ACG/UEGW 2026: Data reflow deck

Job code: CH_ CP-609070 based on CP-607908

Date of preparation: October 2026

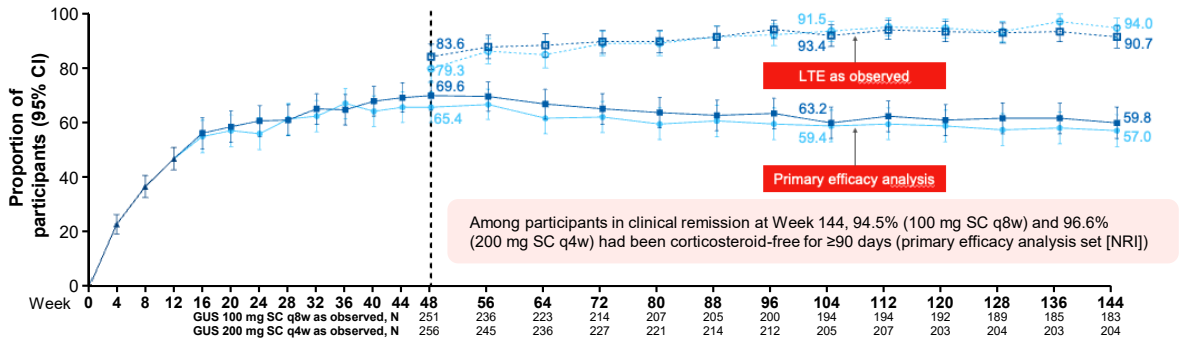
Janssen Cilag AG, a Johnson & Johnson company
Gubelstrasse 34, 6300 Zug, Switzerland

Immunology

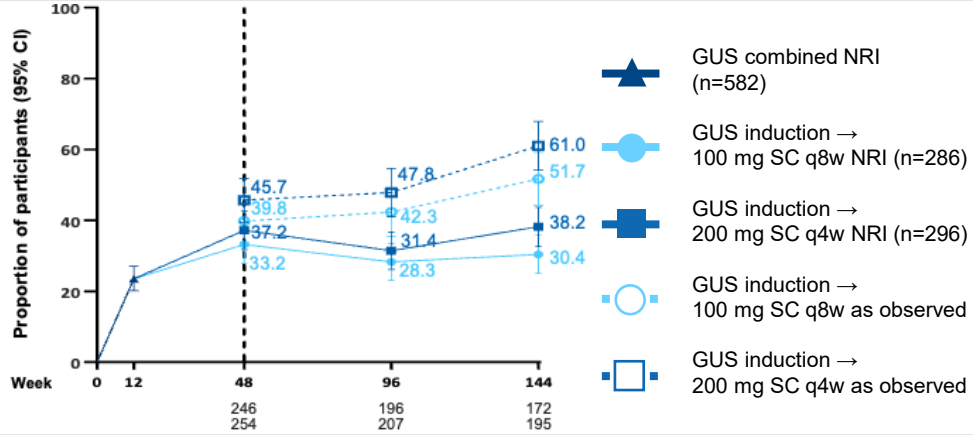
ACG/UEGW 2026 GUS highlights

GALAXI 2 and 3: 3-year efficacy and safety outcomes in CD[1]

Clinical remission through Week 144



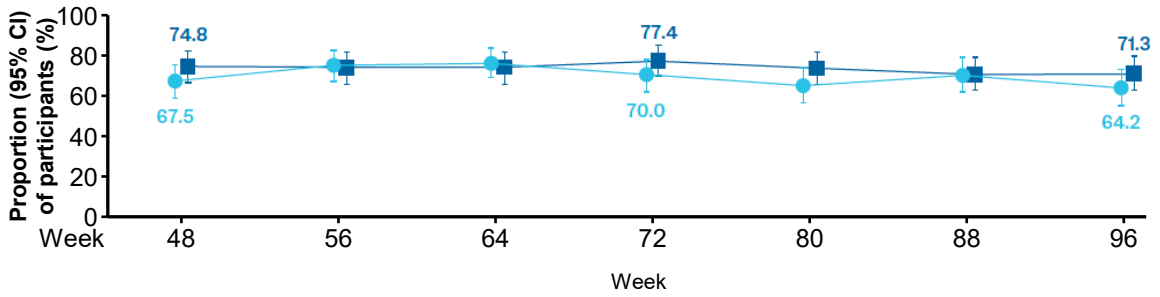
Endoscopic remission through Week 144



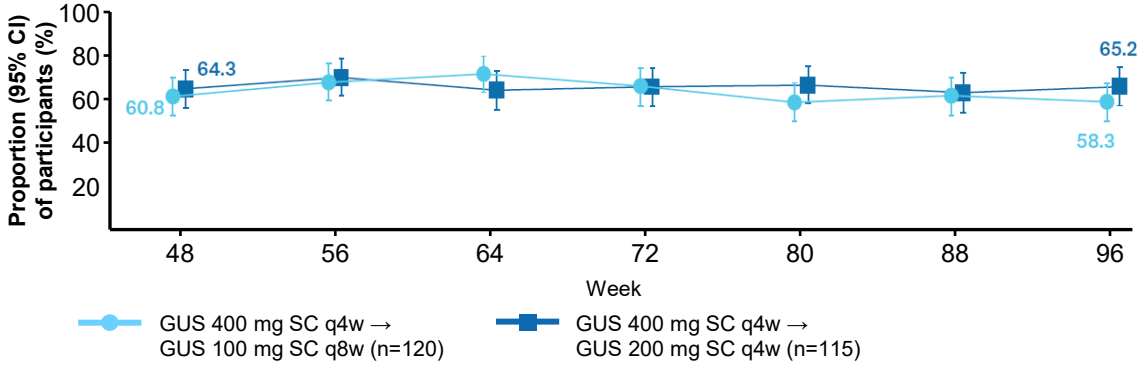
- Both GUS maintenance regimens demonstrated sustained clinical and endoscopic efficacy through Week 144 in participants with CD
- Sustained efficacy was observed in both bio-naïve participants and those with prior inadequate response or intolerance to biologic therapy
- No new safety signals were identified through 3 years; the safety profile remained consistent with the established GUS safety profile

ASTRO: 2-year efficacy and safety outcomes in UC[2]

Symptomatic remission from Week 48 through Week 96 (NRI)



Patients with no bowel urgency from Week 48 through Week 96 (NRI)



- Both GUS maintenance dose regimens demonstrated sustained symptomatic and inflammatory biomarker improvements through Week 96 in participants with UC who continued treatment after Week 48
- The absence of bowel urgency and abdominal pain was also maintained through Week 96
- No new safety concerns were identified; findings remained consistent with the established GUS safety profile

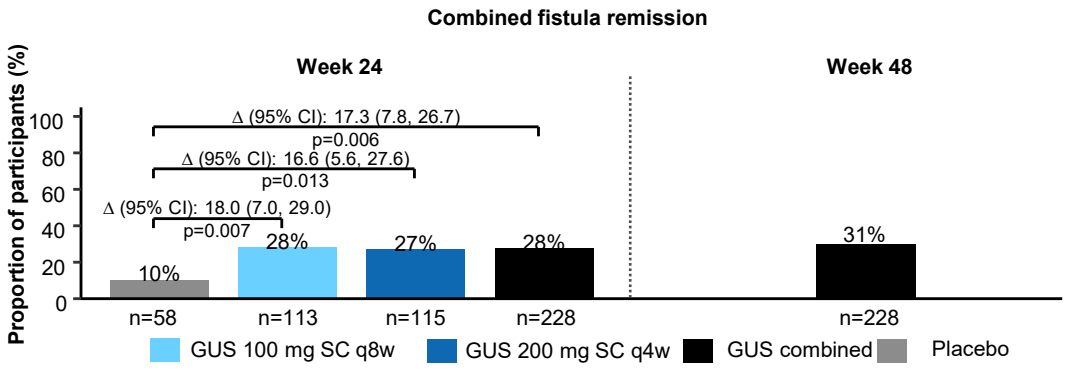
Clinical remission: CDAI score <150. **Deep remission:** Combination of clinical remission and endoscopic remission (SES-CD score ≤4 and ≥2-point reduction from baseline, with no individual component subscore >1).
Symptomatic remission: Stool frequency subscore of 0 or 1 and rectal bleeding subscore of 0. **No bowel urgency:** A rounded daily average (within 1 week) score of 0 in question Q7 of the UC-PRO/SS. Q7: In the past 24 hours, did you feel the need to have a bowel movement right away?
CD, Crohn's disease; CDAI, Crohn's Disease Activity Index; CI, confidence interval; GUS, guselkumab; NRI, nonresponder imputation; q4w, every 4 weeks; q8w, every 8 weeks; SC, subcutaneous; SES-CD, Simple Endoscopic Score for Crohn's Disease; UC, ulcerative colitis; UC-PRO/SS, Ulcerative Colitis Patient-Reported Outcomes Signs and Symptoms.
1. Sands BE, et al. Presented at ACG, Nashville, Tennessee, USA, 9–14 October 2026. P5693; 2. Long M, et al. Presented at ACG, Nashville, Tennessee, USA, 9–14 October 2026. P1177. . Full prescription information: swissmedicinfo-pro.ch.

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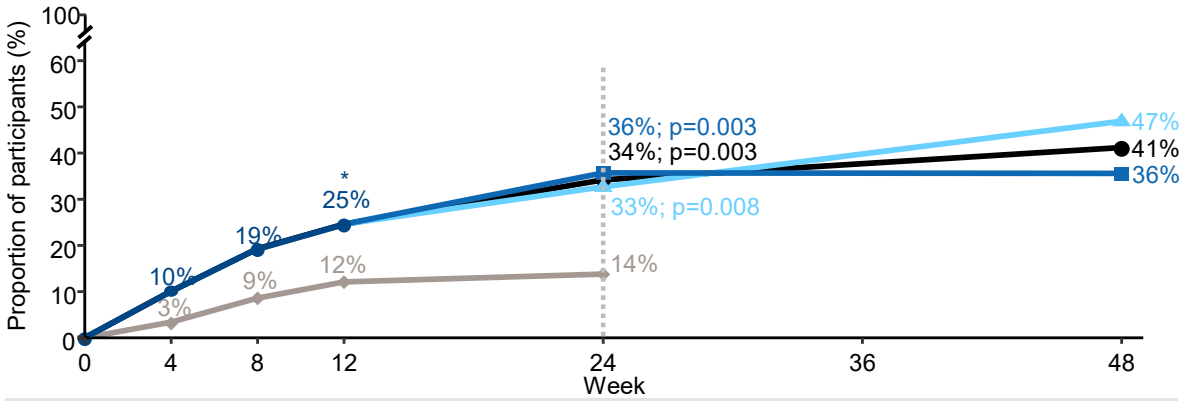
ACG/UEGW 2026 GUS highlights

FUZION:
1-year efficacy and safety outcomes in perianal fistulising CD[1]

Combined fistula remission through Week 48 (primary and secondary endpoints)



Clinically assessed fistula response through Week 48

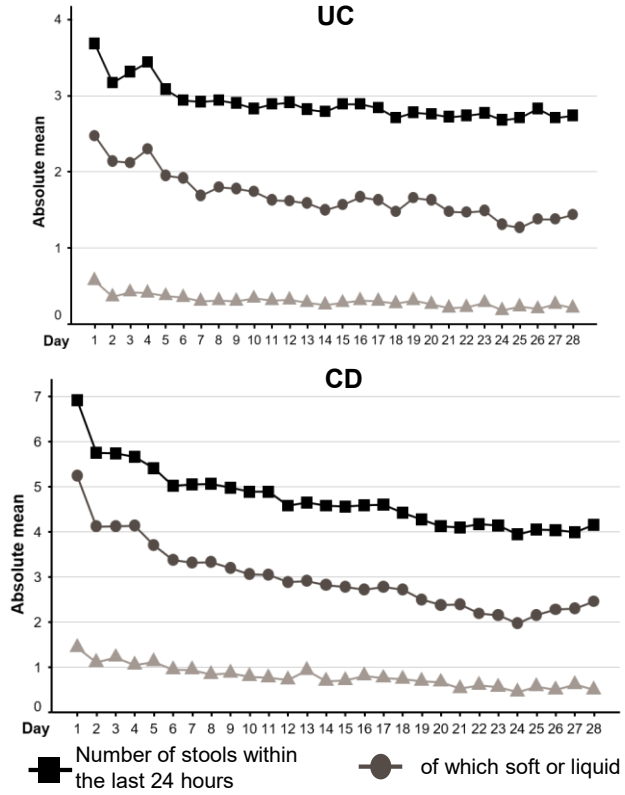


- FUZION is the first large international Phase 3 study dedicated to perianal fistulising CD in more than two decades that showed efficacy of an advanced therapy
- GUS showed superior efficacy to placebo at Week 24
- GUS demonstrated sustained efficacy through Week 48, supporting its continued use for durable fistula remission
- GUS was generally well tolerated through Week 48, with no new safety signals identified

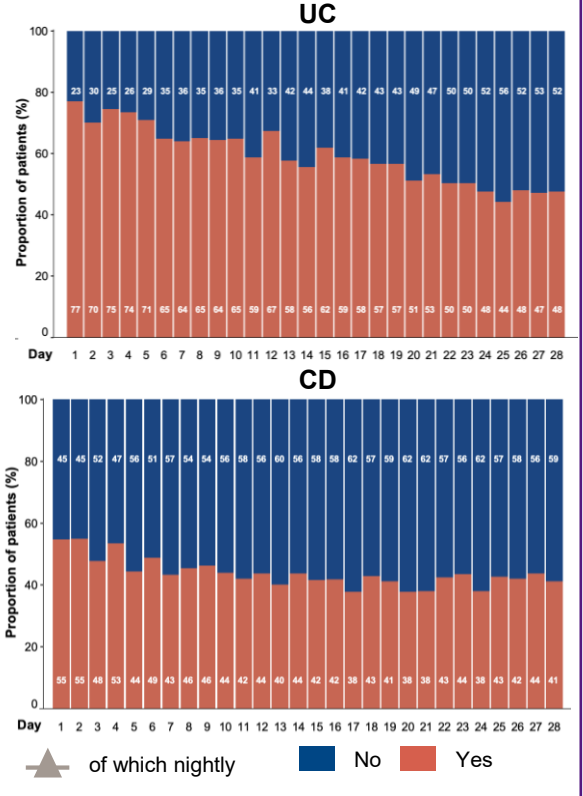
GORGEOUS:
Early symptomatic response in real-world IBD[2]

Symptom response within the first 4 weeks of GUS

Stool frequency



Bowel urgency



- The GORGEOUS study provides early real-world evidence of symptomatic improvement with GUS in patients with CD and UC treated in routine clinical practice in Germany
- Clinically meaningful improvements for most symptomatic endpoints were evident within the first week of treatment, with further improvement through Day 28
- Reductions in stool frequency and bowel urgency were observed in both CD and UC cohorts

Combined fistula remission: 100% closure of all treated external openings, without new fistulas or abscesses and without spontaneous or compression-induced drainage, and absence of perianal fistula collections >2 cm confirmed by blinded central MRI review. **Clinically assessed fistula response:** ≥50% reduction from baseline in the number of open or draining perianal fistulas. **Stool frequency:** Patient-reported number of stools in the preceding 24 hours, including soft/liquid and nightly stools. **Bowel urgency:** Patient-reported presence or absence of bowel urgency in the preceding 24 hours. *GUS 200 mg IV induction at Weeks 0/4/8; †GUS combined included both maintenance doses from Week 12 through Week 48. ‡Placebo non-responders received blinded GUS dose adjustment after Week 24; therefore, there was no placebo-controlled treatment arm beyond Week 24.

CD, Crohn's disease; GUS, guselkumab; IBD, inflammatory bowel disease; MRI, magnetic resonance imaging; q4w, every 4 weeks; q8w, every 8 weeks; SC, subcutaneous; UC, ulcerative colitis.

1. Hart A, et al. Presented at UEGW, Barcelona, Spain, 17–20 October 2026. LB09; 2. Schubert S, et al. Presented at UEGW, Barcelona, Spain, 17–20 October 2026. PP1357. . Full prescription information: swissmedicinfo-pro.ch.

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Reflow deck themes: CD clinical programme and RWE

01 LONG-TERM DISEASE CONTROL

GALAXI | 2 abstracts

- Maintenance efficacy & safety through Week 144
- Deep remission through 3 years

Durable remission and sustained disease control

02 PERIANAL FISTULISING DISEASE

FUZION | 2 abstracts

- Week 24 efficacy: Phase 3 FUZION
- Week 48 efficacy & safety: Phase 3 FUZION

Dedicated evidence in a high-unmet-need population

03 PREDICTORS OF RESPONSE

GALAXI | 3 abstracts

- Efficacy after prior anti-TNF inadequate response / intolerance
- Endoscopic outcomes by disease location
- Early biomarker improvement predicts 1-year outcomes

Signals to inform patient selection and prognosis

04 DISEASE MODIFICATION & STRUCTURAL OUTCOMES

GALAXI + GRAVITI | 4 abstracts

- Changes in intestinal stenosis with GUS
- Fibroblast biology in ileum and rectum (2)

Mechanistic, fibrostenotic and endoscopic evidence beyond symptoms

05 PK & EXPOSURE–RESPONSE

GALAXI + GRAVITI | 1 abstract

- SC maintenance pharmacokinetics and exposure–response

Evidence supporting the maintenance dosing strategy

06 RWE

IBD | 2 abstracts

- GORGEOUS study: GUS RWE from Germany (IBD)
- Early RWE with GUS (IBD)

CD, Crohn's disease; GUS, guselkumab; IBD, inflammatory bowel disease; PK, pharmacokinetics; RWE, real-world evidence; SC, subcutaneous; TNF, tumour necrosis factor.

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Reflow deck themes: UC QUASAR/ASTRO

01 LONG-TERM DISEASE CONTROL

QUASAR + ASTRO | 2 abstracts

- 4-year histology outcomes: QUASAR LTE
- 2-year efficacy & safety: ASTRO Week 96

Durable microscopic and clinical control

02 INDUCTION NON-RESPONDERS

QUASAR | 1 abstract

- Symptoms and biomarkers in patients not in clinical response at Week 12

Understanding benefit beyond early response

03 PREDICTORS OF RESPONSE

ASTRO | 1 abstract

- Machine learning identifies distinct patient-level response patterns

Towards more precise treatment insights

04 DISEASE MODIFICATION & PROGRESSION

QUASAR | 1 abstract

- Time to first indication of disease progression in Phase 3 maintenance

Evaluating impact beyond symptom control towards altering disease course

05 PK & EXPOSURE–RESPONSE

QUASAR + ASTRO | 1 abstract

- SC maintenance pharmacokinetics and exposure–response

Evidence supporting the maintenance dosing strategy

LTE, long-term evidence; PK, pharmacokinetics; SC, subcutaneous; UC, ulcerative colitis.

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Contents (1/3)

Guselkumab data			
Indication	**Study**	**Title**	**Authors**
pfCD	**FUZION**	Efficacy of guselkumab in perianal fistulizing Crohn's disease: Week 24 results from the Phase 3, randomized, double-blind, placebo-controlled, multicenter FUZION study	Sands, et al.
	FUZION	Efficacy and safety of guselkumab in perianal fistulizing Crohn's disease: Week 48 results from the Phase 3, randomized, double-blind, placebo-controlled FUZION trial	Hart, et al.
CD	**GALAXI**	Efficacy and safety of guselkumab maintenance therapy in patients with moderately to severely active Crohn's disease: Results through Week 144 from the Phase 3 GALAXI 2 & 3 studies	Sands, et al.
	GALAXI	Deep remission in guselkumab-treated participants with moderately to severely active Crohn's disease through 3 years: Results from the GALAXI 2 & 3 studies	Rubin, et al.
	GALAXI	Guselkumab efficacy in subgroups of adults with a history of inadequate response or intolerance to anti-tumor necrosis factor-α therapy and moderately to severely active Crohn's disease: Results from the GALAXI 2 & 3 studies	Reinisch, et al.
	GALAXI	Early improvement of inflammatory biomarkers predicts 1-year outcomes in adults with Crohn's disease treated with guselkumab: Pooled GALAXI 2 & GALAXI 3, Phase 3 trials analysis	Ferrante, et al.
	GALAXI	Endoscopic outcomes of ulcer size and ulcerated surface over one year across bowel segments in participants with Crohn's disease treated with guselkumab: Pooled GALAXI 2 and GALAXI 3, Phase 3 trials analysis	Armuzzi, et al.
	GALAXI/GRAVITI	Changes in intestinal stenosis among patients with moderately to severely active Crohn's disease treated with guselkumab: Results from the Phase 3 GALAXI and GRAVITI studies	Reinisch, et al.
	GALAXI/GRAVITI	Pharmacokinetics and exposure–response relationships of subcutaneous guselkumab maintenance in participants with moderately to severely active Crohn's disease	Sands, et al.

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Contents (2/3)

Guselkumab data			
Indication	**Study**	**Title**	**Authors**
UC	**QUASAR**	Histology over time in patients with moderately to severely active ulcerative colitis treated with guselkumab in the QUASAR long-term extension study	Rubin, et al.
	QUASAR	Symptomatic and biomarker outcomes among participants treated with guselkumab not in clinical response at Week 12: Results from the Phase 3 QUASAR induction study	Allegretti, et al.
	QUASAR	Time to first indication of disease progression in patients with moderately to severely active ulcerative colitis: Results from the guselkumab Phase 3 QUASAR maintenance study	Allegretti, et al.
	ASTRO	The efficacy and safety of guselkumab in patients with moderate to severe ulcerative colitis through 2 years: ASTRO Week 96 results	Long, et al.
	QUASAR/ASTRO	Pharmacokinetics and exposure–response relationships of subcutaneous guselkumab maintenance in participants with moderately to severely active ulcerative colitis	Rubin, et al.
IBD	**RWE: GORGEOUS**	Early symptomatic response to guselkumab in inflammatory bowel disease: First real-world data from the German GORGEOUS study	Schubert, et al.

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Contents (3/3)

Other therapies			
Indication	**Study**	**Title**	**Authors**
CD	**DUET: JNJ-4804 in CD**	Efficacy and safety of the first co-antibody therapy, JNJ-78934804, in patients with moderately to severely active Crohn's disease refractory to two or more systemic therapy mechanisms	Jairath, et al.
UC	**DUET: JNJ-4804 in UC**	Efficacy and safety of the first co-antibody therapy, JNJ-78934804, in patients with moderately to severely active ulcerative colitis refractory to two or more systemic therapy mechanisms	Atreya, et al.

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Guselkumab in Crohn’s disease

CH_CP-609070

FUZION: Guselkumab for perianal fistulising CD

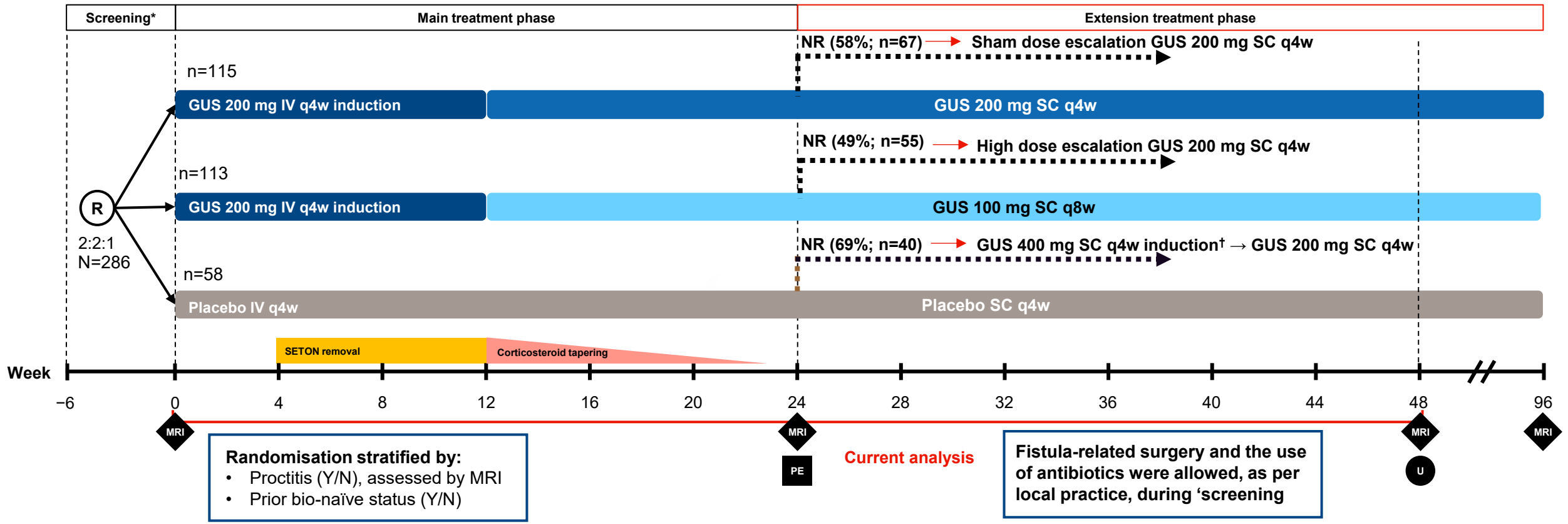
FUZION study design

Key eligibility criteria

- Adults with at least 1 active draining perianal fistula confirmed by blinded central MRI review
- CDAI score <350
- Inadequate response or intolerance to oral corticosteroids, immunomodulators (azathioprine, 6-mercaptopurine, methotrexate) or up to 2 advanced therapy classes (anti-TNF, vedolizumab, JAK inhibitors, excluding prior anti-IL-12/IL-23 inhibitors*)

Non-responders (NR; 57%):

- NR defined as <50% reduction from baseline in the number of open or draining fistulas at Week 24
- NRs in any group received one blinded dose escalation adjustment
- Responders continued with the same treatment and dose regimen through Week 48



Randomisation stratified by:

- Proctitis (Y/N), assessed by MRI
- Prior bio-naïve status (Y/N)

Current analysis

Fistula-related surgery and the use of antibiotics were allowed, as per local practice, during 'screening

*Participants who were exposed to ustekinumab at its approved labelled dosage AND met the required washout criterion (16 weeks) AND had not demonstrated inadequate response or intolerance to ustekinumab AND received one IV induction dose of ~6 mg/kg and one SC maintenance dose of 90 mg. †Included 3 doses q4w from Week 24 to Week 32.

CD, Crohn's disease; CDAI, Crohn's Disease Activity Index; GUS, guselkumab; IL, interleukin; IV, intravenous; JAK, Janus kinase; MOA, mechanism of action; MRI, magnetic resonance imaging; NR, non-responder; PE, primary endpoint; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous; TNF, tumour necrosis factor; U, study unblinding.

Hart A, et al. Presented at UEGW, Barcelona, Spain, 17–20 October 2026. LB09. This specific population is currently still under experimental investigation. Guselkumab is approved for adult moderate-severe CD patients. Full prescription information: swissmedicinfo-pro.ch.

CH_CP-609070 10/26

Efficacy of guselkumab in perianal fistulizing Crohn's disease: Week 24 results from the Phase 3, randomized, double-blind, placebo-controlled, multicenter FUZION study

P5691

Poster presented at ACG

Bruce E Sands,[1] Ailsa Hart,[2] Vipul Jairath,[3] Tadakazu Hisamatsu,[4] Axel Dignass,[5] Antonino Spinelli,[6,7] Susan J. Connor,[8] André D'Hoore,[9] Walter Reinisch,[10] Silvio Danese,[11] David A. Schwartz,[12] Julian Panés,[13] Bram Verstockt,[14] Anton Stift,[15] Maciej Nazar,[16] Jacqueline van Denderen,[17] Takehiko Sakamoto,[19] Koji Masuda,[19] Stephen Xu,[20] Ivana Bravatà,[18] Geert D'Haens,[21] and Laurent Peyrin-Biroulet,[22] on behalf of the FUZION Study Group

[1]Dr. Henry D Janowitz Division of Gastroenterology, Icahn School of Medicine at Mount Sinai, New York, NY, USA; [2]London North-West University Healthcare NHS Trust, London, United Kingdom; [3]Department of Medicine, Schulich School of Medicine & Dentistry I, Western University, London, ON, Canada; [4]Kyorin University School of Medicine, Tokyo, Japan; [5]Department of Medicine, Agaplesion Markus Hospital, Goethe University, Frankfurt, Germany; [6]Department of Biomedical Sciences, Humanitas University, Pieve Emanuele - Milan, Italy; [7]IRCCS Humanitas, Milan, Italy; [8]Department of Gastroenterology, Liverpool Hospital, Liverpool, New South Wales and South West Sydney Clinical Campuses, UNSW Medicine & Health, UNSW Sydney, Sydney, Australia; [9]Department of Abdominal Surgery, University Hospitals Leuven, KU Leuven, Leuven, Belgium; [10]Clinical Department for Gastroenterology and Hepatology, Internal Medicine Clinic III, Medical University of Vienna, Vienna, Austria; [11]IRCCS Ospedale San Raffaele and University Vita-Salute San Raffaele, Milano, Italy; [12]Inflammatory Bowel Disease Center, Vanderbilt University Medical Center, Nashville, TN, United States; [13]Hospital Clínic de Barcelona, IDIBAPS, CIBERehd, Barcelona, Spain; [14]Department of Gastroenterology and Hepatology, University Hospitals Leuven and Department of Chronic Diseases and Metabolism, KU Leuven, Leuven, Belgium; [15]Department of General Surgery, Division of Visceral Surgery, Medical University Vienna, Vienna, Austria; [16]Johnson & Johnson, Warsaw, Poland; [17]Johnson & Johnson, Breda, the Netherlands; [18]Johnson & Johnson, Milan, Italy; [19]Johnson & Johnson, Tokyo, Japan; [20]Johnson & Johnson, Spring House, PA, United States; [21]Department of Gastroenterology, Amsterdam University Medical Centers, Amsterdam, The Netherlands; [22]Northwestern Medicine Digestive Health Institute, Chicago, IL, United States

CH_CP-609070 10//26

Background



- PFCD is a severe and complex complication of CD[1,2]
- PFCD affects approximately one-quarter of patients with CD, and its presence at diagnosis may indicate a more severe clinical course[3]



- GUS is a dual-acting monoclonal antibody targeting IL-23p19, that potentially neutralises IL-23 and binds to CD64, a receptor on cells that produce IL-23[4]
- GUS is approved for adults with moderately to severely active CD[4]



FUZION (NCT05347095)

- Phase 3, randomised, double-blind, placebo-controlled, multicentre study in adults with active PFCD[5]
- GUS demonstrated significantly higher combined fistula remission rates versus placebo at Week 24, with no new safety signals identified[5]

CD, Crohn's disease; CD64, cluster of differentiation 64; GUS, guselkumab; IL, interleukin; MOA, mechanism of action; PFCD, perianal fistulising Crohn's disease; RDBPC, randomised, double-blind, placebo-controlled.

1. Lee JJ, et al. *Clin Exp Gastroenterol*. 2025;18:291–303; 2. Anand E, et al. *Tech Coloproctol*. 2025;29:102; 3. Lichtenstein GR, et al. *Am J Gastroenterol*. 2025;120:1225–1264; 4. Sachen KL, et al. *Front Immunol*. 2025;16:1532852; 5. Peyrin-Biroulet L, et al. Presented at DDW, Chicago, IL, USA, 2–5 May 2026. O1058b. Full prescription information: swissmedicinfo-pro.ch.

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Endpoints and outcomes

Endpoints

- Primary:
  - Combined fistula remission at Week 24 (clinically and radiologically assessed)
- Multiplicity-controlled secondary endpoints:
  - Clinically assessed fistula remission at Week 24
  - Clinically assessed fistula response at Week 24
- Key secondary endpoints:
  - Clinically assessed fistula response at Week 12
  - Change in MAGNIFI-CD score from baseline to Week 24

Statistical considerations

- GUS 200 mg q4w dose then the 100 mg q8w dose were sequentially tested versus placebo for the primary endpoint then for the multiplicity-controlled secondary endpoints
- p-values are based on CMH test for binary endpoints and on MMRM for continuous endpoints
- After applying the ICE strategy, missing data were imputed as not having achieved binary endpoints or not having improvement from baseline for continuous endpoints

Outcomes

Combined fistula remission	100% closure of all external openings, without development of new fistulas or abscesses and without any drainage by the external openings, occurring spontaneously or after gentle finger compression AND absence of collections >2 cm of the perianal fistulas in at least two of three dimensions, confirmed by a blinded central review of the MRI results
Clinically assessed fistula response	≥50% reduction from baseline in number of open or draining perianal fistulas
Clinically assessed fistula remission	100% closure of all external openings, without development of new fistulas or abscesses and without any drainage by the external openings, occurring spontaneously or after gentle finger compression
MAGNIFI-CD score	Score ranges from 0 (no disease activity) to 25 (severe disease activity)

CD, Crohn's disease; CMH, Cochran–Mantel–Haenszel; GUS, guselkumab; ICE, intercurrent event; IL, interleukin; MMRM, mixed model for repeated measures; MOA, mechanism of action; MRI, magnetic resonance imaging; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled.

Sands BE, et al. Presented at ACG, Nashville, Tennessee, USA, 9–14 October 2026. P5691. This specific population is currently still under experimental investigation. Guselkumab is approved for adult moderate-severe CD patients. Full prescription information: swissmedicinfo-pro.ch.

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Baseline characteristics were generally balanced across treatment arms

Baseline characteristics	GUS 100 mg q8w (n=113)	GUS 200 mg q4w (n=115)	Placebo (n=58)
Demographics			
Age, years, mean (SD)	36.2 (12.7)	36.0 (13.0)	38.0 (12.9)
Male, n (%)	79 (70)	85 (74)	41 (71)
Disease characteristics			
CD duration, years, median (IQR)	5.6 (1.9; 15.6)	6.8 (2.1; 15.9)	7.8 (3.4; 17.6)
CDAI score, mean (SD)	154.6 (97.6)	143.2 (84.6)*	147.6 (104.3)†
Participants with open or draining fistula, n (%)			
1 fistula	73 (65)	60 (52)	33 (57)
≥2 fistulas	40 (35)	55 (48)	25 (43)
Participants with complex fistula‡, n (%)	52 (46)	64 (56)	30 (52)
Previous biologic exposure, n (%)			
Bio-naive	43 (38)	38 (33)	16 (28)
Bio-exposed	70 (62)	77 (67)	42 (72)
Proctitis, n (%)	31 (27)	37 (32)	16 (28)
Seton present, n (%)	23 (20)	21 (18)	10 (17)
≥1 fistula-related surgery during screening, n (%)	17 (15)	18 (16)	7 (12)
Participants with collections >2 cm, n (%)	0	4 (4)	2 (3)
MAGNIFI-CD score (0–25), mean (SD)	14.54 (4.8)	15.47 (4.6)	15.05 (5.4)

*n=108; †n=56; ‡Fistula complexity was determined by MRI using MAGNIFI-CD "number of fistula tracts" (single/unbranched or complex).
CD, Crohn's disease; CDAI, Crohn's Disease Activity Index; GUS, guselkumab; IL, interleukin; IQR, interquartile range; MOA, mechanism of action; MRI, magnetic resonance imaging; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous; SD, standard deviation.
Sands BE, et al. Presented at ACG, Nashville, Tennessee, USA, 9–14 October 2026. P5691. This specific population is currently still under experimental investigation. Guselkumab is approved for adult moderate-severe CD patients. Full prescription information: swissmedicinfo-pro.ch.

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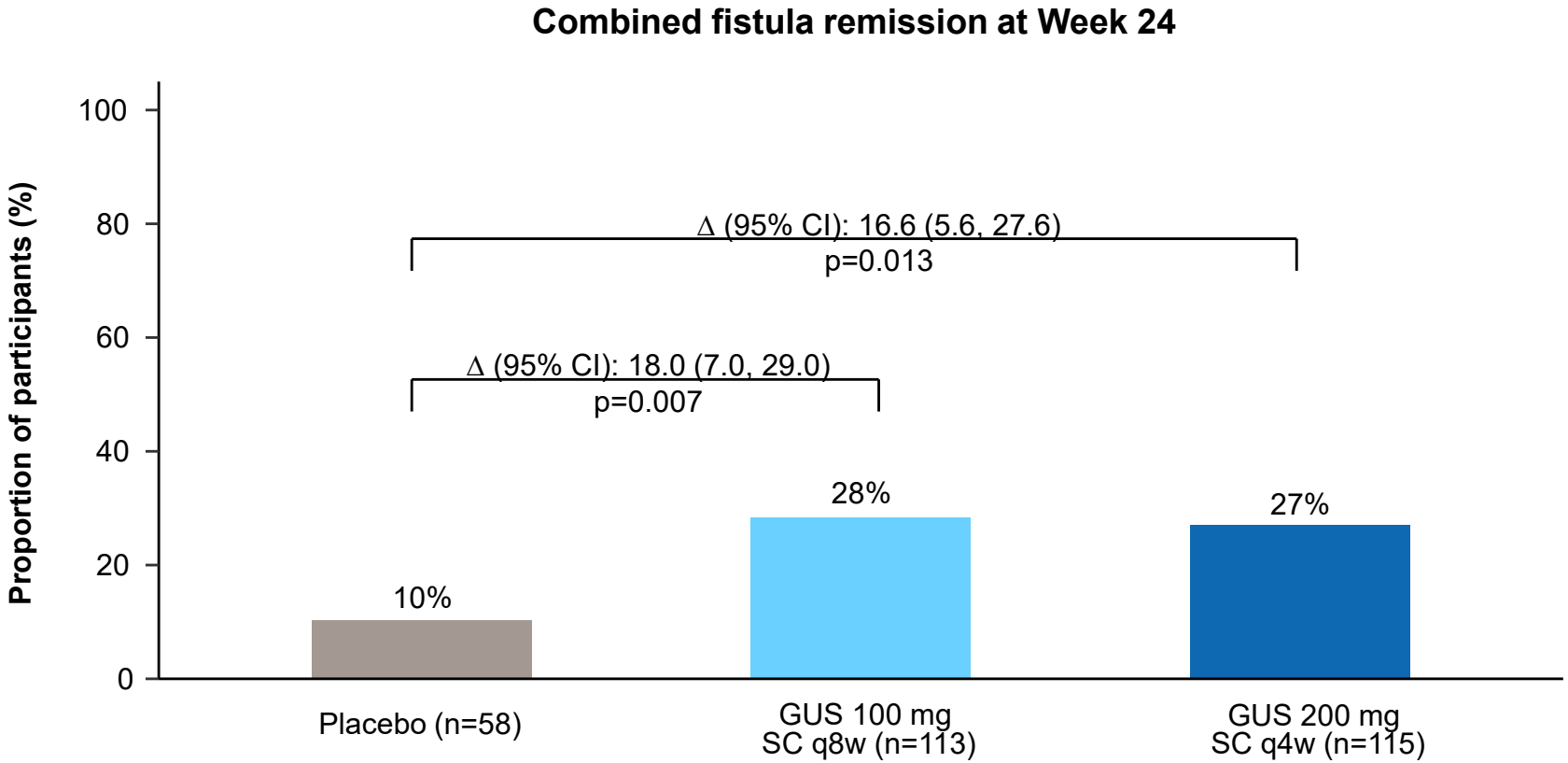
Baseline characteristics	GUS 100 mg q8w (n=113)	GUS 200 mg q4w (n=115)	Placebo (n=58)
Inadequate response or intolerance to Crohn's disease medication, n (%)			
Immunomodulator biologics	70 (62)	77 (67)	42 (72)
Only 1 anti-TNF agent	43 (38)	51 (44)	27 (47)
>1 anti-TNF agent	24 (21)	25 (22)	14 (24)
Only one MOA	62 (55)	69 (60)	35 (60)
Concomitant CD medications, n (%)			
5-ASA	27 (24)	27 (24)	19 (33)
Corticosteroids	10 (9)	9 (8)	3 (5)
Immunosuppressants (AZA, 6-MP, MTX)	34 (30)	35 (30)	16 (28)

5-ASA, 5-aminosalicylic acid; 6-MP, 6-mercaptopurine; AZA, azathioprine; CD, Crohn's disease; GUS, guselkumab; IL, interleukin; MOA, mechanism of action; MTX, methotrexate; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; TNF, tumour necrosis factor.

Sands BE, et al. Presented at ACG, Nashville, Tennessee, USA, 9–14 October 2026. P5691. This specific population is currently still under experimental investigation. Guselkumab is approved for adult moderate-severe CD patients. Full prescription information: swissmedicinfo-pro.ch.

CH_CP-609070 10//26

GUS demonstrated higher combined fistula remission rates at Week 24 versus placebo



Combined fistula remission: 100% closure of all external openings, without development of new fistulas or abscesses and without any drainage by the external openings, occurring spontaneously or after gentle finger compression AND absence of collections >2 cm of the perianal fistulas in at least two of three dimensions, confirmed by a blinded central review of the MRI results.

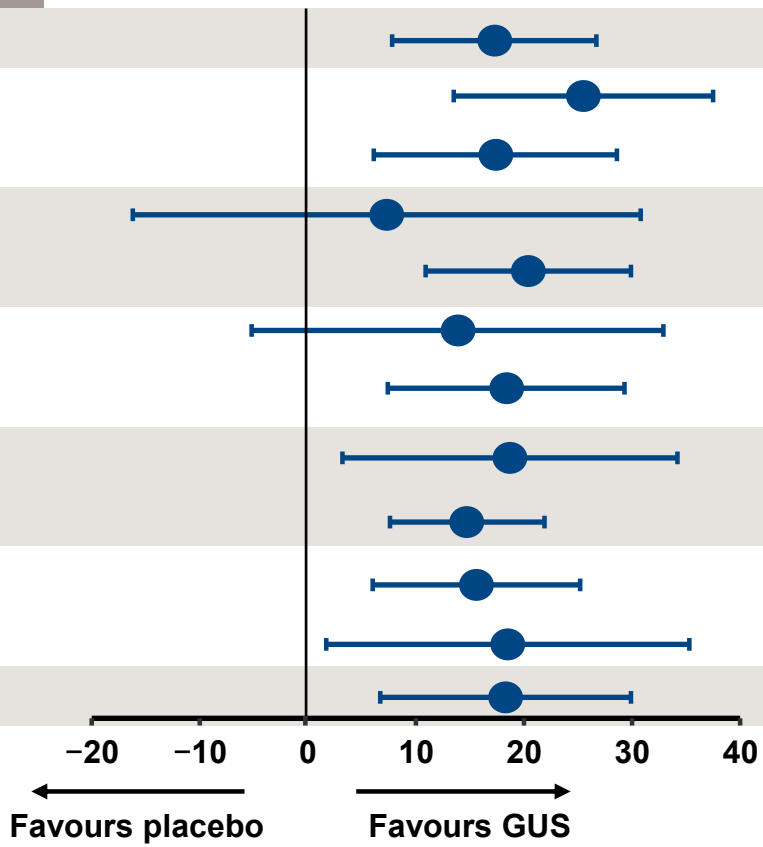
CD, Crohn's disease; CI, confidence interval; GUS, guselkumab; IL, interleukin; MOA, mechanism of action; MRI, magnetic resonance imaging; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; Δ, adjusted risk difference.

Sands BE, et al. Presented at ACG, Nashville, Tennessee, USA, 9–14 October 2026. P5691. This specific population is currently still under experimental investigation. Guselkumab is approved for adult moderate-severe CD patients. Full prescription information: swissmedicinfo-pro.ch.

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GUS achieved higher Week 24 combined fistula remission rates across all subgroups versus placebo

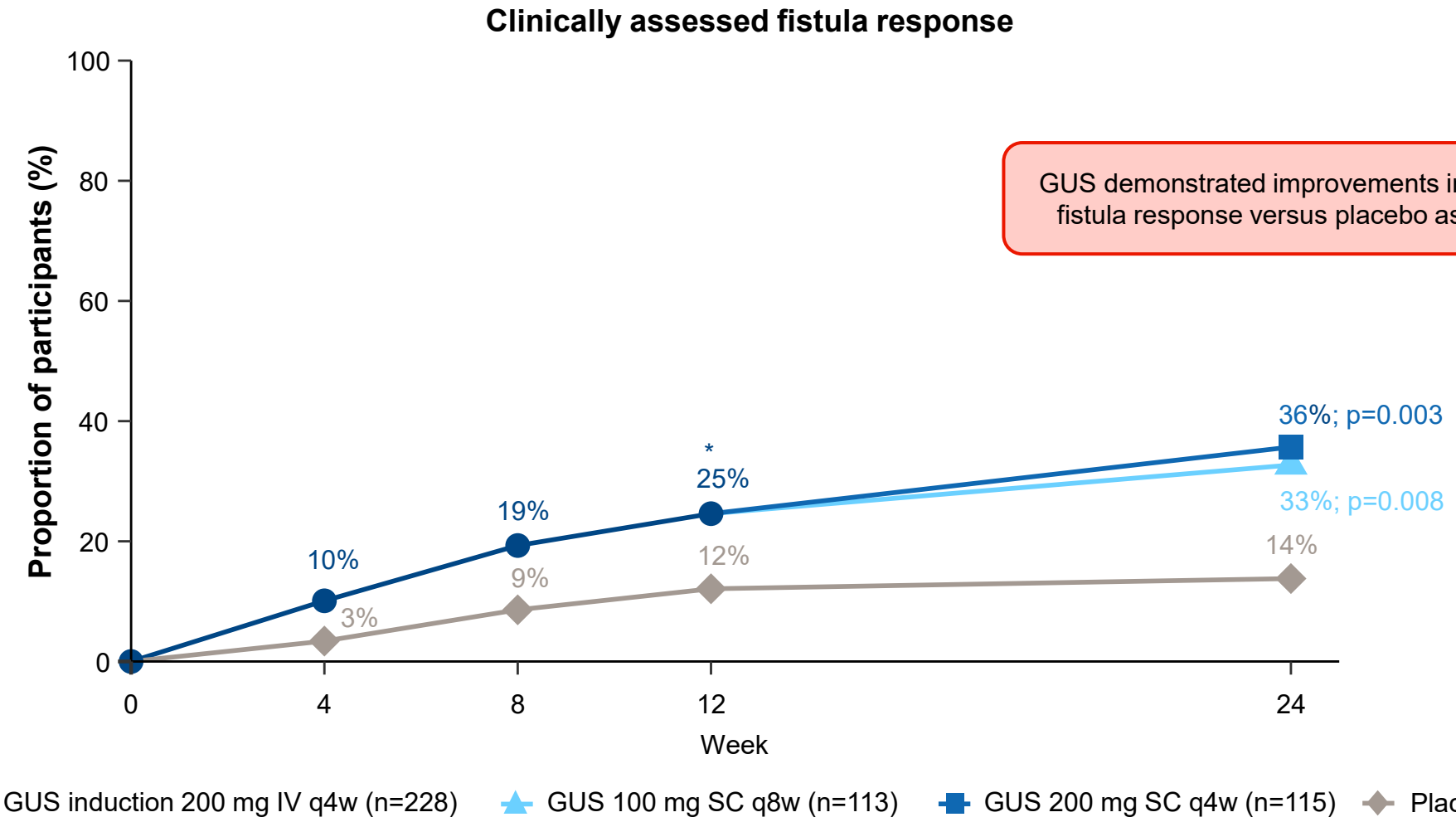
Category	Subgroups	GUS combined* n/N (%)	Placebo n/N (%)
Overall	All participants	63/228 (28)	6/58 (10)
Baseline CDAI	≥220 to ≤450	13/51 (26)	0/9 (0)
	<220	48/170 (28)	5/46 (11)
Biologic exposure	Without prior exposure	26/81 (32)	4/16 (25)
	With prior exposure	37/147 (25)	2/42 (5)
Proctitis at baseline	With proctitis	18/68 (26)	2/16 (12)
	Without proctitis	45/160 (28)	4/42 (10)
Number of fistulas	1 fistula	49/133 (37)	6/33 (18)
	≥2 fistulas	14/95 (15)	0/25 (0)
Fistula complexity	Complex fistula	22/116 (19)	1/30 (3)
	Simple fistula	40/110 (36)	5/28 (18)
Infliximab failure	With prior failure	27/110 (24)	2/32 (6)



Combined fistula remission: 100% closure of all external openings, without development of new fistulas or abscesses and without any drainage by the external openings, occurring spontaneously or after gentle finger compression AND absence of collections >2 cm of the perianal fistulas in at least two of three dimensions, confirmed by a blinded central review of the MRI results.
*GUS combined includes maintenance combined dose of GUS 100 mg SC q8w and 200 mg SC q4w.
CD, Crohn's disease; CDAI, Crohn's Disease Activity Index; CI, confidence interval; GUS, guselkumab; IL, interleukin; MOA, mechanism of action; MRI, magnetic resonance imaging; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous.
Sands BE, et al. Presented at ACG, Nashville, Tennessee, USA, 9–14 October 2026. P5691. This specific population is currently still under experimental investigation. Guselkumab is approved for adult moderate-severe CD patients. Full prescription information: swissmedicinfo-pro.ch.

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GUS demonstrated significantly greater clinically assessed fistula response rates versus placebo at Week 24

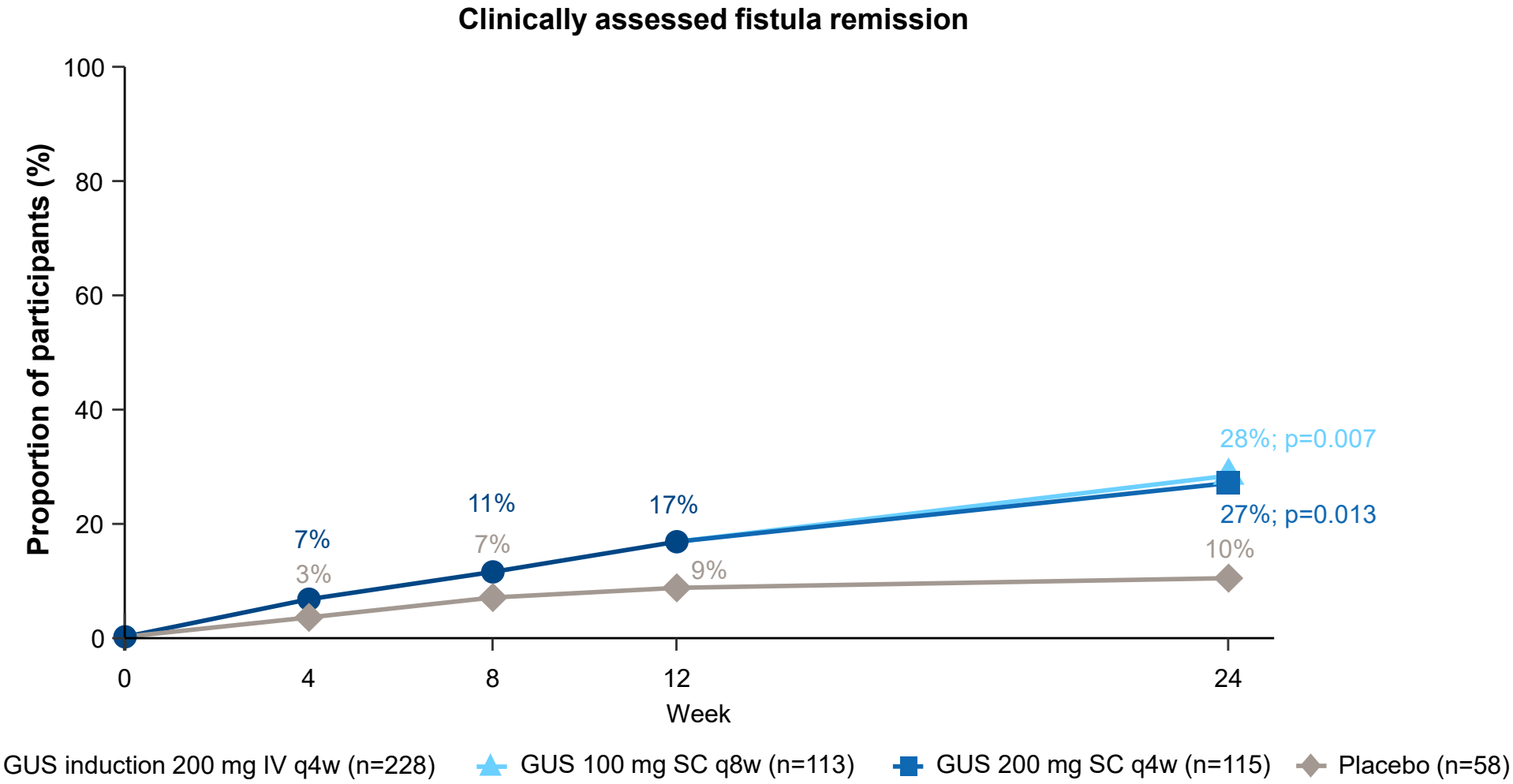


GUS demonstrated improvements in clinically assessed fistula response versus placebo as early as Week 12

Clinically assessed fistula response: ≥50% reduction from baseline in number of open or draining perianal fistulas.
*Nominal p<0.05. Printed p-values are multiplicity-controlled.
CD, Crohn's disease; GUS, guselkumab; IL, interleukin; IV, intravenous; MOA, mechanism of action; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous.
Sands BE, et al. Presented at ACG, Nashville, Tennessee, USA, 9–14 October 2026. P5691. This specific population is currently still under experimental investigation. Guselkumab is approved for adult moderate-severe CD patients. Full prescription information: swissmedicinfo-pro.ch.

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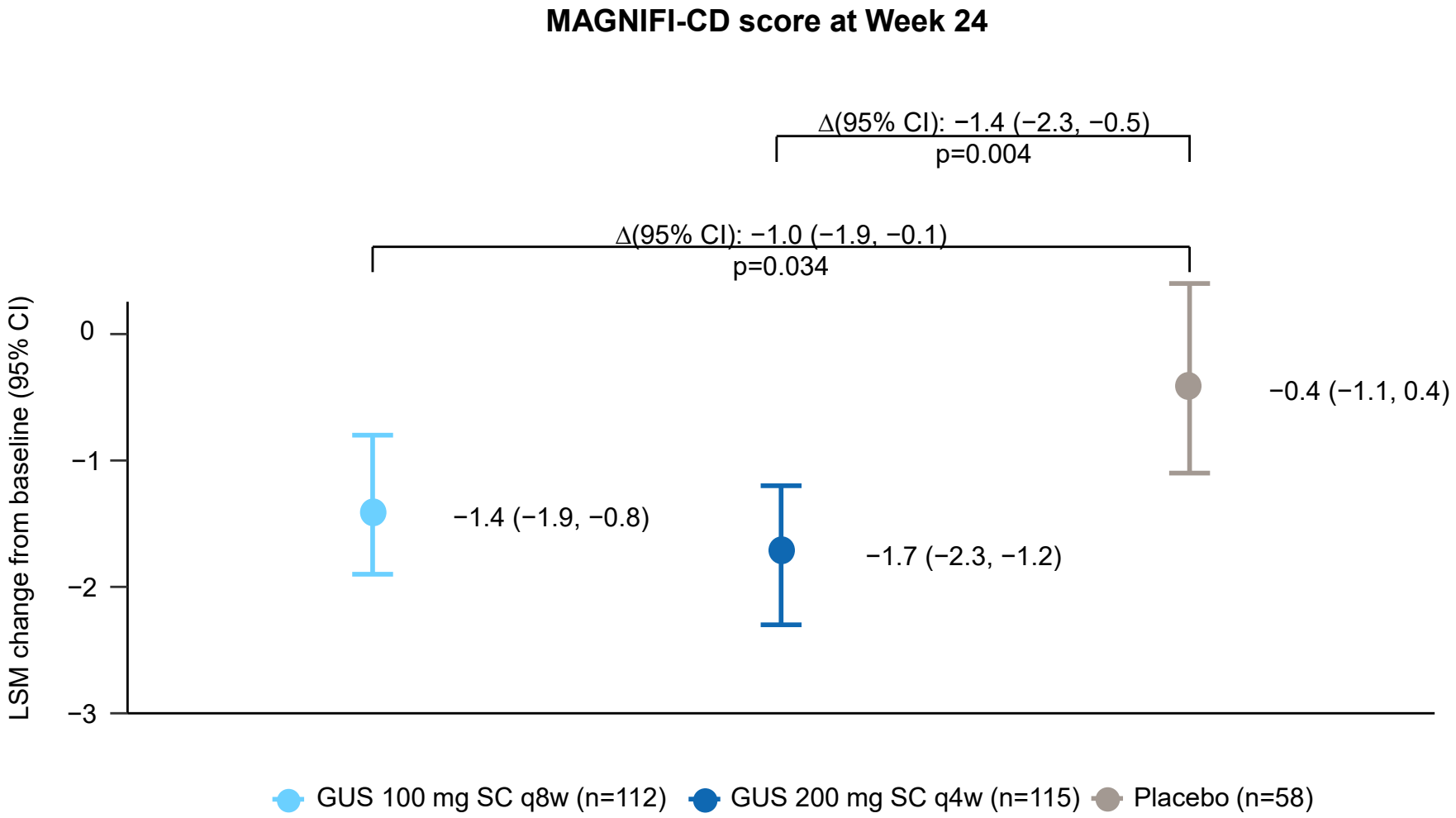
GUS demonstrated significantly greater clinically assessed fistula remission rates versus placebo at Week 24



Clinically assessed fistula remission: 100% closure of all external openings, without development of new fistulas or abscesses and without any drainage by the external openings, occurring spontaneously or after gentle finger compression.
Printed p-values are multiplicity-controlled.
CD, Crohn's disease; GUS, guselkumab; IL, interleukin; IV, intravenous; MOA, mechanism of action; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous.
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Both GUS dose regimens achieved greater MAGNIFI-CD score improvement from baseline to Week 24 versus placebo



MAGNIFI-CD score: Score ranges from 0 (no disease activity) to 25 (severe disease activity).
Nominal p-values.
CD, Crohn's disease; CI, confidence interval; GUS, guselkumab; IL, interleukin; IV, intravenous; LSM, least squares mean; MAGNIFI-CD, Magnetic Resonance Imaging Global Assessment of Fistulas in Crohn's Disease; MOA, mechanism of action; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous; Δ, adjusted difference.
Sands BE, et al. Presented at ACG, Nashville, Tennessee, USA, 9–14 October 2026. P5691. This specific population is currently still under experimental investigation. Guselkumab is approved for adult moderate-severe CD patients. Full prescription information: swissmedicinfo-pro.ch.

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Adverse events rates with GUS were similar to placebo through Week 24

Safety through Week 24	GUS 100 mg SC q8w (n=113)	GUS 200 mg SC q4w (n=115)	Placebo (n=58)
Mean duration of follow-up (weeks)	24.0	24.0	23.8
Mean exposure (number of study agent administrations)	5.7	5.8	5.6
≥1 AE, n (%)	76 (67)	80 (70)	48 (83)
≥1 SAE, n (%)	12 (11)	7 (6)	8 (14)
≥1 AE leading to discontinuation of study agent, n (%)	8 (7)	3 (3)	5 (9)
≥1 infection, n (%)	45 (40)	31 (27)	27 (47)
≥1 serious infection, n (%)	8 (7)	2 (2)	2 (3)

AE, adverse event; CD, Crohn's disease; GUS, guselkumab; IL, interleukin; MOA, mechanism of action; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; SAE, serious adverse event; SC, subcutaneous.

Sands BE, et al. Presented at ACG, Nashville, Tennessee, USA, 9–14 October 2026. P5691. This specific population is currently still under experimental investigation. Guselkumab is approved for adult moderate-severe CD patients. Full prescription information: swissmedicinfo-pro.ch.

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Conclusions[1]

- FUZION is the first large, international Phase 3 study in more than two decades to demonstrate the benefit of an advanced therapy in adults with active perianal fistulising CD
- GUS induction and maintenance therapy demonstrated clinical and radiological benefits versus placebo through Week 24
- At Week 24, GUS achieved numerically higher combined fistula remission rates versus placebo across all subgroups, including participants with prior biologic exposure and infliximab failure
- GUS demonstrated improvements in clinically assessed fistula response as early as Week 12 and achieved greater improvements in MAGNIFI-CD scores versus placebo at Week 24
- GUS demonstrated a favourable benefit–risk profile through Week 24, with no new safety signals identified[2]

CD, Crohn's disease; GUS, guselkumab; IL, interleukin; MAGNIFI-CD, Magnetic Resonance Imaging Global Assessment of Fistulas in Crohn's Disease; MOA, mechanism of action; RDBPC, randomised, double-blind, placebo-controlled.

1. Sands BE, et al. Presented at ACG, Nashville, Tennessee, USA, 9–14 October 2026. P5691; 2. Peyrin-Biroulet L, et al. Presented at DDW, Chicago, IL, USA, 2–5 May 2026. O1058b. This specific population is currently still under experimental investigation. Guselkumab is approved for adult moderate-severe CD patients. Full prescription information: swissmedicinfo-pro.ch.

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Efficacy and safety of guselkumab in perianal fistulizing Crohn's disease: Week 48 results from the Phase 3, randomized, double-blind, placebo-controlled FUZION trial

LB09

Oral presentation at UEGW 2026

Ailsa Hart,[1] Axel Dignass,[2] Vipul Jairath,[3] Tadakazu Hisamatsu,[4] Bruce E. Sands,[5] Antonino Spinelli,[6,7] Susan J. Connor,[8] André D'Hoore,[9] Walter Reinisch,[10] Silvio Danese,[11] David A. Schwartz,[12] Julian Panés,[13] Bram Verstockt,[14] Anton Stift,[15] Maciej Nazar,[16] Jacqueline van Denderen,[17] Takehiko Sakamoto,[18] Koji Masuda,[18] Stephen Xu,[19] Ivana Bravatà,[20] Geert D'Haens,[21] and Laurent Peyrin-Biroulet[22] on behalf of the FUZION Study Group

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Primary and secondary endpoints and outcome definitions in FUZION

Primary endpoint*

Week 24: Combined fistula **remission**† (clinically and radiologically assessed)

Multiplicity-controlled Secondary endpoints*

Week 24:

- Clinically assessed fistula **remission**
- Clinically assessed fistula **response**

Other secondary endpoints

Week 12: clinically assessed fistula **response**

Week 48:

- Combined fistula **remission**
- Clinically assessed fistula **remission**
- Clinically assessed fistula **response**

Assessment Type	Outcome	Definition
Clinical	Clinically assessed fistula **remission**	100% closure of all external openings, without development of new fistulas or abscesses and without any drainage by the external openings, occurring spontaneously or after gentle finger compression
	Clinically assessed fistula **response**	≥50% reduction from baseline in number of open or draining perianal fistulas
Radiological	Absence of collections >2cm	Absence of collections >2 cm of the perianal fistulas, confirmed by a blinded central review of the MRI results

*In the statistical testing procedure, GUS 200 mg q4w dose then the 100 mg q8w dose were sequentially tested versus placebo for the primary endpoint then for the multiplicity-controlled secondary endpoints. †Combined fistula remission definition was previously used in Panés J. *Lancet*. 2016;388:1281–90.
CD, Crohn's disease; GUS, guselkumab; ICE, intercurrent event; IL, interleukin; MOA, mechanism of action; MRI, magnetic resonance imaging; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled.
Hart A, et al. Presented at UEGW, Barcelona, Spain, 17–20 October 2026. LB09. This specific population is currently still under experimental investigation. Guselkumab is approved for adult moderate-severe CD patients.
Full prescription information: swissmedicinfo-pro.ch.

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Demographics and baseline disease characteristics

	GUS 100 mg q8w (n=113)	GUS 200 mg q4w (n=115)	GUS combined (n=228)	Placebo (n=58)
Demographics				
Age, years, mean (SD)	36.2 (12.7)	36.0 (13.0)	36.1 (12.8)	38.0 (12.9)
Male, n (%)	79 (70)	85 (74)	164 (72)	41 (71)
Disease characteristics				
CD duration, years, median (IQ), years	5.6 (1.9; 15.6)	6.8 (2.1; 15.9)	6.1 (1.9; 15.8)	7.75 (3.4; 17.6)
CDAI score	154.6 (97.6)	143.2 (84.6)*	149.0 (91.43)†	147.6 (104.3)‡
> 220, n (%)	25 (22)	23 (21)	48 (22)	10 (18)
≤ 220, n (%)	88 (78)	85 (79)	173 (78)	46 (82)
Participants with open or draining fistula, n (%)				
1 fistula	73 (65)	60 (52)	133 (58)	33 (57)
≥2 fistulas	40 (35)	55 (48)	95 (42)	25 (43)
Participants with complex fistula§, n (%)	52 (46)	64 (56)	116 (51)	30 (52)
Previous biologic exposure, n (%)				
Bio-naïve	43 (38)	38 (33)	81 (36)	16 (28)
Bio-exposed	70 (62)	77 (67)	147 (64)	42 (72)
Proctitis, n (%)	31 (27)	37 (32)	68 (30)	16 (28)
Seton present, n (%)	23 (20)	21 (18)	44 (19)	10 (17)
Fistula-related surgery during screening, n (%)	17 (15)	18 (16)	35 (15)	7 (12)
Participants with collections >2 cm, n (%)	0	4 (4)	4 (2)	2 (3)

Data shown are mean (SD) unless otherwise noted. *n=108; †n=221; ‡n=56; §Fistula complexity was determined by MRI using MAGNIFI-CD "number of fistula tracts" (single/unbranched or complex).
CD, Crohn's disease; CDAI, Crohn's Disease Activity Index; GUS, guselkumab; IL, interleukin; IQR, interquartile range; LTE, long-term extension; MOA, mechanism of action; MRI, magnetic resonance imaging; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; SD, standard deviation.
Hart A, et al. Presented at UEGW, Barcelona, Spain, 17–20 October 2026. LB09. This specific population is currently still under experimental investigation. Guselkumab is approved for adult moderate-severe CD patients.
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Prior and concomitant medications for CD

	GUS 100 mg q8w (n=113)	GUS 200 mg q4w (n=115)	GUS combined (n=228)	Placebo (n=58)
Prior exposure to Crohn's disease medication, n (%)	70 (62)	77 (67)	147 (64)	42 (72)
Infliximab	52 (46)	58 (50)	110 (48)	32 (55)
Adalimumab	38 (34)	42 (37)	80 (35)	23 (40)
Certolizumab	1 (1)	2 (2)	3 (1)	1 (2)
Vedolizumab	8 (7)	6 (5)	14 (6)	3 (5)
Ustekinumab*	4 (4)	3 (3)	7 (3)	5 (9)
Only 1 anti-TNF agent	43 (38)	51 (44)	94 (41)	27 (47)
≥2 anti-TNF agents	24 (21)	25 (22)	49 (21)	14 (24)
Only one MOA	62 (55)	69 (60)	131 (57)	35 (60)
Concomitant CD medications, n (%)				
5-ASA	27 (24)	27 (23)	54 (24)	19 (33)
Corticosteroids	10 (9)	9 (8)	19 (8)	3 (5)
Immunosuppressants (AZA, 6-MP, MTX)	34 (30)	35 (30)	69 (30)	16 (28)

*Participants who were exposed to ustekinumab at its approved labelled dosage AND met the required washout criterion (16 weeks) AND had not demonstrated inadequate response or intolerance to ustekinumab AND received one IV induction dose of ~6 mg/kg and one SC maintenance dose of 90 mg.

5-ASA, 5-aminosalicylic acid; 6-MP, 6-mercaptopurine; AZA, azathioprine; CD, Crohn's disease; GUS, guselkumab; IL, interleukin; MOA, mechanism of action; MTX, methotrexate; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous; TNF, tumour necrosis factor.

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Full prescription information: swissmedicinfo-pro.ch.

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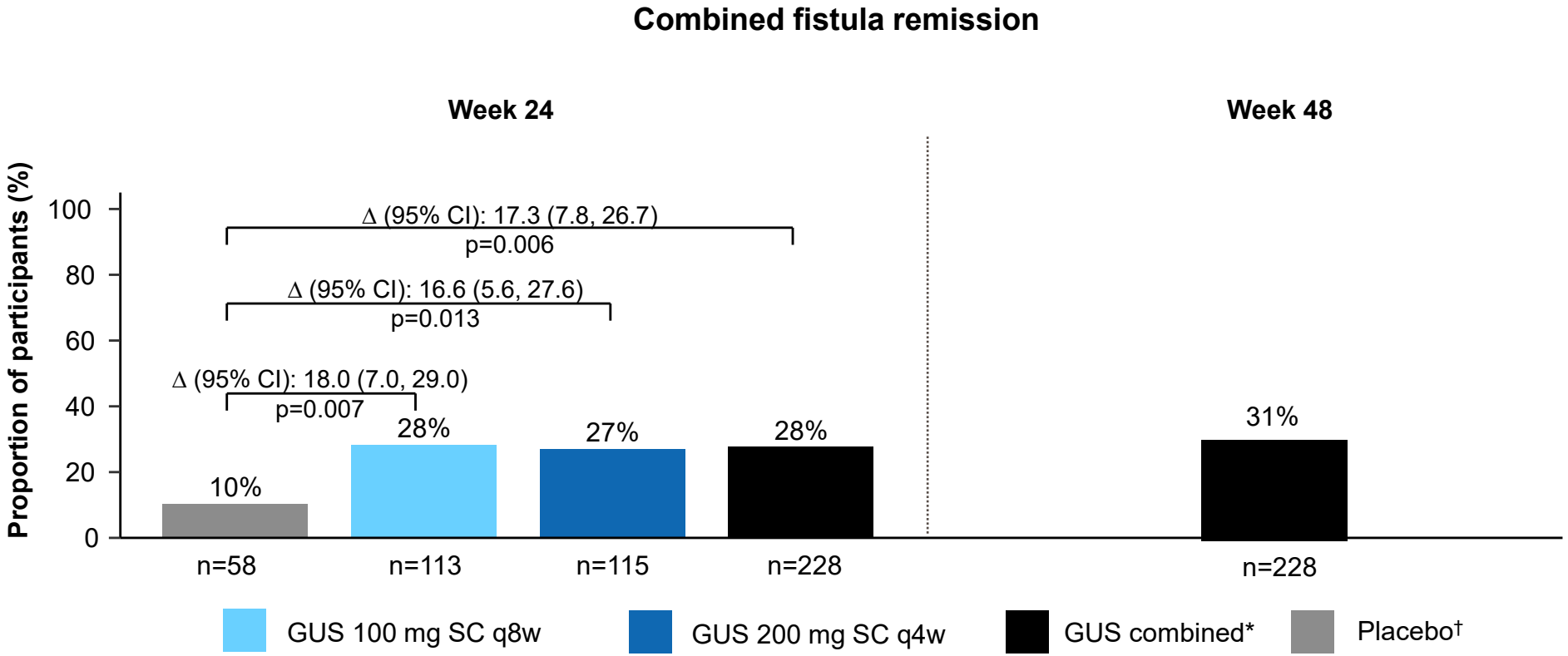
Discontinuation of study agent before Week 48

	GUS 100 mg q8w (n=113)	GUS 200 mg q4w (n=115)	GUS combined (n=228)	Placebo (n=58)
Completed 48 weeks of treatment, n (%)	97 (86)	109 (95)	206 (90)	49 (84)
Discontinued study treatment, n (%)	16 (14)	6 (5)	22 (10)	9 (16)
Reason for discontinuing study treatment, n (%)				
Discontinued due to AEs	10 (9)	5 (4)	15 (7)	5 (9)
AE - other	5 (4)	4 (3)	9 (4)	1 (2)
AE - worsening of CD	5 (4)	1 (1)	6 (3)	4 (7)
Death	0	0	0	0
Lack of efficacy	1 (1)	0	1 (<1)	1 (2)
Lost to follow-up	1 (1)	0	1 (<1)	0
Physician decision	1 (1)	0	1 (<1)	0
Protocol deviation	0	0	0	0
Pregnancy	0	0	0	0
Participant refused further study treatment	0	0	0	1 (2)
Withdrawal of consent	3 (3)	0	3 (1)	0
Prohibited CD-related surgery	0	0	0	0
Other	0	1 (1)	1 (<1)	2 (3)

AE, adverse event; CD, Crohn's disease; GUS, guselkumab; IL, interleukin; MOA, mechanism of action; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous.
Hart A, et al. Presented at UEGW, Barcelona, Spain, 17–20 October 2026. LB09. This specific population is currently still under experimental investigation. Guselkumab is approved for adult moderate-severe CD patients.
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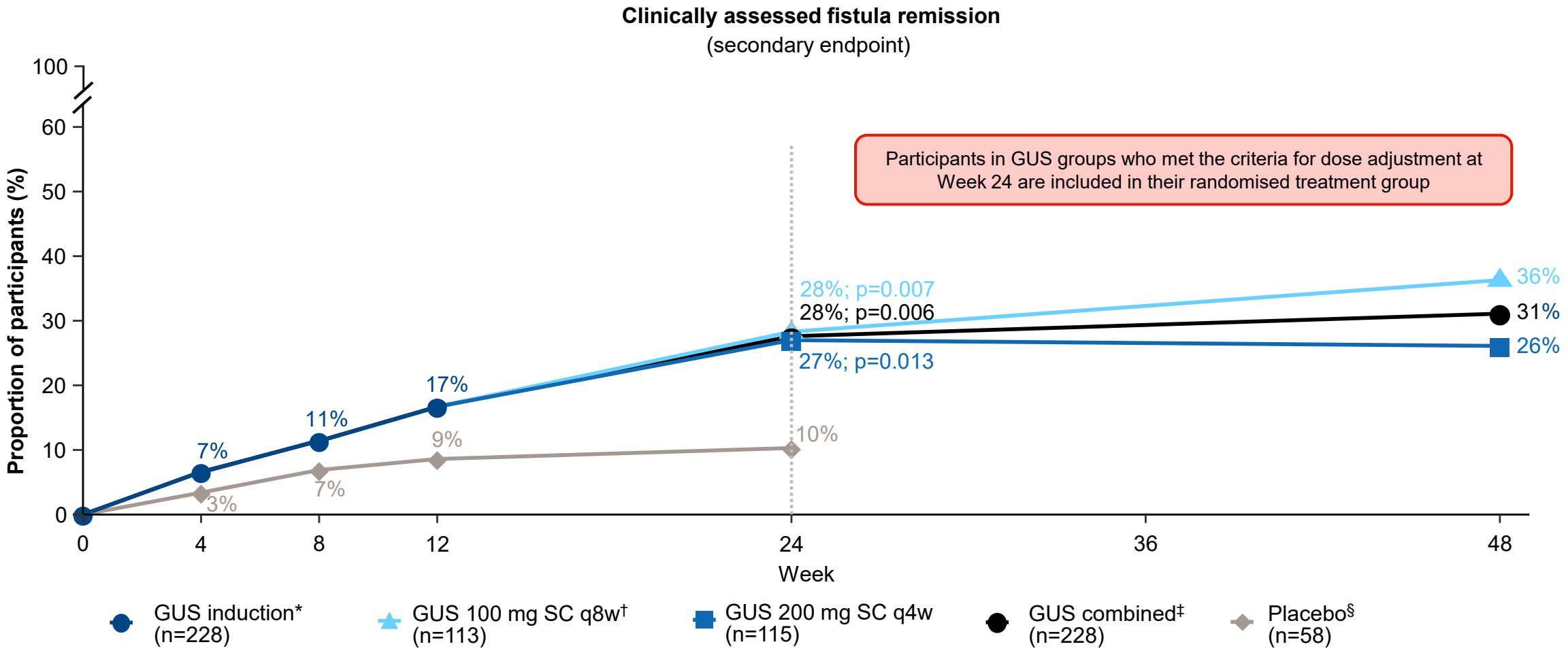
Combined fistula remission through Week 48



Combined fistula remission: 100% closure of all treated external openings, without development of new fistulas or abscesses and without any drainage by the external openings, occurring spontaneously or after gentle finger compression AND absence of collections >2 cm of the perianal fistulas, confirmed by a blinded central review of the MRI results.
After applying the intercurrent event strategy in the statistical analysis, missing data were imputed as not having achieved binary endpoints. *GUS combined included both maintenance doses from Week 12 through Week 48. †Placebo non-responders received GUS after Week 24; therefore, there was no placebo-controlled treatment arm beyond Week 24.
CD, Crohn's disease; CI, confidence interval; GUS, guselkumab; IL, interleukin; MOA, mechanism of action; MRI, magnetic resonance imaging; NR, non-responder; qXw, every X weeks; R, responders; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous.
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Clinically assessed fistula remission through Week 48



Clinically assessed fistula remission: 100% closure of all treated external openings, without development of new fistulas or abscesses and without any drainage by the external openings, occurring spontaneously or after gentle finger compression.

After applying the intercurrent event strategy in the statistical analysis, missing data were imputed as not having achieved binary endpoints. *GUS 200 mg IV induction at Weeks 0/4/8. †At Week 48, GUS 100 mg SC q8w group includes participants on 100 mg SC q8w (R), and those (NRs) with dose adjusted to 200 mg SC q4w at Week 24. ‡GUS combined included both maintenance doses from Week 12 through Week 48. §Placebo non-responders received blinded GUS dose adjustment after Week 24; therefore, there was no placebo-controlled treatment arm beyond Week 24.

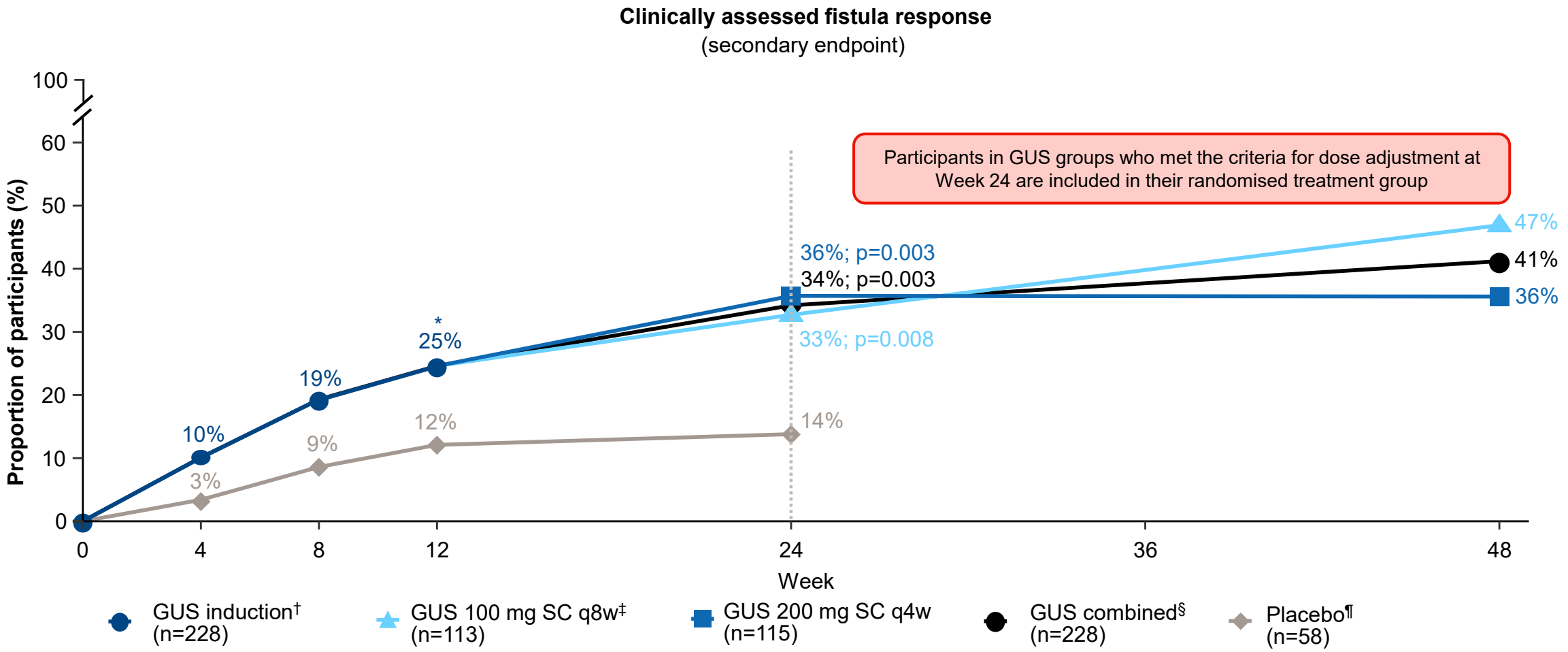
CD, Crohn's disease; GUS, guselkumab; IL, interleukin; IV, intravenous; MOA, mechanism of action; NR, non-responders; qXw, every X weeks; R, responders; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous.

Hart A, et al. Presented at UEGW, Barcelona, Spain, 17–20 October 2026. LB09. This specific population is currently still under experimental investigation. Guselkumab is approved for adult moderate-severe CD patients.

Full prescription information: swissmedicinfo-pro.ch.

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Clinically assessed fistula response through Week 48



Clinically assessed fistula response: ≥50% reduction from baseline in the number of open or draining perianal fistulas.
After applying the intercurrent event strategy in the statistical analysis, missing data were imputed as not having achieved binary endpoints. p-values are multiplicity-controlled, except for GUS combined (nominal).
*Nominal p<0.05 vs placebo. †GUS 200 mg IV induction at Weeks 0/4/8; ‡At Week 48, GUS 100 mg SC q8w group includes participants on 100 mg SC q8w (R), and those (NRs) with dose adjusted to 200 mg SC q4w at Week 24. §GUS combined included both maintenance doses from Week 12 through Week 48. ¶Placebo non-responders received blinded GUS dose adjustment after Week 24; therefore, there was no placebo-controlled treatment arm beyond Week 24.
CD, Crohn's disease; GUS, guselkumab; IL, interleukin; IV, intravenous; MOA, mechanism of action; NR, non-responders; qXw, every X weeks; R, responders; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous.
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Adverse events through Week 48

	GUS 100 mg SC q8w (n=113)*	GUS 200 mg SC q4w (n=115)*	Combined GUS (n=228)*	All GUS (n=268)†	Placebo (n=58)*
Mean duration of follow-up (weeks)	33.6	33.6	33.6	42.8	28.7
Mean exposure (number of study agent administrations)	8.4	8.5	8.4	11.3	6.9
Subjects with ≥1 AE	80 (71%)	88 (77%)	168 (74%)	216 (81%)	48 (83%)
Subjects with ≥1 SAE	18 (16%)	9 (8%)	27 (12%)	34 (13%)	8 (14%)
Subjects with ≥1 AE leading to study agent discontinuation	8 (7%)	5 (4%)	13 (6%)	16 (6%)	5 (9%)
Subjects with ≥1 infection	52 (46%)	41 (36%)	93 (41%)	126 (47%)	28 (48%)
Subjects with ≥1 anal abscess	13 (12%)	6 (5%)	19 (8%)	26 (10%)	3 (5%)
Subjects with ≥1 serious infection	13 (12%)	2 (2%)	15 (7%)	17 (6%)	2 (3%)
Subjects with ≥1 serious gastrointestinal disorders	5 (4%)	3 (3%)	8 (4%)	10 (4%)	4 (7%)

- GUS was well tolerated through Week 48, with overall adverse event rates similar to placebo with both doses
- Two participants with malignancies (plasmablastic lymphoma and NMSC) were reported in the GUS 200 mg group, which were assessed as unrelated to study intervention by investigator
- No reported cases of additional AEs of interest (anaphylactic reactions, opportunistic infections, major adverse cardiovascular events, venous thromboembolisms, active tuberculosis, or clinically important hepatic disorders)

Data shown are n (%) unless otherwise noted. *Excludes data from Week 24 to Week 48 of participants who were dose adjusted to GUS 200 mg q4w at Week 24. †Includes data for participants who received at least one dose of GUS, covering the period from their first GUS dose through Week 48.
AE, adverse event; CD, Crohn's disease; GUS, guselkumab; IL, interleukin; MOA, mechanism of action; NMSC, non-melanoma skin cancer; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; SAE, serious adverse event; SC, subcutaneous.
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Conclusions

- FUZION is the first large international Phase 3 study dedicated to perianal fistulizing Crohn's disease in more than two decades that showed efficacy of an advanced therapy
- GUS showed superior efficacy to placebo at Week 24
- GUS demonstrated sustained efficacy through Week 48, supporting its continued use for durable fistula remission
- GUS was generally well tolerated through Week 48, with no new safety signals identified

CD, Crohn's disease; GUS, guselkumab; IL, interleukin; MOA, mechanism of action; MRI, magnetic resonance imaging; RDBPC, randomised, double-blind, placebo-controlled.
Hart A, et al. Presented at UEGW, Barcelona, Spain, 17–20 October 2026. LB09. This specific population is currently still under experimental investigation. Guselkumab is approved for adult moderate-severe CD patients.
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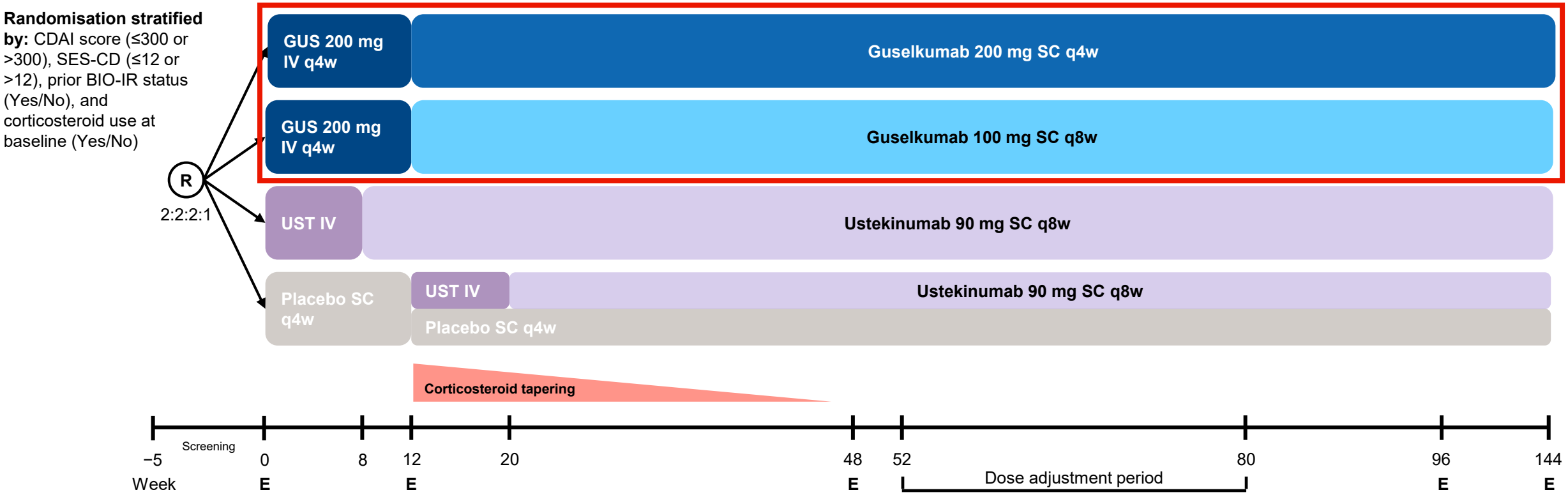
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GALAXI: Guselkumab for luminal CD

GALAXI 2 and 3 study designs through Week 144

Key eligibility criteria

- Moderately to severely active Crohn's disease (CDAI score 220–450 AND either mean daily SF >3 OR AP >1) and SES-CD score ≥6 (or ≥4 for isolated ileal disease), and inadequate response or intolerance to oral corticosteroids, 6-MP/AZA/MTX, or biologic therapies*



*Biologic therapies: TNF antagonists or vedolizumab.
Note: GALAXI 2 & 3 were not designed to compare guselkumab with ustekinumab beyond Week 48; blinding held through the Week 48 analysis and database lock. Participants treated with placebo during the LTE were discontinued after study unblinding. The full study designs can be found in the primary trial publication.
6-MP, 6-mercaptopurine; AP, abdominal pain; AZA, azathioprine; BIO-IR, biologic inadequate response or intolerance; CD, Crohn's disease; CDAI, Crohn's Disease Activity Index; E, endoscopy; GUS, guselkumab; IL, interleukin; IV, intravenous; LTE, long-term extension; MOA, mechanism of action; MTX, methotrexate; qXw, every X weeks; R, randomised; RDBAPC, randomised, double-blind, active- and placebo-controlled; SC, subcutaneous; SES-CD, Simple Endoscopic Score for Crohn's Disease; SF, stool frequency; TNF, tumour necrosis factor; UST, ustekinumab.
Sands BE, et al. Presented at UEGW, Barcelona, Spain, 17–20 October 2026. OP105. Full prescription information: swissmedicinfo-pro.ch.

CH_CP-609070 10//26

Efficacy and safety of guselkumab maintenance therapy in patients with moderately to severely active Crohn's disease: Results through Week 144 from the Phase 3 GALAXI 2 & 3 studies

OP105

Oral presentation at UEGW

First presented as a poster at ACG 2026 (P5693)

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CH_CP-609070 10//26

Endpoints and analysis populations

Aim: Evaluate the long-term efficacy and safety of GUS through Week 144 in the pooled GALAXI 2 and 3 long-term extension

Endpoints

Clinical endpoints

- Clinical remission: CDAI score <150
- 90-day corticosteroid-free clinical remission

Endoscopic endpoints

- Endoscopic response: ≥50% improvement from baseline in SES-CD or SES-CD score ≤2
- Endoscopic remission: SES-CD score ≤4 and at least a 2-point reduction from baseline, with no subscore >1 in any individual component

Composite endpoint

- Deep remission: Combination of clinical remission and endoscopic remission

Analysis populations

- **Primary efficacy analysis set (NRI):** All randomised participants who received ≥1 dose of guselkumab evaluated by randomised group
- **LTE as observed analysis:** Participants who received ≥1 dose in the long-term extension, remained on continuous treatment through each designated time point, and had data available at that time point
- **All treated safety analysis set:** All participants who received ≥1 dose of guselkumab, pooled by actual treatment received

Statistical considerations

- **Primary efficacy analysis (NRI):**
  - Participants with prohibited CD medication changes before Week 48, dose adjustment (including “sham”) during Weeks 52–80, or CD-related surgery or discontinuation of study intervention at any time were considered not to have met the endpoint at the designated time point from the point of incurrent event
  - Missing endpoint data were imputed as non-response
- **LTE as observed analysis:** No nonresponder imputation; only participants with available data and on treatment at the assessment time point were included in the analysis

CD, Crohn’s disease; CDAI, Crohn’s Disease Activity Index; GUS, guselkumab; IL, interleukin; LTE, long-term extension; MOA, mechanism of action; NRI, non-responder imputation; RDBAPC, randomised, double-blind, active- and placebo-controlled; SES-CD, Simple Endoscopic Score for Crohn’s Disease.

Sands BE, et al. Presented at UEGW, Barcelona, Spain, 17–20 October 2026. OP105. Full prescription information: swissmedicinfo-pro.ch.

Baseline demographics and disease characteristics

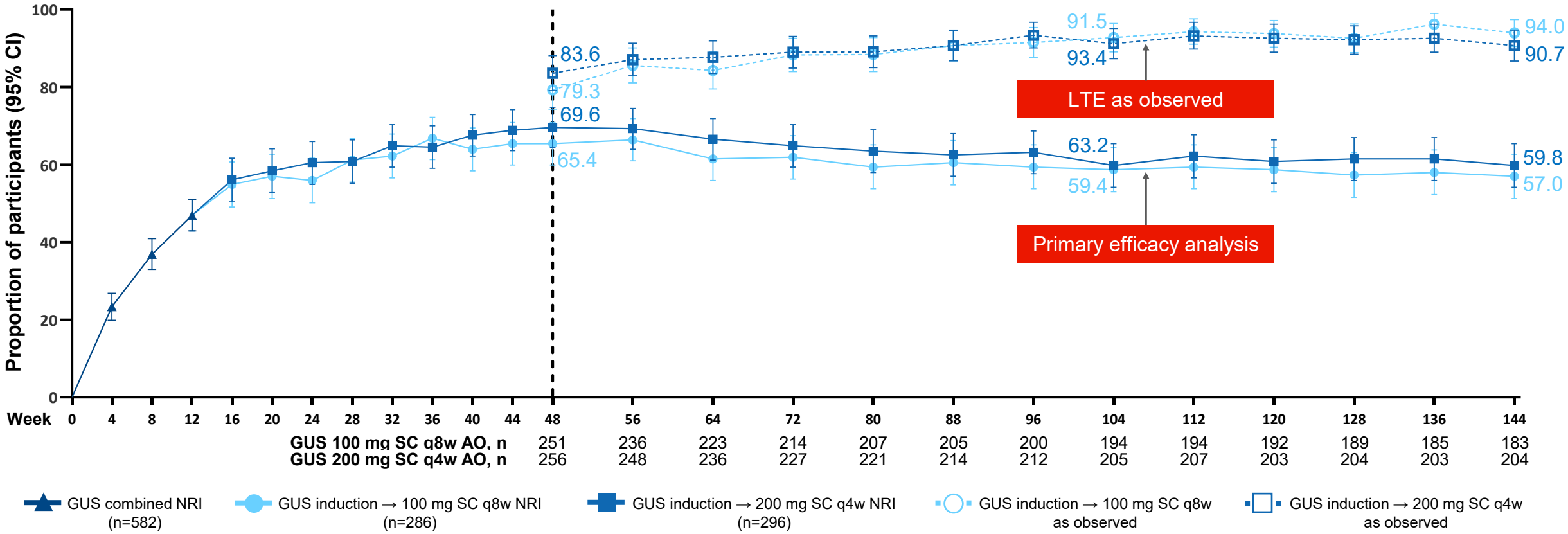
Primary analysis set (NRI)	GUS 200 mg IV → 100 mg SC q8w (n=286)	GUS 200 mg IV → 200 mg SC q4w (n=296)
Demographics		
Age, years, mean (SD)	36.0 (12.24)	36.9 (13.27)
Male, n (%)	154 (53.8)	178 (60.1)
Disease characteristics		
CD duration, years, mean (SD)	7.12 (6.72)	7.07 (7.24)
CDAI score, mean (SD)	296.3 (54.27)	295.9 (52.74)
SES-CD score, mean (SD)	13.2 (7.43)	12.5 (7.24)
Endoscopic disease severity (SES-CD score), n (%)		
Moderate (7–16)	164 (57.3)	147 (49.7)
Severe (>16)	81 (28.3)	79 (26.7)
Involved disease location by central reader, n (%)		
Colon only	113 (39.5)	112 (37.8)
Ileum only	59 (20.6)	80 (27.0)
Ileum and colon	114 (39.9)	104 (35.1)
Biomarkers		
C-reactive protein in mg/L, median (IQR)	7.7 (2.6; 21.8)	6.2 (2.7; 21.3)
Faecal calprotectin in µg/g,* median (IQR)	969.0 (404.0; 2085.0)	1045.5 (323.0; 2006.0)
Biologic medication history, n (%)		
Bio-naïve	116 (40.6)	128 (43.2)
History of inadequate response or intolerance to biologic therapy†	153 (53.5)	147 (49.7)
Concomitant CD medications at baseline, n (%)		
6-MP/AZA	85 (29.7)	89 (30.1)
MTX	2 (0.7)	7 (2.4)
Oral corticosteroids	109 (38.1)	106 (35.8)

Of 582 participants who received GUS IV induction, 432 (74%) continued treatment through Week 144

*Based on n=283 for GUS 100 mg and n=294 for GUS 200 mg; †Primary nonresponse, secondary nonresponse or intolerance.
Note: Baseline demographics and disease characteristics are shown for the primary efficacy analysis set (NRI).
6-MP, 6-mercaptopurine; AZA, azathioprine; CD, Crohn's disease; CDAI, Crohn's Disease Activity Index; GUS, guselkumab; IL, interleukin; IQR, interquartile range; IV, intravenous; MOA, mechanism of action; MTX, methotrexate; NRI, non-responder imputation; qXw, every X weeks; RDBAPC, randomised, double-blind, active- and placebo-controlled; SC, subcutaneous; SD, standard deviation; SES-CD, Simple Endoscopic Score for Crohn's Disease.
Sands BE, et al. Presented at UEGW, Barcelona, Spain, 17–20 October 2026. OP105. Full prescription information: swissmedicinfo-pro.ch.

CH_CP-609070 10//26

Clinical remission through Week 144



Among participants in clinical remission at Week 144, 94.5% (100 mg SC q8w) and 96.6% (200 mg SC q4w) had been corticosteroid-free for ≥90 days; primary efficacy analysis set (NRI)

Clinical remission: CDAI score <150.

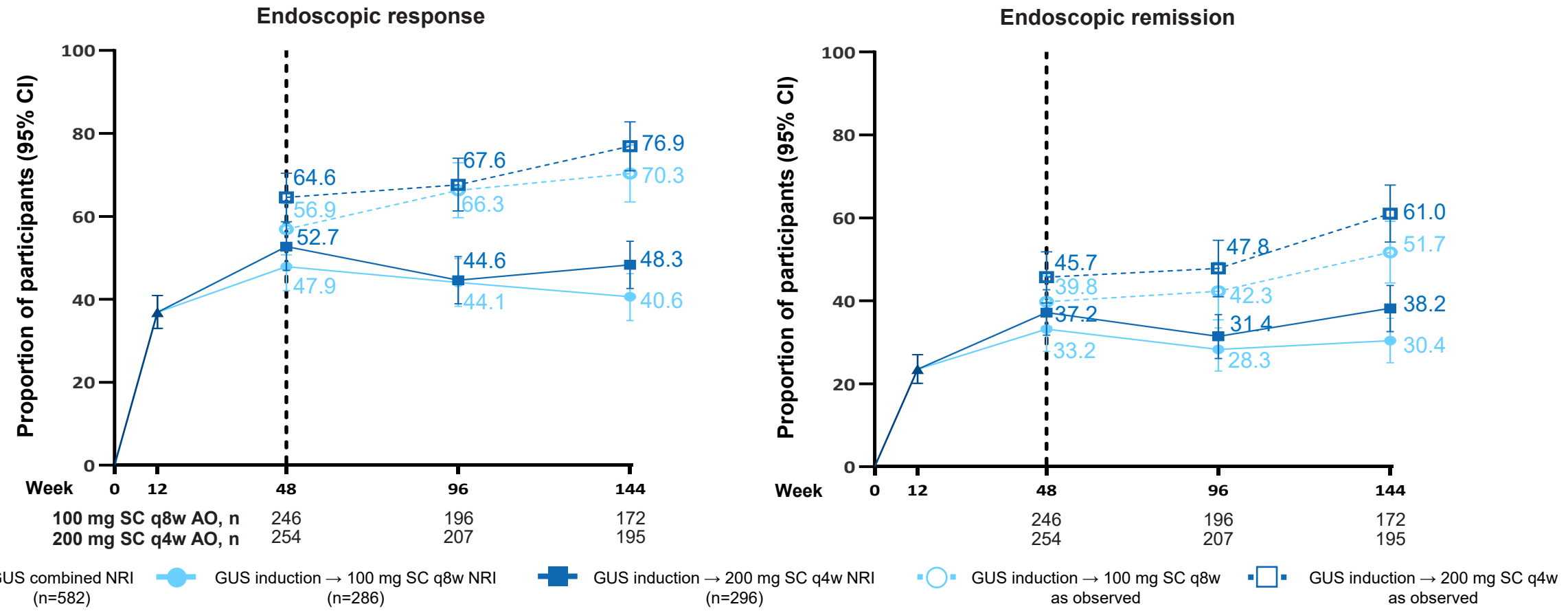
Note: Participants who were not in clinical response (≥100-point reduction in CDAI score from baseline or CDAI <150) and had CDAI ≥220 were eligible for dose adjustment (i.e., GUS 100 mg SC q8w → 200 mg SC q4w or "sham" for participants treated with GUS 200 mg SC q4w) between Weeks 52 and 80. Participants in the LTE as-observed analysis who had a dose adjustment from GUS 100 mg SC q8w to GUS 200 mg SC q4w are not included from the day after the date of dose adjustment.

AO, as observed; CD, Crohn's disease; CDAI, Crohn's Disease Activity Index; CI, confidence interval; GUS, guselkumab; IL, interleukin; LTE, long-term extension; MOA, mechanism of action; NRI, non-responder imputation; qXw, every X weeks; RDBAPC, randomised, double-blind, active- and placebo-controlled; SC, subcutaneous.

Sands BE, et al. Presented at UEGW, Barcelona, Spain, 17–20 October 2026. OP105 and abstract. Full prescription information: swissmedicinfo-pro.ch.

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Endoscopic outcomes through Week 144



Endoscopic response: ≥50% improvement from baseline in SES-CD or SES-CD score ≤2. **Endoscopic remission:** SES-CD score ≤4 and at least a 2-point reduction from baseline, with no subscore >1 in any individual component.

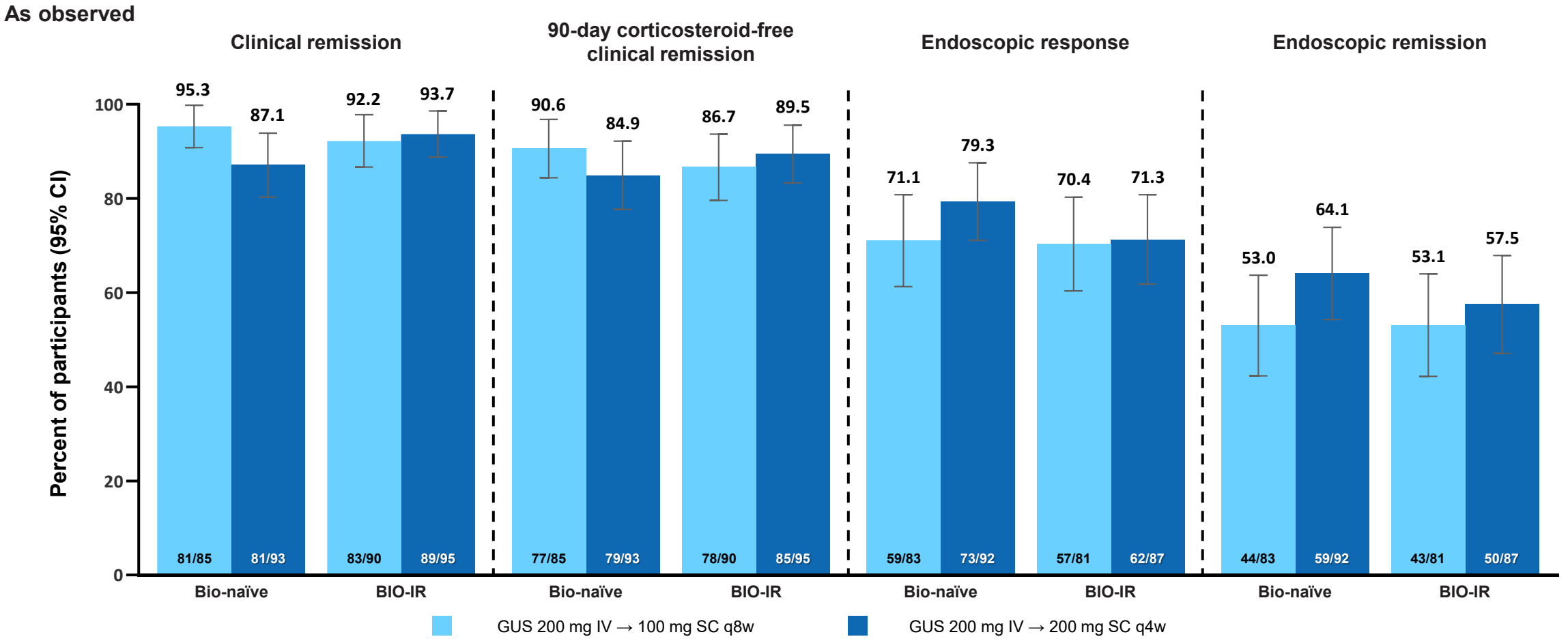
Note: Participants who were not in clinical response (≥100-point reduction in CDAI score from baseline or CDAI <150) and had CDAI ≥220 were eligible for dose adjustment (i.e., GUS 100 mg SC q8w → 200 mg SC q4w or "sham" for participants treated with GUS 200 mg SC q4w) between Weeks 52 and 80. Participants in the LTE as-observed analysis who had a dose adjustment from GUS 100 mg SC q8w to GUS 200 mg SC q4w are not included from the day after the date of dose adjustment.

AO, as observed; CD, Crohn's disease; CDAI, Crohn's Disease Activity Index; CI, confidence interval; GUS, guselkumab; IL, interleukin; LTE, long-term extension; MOA, mechanism of action; NRI, non-responder imputation; qXw, every X weeks; RDBAPC, randomised, double-blind, active- and placebo-controlled; SC, subcutaneous; SES-CD, Simple Endoscopic Score for Crohn's Disease.

Sands BE, et al. Presented at UEGW, Barcelona, Spain, 17–20 October 2026. OP105 and abstract. Full prescription information: swissmedicinfo-pro.ch.

CH_CP-609070 10//26

Outcomes by biologic history at Week 144



Clinical remission: CDAI score <150. **Endoscopic response:** ≥50% improvement from baseline in SES-CD or SES-CD score ≤2. **Endoscopic remission:** SES-CD score ≤4 and at least a 2-point reduction from baseline, with no subscore >1 in any individual component. **Deep remission:** Combination of clinical remission and endoscopic remission.

Note: Participants who were not in clinical response (≥100-point reduction in CDAI score from baseline or CDAI <150) and had CDAI ≥220 were eligible for dose adjustment (i.e., GUS 100 mg SC q8w → 200 mg SC q4w or "sham" for participants treated with GUS 200 mg SC q4w) between Weeks 52 and 80. Participants in the LTE as observed analysis who had a dose adjustment from GUS 100 mg SC q8w to GUS 200 mg SC q4w are not included from the day after the date of dose adjustment.

BIO-IR, biologic inadequate response or intolerance; CD, Crohn's disease; CDAI, Crohn's Disease Activity Index; CI, confidence interval; GUS, guselkumab; IL, interleukin; IV, intravenous; LTE, long-term extension; MOA, mechanism of action; qXw, every X weeks; RDBAPC, randomised, double-blind, active- and placebo-controlled; SC, subcutaneous; SES-CD, Simple Endoscopic Score for Crohn's Disease.

Sands BE, et al. Presented at UEGW, Barcelona, Spain, 17–20 October 2026. OP105. Full prescription information: swissmedicinfo-pro.ch.

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Safety summary from Week 0 through Week 144

All treated safety analysis set	GUS 100 mg SC q8w* (n=296)	All GUS 200 mg SC q4w† (n=407)	Placebo (n=153)*‡
Exposure			
Mean duration of follow-up, weeks	113.5	111.6	30.0
Total participant-years of follow-up	644.1	870.2	88.0
Events per 100 participant-years of follow-up (95% CI)			
AEs	274.2 (261.54, 287.28)	295.8 (284.47, 307.44)	422.8 (380.92, 468.02)
SAEs	11.6 (9.16, 14.60)	10.7 (8.63, 13.09)	27.3 (17.48, 40.59)
AEs leading to discontinuation of study agent	5.1 (3.53, 7.20)	5.1 (3.67, 6.79)	19.3 (11.26, 30.94)
Infections§	74.4 (67.86, 81.34)	76.2 (70.50, 82.21)	79.6 (62.02, 100.52)
Serious infections§	0.9 (0.34, 2.03)	1.6 (0.88, 2.70)	4.5 (1.24, 11.64)
AEs of interest per 100 participant-years of follow-up (95% CI)			
Active tuberculosis	0.2 (0.00, 0.87)	0.1 (0.00, 0.64)	0.0 (0.00, 3.40)
Malignancies	0.2 (0.00, 0.87)	0.3 (0.07, 1.01)	0.0 (0.00, 3.40)
Anaphylactic or serum sickness reactions	0.0 (0.00, 0.47)	0.0 (0.00, 0.34)	1.1 (0.03, 6.33)
Opportunistic infections	0.2 (0.00, 0.87)	1.3 (0.63, 2.26)	2.3 (0.28, 8.21)
Major adverse cardiovascular events	0.2 (0.00, 0.87)	0.0 (0.00, 0.34)	0.0 (0.00, 3.40)
Venous thromboembolism	0.0 (0.00, 0.47)	0.2 (0.03, 0.83)	0.0 (0.00, 3.40)
Clinically important hepatic disorder AEs	0.0 (0.00, 3.40)	0.5 (0.10, 1.36)	0.7 (0.25, 1.50)

*Up to dose adjustment. †Participants who were randomised to receive GUS 200 mg SC q4w maintenance dosing, or had a dose adjustment (including "sham") to GUS 200 mg SC q4w dosing from all treatment groups (including those randomised to placebo and ustekinumab); for participants who had a dose adjustment, only data after their first GUS 200 mg dose are included. ‡Participants treated with placebo during the LTE were discontinued after study unblinding. §Infections are based on MedDRA system organ class "Infections and Infestations".
Note: Adverse events are coded using MedDRA Version 28.0. Occurrences of major adverse cardiovascular events were identified by clinical review. Terms for venous thromboembolism are based on customised MedDRA query. Clinically important hepatic disorders are defined as hepatic disorder AEs reported as SAEs or AEs leading to discontinuation of study intervention. The all-treated safety analysis set includes all participants who received ≥1 dose and differs from the primary efficacy analysis set (NRI) used for the baseline characteristics table.
AE, adverse event; CD, Crohn's disease; CI, confidence interval; GUS, guselkumab; IL, interleukin; LTE, long-term extension; MedDRA, Medical Dictionary for Regulatory Activities; MOA, mechanism of action; NRI, non-responder imputation; qXw, every X weeks; RDBAPC, randomised, double-blind, active- and placebo-controlled; SAE, serious adverse event; SC, subcutaneous.
Sands BE, et al. Presented at UEGW, Barcelona, Spain, 17–20 October 2026. OP105. Full prescription information: swissmedicinfo-pro.ch.

CH_CP-609070 10/26

Conclusions

- GUS-treated participants with moderately to severely active CD maintained clinical and endoscopic efficacy, through Week 144 with both maintenance regimens
- Efficacy was consistent in bio-naïve and BIO-IR participants, supporting durability irrespective of prior biologic exposure
- The 3-year safety profile of GUS was consistent with its known safety profile, with no new safety signals identified
- The treat-through study design enabled long-term efficacy and safety evaluation of all patients initially randomised to GUS induction, regardless of their response status after induction or upon entry into the long-term extension

BIO-IR, biologic inadequate response or intolerance; CD, Crohn's disease; GUS, guselkumab; IL, interleukin; MOA, mechanism of action; RDBAPC, randomised, double-blind, active- and placebo-controlled.
Sands BE, et al. Presented at UEGW, Barcelona, Spain, 17–20 October 2026. OP105. Full prescription information: swissmedicinfo-pro.ch.

CH_CP-609070 10//26

Deep remission in guselkumab-treated participants with moderately to severely active Crohn's disease through 3 years: Results from the GALAXI 2 & 3 studies

OP106

Oral presentation at UEGW

First presented as a poster at ACG 2026 (P3333)

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CH_CP-609070 10//26

Endpoints and analysis populations

Aim: Evaluate deep remission and other long-term outcomes through Week 144 in GUS-treated participants from the pooled GALAXI 2 and 3 studies

Endpoints and criteria

- **Deep remission:** Combination of clinical remission (CDAI score <150) AND endoscopic remission (SES-CD score ≤4 and ≥2-point reduction in SES-CD score and no subscore >1)
- **IBDQ remission:** Total IBDQ score ≥170
- **CD-related hospitalisations and surgeries**
- **Dose adjustment criteria:** Participants with an inadequate response (not in clinical response [≥100-point reduction in CDAI from Week 0 or CDAI <150] AND CDAI ≥220) were eligible to dose adjust once to GUS 200 mg SC between Week 52 and Week 80

Analysis populations

- **Primary efficacy analysis set (NRI):** All randomised participants who received ≥1 dose of guselkumab evaluated by randomised group
- **LTE as-observed analysis:** Participants who received ≥1 dose in the long-term extension, remained on continuous treatment through each designated time point, and had data available at that time point

Statistical considerations

- **Non-responder imputation:**
  - Participants with prohibited CD medication changes before Week 48, dose adjustment (including “sham”) during Weeks 52–80, or CD-related surgery or discontinuation of study intervention at any time were considered not to have met the endpoint at the designated time point
  - Missing endpoint data were imputed as non-response
- **As-observed analysis:** No nonresponder imputation; only participants with available data at the assessment time point were included in the analysis

CD, Crohn’s disease; CDAI, Crohn’s Disease Activity Index; GUS, guselkumab; IBDQ, Inflammatory Bowel Disease Questionnaire; IL, interleukin; LTE, long-term extension; MOA, mechanism of action; NRI, non-responder imputation; RDBAPC, randomised, double-blind, active- and placebo-controlled; SC, subcutaneous; SES-CD, Simple Endoscopic Score for Crohn’s Disease.
Rubin DT, et al. Presented at UEGW, Barcelona, Spain, 17–20 October 2026. OP106. Full prescription information: swissmedicinfo-pro.ch.

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Pooled demographics and disease characteristics in the LTE population

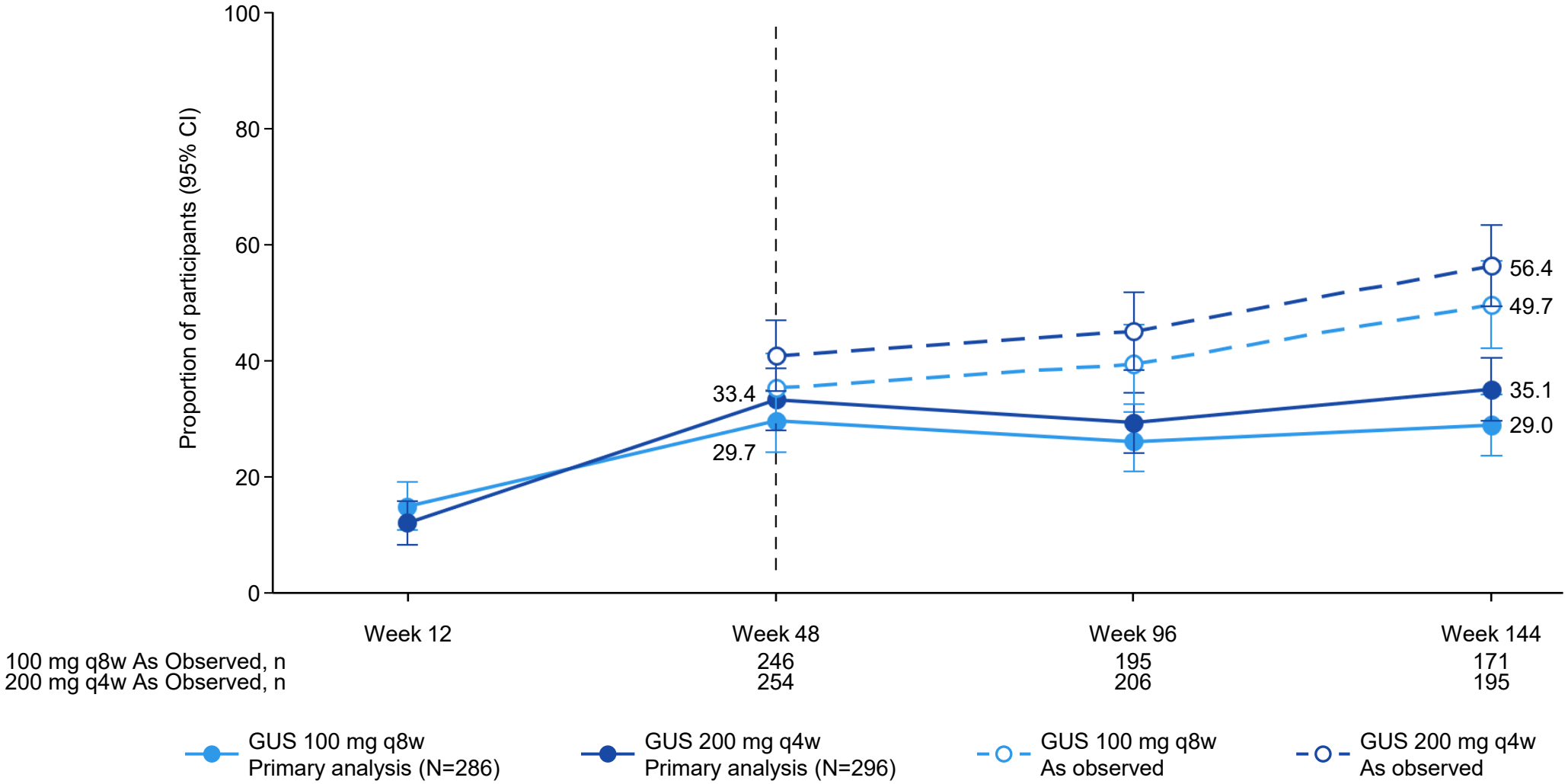
	GUS 100 mg SC q8w* (n=251)	GUS 200 mg SC q4w* (n=256)	GUS combined (n=507)
Primary analysis set, N	**251**	**256**	**507**
Demographics			
Age in years, mean (SD)	35.8 (12.17)	36.6 (13.14)	36.2 (12.66)
Men, n (%)	141 (56.2)	158 (61.7)	299 (59.0)
CD duration in years, mean (SD)	7.00 (6.44)	6.67 (6.60)	6.83 (6.52)
Characteristics, mean (SD)			
CDAI score	293.5 (53.47)	295.2 (53.28)	294.3 (53.33)
SES-CD	13.1 (7.54)	12.7 (7.38)	12.9 (7.46)
IBDQ score	126.8 (32.31)	128.6 (29.80)	127.7 (31.06)
Biomarkers, mean (SD)			
CRP, mg/L	16.0 (21.63)	15.8 (21.92)	15.9 (21.76)
Faecal calprotectin, µg/g	1614.3 (1982.97)	1745.8 (3134.80)	1680.8 (2627.83)
Involved GI areas (as assessed by central reader), n (%)			
Ileum only	49 (19.5)	66 (25.8)	115 (22.7)
Colon only	99 (39.4)	98 (38.3)	197 (38.9)
Ileum and colon	103 (41.0)	92 (35.9)	195 (38.5)

*Treatment group at the start of the LTE period.
CD, Crohn's disease; CDAI, Crohn's Disease Activity Index; CRP, C-reactive protein; GI, gastrointestinal; GUS, guselkumab; IBDQ, Inflammatory Bowel Disease Questionnaire; IL, interleukin; LTE, long-term extension; MOA, mechanism of action; qXw, every X weeks; RDBAPC, randomised, double-blind, active- and placebo-controlled; SC, subcutaneous; SD, standard deviation; SES-CD, Simple Endoscopic Score-Crohn's Disease.
Rubin DT, et al. Presented at UEGW, Barcelona, Spain, 17–20 October 2026. OP106. Full prescription information: swissmedicinfo-pro.ch.

CH_CP-609070 10//26

Deep remission through Week 144

Primary efficacy analysis set[1,2]



Deep remission: Combination of clinical remission (CDAI score <150) AND endoscopic remission (SES-CD score ≤4 and ≥2-point reduction in SES-CD score and no subscore >1).

CD, Crohn's disease; CDAI, Crohn's Disease Activity Index; CI, confidence interval; GUS, guselkumab; IL, interleukin; MOA, mechanism of action; qXw, every X weeks; RDBAPC, randomised, double-blind, active- and placebo-controlled; SES-CD, Simple Endoscopic Score-Crohn's Disease.

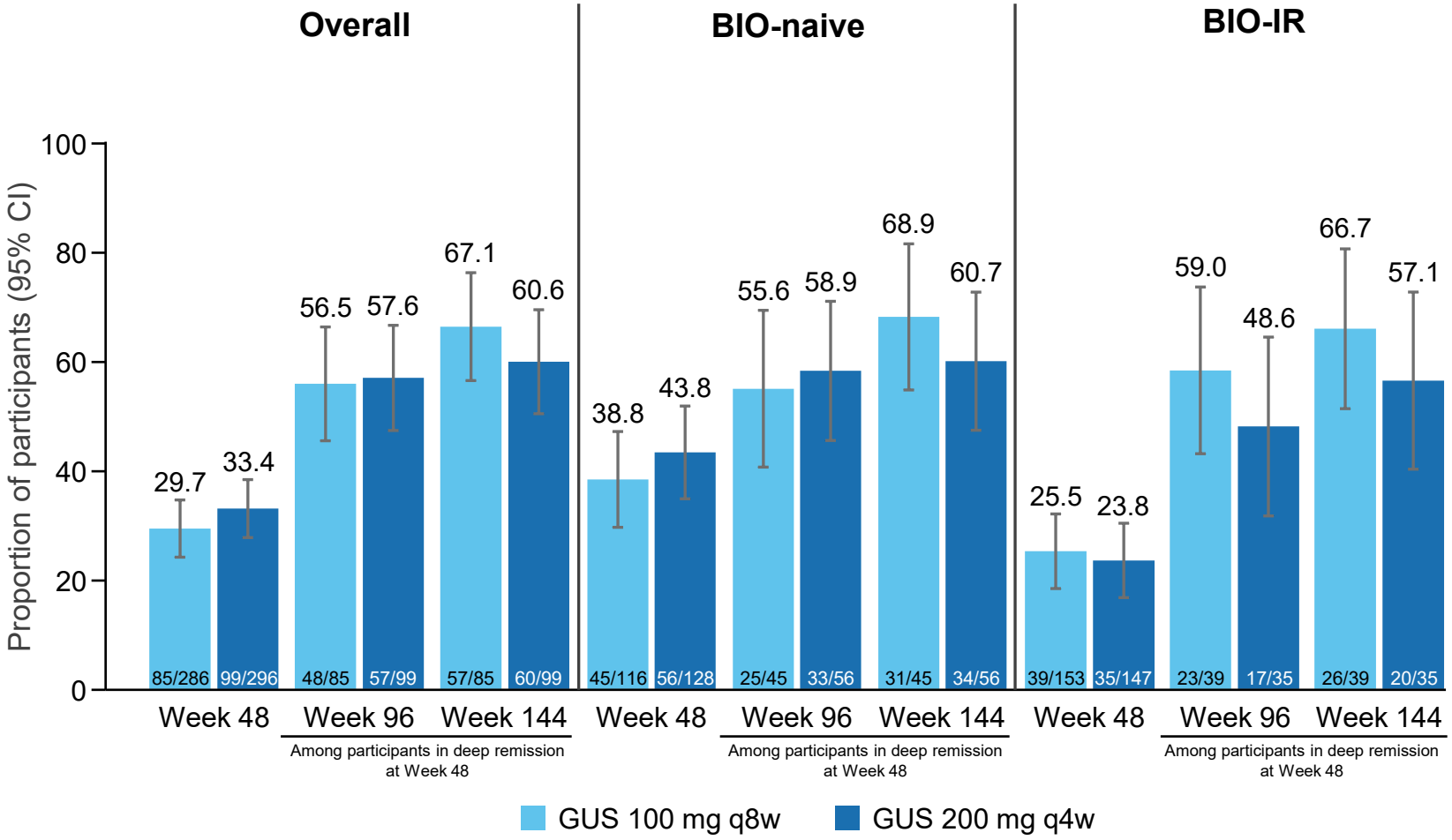
1. Rubin DT, et al. Presented at UEGW, Barcelona, Spain, 17–20 October 2026. OP106; 2. Rubin DT, et al. Presented at ACG, 9–14 October 2026; Nashville, TN, USA. P3333.

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CH_CP-609070 10//26

Deep remission through Week 144 among participants in deep remission at Week 48

Primary efficacy analysis set

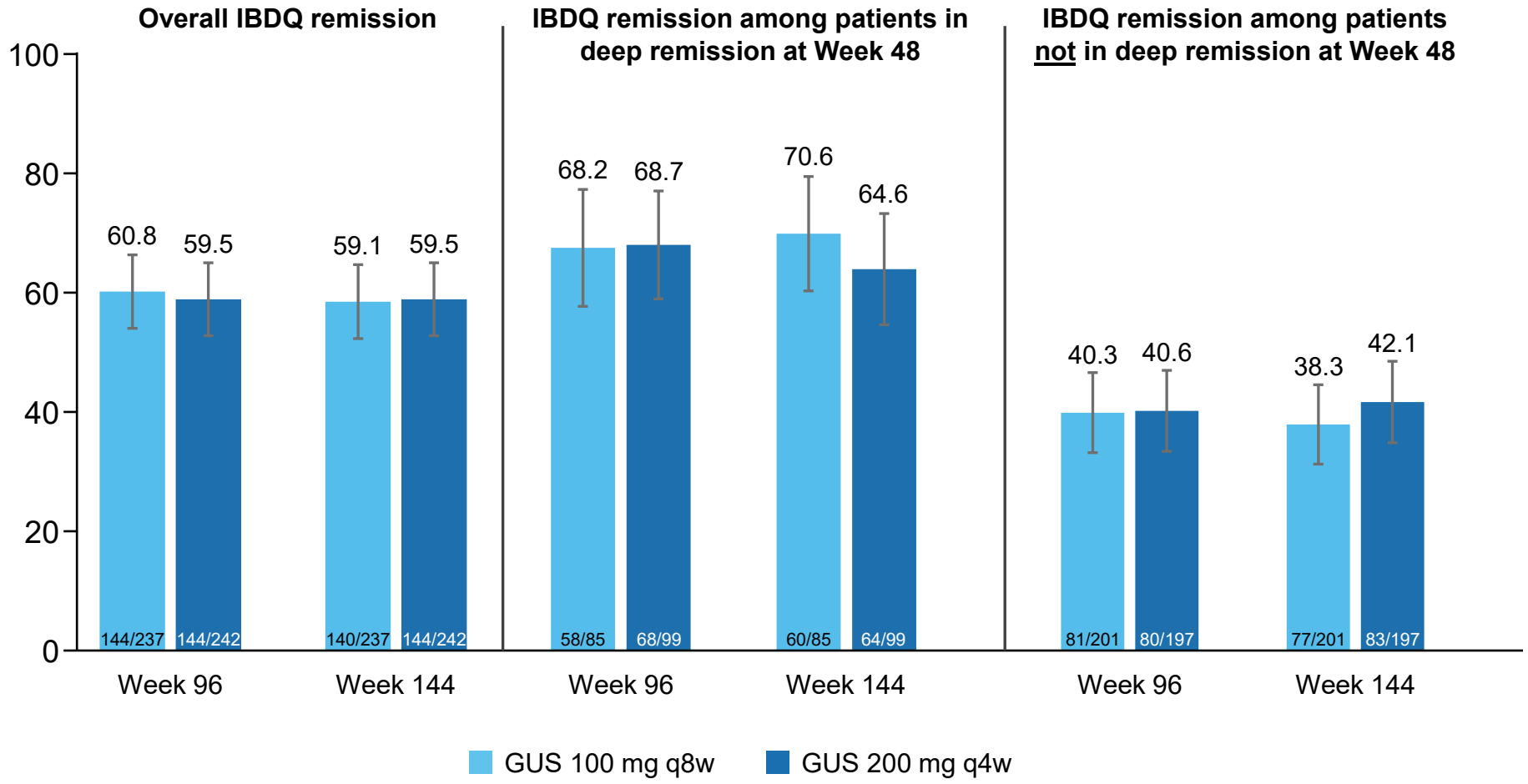


Deep remission: Combination of clinical remission (CDAI score <150) AND endoscopic remission (SES-CD score ≤4 and ≥2-point reduction in SES-CD score and no subscore >1).
BIO-IR, biologic inadequate response or intolerance; CD, Crohn's disease; CDAI, Crohn's Disease Activity Index; CI, confidence interval; GUS, guselkumab; IL, interleukin; MOA, mechanism of action; pts, participants; qXw, every X weeks; RDBAPC, randomised, double-blind, active- and placebo-controlled; SES-CD, Simple Endoscopic Score-Crohn's Disease; Wk, Week.
Rubin DT, et al. Presented at UEGW, Barcelona, Spain, 17–20 October 2026. OP106. Full prescription information: swissmedicinfo-pro.ch.

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IBDQ remission at Weeks 96 and 144 by deep remission status at Week 48

Primary efficacy analysis set

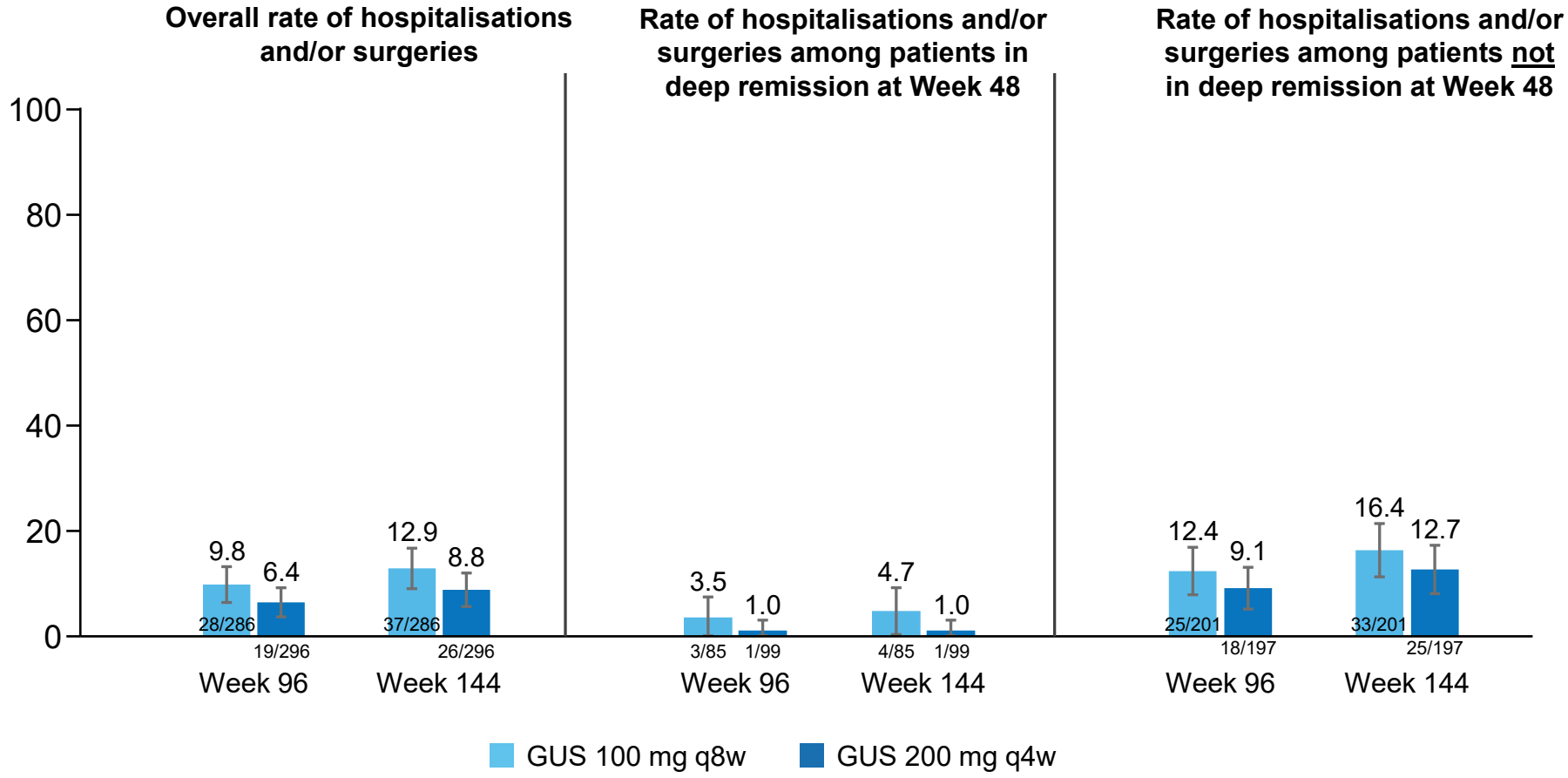


IBDQ remission: Total IBDQ score ≥170. **Deep remission:** Combination of clinical remission (CDAI score <150) AND endoscopic remission (SES-CD score ≤4 and ≥2-point reduction in SES-CD score and no subscore >1).
CD, Crohn's disease; CDAI, Crohn's Disease Activity Index; CI, confidence interval; GUS, guselkumab; IBDQ, Inflammatory Bowel Disease Questionnaire; IL, interleukin; MOA, mechanism of action; qXw, every X weeks; RDBAPC, randomised, double-blind, active- and placebo-controlled; SES-CD, Simple Endoscopic Score-Crohn's Disease.
Rubin DT, et al. Presented at UEGW, Barcelona, Spain, 17–20 October 2026. OP106. Full prescription information: swissmedicinfo-pro.ch.

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Fewer CD-related hospitalisations and/or surgeries were reported among participants who achieved deep remission at Week 48 versus those who did not

Primary efficacy analysis set

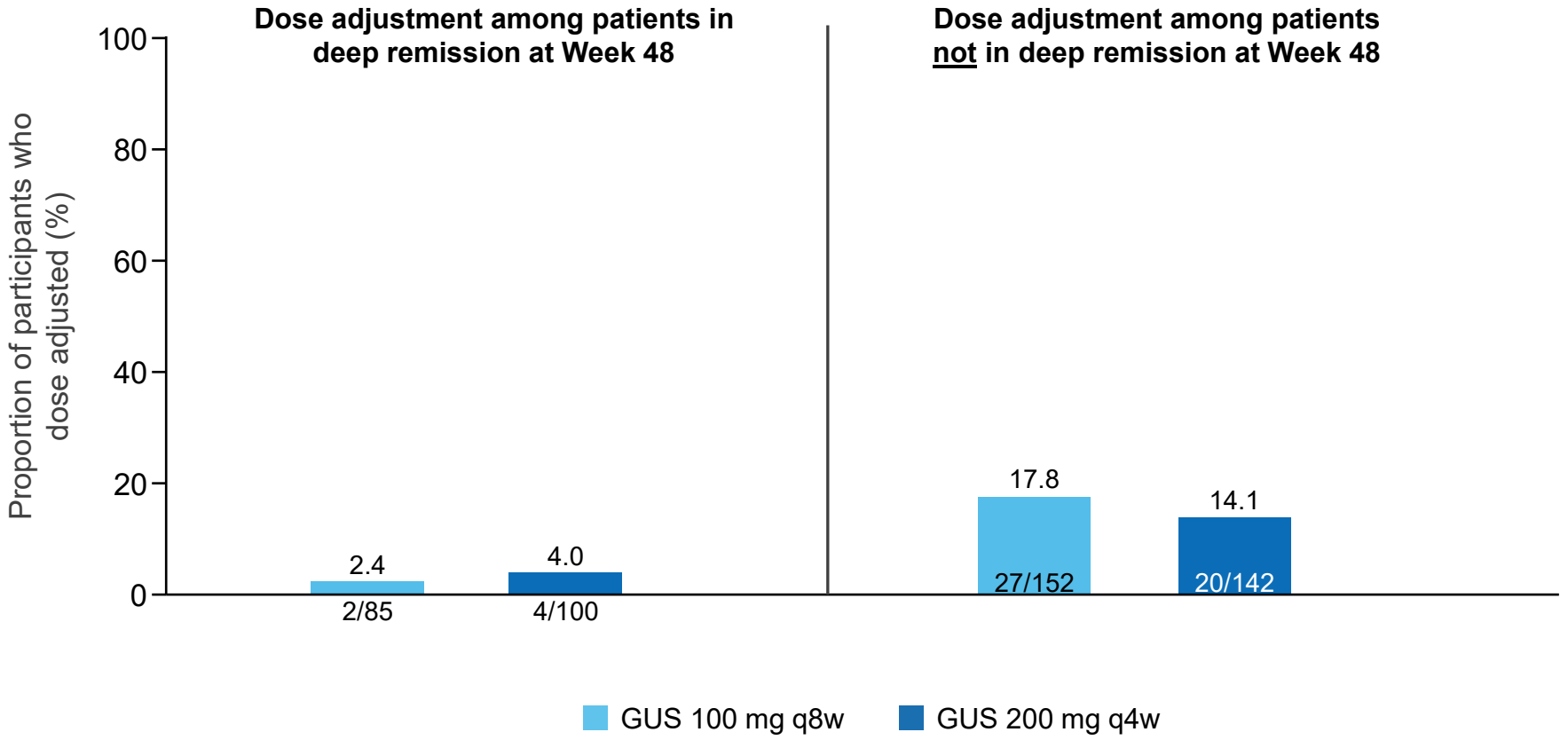


Deep remission: Combination of clinical remission (CDAI score <150) AND endoscopic remission (SES-CD score ≤4 and ≥2-point reduction in SES-CD score and no subscore >1).
CD, Crohn's disease; CDAI, Crohn's Disease Activity Index; CI, confidence interval; GUS, guselkumab; IL, interleukin; MOA, mechanism of action; qXw, every X weeks; RDBAPC, randomised, double-blind, active- and placebo-controlled; SES-CD, Simple Endoscopic Score-Crohn's Disease.
Rubin DT, et al. Presented at UEGW, Barcelona, Spain, 17–20 October 2026. OP106. Full prescription information: swissmedicinfo-pro.ch.

CH_CP-609070 10//26

Fewer participants in deep remission at Week 48 required dose adjustment between Weeks 52 and 80 versus those not in deep remission

Primary efficacy analysis set



Dose-adjustment criteria: Not in clinical response (≥100-point reduction in CDAI from Week 0 or CDAI <150) and CDAI ≥220. **Deep remission:** Combination of clinical remission (CDAI score <150) AND endoscopic remission (SES-CD score ≤4 and ≥2-point reduction in SES-CD score and no subscore >1).

CD, Crohn's disease; CDAI, Crohn's Disease Activity Index; GUS, guselkumab; IL, interleukin; MOA, mechanism of action; qXw, every X weeks; RDBAPC, randomised, double-blind, active- and placebo-controlled; SES-CD, Simple Endoscopic Score-Crohn's Disease.

Rubin DT, et al. Presented at UEGW, Barcelona, Spain, 17–20 October 2026. OP106. Full prescription information: swissmedicinfo-pro.ch.

CH_CP-609070 10//26

Conclusions

- GUS was effective in maintaining the high-bar endpoint of deep remission through 3 years in participants with moderately to severely active CD
- Deep remission was achieved regardless of prior biologic history
- Improved health-related quality of life and fewer hospitalisations and/or surgeries were reported through 3 years in GUS-treated participants in deep remission at Week 48

Deep remission: Combination of clinical remission (CDAI score <150) AND endoscopic remission (SES-CD score ≤4 and ≥2-point reduction in SES-CD score and no subscore >1).
CD, Crohn's disease; CDAI, Crohn's Disease Activity Index; GUS, guselkumab; IL, interleukin; MOA, mechanism of action; RDBAPC, randomised, double-blind, active- and placebo-controlled; SES-CD, Simple Endoscopic Score-Crohn's Disease.
Rubin DT, et al. Presented at UEGW, Barcelona, Spain, 17–20 October 2026. OP106. Full prescription information: swissmedicinfo-pro.ch.

CH_CP-609070 10//26

Guselkumab efficacy in subgroups of adults with a history of inadequate response or intolerance to anti-tumor necrosis factor-α therapy and moderately to severely active Crohn's disease: Results from the GALAXI 2 & 3 studies

MP0299

Poster presented at UEGW

Walter Reinisch,[1] Remo Panaccione,[2] Geert D'Haens,[3] Tadakazu Hisamatsu,[4] Bruce E Sands,[5] Rian Van Rampelbergh,[6] Wilbert van Duijnhoven,[6] Shashi Adsul,[7] Gustavo Scapini,[7] Silvio Danese,[8] Anita Afzali[9]

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CH_CP-609070 10//26

Outcomes and analysis

Aim: These post hoc analyses of pooled GALAXI 2 & 3 data evaluated efficacy outcomes at Week 12 and Week 48 in participants with CD who had an inadequate response or intolerance to anti-TNF therapies

Post hoc analysis cohort

- The analysis cohort was pooled participants from the Phase 3 GALAXI 2 & 3* trials who had moderately to severely active CD and IR to anti-TNF therapies

Anti-TNF-IR subgroups†	
1 anti-TNF-IR	Participants with only 1 prior anti-TNF-IR; may have failed other non-anti-TNF therapies‡
≥2 anti-TNF-IR	Participants with ≥2 prior anti-TNF-IR; may have failed other non-anti-TNF therapies‡
Anti-TNF-IR only	Participants who had previously failed only anti-TNF agents

Outcomes	Analyses
Clinical response§	Proportion of participants achieving clinical response, clinical remission, and endoscopic response at Week 12 with GUS induction vs PBO
Clinical remission	Same analysis as above
Endoscopic response	Proportion of participants achieving clinical remission, endoscopic response, and endoscopic remission at Week 48 with both GUS maintenance dose regimens and UST
Endoscopic remission¶	Same analysis as above

Participants with missing data after applying intercurrent event rules were considered as non-responders

Clinical response: ≥100-point decrease from Week 0 in CDAI or CDAI <150. **Clinical remission:** CDAI score <150. **Endoscopic response:** ≥50% improvement from Week 0 in SES-CD or SES-CD ≤2.
Endoscopic remission: SES-CD ≤4, ≥2-point reduction from Week 0 in SES-CD, and no subscore >1.
*NCT03466411; †Prior exposure to IL-12/IL-23 inhibitors was not allowed; ‡Including conventional (oral corticosteroid, immunomodulators) and biologics (vedolizumab); §Assessed only at Week 12; ¶Assessed only at Week 48.
CD, Crohn's disease; CDAI, Crohn's Disease Activity Index; GUS, guselkumab; IL, interleukin; IR, inadequate response or intolerance; MOA, mechanism of action; PBO, placebo; RDBAPC, randomised, double-blind, active- and placebo-controlled; SES-CD, Simple Endoscopic Score for Crohn's Disease; TNF, tumour necrosis factor; UST, ustekinumab.
Reinisch W, et al. Presented at UEGW, Barcelona, Spain, 17–20 October 2026. MP0299. Full prescription information: swissmedicinfo-pro.ch.

CH_CP-609070 10//26

Baseline participant and disease characteristics were generally balanced across treatment groups in the TNF-IR population

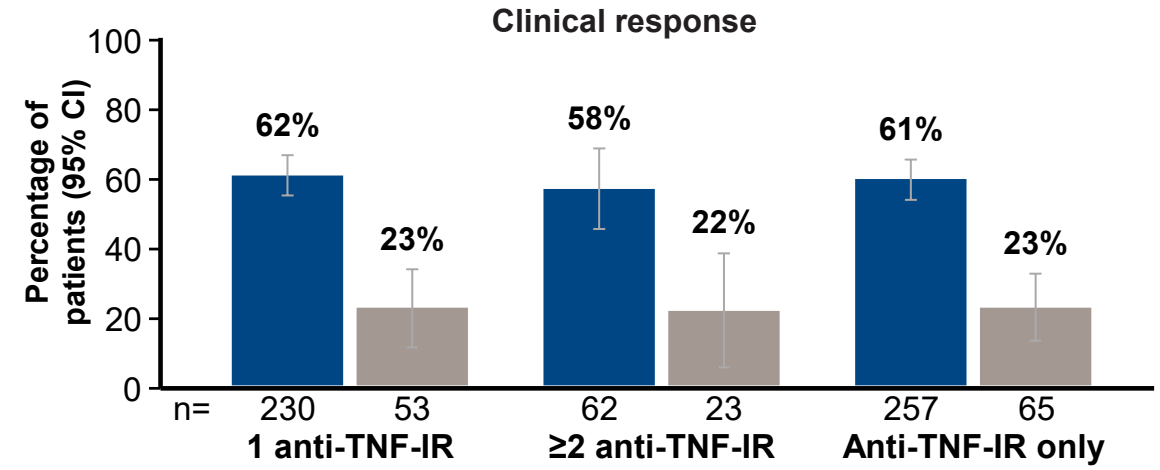
50% of the randomised participants in the GALAXI 2 and 3 studies were eligible for this post hoc analysis

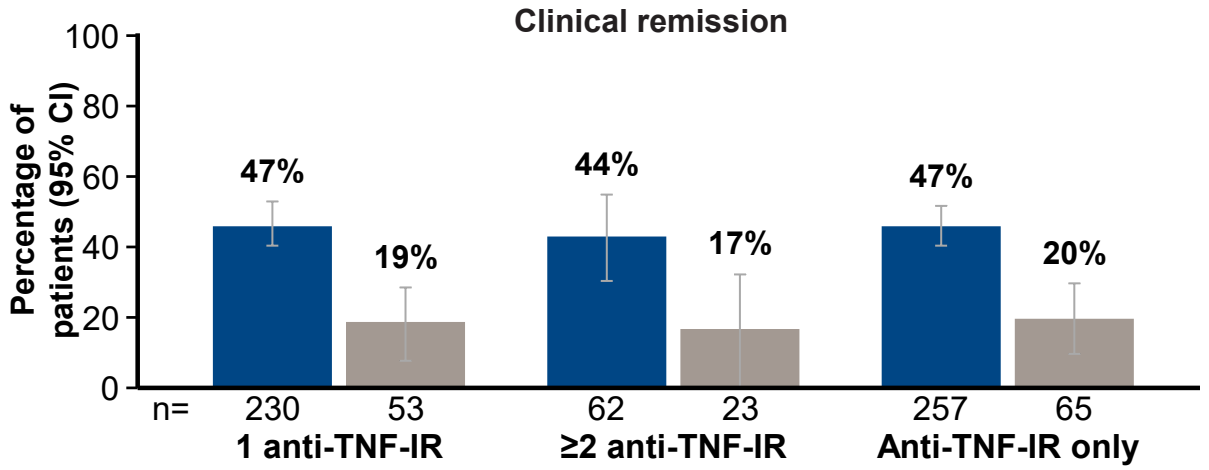
Baseline participant and disease characteristics	GUS 200 mg SC (n=143)	GUS 100 mg SC (n=149)	UST 90 mg SC (n=147)	PBO (n=76)
Demographics				
Age, years	37.0 (13.5)	35.3 (11.7)	38.9 (13.4)	35.7 (11.5)
Male	57%	56%	56%	63%
BMI, kg/m^2	23.3 (5.1)	23.2 (5.0)	23.5 (5.6)	22.7 (6.1)
Disease characteristics				
Disease duration, years	8.2 (7.4)	8.5 (6.8)	8.9 (8.0)	8.7 (8.7)
CDAI, 0–600	295.2 (53.4)	293.5 (52.8)	294.8 (51.1)	296.3 (55.7)
SES-CD, 0–56	13.2 (7.7)	14.1 (7.7)	13.3 (6.8)	14.8 (8.2)
CRP, mg/L	17.3 (22.5)	19.9 (24.7)	15.7 (19.6)	19.6 (26.4)
Involved GI areas*				
Ileum only	26%	17%	22%	18%
Colon only	22%	23%	19%	24%
Ileum and colon	52%	58%	56%	58%
Previous CD medication				
1 anti-TNF agent	78%	79%	69%	70%
≥2 anti-TNF agents	22%	21%	31%	30%
Anti-TNF agent only	90%	86%	85%	86%

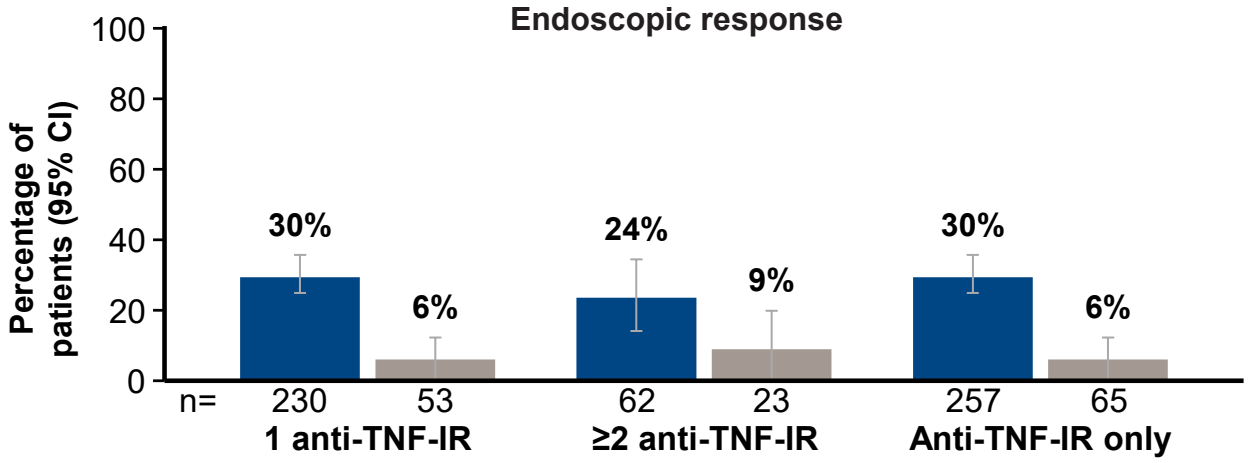
Data shown are mean (SD) unless otherwise noted. *Assessed by central reader.
BMI, body mass index; CD, Crohn's disease; CDAI, Crohn's Disease Activity Index; CRP, C-reactive protein; GI, gastrointestinal; GUS, guselkumab; IL, interleukin; IR, inadequate response or intolerance; MOA, mechanism of action; PBO, placebo; RDBAPC, randomised, double-blind, active- and placebo-controlled; SC, subcutaneous; SD, standard deviation; SES-CD, Simple Endoscopic Score for Crohn's Disease; TNF, tumour necrosis factor; UST, ustekinumab.
Reinisch W, et al. Presented at UEGW, Barcelona, Spain, 17–20 October 2026. MP0299. Full prescription information: swissmedicinfo-pro.ch.

CH_CP-609070 10//26

GUS induction achieved higher rates of clinical and endoscopic outcomes versus placebo at Week 12 across all anti-TNF-IR subgroups



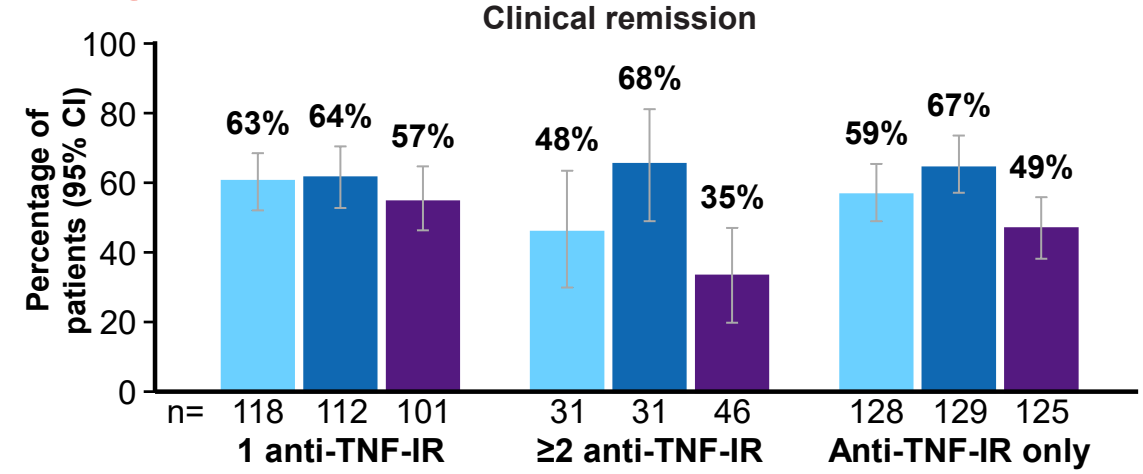


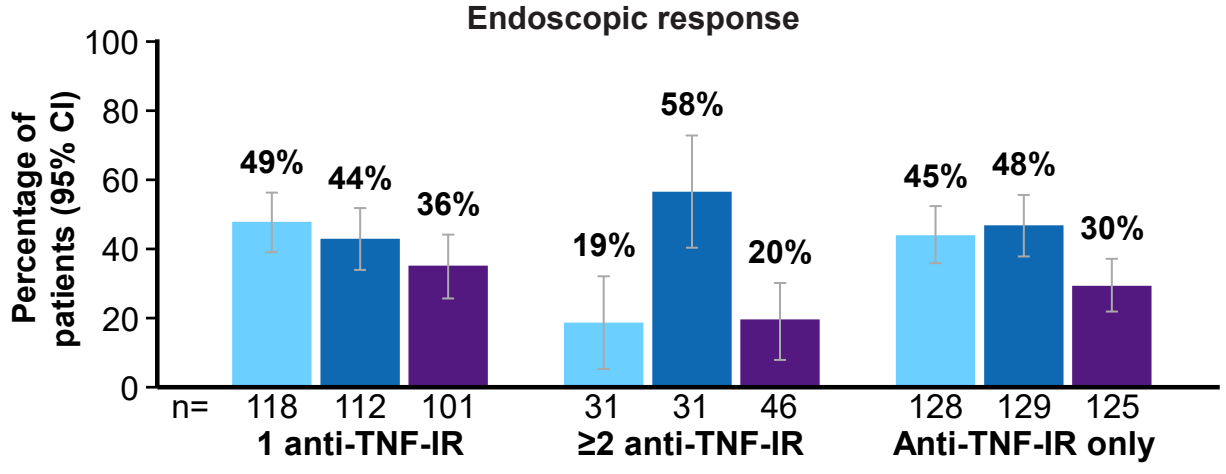


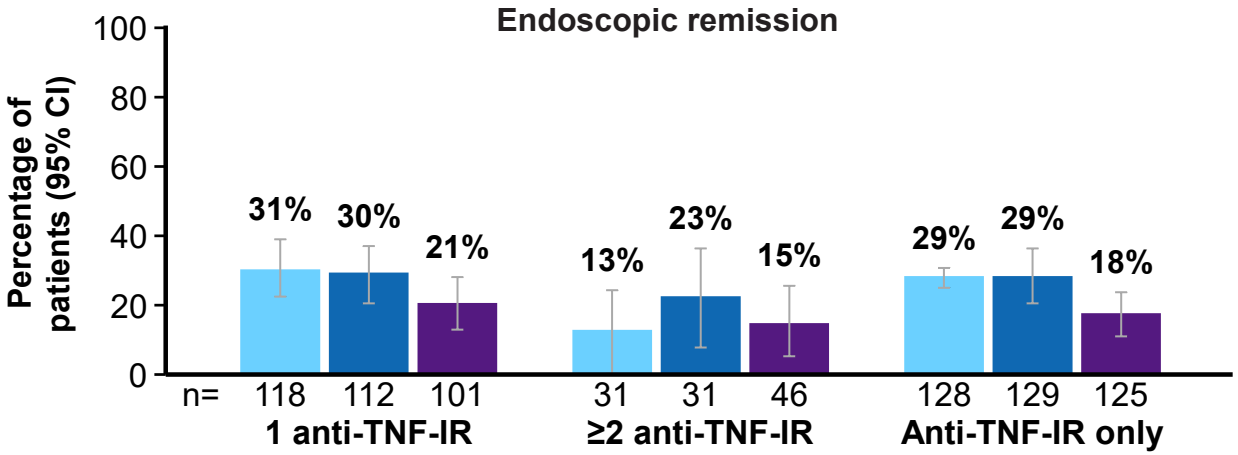
Clinical response: ≥100-point decrease from Week 0 in CDAI or CDAI <150. **Clinical remission:** CDAI score <150**. Endoscopic response:** ≥50% improvement from Week 0 in SES-CD or SES-CD ≤2.
*GUS 200 mg IV at Weeks 0/4/8.
CD, Crohn's disease; CDAI, Crohn's Disease Activity Index; CI, confidence interval; GUS, guselkumab; IL, interleukin; IR, inadequate response or intolerance; IV, intravenous; MOA, mechanism of action; PBO, placebo; RDBAPC, randomised, double-blind, active- and placebo-controlled; SES-CD, Simple Endoscopic Score for Crohn's Disease; TNF, tumour necrosis factor.
Reinisch W, et al. Presented at UEGW, Barcelona, Spain, 17–20 October 2026. MP0299. Full prescription information: swissmedicinfo-pro.ch.

CH_CP-609070 10//26

GUS maintenance generally achieved higher rates of clinical and endoscopic outcomes versus UST at Week 48 across all anti-TNF-IR subgroups







GUS 100mg SC q8w | GUS 200 mg SC q4w | UST 90 mg SC q8w

Clinical remission: CDAI score <150. **Endoscopic response:** ≥50% improvement from Week 0 in SES-CD or SES-CD ≤2. **Endoscopic remission:** SES-CD ≤4, ≥2-point reduction from Week 0 in SES-CD, and no subscore >1.

CD, Crohn's disease; CDAI, Crohn's Disease Activity Index; CI, confidence interval; GUS, guselkumab; IL, interleukin; IR, inadequate response or intolerance; MOA, mechanism of action; qXw, every X weeks; RDBAPC, randomised, double-blind, active- and placebo-controlled; SC, subcutaneous; SES-CD, Simple Endoscopic Score for Crohn's Disease; TNF, tumour necrosis factor; UST, ustekinumab.

Reinisch W, et al. Presented at UEGW, Barcelona, Spain, 17–20 October 2026. MP0299. Full prescription information: swissmedicinfo-pro.ch.

CH_CP-609070 10//26

Conclusions

- In the Phase 3 GALAXI 2 and 3 trials of participants with moderate to severe active CD with prior inadequate response or intolerance to anti-TNF therapies, GUS showed numerically greater clinical and endoscopic outcomes versus placebo at Week 12 and versus UST at Week 48

- Both GUS maintenance dosing regimens provided comparable efficacy at Week 48 in the one anti-TNF-IR and anti-TNF-IR only subgroups
  - In participants with ≥2 prior anti-TNF-IR, GUS 200 mg SC q4w appeared more efficacious than GUS 100 mg SC q8w and UST

- These findings suggest that treatment with GUS SC 200 mg q4w may provide additional benefit over UST and GUS SC 100 mg q8w in participants with CD and extensive prior anti-TNF treatment failure (≥2 anti-TNF-IR)

CD, Crohn's disease; GUS, guselkumab; IL, interleukin; IR, inadequate response or intolerance; MOA, mechanism of action; qXw, every X weeks; RDBAPC, randomised, double-blind, active- and placebo-controlled; SC, subcutaneous; TNF, tumour necrosis factor; UST, ustekinumab.

Reinisch W, et al. Presented at UEGW, Barcelona, Spain, 17–20 October 2026. MP0299. Full prescription information: swissmedicinfo-pro.ch.

CH_CP-609070 10//26

Early improvement of inflammatory biomarkers predicts 1-year outcomes in adults with Crohn's disease treated with guselkumab: Pooled GALAXI 2 & GALAXI 3, Phase 3 trials analysis

MP0081

Poster presented at UEGW

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CH_CP-609070 10/26

Outcomes and analysis

Aim: Post hoc analyses of pooled GALAXI 2 & 3 data evaluating the association between early serum and faecal biomarker response and 1-year clinical/endoscopic outcomes in participants with moderately to severely active CD treated with GUS

Post hoc analysis cohort

- Data were pooled from GUS-treated (200 mg SC q4w and 100 mg SC q8w) participants in the Phase 3 GALAXI 2 & 3 trials with abnormal CRP (>3 mg/L) or FC (>250 µg/g) at Week 0

Early biomarker response at Week 4, Week 8 and Week 12	
CRP response	CRP ≤3 mg/L or ≥50% reduction from Week 0
FC response	FC ≤250 µg/g or ≥50% reduction from Week 0
Clinical and endoscopic outcomes at Week 48	
Clinical remission	CDAI score <150
Endoscopic remission	SES-CD ≤4, ≥2-point reduction from Week 0 in SES-CD, and no SES-CD subscore >1
Deep remission	Clinical and endoscopic remission

For biomarker response, patients with missing data at Week 4, Week 8 and Week 12 were excluded from analysis at the corresponding time point

- For clinical outcomes, participants with missing data after applying intercurrent event rules were considered non-responders
- Associations between early biomarker response and Week 48 clinical and endoscopic outcomes were assessed using:
  - **Unadjusted models:** Chi-squared tests
  - **Adjusted models:** Multivariate logistic regression adjusted for age, sex, GUS maintenance regimen and prior biologic treatment failure status

CD, Crohn's disease; CDAI, Crohn's Disease Activity Index; CRP, C-reactive protein; FC, faecal calprotectin; GUS, guselkumab; IL, interleukin; MOA, mechanism of action; RDBAPC, randomised, double-blind, active- and placebo-controlled; SES-CD, Simple Endoscopic Score for Crohn's Disease.

Ferrante M, et al. Presented at UEGW, Barcelona, Spain, 17–20 October 2026. MP0081. Full prescription information: swissmedicinfo-pro.ch.

CH_CP-609070 10//26

Baseline patient and disease characteristics

	GUS-treated (N=582)
Demographics	
Age, years	36.5 (12.8)
Male	57%
BMI, kg/m^2	23.2 (4.7)
Disease characteristics	
Disease duration	7.1 (7.0)
CDAI, 0–600	296.1 (53.5)
SES-CD, 0–56	12.8 (7.3)
CRP, mg/L	15.9 (21.6)
CRP abnormal (>3mg/L)	72%
FC, µg/g	1788.6 (2974.1)
FC abnormal (>250µg/g)	81%
≥1 failed biological therapy	53%
Involved GI areas*	
Ileum	23%
Colon	25%
Ileum and colon	51%

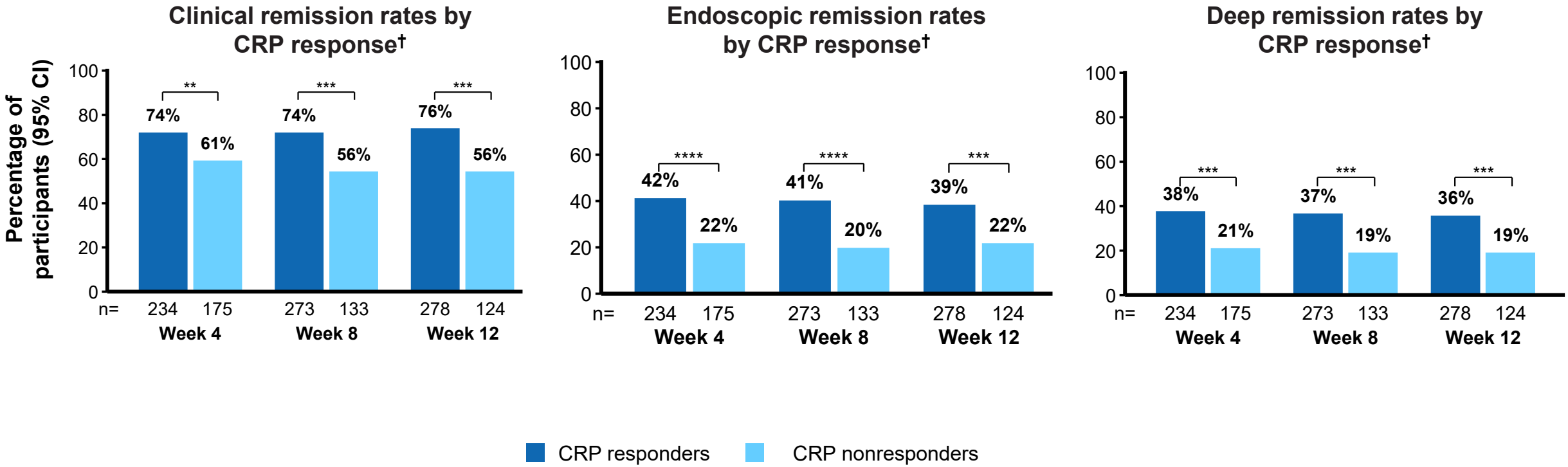
At baseline, 72% and 81% of participants had abnormal CRP and FC levels, respectively

Data shown are mean (SD) unless otherwise noted. *Assessed by central reader.
BMI, body mass index; CD, Crohn's disease; CDAI, Crohn's Disease Activity Index; CRP, C-reactive protein; FC, faecal calprotectin; GI, gastrointestinal; GUS, guselkumab; IL, interleukin; MOA, mechanism of action; RDBAPC, randomised, double-blind, active- and placebo-controlled; SD, standard deviation; SES-CD, Simple Endoscopic Score for Crohn's Disease.
Ferrante M, et al. Presented at UEGW, Barcelona, Spain, 17–20 October 2026. MP0081. Full prescription information: swissmedicinfo-pro.ch.

CH_CP-609070 10//26

CRP response with GUS was observed as early as Week 4 and was associated with higher Week 48 rates of clinical, endoscopic and deep remission

Unadjusted models

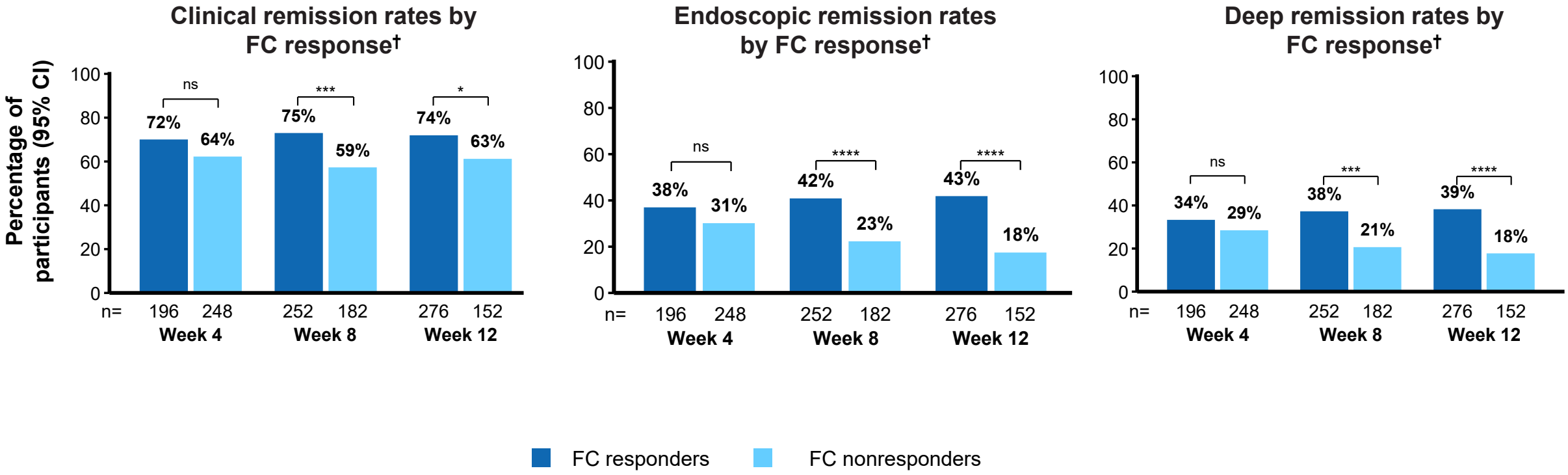


Clinical remission: CDAI score <150. **Endoscopic remission:** SES-CD ≤4, ≥2-point reduction from Week 0 in SES-CD, and no SES-CD subscore >1. **Deep remission:** clinical and endoscopic remission. **CRP response:** CRP ≤3 mg/L or ≥50% reduction from Week 0.
Nominal **p<0.01, ***p<0.001, ****p<0.0001 for CRP responders vs nonresponders. †Among participants with abnormal CRP (>3 mg/L) at Week 0.
CD, Crohn's disease; CDAI, Crohn's Disease Activity Index; CI, confidence interval; CRP, C-reactive protein; GUS, guselkumab; IL, interleukin; MOA, mechanism of action; RDBAPC, randomised, double-blind, active- and placebo-controlled; SES-CD, Simple Endoscopic Score for Crohn's Disease.
Ferrante M, et al. Presented at UEGW, Barcelona, Spain, 17–20 October 2026. MP0081. Full prescription information: swissmedicinfo-pro.ch.

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FC response with GUS was observed as early as Week 8 and was associated with higher Week 48 rates of clinical, endoscopic and deep remission

Unadjusted models



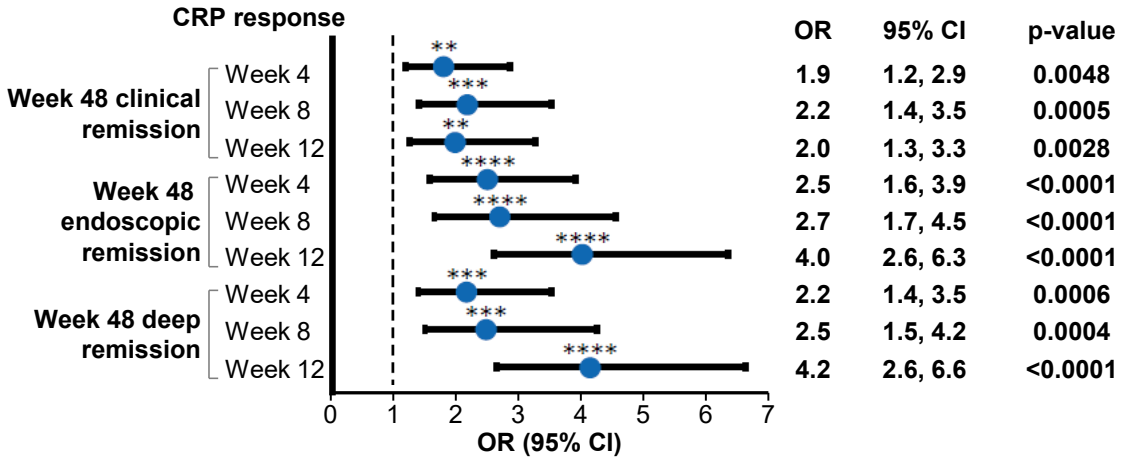
Clinical remission: CDAI score <150. **Endoscopic remission:** SES-CD ≤4, ≥2-point reduction from Week 0 in SES-CD, and no SES-CD subscore >1. **Deep remission:** clinical and endoscopic remission. **FC response:** FC ≤250 µg/g or ≥50% reduction from Week 0.
Nominal *p<0.05, ***p<0.001, ****p<0.0001 for FC responders vs nonresponders. †Among participants with abnormal FC (>250 µg/g) at Week 0.
CD, Crohn's disease; CDAI, Crohn's Disease Activity Index; CI, confidence interval; FC, faecal calprotectin; GUS, guselkumab; IL, interleukin; MOA, mechanism of action; ns, not significant; RDBAPC, randomised, double-blind, active- and placebo-controlled; SES-CD, Simple Endoscopic Score for Crohn's Disease.
Ferrante M, et al. Presented at UEGW, Barcelona, Spain, 17–20 October 2026. MP0081. Full prescription information: swissmedicinfo-pro.ch.

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CRP response from Week 4 onward and FC response from Week 8 onward was significantly associated with Week 48 outcomes in GUS-treated participants

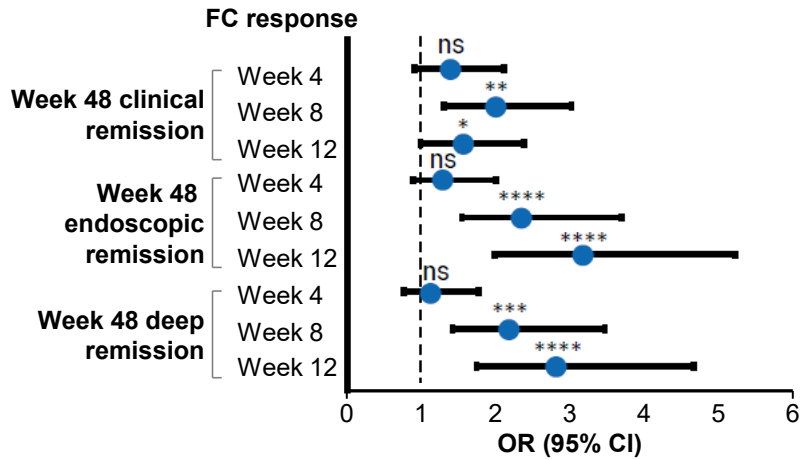
Adjusted models

Achievement of Week 48 clinical/endoscopic outcomes by early biomarker response†



	OR	95% CI	p-value
Week 48 clinical remission, Week 4	1.9	1.2, 2.9	0.0048
Week 48 clinical remission, Week 8	2.2	1.4, 3.5	0.0005
Week 48 clinical remission, Week 12	2.0	1.3, 3.3	0.0028
Week 48 endoscopic remission, Week 4	2.5	1.6, 3.9	<0.0001
Week 48 endoscopic remission, Week 8	2.7	1.7, 4.5	<0.0001
Week 48 endoscopic remission, Week 12	4.0	2.6, 6.3	<0.0001
Week 48 deep remission, Week 4	2.2	1.4, 3.5	0.0006
Week 48 deep remission, Week 8	2.5	1.5, 4.2	0.0004
Week 48 deep remission, Week 12	4.2	2.6, 6.6	<0.0001

Achievement of Week 48 clinical/endoscopic outcomes by early biomarker response‡



	OR	95% CI	p-value
Week 48 clinical remission, Week 4	1.4	0.9, 2.1	0.1124
Week 48 clinical remission, Week 8	2.0	1.3, 3.0	0.0012
Week 48 clinical remission, Week 12	1.6	1.0, 2.4	0.0474
Week 48 endoscopic remission, Week 4	1.3	0.9, 2.0	0.1451
Week 48 endoscopic remission, Week 8	2.4	1.6, 3.7	<0.0001
Week 48 endoscopic remission, Week 12	3.2	2.0, 5.2	<0.0001
Week 48 deep remission, Week 4	1.2	0.8, 1.8	0.419
Week 48 deep remission, Week 8	2.2	1.4, 3.5	0.0004
Week 48 deep remission, Week 12	2.9	1.8, 4.7	<0.0001

CRP response: CRP ≤3 mg/L or ≥50% reduction from Week 0. **FC response:** FC ≤250 µg/g or ≥50% reduction from Week 0.
Nominal *p<0.05, **p<0.01, ***p<0.001, ****p<0.0001 for CRP/FC responders vs nonresponders. †Among participants with abnormal CRP (>3 mg/L) at Week 0. ‡Among participants with abnormal FC (>250 µg/g) at Week 0.
CD, Crohn's disease; CI, confidence interval; CRP, C-reactive protein; FC, faecal calprotectin; GUS, guselkumab; IL, interleukin; MOA, mechanism of action; ns, not significant; OR, odds ratio; RDBAPC, randomised, double-blind, active- and placebo-controlled.
Ferrante M, et al. Presented at UEGW, Barcelona, Spain, 17–20 October 2026. MP0081. Full prescription information: swissmedicinfo-pro.ch.

CH_CP-609070 10//26

Conclusions

In the Phase 3 GALAXI 2 and 3 trials, participants with moderately to severely active CD and abnormal baseline biomarker levels were treated with GUS:[1]

- Early serum and faecal biomarker responses with GUS were associated with significantly higher rates of Week 48 clinical, endoscopic and deep remission
- These associations were observed as early as Week 4 for CRP and Week 8 for FC

Among participants without a CRP response during GUS induction, prior GALAXI 2 and 3 analyses showed greater Week 48 benefit with GUS 200 mg SC q4w vs 100 mg SC q8w maintenance[2]

Findings support the potential utility of early biomarker changes to inform longer-term outcomes and treatment optimisation in CD[1]

CD, Crohn's disease; CRP, C-reactive protein; FC, faecal calprotectin; GUS, guselkumab; IL, interleukin; MOA, mechanism of action; qXw, every X weeks; RDBAPC, randomised, double-blind, active- and placebo-controlled; SC, subcutaneous.

1. Ferrante M, et al. Presented at UEGW, Barcelona, Spain, 17–20 October 2026. MP0081; 2. Panaccione R. *Lancet* 2025;358–75 [supplementary appendix]. Full prescription information: swissmedicinfo-pro.ch.

CH_CP-609070 10//26

Endoscopic outcomes of ulcer size and ulcerated surface over one year across bowel segments in participants with Crohn's disease treated with guselkumab: Pooled GALAXI 2 and GALAXI 3, Phase 3 trials analysis

P5695

Poster presented at ACG 2026

Also presented as an encore at UEGW 2026 (MP0993)

Armuzzi A,[1,2] Fumery M,[3] Sands BE,[4] Ferrante M,[5] Raine T,[6] Van Rampelbergh R,[7] Nazar M,[8] McCaffrey V,[9] Van Denderen J,[10] Atreya R[11]

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CH_CP-609070 10/26

Study objective, outcomes and analysis[1]

Objective

Post hoc analyses of pooled GALAXI 2 & 3 data evaluating changes in ulcer presence/size and percentage of ulcerated surfaces across bowel segments through 1 year in GUS-treated participants with moderately to severely active CD

Post hoc analysis cohort

- Pooled from GUS-treated (200 mg SC q4w and 100 mg SC q8w) participants in the Phase 3 GALAXI 2 and GALAXI 3 trials with ≥1 ulcer in the ileum, colon and/or rectum at Week 0

- The SES-CD endoscopic scoring system (range, 0–56)[2] includes assessment of ulcer size and percent ulcerated surface (each scored 0–3) across five bowel segments, as assessed by central reader
- Changes over time were analysed as observed using the Wilcoxon signed-rank test
- All p-values are nominal

SES-CD assessments at Week 0, Week 12 and Week 48

Bowel segments	Variable of interest	Categories
Ileum Right colon Transverse colon Left colon Rectum	Presence/ulcer size	None Aphthous (0.1–0.5 cm) Large (0.5–2 cm) Very large (>2 cm)
	Percent ulcerated Surface	None <10% 10–30% >30%

Crohn's disease; GUS, guselkumab; IL, interleukin; MOA, mechanism of action; RDBPC, randomised, double-blind, placebo-controlled; SES-CD, Simple Endoscopic Score for Crohn's Disease.

1. Armuzzi A, et al. Presented at ACG, 9–14 October 2026; Nashville, TN, USA. PO5695; 2. Daperno M, et al. *Gastrointest Endosc* 2004;60:505–12. Full prescription information: swissmedicinfo-pro.ch.

CH_CP-609070 10//26

Baseline demographics and disease characteristics

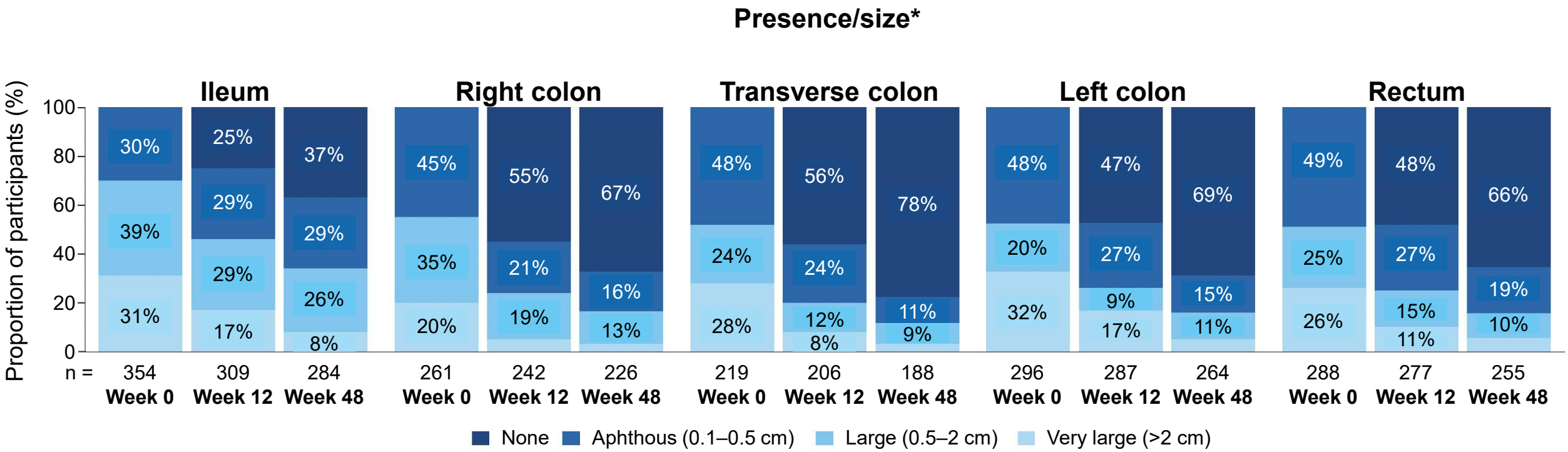
Baseline participant and disease characteristics	GUS-treated patients (N=582)
Demographics	
Age, years	36.5 (12.8)
Male	57%
BMI, kg/m^2	23.2 (4.7)
Disease characteristics	
Disease duration, years	7.1 (7.0)
CDAI score, 0–600	296.1 (53.5)
SES-CD, 0–56	12.8 (7.3)
Involved GI areas*	
Ileum only	23%
Colon only	25%
Ileum and colon	51%
CRP, mg/L	15.9 (21.6)
Faecal calprotectin, µg/g	1788.6 (2974.1)
≥1 failed biological therapy	53%
Ulcer location	
Ileum	74%
Colonic	39%
Rectal	50%

- At baseline, higher proportions of participants had ulcers in the ileum (74%) than in the rectal (50%) and colonic (39%) segments

Data are % for categorical variables and mean (SD) for continuous variables. *Assessed by central reader.
BMI, body mass index; CD, Crohn's disease; CDAI, Crohn's Disease Activity Index; CRP, C-reactive protein; GI, gastrointestinal; GUS, guselkumab; IL, interleukin; MoA, mechanism of action; RDBPC, randomised, double-blind, placebo-controlled; SD, standard deviation; SES-CD, Simple Endoscopic Score for Crohn's Disease. Armuzzi A, et al. Presented at ACG, 9–14 October 2026; Nashville, TN, USA. PO5695.
Full prescription information: swissmedicinfo-pro.ch.

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Presence/size of ulcer from Week 0 through Week 48

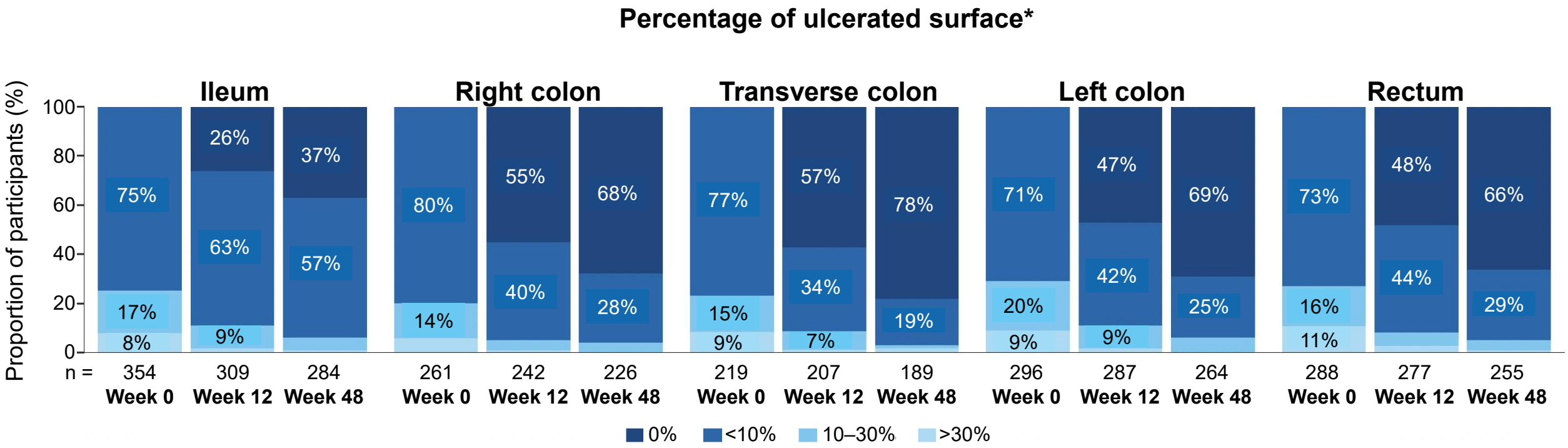


- GUS significantly reduced the ulcer size from Week 0 to Week 12 across all bowel segments, with sustained and enhanced reductions through Week 48
- Greater reductions in ulcer size from Week 0 were observed in colonic and rectal segments than in the ileum at Week 12 and Week 48

All nominal p<0.0001 for Week 12 vs Week 0, Week 48 vs Week 0, and Week 48 vs Week 12. Labels are not shown for the 'Very large' category at Week 12 (right colon) and Week 48 (right colon, transverse colon, left colon and rectum) due to space limitation.
*Among participants with ulcers within a given bowel segment at Week 0.
CD, Crohn's disease; GUS, guselkumab; IL, interleukin; MoA, mechanism of action; RDBPC, randomised, double-blind, placebo-controlled.
Armuzzi A, et al. Presented at ACG, 9–14 October 2026; Nashville, TN, USA. PO5695. Full prescription information: swissmedicinfo-pro.ch.

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Percentage of ulcerated surface from Week 0 through Week 48

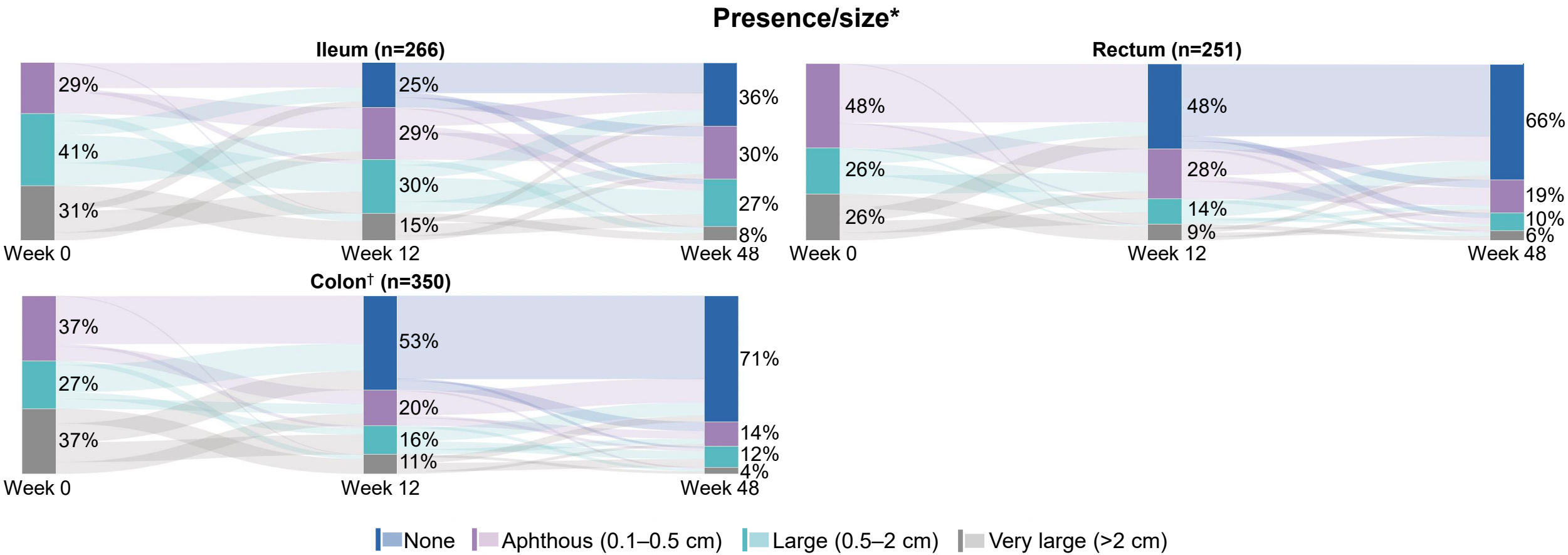


- GUS significantly reduced the percentage of ulcerated surface from Week 0 to Week 12, with sustained and enhanced reductions through Week 48
- Greater reductions in the percentage of ulcerated surface were observed in colonic and rectal segments than in the ileum at Weeks 12 and 48

All nominal p<0.0001 for Week 12 vs Week 0, Week 48 vs Week 0, and Week 48 vs Week 12. Labels are not shown for the 10–30% and/or >30% categories at Week 12 (right colon, transverse colon, left colon and rectum) and Week 48 (ileum, right colon, transverse colon, left colon, and rectum) due to space limitation.
*Among participants with ulcers within a given bowel segment at Week 0.
CD, Crohn's disease; GUS, guselkumab; IL, interleukin; MoA, mechanism of action; RDBPC, randomised, double-blind, placebo-controlled.
Armuzzi A, et al. Presented at ACG, 9–14 October 2026; Nashville, TN, USA. PO5695. Full prescription information: swissmedicinfo-pro.ch.

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Shifts in ulcer size across bowel segments through Week 48



- GUS shifted ulcer size towards resolution across bowel segments through Week 48

*Among participants with ulcers within a given bowel segment at Week 0 and assessment available at each timepoint. †Data from the three colonic segments (right colon, transverse colon, and left colon) were collapsed.
CD, Crohn's disease; GUS, guselkumab; IL, interleukin; MoA, mechanism of action; RDBPC, randomised, double-blind, placebo-controlled.
Armuzzi A, et al. Presented at ACG, 9–14 October 2026; Nashville, TN, USA. PO5695. Full prescription information: swissmedicinfo-pro.ch.

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Conclusions

Among GUS-treated adults with moderately to severely active CD in the Phase 3 GALAXI 2 & 3 trials:

- Baseline ulcers were more common in the ileum (74%) than in the rectum (50%) and colon (39%), and were large or very large in 70% of ileal and ~50% of colonic and rectal ulcers
- Complete ulcer resolution at Week 48 was achieved by 37%, 67–78% and 66% of participants with baseline ileal, colonic or rectal ulcers, respectively
- GUS markedly reduced ulcer size and ulcerated surface across all bowel segments as early as Week 12, with effects sustained through Week 48
    - Improvements in ulcer size were greater in colonic and rectal than ileal segments

These findings suggest early and durable endoscopic improvement with GUS across bowel segments in moderately to severely active CD

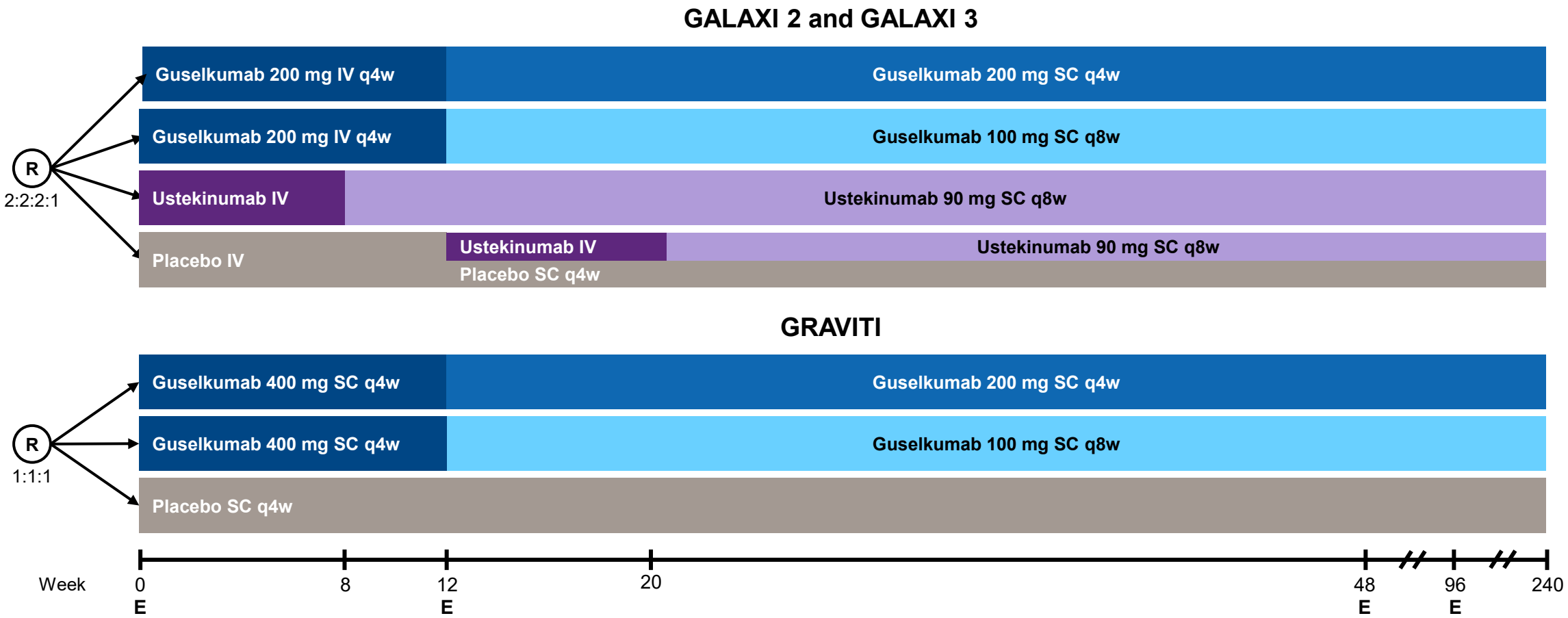
CD, Crohn's disease; GUS, guselkumab; IL, interleukin; MoA, mechanism of action; RDBPC, randomised, double-blind, placebo-controlled.
Armuzzi A, et al. Presented at ACG, 9–14 October 2026; Nashville, TN, USA. PO5695. Full prescription information: swissmedicinfo-pro.ch.

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GALAXI/GRAVITI: Guselkumab for luminal CD

Phase 3 GALAXI and GRAVITI study designs

- **Key eligibility criteria:** Moderately to severely active CD (CDAI score 220–450 and either mean daily SF or AP above protocol thresholds*), SES-CD score ≥6 (or ≥4 for isolated ileal disease), and inadequate response/intolerance to oral corticosteroids, 6-MP/AZA/MTX or biologic therapies†
- **Randomisation stratified by:** CDAI score (≤300 or >300), SES-CD (≤12 or >12), BIO-IR status and corticosteroid use at baseline (GALAXI only)



*__GALAXI:__ mean daily SF count >3 or AP score >1; __GRAVITI:__ mean daily SF count ≥4 or AP score ≥2. †Biologic therapies: TNF antagonists or vedolizumab.
Note: Entry into the LTE was determined based on investigator discretion.
6-MP, 6-mercaptopurine; AP, abdominal pain; AZA, azathioprine; BIO, biologic; CD, Crohn's disease; CDAI, Crohn's Disease Activity Index; E, endoscopy; GUS, guselkumab; IL, interleukin; IR, inadequate response or intolerance; IV, intravenous; LTE, long-term extension; MoA, mechanism of action; MTX, methotrexate; qXw, every X weeks; R, randomisation; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous; SES-CD, Simple Endoscopic Score for Crohn's Disease; SF, stool frequency; TNF, tumour necrosis factor.
Reinisch W, et al. Presented at ACG, 9–14 October 2026; Nashville, TN, USA. OP38. Full prescription information: swissmedicinfo-pro.ch.

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Changes in intestinal stenosis among patients with moderately to severely active Crohn's disease treated with guselkumab: Results from the Phase 3 GALAXI and GRAVITI studies

OP38

Oral presentation at ACG 2026

Also presented as an encore at UEGW 2026 (MP0083)

Walter Reinisch,[1] Anita Afzali,[2] Hamid Bassiri,[3] Rian Van Rampelbergh,[4] Wilbert van Duijnhoven,[4] Shashi Adsul,[5] Dylan J Richards,[3] Nat A Terry,[3] David T Rubin[6] on behalf of the GALAXI and GRAVITI Investigators

[1]Division of Gastroenterology & Hepatology, Medical University of Vienna, Vienna, Austria; [2]Division of Digestive Diseases, University of Cincinnati, College of Medicine Ohio, USA; [3]Johnson & Johnson, Spring House, PA, USA; [4]Johnson & Johnson, Antwerp, Belgium; [5]Johnson & Johnson, Horsham, PA, USA; [6]University of Chicago Medicine Inflammatory Bowel Disease Center, Chicago, IL, USA

CH_CP-609070 10/26

Analysis considerations

- Participants with symptomatic stenoses, or stenoses requiring surgical intervention, were excluded from enrolment
- Analyses were *post hoc*, as observed and descriptive; no formal statistical hypothesis testing was performed
- In GALAXI, participants meeting prespecified criteria could dose adjust from GUS 100 mg to 200 mg between Weeks 52–80; these participants were analysed within their original randomised arm
- Stenosis status was determined using the narrowing component of the SES-CD at Weeks 12, 48 and 96
- Participants with non-passable stenosis (SES-CD narrowing score of 3) at baseline were evaluated at each post-baseline endoscopic assessment
- Participants with missing SES-CD data at a given timepoint were excluded from the analysis at that timepoint because stenosis status could not be determined

SES-CD narrowing (stenosis) component

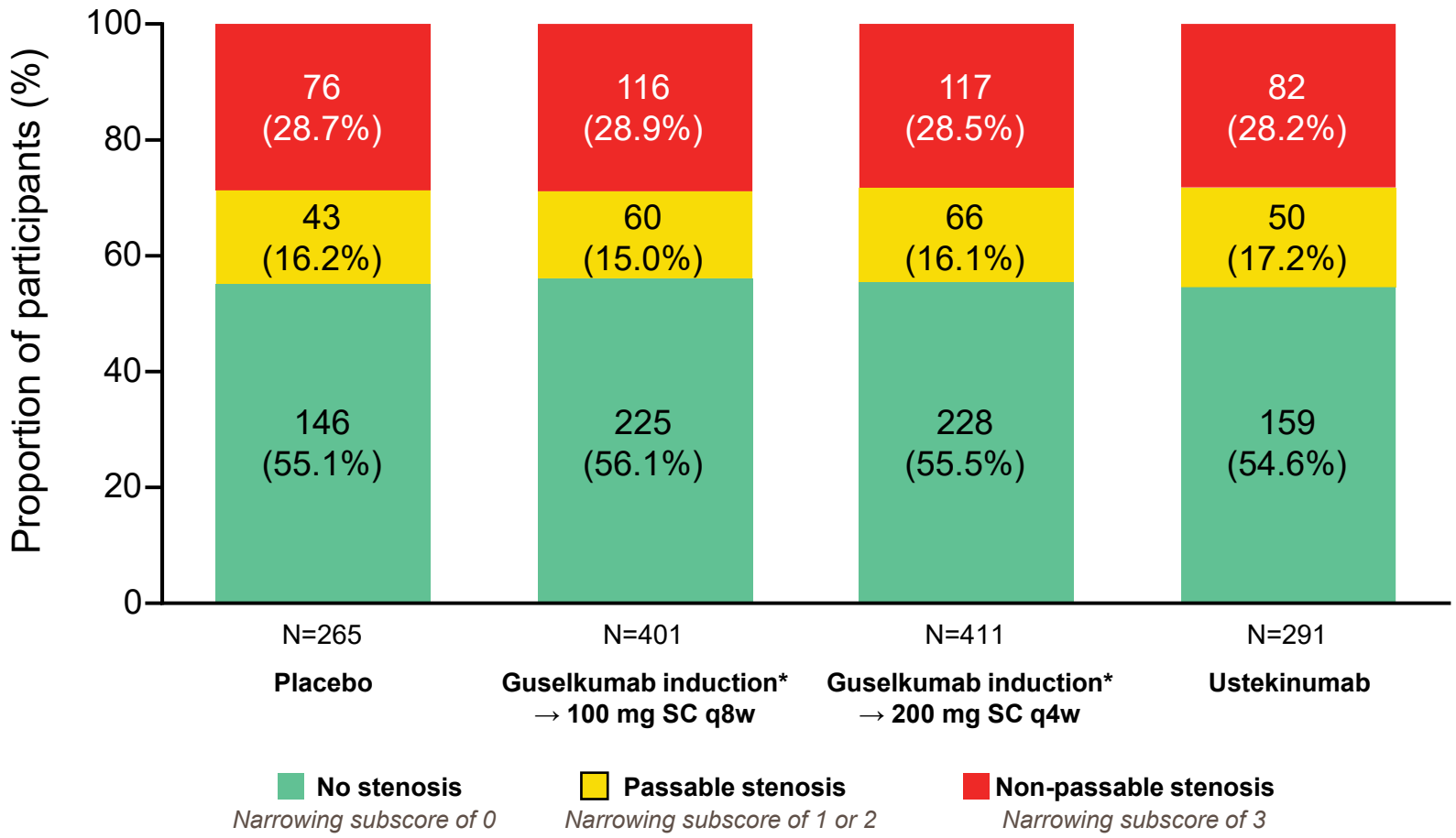
0	No narrowing
1	Single narrowing, can be passed
2	Multiple narrowings, can be passed
3	**Narrowing that cannot be passed (non-passable)**

Scored per ileocolonic segment; narrowing is 1 of 4 SES-CD components

CD, Crohn's disease; GUS, guselkumab; IL, interleukin; MoA, mechanism of action; RDBPC, randomised, double-blind, placebo-controlled; SES-CD, Simple Endoscopic Score for Crohn's Disease.
Reinisch W, et al. Presented at ACG, 9–14 October 2026; Nashville, TN, USA. OP38. Full prescription information: swissmedicinfo-pro.ch.

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Baseline distribution of stenoses across treatment arms



Percentages may not total 100% due to rounding.
*Guselkumab 200 mg IV or 400 mg SC at Weeks 0, 4 and 8.
CD, Crohn's disease; GUS, guselkumab; IL, interleukin; IV, intravenous; MoA, mechanism of action; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous.
Reinisch W, et al. Presented at ACG, 9–14 October 2026; Nashville, TN, USA. OP38. Full prescription information: swissmedicinfo-pro.ch.

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Baseline disease characteristics in participants with non-passable stenoses at study entry

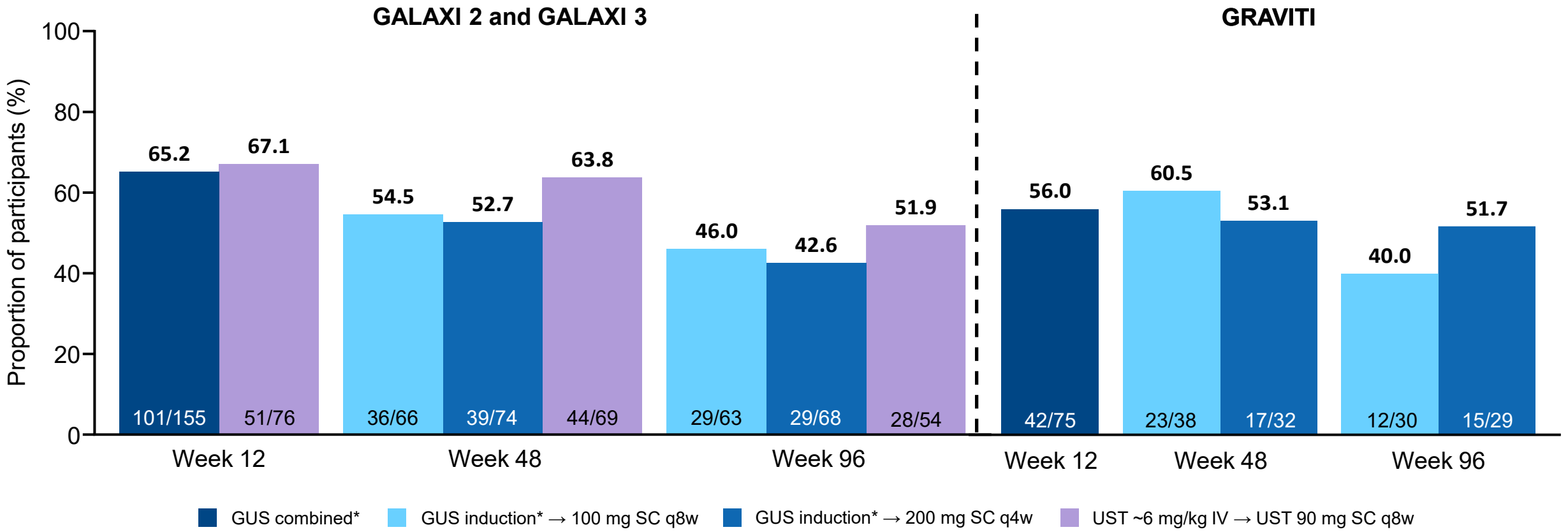
	GALAXI 2 and GALAXI 3			GRAVITI	
	Non-passable stenosis at baseline			Non-passable stenosis at baseline	
	GUS 100* (N=76)	GUS 200† (N=82)	UST‡ (N=82)	GUS 100§ (N=40)	GUS 200‖ (N=35)
CD duration in years, median (IQR)	4.88 (1.69–9.43)	5.02 (1.85–10.98)	6.05 (3.02–10.81)	3.90 (1.15–9.28)	5.80 (3.61–12.84)
CDAI score, median (IQR)	298.0 (253.5–342.0)	274.0 (251.0–336.0)	290.0 (261.0–331.0)	280.5 (250.0–320.0)	291.0 (264.0–351.0)
SES-CD score, median (IQR)	10.0 (7.0–15.0)	9.5 (7.0–15.0)	10.0 (7.0–16.0)	9.0 (7.0–12.5)	10.0 (6.0–16.0)
Ulcer component¶	5.0 (3.0–8.0)	4.0 (3.0–7.0)	5.0 (3.0–8.0)	4.0 (2.5–6.0)	5.0 (2.0–9.0)
Stricture component#	3.0 (3.0–3.0)	3.0 (3.0–3.0)	3.0 (3.0–3.0)	3.0 (3.0–3.0)	3.0 (3.0–3.0)
No history of intestinal resection surgery,** n (%)	63 (82.9)	61 (74.4)	58 (70.7)	27 (67.5)	29 (82.9)
Non-passable stenosis location by central reader,†† n (%)					
Ileum	31 (40.8)	36 (43.9)	36 (43.9)	18 (45.0)	16 (45.7)
Right colon	35 (46.1)	32 (39.0)	38 (46.3)	17 (42.5)	16 (45.7)
Transverse colon	3 (3.9)	7 (8.5)	5 (6.1)	1 (2.5)	3 (8.6)
Left colon	6 (7.9)	6 (7.3)	2 (2.4)	4 (10.0)	0
Rectum	1 (1.3)	1 (1.2)	1 (1.2)	0	0
Biologic medication history, n (%)					
Biologic-naïve	35 (46.1)	37 (45.1)	31 (37.8)	23 (57.5)	13 (37.1)
History of inadequate response/intolerance‡‡ to biologic therapy	32 (42.1)	39 (47.6)	49 (59.8)	14 (35.0)	19 (54.3)

*Participants randomised to guselkumab 200 mg IV q4w → 100 mg SC q8w. †Participants randomised to guselkumab 200 mg IV q4w → 200 mg SC q4w. ‡Participants randomised to ustekinumab ~6 mg/kg IV → 90 mg SC q8w. §Participants randomised to guselkumab 400 mg SC q4w → 100 mg SC q8w. ‖Participants randomised to guselkumab 400 mg SC q4w → 200 mg SC q4w. ¶Includes the presence and size of ulcers component and extent of ulcerated surface component. #Includes the presence and type of narrowings component across all evaluable segments. **Includes participants who did not have a 'total or sub-total colectomy' or a 'partial bowel resection' or 'other Crohn's disease–related surgery' prior to study entry. ††The first non-passable stenosis encountered (distal to proximal) is the one whose location is being reported. ‡‡Primary non-response, secondary non-response or intolerance.
CD, Crohn's disease; CDAI, Crohn's Disease Activity Index; GUS, guselkumab; IL, interleukin; IQR, interquartile range; IV, intravenous; MoA, mechanism of action; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous; SES-CD, Simple Endoscopic Score for Crohn's Disease; UST, ustekinumab.
Reinisch W, et al. Presented at ACG, 9–14 October 2026; Nashville, TN, USA. OP38. Full prescription information: swissmedicinfo-pro.ch.

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Persistence of non-passable stenosis through Week 96

Participants with non-passable stenosis at baseline

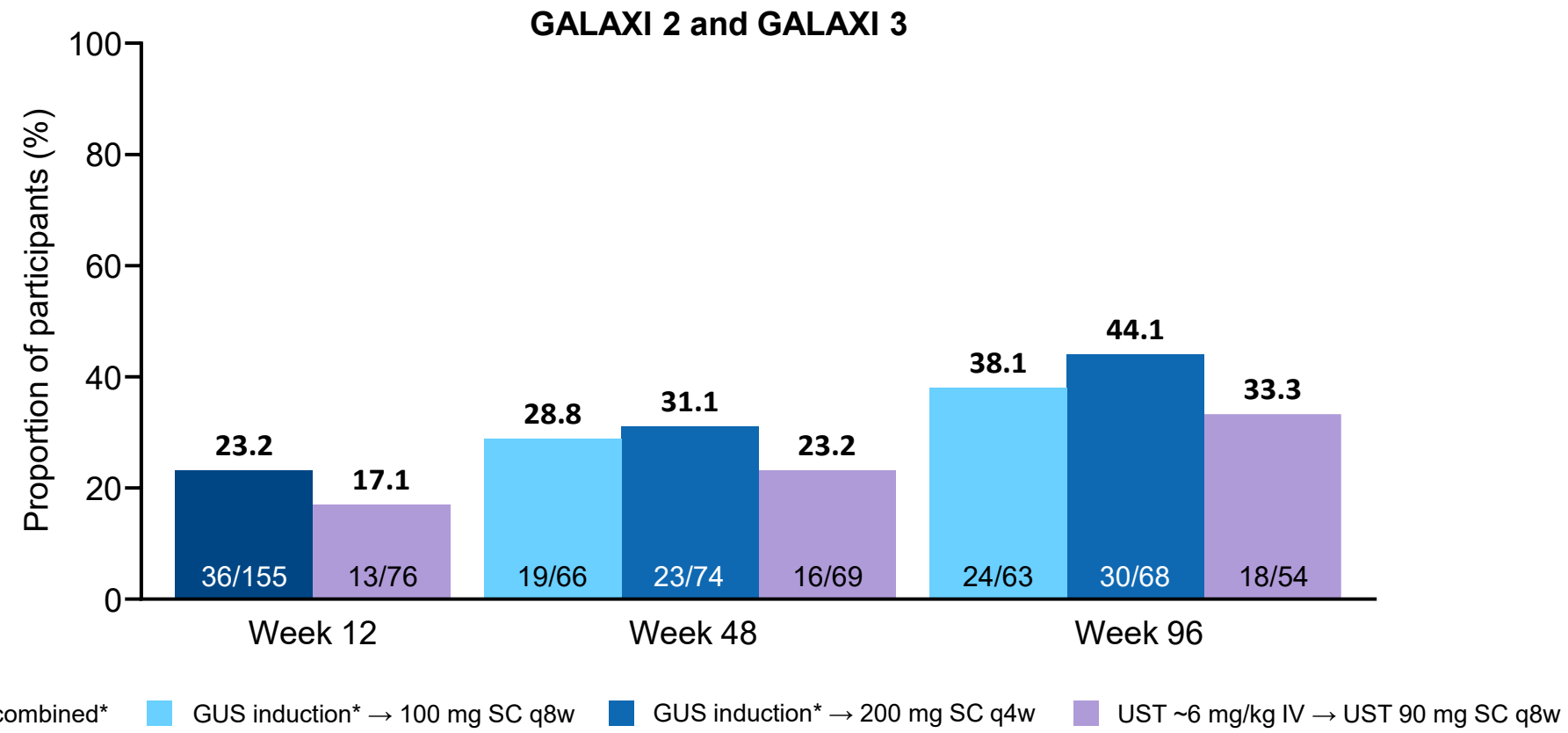


*Guselkumab 200 mg IV or 400 mg SC at Weeks 0, 4 and 8.
Note: Denominators are defined as participants with non-passable stenosis at baseline minus the missing SES-CD data at the respective timepoint. Missing SES-CD data are defined as participants who had a non-passable stenosis at baseline and did not have a dilation procedure or an intestinal resection surgery prior to the respective timepoint, and did not have any SES-CD data at the respective timepoint, so it was unknown whether they improved.
CD, Crohn's disease; GUS, guselkumab; IL, interleukin; IV, intravenous; MoA, mechanism of action; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous; SES-CD, Simple Endoscopic Score for Crohn's Disease; UST, ustekinumab.
Reinisch W, et al. Presented at ACG, 9–14 October 2026; Nashville, TN, USA. OP38. Full prescription information: swissmedicinfo-pro.ch.

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Resolution to no stenosis through Week 96

Participants with non-passable stenosis at baseline

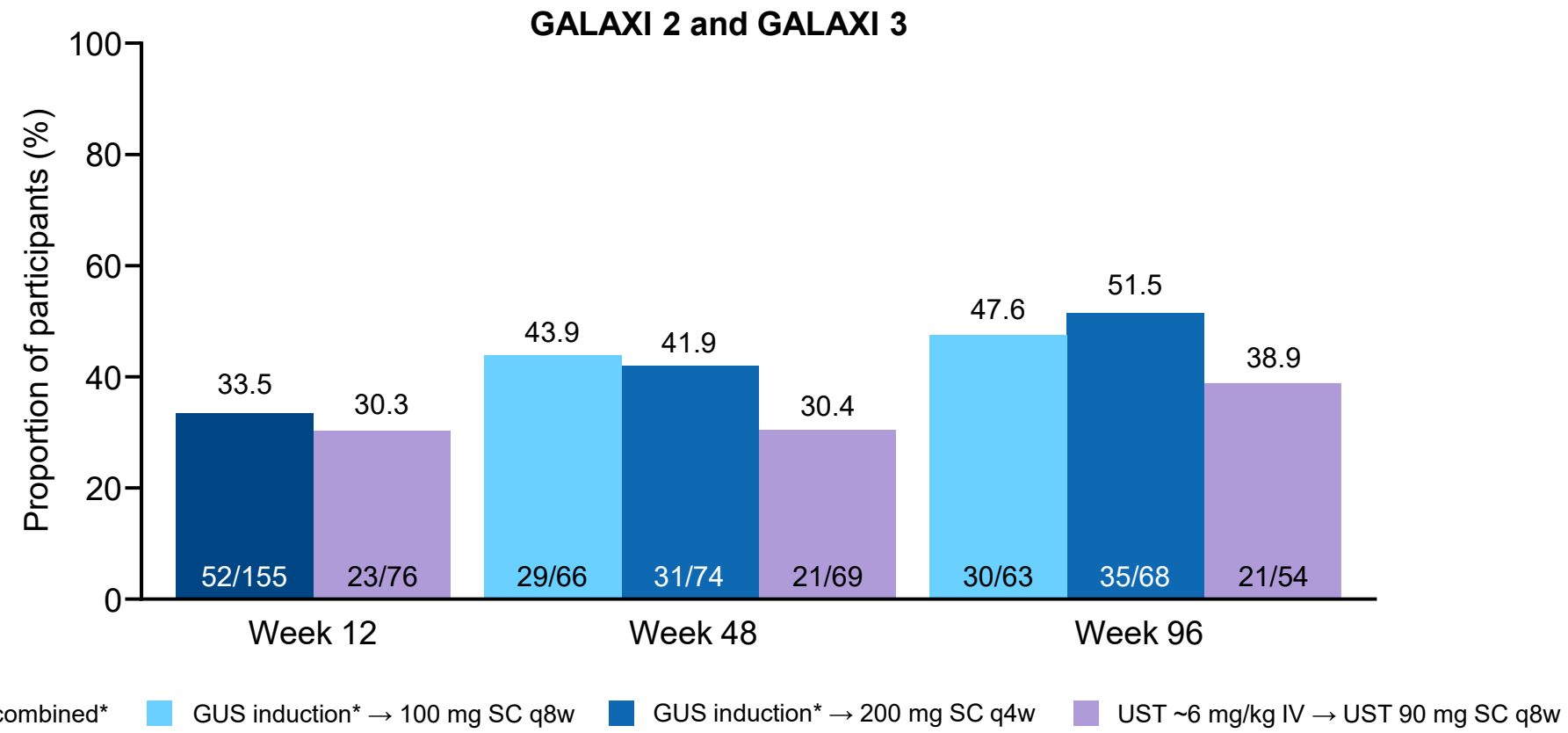


*Guselkumab 200 mg IV at Weeks 0, 4, and 8.
Note: Denominators are defined as participants with non-passable stenosis at baseline minus the missing SES-CD data at the respective timepoint. Missing SES-CD data are defined as participants who had a non-passable stenosis at baseline and did not have a dilation procedure or an intestinal resection surgery prior to the respective timepoint, and did not have any SES-CD data at the respective timepoint, so it was unknown whether they improved.
CD, Crohn's disease; GUS, guselkumab; IL, interleukin; IV, intravenous; MoA, mechanism of action; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous; SES-CD, Simple Endoscopic Score for Crohn's Disease; UST, ustekinumab.
Reinisch W, et al. Presented at ACG, 9–14 October 2026; Nashville, TN, USA. OP38. Full prescription information: swissmedicinfo-pro.ch.

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Improvement to passable or no stenosis through Week 96

Participants with non-passable stenosis at baseline



*Guselkumab 200 mg IV at Weeks 0, 4, and 8.
Note: Denominators are defined as participants with non-passable stenosis at baseline minus the missing SES-CD data at the respective timepoint. Missing SES-CD data are defined as participants who had a non-passable stenosis at baseline and did not have a dilation procedure or an intestinal resection surgery prior to the respective timepoint, and did not have any SES-CD data at the respective timepoint, so it was unknown whether they improved.
CD, Crohn's disease; GUS, guselkumab; IL, interleukin; IV, intravenous; MoA, mechanism of action; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous; SES-CD, Simple Endoscopic Score for Crohn's Disease; UST, ustekinumab.
Reinisch W, et al. Presented at ACG, 9–14 October 2026; Nashville, TN, USA. OP38. Full prescription information: swissmedicinfo-pro.ch.

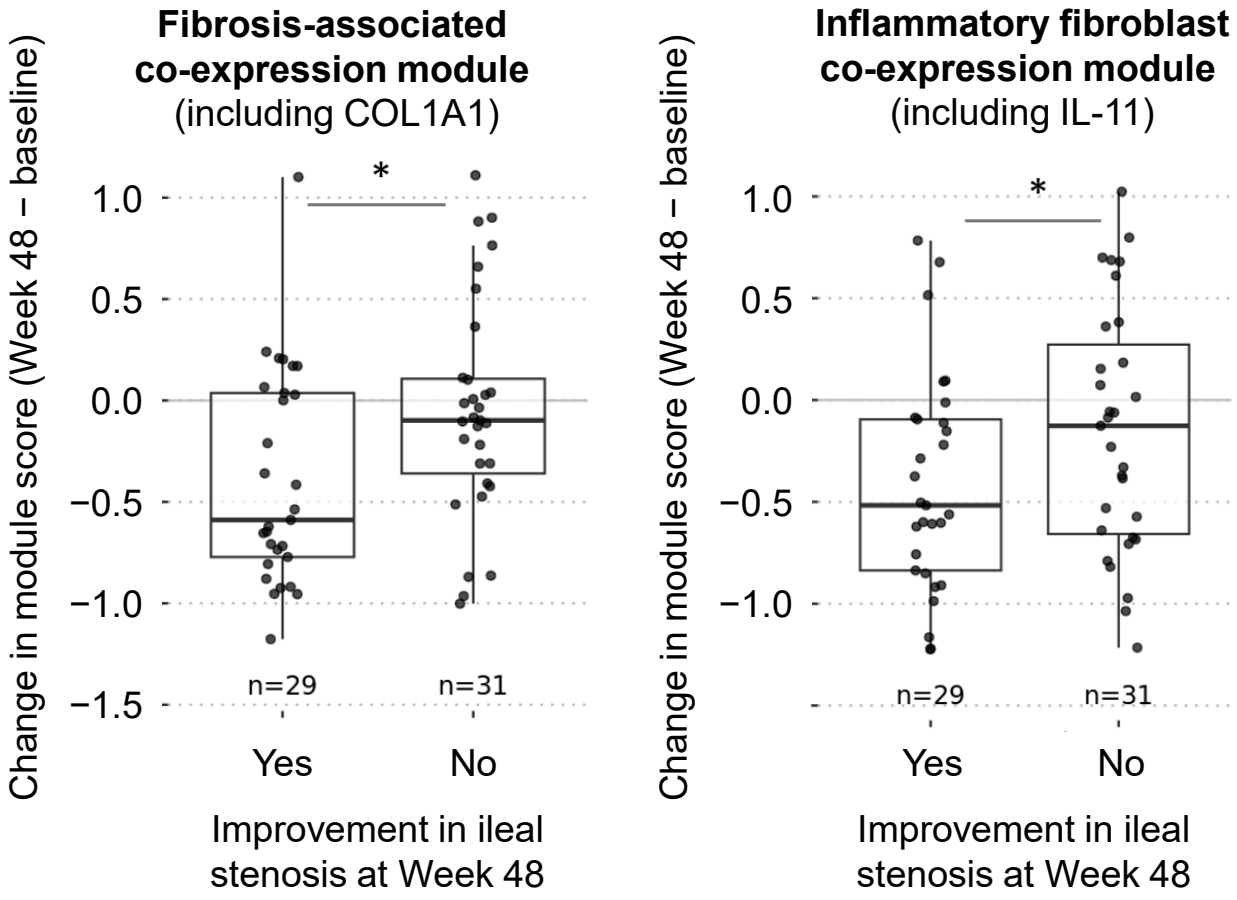
CH_CP-609070**10//26**

Inflammatory and fibrosis-related biology is reduced in participants with improved ileal stenosis

GALAXI data

Analysis approach

- Single cell-based gene correlation network analysis in IBD data defines fibroblast-derived gene co-expression modules for bulk disease interpretation[2]
- RNA sequencing of ileum biopsies in a subset of participants in GALAXI (total set) was performed at Weeks 0, 12 and 48 to evaluate changes in fibroblast-associated biology[1]
  - ~33% of samples were from participants with any ileal stenosis at baseline (stenotic subset)



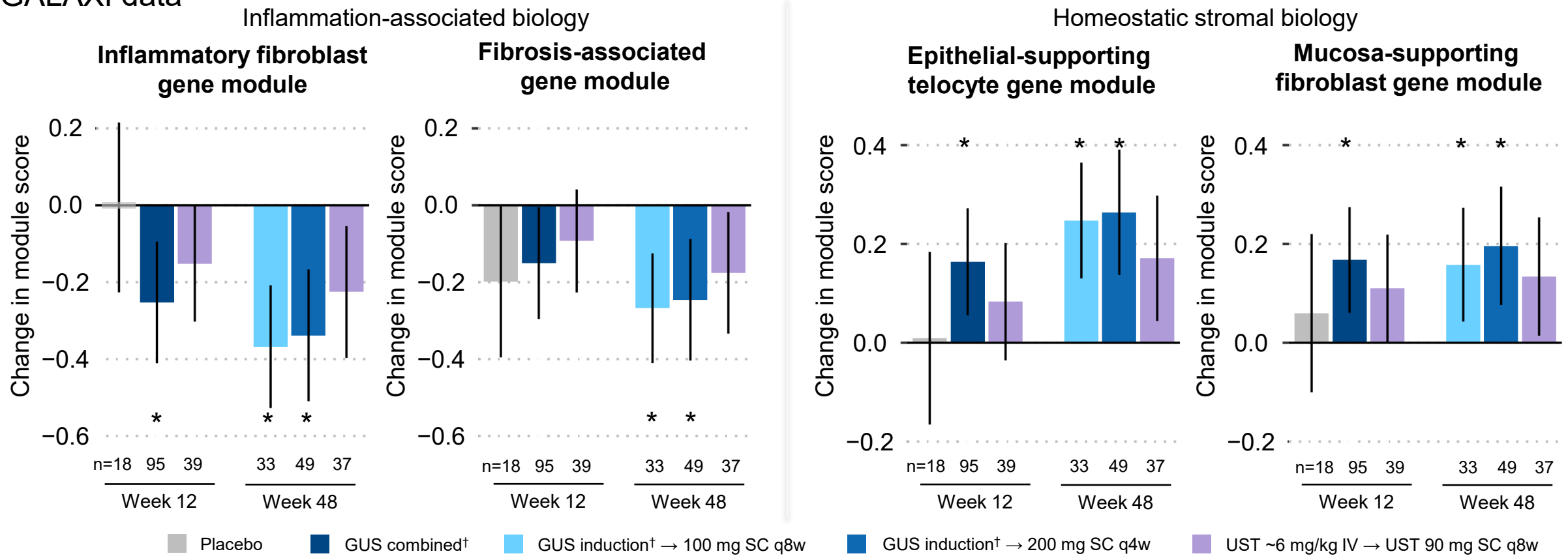
- Participants with improved ileal stenosis at Week 48 had greater reductions in inflammatory fibroblast and fibrosis-associated gene module expression than those without improvement[1]

*p<0.05, independent *t*-test; analysis in stenotic subset.
CD, Crohn's disease; COL1A1, collagen type I alpha 1 chain; GUS, guselkumab; IBD, inflammatory bowel disease; IL, interleukin; MoA, mechanism of action; RDBPC, randomised, double-blind, placebo-controlled.
1. Reinisch W, et al. Presented at ACG, 9–14 October 2026; Nashville, TN, USA. OP38; 2. Hart A, et al. *BMJ Open Gastroenterol* 2026;13:e002153. Full prescription information: swissmedicinfo-pro.ch.

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GUS reduced inflammatory fibroblast biology at Week 12 and fibrosis-associated biology by Week 48, with increases in homeostatic stromal biology, in the ileum

GALAXI data



*Adjusted p<0.05; analysis in total set from pooled GALAXI 2 and GALAXI 3. †GUS 200 mg IV at Weeks 0, 4 and 8. Linear mixed-effect model: fibroblast modules ~ treatment time contrasts with participant random effect.
CD, Crohn's disease; GUS, guselkumab; IL, interleukin; IV, intravenous; MoA, mechanism of action; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous; UST, ustekinumab.
Reinisch W, et al. Presented at ACG, 9–14 October 2026; Nashville, TN, USA. OP38. Full prescription information: swissmedicinfo-pro.ch.

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Conclusions

- Among participants with moderately to severely active CD and non-passable stenosis at baseline, improvement to passable or no stenosis was observed as early as Week 12 with GUS and was maintained through 2 years
- GUS induced greater and earlier improvement in disease-associated fibroblast gene module expression in the ileum, while UST showed limited changes
- These findings suggest that GUS may improve endoscopic intestinal stenosis while inducing early changes in stenosis-associated fibroblast gene module expression

CD, Crohn's disease; GUS, guselkumab; IL, interleukin; MoA, mechanism of action; RDBPC, randomised, double-blind, placebo-controlled; UST, ustekinumab.
Reinisch W, et al. Presented at ACG, 9–14 October 2026; Nashville, TN, USA. OP38. Full prescription information: swissmedicinfo-pro.ch.

CH_CP-609070 10//26

Pharmacokinetics and exposure-response relationships of subcutaneous guselkumab maintenance in participants with moderately to severely active Crohn's disease

P5692

Poster presented at ACG 2026

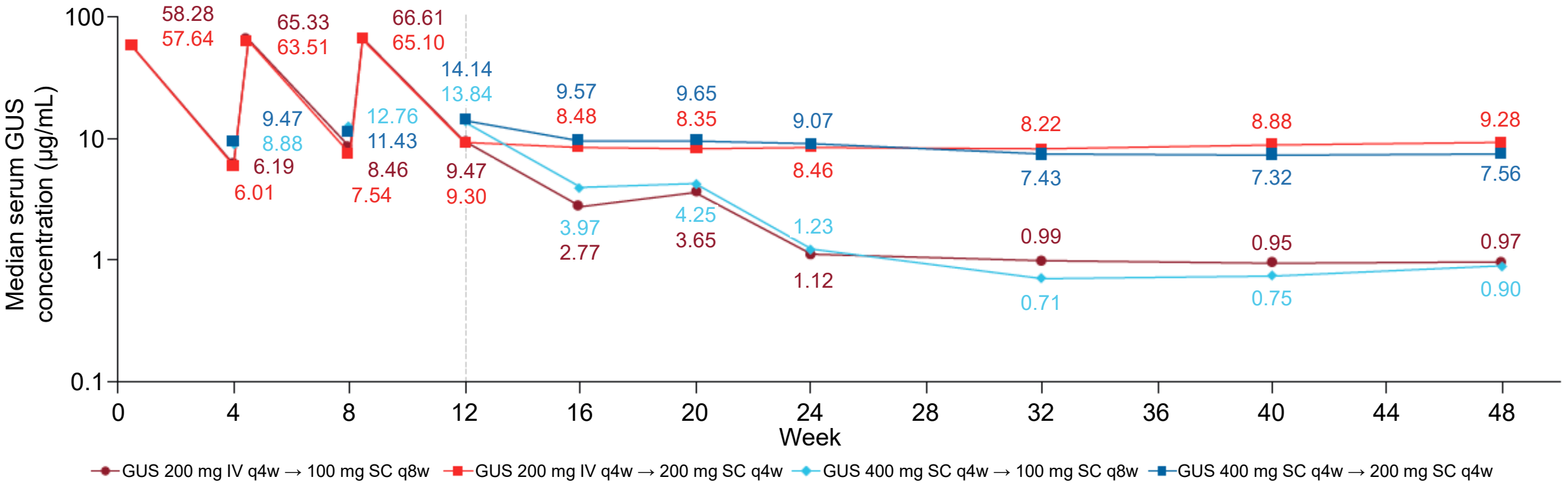
Also presented as an encore at UEGW 2026 (MP0229)

Bruce E. Sands,[1†] Geert R. D'Haens,[2] Omoniyi J. Adedokun,[3] Tae Eun Yang,[3] David T. Rubin,[4] Nat A. Terry,[3] Rian Van Rampelbergh,[5] Marion L. Vetter,[3] Tadakazu Hisamatsu,[6] Remo Panaccione,[7] Silvio Danese[8]

[1]Dr. Henry D. Janowitz Division of Gastroenterology, Icahn School of Medicine at Mount Sinai, New York, NY, USA; [2]Department of Gastroenterology, Amsterdam University Medical Centers, Amsterdam, The Netherlands; [3]Johnson & Johnson, Spring House, PA, USA; [4]University of Chicago Medicine Inflammatory Bowel Disease Centre, Chicago, IL, USA; [5]Johnson & Johnson Research & Development, Antwerp, Belgium; [6]Department of Gastroenterology and Hepatology, Kyorin University, Tokyo, Japan; [7]Inflammatory Bowel Disease Unit, Division of Gastroenterology and Hepatology, University of Calgary, Calgary, AB, Canada; [8]Gastroenterology and Endoscopy, IRCCS San Raffaele Hospital and Vita-Salute San Raffaele University, Milan, Italy

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Median serum concentrations of GUS through Week 48

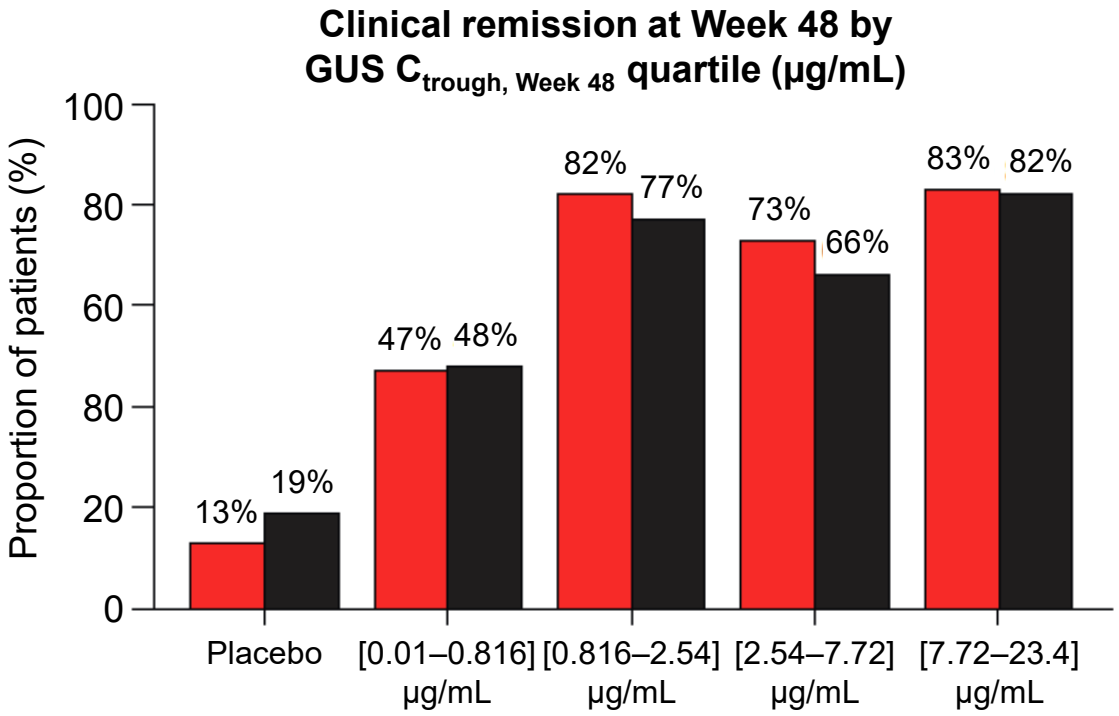


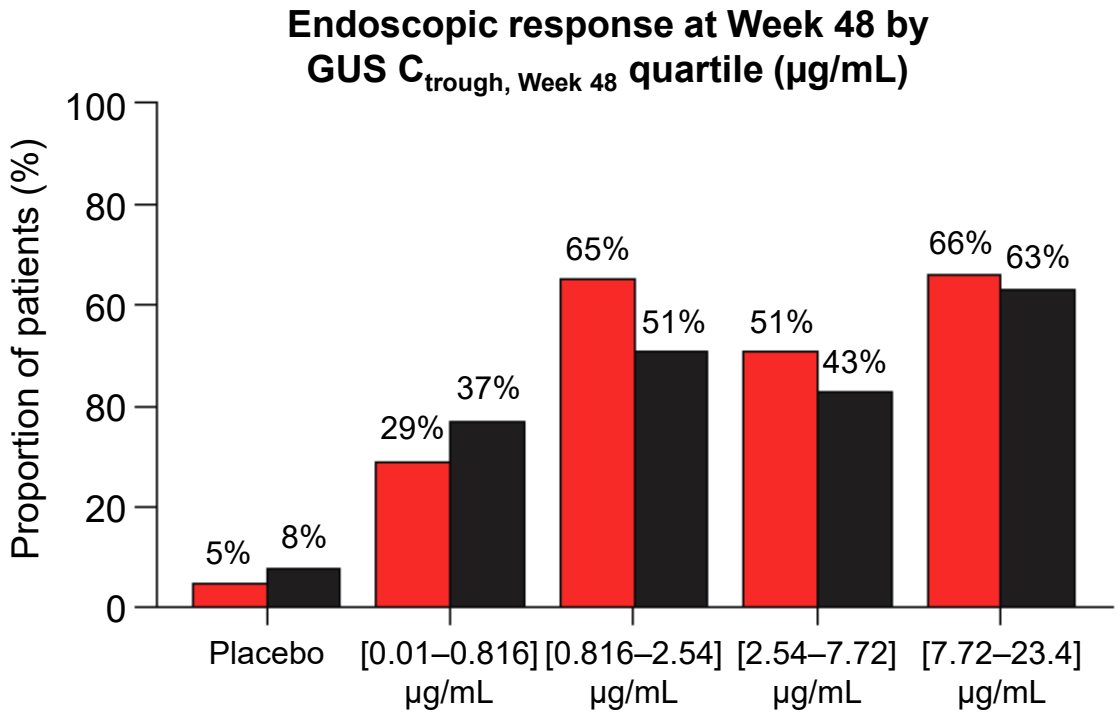
- Median GUS concentrations converged following initiation of SC maintenance dosing, with similar concentrations within each maintenance dose group regardless of IV or SC induction; steady state was reached by approximately Week 24
- Overall, the two maintenance dose regimens (100 mg SC q8w and 200 mg SC q4w) provide a four-fold dose range and an approximately eight-fold range of steady-state trough concentrations

Post-infusion concentrations for the IV induction doses are shown at Weeks 0, 4 and 8; all other values are pre-dose (trough) concentrations.
Maintenance dosing starts at Week 12 for 200 mg q4w and at Week 16 for 100 mg q8w. Concentrations are plotted on a logarithmic scale.
CD, Crohn's disease; GUS, guselkumab; IL, interleukin; IV, intravenous; MoA, mechanism of action; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous.
Sands BE, et al. Presented at ACG, 9–14 October 2026; Nashville, TN, USA. P5692. Full prescription information: swissmedicinfo-pro.ch.

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Exposure–response trends at Week 48





GALAXI 1/2/3: 200 mg IV q4w followed by 100 mg SC q8w or 200 mg SC q4w

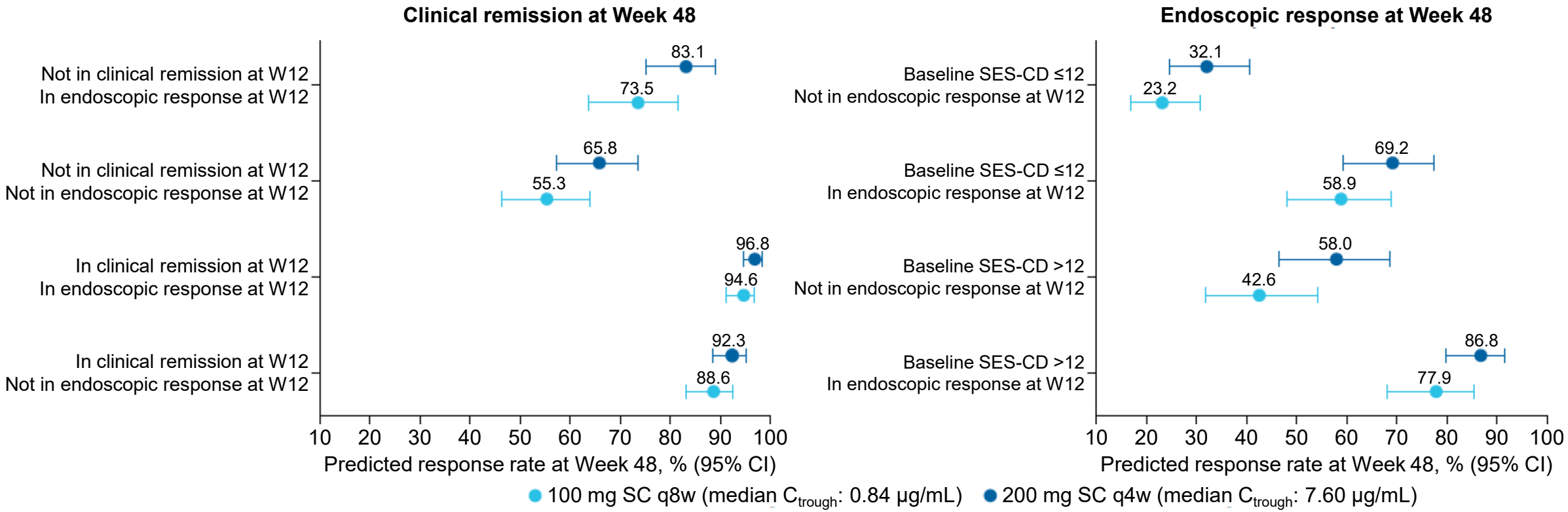
GRAVITI: 400 mg SC q4w followed by 100 mg SC q8w or 200 mg SC q4w

- Positive E-R trends were observed between steady-state GUS concentration and Week 48 efficacy (clinical remission and endoscopic response) and remained after accounting for clearance or half-life

Clinical remission: CDAI score <150. **Endoscopic response: GALAXI:** ≥50% improvement from baseline in SES-CD or SES-CD ≤2; **GRAVITI:** ≥50% improvement from baseline in SES-CD.
CD, Crohn's disease; CDAI, Crohn's Disease Activity Index; C_{trough}, trough concentration; E-R, exposure–response; GUS, guselkumab; IL, interleukin; IV, intravenous; MoA, mechanism of action; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous; SES-CD, Simple Endoscopic Score for Crohn's Disease.
Sands BE, et al. Presented at ACG, 9–14 October 2026; Nashville, TN, USA. P5692. Full prescription information: swissmedicinfo-pro.ch.

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Model-predicted Week 48 response rates

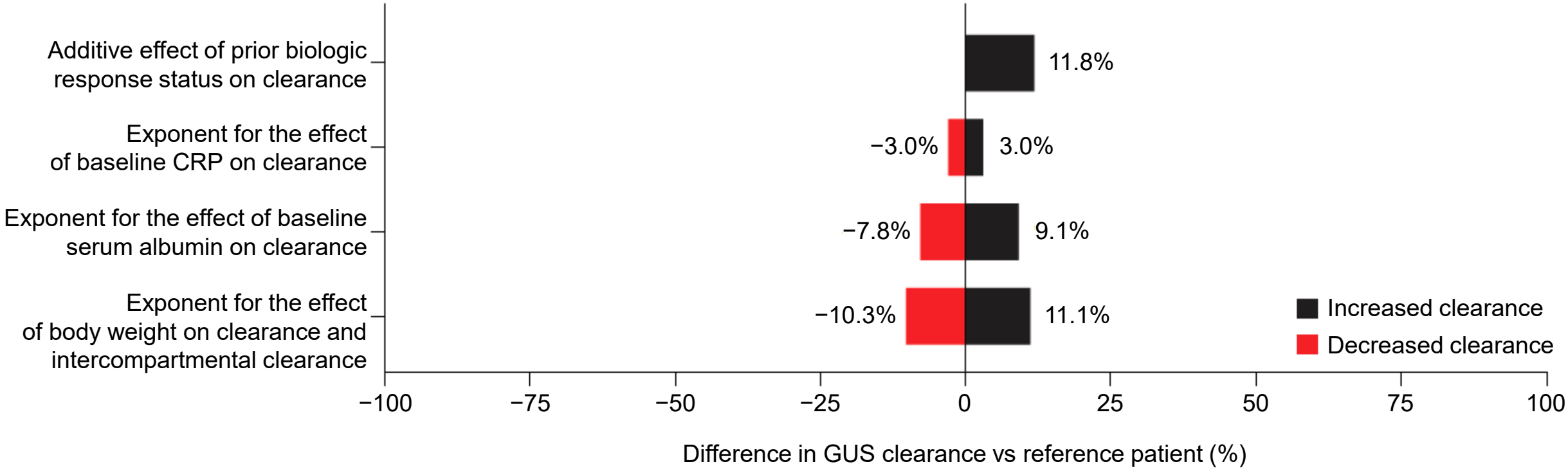


- In the exposure-response (E-R) model, participants who had not achieved clinical remission by Week 12, and those with a baseline SES-CD >12 who had not achieved an endoscopic response by Week 12, were more likely to achieve Week 48 outcomes when treated with the GUS 200 mg maintenance regimen compared with the GUS 100 mg maintenance regimen

Model-predicted Week 48 response rates for SC maintenance treatment groups, stratified by covariates. Response rate was calculated based on model-predicted median $C_{trough,\ Week\ 48}$, which is 0.84 µg/mL for the 100 mg SC q8w regimen and 7.60 µg/mL for the 200 mg SC q4w regimen.
Clinical remission: CDAI score <150. **Endoscopic response: GALAXI:** ≥50% improvement from baseline in SES-CD or SES-CD ≤2; **GRAVITI:** ≥50% improvement from baseline in SES-CD.
CD, Crohn's disease; CDAI, Crohn's Disease Activity Index; CI, confidence interval; Ctrough, trough concentration; E-R, exposure–response; GUS, guselkumab; IL, interleukin; MoA, mechanism of action; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous; SES-CD, Simple Endoscopic Score for Crohn's Disease; W, Week.
Sands BE, et al. Presented at ACG, 9–14 October 2026; Nashville, TN, USA. P5692. Full prescription information: swissmedicinfo-pro.ch.

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Impact of covariates on GUS clearance in the population PK model



- Higher body weight, lower serum albumin, higher CRP level, and inadequate response or intolerance to biologic therapies were associated with faster clearance; however, the magnitude of these effects did not warrant covariate-based dose adjustment

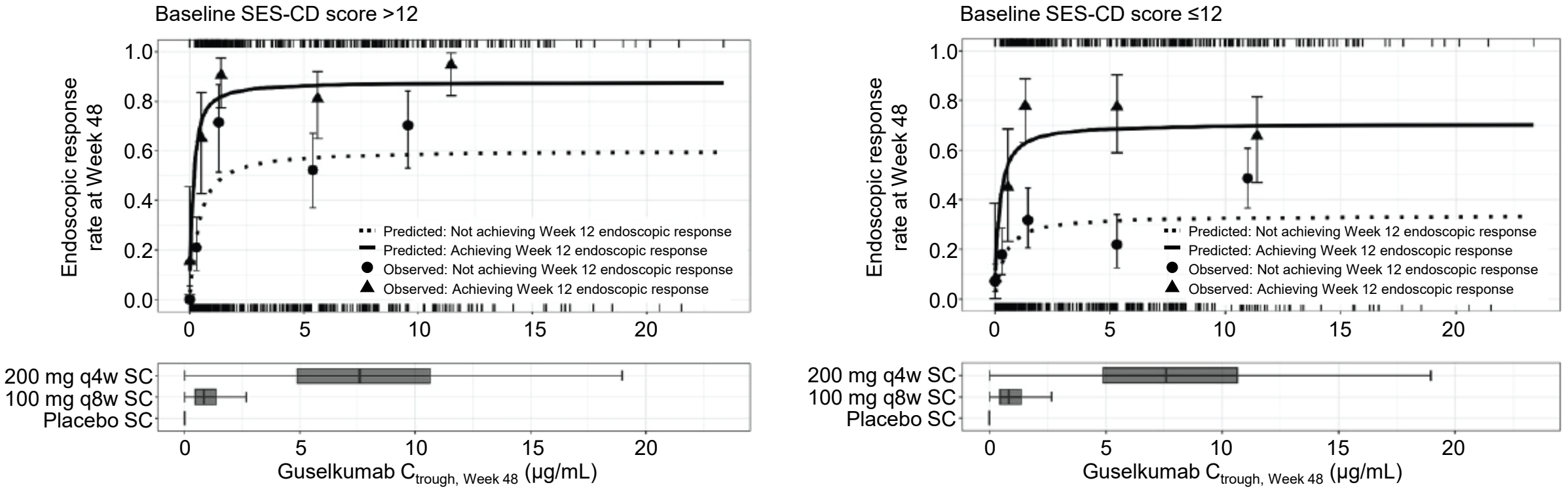
Parameter estimates from the final population PK model for GUS in participants with moderately to severely active CD.
The magnitude of change in the parameter estimate as a result of a continuous covariate was expressed as a range (i.e. percentage change from the median value, when the covariate factor varied from the 25th percentile to the 75th percentile of the population).

$$CL\left(\frac{L}{day}\right) = 0.304 \times \left(\frac{Body\ weight}{70}\right)^{0.618} \times \left(\frac{Albumin}{43}\right)^{-1.20} \times \left(\frac{CRP}{6.4}\right)^{0.0281} \times (1.118)^{prior\ biologic\ failure\ status}$$

CD, Crohn's disease; CL, clearance; CRP, C-reactive protein; GUS, guselkumab; IL, interleukin; MoA, mechanism of action; PK, pharmacokinetic; RDBPC, randomised, double-blind, placebo-controlled.
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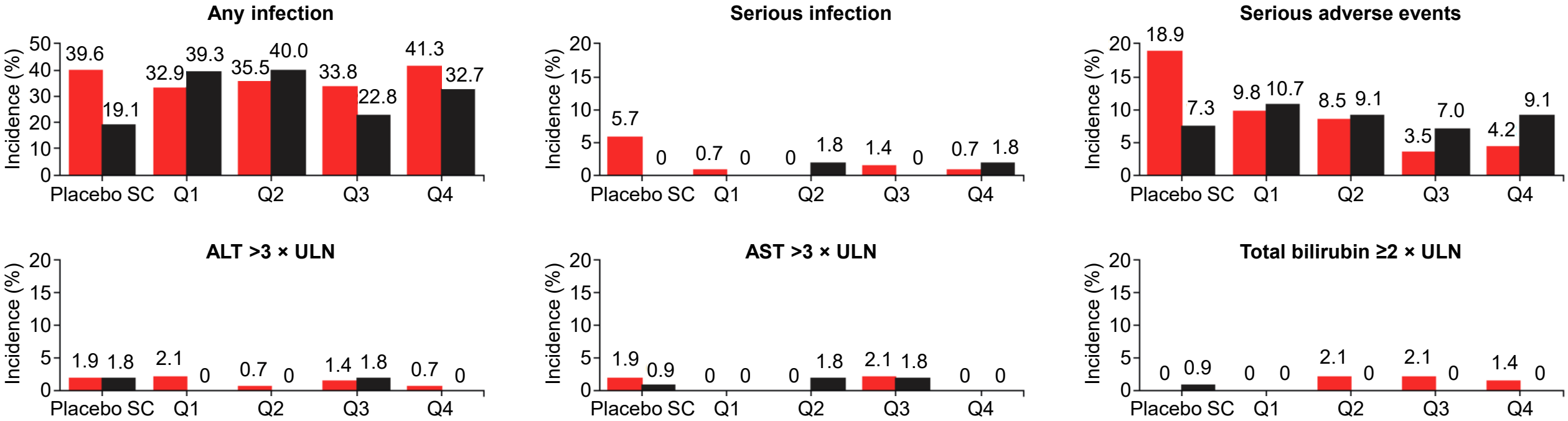
Model-predicted exposure–response relationships for endoscopic response at Week 48*



- Exposures for both GUS maintenance dose regimens were associated with substantial efficacy for most participants; 200 mg SC q4w exposures were at the plateau of the E-R curves, suggesting that maximal response is likely achieved with this regimen

Model-predicted E-R relationships for endoscopic response at Week 48 using $C_{trough,\ Week\ 48}$ as the PK exposure metric in participants with CD, stratified by baseline SES-CD and endoscopic response at Week 12.
CD, Crohn's disease; C_{trough}, trough concentration; E-R, exposure–response; GUS, guselkumab; IL, interleukin; MoA, mechanism of action; PK, pharmacokinetic; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous; SES-CD, Simple Endoscopic Score for Crohn's Disease.
Sands BE, et al. Presented at ACG, 9–14 October 2026; Nashville, TN, USA. P5692. Full prescription information: swissmedicinfo-pro.ch.

No associations between GUS concentrations and infections, serious infections, serious adverse events, or liver function tests were observed



GALAXI 1/2/3: 200 mg IV q4w followed by 100 mg SC q8w or 300 mg SC q4w

GRAVITI: 400 mg SC q4w followed by 100 mg SC q8w or 200 mg SC q4w

- Immunosuppressants did not impact GUS concentrations (data not shown)

The PK analysis set includes participants randomised to GUS 200 mg IV q4w or GUS 400 mg SC q4w in induction who had evaluable GUS trough concentrations during the induction period (Week 0 through Week 12) or at steady state during the maintenance period (Week 12 through Week 48). Data for placebo participants are also included as a reference. Includes only participants with a baseline SES-CD ≥6 (≥4 for those with isolated ileal disease). Concentrations below the lowest quantifiable concentration were treated as zero in calculating the summary statistics. For GALAXI, includes participants who were in clinical response (as assessed by IWRS) to placebo after induction treatment. For GRAVITI, includes all placebo participants excluding data after a participant was rescued with GUS. GALAXI serum GUS concentration quartiles include data from Week 12 to Week 48 and are based on participants randomised to GUS 200 mg IV q4w in induction as follows: Q1 = <0.96 µg/mL, Q2 = ≥0.96 to <2.68 µg/mL, Q3 = ≥2.68 to 8.34 µg/mL, Q4 = ≥8.34. GRAVITI serum GUS concentration quartiles include data from Week 12 to Week 48 and are based on participants randomised to GUS 400 mg SC q4w in induction as follows: Q1 = <0.92 µg/mL, Q2 = ≥0.92 to <2.87 µg/mL, Q3 = ≥2.87 to <7.61 µg/mL, Q4 = ≥7.61. Average steady-state trough concentrations were calculated based on the following visits: 1) Weeks 24, 32, 40 and 48 for participants randomised to receive GUS 100 mg SC q8w; 2) Weeks 20, 24, 28, 32, 36, 40, 44 and 48 for participants randomised to receive GUS 200 mg SC q4w. ALT, alanine aminotransferase; AST, aspartate aminotransferase; CD, Crohn's disease; GUS, guselkumab; IL, interleukin; IWRS, Interactive Web Response System; IV, intravenous; MoA, mechanism of action; PK, pharmacokinetic; Q, quartile; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous; SES-CD, Simple Endoscopic Score for Crohn's Disease; ULN, upper limit of normal.

Sands BE, et al. Presented at ACG, 9–14 October 2026; Nashville, TN, USA. P5692. Full prescription information: swissmedicinfo-pro.ch.

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Conclusions

- Maintenance PK and efficacy data were consistent regardless of induction route (IV or SC), supporting a common maintenance dosing strategy in CD irrespective of route of induction regimen
- E-R modelling showed efficacy with both maintenance regimens, but exposures with 200 mg q4w were near the plateau of the E-R curves, suggesting that this regimen is more likely to achieve a maximal treatment response
- Patients with higher residual disease activity after induction may benefit more from the 200 mg maintenance dose
- There were no associations between GUS concentrations and safety events

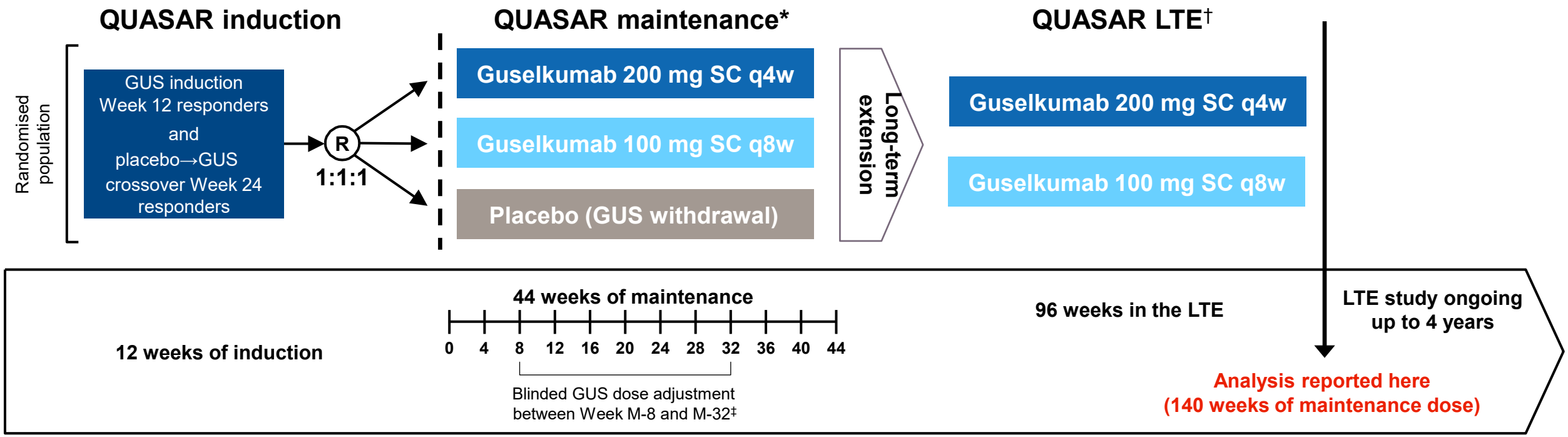
CD, Crohn's disease; E-R, exposure–response; GUS, guselkumab; IV, intravenous; PK, pharmacokinetic; qXw, every X weeks; SC, subcutaneous.
Sands BE, et al. Presented at ACG, 9–14 October 2026; Nashville, TN, USA. P5692. Full prescription information: swissmedicinfo-pro.ch.

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Guselkumab in ulcerative colitis

QUASAR: Guselkumab for UC

QUASAR study design



*Patients who completed the safety and efficacy evaluations (including the required endoscopy procedure) at maintenance Week M-44 and who may benefit from continued study intervention, in the opinion of the investigator, had the opportunity to participate in the LTE of the maintenance study. †The study blinding was maintained during the LTE until the last patient in the maintenance study completed the M-44 visit. After the maintenance study was unblinded, patients had their study visits scheduled to coincide with their dose regimen (either q4w or q8w) and patients receiving placebo were terminated from study participation. ‡Randomised patients meeting loss of clinical response criteria (based on the mMayo score and required an endoscopic assessment) were eligible for a blinded dose adjustment as follows: placebo SC → GUS 200 mg SC q4w; GUS 100 mg SC q8w → GUS 200 mg SC q4w; GUS 200 mg SC q4w → GUS 200 mg SC q4w (sham adjustment).

GUS, guselkumab; IL, interleukin; LTE, long-term extension; M, maintenance; mMayo, modified Mayo; MOA, mechanism of action; qXw, every X weeks; R, randomisation; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous; UC, ulcerative colitis.

Rubin DT, et al. Presented at ACG, Nashville, Tennessee, USA, 9–14 October 2026. OP43. Full prescription information: swissmedicinfo-pro.ch.

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Histology outcomes over time in patients with moderately to severely active ulcerative colitis treated with guselkumab in the QUASAR long-term extension study

OP43

Oral presentation at ACG

David T. Rubin,[1] Jessica R. Allegretti,[2] Yelina Alvarez,[3] Thomas Baker,[3] Shashi Adsul,[4] Darren Piscitelli,[4] Ye Miao,[3] Laurent Peyrin-Biroulet[5]

[1]University of Chicago Medicine Inflammatory Bowel Disease Center, Chicago, IL, USA; [2]Division of Gastroenterology, Hepatology and Endoscopy, Brigham and Women's Hospital, Harvard Medical School, Boston, MA, USA; [3]Johnson & Johnson, Spring House, PA, USA; [4]Johnson & Johnson, Horsham, PA, USA; [5]Northwestern Medicine Digestive Health Institute, Chicago, IL, USA

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Endpoints, Geboes score categories and statistical considerations

- Sankey diagrams of Geboes score categories were created for patients randomised to GUS in the maintenance study, continued in the LTE study, and had Geboes score evaluations at all time points from induction baseline through Week M-140 (100 mg q8w or dose adjusted, n=137; 200 mg q4w, n=130)

Geboes score categories

- **5.4:** neutrophils in lamina propria and epithelium; crypt destruction with ulceration
- **5.3:** neutrophils in lamina propria and epithelium; crypt destruction with erosion of the surface epithelium
- **3.2–5.2:** neutrophils in lamina propria and epithelium; varying degrees of crypt damage, possibly including surface epithelium; no erosion or ulceration
- **2B.1–3.1:** neutrophils in lamina propria, few neutrophils in epithelium in <5% of crypts
- **0.0–2B.0:** no neutrophils (no histological activity)

Statistical considerations

- Patients who had an ostomy or colectomy, or discontinued study agent due to lack of efficacy or an adverse event of worsening UC before the designated post-maintenance baseline time point had their induction baseline value carried forward from the time of the event onward
- For patients who discontinued study agent for any other reason before the designated post-maintenance baseline time point, their observed values were used, if available
- After accounting for these events, patients missing any histology components at any time point were excluded

GUS, guselkumab; IL, interleukin; LTE, long-term extension; M, maintenance; MOA, mechanism of action; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; UC, ulcerative colitis.
Rubin DT, et al. Presented at ACG, Nashville, Tennessee, USA, 9–14 October 2026. OP43. Full prescription information: swissmedicinfo-pro.ch.

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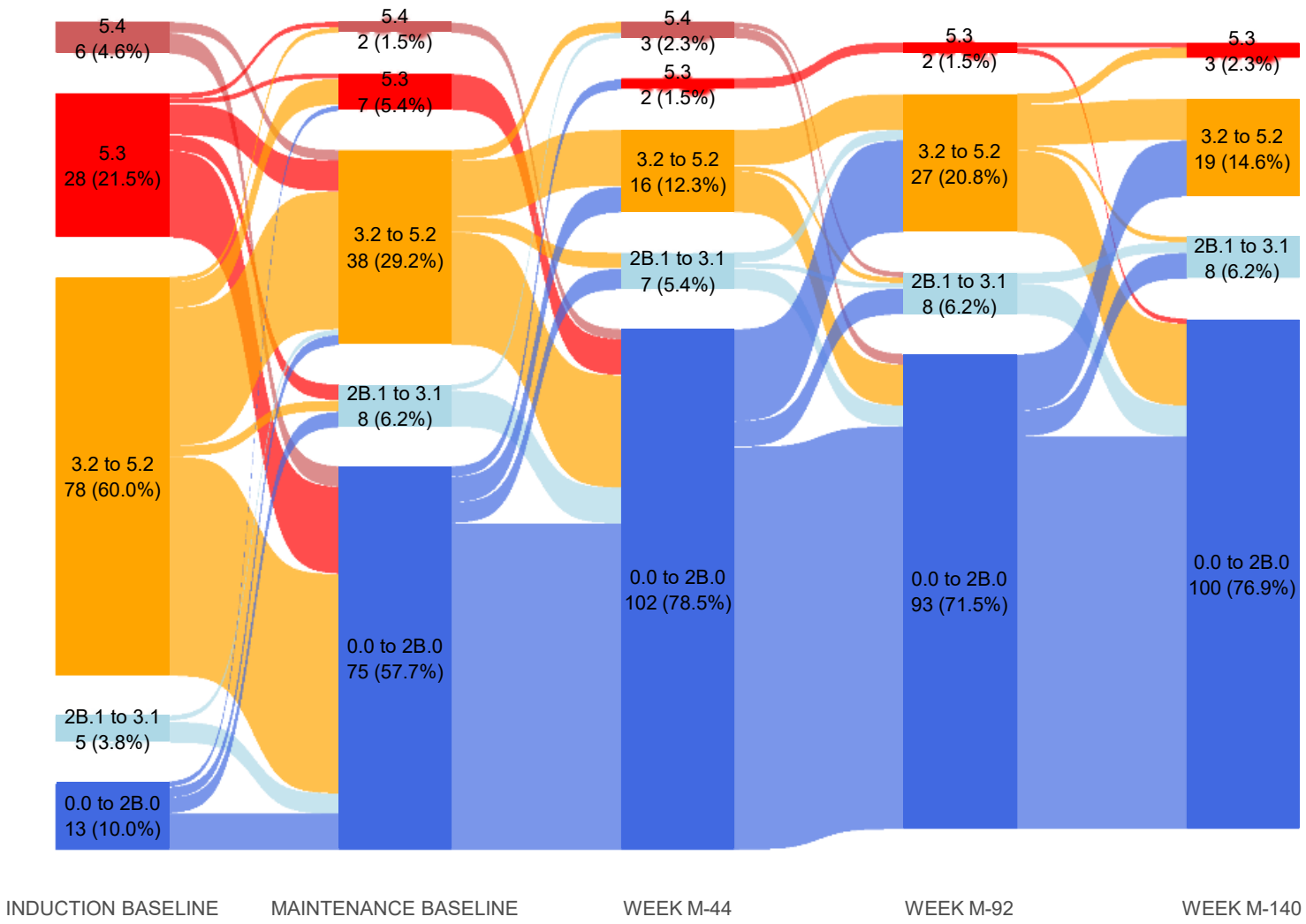
Induction baseline demographics and disease characteristics (LTE randomised full analysis set)

	Guselkumab	
	100 mg q8w n=155	200 mg q4w n=148
Demographics		
Age, years, mean (SD)	40.2 (12.8)	40.6 (15.1)
Male, n (%)	83 (53.5)	75 (50.7)
Disease characteristics		
UC disease duration, years, mean (SD)	8.2 (9.0)	8.2 (8.5)
mMayo score (0–9), mean (SD)	6.8 (1.2)	6.9 (1.1)
mMayo score of 7–9 (severe), n (%)	94 (60.6)	97 (65.5)
C-reactive protein, mg/L, median (IQR)	4.0 (1.4–10.4)*	3.9 (1.5–9.5)†
Faecal calprotectin, mg/kg, median (IQR)	1709.0 (815.0–3607.0)‡	1605.5 (596.0–3253.0)§
Prior inadequate response/intolerance to ADT, n (%)	60 (38.7)	62 (41.9)
ADT-naive, n (%)	90 (58.1)	81 (54.7)
Concomitant UC medications		
Oral corticosteroids, n (%)	56 (36.1)	54 (36.5)
Immunomodulatory drugs, n (%)	39 (25.2)	36 (24.3)

*n=153; †n=145; ‡n=133; §n=134.
ADT, advanced therapy; GUS, guselkumab; IL, interleukin; IQR, interquartile range; mMayo, modified Mayo; MOA, mechanism of action; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; SD, standard deviation; UC, ulcerative colitis.
Rubin DT, et al. Presented at ACG, Nashville, Tennessee, USA, 9–14 October 2026. OP43. Full prescription information: swissmedicinfo-pro.ch.

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Geboes score through 3 years with GUS SC 200 mg q4w (n=130)



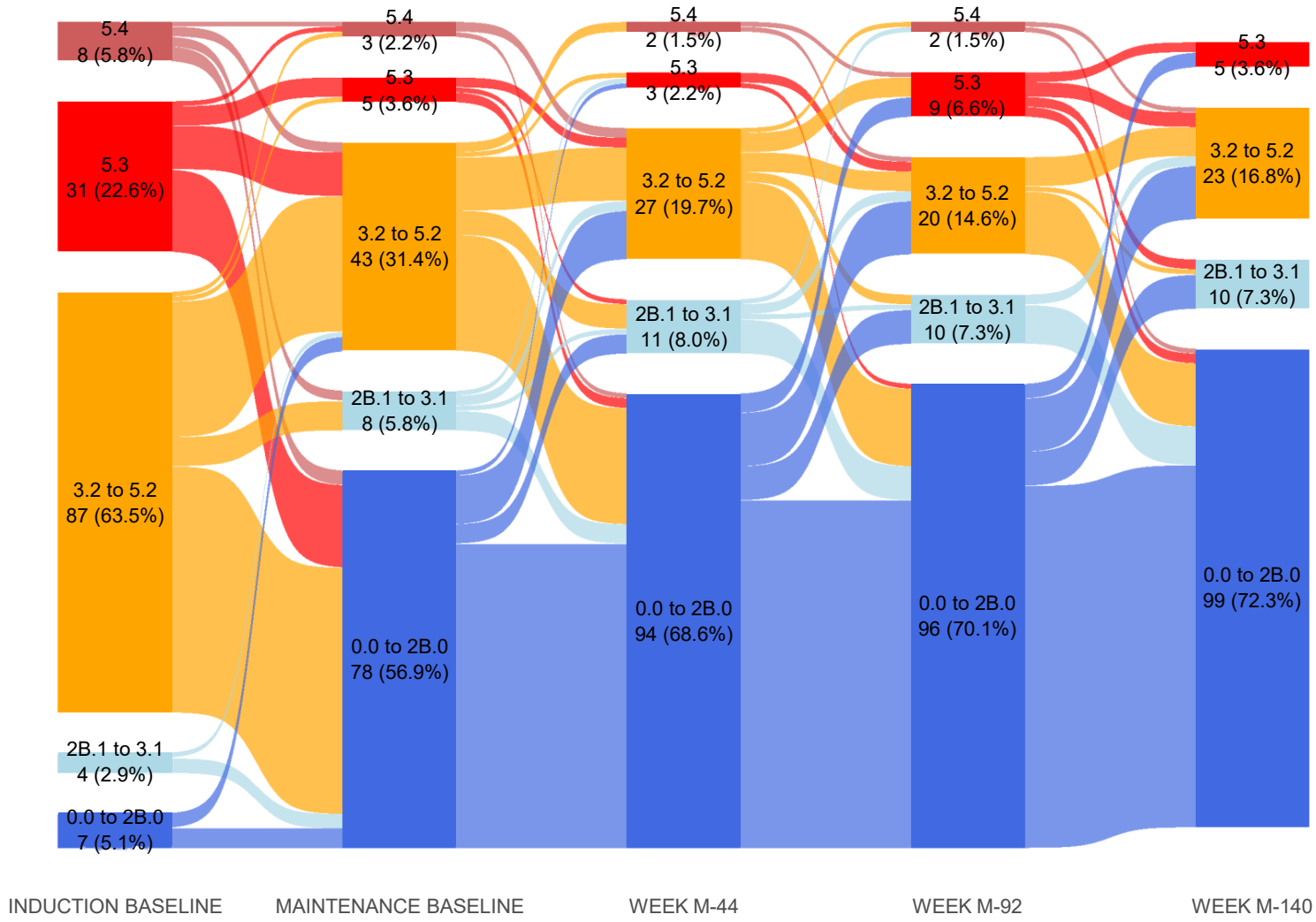
Geboes score key

- 5.4: neutrophils in lamina propria and epithelium; crypt destruction with ulceration
- 5.3: neutrophils in lamina propria and epithelium; crypt destruction with erosion of the surface epithelium
- 3.2 to 5.2: neutrophils in lamina propria and epithelium; varying degrees of crypt damage, possibly including surface epithelium; no erosion or ulceration
- 2B.1 to 3.1: neutrophils in lamina propria, few neutrophils in epithelium in <5% of crypts
- 0.0 to 2B.0: no neutrophils (no histological activity)

GUS, guselkumab; IL, interleukin; M, maintenance; MOA, mechanism of action; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous; UC, ulcerative colitis.
Rubin DT, et al. Presented at ACG, Nashville, Tennessee, USA, 9–14 October 2026. OP43. Full prescription information: swissmedicinfo-pro.ch.

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Geboes score through 3 years with GUS SC 100 mg q8w (200 mg q4w after dose adjustment) (n=137)



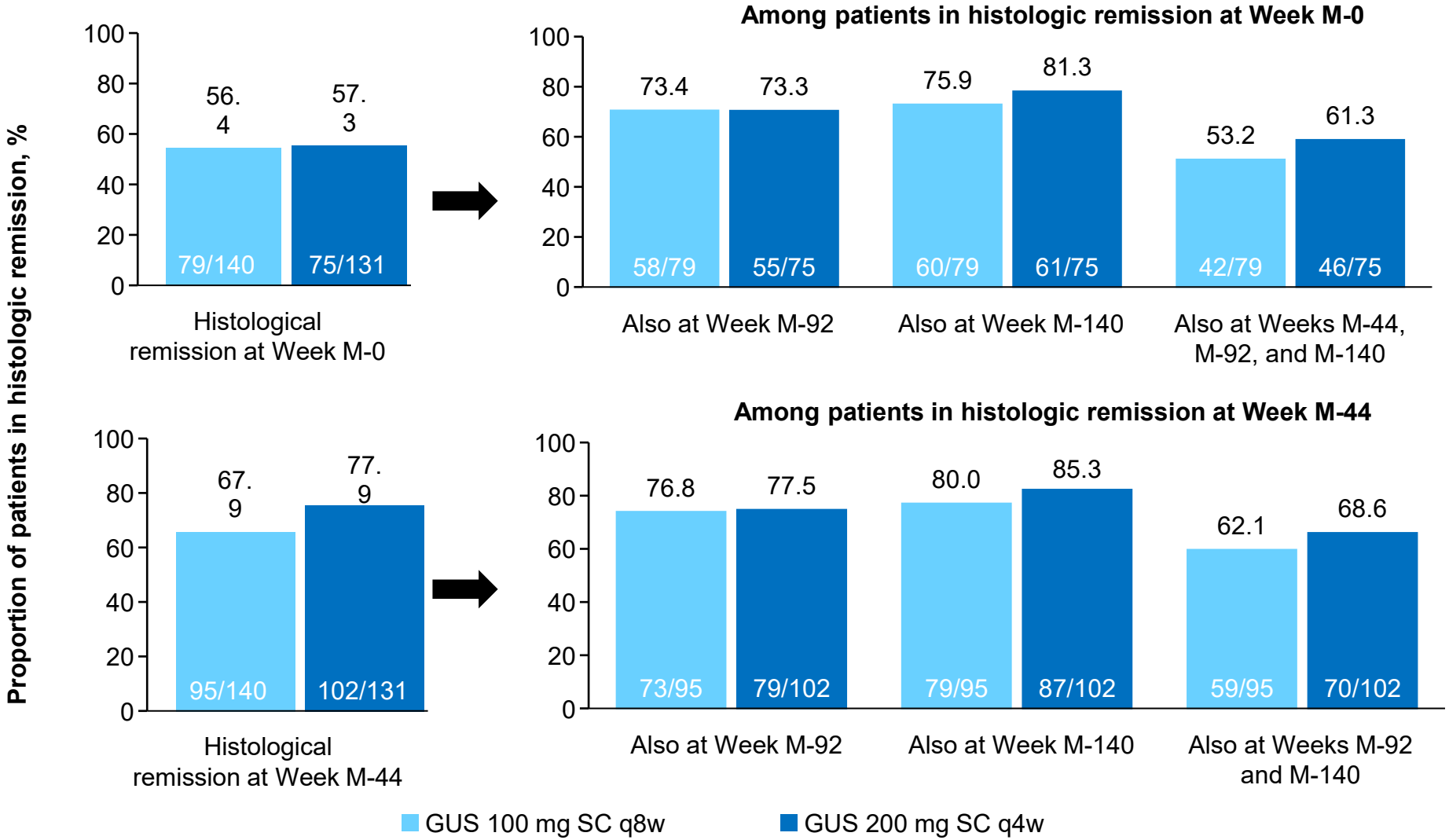
Geboes score key

- 5.4: neutrophils in lamina propria and epithelium; crypt destruction with ulceration
- 5.3: neutrophils in lamina propria and epithelium; crypt destruction with erosion of the surface epithelium
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- 2B.1 to 3.1: neutrophils in lamina propria, few neutrophils in epithelium in <5% of crypts
- 0.0 to 2B.0: no neutrophils (no histological activity)

GUS, guselkumab; IL, interleukin; M, maintenance; MOA, mechanism of action; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous; UC, ulcerative colitis.
Rubin DT, et al. Presented at ACG, Nashville, Tennessee, USA, 9–14 October 2026. OP43. Full prescription information: swissmedicinfo-pro.ch.

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Maintenance of histological remission



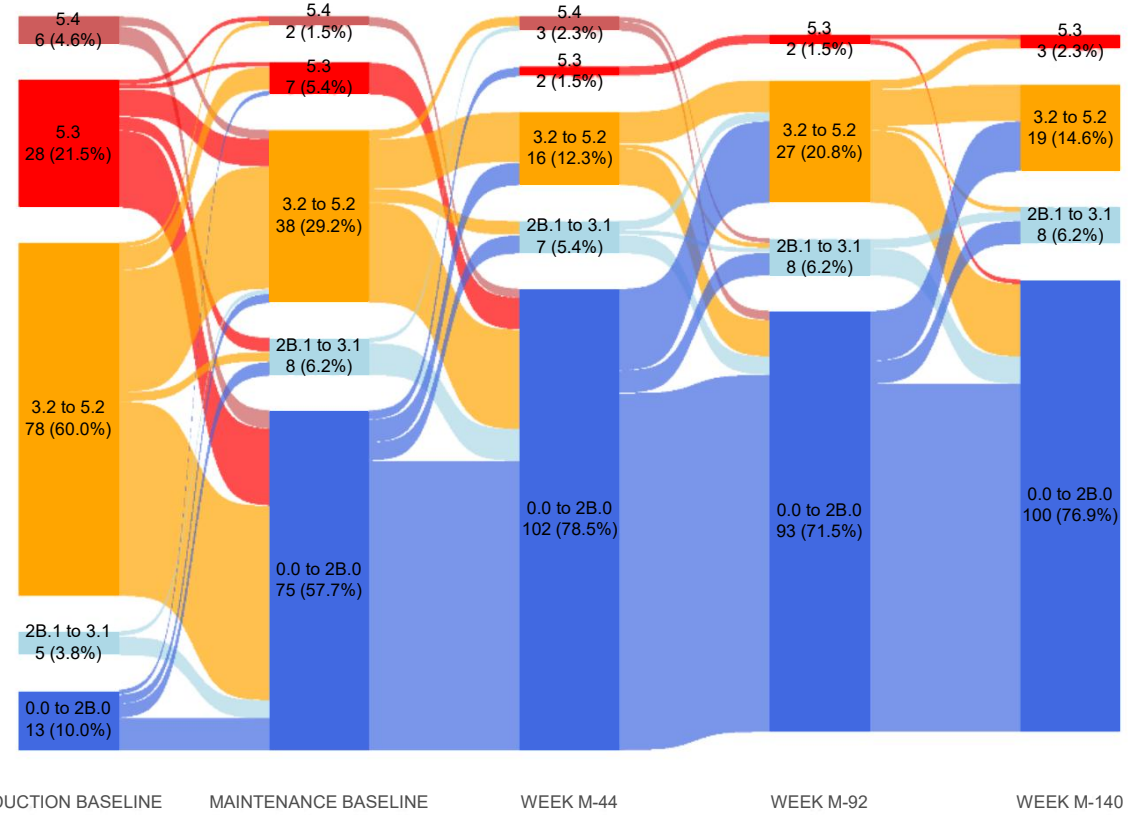
Most patients who achieved histological remission after induction or maintenance remained in histological remission at 3 years

Histologic remission: Absence of neutrophils from the mucosa (both lamina propria and epithelium), no crypt destruction, and no erosions, ulcerations, or granulation tissue.
GUS, guselkumab; IL, interleukin; M, maintenance; MOA, mechanism of action; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous; UC, ulcerative colitis.
Rubin DT, et al. Presented at ACG, Nashville, Tennessee, USA, 9–14 October 2026. OP43. Full prescription information: swissmedicinfo-pro.ch.

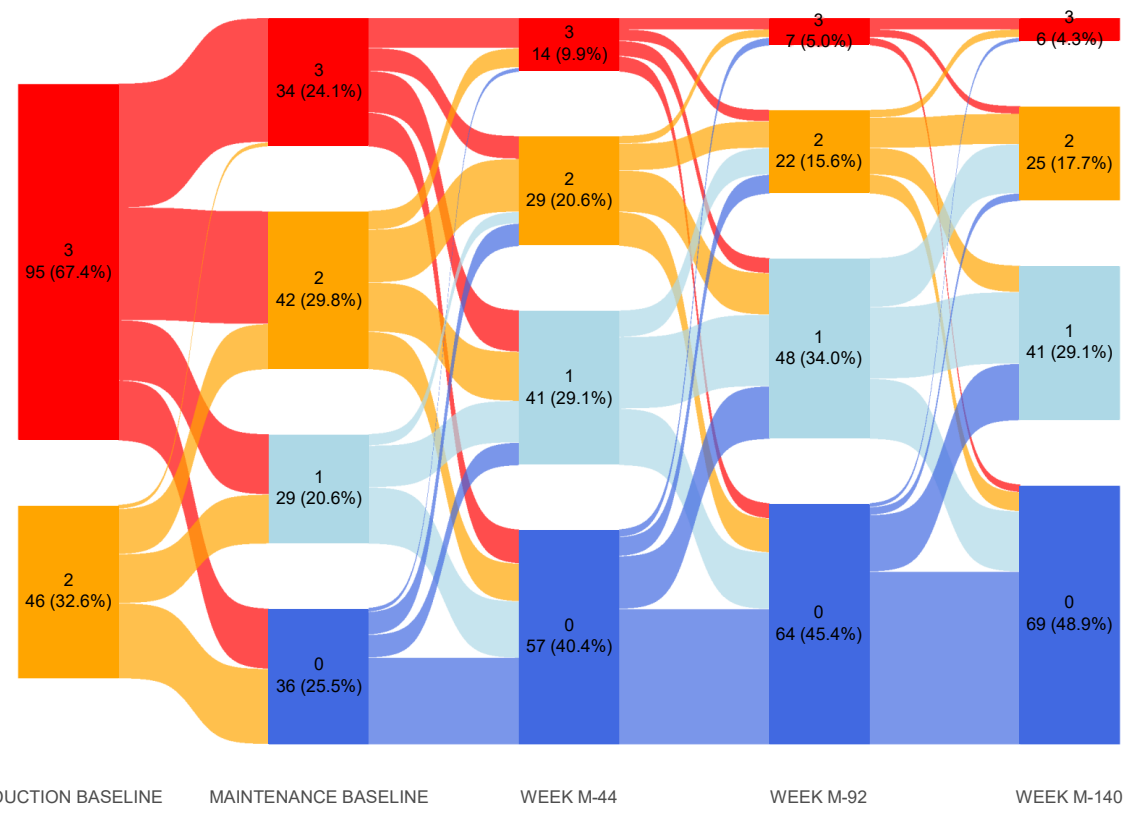
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Geboes score and Mayo Endoscopic Score through 3 years with GUS SC 200 mg q4w

Geboes score (n=130)[1]



Mayo Endoscopic Score (n=141)[2]



Geboes score = 5.4
Geboes score = 5.3
Geboes score = 3.2 to 5.2
Geboes score = 2B.1 to 3.1
Geboes score = 0.0 to 2B.0

MES = 3
MES = 2
MES = 1
MES = 0

Similar improvements in Geboes score and Mayo Endoscopic Score from baseline to Week M-140 were observed with GUS

GUS, guselkumab; IL, interleukin; M, maintenance; MOA, mechanism of action; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous; UC, ulcerative colitis.

1. Rubin DT, et al. Presented at ACG, Nashville, Tennessee, USA, 9–14 October 2026. OP43; 2. Rubin DT, et al. Presented at DDW, Chicago, IL, USA, 2–5 May 2026. Mo1516. Full prescription information: swissmedicinfo-pro.ch.

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Conclusions

- Both guselkumab maintenance doses were associated with histological improvements over 3 years[1]
- Although approximately 40% of induction clinical responders had moderate to severe histological disease activity (Geboes score = 3.2–5.4) at the beginning of maintenance, continued histological improvement was observed during guselkumab maintenance treatment up to 1 year and was generally maintained through 3 years[1]
- Histology improvement trends over time were similar to previously reported endoscopy improvement trends over time[2]

GUS, guselkumab; IL, interleukin; MOA, mechanism of action; RDBPC, randomised, double-blind, placebo-controlled; UC, ulcerative colitis.
1. Rubin DT, et al. Presented at ACG, Nashville, Tennessee, USA, 9–14 October 2026. OP43; 2. Rubin DT, et al. Presented at DDW, Chicago, IL, USA, 2–5 May 2026. Mo1516. Full prescription information: swissmedicinfo-pro.ch.

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Symptomatic and biomarker outcomes among participants treated with guselkumab not in clinical response at Week 12: Results from the Phase 3 QUASAR induction study

P3359

Presented at ACG 2026

Jessica R. Allegretti,[1] Alessandro Armuzzi,[2] Jordan Axelrad,[3] Yelina Alvarez,[4] Thomas Baker,[4] Caroline Kerner,[5] Shashi Adsul,[5] Ye Miao,[4] Hongyan Zhang,[4] Axel Dignass[6]

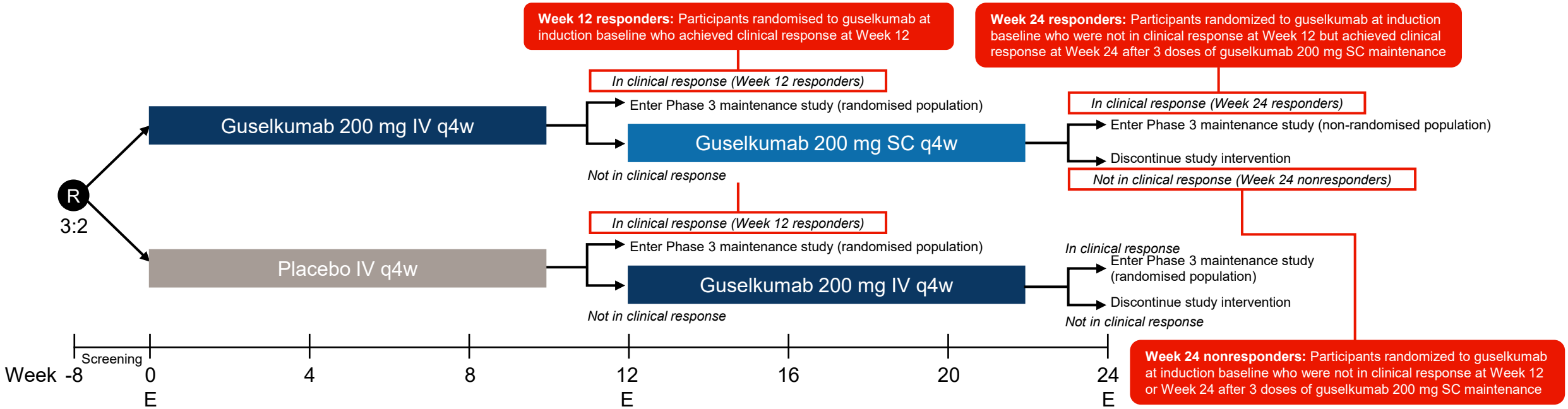
[1]Division of Gastroenterology, Hepatology and Endoscopy, Brigham and Women's Hospital, Harvard Medical School, Boston, MA, USA; [2]IBD Center, Humanitas Research Hospital, Humanitas University, Milan, Italy; [3]Division of Gastroenterology, Department of Medicine, NYU Grossman School of Medicine, New York, NY, USA; [4]Johnson & Johnson, Spring House, PA, USA; [5]Johnson & Johnson, Horsham, PA, USA; [6]Department of Medicine I, Agaplesion Markus Hospital, Goethe University, Frankfurt, Germany

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Phase 3 QUASAR induction study design through Week 24

Aim: post hoc analysis evaluating clinical characteristics, symptomatic response, and biomarkers according to Week 24 response status in the Phase 3 QUASAR induction study

- In the Phase 3 QUASAR induction study, clinical responders to guselkumab IV induction at Week 12 entered the randomised-withdrawal maintenance study, and clinical nonresponders received guselkumab 200 mg SC q4w and were evaluated through Week 24



Key eligibility criteria
- Baseline modified Mayo score of 5 to 9
- Induction baseline Mayo rectal bleeding subscore ≥1, Mayo endoscopic subscore ≥2 (centrally reviewed)
- Inadequate response/intolerance to oral corticosteroids, 6-MP/AZA, biologics* or JAKi

Randomisation stratified by:
- Prior BIO/JAKi-IR status (yes/no)
- Region (Eastern Europe, Asia or rest of world)
- Concomitant corticosteroid use at baseline (yes/no)

Clinical response was defined as a ≥30% and ≥2-point decrease from baseline in the modified Mayo score, with either a ≥1-point decrease from baseline in the rectal bleeding subscore or a rectal bleeding subscore of 0 or 1.
*Biologic therapies: TNF antagonists or vedolizumab.
6-MP, 6-mercaptopurine; AZA, azathioprine; BIO, biologic; E, endoscopy; GUS, guselkumab; IL, interleukin; IR, inadequate response/intolerance; IV, intravenous; JAKi, Janus kinase inhibitor; MoA, mechanism of action; qXw, every X weeks; R, randomisation; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous; UC, ulcerative colitis.
Allegretti JR, et al. Presented at ACG, 9–14 October 2026; Nashville, TN, USA. P3359. Full prescription information: swissmedicinfo-pro.ch.

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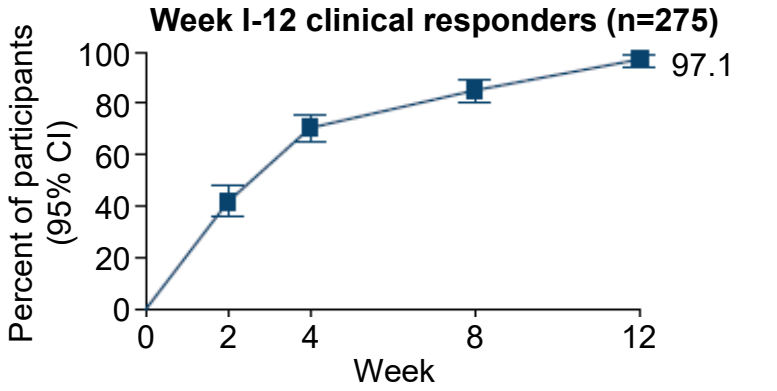
Baseline demographics and disease characteristics

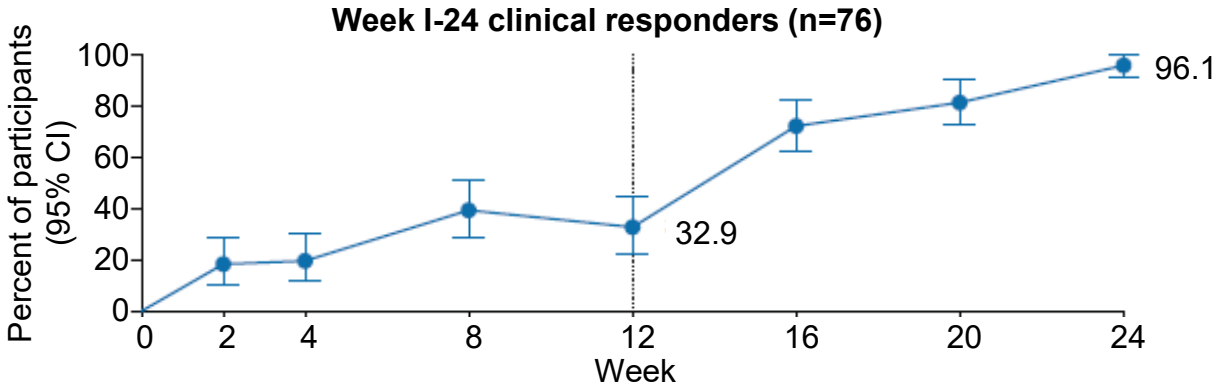
	GUS 200 mg IV → Week 12 clinical responder* (n=275)	GUS 200 mg IV → 200 mg SC q4w Week 24 clinical responder* (n=76)	GUS 200 mg IV → 200 mg SC q4w Week 24 clinical nonresponder* (n=35)
Demographics			
Age in years, mean (SD)	41.2 (14.01)	40.3 (14.13)	38.9 (11.71)
Male, n (%)	146 (53.1%)	47 (61.8%)	22 (62.9%)
Disease characteristics			
UC duration in years, mean (SD)	7.90 (8.21)	6.91 (5.53)	8.99 (8.91)
Modified Mayo score (0–9), mean (SD)	6.9 (1.18)	6.9 (0.95)	6.9 (1.04)
Modified Mayo score of 7–9 (severe), n (%)	169 (61.5%)	56 (73.7%)	23 (65.7%)
Mayo endoscopic subscore of 3 (severe), n (%)	182 (66.2%)	60 (78.9%)	27 (77.1%)
Extensive UC, n (%)	122 (44.4%)	31 (40.8%)	20 (57.1%)
Biomarkers			
C-reactive protein in mg/L, N	272	76	34
C-reactive protein, median (IQR)	3.94 (1.39; 9.11)	5.39 (1.51; 15.45)	6.46 (2.54; 9.54)
Faecal calprotectin in mg/kg, N	243	68	28
Faecal calprotectin, median (IQR)	1641.0 (590.0; 3741.0)	1920.0 (807.5; 3723.5)	1624.0 (1336.0; 4382.0)
Concomitant UC medications at baseline, n (%)			
6-mercaptopurine/azathioprine	60 (21.8%)	17 (22.4%)	8 (22.9%)
Methotrexate	2 (0.7%)	0	0
Oral corticosteroids	108 (39.3%)	37 (48.7%)	21 (60.0%)
Biologic history, n (%)			
BIO/JAKi naïve	152 (55.3%)	30 (39.5%)	11 (31.4%)
BIO/JAKi-IR[b]	114 (41.5%)	45 (59.2%)	23 (65.7%)

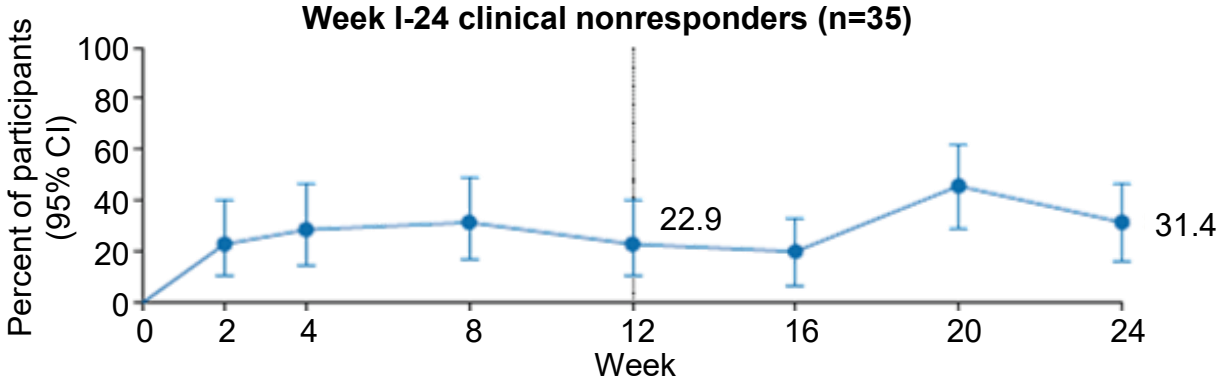
Includes only participants with modified Mayo score 5–9 at induction baseline. Response groups were defined sequentially using observed IWRS data. Among 421 GUS-randomised participants, 21 discontinued before Week 12, leaving 400 classified as either Week 12 responders (n=275) or Week 12 non-responders (n=125). Among 125 Week 12 non-responders, 14 discontinued before Week 24, leaving 111 classified as Week 24 responders (n=76) or Week 24 non-responders (n=35).
*Clinical responders/non-responders were determined by IWRS using data observed at the designated visit (Week 12 or Week 24). †Primary non-response, secondary non-response or intolerance.
BIO, biologic; GUS, guselkumab; IL, interleukin; IQR, interquartile range; IR, inadequate response/intolerance; IV, intravenous; IWRS, Interactive Web Response System;
JAKi, Janus kinase inhibitor; MoA, mechanism of action; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous; SD, standard deviation; UC, ulcerative colitis.
Allegretti JR, et al. Presented at ACG, 9–14 October 2026; Nashville, TN, USA. P3359. Full prescription information: swissmedicinfo-pro.ch.

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Symptomatic response over time in Week 12 and Week 24 responders, and Week 24 non-responders





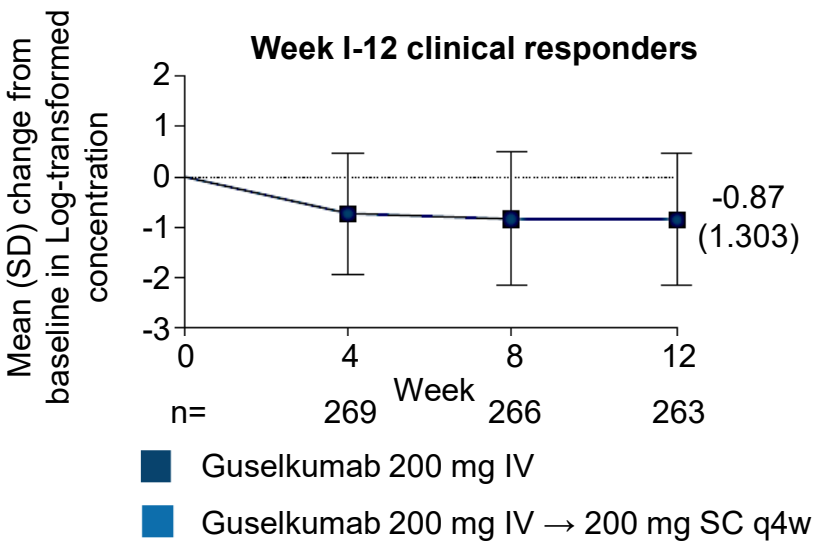


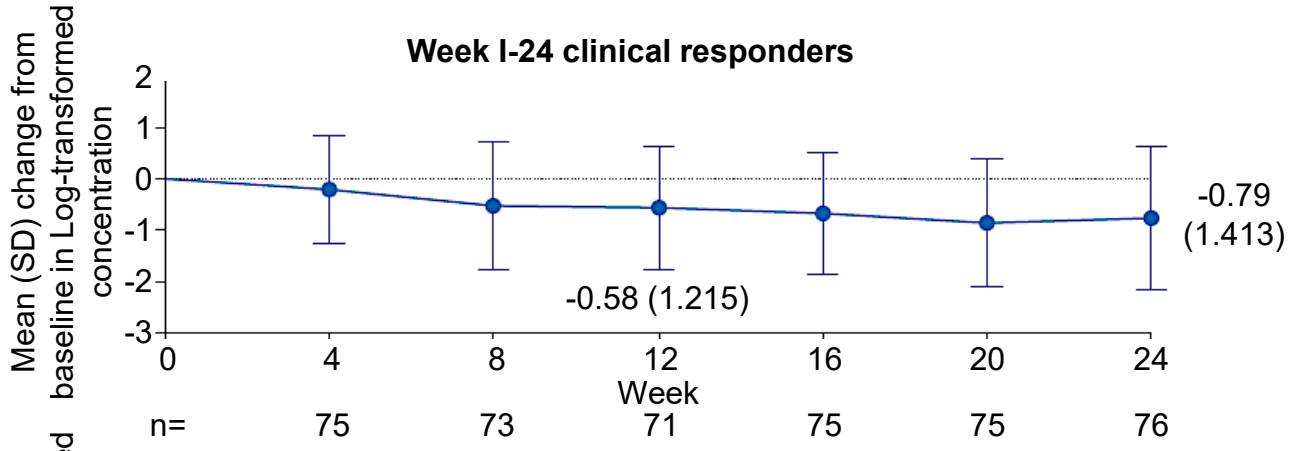
Among participants without clinical response at Week 12 after guselkumab IV induction, a subset benefited after three additional guselkumab 200 mg SC q4w injections

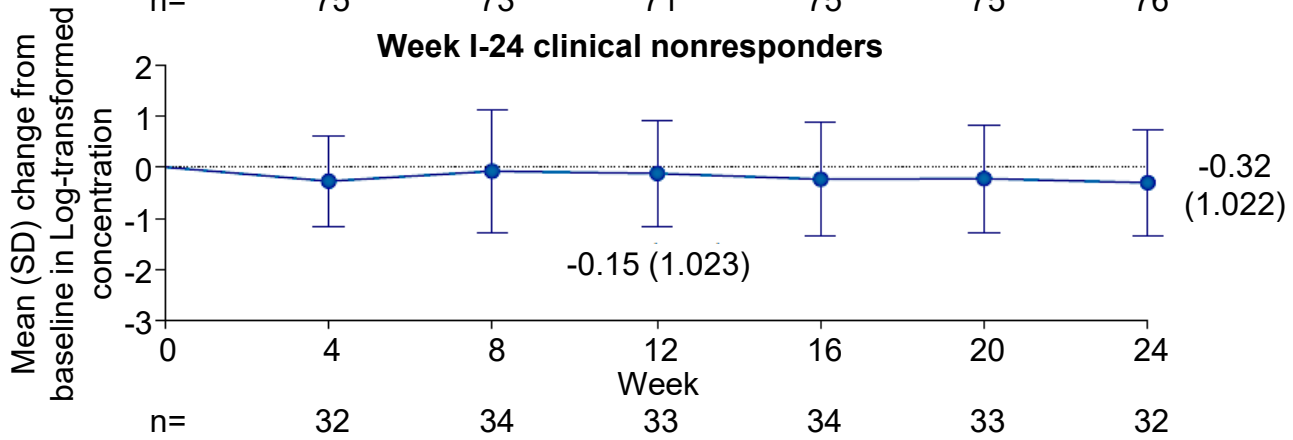
Symptomatic response: A decrease from induction baseline in the symptomatic Mayo score by ≥30% and ≥1 point, with either a ≥1-point decrease from baseline in the rectal bleeding subscore or a rectal bleeding subscore of 0 or 1; the symptomatic Mayo score is defined as the sum of the stool frequency and the rectal bleeding subscores.
Includes only participants with modified Mayo score 5–9 at induction baseline. Response groups were defined sequentially using observed IWRS data. Among 125 Week 12 non-responders, 14 discontinued before Week 24, leaving 111 classified as Week 24 responders (N=76) or Week 24 non-responders (N=35). Participants in clinical response to GUS at Week 12 were re-randomised into the QUASAR maintenance study. Participants who had an ostomy or colectomy, a prohibited change in UC medication, or discontinued study intervention for any reason (other than COVID-19–related reasons [excluding SARS-CoV-2 infection] or regional crisis) were considered not to have achieved symptomatic response at the designated timepoint.
Participants who discontinued study intervention due to COVID-19–related reasons (excluding SARS-CoV-2 infection) or regional crisis had their observed data used, if available. After accounting for these intercurrent event rules, participants who were missing stool frequency and/or rectal bleeding data at a designated timepoint were considered not to have achieved symptomatic response.
CIs for symptomatic response are based on normal approximation confidence limits.
CI, confidence interval; COVID-19, coronavirus disease 2019; GUS, guselkumab; I, induction; IL, interleukin; IV, intravenous; IWRS, Interactive Web Response System;
MoA, mechanism of action; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; SARS-CoV-2, severe acute respiratory syndrome coronavirus 2; SC, subcutaneous; UC, ulcerative colitis.
Allegretti JR, et al. Presented at ACG, 9–14 October 2026; Nashville, TN, USA. P3359. Full prescription information: swissmedicinfo-pro.ch.

Change from baseline in CRP concentration over time in Week 12 and Week 24 responders, and Week 24 non-responders

Log-transformed CRP concentration (mg/L) over time







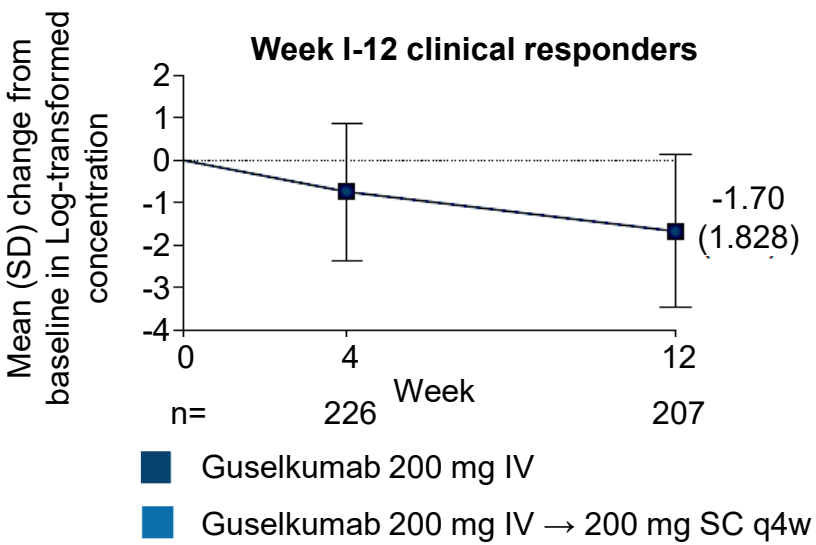
Week 24 responders showed numerically greater reductions in log-transformed CRP by Week 12 than Week 24 nonresponders

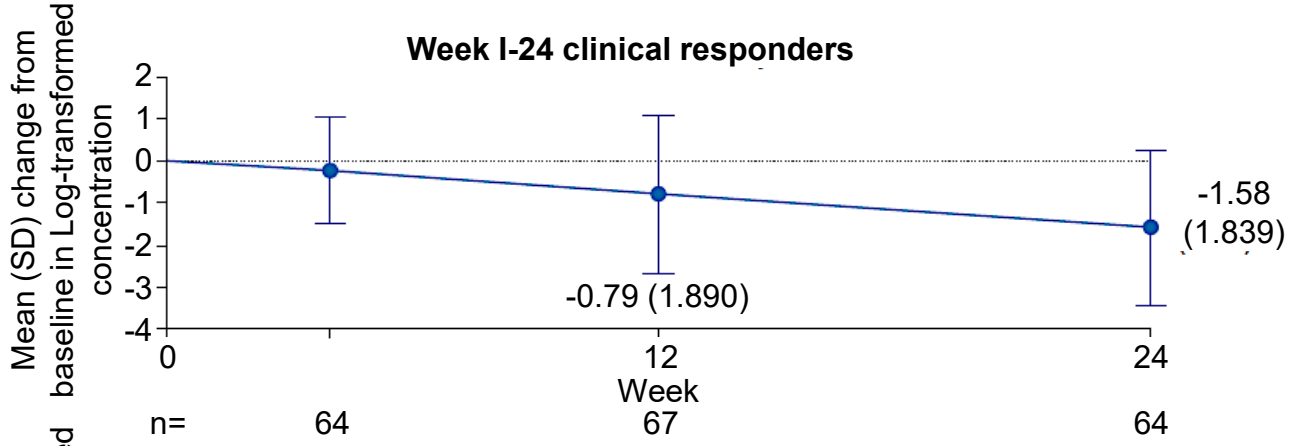
Changes in CRP are shown on a natural log scale. A decrease of 0.7 corresponds to a 50% reduction from baseline, a decrease of 1.0 corresponds to a 63% reduction from baseline, and a decrease of 2.0 corresponds to an 87% reduction from baseline. Includes only participants with modified Mayo score 5–9 at induction baseline. Response groups were defined sequentially using observed IWRS data. Among 125 Week 12 non-responders, 14 discontinued before Week 24, leaving 111 classified as Week 24 responders (N=76) or Week 24 non-responders (N=35). Participants in clinical response to GUS at Week 12 were re-randomised into the QUASAR maintenance study. Participants who had an ostomy or colectomy, a prohibited change in UC medication, or discontinued study intervention for any reason (other than COVID-19–related reasons [excluding SARS-CoV-2 infection] or regional crisis) were considered not to have achieved symptomatic response at the designated timepoint and, for CRP and fCal concentrations, had their baseline value carried forward from the time of the event onwards. Participants who discontinued study intervention due to COVID-19–related reasons (excluding SARS-CoV-2 infection) or regional crisis had their observed data used, if available. After accounting for these intercurrent event rules, participants who were missing stool frequency and/or rectal bleeding data at a designated timepoint were considered not to have achieved symptomatic response.
For CRP and fCal, change from baseline was based on the participants with evaluable data at both baseline and the designated timepoint.
COVID-19, coronavirus disease 2019; CRP, C-reactive protein; fCal, faecal calprotectin; GUS, guselkumab; I, induction; IL, interleukin; IV, intravenous; IWRS, Interactive Web Response System; MoA, mechanism of action; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; SARS-CoV-2, severe acute respiratory syndrome coronavirus 2; SC, subcutaneous; SD, standard deviation; UC, ulcerative colitis.
Allegretti JR, et al. Presented at ACG, 9–14 October 2026; Nashville, TN, USA. P3359. Full prescription information: swissmedicinfo-pro.ch.

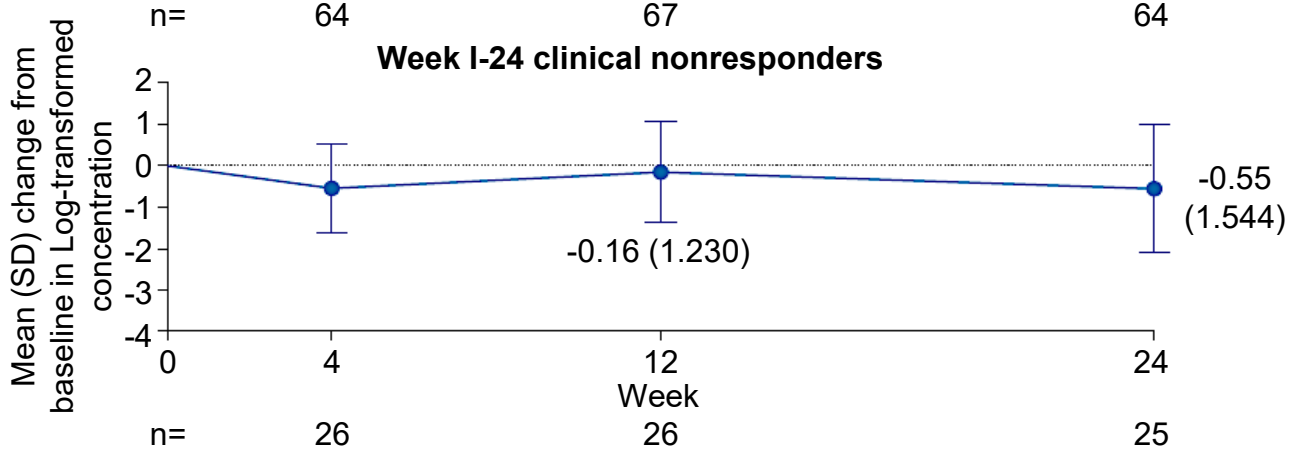
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Change from baseline in fCal concentration over time in Week 12 and Week 24 responders, and Week 24 non-responders

Log-transformed fCal concentration (mg/kg) over time







Week 24 responders showed numerically greater reductions in log-transformed fCal by Week 12 than Week 24 nonresponders

Changes in fCal are shown on a natural log scale. A decrease of 0.7 corresponds to a 50% reduction from baseline, a decrease of 1.0 corresponds to a 63% reduction from baseline, and a decrease of 2.0 corresponds to an 87% reduction from baseline. Includes only participants with modified Mayo score 5–9 at induction baseline. Response groups were defined sequentially using observed IWRS data. Among 125 Week 12 non-responders, 14 discontinued before Week 24, leaving 111 classified as Week 24 responders (N=76) or Week 24 non-responders (N=35). Participants in clinical response to GUS at Week 12 were re-randomised into the QUASAR maintenance study. Participants who had an ostomy or colectomy, a prohibited change in UC medication, or discontinued study intervention for any reason (other than COVID-19–related reasons [excluding SARS-CoV-2 infection] or regional crisis) were considered not to have achieved symptomatic response at the designated timepoint and, for CRP and fCal concentrations, had their baseline value carried forward from the time of the event onwards. Participants who discontinued study intervention due to COVID-19–related reasons (excluding SARS-CoV-2 infection) or regional crisis had their observed data used, if available. After accounting for these intercurrent event rules, participants who were missing stool frequency and/or rectal bleeding data at a designated timepoint were considered not to have achieved symptomatic response.
For CRP and fCal, change from baseline was based on the participants with evaluable data at both baseline and the designated timepoint.
COVID-19, coronavirus disease 2019; CRP, C-reactive protein; fCal, faecal calprotectin; GUS, guselkumab; I, induction; IL, interleukin; IV, intravenous; IWRS, Interactive Web Response System; MoA, mechanism of action; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; SARS-CoV-2, severe acute respiratory syndrome coronavirus 2; SC, subcutaneous; SD, standard deviation; UC, ulcerative colitis.
Allegretti JR, et al. Presented at ACG, 9–14 October 2026; Nashville, TN, USA. P3359. Full prescription information: swissmedicinfo-pro.ch.

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Conclusions

- Among participants with moderately to severely active UC without clinical response at Week 12 after guselkumab IV induction, a subset benefited after three additional GUS 200 mg SC q4w injections
- Baseline clinical characteristics did not distinguish Week 12 or Week 24 clinical response status
- However, Week 24 responders showed numerically greater reductions in log-transformed CRP and fCal by Week 12 than Week 24 non-responders, suggesting that early biomarker improvement may help to identify patients likely to respond with continued treatment

CRP, C-reactive protein; fCal, faecal calprotectin; GUS, guselkumab; IL, interleukin; IV, intravenous; MoA, mechanism of action; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous; UC, ulcerative colitis.
Allegretti JR, et al. Presented at ACG, 9–14 October 2026; Nashville, TN, USA. P3359. Full prescription information: swissmedicinfo-pro.ch.

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Time to first indication of disease progression in patients with moderately to severely active ulcerative colitis: Results from the guselkumab Phase 3 QUASAR maintenance study

P3358

Poster presented at ACG 2026

Key data only

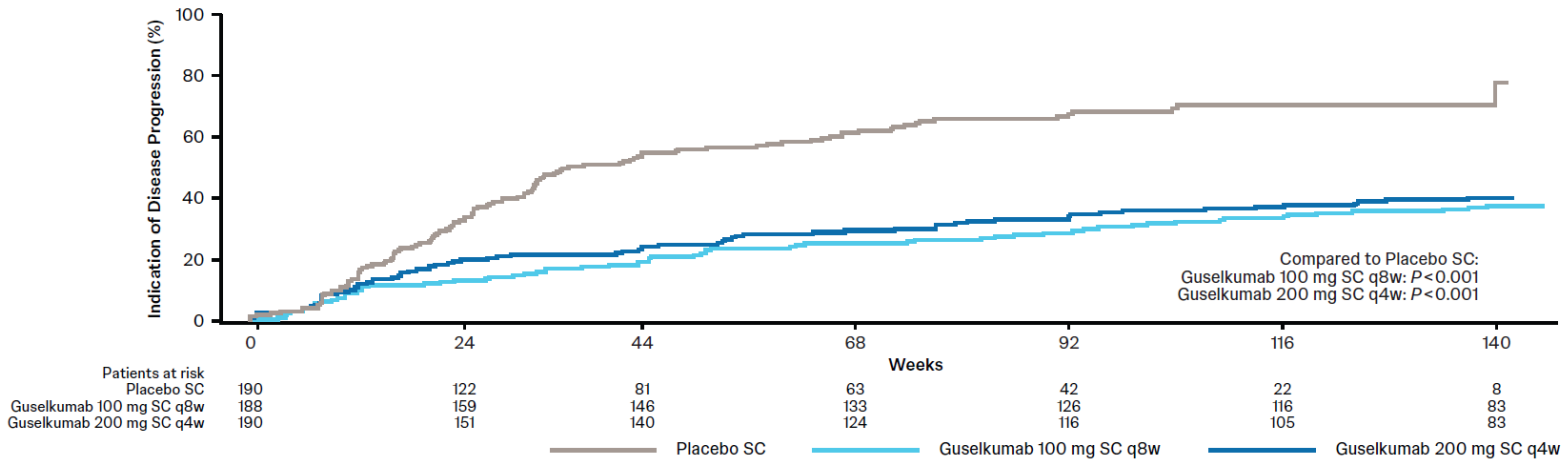
Jessica R. Allegretti,[1] Brian Bressler,[2] Tadakazu Hisamatsu,[3] Bruce E. Sands,[4] Yelina Alvarez,[5] Thomas Baker,[5] Shashi Adsul,[5] Ye Miao,[5] Hongyan Zhang,[5] David T. Rubin,[6] Axel Dignass[7]

[1]Division of Gastroenterology, Hepatology, and Endoscopy, Brigham and Women's Hospital, Harvard Medical School, Boston, MA, USA; [2]University of British Columbia, Vancouver, BC, Canada; [3]Kyorin University, Tokyo, Japan; [4]Dr. Henry D. Janowitz Division of Gastroenterology, Icahn School of Medicine at Mount Sinai, New York, NY, USA; [5]Johnson & Johnson, Spring House, PA, USA; [6]University of Chicago Medicine Inflammatory Bowel Disease Center, Chicago, IL, USA; [7]Department of Medicine I, Agaplesion Markus Hospital, Goethe University, Frankfurt, Germany

CH_CP-609070 10//26

Aim: Post hoc analysis to assess time to first indication of disease progression over approximately 3 years in the Phase 3 QUASAR maintenance study and LTE

Time to first indication of disease progression in each GUS maintenance treatment group was longer than in the placebo group and appeared similar for both GUS doses (analysis included all patients)*

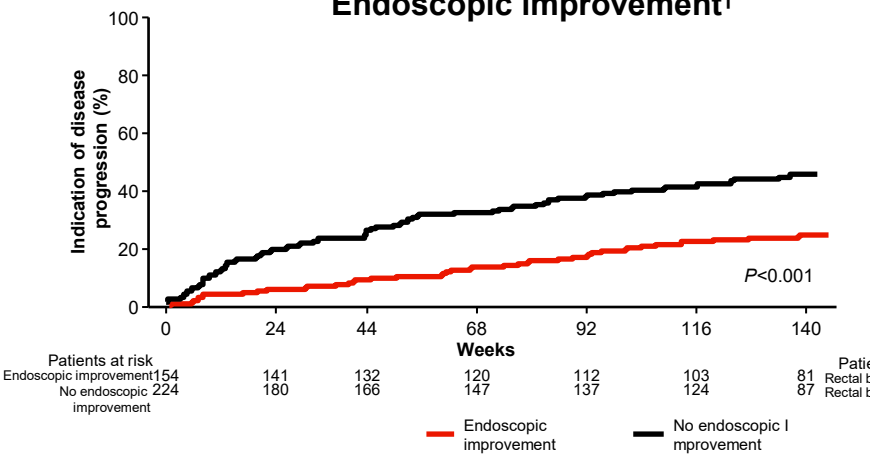


Conclusions

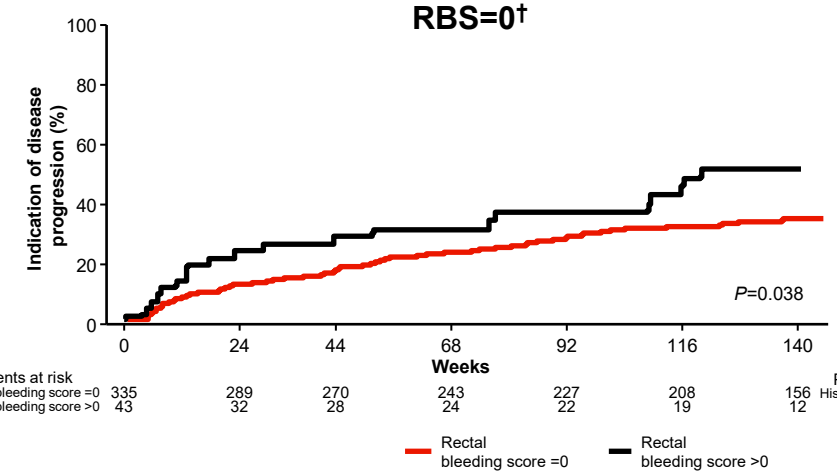
- This *post hoc* analysis of the QUASAR study showed that both GUS maintenance doses delayed disease progression, suggesting potential disease modification in patients with moderately to severely active UC through approximately 3 years of maintenance treatment
- The data further suggest that among patients receiving GUS maintenance, disease progression occurred later in patients who had achieved rectal bleeding resolution, endoscopic improvement or histologic remission after GUS induction

Time to first indication of disease progression among patients who achieved each respective endpoint after GUS induction

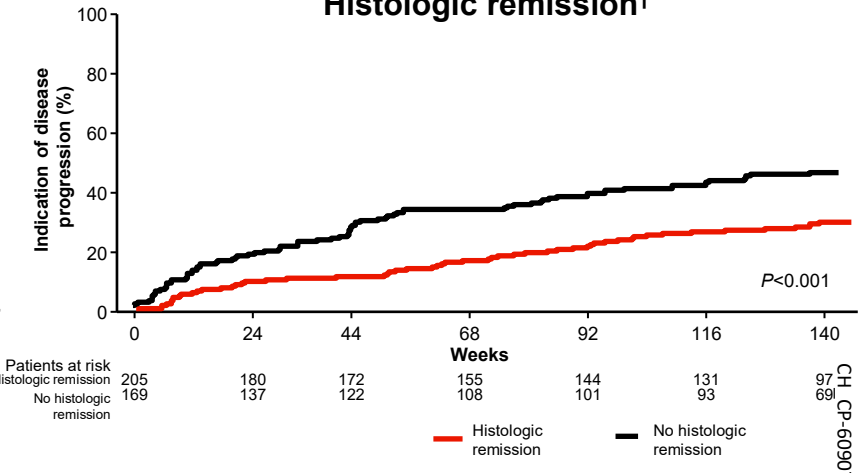
Endoscopic improvement†



RBS=0†



Histologic remission†



*Nominal p-values are based on stratified log-rank test, adjusting for clinical remission status at Week M-0 (yes vs no) and induction treatment (GUS 400 mg IV, GUS 200 mg IV, placebo IV crossover to GUS 200 mg IV). †Nominal p-values are based on log-rank test. **Endoscopic improvement:** a Mayo endoscopy subscore of 0 or 1 with no friability. **Histologic remission:** absence of neutrophils from the mucosa (both lamina propria and epithelium); no crypt destruction; and no erosions, ulcerations or granulation tissue according to the Geboes grading system.

GUS, guselkumab; IL, interleukin; IV, intravenous; LTE, long-term extension; M, maintenance; MoA, mechanism of action; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous; UC, ulcerative colitis.

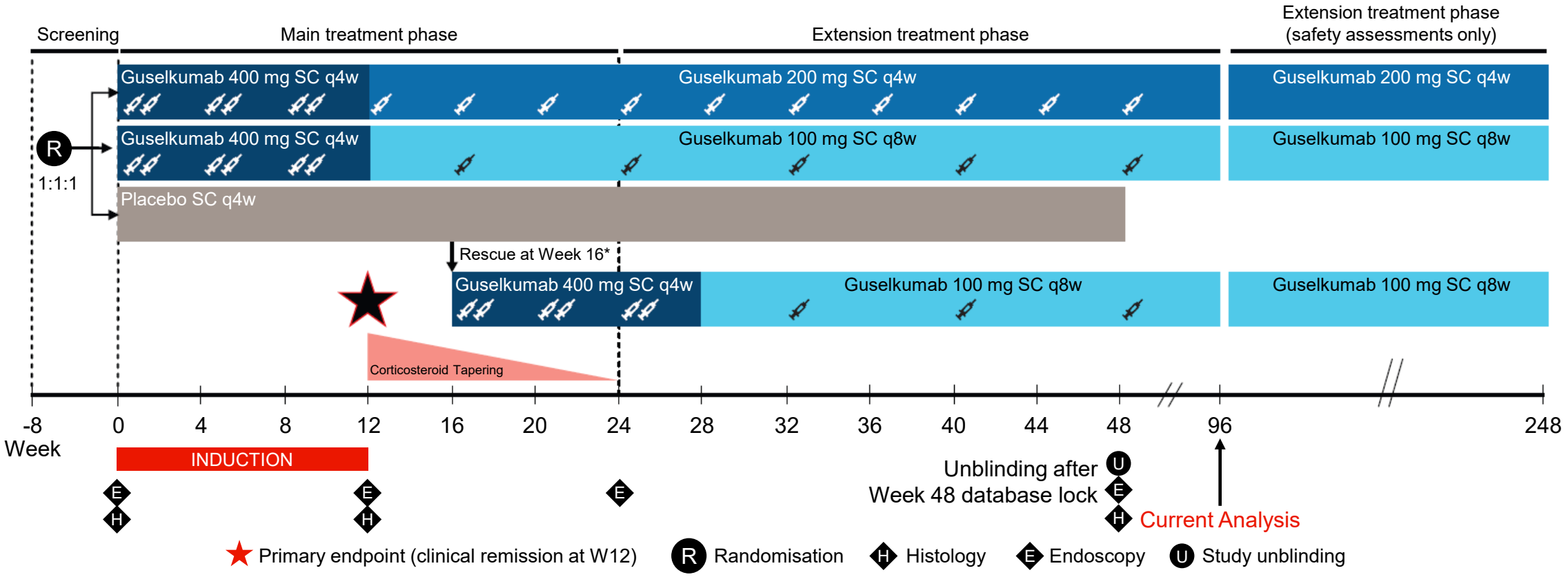
Allegretti JR, et al. Presented at ACG, 9–14 October 2026; Nashville, TN, USA. P3358. Full prescription information: swissmedicinfo-pro.ch.

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ASTRO: Guselkumab for UC

Phase 3, randomised, double-blind, placebo-controlled, treat-through design: ASTRO

Stratified randomisation
- BIO/JAKi/S1Pi-IR status: yes or no
- Mayo endoscopic subscore at baseline: moderate (2) or severe (3)



BIO, biologic; E, endoscopy; GUS, guselkumab; H, histology; IL, interleukin; IR, inadequate response or intolerance; JAKi, Janus kinase inhibitor; MoA, mechanism of action; qXw, every X weeks; R, randomisation; RDBPC, randomised, double-blind, placebo-controlled; S1Pi, sphingosine-1-phosphate receptor modulator; SC, subcutaneous; U, study unblinding; UC, ulcerative colitis.

Long M, et al. Presented at ACG, 9–14 October 2026; Nashville, TN, USA. P1177. Full prescription information: swissmedicinfo-pro.ch.

CH_CP-609070 10//26

The efficacy and safety of guselkumab in patients with moderate to severe ulcerative colitis through 2 years: ASTRO Week 96 results

P1177

Poster presented at ACG

Also presented as an encore at UEGW 2026 (MP0297)

M. Long,[1] L. Peyrin-Biroulet,[2] T. Hisamatsu,[3] J. R. Allegretti,[4] Y. Alvarez,[5] T. Baker,[5] K. Hertzog,[5] C. Han,[6] H. Zhang,[5] Y. Miao,[5] D. T. Rubin,[7] S. Danese[8]

[1]Division of Gastroenterology and Hepatology, University of North Carolina at Chapel Hill, Chapel Hill, NC, USA; [2]Northwestern Medicine Digestive Health Institute, Chicago, IL, USA; [3]Department of Gastroenterology and Hepatology, Kyorin University School of Medicine, Tokyo, Japan; [4]Division of Gastroenterology, Hepatology, and Endoscopy, Brigham and Women's Hospital, Harvard Medical School, Boston, MA, USA; [5]Johnson & Johnson, Spring House, PA, USA; [6]Johnson & Johnson, Malvern, PA, USA; [7]University of Chicago Medicine Inflammatory Bowel Disease Centre, Chicago, IL, USA; [8]Gastroenterology and Endoscopy, IRCCS Ospedale San Raffaele and University Vita-Salute San Raffaele, Milan, Italy

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Demographics and baseline disease characteristics

	Placebo	GUS 400 mg SC → 100 mg SC q8w	GUS 400 mg SC → 200 mg SC q4w
Full analysis set*, N	139	139	140
Age in years, mean (SD)	39.5 (13.6)	42.1 (14.6)	43.6 (14.3)
Male, n (%)	90 (64.7%)	79 (56.8%)	87 (62.1%)
UC disease duration in years, mean (SD)	6.61 (6.2)	8.39 (7.3)	7.69 (6.4)
Modified Mayo score† (0–9), mean (SD)	6.8 (1.1)‡	6.8 (1.2)	6.6 (1.2)
Modified Mayo score of 7–9 (severe), n (%)	87 (63.0%)‡	95 (68.3%)	77 (55.0%)
Mayo endoscopic subscore of 3 (severe), n (%)	78 (56.1%)	78 (56.1%)	78 (55.7%)
Extensive UC, n (%)	73 (52.5%)	69 (49.6%)	82 (58.6%)
C-reactive protein§ median in mg/L (IQR)	3.8 (1.2; 10.9)	3.7 (1.3; 7.2)	4.7 (1.7; 9.1)
C-reactive protein§ >3 mg/L, n (%)	77 (55.8%)	75 (55.1%)	86 (61.4%)
Faecal calprotectin¶ median in mg/kg (IQR)	1749.0 (617.0; 3202.0)	1351.5 (609.0; 2805.0)	1594.0 (838.0; 3336.0)
Faecal calprotectin¶ >250 mg/kg, n (%)	119 (90.8%)	107 (84.9%)	119 (93.0%)

*All randomised patients who received at least one (partial or complete) dose of study intervention. †Modified Mayo score: three-component (stool frequency, rectal bleeding and endoscopy subscores) Mayo score without the physician's global assessment. ‡Based on n=138. §Based on n=138 for placebo, n=136 for GUS 400 mg SC→100 mg SC q8w, n=140 for GUS 400 mg SC→200 mg SC q4w. ¶Based on n=131 for placebo, n=126 for GUS 400 mg SC→100 mg SC q8w, n=128 for GUS 400 mg SC→200 mg SC q4w.
GUS, guselkumab; IL, interleukin; IQR, interquartile range; MoA, mechanism of action; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous; SD, standard deviation; UC, ulcerative colitis.
Long M, et al. Presented at ACG, 9–14 October 2026; Nashville, TN, USA. P1177. Full prescription information: swissmedicinfo-pro.ch.

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Treatment disposition after Week 48 through Week 96

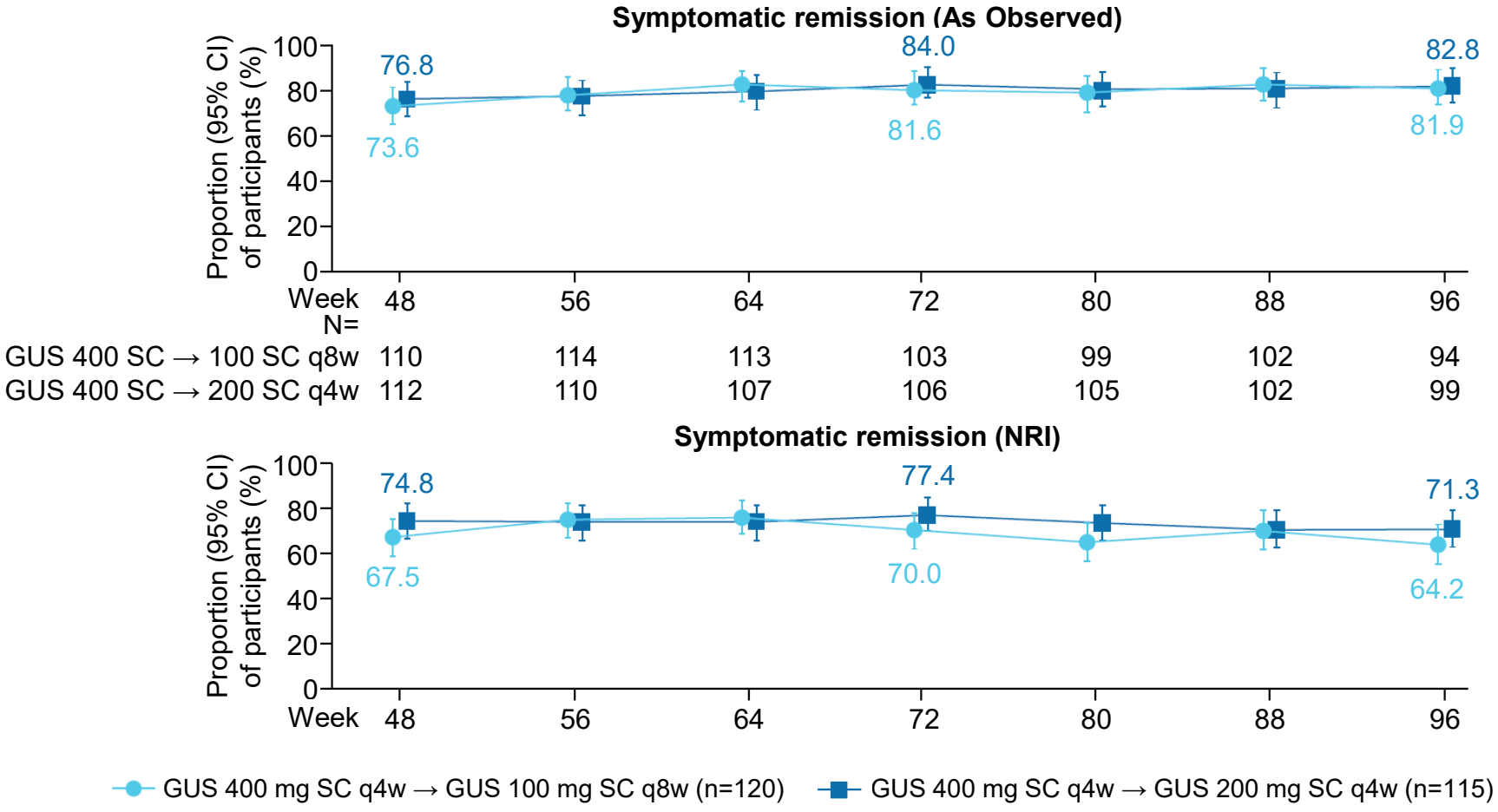
	Placebo SC	GUS 400 mg SC q4w → GUS 100 mg SC q8w	GUS 400 mg SC q4w → GUS 200 mg SC q4w	Total
Safety post Week 48 extension analysis set*, N	86	120	115	321
Patients who discontinued study treatment prior to Week 96, n (%)	47 (54.7%)	15 (12.5%)	9 (7.8%)	71 (22.1%)
Reason for discontinuation, n (%)				
Adverse event – worsening of UC	0	1 (0.8%)	1 (0.9%)	2 (0.6%)
Adverse event – other than worsening of UC	1 (1.2%)	1 (0.8%)	2 (1.7%)	4 (1.2%)
Lack of efficacy	5 (5.8%)	6 (5.0%)	4 (3.5%)	15 (4.7%)
Withdrawal by participant	3 (3.5%)	7 (5.8%)	2 (1.7%)	12 (3.7%)
Week 48 unblinding	38 (44.2%)	0	0	38 (11.8%)
Lost to follow-up	0	0	0	0
Death	0	0	0	0
Initiated prohibited medication	0	0	0	0
Protocol deviation	0	0	0	0
Other	0	0	0	0

- A total of 235 patients who were randomised to GUS continued treatment after Week 48

*Patients who received at least one dose of study intervention (including a partial dose) at or after Week 48.
GUS, guselkumab; IL, interleukin; MoA, mechanism of action; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous; UC, ulcerative colitis; W, Week.
Long M, et al. Presented at ACG, 9–14 October 2026; Nashville, TN, USA. P1177. Full prescription information: swissmedicinfo-pro.ch.

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Symptomatic remission was maintained from Week 48 through Week 96 in the GUS 100 mg SC q8w and GUS 200 mg SC q4w treatment arms

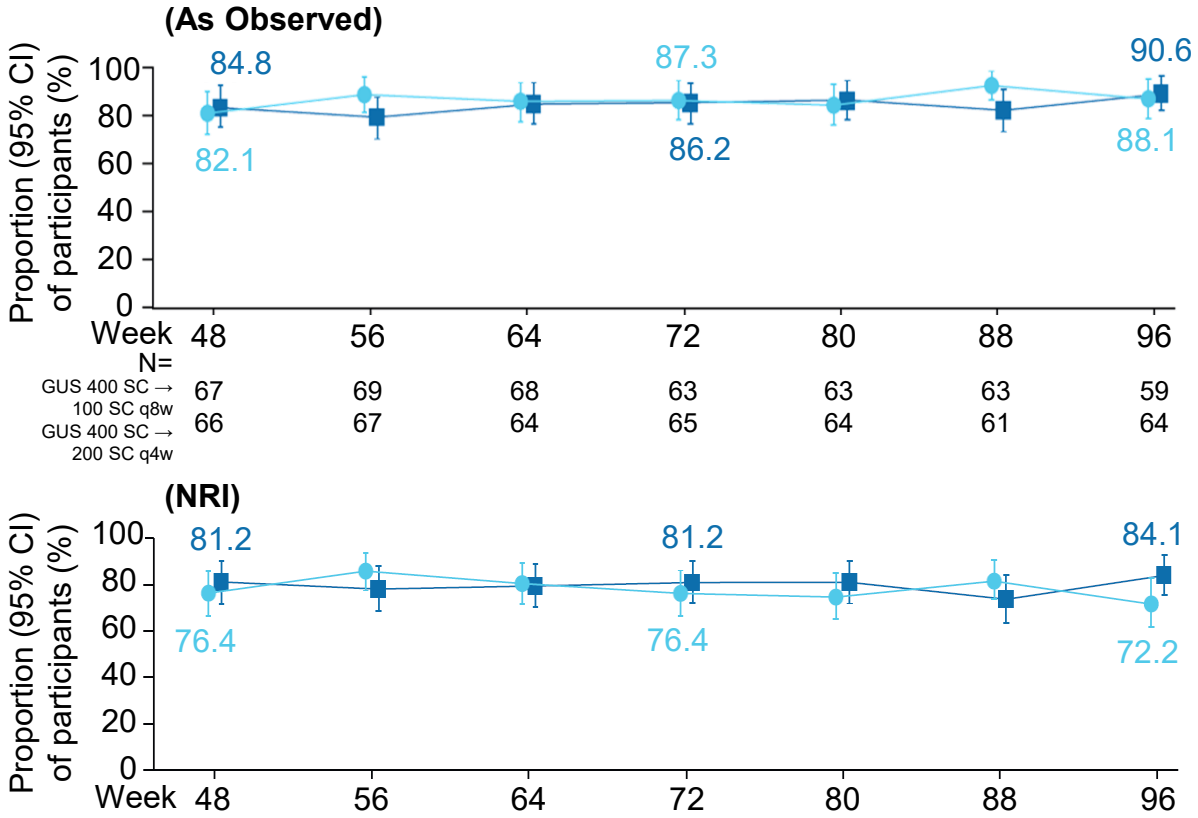


Symptomatic remission: a stool frequency subscore of 0 or 1 and not increased from baseline, and a rectal bleeding subscore of 0 as assessed by two PRO questions from Mayo score.
CI, confidence interval; GUS, guselkumab; IL, interleukin; MoA, mechanism of action; NRI, non-responder imputation; PRO, patient-reported outcome; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous; UC, ulcerative colitis.
Long M, et al. Presented at ACG, 9–14 October 2026; Nashville, TN, USA. P1177. Full prescription information: swissmedicinfo-pro.ch.

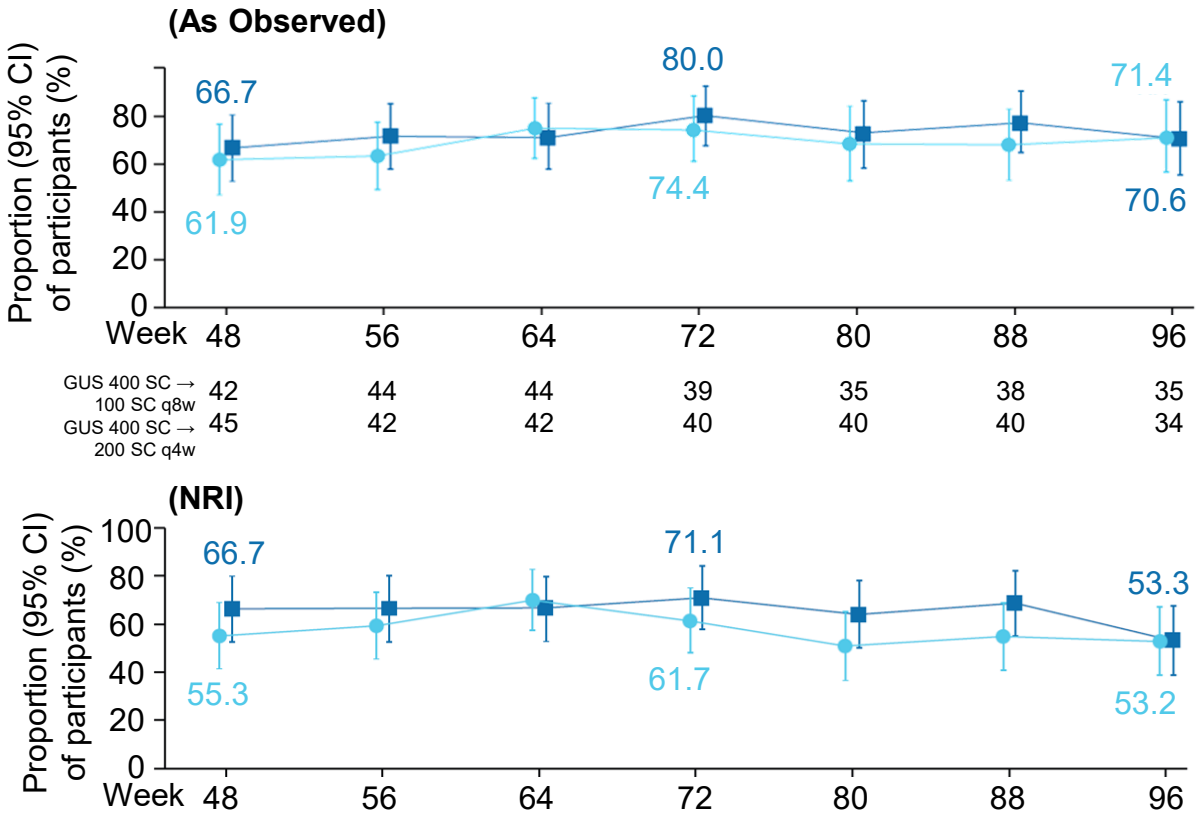
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Maintenance of symptomatic remission was generally similar in the ADT-naïve and ADT-IR subgroups

Symptomatic remission from Week 48 to Week 96 among ADT-naïve patients



Symptomatic remission from Week 48 to Week 96 among ADT-IR patients



GUS 400 mg SC q4w → GUS 100 mg SC q8w (n=47) GUS 400 mg SC q4w → GUS 200 mg SC q4w (n=45)

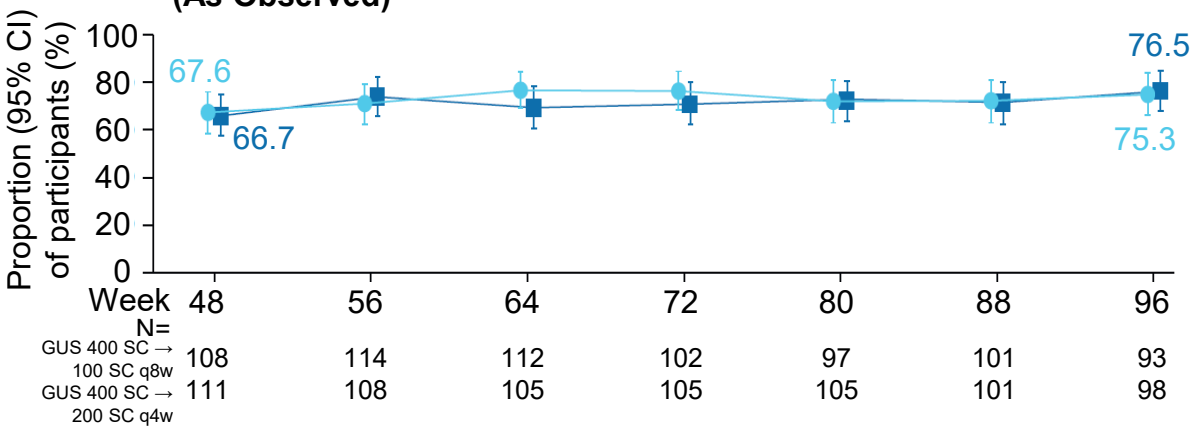
Symptomatic remission: a stool frequency subscore of 0 or 1 and not increased from baseline, and a rectal bleeding subscore of 0 as assessed by two PRO questions from Mayo score.
ADT, advanced therapy; CI, confidence interval; GUS, guselkumab; IL, interleukin; IR, inadequate response or intolerance; MoA, mechanism of action; NRI, non-responder imputation; PRO, patient-reported outcome; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous; UC, ulcerative colitis.
Long M, et al. Presented at ACG, 9–14 October 2026; Nashville, TN, USA. P1177. Full prescription information: swissmedicinfo-pro.ch.

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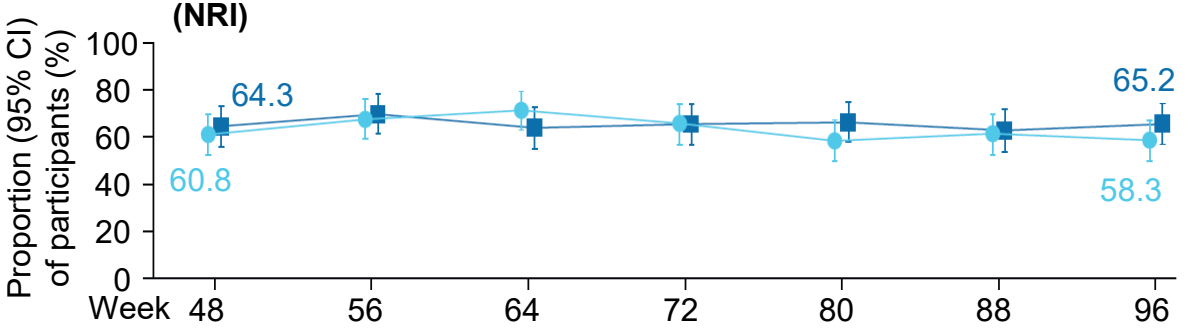
The absence of bowel urgency and abdominal pain was maintained from Week 48 through Week 96

No bowel urgency

(As Observed)

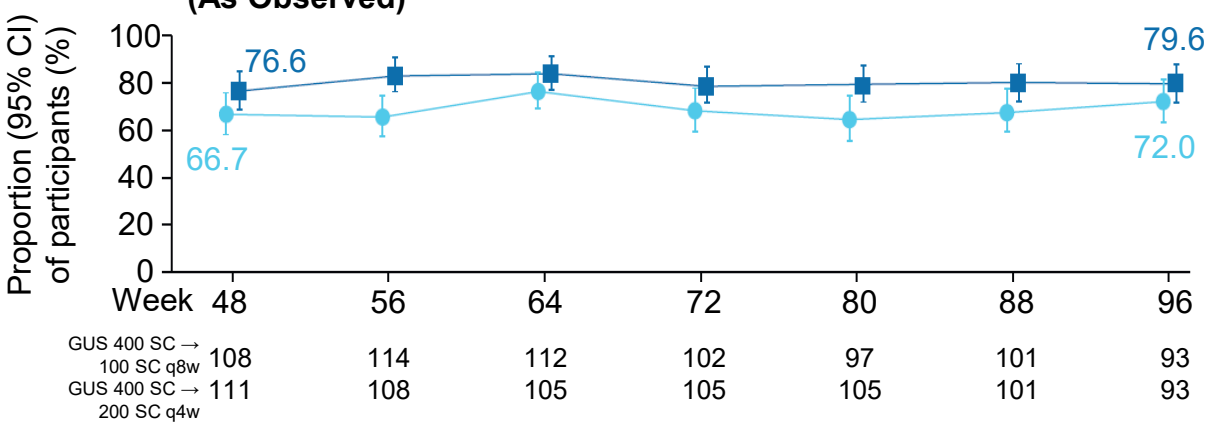


(NRI)

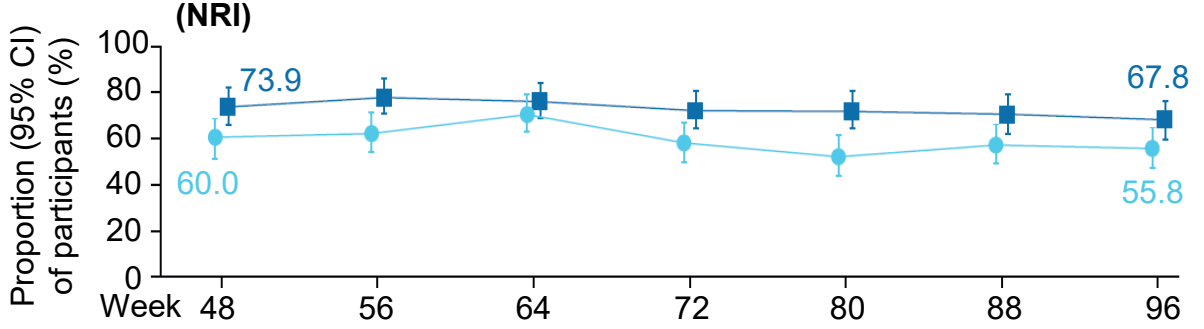


No abdominal pain

(As Observed)



(NRI)

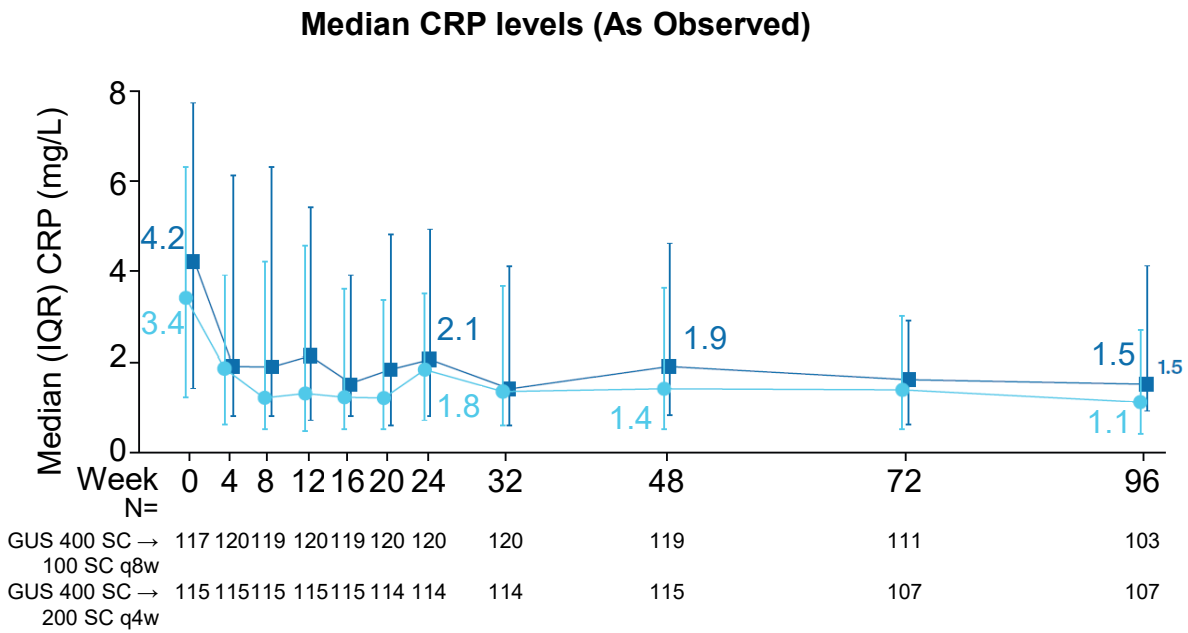


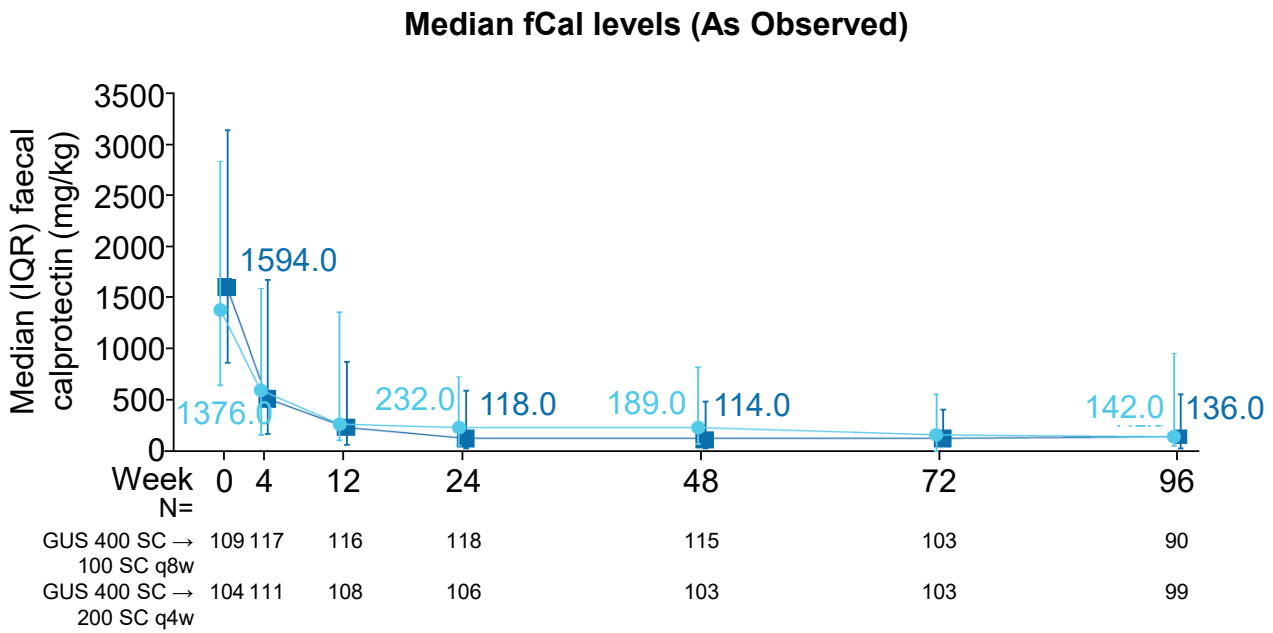
GUS 400 mg SC q4w → GUS 100 mg SC q8w (n=120) — GUS 400 mg SC q4w → GUS 200 mg SC q4w (n=115)

No bowel urgency: defined as a rounded daily average (within 1 week) score of 0 in question 7 of the UC-PRO/SS. *Q7: In the past 24 hours, did you feel the need to have a bowel movement right away?*
No abdominal pain: defined as a rounded daily average (within 1 week) score of 0 in question 8 of the UC-PRO/SS. *Q8: In the past 24 hours, did you feel pain in your belly?*
CI, confidence interval; GUS, guselkumab; IL, interleukin; MoA, mechanism of action; NRI, non-responder imputation; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous; UC, ulcerative colitis; UC-PRO/SS, Ulcerative Colitis Patient-Reported Outcomes Signs and Symptoms.
Long M, et al. Presented at ACG, 9–14 October 2026; Nashville, TN, USA. P1177. Full prescription information: swissmedicinfo-pro.ch.

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Median CRP levels and median fCal levels were maintained over time from Week 48 through Week 96





GUS 400 mg SC q4w → GUS 100 mg SC q8w

GUS 400 mg SC q4w → GUS 200 mg SC q4w

CRP, C-reactive protein; fCal, faecal calprotectin; GUS, guselkumab; IL, interleukin; IQR, interquartile range; MoA, mechanism of action; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous; UC, ulcerative colitis.

Long M, et al. Presented at ACG, 9–14 October 2026; Nashville, TN, USA. P1177. Full prescription information: swissmedicinfo-pro.ch.

CH_CP-609070 10//26

Key safety findings in all treated patients from Week 0 through Week 96

TEAEs per 100 PY through Week 96 following randomised GUS 100 mg SC q8w and GUS 200 mg SC q4w vs placebo SC

	Placebo SC (n=139)	GUS 400 mg SC q4w → GUS 100 mg SC q8w (n=139)	GUS 400 mg SC q4w → GUS 200 mg SC q4w (n=140)
Average duration of follow-up (weeks)	40.3	86.1	83.5
Total PY of follow-up	107.4	229.4	224.0
Number of events per 100 PY of follow up (95% CI)			
AEs	289.45 (258.17, 323.48)	266.34 (245.64, 288.32)	274.05 (252.80, 296.61)
Serious AEs	30.71 (21.14, 43.13)	5.67 (3.02, 9.69)	6.70 (3.75, 11.04)
AEs leading to discontinuation of study agent	14.89 (8.51, 24.18)	3.05 (1.23, 6.29)	3.57 (1.54, 7.04)
Infections*	67.01 (52.43, 84.39)	75.85 (65, 87.99)	62.93 (52.97, 74.22)
Serious infections*	2.79 (0.58, 8.16)	0.87 (0.11, 3.15)	1.34 (0.28, 3.91)
Death	0.93 (0.02, 5.19)	0.44 (0.01, 2.43)	0 (0, 1.34)

*Infections are based on MedDRA system organ class 'Infections and infestations'.
AE, adverse event; CI, confidence interval; GUS, guselkumab; IL, interleukin; MedDRA, Medical Dictionary for Regulatory Activities; MoA, mechanism of action; PY, patient-years; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous; TEAE, treatment-emergent adverse event; UC, ulcerative colitis.
Long M, et al. Presented at ACG, 9–14 October 2026; Nashville, TN, USA. P1177. Full prescription information: swissmedicinfo-pro.ch.

CH_CP-609070 10//26

Conclusions

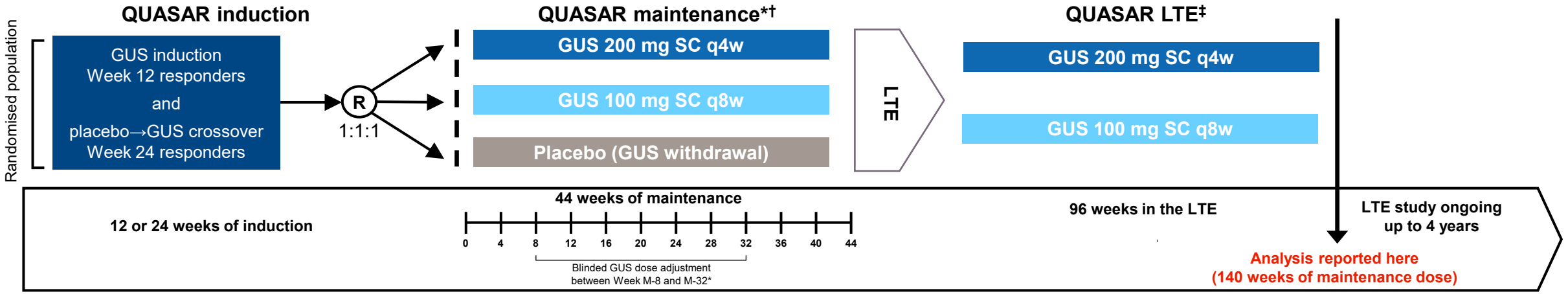
- GUS SC maintenance therapy showed sustained symptomatic and inflammatory biomarker improvements among patients who continued treatment after Week 48
- GUS SC maintenance therapy also maintained no bowel urgency or abdominal pain among patients who continued treatment after Week 48
- No new safety concerns were identified; safety data remain consistent with the well-characterised profile of GUS in its approved indications

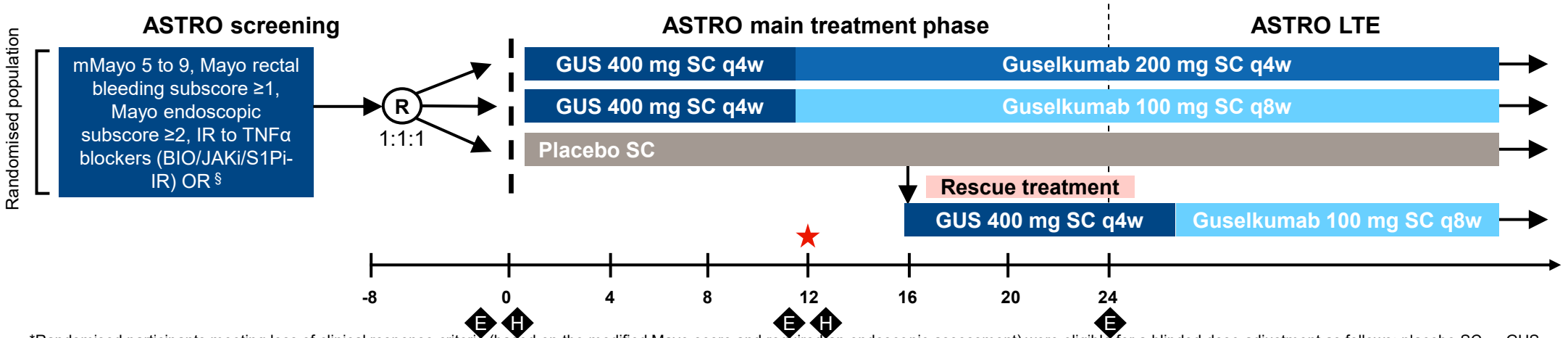
GUS, guselkumab; IL, interleukin; MoA, mechanism of action; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous; UC, ulcerative colitis.
Long M, et al. Presented at ACG, 9–14 October 2026; Nashville, TN, USA. P1177. Full prescription information: swissmedicinfo-pro.ch.

CH_CP-609070 10//26

QUASAR/ASTRO: Guselkumab for UC

QUASAR and ASTRO study designs[1,2]





*Randomised participants meeting loss of clinical response criteria (based on the modified Mayo score and required an endoscopic assessment) were eligible for a blinded dose adjustment as follows: placebo SC → GUS 200 mg SC q4w; GUS 100 mg SC q8w → GUS 200 mg SC q4w; GUS 200 mg SC q4w → GUS 200 mg SC q4w (sham adjustment); †Participants who completed the safety and efficacy evaluations (including the required endoscopy procedure) at maintenance Week M-44 and who may benefit from continued study intervention, in the opinion of the investigator, had the opportunity to participate in the LTE of the Maintenance Study; ‡The study blinding was maintained during the LTE until the last participant in the Maintenance Study completed the M-44 visit. After the Maintenance Study was unblinded, participants had their study visits scheduled to coincide with their dose regimen (either q4w or q8w) and participants receiving placebo were terminated from study participation; §Naïve to BIO/JAKi/S1Pi (or exposed to BIO/JAKi/S1Pi without IR) and IR to corticosteroids, 6-MP, or AZA.
BIO, biologic; CD, Crohn's disease; E, endoscopy; GUS, guselkumab; H, histology; IL, interleukin; IR, inadequate response or intolerance; IV, intravenous; JAKi, Janus kinase inhibitor; LTE, long-term extension; M, maintenance; MES, Mayo Endoscopic Subscore; mMayo, modified Mayo score; MOA, mechanism of action; q4w, every 4 weeks; q8w, every 8 weeks; R, randomisation; RDBPC, randomised, double-blind, placebo-controlled; S1Pi, sphingosine-1-phosphate receptor modulator; SC, subcutaneous; TNFα, tumour necrosis factor alpha; UC, ulcerative colitis.

1. Rubin DT, et al. Presented at DDW, Chicago, IL, USA, 2–5 May 2026. Mo1516; 2. Peyrin-Biroulet L, et al. Presented at ECCO, Berlin, Germany, 19–22 February 2025. OP10. Full prescription information: swissmedicinfo-pro.ch.

CH_CP-609070 10/26

Pharmacokinetics and exposure-response relationships of subcutaneous guselkumab maintenance in participants with moderately to severely active ulcerative colitis

OP243

Oral presentation at UEGW

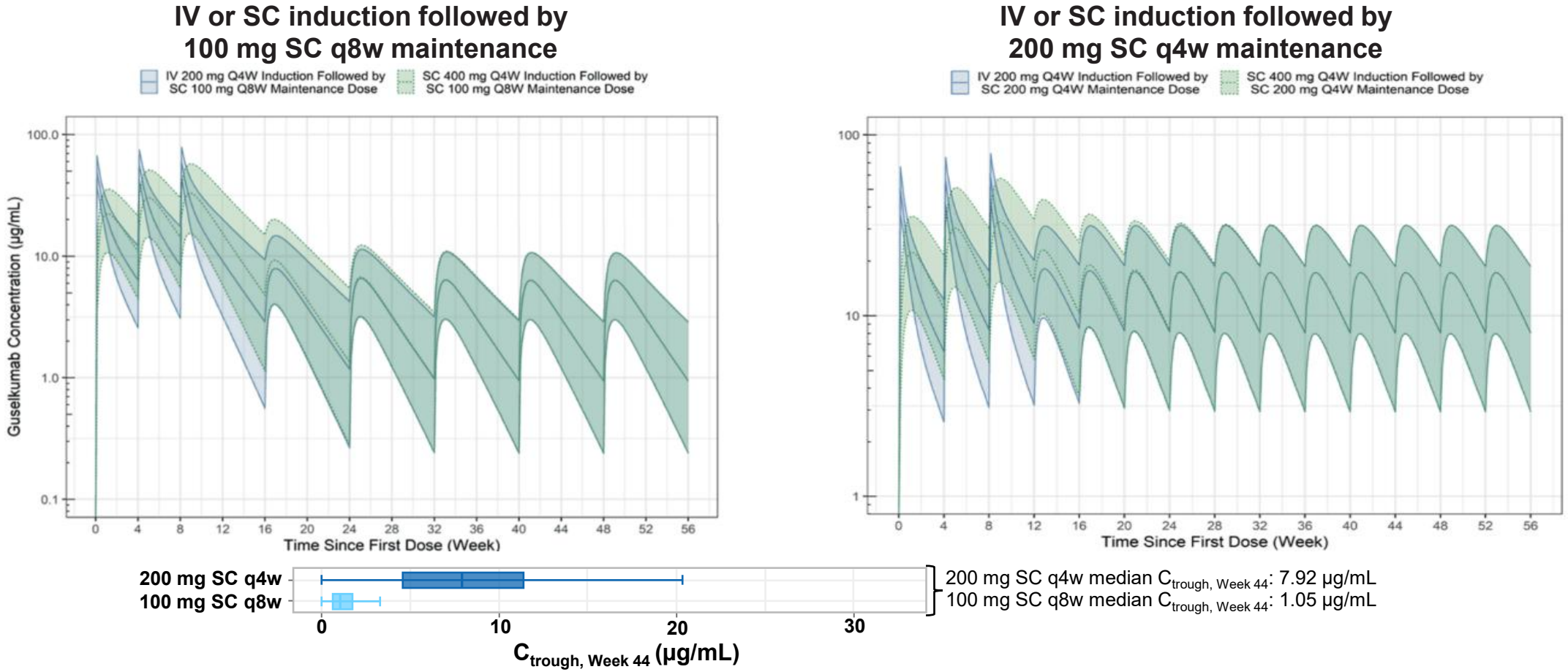
Also presented as a poster at ACG 2026 (P3334)

David T Rubin,[1] Laurent Peyrin-Biroulet,[2] Jie Shao,[3] Yu Kyoung Cho,[3] Jafar Sadik B Shaik,[3] Millie Long,[4] Silvio Danese,[5] Yelina Alvarez,[3] Marion L. Vetter,[3] Thomas Baker,[3] Jessica R Allegretti,[6] Tadakazu Hisamatsu[7]

[1]University of Chicago Medicine Inflammatory Bowel Disease Centre, Chicago, IL, USA; [2]Northwestern Medicine Digestive Health Institute, Chicago, IL, USA; [3]Johnson & Johnson, Spring House, PA, USA; [4]Division of Gastroenterology and Hepatology, University of North Carolina at Chapel Hill, Chapel Hill, NC, USA; [5]Gastroenterology and Endoscopy, IRCCS San Raffaele Hospital and Vita-Salute San Raffaele University, Milan, Italy; [6]Division of Gastroenterology, Hepatology and Endoscopy, Brigham and Women's Hospital, Harvard Medical School, Boston, MA, USA; [7]Department of Gastroenterology and Hepatology, Kyorin University, Tokyo, Japan

CH_CP-609070 10//26

Simulated pharmacokinetic profiles: Maintenance concentrations within each dose were similar regardless of induction route, with steady state achieved by Week 24

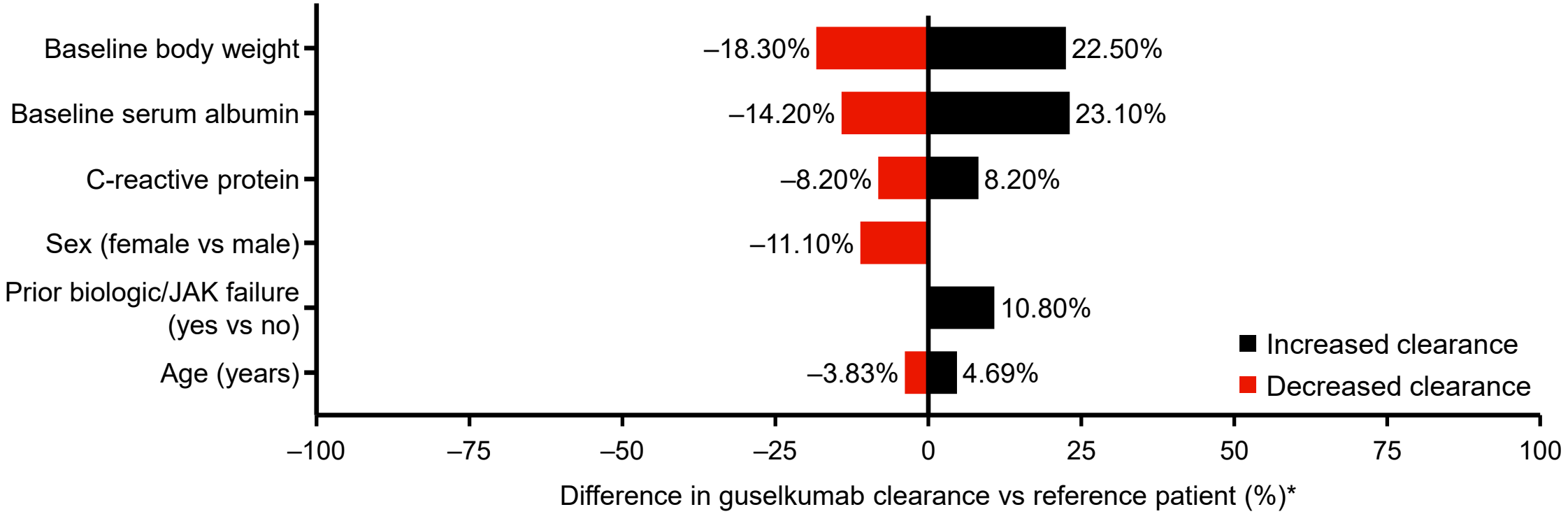


The two maintenance regimens provide a 4-fold dose range, which results in an ~8-fold range in steady-state trough concentrations

The solid line represents the median and the shaded area represents the 90% prediction interval.
GUS, guselkumab; IL, interleukin; IV, intravenous; MOA, mechanism of action; PK, pharmacokinetics; qXw, every X weeks; SC, subcutaneous; UC, ulcerative colitis.
Rubin DT, et al. Presented at UEGW, Barcelona, Spain, 17–20 October 2026. OP243. Full prescription information: swissmedicinfo-pro.ch.

CH_CP-609070 10//26

Parameter estimates in the population PK model



Covariate effects were modest relative to exposure differences between maintenance regimens, and were insufficient to justify covariate-adjusted dosing, supporting the use of fixed doses. Patients who require higher exposure may benefit from the 200 mg q4w dose

*The magnitude of change in the parameter estimate caused by a continuous covariate was expressed as a range, i.e., % change from the median value when the covariate factor varied from the 5th percentile (red) to the 95th percentile (black) of the population.

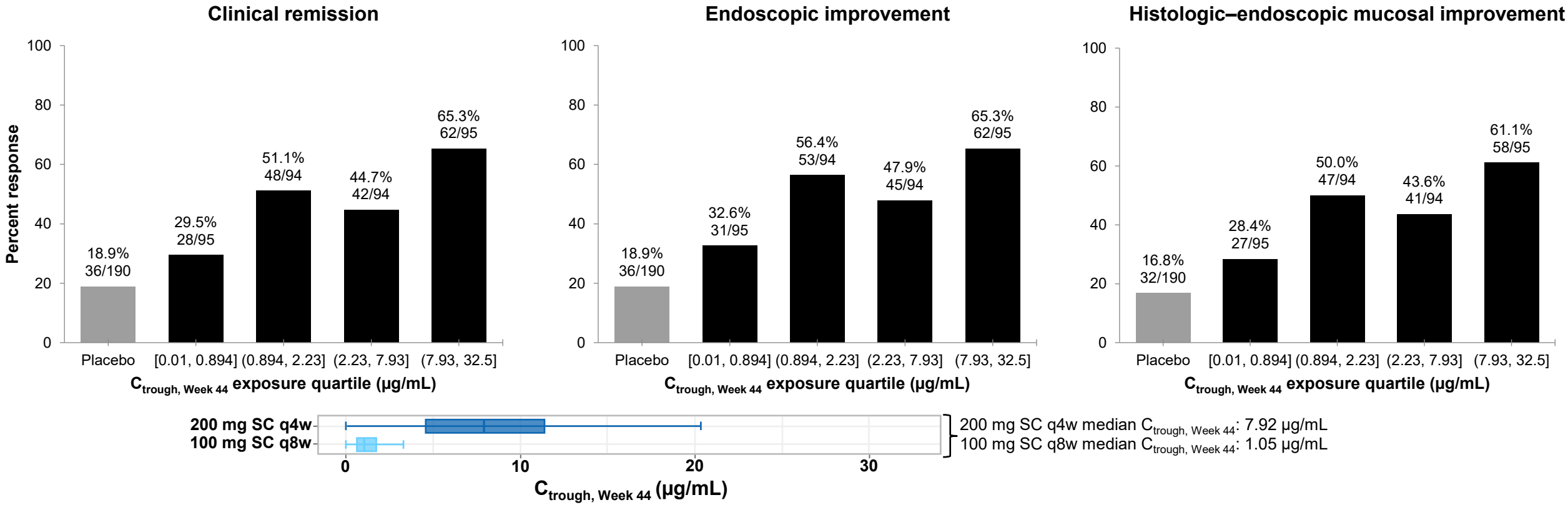
$$\mathrm{CL}\left(\frac{\mathrm{L}}{\mathrm{day}}\right) = 0.312 \times \left(\frac{\mathrm{BWT}}{70}\right)^{0.538} \times \left(\frac{\mathrm{ALB}}{43}\right)^{-1.17} \times \left(\frac{\mathrm{CRP}}{4.28}\right)^{0.0365} \times \left(\frac{\mathrm{AGE}}{39}\right)^{-0.0741} \times (1.108)^{\mathrm{FBIO}} \times (0.889)^{\mathrm{SEX}}$$

ALB, albumin; BWT, bodyweight; CL, clearance; CRP, C-reactive protein; FBIO, prior biologic exposure; GUS, guselkumab; IL, interleukin; JAK, Janus kinase; MOA, mechanism of action; PK, pharmacokinetics; qXw, every X weeks; UC, ulcerative colitis.

Rubin DT, et al. Presented at UEGW, Barcelona, Spain, 17–20 October 2026. OP243. Full prescription information: swissmedicinfo-pro.ch.

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Guselkumab exposure–response at maintenance Week 44



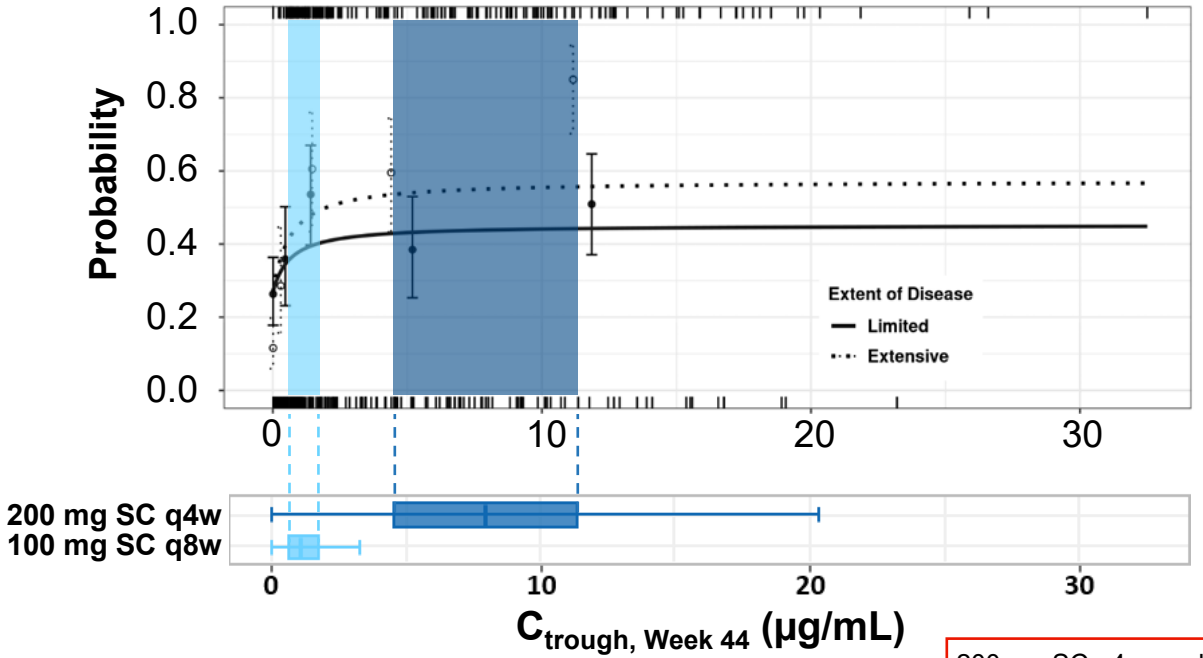
E–R analyses showed that higher serum GUS concentrations during maintenance were associated with greater efficacy at maintenance Week 44 regardless of clearance or half-life, suggesting that some participants may benefit from higher exposure

Clinical remission: Stool frequency subscore of 0 or 1 (with no increase from induction baseline), a rectal bleeding subscore of 0, and an endoscopic subscore of 0 or 1 with no friability present on the endoscopy.
Endoscopic improvement: Endoscopic subscore of 0 or 1 with no friability present on the endoscopy. **HEMI:** Histological improvement and endoscopic improvement. **Histological improvement:** Neutrophil infiltration in <5% of crypts, no crypt destruction, and no erosions, ulcerations, or granulation tissue according to the Geboes grading system.
C_{trough}, trough serum concentration; E–R, exposure–response; GUS, guselkumab; HEMI, histologic–endoscopic mucosal improvement; IL, interleukin; MOA, mechanism of action; PK, pharmacokinetics; qXw, every X weeks; SC, subcutaneous; UC, ulcerative colitis.
Rubin DT, et al. Presented at UEGW, Barcelona, Spain, 17–20 October 2026. OP243. Full prescription information: swissmedicinfo-pro.ch.

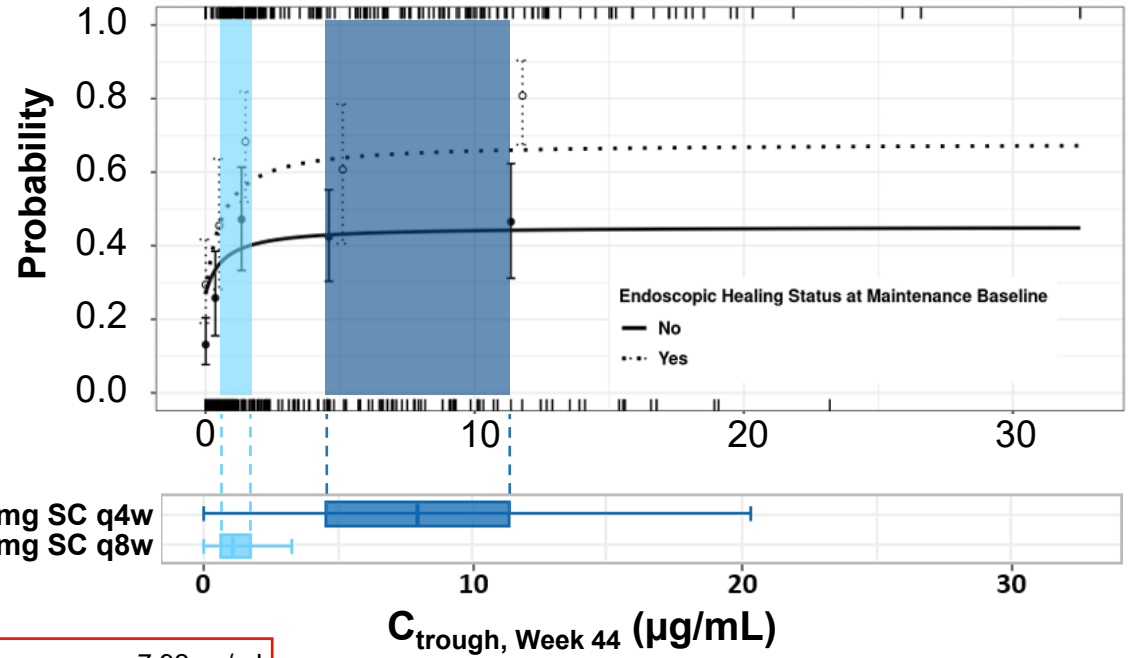
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Exposures for both maintenance dose regimens were associated with substantial efficacy for most participants

Endoscopic improvement at maintenance Week 44, final model stratified by extent of disease



Endoscopic improvement at maintenance Week 44, final model stratified by endoscopic improvement status at maintenance baseline



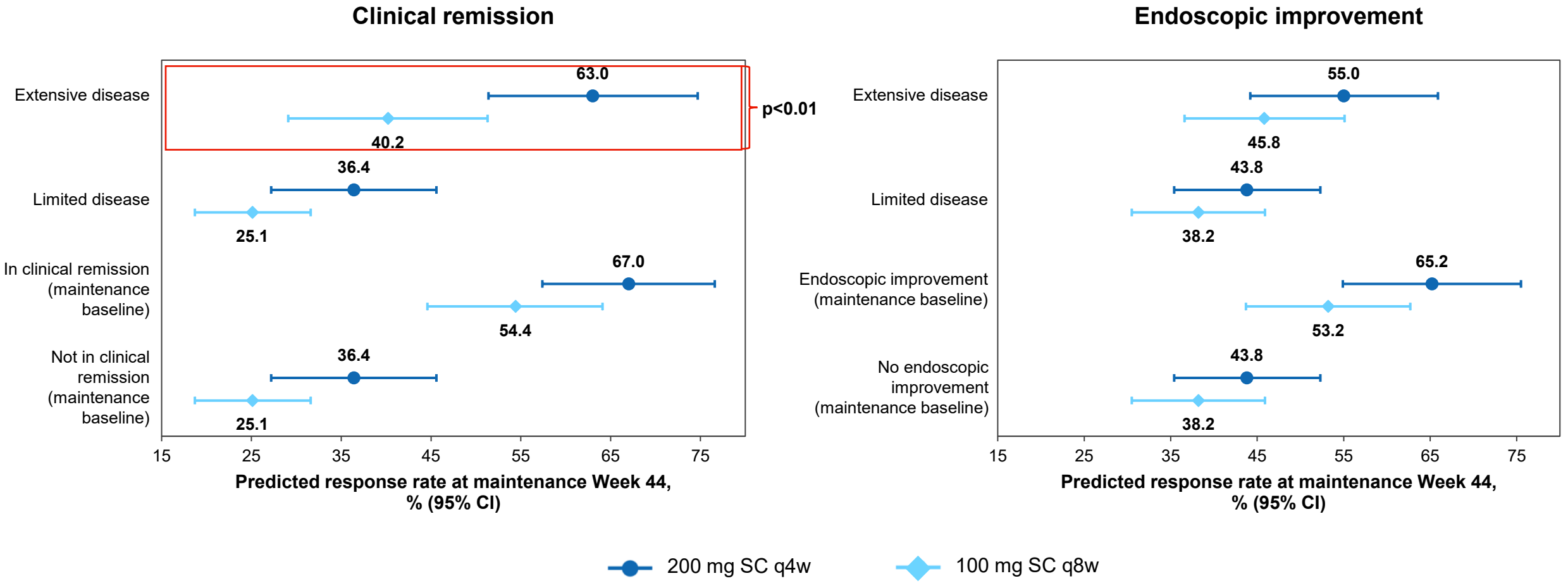
200 mg SC q4w median $C_{trough,\ Week\ 44}$: 7.92 µg/mL
100 mg SC q8w median $C_{trough,\ Week\ 44}$: 1.05 µg/mL

200 mg SC q4w exposures were at the plateau of the E–R curves, suggesting that maximal response is more likely with this regimen and higher doses would not be expected to provide greater efficacy

C_{trough}, trough serum concentration; E–R, exposure–response; GUS, guselkumab; IL, interleukin; MOA, mechanism of action; PK, pharmacokinetics; qXw, every X weeks; SC, subcutaneous; UC, ulcerative colitis.
Rubin DT, et al. Presented at UEGW, Barcelona, Spain, 17–20 October 2026. OP243. Full prescription information: swissmedicinfo-pro.ch.

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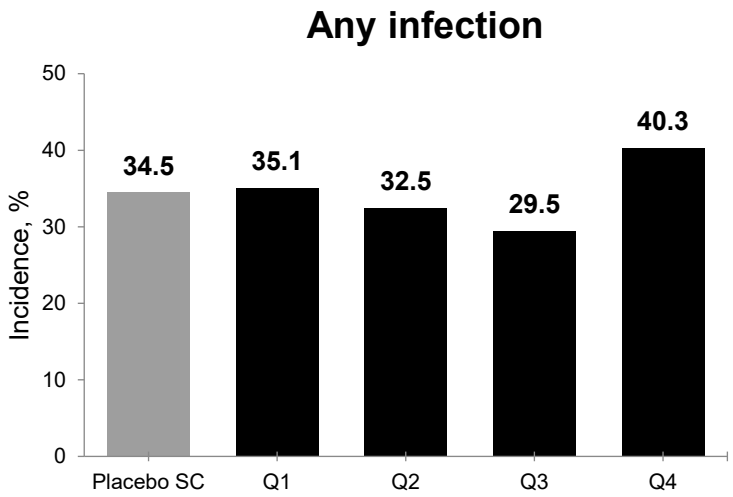
Participants with extensive UC at induction baseline showed a greater treatment effect with 200 mg q4w versus 100 mg SC q8w

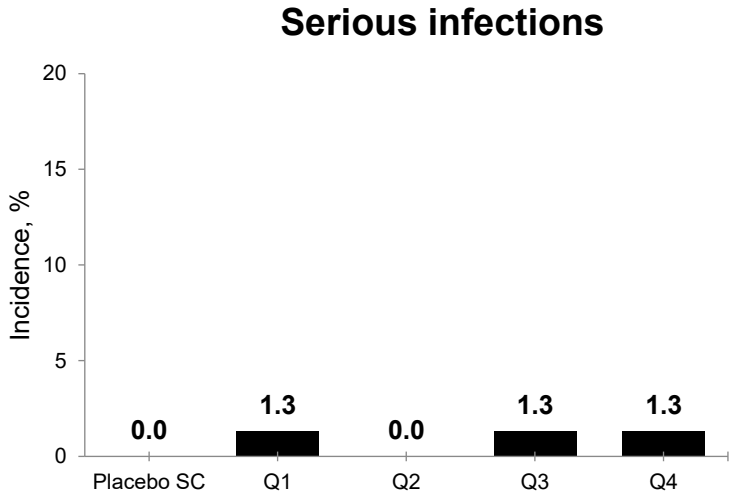


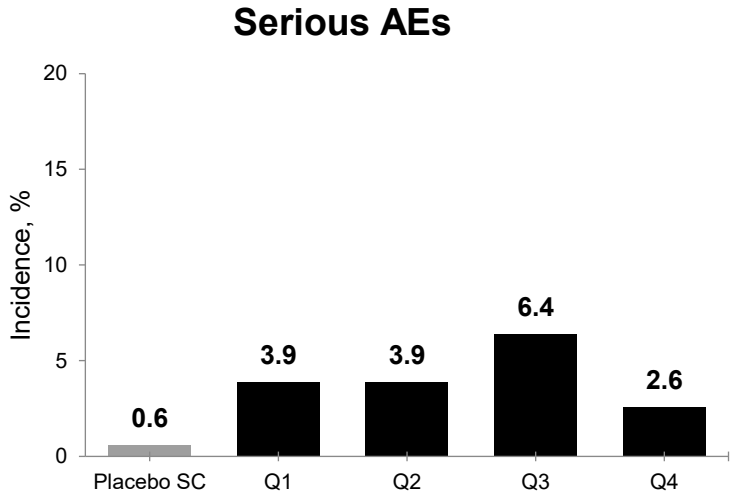
Clinical remission: Stool frequency subscore of 0 or 1 (with no increase from induction baseline), a rectal bleeding subscore of 0, and an endoscopic subscore of 0 or 1 with no friability present on the endoscopy.
Endoscopic improvement: Endoscopic subscore of 0 or 1 with no friability present on the endoscopy.
CI, confidence interval; E–R, exposure–response; GUS, guselkumab; IL, interleukin; MOA, mechanism of action; PK, pharmacokinetics; qXw, every X weeks; SC, subcutaneous; UC, ulcerative colitis.
Rubin DT, et al. Presented at UEGW, Barcelona, Spain, 17–20 October 2026. OP243. Full prescription information: swissmedicinfo-pro.ch.

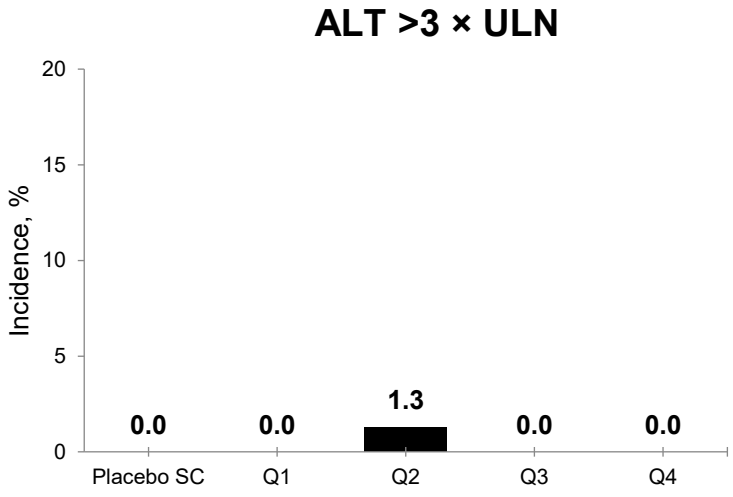
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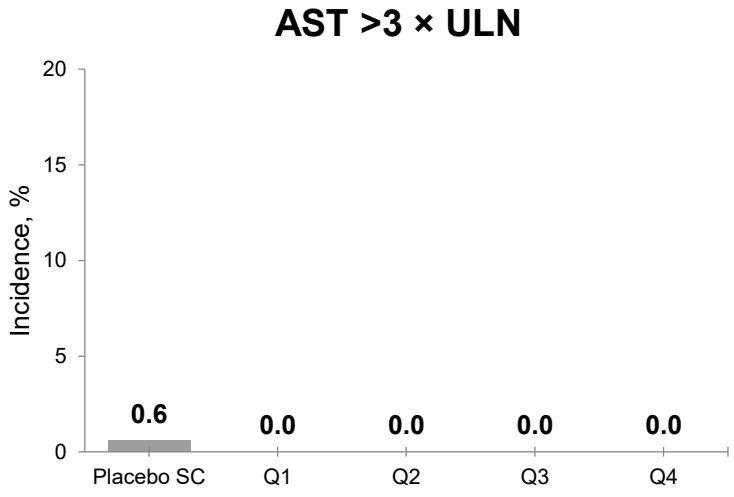
No associations were found between GUS concentrations and infections, serious infections, serious AEs, or abnormal liver function tests

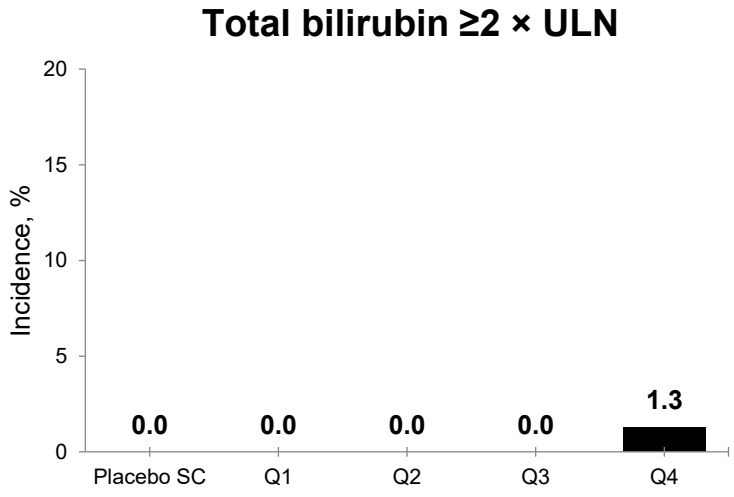












Includes only participants with a modified Mayo score of 5–9 at induction baseline. Quartiles are based on participants in GUS 100 mg SC q8w and 200 mg SC q4w treatment groups as follows: Q1=<1.22 µg/mL, Q2=1.22–3.82 µg/mL, Q3=3.82–9.49 µg/mL, Q4=≥9.49 µg/mL.

AE, adverse event; ALT, alanine aminotransferase; AST, aspartate aminotransferase; GUS, guselkumab; IL, interleukin; MOA, mechanism of action; PK, pharmacokinetics; Q, quartile; qXw, every X weeks; SC, subcutaneous; UC, ulcerative colitis; ULN, upper limit of normal.

Rubin DT, et al. Presented at UEGW, Barcelona, Spain, 17–20 October 2026. OP243. Full prescription information: swissmedicinfo-pro.ch.

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Conclusions

- GUS induction may be given intravenously (200 mg q4w) or subcutaneously (400 mg q4w), with two SC maintenance dosing options (100 mg q8w or 200 mg q4w)
- GUS maintenance PK and efficacy were consistent regardless of IV or SC induction, supporting a common maintenance dosing strategy in UC irrespective of route of induction regimen
- E–R efficacy trends suggest that some participants with extensive UC may further benefit from the higher guselkumab exposure with the 200 mg q4w maintenance dose
- E–R modeling showed efficacy with both maintenance regimens, but exposures with the 200 mg q4w dose were on the plateau of the E–R curve, suggesting no additional efficacy expected with higher doses

E–R, exposure–response; GUS, guselkumab; IL, interleukin; IV, intravenous; MOA, mechanism of action; PK, pharmacokinetics; qXw, every X weeks; SC, subcutaneous; UC, ulcerative colitis.
Rubin DT, et al. Presented at UEGW, Barcelona, Spain, 17–20 October 2026. OP243. Full prescription information: swissmedicinfo-pro.ch.

CH_CP-609070 10//26

Guselkumab in IBD

RWE of GUS in IBD

Early symptomatic response to guselkumab in inflammatory bowel disease: First real-world data from the German GORGEOUS study

PP1357

Poster presented at UEGW

S. Schubert,[1] R. Ehehalt,[2] W. Landry,[3] C. Jochum,[4] E. Schnoy,[5] F. Kreimendahl,[6] T.-L. Ly,[6] E. Hundt,[6] K. Vogel,[6] L. Scharfenberger[6]

[1]MVZ Gastroenterologie am Bayrischen Platz, Berlin, Germany; [2]Praxis für Gastroenterologie Prof. Dr. R. Ehehalt, Heidelberg, Germany; [3]MVZ Dachau, Dachau, Germany; [4]Charité Universitätsmedizin, Berlin, Germany; [5]Universitätsklinikum Augsburg, Augsburg, Germany; [6]Johnson&Johnson, Neuss, Germany.

CH_CP-609070 10//26

Methods

Aim: To assess early symptomatic response in patients with CD or UC using daily diary data from the first 28 days following the first GUS dose

- Since August 2025, the ongoing GORGEOUS study has been collecting physician-documented and patient-reported data from patients with CD or UC initiating GUS in routine clinical care according to the approved label across 65 sites in Germany
- This interim analysis reports diary data from patients who completed the 28-day assessment period, reflecting early symptom changes following the first induction dose of GUS administered on Day 0
- Following abstract acceptance, an updated data cut (10 Jul 2026) was performed to increase the evaluable patient population and to enable a more robust assessment of early response outcomes; findings remained consistent with those from the initial dataset
- Patients completed a daily symptom diary via a smartphone app during the first 28 days of treatment. Assessed symptoms included: stool frequency, soft/liquid stools, nightly stools, bowel urgency, rectal bleeding, and abdominal pain in the last 24 hours
- Results are presented descriptively and stratified by disease cohort (CD and UC)
- Further analyses will be performed as follow-up data accrue

CD, Crohn's disease; GUS, guselkumab; IBD, inflammatory bowel disease; IL, interleukin; MOA, mechanism of action; RWE, real-world evidence; UC, ulcerative colitis.
Schubert S, et al. Presented at UEGW, Barcelona, Spain, 17–20 October 2026. PP1357. Full prescription information: swissmedicinfo-pro.ch.

CH_CP-609070 10//26

Study population and baseline characteristics (1/2)

	CD cohort n=236	UC cohort n=153
Gender – Female	51.3%	52.9%
Age, years; median (Q1/ Q3)	37 (29/51.5)	38 (27/50)
Disease duration ≤2 years / >2 years	21.2% / 78.8%	25.5% / 74.5%
CD location,*,† available N	202	
L1 – terminal ileum	33.1%	*NA*
L2 – colon	17.4%	*NA*
L3 – ileocolonic	42.0%	*NA*
L4 – upper gastrointestinal	15.3%	*NA*
CD disease behaviour,*,† available N	202	
B1 – non-stricturing, non-penetrating	49.6%	*NA*
B2 – stricturing	30.9%	*NA*
B3 – penetrating	12.3%	*NA*
P – perianal	8.9%	*NA*
UC extent of disease,* available N		148
E1 – Proctitis	*NA*	17.6%
E2 – Left sided colitis	*NA*	35.8%
E3 – Pancolitis	*NA*	46.6%

Baseline characteristics reflect real-world IBD population and suggest that treatment decisions were informed by factors beyond symptom burden, such as persistent inflammatory activity or prior treatment failure. Most patients (~70%) were ADT-experienced

Data are presented as percent unless specified otherwise. Cohort specific variables are marked as NA. All assessments have only been documented if assessed during local clinical routine.
*Montreal classification; †Multiple answers possible.
ADT, advanced drug therapy; CD, Crohn's disease; GUS, guselkumab; IBD, inflammatory bowel disease; IL, interleukin; MOA, mechanism of action; N, number of patients; NA, not applicable; Q1, first quartile; Q3, third quartile; RWE, real-world evidence; UC, ulcerative colitis.
Schubert S, et al. Presented at UEGW, Barcelona, Spain, 17–20 October 2026. PP1357. Full prescription information: swissmedicinfo-pro.ch.

CH_CP-609070 10//26

Study population and baseline characteristics (2/2)

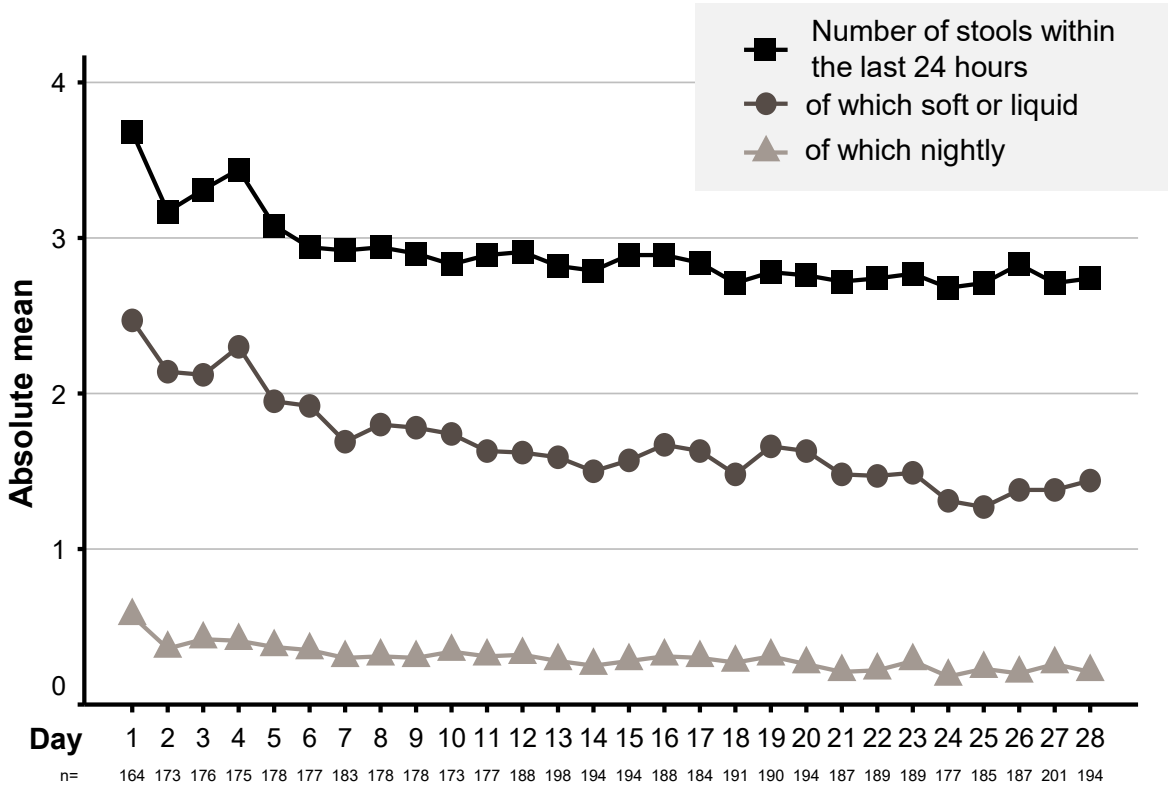
	CD cohort n=236	UC cohort n=153
UC severity of disease,* available N		
S0 – asymptomatic / S1 – mild	*NA*	1.3% / 8.7%
S2 – moderate / S3 – severe	*NA*	63.8% / 26.2%
Disease activity status, available N	182	121
Remission or inactive disease	HBI 0–4: 35.7%	PMS 0–2: 13.2%
Mild disease	HBI 5–7: 31.9%	PMS 3–4: 28.1%
Moderate disease	HBI 8–16: 32.8%	PMS 5–6: 32.2%
Severe disease	HBI >16: 0.0%	PMS 7–9: 26.4%
Abnormal endoscopic activity, available N	164	106
CD: moderate/severe or SES-CD score ≥7; UC: MES ≥2	61.6%	76.4%
Number of previous ADTs		148
0	23.3%	28.8%
1 / 2	37.7% / 21.2%	32.0% / 24.2%
3 / 4	11.0% / 6.8%	7.2% / 7.8%
Corticosteroid use at baseline		
Yes	32.2%	49.0%
Laboratory inflammatory parameters		
Available N	181	132
CRP >5 mg/L	54.7%	45.5%
Available N	100	75
fCAL >250 µg/g	58.0%	64.0%

Data are presented as percent unless specified otherwise. Cohort specific variables are marked as NA. All assessments have only been documented if assessed during local clinical routine.
*Montreal classification.
ADT, advanced drug therapy; CD, Crohn's disease; CRP, C-reactive protein; fCal, faecal calprotectin; GUS, guselkumab; IBD, inflammatory bowel disease; IL, interleukin; MES, Mayo Endoscopic Subscore; MOA, mechanism of action; N, number of patients; NA, not applicable; Q1, first quartile; Q3, third quartile; RWE, real-world evidence; SES-CD, Simple Endoscopic Score for Crohn's Disease; UC, ulcerative colitis.
Schubert S, et al. Presented at UEGW, Barcelona, Spain, 17–20 October 2026. PP1357. Full prescription information: swissmedicinfo-pro.ch.

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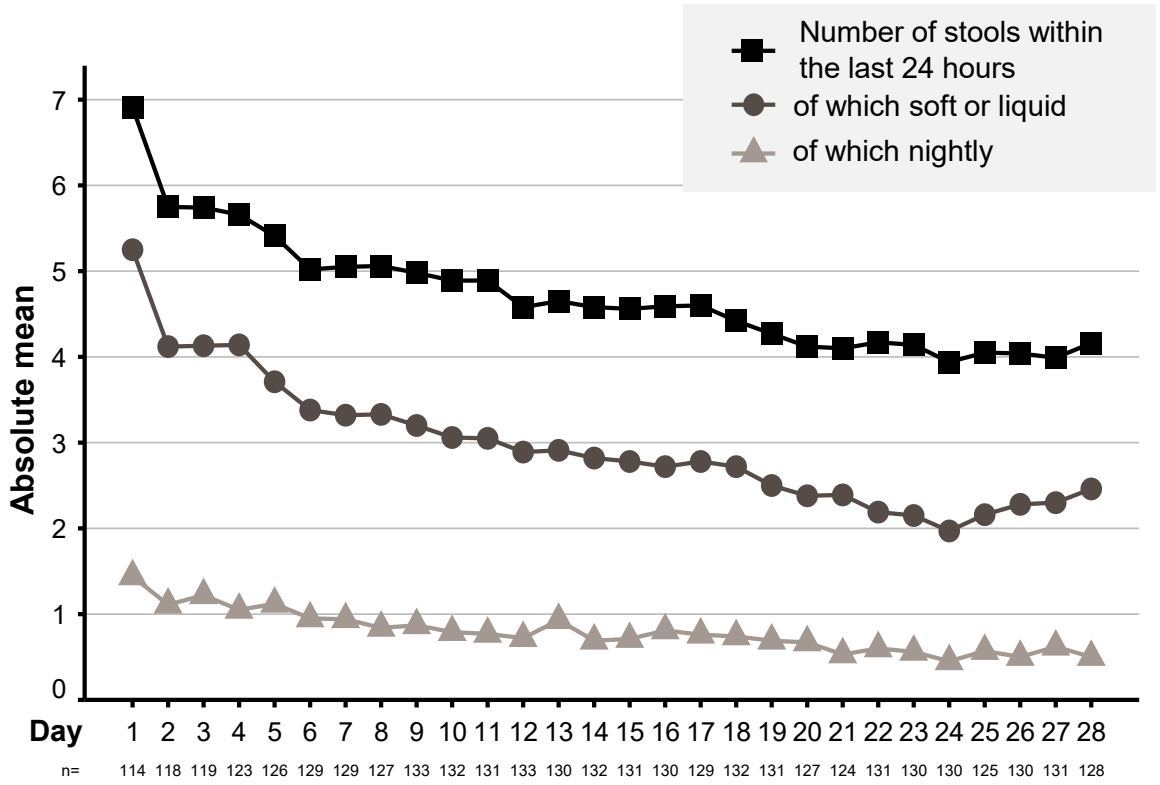
Stool frequency within the first 4 weeks of GUS

CD cohort



In patients with CD, mean stool frequency decreased from 3.7 to 2.7. Soft or liquid stools were decreased from 2.5 to 1.4, from Day 1 to Day 28, respectively.

UC cohort

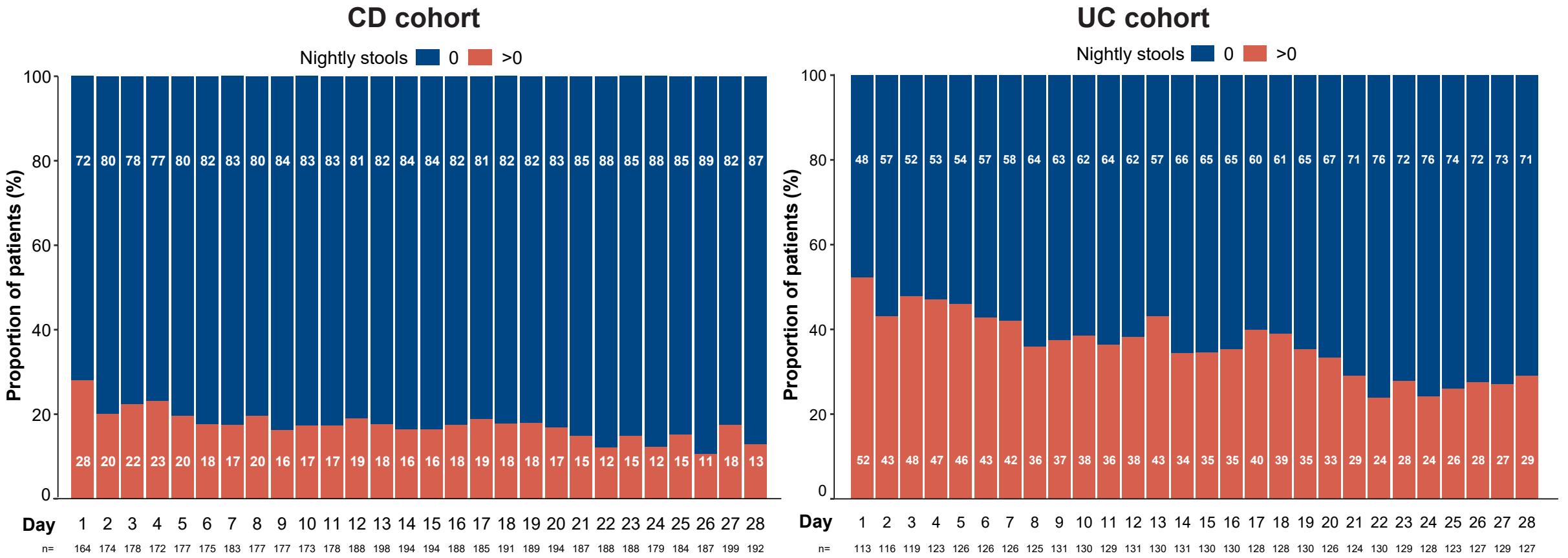


In patients with UC, the stool frequency mean decreased from 6.9 (Day 1) to 4.2 (Day 28). Soft or liquid stools were decreased from 5.3 (Day 1) to 2.5 (Day 28).

CD, Crohn's disease; GUS, guselkumab; IBD, inflammatory bowel disease; IL, interleukin; MOA, mechanism of action; N, number of patients; RWE, real-world evidence; UC, ulcerative colitis.

Schubert S, et al. Presented at UEGW, Barcelona, Spain, 17–20 October 2026. PP1357. Full prescription information: swissmedicinfo-pro.ch.

Nightly stools within the first 4 weeks of GUS

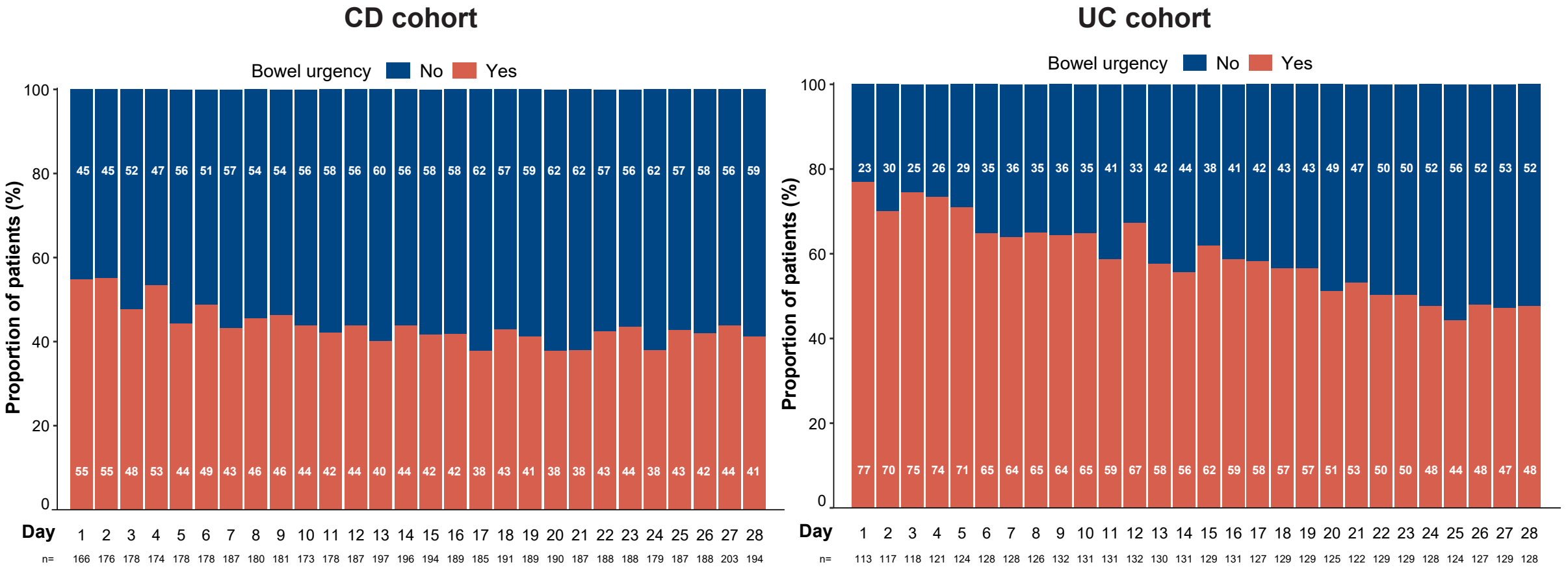


Nightly stools, a key driver of sleep interruption, were reported by 28.1% and 52.2% of patients, respectively, with CD and UC at Day 1. By Day 28, the proportion had lowered to 13.0% in CD and 29.1% in UC, corresponding to relative reductions of 53.7% and 44.3%, respectively

CD, Crohn's disease; GUS, guselkumab; IBD, inflammatory bowel disease; IL, interleukin; MOA, mechanism of action; N, number of patients; RWE, real-world evidence; UC, ulcerative colitis.
Schubert S, et al. Presented at UEGW, Barcelona, Spain, 17–20 October 2026. PP1357. Full prescription information: swissmedicinfo-pro.ch.

CH_CP-609070 10//26

Bowel urgency within the first 4 weeks of GUS

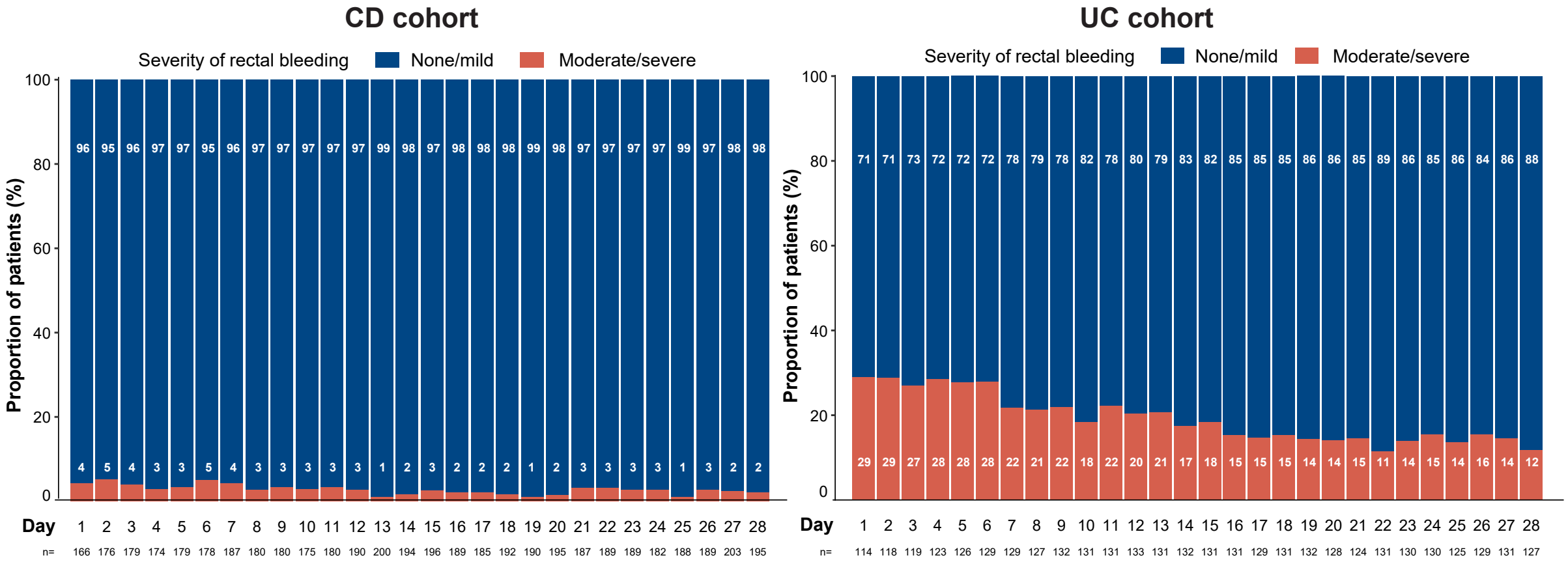


At Day 1, bowel urgency was reported by the majority of patients in both cohorts. In CD, rate decreased from 54.8% to 41.2% by Day 28 (a 24.8% relative reduction); in UC, from 77.0% to 47.7% (a 38.1% relative reduction).

CD, Crohn's disease; GUS, guselkumab; IBD, inflammatory bowel disease; IL, interleukin; MOA, mechanism of action; N, number of patients; RWE, real-world evidence; UC, ulcerative colitis.

Schubert S, et al. Presented at UEGW, Barcelona, Spain, 17–20 October 2026. PP1357. Full prescription information: swissmedicinfo-pro.ch.

Rectal bleeding within the first 4 weeks of GUS



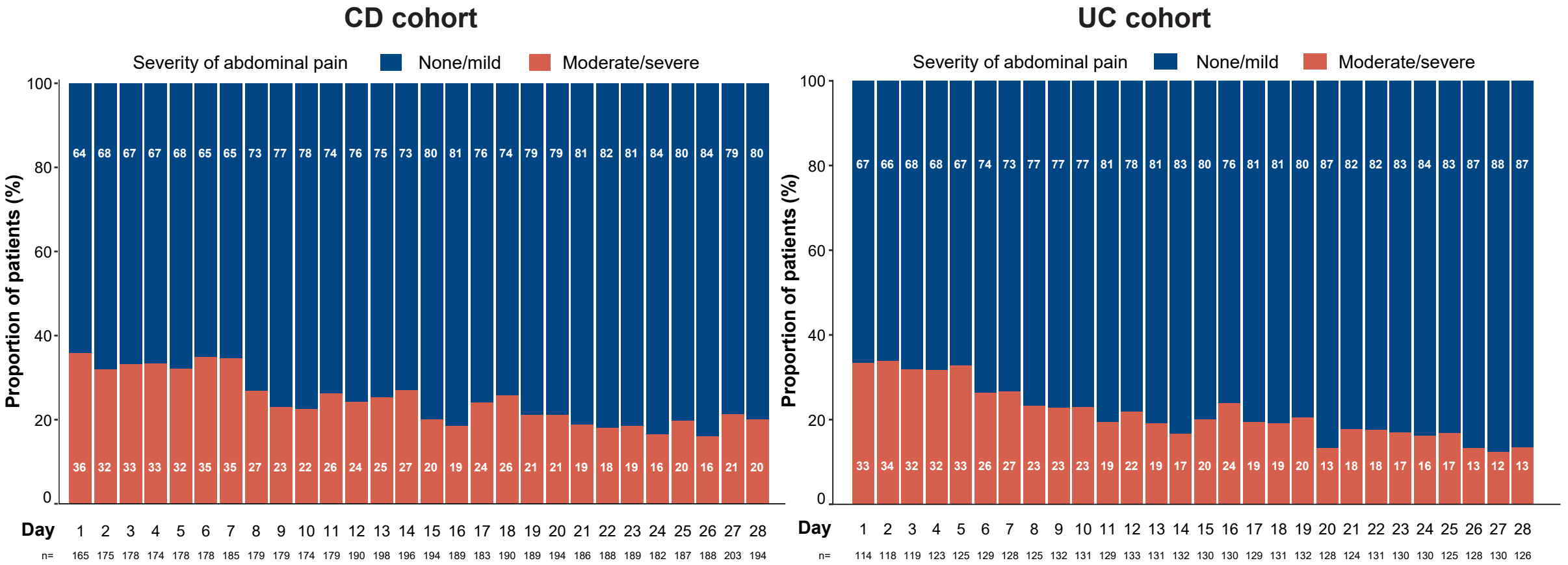
Moderate or severe rectal bleeding was infrequent in CD, decreasing from 4.2% at Day 1 to 2.1% at Day 28. In UC, where rectal bleeding was predominantly reported, the proportion decreased from 29.0% at Day 1 to 11.8% at Day 28, representing a relative reduction of 58.9%.

CD, Crohn's disease; GUS, guselkumab; IBD, inflammatory bowel disease; IL, interleukin; MOA, mechanism of action; N, number of patients; RWE, real-world evidence; UC, ulcerative colitis.

Schubert S, et al. Presented at UEGW, Barcelona, Spain, 17–20 October 2026. PP1357. Full prescription information: swissmedicinfo-pro.ch.

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Abdominal pain within the first 4 weeks of GUS



Both CD and UC cohorts reported reductions of abdominal pain, with more prominent relief in UC, where the proportion of patients with no or mild pain increased from 66.7% at Day 1 to 86.5% at Day 28.

CD, Crohn's disease; GUS, guselkumab; IBD, inflammatory bowel disease; IL, interleukin; MOA, mechanism of action; N, number of patients; RWE, real-world evidence; UC, ulcerative colitis.

Schubert S, et al. Presented at UEGW, Barcelona, Spain, 17–20 October 2026. PP1357. Full prescription information: swissmedicinfo-pro.ch.

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Conclusions

- GORGEOUS is the largest real-world study of patients treated with GUS in IBD to date. Its population reflects patients typically seen in routine clinical practice in Germany, with long disease duration and heterogeneous treatment histories
- Daily patient-reported symptom diary entries capture symptom relief information, without recall bias
- For most symptomatic endpoints, clinically meaningful improvements were already evident within the first week following a single dose of GUS, with a substantial proportion of the overall 28-day improvement achieved by Days 6–8 and modest additional gains thereafter
- Reductions in nocturnal stools and abdominal pain particularly highlight the relevance for patients' quality of life
- No new safety signals were identified through Week 4
- These interim findings provide the first real-world evidence of early symptomatic response to GUS in routine clinical practice in Germany

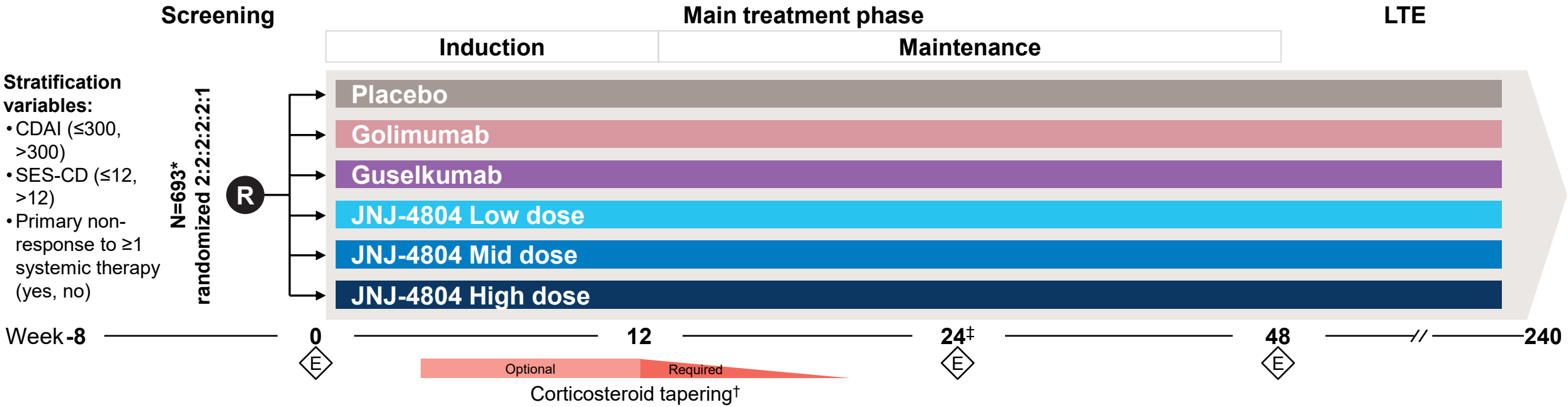
CD, Crohn's disease; GUS, guselkumab; IBD, inflammatory bowel disease; IL-23, interleukin-23; MOA, mechanism of action; N, number of patients; RWE, real-world evidence; UC, ulcerative colitis.
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Other therapies in IBD

DUET-CD: JNJ-4804 in CD

DUET-CD study design



*Full analysis set. †All participants taking corticosteroids at Week 0 could begin tapering as early as Week 4 but no later than Week 12.
‡Patients who met inadequate response criteria, regardless of treatment assignment, received a JNJ-4804 regimen based on their initial study intervention group assignment.
CD, Crohn's disease; CDAI, Crohn's Disease Activity Index; E, endoscopy; IL, interleukin; LTE, long-term extension; MoA, mechanism of action;
R, randomisation; RDBPC, randomised, double-blind, placebo-controlled; SES-CD, Simple Endoscopic Score for Crohn's Disease; TNF, tumour necrosis factor.
Jairath V, et al. Presented at ACG, 9–14 October 2026; Nashville, TN, USA. P5694.
JNJ-7893480 is investigational. JNJ-7893480 and golimumab (Simponi) are not approved by Swissmedic for Crohn's disease. Full prescribing information: swissmedicinfo-pro.ch.

CH_CP-609070 10//26

Efficacy and safety of the first co-antibody therapy, JNJ-78934804, in patients with moderately to severely active Crohn's disease refractory to two or more systemic therapy mechanisms

P5694

Presented at ACG 2026

Also presented as an encore at UEGW 2026 (MP0562)

Vipul Jairath,[1] Geert D'Haens,[2] Iris Dotan,[3] Nat A. Terry,[4] Monica Walker,[4] Vanessa Bundy,[4] Hayley Perry,[5] Dylan Richards,[4] Marion L. Vetter,[4] Taku Kobayashi,[6] Stefan Schreiber,[7] Bruce E. Sands[8]

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DUET-CD background and objective

Background

- Despite therapeutic advances, substantial unmet need remains in CD as many patients develop disease refractory to STs (biologics and novel oral agents), with diminishing efficacy after each treatment
- We previously reported results from the DUET-CD study of JNJ-7893480 (JNJ-4804), the first fixed-dose, co-antibody therapy with GUS and GOL targeting the complementary IL-23 and TNF pathways
- In DUET-CD, JNJ-4804 high dose showed the most substantial and clinically meaningful benefit versus GUS and GOL monotherapies in the subpopulation of participants with CD refractory to ≥2 ST mechanisms (anti-TNFs, anti-IL-12/23s, anti-IL-23p19s, anti-integrins and JAK inhibitors)

Objective

- Here, we present efficacy data from this treatment-refractory subpopulation as well as additional faecal calprotectin and safety data

CD, Crohn's disease; GOL, golimumab; GUS, guselkumab; IL, interleukin; JAK, Janus kinase; MOA, mechanism of action; RDBPC, randomised, double-blind, placebo-controlled; ST, systemic therapy; TNF, tumour necrosis factor. Jairath V, et al. Presented at ACG, 9–14 October 2026; Nashville, TN, USA. P5694.
JNJ-7893480 is investigational. JNJ-7893480 and golimumab (Simponi) are not approved by Swissmedic for Crohn's disease. Full prescribing information: swissmedicinfo-pro.ch.

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Baseline demographics and disease characteristics

- Baseline demographics and disease characteristics show that DUET-CD evaluated one of the most treatment-refractory CD populations studied to date

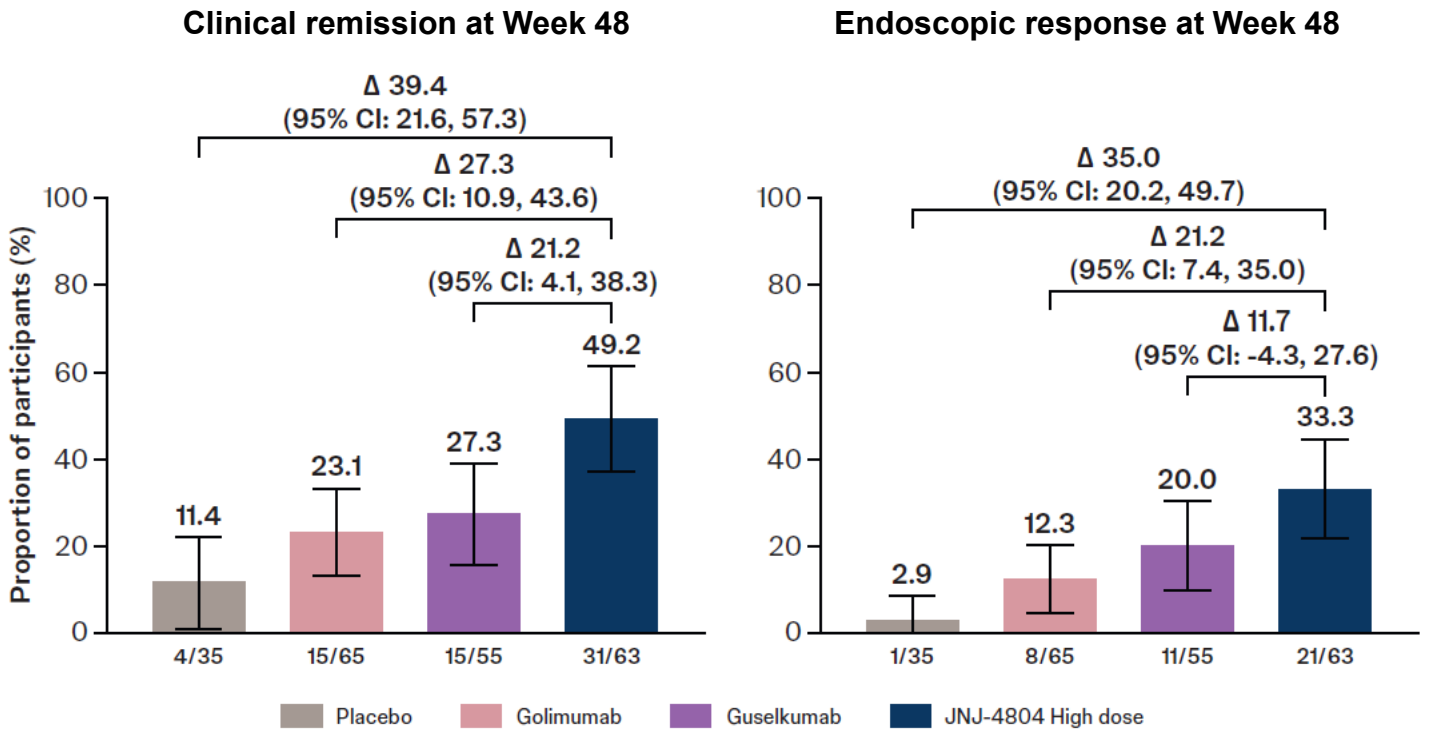
	Placebo	Golimumab	Guselkumab	JNJ-4804 Low dose	JNJ-4804 Mid dose	JNJ-4804 High dose
Full analysis set	64	126	127	127	123	126
Participants with IR to ≥2 ST mechanisms	35	65	55	73	55	63
Demographics						
Age in years, mean (SD)	35.9 (13.62)	39.0 (12.82)	35.7 (11.22)	37.8 (12.34)	37.5 (10.84)	37.4 (2.62)
Sex, n (%) Male	26 (74.3%)	38 (58.5%)	27 (49.1%)	37 (50.7%)	33 (60.0%)	23 (36.5%)
Crohn's disease duration in years, mean (SD)	12.04 (9.916)	11.29 (7.389)	13.17 (8.125)	13.47 (9.235)	11.68 (8.291)	13.91 (9.286)
Disease characteristics						
Disease location, n (%) Ileum only	7 (20.0%)	22 (33.8%)	15 (27.3%)	14 (19.2%)	12 (21.8%)	14 (22.2%)
CDAI score, mean (SD)*	325.57 (61.583)	324.14 (61.661)	338.22 (49.532)	324.75 (60.935)	312.26 (53.835)	335.52 (64.701)
SES-CD, mean (SD)	14.46 (8.023)	13.28 (8.020)	14.67 (8.367)	15.03 (8.617)	13.98 (8.038)	14.38 (9.042)
Concomitant medications						
Immunomodulators, n (%)	6 (17.1%)	7 (10.8%)	8 (14.5%)	14 (19.2%)	4 (7.3%)	10 (15.9%)
Oral corticosteroids, n (%)	13 (37.1%)	20 (30.8%)	18 (32.7%)	23 (31.5%)	16 (29.1%)	22 (34.9%)
Inflammatory pharmacodynamic markers						
CRP (mg/L), median†	8.70	7.50	10.70	10.80	8.10	8.90
Faecal calprotectin (mg/kg), median‡	1705.00	1133.40	1373.35	1747.80	978.00	1145.00
Prior ST inadequate response history						
2 ST mechanisms, n (%)	24 (68.6%)	45 (69.2%)	37 (67.3%)	49 (67.1%)	37 (67.3%)	39 (61.9%)
>2 ST mechanisms, n (%)	11 (31.4%)	20 (30.8%)	18 (32.7%)	24 (32.9%)	18 (32.7%)	24 (38.1%)
Anti-IL12/23 and anti-TNF, n (%)	21 (60.0%)	37 (56.9%)	26 (47.3%)	35 (47.9%)	28 (50.9%)	31 (49.2%)

*Means are based on the following numbers of participants with non-missing data: 35 (placebo), 63 (GOL), 54 (GUS), 73 (JNJ-4804 low dose), 54 (JNJ-4804 mid dose), 63 (JNJ-4804 high dose).
†Medians are based on the following numbers of participants with non-missing data: 35 (placebo), 62 (GOL), 55 (GUS), 73 (JNJ-4804 low dose), 55 (JNJ-4804 mid dose), 63 (JNJ-4804 high dose).
‡Medians are based on the following numbers of participants with non-missing data: 31 (placebo), 57 (GOL), 46 (GUS), 66 (JNJ-4804 low dose), 49 (JNJ-4804 mid dose), 55 (JNJ-4804 high dose).
CD, Crohn's disease; CDAI, Crohn's Disease Activity Index; CRP, C-reactive protein; GOL, golimumab; GUS, guselkumab; IL, interleukin; IR, inadequate response or intolerance; MoA, mechanism of action; RDBPC, randomised, double-blind, placebo-controlled; SD, standard deviation; SES-CD, Simple Endoscopic Score for Crohn's Disease; ST, systemic therapy; TNF, tumour necrosis factor.
Jairath V, et al. Presented at ACG, 9–14 October 2026; Nashville, TN, USA. P5694.
JNJ-7893480 is investigational. JNJ-7893480 and golimumab (Simponi) are not approved by Swissmedic for Crohn's disease. Full prescribing information: swissmedicinfo-pro.ch.

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Clinical remission and endoscopic response at Week 48

Co-primary endpoints in patients with CD refractory to ≥2 systemic therapy mechanisms



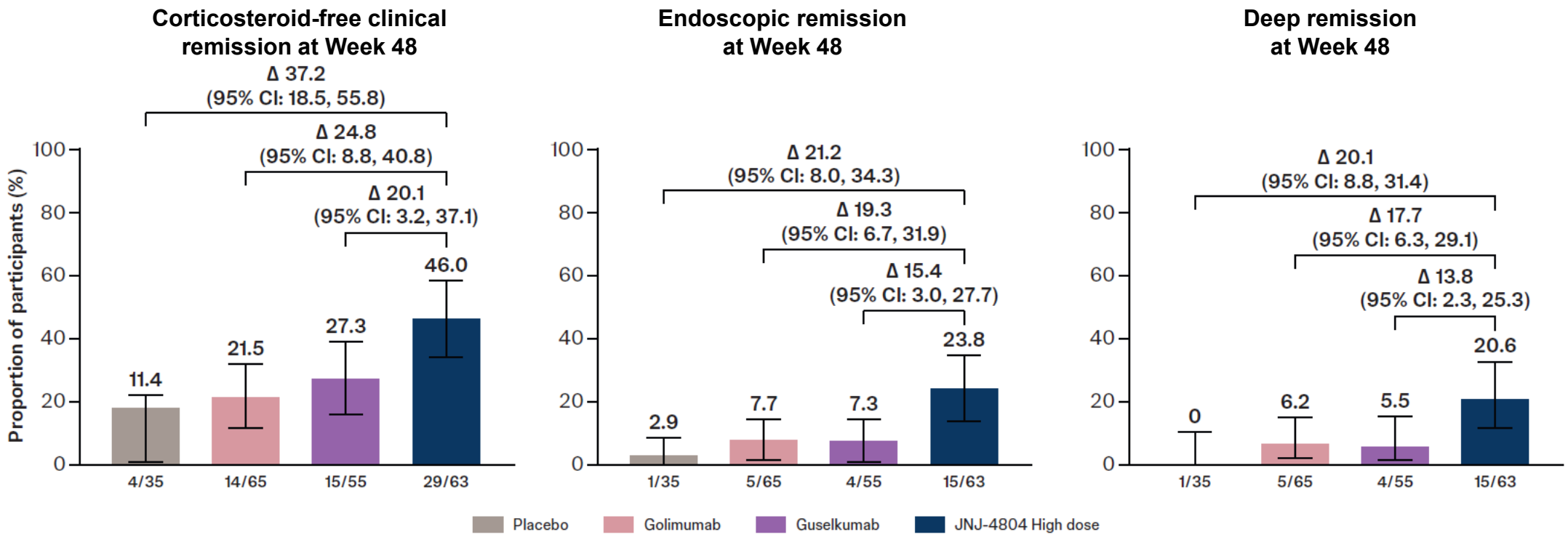
- Among participants with CD refractory to ≥2 systemic therapy mechanisms, JNJ-4804 high dose led to clinically meaningful differences at Week 48 in rates of clinical remission and endoscopic response vs golimumab, guselkumab and placebo monotherapies

Clinical remission: CDAI score of <150. **Endoscopic response:** >50% improvement from baseline in SES-CD or an SES-CD ≤2, per central endoscopy.
CD, Crohn's disease; CDAI, Crohn's Disease Activity Index; CI, confidence interval; IL, interleukin; MoA, mechanism of action;
RDBPC, randomised, double-blind, placebo-controlled; SES-CD, Simple Endoscopic Score for Crohn's Disease; TNF, tumour necrosis factor.
Jairath V, et al. Presented at ACG, 9–14 October 2026; Nashville, TN, USA. P5694.
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Corticosteroid-free, endoscopic and deep remission at Week 48

Key endpoints in patients with CD refractory to ≥2 systemic therapy mechanisms

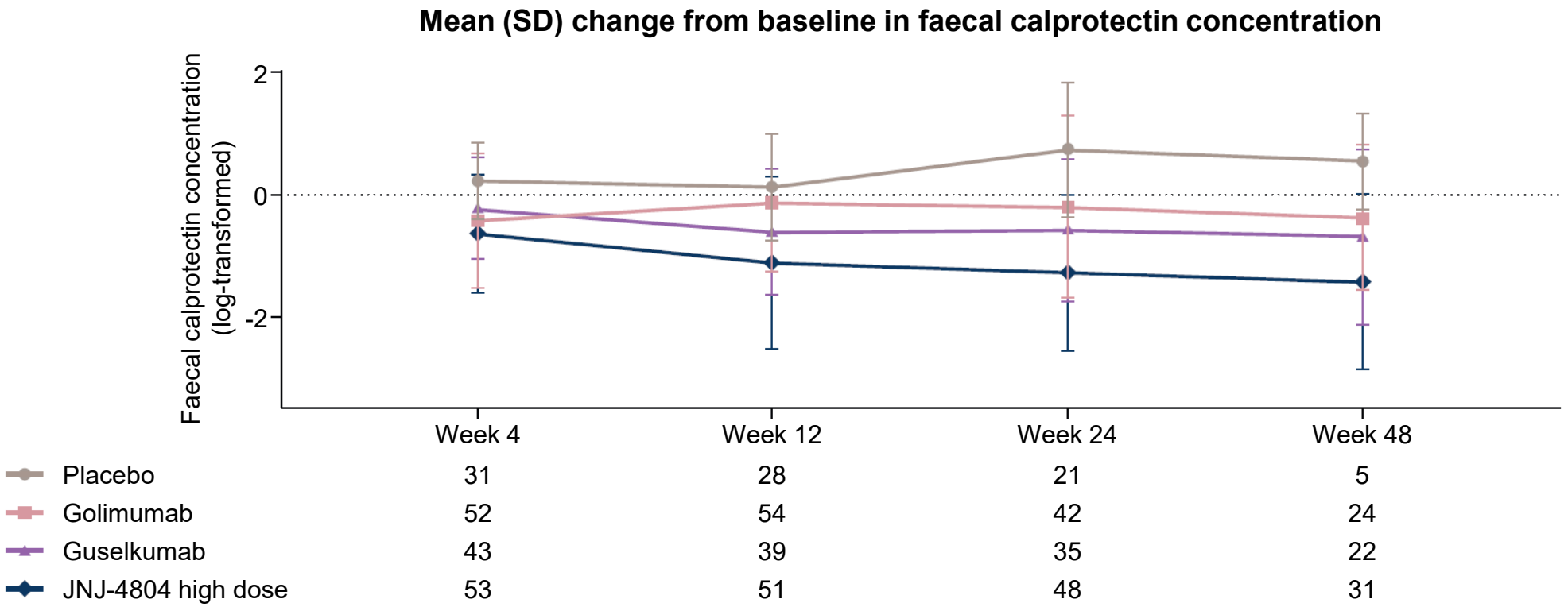


- Rates of meeting high-bar outcomes such as endoscopic remission and deep remission with JNJ-4804 high dose exceeded the sum of the rates in the individual monotherapy arms, suggesting a synergistic effect

Clinical remission: CDAI score of <150. **Endoscopic remission:** SES-CD ≤4, with a ≥2-point reduction from baseline and no subscore >1 on any component.
Deep remission: both clinical remission and endoscopic remission. **Corticosteroid-free (60-day) clinical remission:** clinical remission with no corticosteroids received for ≥60 days.
CD, Crohn's disease; CDAI, Crohn's Disease Activity Index; CI, confidence interval; IL, interleukin; MoA, mechanism of action;
RDBPC, randomised, double-blind, placebo-controlled; SES-CD, Simple Endoscopic Score for Crohn's Disease; TNF, tumour necrosis factor.
Jairath V, et al. Presented at ACG, 9–14 October 2026; Nashville, TN, USA. P5694.

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Mean change from baseline in faecal calprotectin to Week 48



- Mean change from baseline in faecal calprotectin was greater with JNJ-4804 high dose vs golimumab, guselkumab and placebo monotherapies through Week 48

Mean change from baseline in faecal calprotectin with JNJ-4804 high dose vs the other groups in participants with CD refractory to ≥2 ST mechanisms.
CD, Crohn's disease; IL, interleukin; MoA, mechanism of action; RDBPC, randomised, double-blind, placebo-controlled; SD, standard deviation; ST, systemic therapy; TNF, tumour necrosis factor.
Jairath V, et al. Presented at ACG, 9–14 October 2026; Nashville, TN, USA. P5694.
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CH_CP-609070 **10/26**

Exposure-adjusted safety event rates[1]

	Placebo	Golimumab	Guselkumab	JNJ-4804 Low dose	JNJ-4804 Mid dose	JNJ-4804 High dose	JNJ-4804 Combined
Full analysis set	64	126	127	127	123	126	376
Participants with IR to ≥2 ST mechanisms	35	65	55	73	55	63	191
Total patient-years of follow-up	20.5	44.9	38.9	54.2	42.6	50	146.8
Event rates per hundred patient-years [number of events] (95% CI).							
AEs	606.0 [124] (504.02, 722.51)	695.1 [312] (620.08, 776.64)	798.8 [311] (712.50, 892.72)	631.4 [342] (566.27, 702.02)	615.4 [262] (543.17, 694.66)	585.6 [293] (520.46, 656.64)	611.2 [897] (571.82, 652.51)
SAEs	24.4 [5] (7.93, 57.02)	44.6 [20] (27.22, 68.81)	20.5 [8] (8.87, 40.49)	42.5 [23] (26.92, 63.72)	18.8 [8] (8.11, 37.03)	24.0 [12] (12.39, 41.89)	29.3 [43] (21.20, 39.46)
AEs leading to discontinuation of study treatment	53.8 [11] (26.83, 96.18)	29.0 [13] (15.42, 49.53)	23.1 [9] (10.57, 43.88)	11.1 [6] (4.07, 24.11)	11.7 [5] (3.81, 27.41)	10.0 [5] (3.24, 23.32)	10.9 [16] (6.23, 17.70)
Infections	102.6 [21] (63.53, 156.87)	151.5 [68] (117.64, 192.05)	120.7 [47] (88.70, 160.54)	155.1 [84] (123.70, 192.01)	133.9 [57] (101.41, 173.48)	143.9 [72] (112.59, 181.22)	145.1 [213] (126.29, 165.98)
Serious infections	4.9 [1] (0.12, 27.23)	2.2 [1] (0.06, 12.41)	7.7 [3] (1.59, 22.52)	11.1 [6] (4.07, 24.11)	4.7 [2] (0.57, 16.97)	2.0 [1] (0.05, 11.14)	6.1 [9] (2.80, 11.64)
Deaths	0.0 [0] (0.00, 14.64)	0.0 [0] (0.00, 6.67)	0.0 [0] (0.00, 7.69)	0.0 [0] (0.00, 5.53)	0.0 [0] (0.00, 7.04)	0.0 [0] (0.00, 5.99)	0.0 [0] (0.00, 2.04)

- Exposure-adjusted safety event rates with JNJ-4804 in the subpopulation were overall similar to or lower than those with placebo, golimumab or guselkumab, and were similar to those in the overall population[2]

AE, adverse event; CD, Crohn's disease; CI, confidence interval; IL, interleukin; IR, inadequate response or intolerance; MoA, mechanism of action; RDBPC, randomised, double-blind, placebo-controlled; SAE, serious adverse event; ST, systemic therapy; TNF, tumour necrosis factor.

1. Jairath V, et al. Presented at ACG, 9–14 October 2026; Nashville, TN, USA. P5694; 2. Abreu MT, et al. Presented at DDW; 2–5 May 2026. Chicago, IL, USA [Abstract 1058d].

JNJ-7893480 is investigational. JNJ-7893480 and golimumab (Simponi) are not approved by Swissmedic for Crohn's disease. Full prescribing information: swissmedicinfo-pro.ch.

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Conclusions

- In treatment-refractory participants with inadequate response to ≥2 systemic therapy mechanisms, JNJ-4804, the first co-antibody therapy in development for IBD, delivered substantial and clinically meaningful efficacy, with benefits that exceeded those of golimumab and guselkumab monotherapy, and the greatest improvements observed with the high dose
- Clinical results were supported by improvements in objective inflammatory and clinically relevant biomarkers
- The JNJ-4804 safety profile in this subpopulation was similar to those of the golimumab and guselkumab monotherapies
- These results support advancement to Phase 3 in the rapidly growing population of patients with disease refractory to multiple systemic therapy mechanisms

CD, Crohn's disease; IBD, inflammatory bowel disease; IL, interleukin; MoA, mechanism of action; RDBPC, randomised, double-blind, placebo-controlled; TNF, tumour necrosis factor.

Jairath V, et al. Presented at ACG, 9–14 October 2026; Nashville, TN, USA. P5694.

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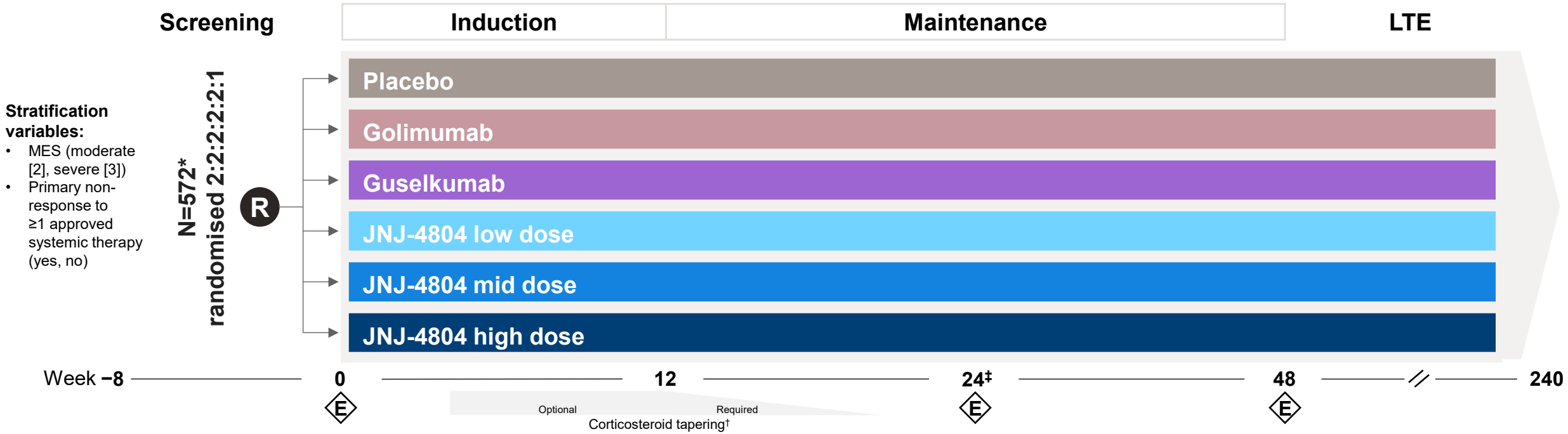
DUET: JNJ-4804 in UC

DUET-UC study design

Aim: Evaluate the efficacy and safety of JNJ-4804 through Week 240 in this treatment-refractory subpopulation

Key eligibility criteria

- Moderately to severely active UC
- Inadequate response or intolerance to ≥1 systemic therapy mechanism (anti-TNF, IL-12/23, IL-23p19, integrin, JAK inhibitor or S1PR modulator)
- Caps for prior approved systemic therapy mechanisms: 1 (50%), 2 (35%), >2 (15%)
- All study medications were administered subcutaneously every 4 weeks



*Full analysis set; 559 were included in the modified full analysis set (13 did not meet mMayo inclusion criteria at baseline and were excluded). †Participants taking corticosteroids at Week 0 could begin tapering as early as Week 4 but no later than Week 12. ‡Participants who met inadequate response criteria at Week 24 received a JNJ-4804 regimen (regardless of treatment assignment).
E, endoscopy; IL, interleukin; JAK, Janus kinase; LTE, long-term extension; MES, Mayo Endoscopic Subscore; mMayo, modified Mayo score; MOA, mechanism of action; R, randomisation; RDBAPC, randomised, double-blind, active- and placebo-controlled; S1PR, sphingosine-1-phosphate receptor; SC, subcutaneous; TNF, tumour necrosis factor; UC, ulcerative colitis.
Atreya R, et al. Presented at UEGW, Barcelona, Spain, 17–20 October 2026. OP255.
JNJ-7893480 is investigational. JNJ-7893480 is not approved by Swissmedic for UC. Full prescribing information: swissmedicinfo-pro.ch.

CH_CP-609070 10//26

Efficacy and safety of the first co-antibody therapy, JNJ-78934804, in patients with moderately to severely active ulcerative colitis refractory to two or more systemic therapy mechanisms

OP255

Oral presentation at UEGW 2026

Raja Atreya,[1] Bruce E. Sands,[2] Séverine Vermeire,[3] Remo Panaccione,[4] Gregory T Moore,[5] John Lynch,[6] Melissa G. Marko,[6] Eun Suk Jung,[6] Ruchi Patel,[6] Siyka Alexandrova,[6] Hayley Perry,[7] Marion L. Vetter,[6] Julián Panés,[8] Maria T. Abreu[9]

[1]Department of Medicine, Erlangen University Hospital, Friedrich-Alexander-Universität Erlangen-Nürnberg, Erlangen, Germany; [2]Dr. Henry D Janowitz Division of Gastroenterology, Icahn School of Medicine at Mount Sinai, New York, NY, USA; [3]University Hospitals Leuven, Leuven, Belgium; [4]Inflammatory Bowel Disease Unit, Division of Gastroenterology and Hepatology, University of Calgary, Calgary, AB, Canada; [5]Monash Health, and Monash University, Melbourne, Australia; [6]Johnson & Johnson, Spring House, PA, USA; [7]Johnson & Johnson Innovative Medicine UK, High Wycombe, England; [8]Hospital Clínic de Barcelona, IDIBAPS, CIBERehd, Barcelona, Spain; [9]Cedars-Sinai, Los Angeles, CA, USA.

CH_CP-609070 10//26

Background

There is a high unmet need for the treatment of patients with UC refractory to multiple prior systemic therapies (biologics and oral targeted therapies), a growing population identified as “difficult-to-treat” by the IOIBD

We previously reported results from the DUET-UC study of JNJ-78934804 (JNJ-4804), the first fixed-dose, co-antibody therapy for IBD, developed using a biologically and clinically validated approach targeting the complementary IL-23 and TNF pathways

In DUET-UC, JNJ-4804 conferred the greatest clinical benefit versus guselkumab and golimumab monotherapies in participants with moderately to severely active UC refractory to 2 or more systemic therapy mechanisms,* a subgroup comprising nearly half the study population

*Anti-TNFs, anti-IL-12/23s, anti-IL-23p19s, anti-integrins, JAK inhibitors, S1PR modulators.
IBD, inflammatory bowel disease; IL, interleukin; IOIBD, International Organization for the Study of Inflammatory Bowel Diseases; JAK, Janus kinase; MOA, mechanism of action; RDBAPC, randomised, double-blind, active- and placebo-controlled; S1PR, sphingosine-1-phosphate receptor; TNF, tumour necrosis factor; UC, ulcerative colitis.
Atreya R, et al. Presented at UEGW, Barcelona, Spain, 17–20 October 2026. OP255.
JNJ-7893480 is investigational. JNJ-7893480 is not approved by Swissmedic for UC. Full prescribing information: swissmedicinfo-pro.ch.

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Endpoints and statistical considerations

Based on the totality of efficacy findings in the overall population and prespecified subgroups, the JNJ-4804 high dose regimen was selected for focused exploratory comparisons with the monotherapies and placebo in participants with inadequate response or intolerance to ≥2 approved systemic therapy mechanisms

Endpoints (Week 48)

- Clinical remission (primary endpoint in the overall population)
- Endoscopic improvement
- Histologic remission AND endoscopic improvement
- Corticosteroid-free clinical remission
- Faecal calprotectin (change from baseline)

Statistical considerations

- Participants who met prespecified treatment failure rules or had missing data were considered not to have met endpoints*
- Participants who met rescue criteria were considered treatment failures at Week 48*
- Post hoc analyses were conducted in the combined subpopulation with inadequate response or intolerance to 2 or more systemic therapy mechanisms, informed by prespecified analyses of 2 and >2 prior mechanism failures†
- All subgroup analyses were exploratory and were not controlled for multiplicity

*Participants were considered not to have met the Week 48 endpoint if any of the following occurred prior to Week 48: An ostomy or colectomy (partial or total); prohibited change in UC medication; treatment escalation due to inadequate response at Week 24; discontinuation of study treatment due to lack of efficacy or an AE of worsening UC; discontinuation of study treatment due to COVID-19 infection or any other reason. Participants who discontinued study treatment for COVID-19-related reasons (excluding COVID-19 infection) had their observed data used, if available. Missing data imputation: After accounting for these conditions, participants with missing Mayo subscores (any or all) at Week 48 (for clinical remission and corticosteroid-free clinical remission), missing endoscopy subscores at Week 48 (for endoscopic improvement and HREI), or missing histology data at Week 48 (for HREI) were considered not to have met the endpoint at Week 48; †Analyses of subpopulations with 1, 2 and >2 prior systemic therapy mechanisms-IR were prespecified; the combined ≥2 systemic therapy mechanisms-IR subpopulation was evaluated post hoc based on these results.

AE, adverse event; COVID-19, coronavirus disease 2019; HREI, histologic remission and endoscopic improvement; IL, interleukin; IR, inadequate response or intolerance; MOA, mechanism of action; RDBAPC, randomised, double-blind, active- and placebo-controlled; TNF, tumour necrosis factor; UC, ulcerative colitis.

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CH_CP-609070 10/26

Baseline demographics and disease characteristics

Modified full analysis set*	Placebo (n=51)	Golimumab (n=103)	Guselkumab (n=98)	JNJ-4804 Low dose (n=101)	JNJ-4804 Mid dose (n=103)	JNJ-4804 High dose (n=103)	Total (N=559)
Participants with IR to ≥2 ST mechanisms	21	47	50	38	45	45	246
Demographics and disease characteristics							
Age in years, mean (SD)	36.3 (11.41)	39.6 (13.20)	39.7 (12.93)	35.4 (12.36)	36.6 (13.37)	36.8 (12.22)	37.7 (12.71)
Male, n (%)	14 (66.7)	29 (61.7)	28 (56.0)	22 (57.9)	32 (71.1)	35 (77.8)	160 (65.0)
UC duration, years, mean (SD)	8.10 (5.185)	7.92 (7.419)	9.69 (6.556)	8.77 (6.765)	8.46 (5.714)	8.15 (6.376)	8.56 (6.450)
Extent of disease, n (%)	11 (52.4)	22 (46.8)	32 (64.0)	21 (55.3)	28 (62.2)	18 (40.0)	132 (53.7)
mMayo score, mean (SD)	7.0 (1.20)	7.3 (0.93)	7.0 (1.01)	7.0 (1.01)	7.1 (1.19)	7.2 (1.11)	7.1 (1.07)
mMayo 7–9 (severe UC), n (%)	13 (61.9)	37 (78.7)	32 (64.0)	28 (73.7)	31 (68.9)	34 (75.6)	175 (71.1)
Mayo Endoscopy Subscore of 3 (severe UC), n (%)	17 (81.0)	38 (80.9)	37 (74.0)	25 (65.8)	31 (68.9)	33 (73.3)	181 (73.6)
Concomitant medications							
Immunomodulators, n (%)	2 (9.5)	5 (10.6)	4 (8.0)	3 (7.9)	4 (8.9)	5 (11.1)	23 (9.3)
Oral corticosteroids, n (%)	8 (38.1)	20 (42.6)	24 (48.0)	18 (47.4)	25 (55.6)	28 (62.2)	123 (50.0)
Inflammatory pharmacodynamic markers							
Elevated faecal calprotectin (>250 mg/kg), n (%)†	18 (85.7)	42 (97.7)	38 (86.4)	33 (100.0)	35 (92.1)	38 (92.7)	204 (92.7)
Elevated CRP (>5 mg/L), n (%)‡	10 (47.6)	25 (56.8)	27 (54.0)	19 (50.0)	20 (44.4)	20 (44.4)	121 (49.8)

Baseline demographics and disease characteristics in the ≥2 systemic therapy mechanisms-IR subpopulation reflect a refractory population

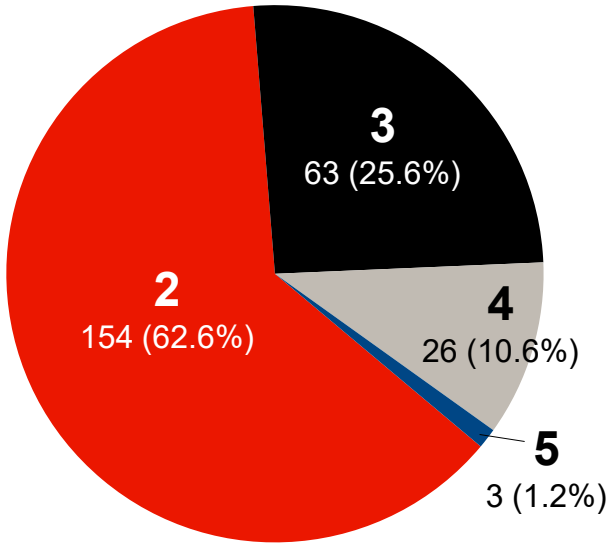
*Modified full analysis set: full analysis set excluding participants with a modified Mayo <5 or missing at baseline. †Numbers and percentages are based on the following numbers of participants with non-missing data: 21 (PBO), 43 (GOL), 44 (GUS), 33 (JNJ-4804 low dose), 38 (JNJ-4804 mid dose), 41 (JNJ-4804 high dose). ‡Numbers and percentages are based on the following numbers of participants with non-missing data: 21 (PBO), 44 (GOL), 50 (GUS), 38 (JNJ-4804 low dose), 45 (JNJ-4804 mid dose), 45 (JNJ-4804 high dose).
CRP, C-reactive protein; GOL, golimumab; GUS, guselkumab; IL, interleukin; IR, inadequate response or intolerance; mMayo, modified Mayo score; MOA, mechanism of action; PBO, placebo; RDBAPC, randomised, double-blind, active- and placebo-controlled; SD, standard deviation; ST, systemic therapy; TNF, tumour necrosis factor; UC, ulcerative colitis.
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Treatment-refractory population with inadequate response or intolerance to ≥2 systemic therapy mechanisms

Modified full analysis set*	Placebo (n=51)	Golimumab (n=103)	Guselkumab (n=98)	JNJ-4804 Low dose (n=101)	JNJ-4804 Mid dose (n=103)	JNJ-4804 High dose (n=103)	Total (N=559)
Participants with IR to ≥2 ST mechanisms, n (%)†	21 (41.2)	47 (45.6)	50 (51.0)	38 (37.6)	45 (43.7)	45 (43.7)	246 (44.0)

Number of systemic therapy mechanisms failed



Previously failed ST mechanism	Participants, n	% of ≥ST mechanisms-IR subset (N=246)
Anti-TNF	222	90.2%
Anti-integrin	169	68.7%
JAK inhibitor	113	45.9%
IL-12/23 antagonist	93	37.8%
S1PR modulator	15	6.1%
IL-23 antagonist	4	1.6%

Most common combinations:

- Anti-TNF + anti-integrin (28.5%)
- Anti-TNF + JAK inhibitor (14.2%)
- Anti-TNF + anti-integrin + JAK inhibitor (13.0%)

*Modified full analysis set: Full analysis set excluding participants with an mMayo <5 or missing at baseline; †Anti-TNF, anti-IL-12/23, anti-IL-23p19, anti-integrin, JAK inhibitor and S1PR modulator.
IL, interleukin; IR, inadequate response or intolerance; JAK, Janus kinase; mMayo, modified Mayo score; MOA, mechanism of action; RDBAPC, randomised, double-blind, active- and placebo-controlled; S1PR, sphingosine-1-phosphate receptor; ST, systemic therapy; TNF, tumour necrosis factor; UC, ulcerative colitis.
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Treatment disposition through Week 48 in the ≥2 systemic therapy mechanisms-IR subpopulation

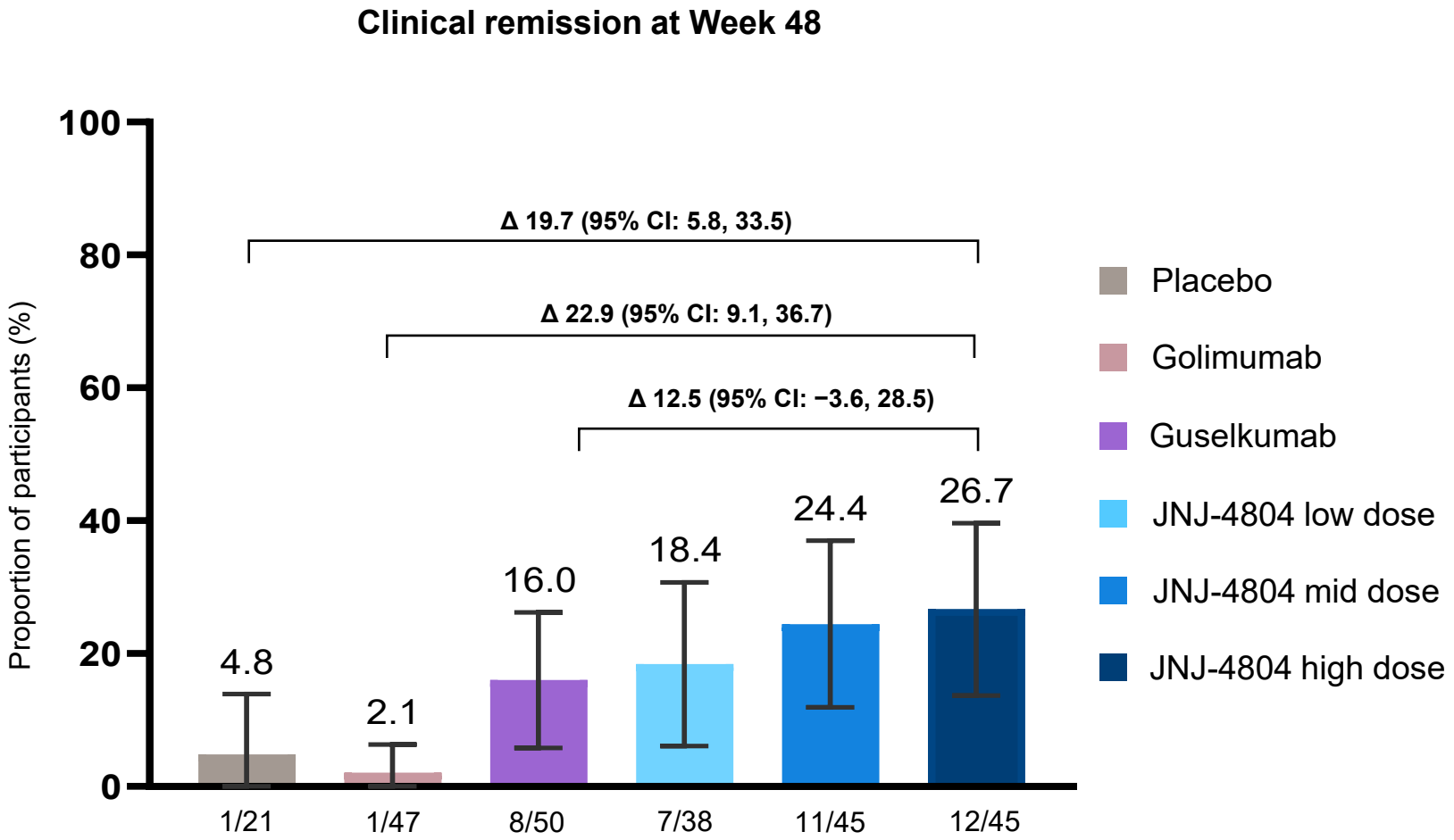
Modified full analysis set*	Placebo (n=51)	Golimumab (n=103)	Guselkumab (n=98)	JNJ-4804 Low dose (n=101)	JNJ-4804 Mid dose (n=103)	JNJ-4804 High dose (n=103)
Participants with IR to ≥2 ST mechanisms, n	21	47	50	38	45	45
Discontinued study treatment before Week 48, n (%)	8 (38.1)	14 (29.8)	18 (36.0)	10 (26.3)	11 (24.4)	9 (20.0)
Reasons for discontinuing study treatment, n (%)						
Lack of efficacy	2 (9.5)	3 (6.4)	10 (20.0)	4 (10.5)	2 (4.4)	4 (8.9)
Adverse event – worsening of UC	3 (14.3)	5 (10.6)	3 (6.0)	2 (5.3)	3 (6.7)	4 (8.9)
Withdrawal by participant	3 (14.3)	4 (8.5)	3 (6.0)	1 (2.6)	5 (11.1)	1 (2.2)
Adverse event – other	0	1 (2.1)	0	2 (5.3)	1 (2.2)	0
Initiated prohibited medication	0	0	2 (4.0)	0	0	0
Protocol deviation	0	0	0	1 (2.6)	0	0
Other	0	1 (2.1)	0	0	0	0

Participants are presented in the treatment group assigned at Week 0. *Modified full analysis set: full analysis set excluding participants with an mMayo <5 or missing at baseline.
IL, interleukin; IR, inadequate response or intolerance; mMayo, modified Mayo score; MOA, mechanism of action; RDBAPC, randomised, double-blind, active- and placebo-controlled; ST, systemic therapy; TNF, tumour necrosis factor; UC, ulcerative colitis.
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Clinical remission at Week 48

≥2 systemic therapy mechanisms-IR subpopulation: Modified full analysis set*



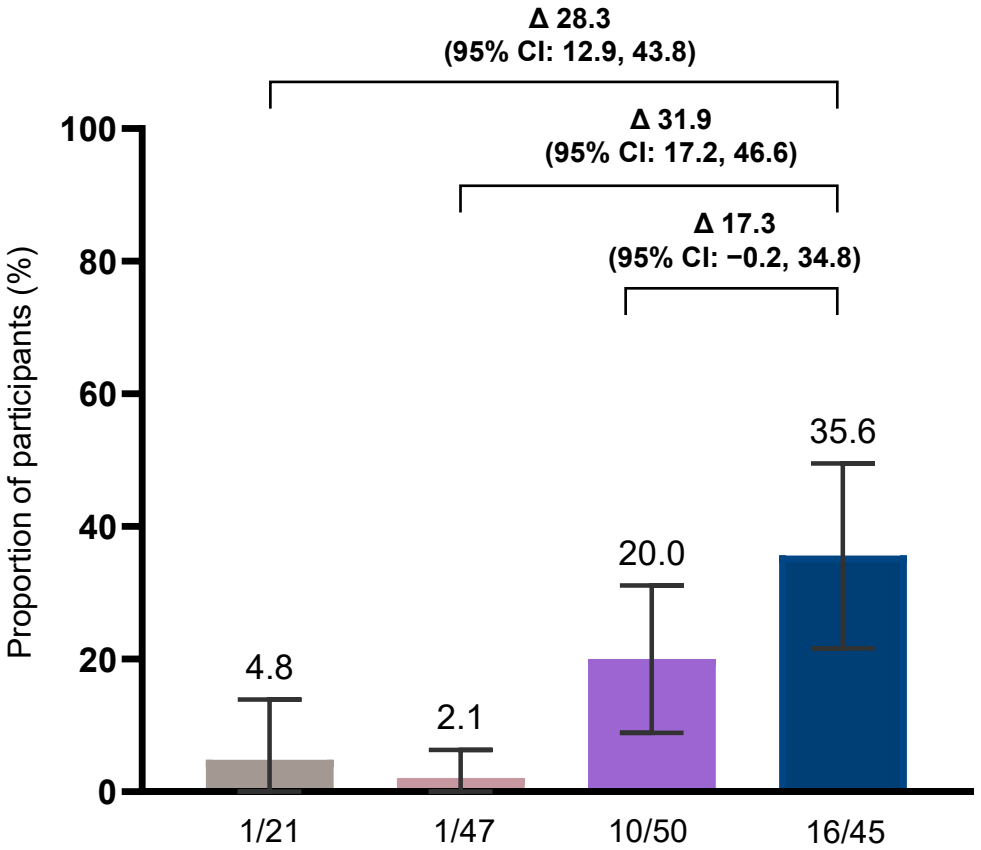
Clinical remission: A stool frequency subscore of 0 or 1, a rectal bleeding subscore of 0 and an endoscopy subscore of 0 or 1 per central review of the video endoscopy.
*Modified full analysis set: Full analysis set excluding participants with an mMayo <5 or missing at baseline.
CI, confidence interval; IL, interleukin; IR, inadequate response or intolerance; mMayo, modified Mayo score; MOA, mechanism of action; RDBAPC, randomised, double-blind, active- and placebo-controlled; TNF, tumour necrosis factor; UC, ulcerative colitis; Δ, adjusted risk difference.
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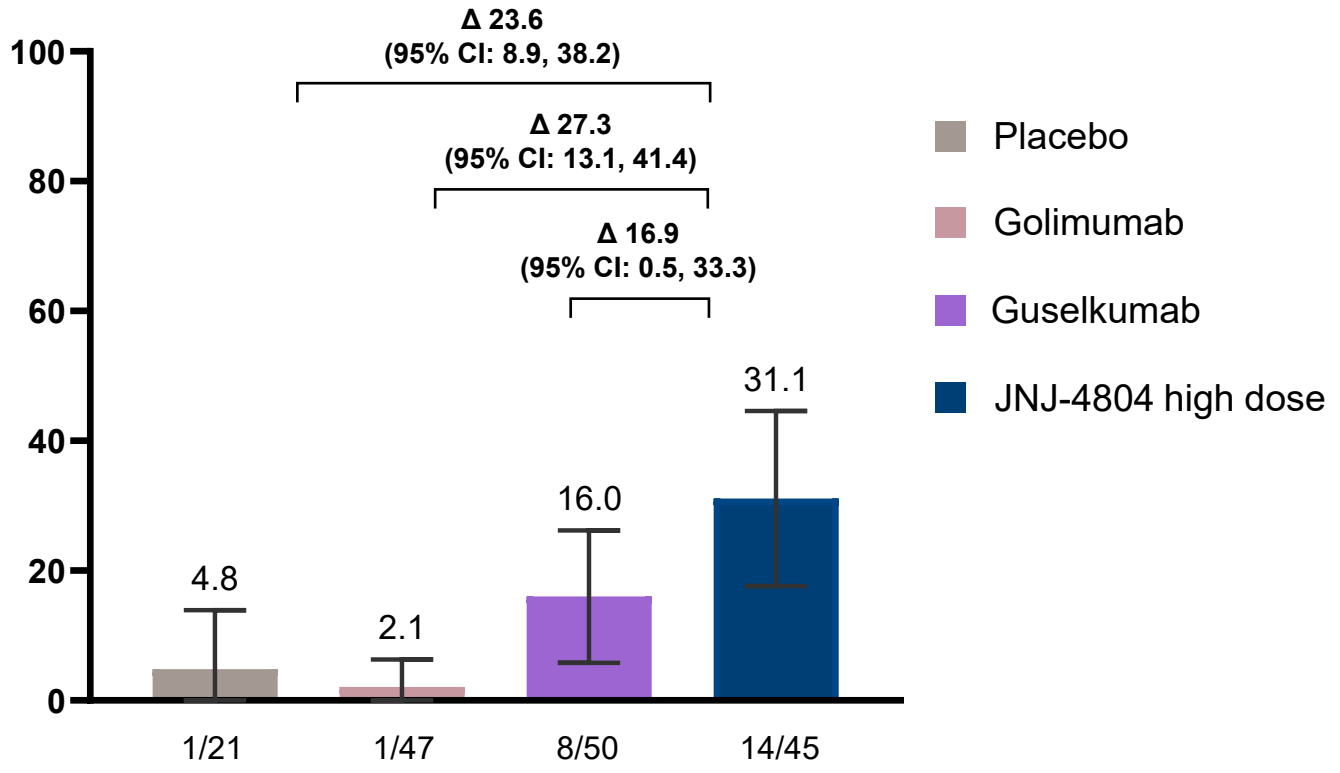
Endoscopic and histological endpoints at Week 48

≥2 systemic therapy mechanisms-IR subpopulation: Modified full analysis set*

Endoscopic improvement at Week 48



Histological remission and endoscopic improvement at Week 48



Endoscopic improvement: An endoscopy subscore of 0 or 1 per central review of the video endoscopy. **HREI:** Both histologic remission (absence of neutrophils from the mucosa [both lamina propria and epithelium], no crypt destruction, and no erosions, ulcerations or granulation tissue [Geboes score ≤2B.0]) and endoscopic improvement.

*Modified full analysis set: Full analysis set excluding participants with an mMayo <5 or missing at baseline.

CI, confidence interval; HREI, histologic remission and endoscopic improvement; IL, interleukin; IR, inadequate response or intolerance; mMayo, modified Mayo score; MOA, mechanism of action; RDBAPC, randomised, double-blind, active- and placebo-controlled; TNF, tumour necrosis factor; UC, ulcerative colitis; Δ, adjusted rate difference.

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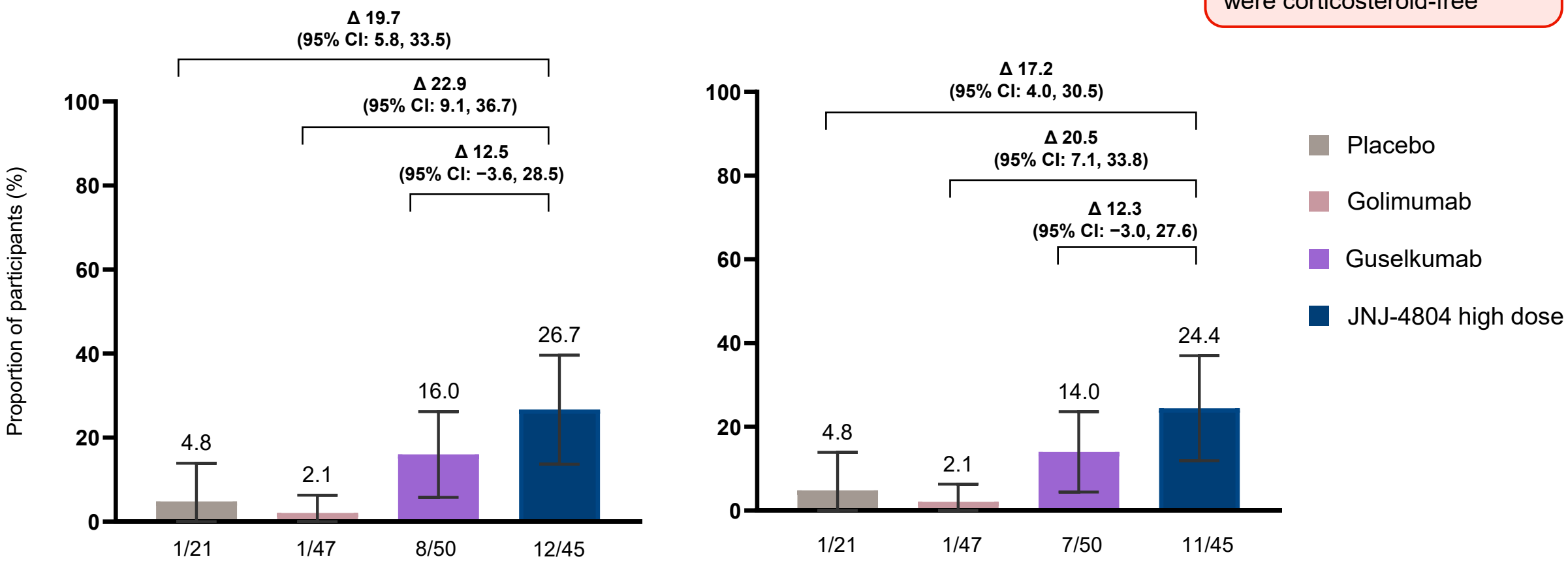
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Corticosteroid-free clinical remission at Week 48

≥2 systemic therapy mechanisms-IR subpopulation: Modified full analysis set*

Nearly all participants in clinical remission at Week 48 were corticosteroid-free



Corticosteroid-free clinical remission (60-day): Clinical remission with no concomitant corticosteroids for ≥60 days prior to Week 48. **Clinical remission:** A stool frequency subscore of 0 or 1, a rectal bleeding subscore of 0 and an endoscopy subscore of 0 or 1 per central review of the video endoscopy.

*Modified full analysis set: Full analysis set excluding participants with an mMayo <5 or missing at baseline.

CI, confidence interval; IL, interleukin; IR, inadequate response or intolerance; mMayo, modified Mayo score; MOA, mechanism of action; RDBAPC, randomised, double-blind, active- and placebo-controlled; TNF, tumour necrosis factor; UC, ulcerative colitis; Δ, adjusted rate difference.

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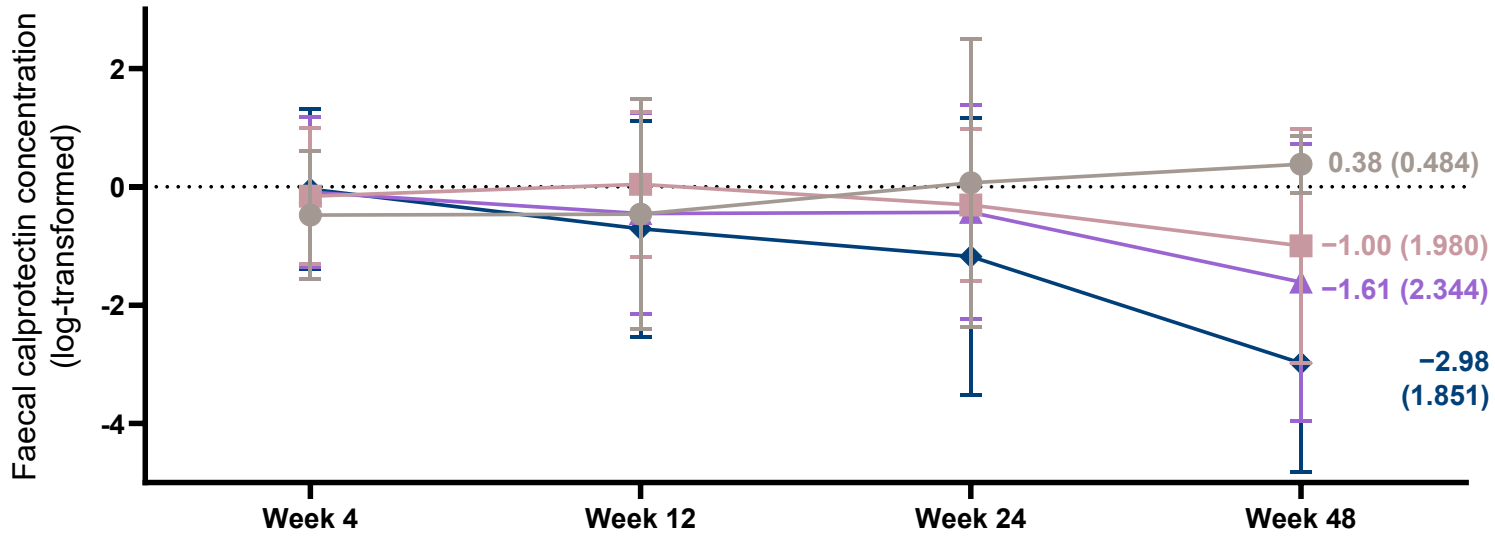
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Faecal calprotectin: Mean change from baseline to Week 48

≥2 systemic therapy mechanisms-IR subpopulation: Modified full analysis set*

Mean (SD) change from baseline in faecal calprotectin concentration



	Week 4	Week 12	Week 24	Week 48
JNJ-4804 high dose	n=38	34	39	16
Placebo	n=18	17	13	2
Golimumab	n=41	40	33	9
Guselkumab	n=38	37	35	18

Data are as-observed, log-transformed (natural log) and exclude inadequate responders after Week 24 treatment escalation.
*Modified full analysis set: Full analysis set excluding participants with an mMayo <5 or missing at baseline.
IL, interleukin; IR, inadequate response or intolerance; mMayo, modified Mayo score; MOA, mechanism of action; RDBAPC, randomised, double-blind, active- and placebo-controlled; SD, standard deviation; TNF, tumour necrosis factor; UC, ulcerative colitis.
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Summary of exposure-adjusted adverse events through Week 48

≥2 systemic therapy mechanisms-IR subpopulation

Safety analysis set	Placebo (n=52)	Golimumab (n=104)	Guselkumab (n=103)	JNJ-4804 Low dose (n=103)	JNJ-4804 Mid dose (n=105)	JNJ-4804 High dose (n=105)
Participants with IR to ≥2 ST mechanisms	21 (40.4%)	48 (46.2%)	52 (50.5%)	40 (38.8%)	47 (44.8%)	46 (43.8%)
Total patient-years of follow-up	9.3	27.0	33.0	25.1	33.8	33.8
Event rate per hundred patient-years [number of events] (95% CI)						
AEs	568.4 [53] (425.74, 743.43)	618.0 [167] (527.82, 719.17)	587.6 [194] (507.82, 676.36)	454.3 [114] (374.72, 545.72)	497.3 [168] (424.98, 578.49)	679.5 [230] (594.52, 773.23)
SAEs	32.2 [3] (6.63, 94.02)	29.6 [8] (12.78, 58.33)	18.2 [6] (6.67, 39.56)	19.9 [5] (6.47, 46.50)	11.8 [4] (3.23, 30.32)	23.6 [8] (10.20, 46.57)
AEs leading to discontinuation of study treatment	32.2 [3] (6.63, 94.02)	22.2 [6] (8.15, 48.33)	9.1 [3] (1.87, 26.55)	19.9 [5] (6.47, 46.50)	11.8 [4] (3.23, 30.32)	8.9 [3] (1.83, 25.90)
Infections	64.3 [6] (23.61, 140.05)	129.5 [35] (90.22, 180.13)	121.2 [40] (86.55, 164.98)	107.6 [27] (70.90, 156.54)	136.2 [46] (99.70, 181.64)	100.4 [34] (69.56, 140.37)
Serious infections	0.0 [0] (0.00, 32.13)	7.4 [2] (0.90, 26.74)	0.0 [0] (0.00, 9.07)	4.0 [1] (0.10, 22.20)	5.9 [2] (0.72, 21.39)	8.9 [3] (1.83, 25.90)
Deaths	0.0 [0] (0.00, 32.13)	0.0 [0] (0.00, 11.09)	0.0 [0] (0.00, 9.07)	0.0 [0] (0.00, 11.94)	0.0 [0] (0.00, 8.87)	0.0 [0] (0.00, 8.85)

Safety events per 100 patient-years with JNJ-4804 in the subpopulation were overall similar to or lower than those in the monotherapy groups

Excludes inadequate responder events after treatment escalation at Week 24. There were no deaths per the Case Report Form.
AE, adverse event; CI, confidence interval; IL, interleukin; IR, inadequate response or intolerance; MOA, mechanism of action; RDBAPC, randomised, double-blind, active- and placebo-controlled; SAE, serious adverse event; ST, systemic therapy; TNF, tumour necrosis factor; UC, ulcerative colitis.
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Treatment-emergent adverse events of interest through Week 48

≥2 systemic therapy mechanisms-IR subpopulation

Safety analysis set	Placebo (n=52)	Golimumab (n=104)	Guselkumab (n=103)	JNJ-4804 Low dose (n=103)	JNJ-4804 Mid dose (n=105)	JNJ-4804 High dose (n=105)
Participants with IR to ≥2 ST mechanisms	21 (40.4%)	48 (46.2%)	52 (50.5%)	40 (38.8%)	47 (44.8%)	46 (43.8%)
Participants with ≥1 AE of interest	0	2 (4.2%)	1 (1.9%)	2 (5.0%)	2 (4.3%)	3 (6.5%)
Participants with ≥1 AE of special interest	0	2 (4.2%)	0	2 (5.0%)	2 (4.3%)	2 (4.3%)
Serious infection	0	2 (4.2%)	0	1 (2.5%)	2 (4.3%)	2 (4.3%)
Invasive fungal infection	0	0	0	0	0	0
Hepatitis B reactivation	0	0	0	0	0	0
Tuberculosis	0	0	0	0	0	0
Malignancy	0	0	0	0	0	0
Hypersensitivity reaction	0	0	0	1 (2.5%)	0	0
Congestive heart failure	0	0	0	0	0	0
Demyelinating disorders	0	0	0	0	0	0
Lupus-like syndrome	0	0	0	0	0	0
Major adverse cardiovascular events	0	0	0	0	0	0
Venous thromboembolism	0	1 (2.1%)	0	0	0	1 (2.2%)
Drug-related hepatic disorders	0	0	0	0	0	0
Opportunistic infection	0	0	1 (1.9%)	0	0	0

Excludes inadequate responder events after treatment escalation at Week 24.
AE, adverse event; IL, interleukin; IR, inadequate response or intolerance; MOA, mechanism of action; RDBAPC, randomised, double-blind, active- and placebo-controlled; ST, systemic therapy; TNF, tumour necrosis factor; UC, ulcerative colitis.
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Conclusions

- JNJ-4804, the first co-antibody therapy in development for IBD, delivered substantial and clinically meaningful efficacy across all endpoints in participants with UC refractory to 2 or more approved systemic therapy mechanisms, a growing population of high unmet need identified as “difficult to treat” by the IOIBD
- The magnitude of benefit observed with JNJ-4804 high dose was greater than that observed with either component monotherapy across multiple symptoms-based and objective endpoints
- No new safety signals were identified in this exploratory subgroup through Week 48, though interpretation is limited by small treatment groups and low numbers of individual events
- These exploratory findings should be interpreted in the context of the post hoc combined subgroup, limited sample sizes, and lack of multiplicity control
- These results support advancement to Phase 3 in the rapidly increasing population of participants with disease refractory to systemic therapies

IBD, inflammatory bowel disease; IL, interleukin; IOIBD, International Organization for the Study of Inflammatory Bowel Diseases; MOA, mechanism of action; RDBAPC, randomised, double-blind, active- and placebo-controlled; TNF, tumour necrosis factor; UC, ulcerative colitis.

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Tremfya® Abbreviated Information for Professionals

TREMFYA® (Guselkumab, humaner IgG1λ-mAb) Injektionslösung zur subkutanen oder intravenösen Anwendung.
I: indiziert zur Behandlung von erwachsenen und pädiatrischen Patienten ab 6 Jahren mit einem Körpergewicht von mindestens 12 kg mit mittelschwerer bis schwerer, chronischer Plaque-Psoriasis, die für eine systemische Therapie in Frage kommen, aktiver Psoriasis-Arthritis (PsA)– allein oder in Kombination mit Methotrexat, die auf eine vorgängige Therapie mit einem krankheitsmodifizierenden Antirheumatikum (dmARD) unzureichend angesprochen oder nicht vertragen haben, sowie mittelschwerer bis schwerer aktiver Colitis ulcerosa (UC) oder Morbus Crohn (CD), die entweder auf eine konventionelle Therapie oder ein Biologikum unzureichend angesprochen haben, nicht mehr ansprechen oder diese nicht vertragen haben. **D:** Anwendung unter Anleitung und Aufsicht eines in der Behandlung dieser Erkrankungen erfahrenen Arztes. Erwachsene: Nach Schulung ist Selbstinjektion möglich. Für Psoriasis und Psoriasis-Arthritis beträgt die empfohlene Dosis 100mg als s.c. Injektion in Woche 0 und 4, danach alle 8 Wochen (q8w). Bei Colitis ulcerosa oder Morbus Crohn beträgt die Induktionsdosis 200mg i.v. oder alternativ 400mg s.c. in Woche 0, 4 und 8, gefolgt von einer Erhaltungsdosis von 100mg s.c. ab Woche 16 danach q8w. Alternativ kann nach klinischem Ermessen eine Erhaltungsdosierung von 200mg als s.c. Injektion in Woche 12 danach q4w erwogen werden. Bei fehlendem Ansprechen nach 12 (UC und CD), nach 16 (PsO) oder 24 (PsA) Wochen, Therapieabbruch erwägen. Kinder und Jugendliche ab 6 Jahren mit einem Körpergewicht von mindestens 12kg: ≥40kg:100 mg mit der 1-ml-Fertigspritze verabreicht. Patienten <40kg Verabreichung mit 45-mg-/0,45-ml-Fertigpen bis zu einer maximalen Dosis von 45 mg. Empfohlene Dosierung: 12-15kg: 20mg, 16-19kg: 25mg, 20-23kg: 30mg, 24-26kg: 35mg, 27-30kg: 40mg, 31-39kg: 45mg s.c.

KI: Schwere Überempfindlichkeit gegenüber Wirkstoff/Hilfsstoffe. Klinisch relevante aktive Infektionen (z. B. aktive Tuberkulose). **VM:** Bei schweren Infektionen: engmaschige Überwachung und Therapiepause bis zur Ausheilung. Keine Lebendimpfstoffe während der Therapie. Vor Therapiebeginn Abklärung auf Tuberkulose (TB), ggf. prophylaktische Behandlung bei latenter TB. Bei schwerwiegenden Überempfindlichkeitsreaktionen Therapie absetzen. **UAW:** Sehr häufig: Atemwegsinfektionen; Häufig: Herpes-simplex-Infektionen, Tinea-Infektionen, Gastroenteritis, Kopfschmerzen, Diarrhö, Transaminasen erhöht, Arthralgie, Reaktionen an der Injektionsstelle, Überempfindlichkeit, Urtikaria, Ausschläge. Weitere UAW s. FI. **IA:** Bisher keine klinisch relevanten Interaktionen beobachtet. **Packungen:** Injektionslösung in Fertigspritze, Fertigpen oder Fertigpen - PushPen (100mg/1ml oder 200mg/2ml); 45 mg/0,45 ml Fertigpen; Konzentrat zur Herstellung einer Infusionslösung in einer Durchstechflasche (200mg/20ml). **Abgabekat.:** B. **Ausführliche Informationen:** www.swissmedic.ch oder www.swissmedicinfo-pro.ch **Zulassungsinhaberin:** Janssen-Cilag AG, Gubelstrasse 34, 6300 Zug (CP-586349-06/26)

Ulcerative colitis at 40x magnification