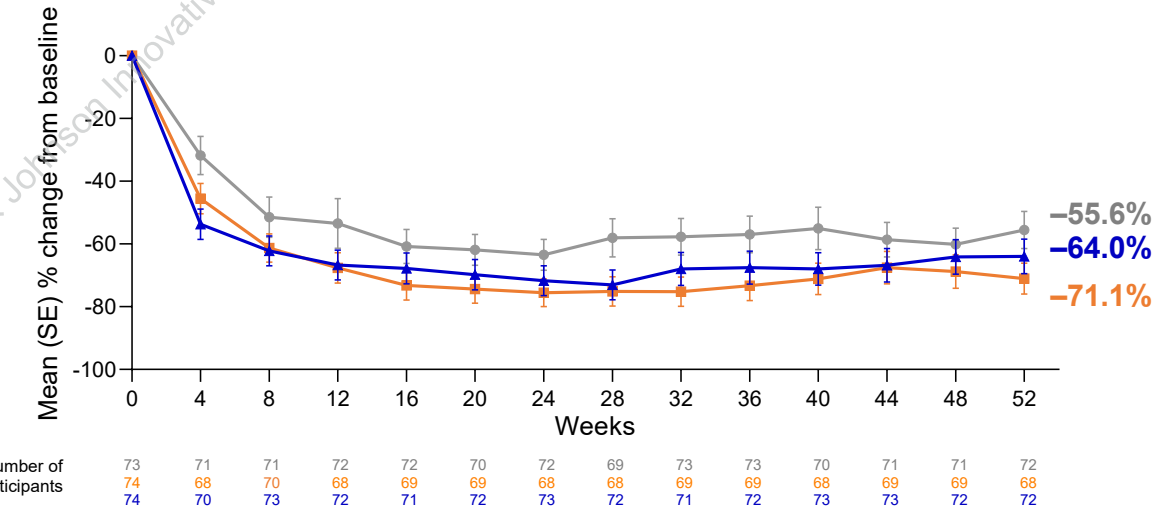
Active Joint Count Through Week 52

Improvements with nipocalimab versus placebo were observed in active joint counts

Change from baseline
(all study participants in the mFAS)



% change from baseline
(all study participants in the mFAS)



Placebo Nipocalimab 5 mg/kg IV Q2W Nipocalimab 15 mg/kg IV Q2W

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Exploratory endpoints. Participants who initiated or adjusted concomitant medication that meets Treatment Failure criteria (ICE1) or discontinued study intervention for any reason other than COVID-19-related operational or logistical issues (ICE2) were considered as no change from baseline. An active joint was reported as painful by the participant and demonstrated tenderness and at least one additional sign of inflammation (eg, observed swelling such as edema or effusion) on physical examination as determined by the joint assessor.
ICE=intercurrent event, **IV**=intravenous, **mFAS**=modified full analysis set, **Q2W**=every 2 weeks, **SE**=standard error.

Predefined Biomarker-Based Populations

Efficacy was evaluated in 3 predefined biomarker-based populations based on nipocalimab's MOA

aAb-positive (79%)

- Positive for ≥1:
  - Anti-dsDNA
  - Anti-Smith
  - ANA with anti-RNP, anti-Ro, or historical anti-dsDNA
- Positive for a range of additional autoantibodies based on extended profiling

aAb-high (15%)

- Identified using machine learning through unsupervised clustering of baseline aAb levels
- aAb-high population demonstrates:
  - Increased CICs
  - High IFN signature
  - Low C3

IFN-high (66%)

- Defined using a peripheral blood RNA signature[1]

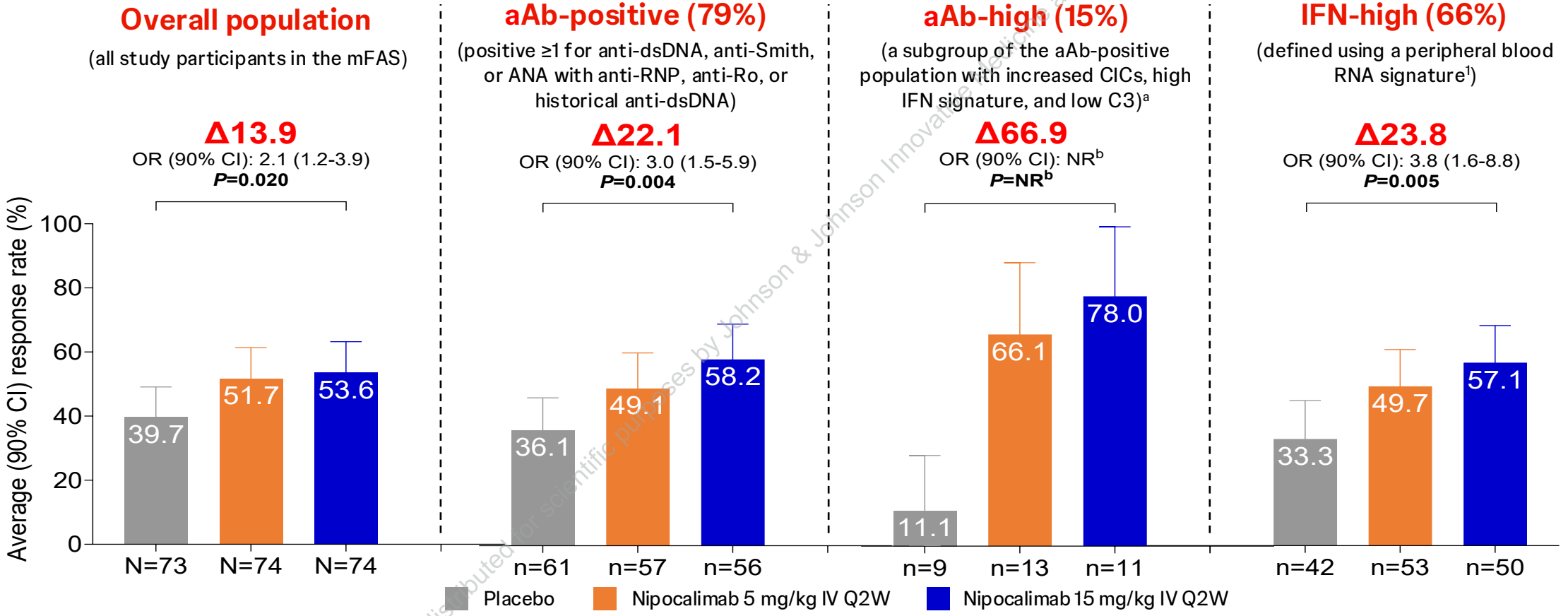
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aAb=autoantibody, **ANA**=antinuclear antibody, **C3**=complement component 3, **CIC**=circulating immune complex, **dsDNA**=double-stranded deoxyribonucleic acid, **IFN**=interferon, **MOA**=mechanism of action, **RNP**=ribonucleoprotein, **SLE**=systemic lupus erythematous.

1. Cesaroni M, et al. Type i interferon signatures and methods of use. WO2020084591A1. Issued October 25, 2019.

SRI-4 Composite Response at Week 52

Nipocalimab improved Week 52 SRI-4 composite response rates compared with placebo, with greater response rates in predefined biomarker-based populations



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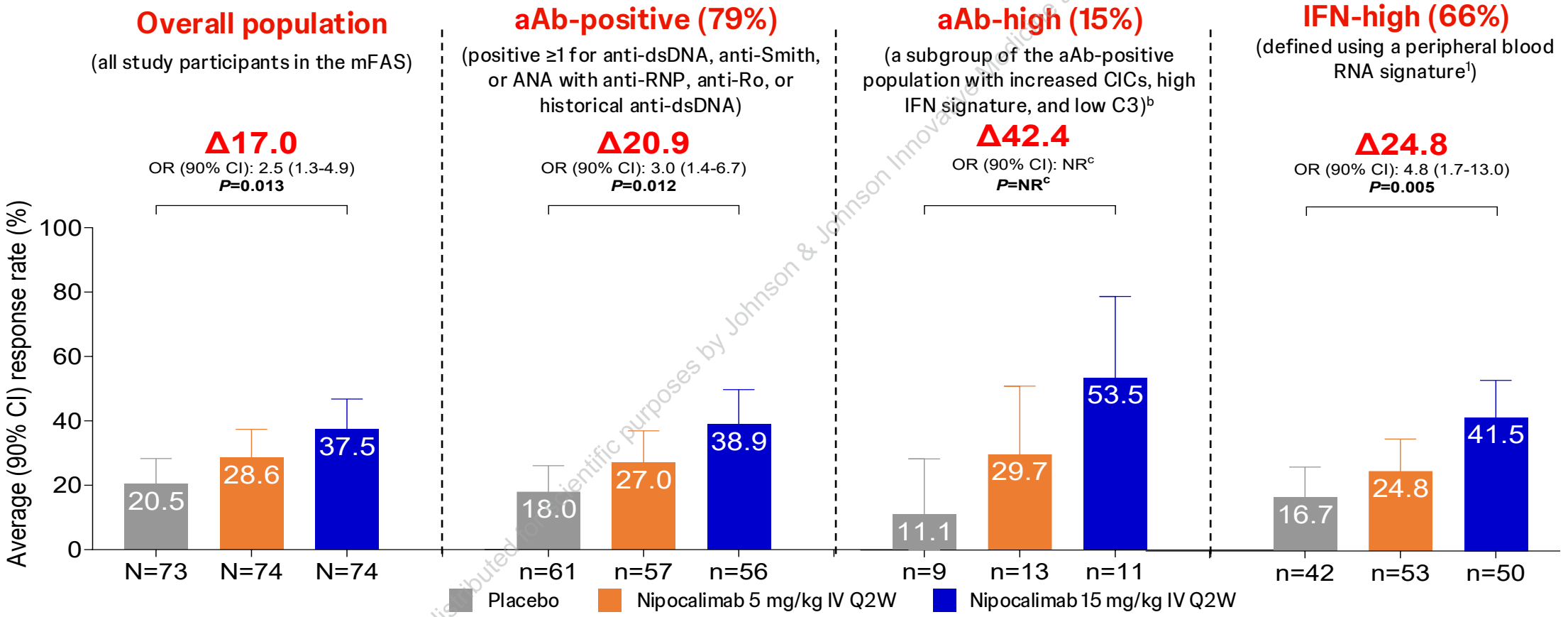
Subgroup analyses were conducted post-hoc. Analyses based on the mFAS with imputed data. All *P* values reported here are nominal. [a]Identified through unsupervised consensus clustering based on semiquantitative baseline aAb intensities. [b]Due to small sample sizes and large variability, the OR and *P* value are NR.

aAb=autoantibody, **ANA**=antinuclear antibody, **C3**=complement component 3, **CI**=confidence interval, **CIC**=circulating immune complex, **dsDNA**=double-stranded deoxyribonucleic acid, **IFN** =interferon, **IV**=intravenous, **mFAS**=modified full analysis set, **NR**=not reported, **OR**=odds ratio, **Q2W**=every 2 weeks, **RNP**=ribonucleoprotein, **SRI-4**=Systemic Lupus Erythematosus Responder Index.

1. Cesaroni M, et al. Type i interferon signatures and methods of use. WO2020084591A1. Issued October 25, 2019.

LLDAS Response at Week 52

Nipocalimab improved Week 52 LLDAS[a] response rates compared with placebo, with greater response rates in predefined biomarker-based populations



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Subgroup analyses were conducted post-hoc. Analyses based on the mFAS with imputed data. All *P* values reported here are nominal. [a]LLDAS-7.5. [b]Identified through unsupervised consensus clustering based on semiquantitative baseline aAb intensities. [c]Due to small sample sizes and large variability, the OR and *P* value are NR.

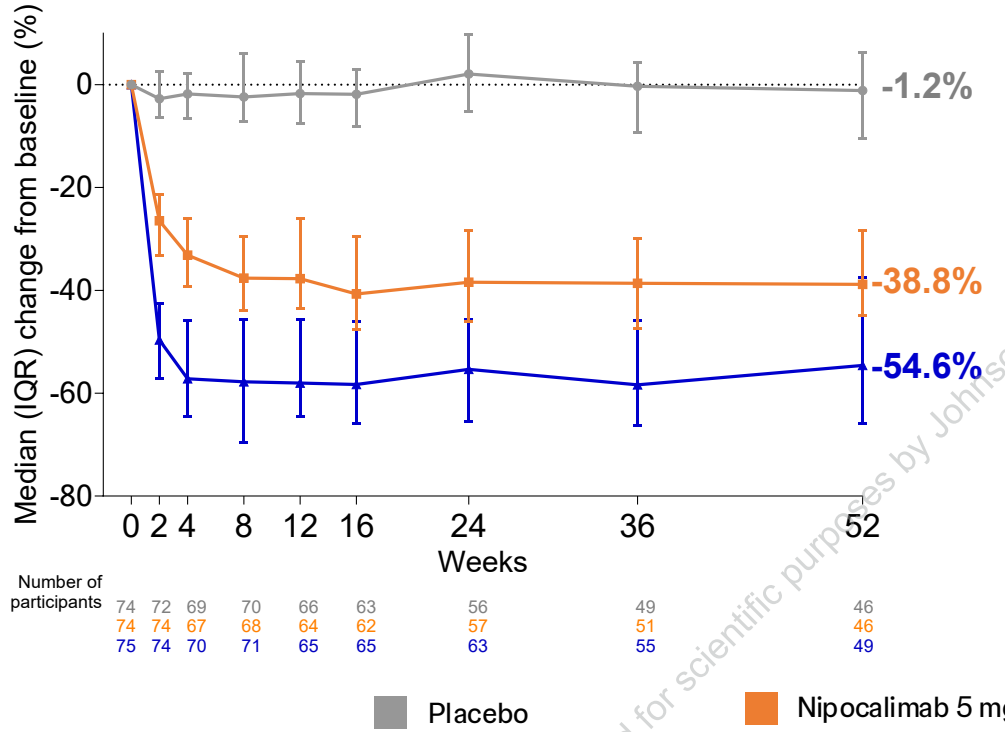
aAb=autoantibody, **ANA**=antinuclear antibody, **C3**=complement component 3, **CI**=confidence interval, **CIC**=circulating immune complex, **dsDNA**=double-stranded deoxyribonucleic acid, **IFN**=interferon, **IV**=intravenous, **LLDAS**=Lupus Low Disease Activity State, **mFAS**=modified full analysis set, **NR**=not reported, **OR**=odds ratio, **Q2W**=every 2 weeks, **RNP**=ribonucleoprotein.

1. Cesaroni M, et al. Type i interferon signatures and methods of use. WO2020084591A1. Issued October 25, 2019.

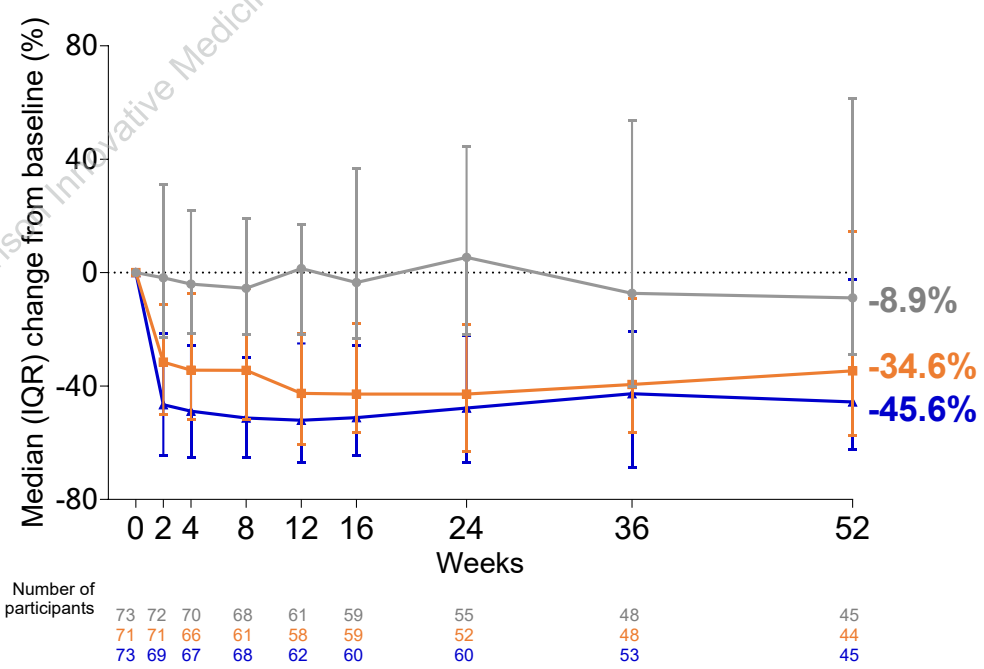
Biomarkers Through Week 52

Nipocalimab reduced total IgG and CIC levels across the treatment period[a,b]

Total IgG (Pre-Dose)[c,d]



CIC



Placebo | Nipocalimab 5 mg/kg IV Q2W | Nipocalimab 15 mg/kg IV Q2W

- No notable changes from baseline in IgM, IgE, or IgA levels were observed in any group

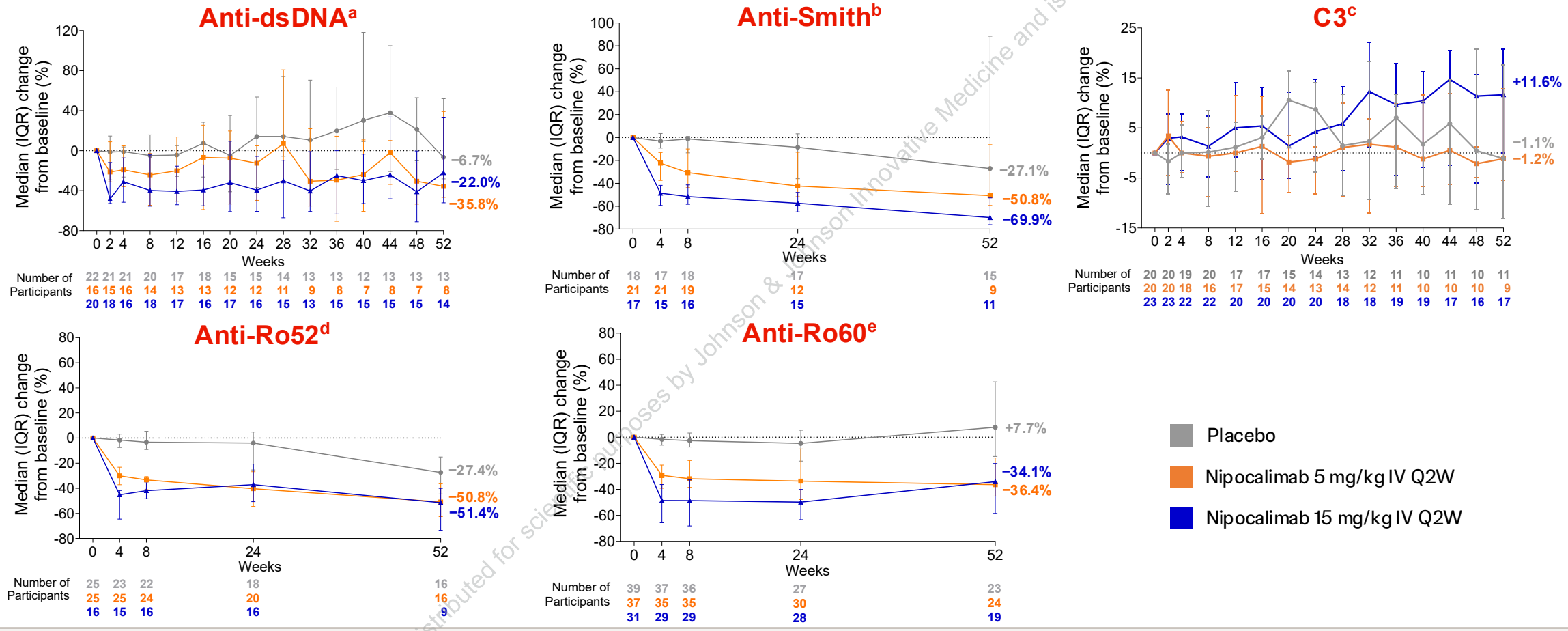
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Exploratory endpoints. [a]PD was assessed pre-dose and does not represent maximal reduction. [b]Based on the FAS with ≥1 valid post-dose blood sample drawn for pharmacodynamic analysis. If a participant missed a planned dose of study intervention at any visit, their data was excluded from all subsequent visits after the first occurrence of a missed dose. [c]20 (27%) participants in the nipocalimab 5 mg/kg group and 47 (63%) in the nipocalimab 15 mg/kg group had minimum IgG <LLN (6 g/L). [d]Model-predicted maximal IgG reduction was 71%.

CIC=circulating immune complexes, **FAS**=full analysis set, **Ig**=immunoglobulin, **IQR**=interquartile range, **IV**=intravenous, **LLN**=lower limit of normal, **PD**=pharmacodynamics, **Q2W**=every 2 weeks.

Biomarkers Through Week 52

Nipocalimab reduced anti-dsDNA, anti-Smith, anti-Ro52, and anti-Ro60 autoantibody levels while increasing C3 levels across the treatment period



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Exploratory endpoints. PD was assessed pre-dose and does not represent maximal reduction.
Based on the FAS with ≥1 valid postdose blood sample drawn for pharmacodynamic analysis. If a participant missed a planned dose of study intervention at any visit, their data was excluded from all subsequent visits after the first occurrence of a missed dose. [a]In participants positive for anti-dsDNA (≥75 IU/mL) at baseline. [b]In participants positive for anti-Smith (≥20 CU) at baseline. [c]In participants with low baseline C3. [d]In participants positive for anti-Ro52 (≥20 CU) at baseline. [e]In participants positive for anti-Ro60 (≥20 CU) at baseline.
C3=complement component 3, **CU**=chemiluminescence units, **FAS**=full analysis set, **IV**=intravenous, **PD**=pharmacodynamics, **Q2W**=every 2 weeks.

Safety Through Week 58

Nipocalimab was well tolerated, with no new safety signals observed

Participants with ≥1 AE,[a] n (%)	Placebo (N=75)	Nipocalimab 5 mg/kg IV Q2W (N=77)	Nipocalimab 15 mg/kg IV Q2W (N=76)	Combined nipocalimab (N=153)
AEs	57 (76.0)	69 (89.6)	63 (82.9)	132 (86.3)
Serious AEs	6 (8.0)	6 (7.8)	10 (13.2)	16 (10.5)
AEs leading to death	0	1 (1.3)	1 (1.3)	2 (1.3)
Infections and infestations	42 (56.0)	45 (58.4)	46 (60.5)	91 (59.5)
Serious infections and infestations	3 (4.0)	4 (5.2)	3 (3.9)	7 (4.6)
Opportunistic infections	0	0	1 (1.3)	1 (0.7)
Infusion reactions	6 (8.0)	10 (13.0)	5 (6.6)	15 (9.8)
Hypersensitivity reactions[b]	9 (12.0)	10 (13.0)	14 (18.4)	24 (15.7)
MACE[c]	1 (1.3)	1 (1.3)	1 (1.3)	2 (1.3)

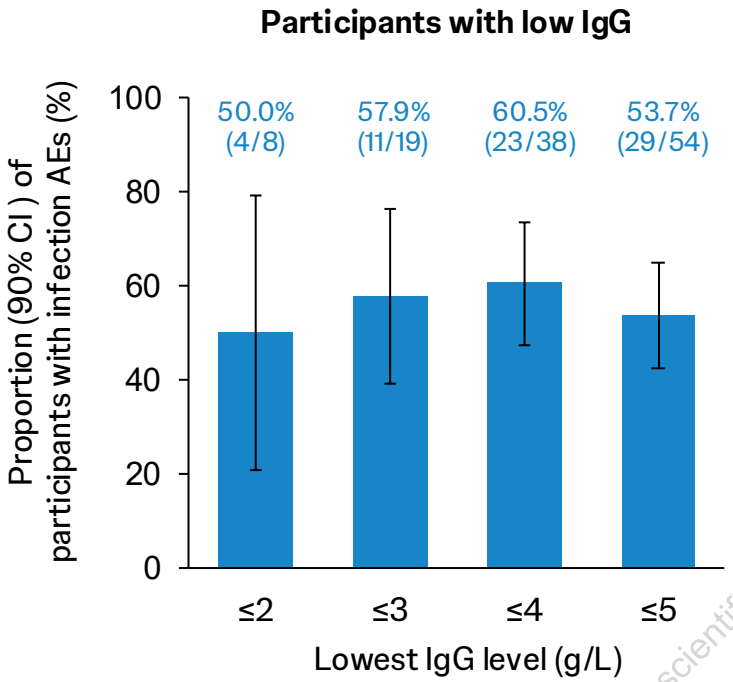
- Rates of AEs were not dose dependent
- No severe hypoalbuminemia (<20 g/L) was observed
- Total cholesterol, HDL, and LDL mean (SD) % change from baseline at Week 48 were 3.02% (16.352), 9.41% (23.768) and 2.29% (25.441), respectively, in the nipocalimab 15 mg/kg group.
- Two deaths were reported in participants from each nipocalimab treatment group due to septic shock. Both events were assessed by the investigator as unrelated to the study intervention

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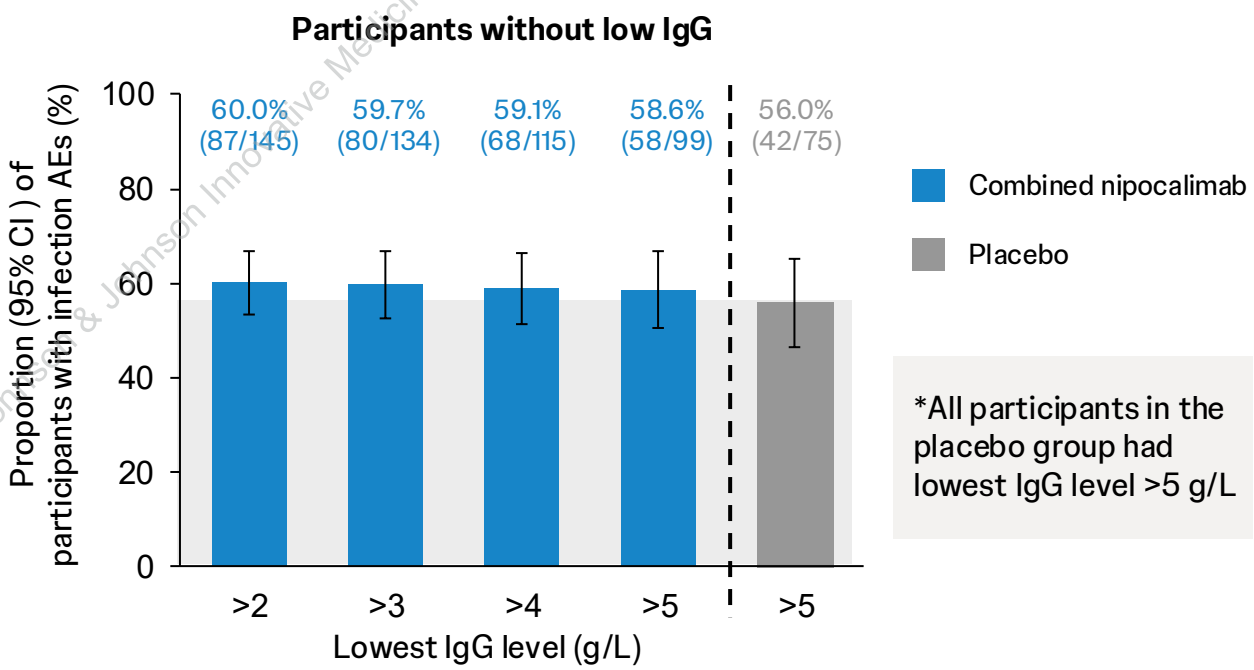
[a]Based on the FAS. [b]Defined by the Standardized Medical Dictionary for Regulatory Activities (MedDRA) Queries. [c]Cardiovascular death, nonfatal myocardial infarction, and nonfatal stroke.
AE=adverse event, **FAS**=full analysis set, **IgG**=immunoglobulin G, **IV**=intravenous, **MACE**=major adverse cardiovascular events, **Q2W**=every 2 weeks.

Lack of Association Between Low IgG Level and Occurrence of Infection

Infection frequency did not increase with lower IgG levels[a]



Infection rates were consistent between nipocalimab and placebo across IgG levels



*All participants in the placebo group had lowest IgG level >5 g/L

- Nadir IgG ≤1 g/L was observed in only 1 participant treated with nipocalimab and no serious AEs (including serious infections) were reported through Week 58

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Analyses were conducted post-hoc. [a]Infections occurred after the time point when the lowest IgG level occurred.
AE=adverse event, **IgG**=immunoglobulin G, **IV**=intravenous, **NA**=not applicable, **Q2W**=every 2 weeks.

Key Takeaways

In JASMINE-SLE, the first study of an FcRn blocker in SLE, nipocalimab 15 mg/kg IV Q2W treatment led to:

- ✓ Significant improvement over placebo in SRI-4 composite response at Week 24 and improved SRI-4 composite and LLDAS responses at Week 52
- ✓ Sustained oral GC reduction
- ✓ Greater clinical effects in populations of aAb-positive, aAb-high, and IFN-high participants
- ✓ Reductions in total IgG, IgG autoantibodies, CICs; and increase in C3 levels

✓ Nipocalimab was well tolerated among participants

These results establish proof of concept for FcRn blockade with nipocalimab in SLE and support the evaluation of nipocalimab in an ongoing phase 3 trial (GARDENIA; NCT07438496)

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aAb=autoantibody, **FcRn**=neonatal crystallizable fragment receptor, **GC**=glucocorticoid, **IFN**=interferon, **IV**=intravenous, **LLDAS**=Lupus Low Disease Activity State, **Q2W**=every 2 weeks, **SLE**=systemic lupus erythematous, **SRI-4**=Systemic Lupus Erythematosus Responder Index.

Additional JASMINE Presentations at ACR 2026

Pharmacodynamics of Nipocalimab, a Fully Human FcRn-Blocking Monoclonal Antibody, in Adults With Systemic Lupus Erythematosus: Results From a Phase 2, Multicenter, Randomized, Placebo-Controlled, Double-Blind Study (JASMINE-SLE)

Nipocalimab led to dose-dependent reductions in total IgG and all IgG subclasses; multiple IgG aAbs; and CICs; as well as an increase in C3.

Poster Session A: Sun 11 Nov, 10:30-12:30, Hall WB1&2
0275 (Petri M, et al.)

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Relationship Between IgG Levels and Infection Rates in Nipocalimab Treated Participants With Systemic Lupus Erythematosus (SLE) in the Phase 2 JASMINE-SLE Study

Nipocalimab reduced levels of IgG, but the incidence of infection was comparable with placebo and there was no predictive association between IgG levels and infection risk

Poster Session A: Sun 11 Nov, 10:30-12:30, Hall WB1&2
0279 (Furie R, et al.)

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Acknowledgments

- We thank all study participants, steering committee members, investigators, and study site staff for their contributions to the JASMINE-SLE study
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- This work was previously presented at the European Alliance of Associations for Rheumatology (EULAR) European Congress; 3-6 June 2026; London, England, UK.

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