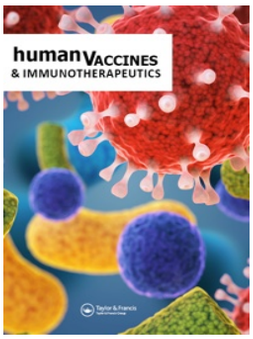

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Effect of nipocalimab on IgG responses to vaccinations and viral infections in patients with IgG autoantibody-mediated diseases: Post hoc analyses of three randomized, placebo-controlled trials

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ABSTRACT

Nipocalimab is a neonatal Fc receptor (FcRn)–blocking monoclonal antibody approved for the treatment of generalized myasthenia gravis (gMG) and is being evaluated for other immunoglobulin G (IgG) autoantibody- or alloantibody-mediated diseases. Nipocalimab binds to FcRn with high specificity and affinity, eliciting increased clearance of IgG antibodies without affecting IgG production or other immune functions. However, nipocalimab's impact on vaccine responses in patients has not been previously reported. The effect of nipocalimab on pre-existing antibodies against tetanus toxoid (TT) and herpes zoster virus (HZV) vaccines as well as humoral responses to TT vaccines, severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) vaccines, and SARS-CoV-2 infections was examined in post hoc analyses of data from three randomized, placebo-controlled trials in participants with gMG, rheumatoid arthritis, and Sjögren's disease. The levels of pre-existing anti-TT and anti-HZV IgG followed the kinetics of total IgG during nipocalimab treatment (60%–65% and 53%–68% IgG reduction, respectively) and returned to baseline after discontinuation. Nevertheless, the majority of nipocalimab-treated participants maintained pre-existing anti-HZV (66/90, 73.3% above ≥100 IU/L reference threshold) and anti-TT IgG levels (62/81, 76.5% ≥0.16 IU/mL protective threshold) throughout the study period. Participants treated with nipocalimab elicited positive IgG responses to TT and SARS-CoV-2 vaccination, similar to placebo-treated participants. SARS-CoV-2 infections during the studies were mild to moderate in severity with no complications. These results suggest that nipocalimab does not impair the development of humoral responses to vaccines or viral infections in patients with IgG autoantibody-mediated diseases.

Trial registration number: NCT04951622, NCT04991753, NCT04968912.

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KEYWORDS
Non-live vaccine; infections; humoral response; nipocalimab; FcRn blocker; immunoglobulin G

Introduction

Pathogenic immunoglobulin G (IgG) autoantibodies play a key role in many autoimmune diseases, such as generalized myasthenia gravis (gMG), rheumatoid arthritis (RA), and Sjögren's disease (SjD).[1–3] The neonatal Fc receptor (FcRn) regulates serum IgG levels by protecting IgG from degradation, thereby prolonging its serum half-life.[4] Blockade of FcRn has been validated as an effective treatment approach for some IgG autoantibody-mediated diseases through the reduction of IgG levels by increasing clearance of IgG antibodies without affecting IgG production.[5–7] Nipocalimab is an FcRn blocker that has been recently approved for the treatment of gMG[7] and is currently under evaluation for the treatment of a wide range of IgG autoantibody- or alloantibody-mediated diseases.[2,3,8,9]

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Nipocalimab is a fully human, aglycosylated IgG1 monoclonal antibody that binds to the IgG binding site of FcRn with high specificity and affinity in a pH-independent manner and lacks Fc-mediated effector functions.[9] Across multiple phase 1, 2, and 3 studies in healthy participants and in participants with IgG autoantibody- or alloantibody-mediated diseases, including gMG, RA, SjD, and hemolytic disease of the fetus and newborn, nipocalimab has demonstrated rapid, substantial, dose-dependent, and reversible lowering of circulating IgG levels, with a maximum reduction of up to 85% from baseline within 2 weeks after administration without impacting IgG production and other cell-mediated immune functions.[2,3,10–12]

In an immunotoxicity study in cynomolgus monkeys, nipocalimab did not affect the immune response to T-cell-dependent keyhole limpet hemocyanin (KLH) antigen.[9] In a phase 1, open-label study in healthy adults, nipocalimab did not impact the development of an adequate IgG response to T-cell-dependent and -independent vaccines (ie, tetanus toxoid [TT], diphtheria, and acellular pertussis vaccine [Tdap] and 23-polysaccharide pneumococcal vaccine [PPSV®23], respectively), despite a median (predose, minimal) reduction in total IgG levels of 65.9% from baseline to Week 4.[13] However, as vaccine responses can be diminished in individuals with autoimmune diseases due to their disease states and/or treatments (eg, methotrexate, corticosteroids), we investigated the impact of nipocalimab on pre-existing anti-vaccine antibodies and the IgG responses to routine vaccinations or infection in patients with autoimmune diseases. Here, we report results from post hoc analyses of data from three randomized, placebo-controlled trials to explore the effect of nipocalimab on the levels of pre-existing antibodies against vaccines (TT and herpes zoster virus [HZV]) and on humoral responses to vaccines (severe acute respiratory syndrome coronavirus 2 [SARS-CoV-2] and TT) and viral infections (SARS-CoV-2) in participants with gMG, RA, and SjD on nonbiologic standard of care (SOC) therapy.

Materials and methods

Participants, trials designs, and interventions

These post hoc analyses included participants from Vivacity-MG3 (ClinicalTrials.gov: NCT04951622), IRIS-RA (NCT04991753), and DAHLIAS (NCT04968912). Details of study designs, inclusion and exclusion criteria, and primary results have been reported.[2,3,10]

Vivacity-MG3 is a phase 3, randomized, double-blind, multicenter study comprising a placebo-controlled period (24 weeks) and an ongoing open-label extension phase in adult participants with gMG inadequately controlled with SOC therapy.[2] Participants were randomly assigned (1:1) to intravenous (IV) treatment with either nipocalimab (30 mg/kg loading dose then 15 mg/kg every 2 weeks [Q2W] for maintenance dosing) or placebo Q2W, which was administered in conjunction with SOC therapy during the double-blind period from Week 0 through Week 22.[2] The post hoc analysis included participants from Vivacity-MG3 through Week 24.

In the IRIS-RA phase 2a, randomized, double-blind study, adult participants with moderate-to-severe, active RA seropositive for anticitrullinated protein antibodies or rheumatoid factors were randomized (3:2) to nipocalimab 15 mg/kg or placebo IV Q2W from Week 0 through Week 10.[3] The post hoc analysis included participants from IRIS-RA through Week 18.

DAHLIAS was a phase 2, double-blind, multicenter trial of adult participants with moderate-to-severe, active SjD who were seropositive for anti-Ro IgG autoantibodies and were randomized (1:1:1) to receive nipocalimab 5 mg/kg, 15 mg/kg, or placebo IV Q2W from Week 0 through Week 22.[10] This post hoc analysis included participant data through Week 30 and included only participants treated with 15 mg/kg IV Q2W, as this dose regimen is clinically relevant for the conditions being investigated.

Assessments

Anti-TT and anti-HZV IgG levels were assessed in all participants from the IRIS-RA and DAHLIAS studies and in a subset of participants (51 of 206 randomized) from the Vivacity-MG3 study who had available biomarker samples at relevant time points (IRIS-RA: Weeks 0, 4, 8, 12, 18; DAHLIAS: Weeks 0, 2, 4, 8, 16, 24, 30; Vivacity-MG3: 0, 2, 4, 8, 12, 16, 20, 24). Anti-TT-specific IgG level evaluations were performed using the anti-TT enzyme-linked immunosorbent assay (ELISA) IgG kit

(EUROIMMUN, EI 2060–9601 G) according to manufacturer's instructions. Anti-HZV IgG levels were assessed using the anti-HZV ELISA IgG kit (EUROIMMUN, EI 2650–9601 G) according to manufacturer's instructions. The effects of nipocalimab on pre-existing anti-TT and HZV IgG levels were evaluated in participants with baseline levels above respective thresholds (protective threshold ≥0.16 IU/mL for TT[14]; reference threshold ≥100 IU/L for HZV[15–17]) that generally suggest a sufficient level of immunity to provide protection. In Vivacity-MG3, participants who received TT vaccines during the double-blind, placebo-controlled period were also evaluated for anti-TT IgG levels.

Participants who received SARS-CoV-2 vaccines and/or had documented SARS-CoV-2 infections during the studies were evaluated for total IgG and anti-SARS-CoV-2-specific IgG (against spike protein, spike protein S1 receptor-binding domain [S1 RBD], and nucleocapsid) levels at the relevant time points. Serum concentration of total IgG was measured using the Tina-quant IgG Gen.2 assay (Roche Diagnostics, 05220718190), and anti-SARS-CoV-2-specific IgG levels were measured using the V-PLEX COVID-19 Coronavirus Panel 3 (IgG) Kit (Meso Scale Diagnostics, K15399U-2) according to manufacturer's instructions with data analysis using Discovery Workbench software version 4 (Meso Scale Diagnostics, Rockville, MD).

Data analysis

All participants with a baseline sample and ≥1 other time point available for comparison were included in the analyses. All analyses were based on actual intervention received, and data were summarized using descriptive statistics for continuous and categorical variables (using the number of observations and percentages as appropriate). Pre-existing vaccine-induced IgG were summarized as median (interquartile range) by treatment group and time point. Total serum IgG and antibody responses against vaccination/infections during the study period were plotted graphically.

Ethics

All three studies were conducted in accordance with the Declaration of Helsinki and were consistent with Good Clinical Practice guidelines. Participants or their legally acceptable representatives provided written informed consent to participate in the studies. Protocols and other relevant documents were reviewed and approved by Independent Ethics Committees and Institutional Review Boards.

Results

Participants

Baseline characteristics of the participants in Vivacity-MG3, IRIS-RA, and DAHLIAS were generally well balanced between the nipocalimab and placebo groups, with detailed baseline and clinical characteristics previously reported for each study population.[2,3,10] All participants in Vivacity-MG3 received concomitant SOC therapy with corticosteroids or other immunosuppressive agents, whereas approximately 96% of participants in IRIS-RA and 80% of participants in DAHLIAS received concomitant SOC therapy with these agents.[2,3,10] Participants in Vivacity-MG3 received multiple concomitant SOC medications for gMG, including pyridostigmine, immunosuppressants (eg, mycophenolate mofetil, tacrolimus, azathioprine), and glucocorticoids (eg, prednisone), as well as medications for common comorbidities, such as hypertension, hyperlipidemia, and type 2 diabetes. Participants in IRIS-RA who received concomitant SOC therapy for RA were treated with conventional synthetic disease-modifying antirheumatic drugs (csDMARDS; eg, methotrexate, sulfasalazine, hydroxychloroquine/chloroquine, leflunomide), glucocorticoids (eg, methylprednisolone, prednisolone, prednisone), as well as nonsteroidal anti-inflammatory drugs. Participants in DAHLIAS who received concomitant SOC therapy for SjD were treated with antimalarials (eg, hydroxychloroquine/chloroquine), immunomodulators (eg, azathioprine, methotrexate, mycophenolate mofetil, leflunomide, sulfasalazine), and systemic glucocorticoids (eg, methylprednisolone,

prednisolone, prednisone, budesonide) at ≤10 mg/d prednisone-equivalent doses. Concomitant use of biologic SOC was not permitted in any of these trials, because of the potential for drug–drug interactions with nipocalimab due to its mechanism of action.

Concomitant therapies used by participants who received TT or SARS-CoV-2 vaccines in this analysis are shown in Table S1. Among participants who received a TT vaccine during the studies, all received concomitant SOC therapy, including corticosteroids and other immunosuppressive agents. Of the participants who received a SARS-CoV-2 vaccine during the study, nearly all from Vivacity-MG3 (22/24) and IRIS-RA (6/6) received concomitant SOC therapy with corticosteroids and/or other immunosuppressive agents, whereas none of the four participants from DAHLIAS received concomitant therapy with these agents.

Pre-existing anti-HZV IgG levels

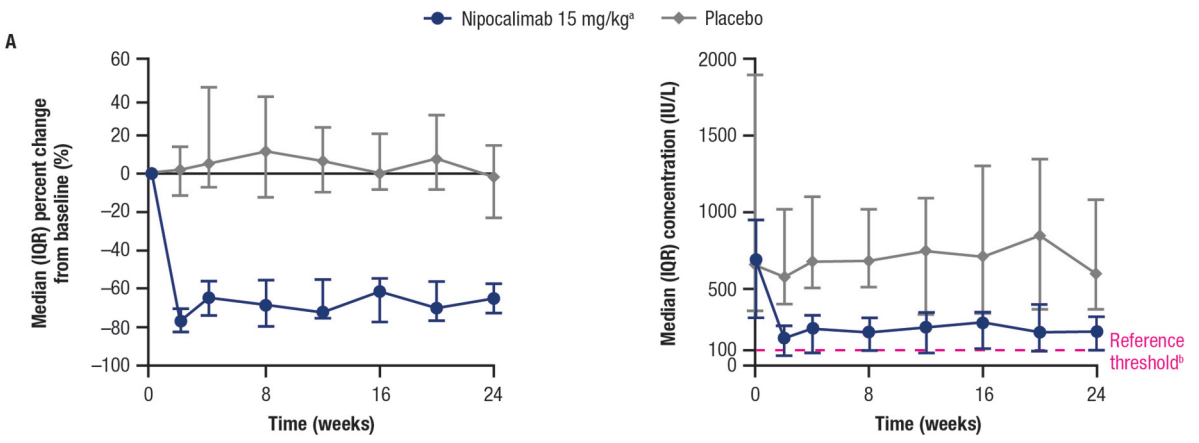
The anti-HZV analysis included 38 participants (nipocalimab: n = 20; placebo: n = 18) from Vivacity-MG3, 45 (nipocalimab: n = 28; placebo: n = 17) from IRIS-RA, and 105 (nipocalimab: n = 50; placebo: n = 55) from DAHLIAS. Not all participants included in the analysis completed the study and/or had samples available at every time point. For this analysis, all participants who had samples at baseline and ≥1 other time point (nipocalimab: n = 98; placebo: n = 90) were included. Across the three studies, 91.8% (90/98) of participants receiving nipocalimab had a baseline anti-HZV IgG level ≥100 IU/L, a reference threshold that generally suggests a sufficient level of immunity to provide protection against HZV.[15–17] Nipocalimab reduced the pre-existing anti-HZV IgG by 60% to 65%, consistent with the effect on total IgG in the overall trial populations (Table 1).[3,10,18] Despite this reduction, 73.3% (66/90) of nipocalimab-treated participants with a baseline anti-HZV IgG level ≥100 IU/mL maintained anti-HZV IgG levels ≥100 IU/L at all available time points measured during the respective study periods included in this post hoc analysis (Figure 1).

Within 8 weeks of the cessation of nipocalimab treatment in IRIS-RA and DAHLIAS, anti-HZV IgG levels returned to baseline values in nearly all participants with available samples at the analyzed time point. In IRIS-RA, 63.6% (14/22) of nipocalimab-treated participants maintained anti-HZV IgG levels ≥100 IU/L at Week 12 (ie, 2 weeks after the last dose) and 91.3% (21/23) at Week 18 (ie, 8 weeks after the last dose), while 56.0% (14/25) maintained levels at all available time points measured. In DAHLIAS, 90.0% (36/40) of participants receiving nipocalimab 15 mg/kg maintained anti-HZV IgG levels ≥100 IU/L at Week 24 (ie, 2 weeks after the last dose) and 100% (39/39) at Week 30 (ie, 8 weeks after the last dose), compared with 87.0% (40/46) who maintained levels at all available time points measured. In Vivacity-MG3, 61.1% (11/18) of nipocalimab-treated participants maintained anti-HZV IgG levels ≥100 IU/L at Week 24 (ie, 2 weeks after the last double-blind dose, prior to entry in the open-label extension), while 63.2% (12/19) maintained levels ≥100 IU/L at all available time points measured.

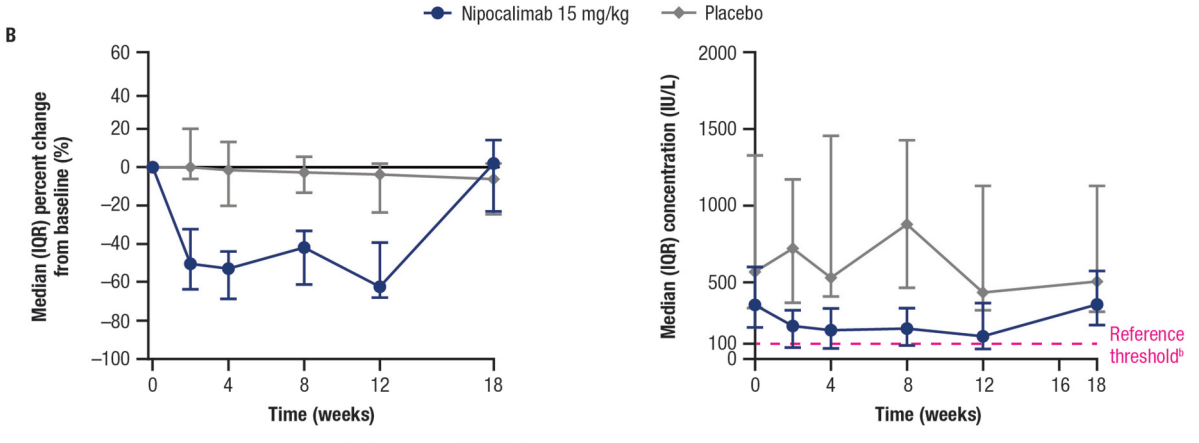
Table 1. Median IgG level reduction from baseline.

Median (IQR) reduction from baseline, %	Vivacity-MG3 Week 24		IRIS-RA Week 12		DAHLIAS Week 24	
	Nipocalimab	Placebo	Nipocalimab	Placebo	Nipocalimab 15 mg/kg	Placebo
Total IgG[a]	(n = 97) −68.8 (−75.3, −62.2)	(n = 97) −0.4 (−5.0, 5.4)	(n = 32) −62.1 (−50.3, −70.8)	(n = 20) 3.8 (−5.2, 12.3)	(n = 54) −60.9 (−65.9, −45.7)	(n = 56) −0.5 (−6.8, 5.0)
Anti-HZV IgG	(n = 18) −65.0 (−72.6, −57.0)	(n = 14) −1.5 (−22.8, 15.1)	(n = 22) −61.3 (−66.9, −38.6)	(n = 15) −3.7 (−23.2, 1.9)	(n = 40) −60.0 (−70.2, −45.1)	(n = 46) −2.1 (−14.4, 17.7)
Anti-TT IgG	(n = 15) −68.3 (−74.5, −49.8)	(n = 12) 5.7 (−7.5, 10.0)	(n = 25) −64.7 (−72.7, −60.2)	(n = 14) 0.0 (−11.0, 2.5)	(n = 31) −52.6 (−60.5, −32.3)	(n = 39) −2.4 (−11.9, 8.0)

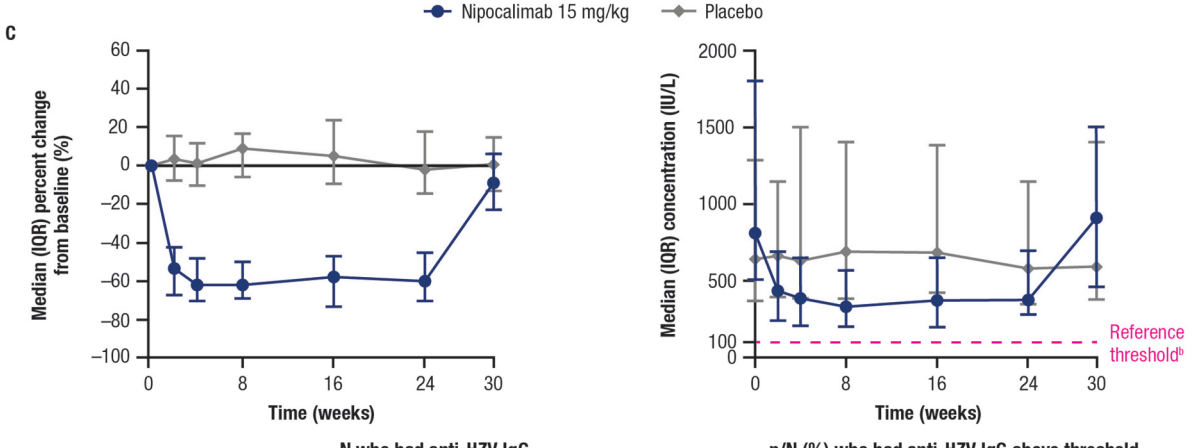
HZV, herpes zoster virus; IgG, immunoglobulin G; IQR, interquartile range; TT, tetanus toxoid.
[a]Total IgG data based on results from overall population in each trial.



Treatment group	Total N	N who had anti-HZV IgG above threshold at Week 0	n/N (%) who had anti-HZV IgG above threshold at Week 24	n/N (%) who had anti-HZV IgG above threshold at all available time points
Nipocalimab	20	19	11/18 (61%)	12/19 (63%)
Placebo	18	16	14/14 (100%)	15/16 (94%)



Treatment group	Total N	N who had anti-HZV IgG above threshold at Week 0	n/N (%) who had anti-HZV IgG above threshold at Week 12	n/N (%) who had anti-HZV IgG above threshold at Week 18	n/N (%) who had anti-HZV IgG above threshold at all available time points
Nipocalimab	28	25	14/22 (64%)	21/23 (91%)	14/25 (56%)
Placebo	17	17	15/15 (100%)	17/17 (100%)	17/17 (100%)



Treatment group	Total N	N who had anti-HZV IgG above threshold at Week 0	n/N (%) who had anti-HZV IgG above threshold at Week 24	n/N (%) who had anti-HZV IgG above threshold at Week 30	n/N (%) who had anti-HZV IgG above threshold at all available time points
Nipocalimab	50	46	36/40 (90%)	39/39 (100%)	40/46 (87%)
Placebo	55	53	46/46 (100%)	44/44 (100%)	53/53 (100%)

Figure 1. Percent change from baseline, concentration, and the proportions of participants who had pre-existing anti-HZV IgG above the reference threshold over time in (A) Vivacity-MG3, (B) IRIS-RA, and (C) DAHLIAS. HZV, herpes zoster virus; IgG, immunoglobulin G; IQR, interquartile range. [a]30 mg/kg loading dose at Week 0. [b]Reference threshold (≥100 IU/L) that generally suggests a sufficient level of immunity to provide protection against developing HZV.[15–17]

Pre-existing anti-TT IgG levels

The anti-TT analysis included 37 participants (nipocalimab: n = 21; placebo: n = 16) from Vivacity-MG3, 44 (nipocalimab: n = 28; placebo: n = 16) from IRIS-RA, and 90 (nipocalimab: n = 41; placebo: n = 49) from DAHLIAS. As with anti-HZV IgG analyses, all participants who had samples at baseline and ≥1 other time point (nipocalimab: n = 83; placebo: n = 72) were included in the analysis. A total of 92.2% (83/90) of participants receiving nipocalimab had a baseline anti-TT IgG level ≥0.16 IU/mL, a protective threshold of sufficient immunity to provide protection against tetanus infection.[14] Consistent with the reduction in pre-existing anti-HZV IgG levels, nipocalimab reduced pre-existing anti-TT IgG levels by 53% to 68% (median observed predose reduction at Weeks 24, 12, and 24 of the Vivacity-MG3, IRIS-RA, and DAHLIAS trials, respectively; Table 1). Despite this reduction, anti-TT IgG levels were maintained at the protective threshold (≥0.16 IU/mL) in 77.1% (64/83) of nipocalimab-treated participants with a baseline anti-TT IgG level ≥0.16 IU/mL at all available time points measured during the study periods included in this post hoc analysis across the three trials (Figure 2).

Within 8 weeks of the cessation of nipocalimab treatment in IRIS-RA and DAHLIAS, anti-TT IgG levels returned to baseline values in most participants with available samples at the analyzed time points. In IRIS-RA, 80.0% (20/25) of nipocalimab-treated participants maintained protective levels of anti-TT IgG at Week 12 (ie, 2 weeks after the last dose) and 92.3% (24/26) at Week 18 (ie, 8 weeks after the last dose), compared with 70.4% (19/27) who maintained levels at all time points measured. In DAHLIAS, 90.3% (28/31) of participants receiving nipocalimab 15 mg/kg IV Q2W maintained protective levels of anti-TT IgG at Week 24 (ie, 2 weeks after the last dose) and 96.8% (30/31) at Week 30 (ie, 8 weeks after the last dose), compared with 84.2% (32/38) who maintained levels at all available time points measured. In Vivacity-MG3, 70.6% (12/17) of nipocalimab-treated participants maintained protective levels of anti-TT IgG at Week 24 (ie, 2 weeks after the last double-blind dose, prior to entry in the open-label extension), while 72.2% (13/18) maintained protective levels at all available time points measured. No incidence of TT infection was reported across all three trials.

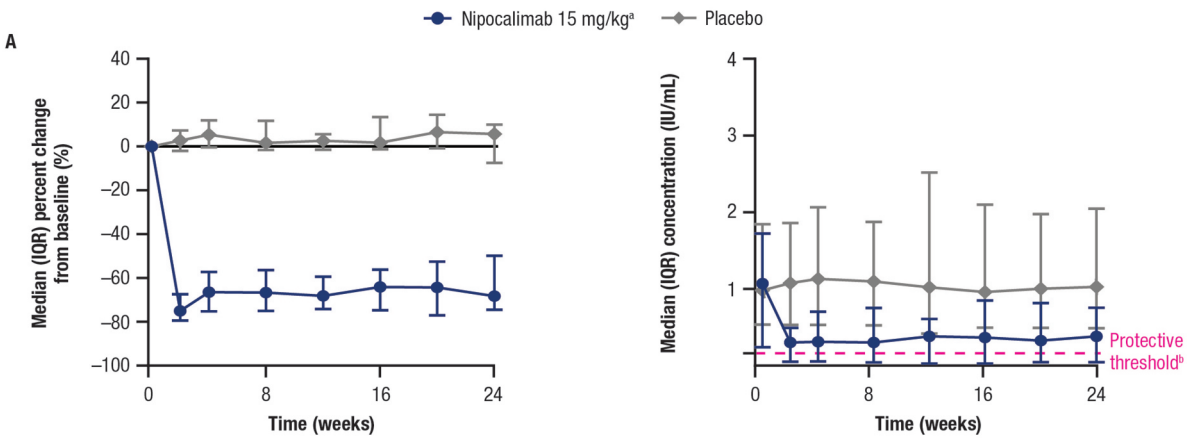
Responses to TT vaccination

In Vivacity-MG3, 3 participants (nipocalimab: n = 1; placebo: n = 2) received a TT vaccine during the study. The nipocalimab-treated participant who received TT vaccination during the study (n = 1) had a markedly increased anti-TT IgG level post-vaccination despite a reduced total IgG level. The anti-TT level was sustained above the protective threshold (0.16 IU/mL) after vaccination and through the end of the double-blind period (Figure 3). Placebo-treated participants receiving TT vaccination (n = 2) also mounted clear IgG response post vaccination (Figure S1). No apparent difference in antibody responses was observed in these participants despite receiving concomitant SOC of corticosteroids and/or immunosuppressive agents.

Responses to SARS-CoV-2 vaccination

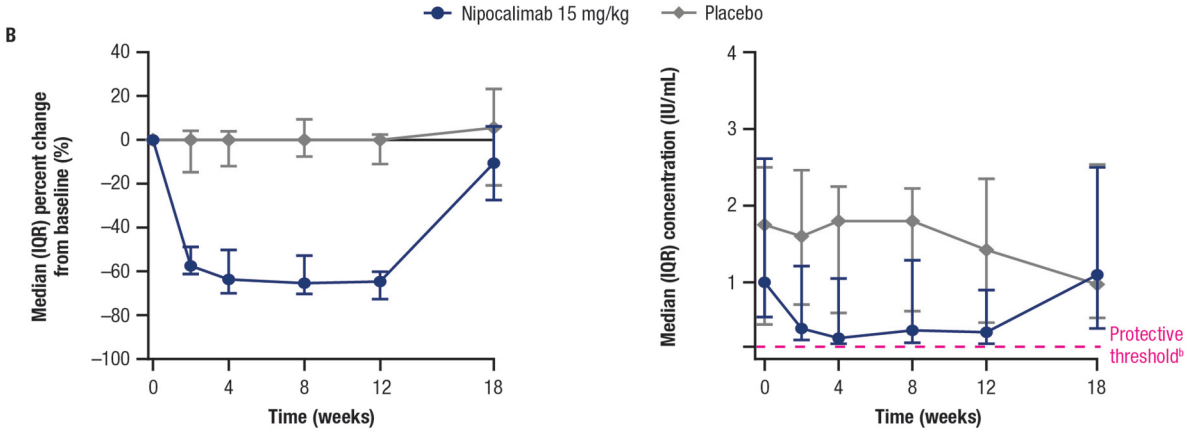
A total of 24 participants (nipocalimab: n = 12; placebo: n = 12) from Vivacity-MG3, 6 (nipocalimab: n = 3; placebo: n = 3) from IRIS-RA, and 4 (nipocalimab: n = 2; placebo: n = 2) from DAHLIAS received a SARS-CoV-2 vaccine during the trial and were included in the anti-SARS-CoV-2 analysis. Despite the reduction in total IgG levels (Figure S2), most participants who received the SARS-CoV-2 vaccine during nipocalimab treatment elicited IgG responses to the vaccine (ie, anti-spike and anti-S1 RBD), while no responses to anti-nucleocapsid levels, included as a negative control, were observed (Figure 4). The magnitude and duration of these vaccine responses varied individually, similar to the anti-SARS-CoV-2 IgG responses observed in placebo-treated participants (Figure 5).

Across studies, no apparent difference in antibody responses to the SARS-CoV-2 vaccine was observed, irrespective of concomitant SOC therapy with corticosteroids and/or immunosuppressive agents (Tables 2 and S1). Two participants treated with nipocalimab (nipocalimab 6 and 7) and 3 participants treated with placebo (placebo 3, 6, and 12) in Vivacity-MG3 also received mycophenolate mofetil, which is known to inhibit antibody responses.[19] No apparent difference in antibody responses was observed in these participants compared with those who did not receive mycophenolate mofetil.



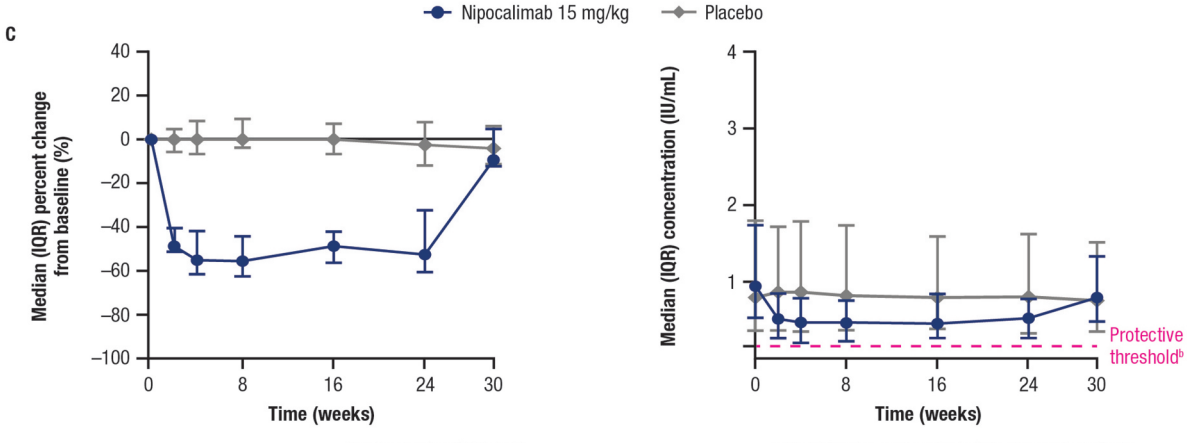
Treatment group	Total N	N who had anti-TT IgG above threshold at Week 0
Nipocalimab	21	18
Placebo	16	14

n/N (%) who had anti-TT IgG above threshold	
at Week 24	at all available time points
12/17 (71%)	13/18 (72%)
12/12 (100%)	12/12 (100%)



Treatment group	Total N	N who had anti-TT IgG above threshold at Week 0
Nipocalimab	28	27
Placebo	16	14

n/N (%) who had anti-TT IgG above threshold		
at Week 12	at Week 18	at all available time points
20/25 (80%)	24/26 (92%)	19/27 (70%)
14/14 (100%)	14/14 (100%)	14/14 (100%)



Treatment group	Total N	N who had anti-TT IgG above threshold at Week 0
Nipocalimab	41	38
Placebo	49	44

n/N (%) who had anti-TT IgG above threshold		
at Week 24	at Week 30	at all available time points
28/31 (90%)	30/31 (97%)	32/38 (84%)
38/39 (97%)	35/36 (97%)	42/44 (95%)

Figure 2. Percent change from baseline, concentration, and the proportions of participants who had pre-existing anti-TT IgG above the protective threshold over time in (A) Vivacity-MG3, (B) IRIS-RA, and (C) DAHLIAS. IgG, immunoglobulin G; IQR, interquartile range; TT, tetanus toxoid. [a]30 mg/kg loading dose at Week 0. [b]Protective threshold (≥0.16 IU/mL) that generally suggests a sufficient level of immunity to tetanus.[14]

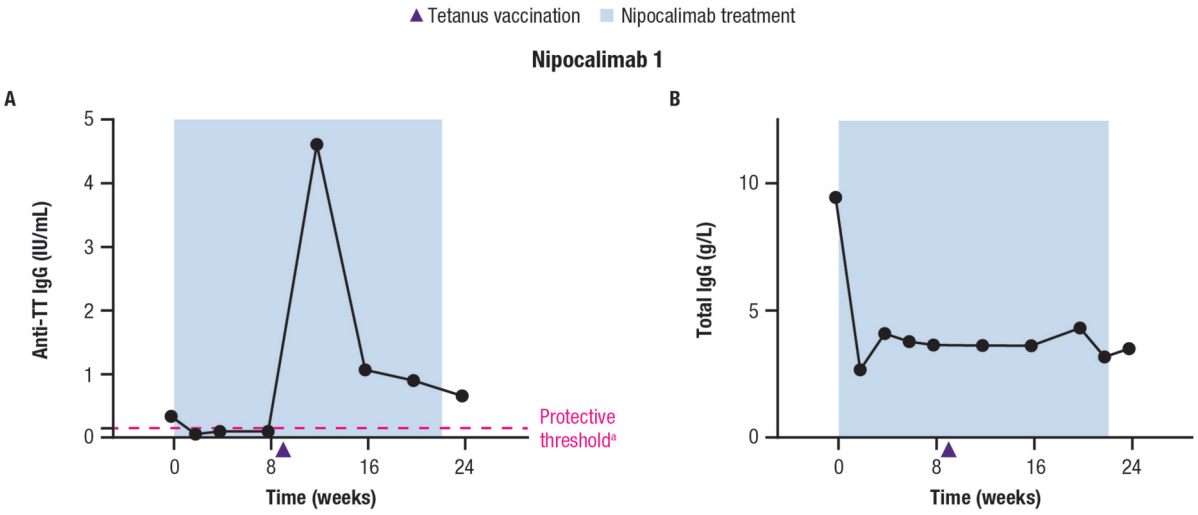


Figure 3. Anti-TT (A) specific IgG and (B) total IgG responses to the TT vaccination in individual participants treated with nipocalimab in the Vivacity-MG3 study. IgG, immunoglobulin G; TT, tetanus toxoid. [a]Protective threshold (≥0.16 IU/mL) that generally suggests a sufficient level of immunity to tetanus.[14]

Responses to SARS-CoV-2 infections and severity outcomes

A total of 18 participants (nipocalimab: n = 10; placebo: n = 8) from Vivacity-MG3, 4 (nipocalimab: n = 4; placebo: n = 0) from IRIS-RA, and 6 (nipocalimab: n = 3; placebo: n = 3) from DAHLIAS had SARS-CoV-2 infections during the studies and were included in the anti-SARS-CoV-2 analysis. Across studies, participants who developed SARS-CoV-2 infections during nipocalimab treatment mounted IgG responses against spike protein, S1 RBD, and nucleocapsid (Figure 6), similar to the responses observed in placebo-treated participants who had SARS-CoV-2 infections during the studies (Figure 7). Notably, 1 participant in Vivacity-MG3 experienced a SARS-CoV-2 infection and subsequently received SARS-CoV-2 vaccination (nipocalimab 12; Figure 6(A)).

While the reduction in total IgG levels was observed in all participants who developed SARS-CoV-2 infections during nipocalimab treatment (Figure S3), all SARS-CoV-2 infections were mild to moderate in severity. Among participants receiving nipocalimab, 12 had mild infections (7 in Vivacity-MG3, 3 in IRIS-RA, and 2 in the nipocalimab 15 mg/kg group of DAHLIAS) and 5 had moderate infections (3 in Vivacity-MG3, 1 in IRIS-RA, and 1 in the nipocalimab 15 mg/kg group of DAHLIAS); among participants receiving placebo, 8 had mild infections (7 in Vivacity-MG3 and 1 in DAHLIAS) and 3 had moderate infections (1 in Vivacity-MG3 and 2 in DAHLIAS). All infections were resolved without complications or need for hospitalization.

Discussion

These post hoc analyses of Vivacity-MG3, IRIS-RA, and DAHLIAS demonstrated that the levels of pre-existing anti-TT and anti-HZV IgG followed the kinetics of total IgG reduction during nipocalimab treatment and returned to baseline after treatment discontinuation. Despite the reduction in IgG levels with nipocalimab treatment, the levels of pre-existing anti-HZV were at or above the reference threshold of 100 IU/L in the majority of participants. This reference threshold was established based on population studies and has moderate correlation with infection risk, as cellular immunity and rapid anamnestic responses also contribute significantly to the protection against HZV infection.[15–17] Similarly, the levels of pre-existing anti-TT IgG levels remained above the protective threshold of 0.16 IU/mL[14] in the majority of participants. As tetanus immunity is primarily mediated by antibodies, the anti-TT threshold is well standardized and correlates strongly with protection.[14] In the small minority of patients whose anti-vaccine IgG levels fell below protective levels during nipocalimab treatment, the risk of infection with the respective pathogens might be increased and closer monitoring may be warranted. However, this risk may be mitigated by the reversibility of the

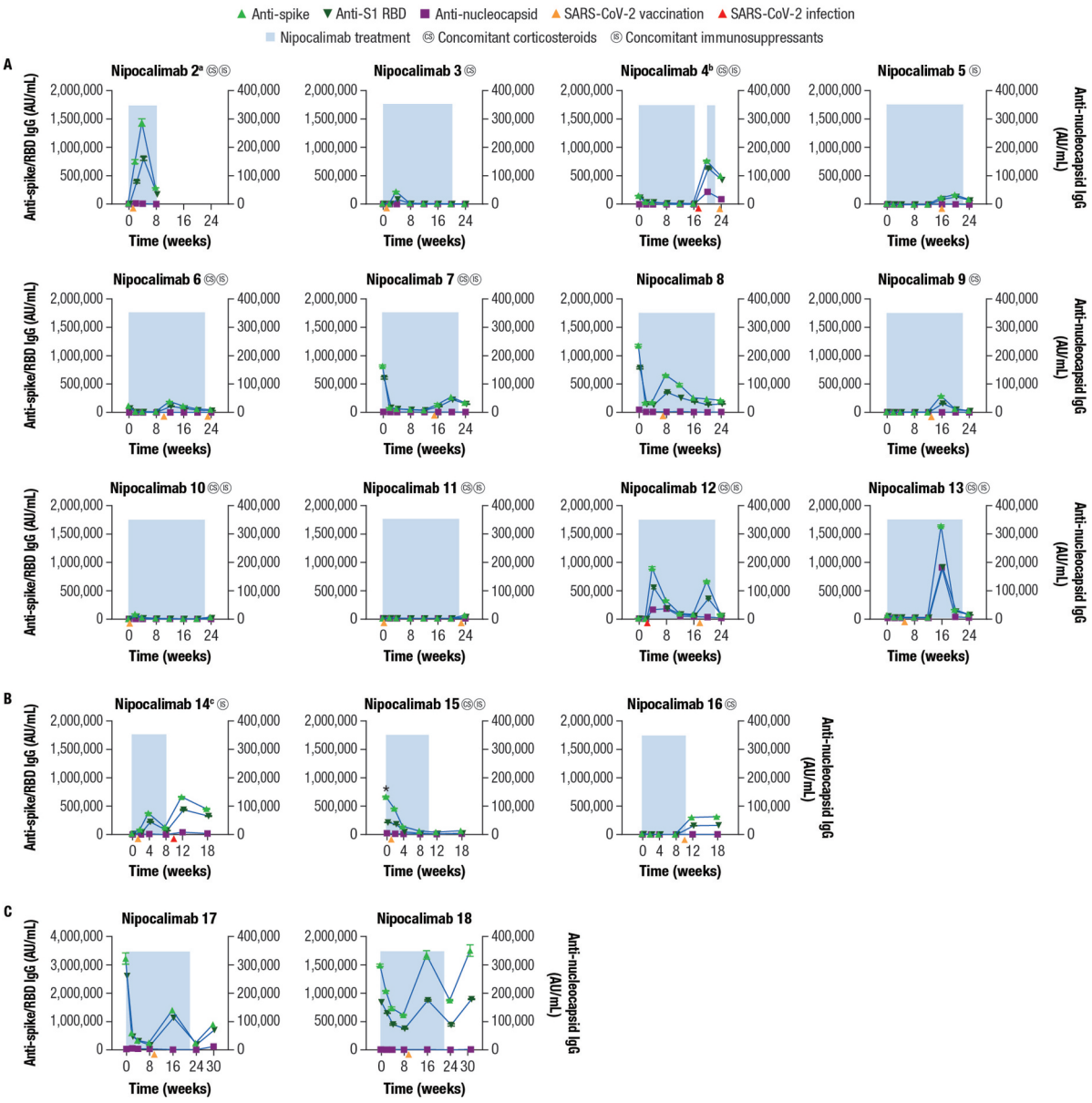


Figure 4. SARS-CoV-2 anti-spike, anti-S1 RBD, and anti-nucleocapsid IgG levels in individual participants vaccinated with SARS-CoV-2 vaccine and treated with nipocalimab in (A) Vivacity-MG3, (B) IRIS-RA, and (C) DAHLIAS. AU, arbitrary unit; COVID-19, coronavirus disease 2019; IgG, immunoglobulin G; S1 RBD, spike protein S1 receptor-binding domain; SARS-CoV-2, severe acute respiratory syndrome coronavirus 2. *High anti-spike IgG level was achieved in nipocalimab 15 after first dose of vaccination (17 d prior to study start). Therefore, the second vaccination may not further increase the anti-spike IgG level. [a]Participant received the last dose at Week 8. [b]Participant missed the dose at Week 18 due to infection. [c]Participant had both SARS-CoV-2 vaccination and infection during the treatment period and missed the dose at Week 10 due to infection.

anti-vaccine IgG reductions and the preserved humoral response to vaccination during nipocalimab treatment when vaccine boosters are needed.[13] Indeed, nipocalimab-treated participants, the majority of whom also received concomitant SOC therapy including corticosteroids and immunosuppressants, were able to elicit IgG responses to the SARS-CoV-2 vaccination/infection and TT vaccination during treatment, and anti-vaccine IgG titers returned to baseline within 6 to 8 weeks of the last nipocalimab dose. These results suggest that there is not a lasting impact of nipocalimab treatment on pre-existing

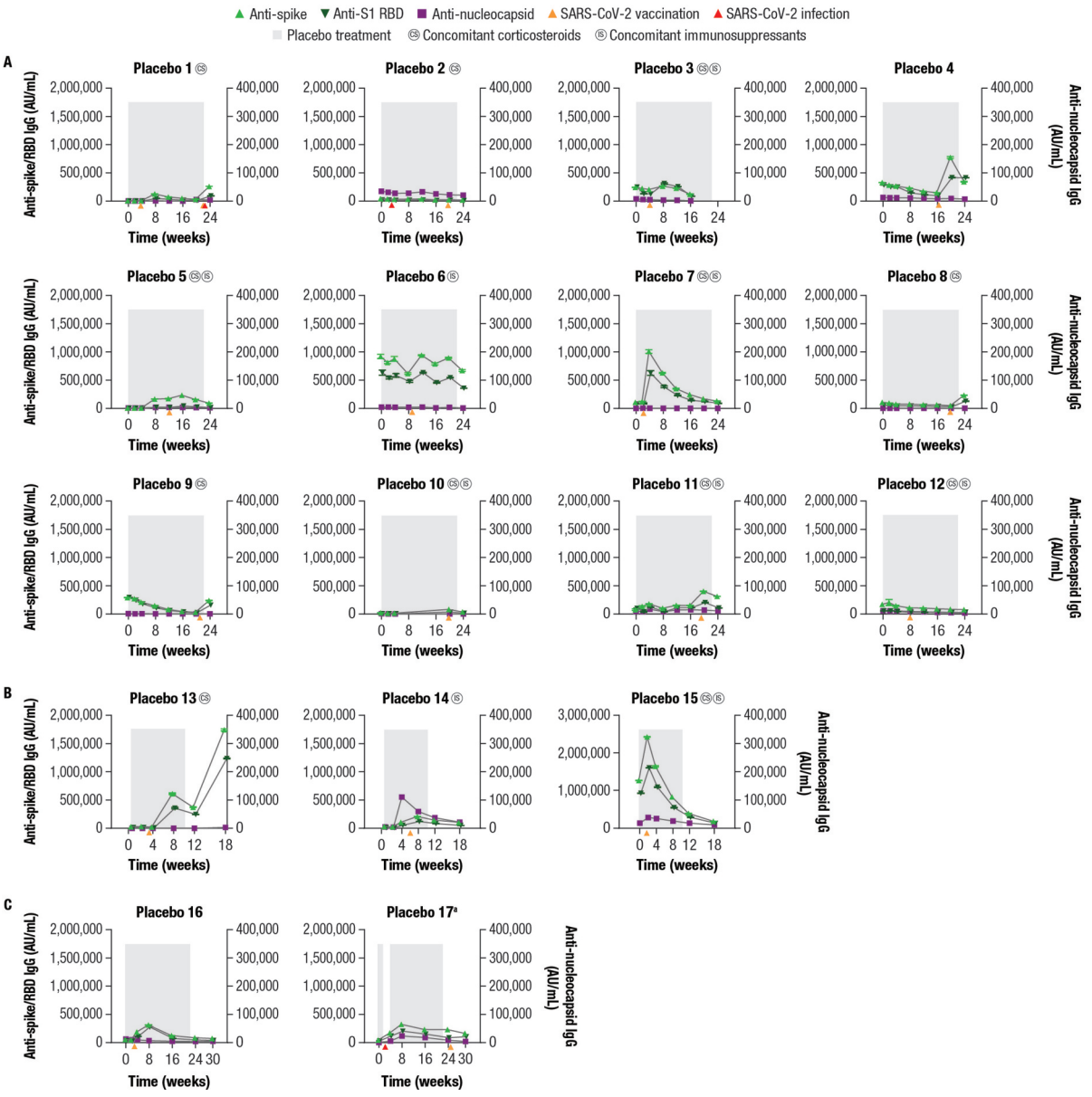


Figure 5. SARS-CoV-2 anti-spike, anti-S1 RBD, and anti-nucleocapsid IgG levels in individual participants vaccinated with SARS-CoV-2 vaccine and treated with placebo in (A) Vivacity-MG3, (B) IRIS-RA, and (C) DAHLIAS. AU, arbitrary unit; COVID-19, coronavirus disease 2019; IgG, immunoglobulin G; S1 RBD, spike protein S1 receptor-binding domain; SARS-CoV-2, severe acute respiratory syndrome coronavirus 2. [a]Participant had both SARS-CoV-2 vaccination and infection during the treatment period and missed the dose at Week 2.

protective antibody levels from vaccination, and that nipocalimab does not impair the development of humoral responses to vaccines or viral infections in patients with IgG autoantibody-mediated diseases, despite ongoing treatment with immunomodulator or immunosuppressant concomitant therapies. For patients whose anti-vaccine IgG levels fell below protective levels during treatment, a booster vaccination may be considered to enhance protective immunity. However, future prospective studies with larger sample sizes that incorporate functional antibody assays and capture infection outcomes will be important to more directly define any clinically meaningful effect of nipocalimab treatment on vaccination responses.

Table 2. SARS-CoV-2 anti-spike and anti-S1 RBD IgG responses stratified by concomitant SOC therapy and mycophenolate mofetil use in individual participants who received a SARS-CoV-2 vaccine.

Treatment group	Concomitant SOC use	N (%)	Range of individual peak anti-spike IgG responses post-vaccination (AU/mL)	Range of individual peak anti-S1 RBD IgG responses post-vaccination (AU/mL)
Nipocalimab (N = 17)	No corticosteroids or immunosuppressants *(reference)*	3 (17.6)	659,539–1,673,150	353,210–1,137,800
	Corticosteroids and/or immunosuppressants	14 (82.4)	9507–1,638,290	3792–901,584
	Mycophenolate mofetil	2 (11.7)	193,007–267,128	107,632–218,785
Placebo (N = 17)	No corticosteroids or immunosuppressants *(reference)*	3 (17.6)	162,188–769,896	102,559–411,227
	Corticosteroids and/or immunosuppressants	14 (82.4)	106,029–2,425,370	7976–1,603,740
	Mycophenolate mofetil	3 (17.6)	106,029–941,162	7976–631,653

AU, arbitrary unit; IgG, immunoglobulin G; S1 RBD, spike protein S1 receptor-binding domain; SARS-CoV-2, severe acute respiratory syndrome coronavirus 2; SOC, standard of care.

Observed IgG responses to vaccination are also consistent with previous observations of nipocalimab in preclinical data and a phase 1 vaccination study.[9,13] Nipocalimab decreased the levels of circulating neoantigen (KLH) IgG but did not affect the generation of neoantigen IgG responses in cynomolgus monkeys.[9] In a phase 1 study, healthy participants treated with nipocalimab had numerically lower IgG responses to TT and PPSV®23 at Week 4, while comparable responses were observed by Week 16 (ie, within 12 weeks of the last dose). Comparable antigen-specific responses were also observed at Week 2, when the IgG-lowering effect of nipocalimab was close to the nadir reached at Week 4, and showed a clear increase in both arms with respect to pre-vaccination titers, thereby demonstrating that nipocalimab did not prevent the triggering of antigen-specific responses. Throughout the study, anti-TT IgG levels remained above the protective threshold for all participants, and anti-pneumococcal capsular polysaccharide (PCP) IgG levels showed a 2-fold increase from baseline in both arms.[13] These findings are also consistent with previous observations of another FcRn blocker, efgartigimod, in patients with IgG autoantibody-mediated pemphigus and gMG,[20] supporting FcRn blockade as a highly immunoselective targeting strategy for the treatment of IgG autoantibody-mediated diseases while preserving humoral immune function.

In addition to having no detectable impact on IgG production, nipocalimab has shown no clinically significant changes to levels of immunoglobulin M (IgM), E (IgE), and A (IgA) or cellular responses in preclinical and phase 1 to 3 trials to date.[2,3,9–11] IgG, IgA, and IgM all contribute to anti-vaccine responses and humoral immunity against viral infections.[21,22] Current prescribing guidance recommends that clinicians evaluate the need to administer age-appropriate vaccines in accordance with applicable immunization guidelines before initiating nipocalimab.[7] The findings of these analyses, together with previous observations, suggest that nipocalimab treatment does not interfere with the development of effective immunity following vaccination and/or infection, and patients treated with nipocalimab should adhere to recommended vaccination schedules and may receive non-live vaccines, as needed, at any time during treatment.[7] These individuals can mount IgG responses to vaccines and benefit from vaccine-specific IgG, IgM, and cellular immunity despite the inhibition of IgG recycling by nipocalimab.

Limitations of these post hoc analyses include the small number of vaccinated participants, particularly in IRIS-RA and DAHLIAS cohorts, and the reliance on a limited number of serum samples across all 3 studies to assess anti-vaccine IgG responses. In addition, the absence of more comprehensive immunoassays, such as measures of cellular immunity, further restrict the interpretation of these findings.

In conclusion, despite the reduction of total IgG by nipocalimab, most participants treated with nipocalimab maintained pre-existing vaccine-induced IgG levels ≥100 IU/L for anti-HZV and above the

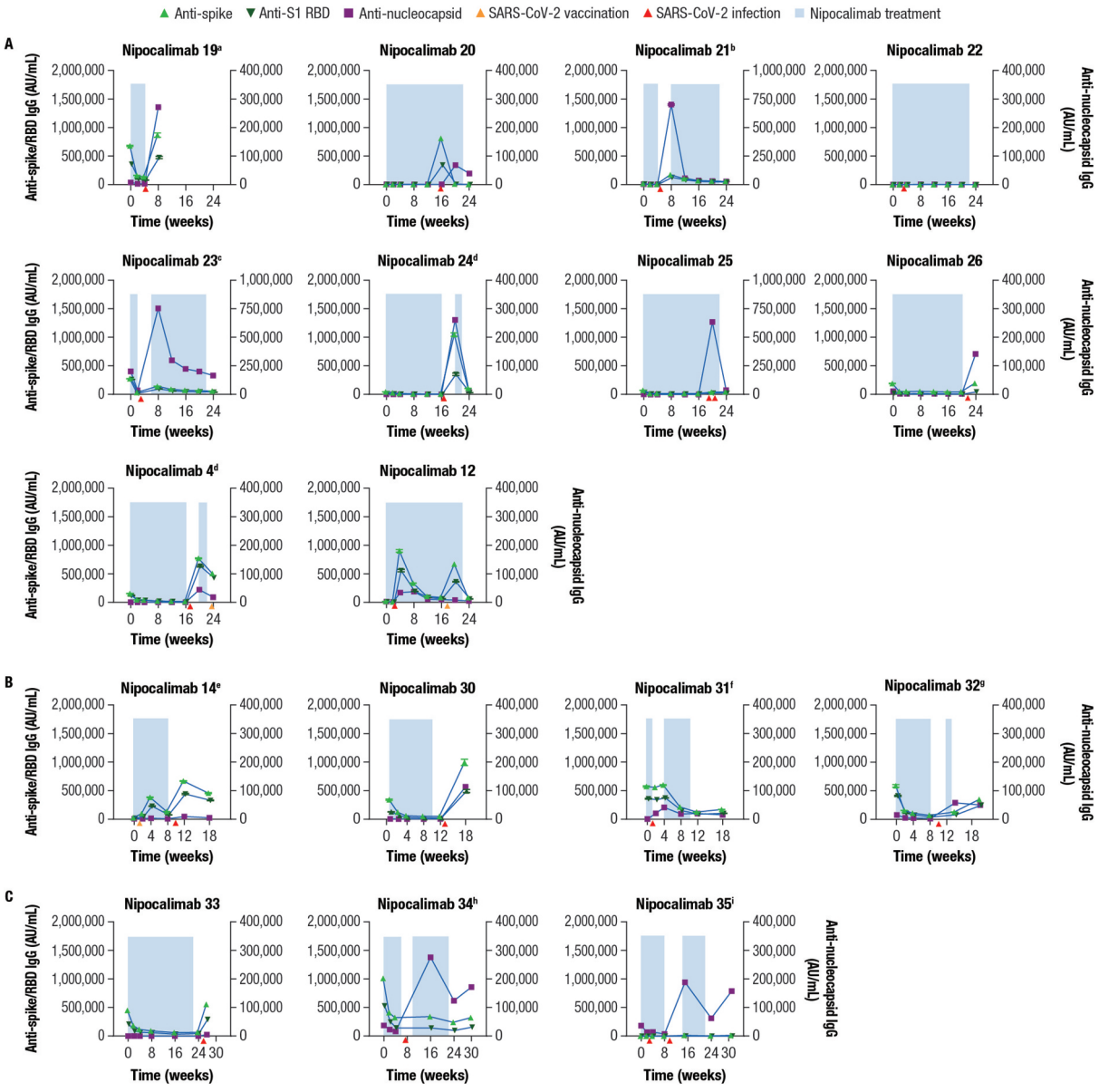


Figure 6. SARS-CoV-2 anti-spike, anti-S1 RBD, and anti-nucleocapsid IgG levels in individual participants who had SARS-CoV-2 infections and were treated with nipocalimab in (A) Vivacity-MG3, (B) IRIS-RA, and (C) DAHLIAS. AU, arbitrary unit; COVID-19, coronavirus disease 2019; IgG, immunoglobulin G; RA, rheumatoid arthritis; S1 RBD, spike protein S1 receptor-binding domain; SARS-CoV-2, severe acute respiratory syndrome coronavirus 2. [a]Participant received the last dose at Week 4. [b]Participant missed the dose at Week 6 due to infection. [c]Participant missed the dose at Week 4 due to infection. [d]Participant missed the dose at Week 18 due to infection. [e]Participant had both SARS-CoV-2 vaccination and infection during the treatment period and missed the dose at Week 10 due to infection. [f]Participant missed the dose at Week 2 due to infection. [g]Participant delayed the dose from Week 10 to Week 12 due to infection. [h]Participant missed the dose at Week 8. [i]Participant missed the doses at Weeks 10 and 12.

protective threshold of 0.16 IU/mL for anti-TT. The majority of participants treated with nipocalimab also mounted positive IgG responses to selected vaccines and viral infections (SARS-CoV-2 and TT), in line with previous findings in healthy participants which showed that nipocalimab does not impact the development of IgG responses to T-cell-dependent and -independent vaccines.[13] These findings were consistent across 3 trials in different autoantibody-mediated diseases on distinct background SOC. This consistent evidence supports that nipocalimab does not impair the humoral immune response to vaccines or viral infections.

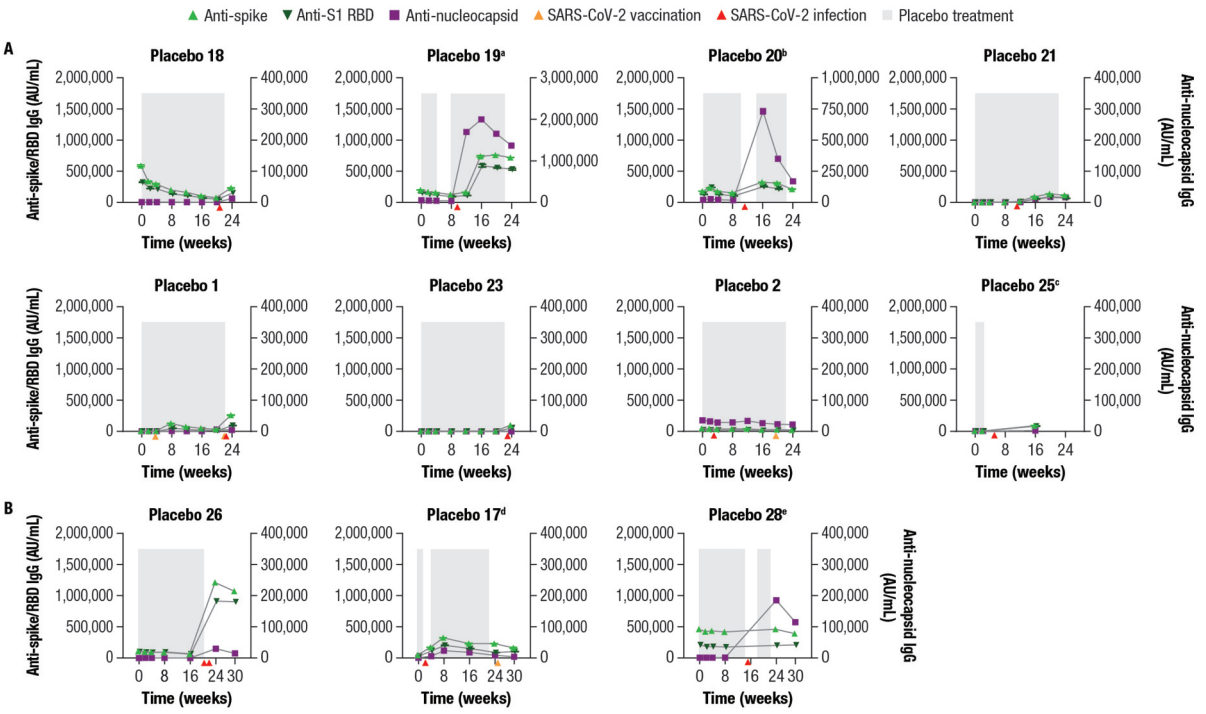


Figure 7. SARS-CoV-2 anti-spike, anti-S1 RBD, and anti-nucleocapsid IgG levels in individual participants who had SARS-CoV-2 infections and were treated with placebo in (A) Vivacity-MG3 and (B) DAHLIAS. AU, arbitrary unit; COVID-19, coronavirus disease 2019; IgG, immunoglobulin G; S1 RBD, spike protein S1 receptor-binding domain; SARS-CoV-2, severe acute respiratory syndrome coronavirus 2. [a]Participant missed the dose at Week 4. [b]Participant missed the dose at Week 12 due to infection. [c]Participant received the last dose at Week 2. [d]Participant had both SARS-CoV-2 vaccination and infection during the treatment period and missed the dose at Week 2. [e]Participant missed the dose at Week 16.

Therefore, these findings may suggest that patients receiving nipocalimab should adhere to recommended vaccination schedules of non-live vaccines. Additional ongoing studies will provide data to further ascertain the safety and risk of infections in patients treated with nipocalimab.

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Author contributions

CRediT: **Faye Yu:** Conceptualization, Data curation, Formal analysis, Investigation, Methodology, Project administration, Visualization, Writing – review & editing; **Eugene Myshkin:** Conceptualization, Data curation, Formal analysis, Investigation, Methodology, Visualization, Writing – review & editing; **Brandon Nguyen:** Investigation, Formal analysis, Writing – review & editing; **Carolina Bobadilla Mendez:** Conceptualization, Data curation, Investigation, Writing – review & editing; **Marta Cossu:** Conceptualization, Data curation, Investigation, Writing – review & editing; **Kaiyin Fei:** Data curation, Investigation, Writing – review & editing; **Qingmin Wang:** Data curation, Investigation, Writing – review & editing; **Jonathan J. Hubbard:** Data curation, Investigation, Writing – review & editing; **Kim Campbell:** Data curation, Investigation, Writing – review & editing; **Sindhu Ramchandren:** Data curation, Investigation, Writing – review & editing; **Ricardo Rojo Cella:** Data curation, Writing – review & editing; **Robert Edwards:** Data curation, Writing – review & editing; **Peter C. Taylor:** Investigation, Writing – review & editing; **Jacques-Eric Gottenberg:** Investigation, Writing – review &

editing; **Ghaith Noaiseh:** Investigation, Writing – review & editing; **Tuan Vu:** Investigation, Writing – review & editing; **Carlo Antozzi:** Investigation, Writing – review & editing; **Kevin L. Winthrop:** Investigation, Writing – review & editing; **Carolyn A. Cuff:** Conceptualization, Data curation, Resources, Supervision, Writing – review & editing; **Matthew J. Loza:** Data curation, Formal analysis, Investigation, Methodology, Visualization, Writing – review & editing; **Dessislava Dimitrova:** Conceptualization, Data curation, Supervision, Writing – review & editing; **Sheng Gao:** Conceptualization, Data curation, Formal analysis, Methodology, Project administration, Supervision, Visualization, Writing – review & editing.

Disclosure statement

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Data availability statement

The data sharing policy of Johnson & Johnson is available at https://www.jnj.com/innovativemedicine/our-innovation/clinical-trials/transparency. As noted on this site, requests for access to the study data can be submitted through Yale Open Data Access [YODA] Project site at http://yoda.yale.edu.

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