ECCO 2026: Data reflow deck

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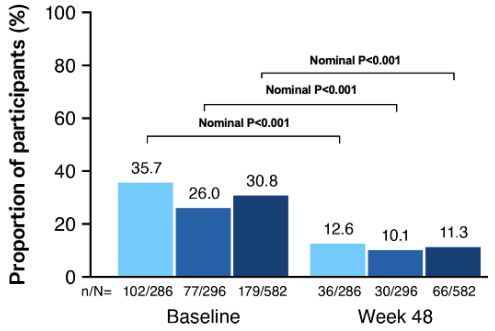
Immunology

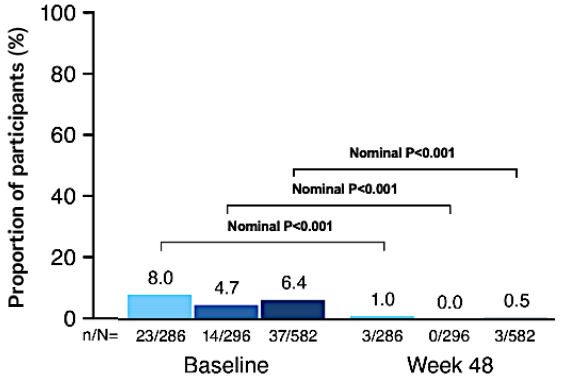
ECCO 2026 GUS highlights

GALAXI 2 and 3: Extraintestinal manifestations in CD[1]

Arthritis/arthralgia



Erythema nodosum / pyoderma gangrenosum

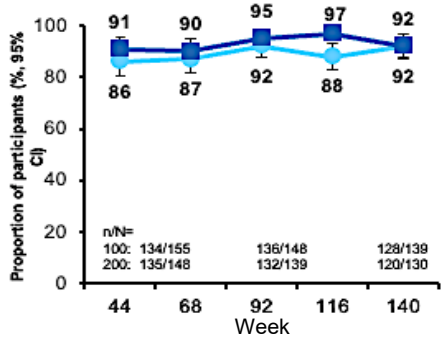


GUS 200 mg IV → 100 mg SC q8w | GUS 200 mg IV → 200 mg SC q4w | GUS combined

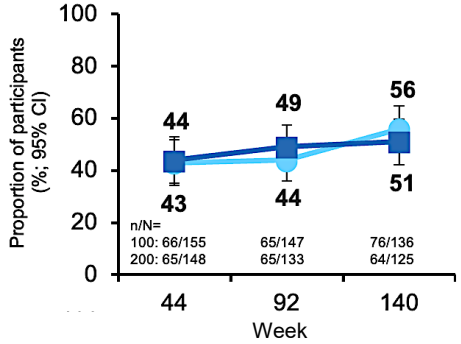
- GUS-treated participants with CD had greater EIM resolution and lower rates of *de novo* EIMs at Week 12 vs placebo
- EIM resolution continued through Week 48 and was not dependent on corticosteroid use
- These results suggest GUS may improve and prevent EIMs in patients with CD

QUASAR: Efficacy and safety at Week 140 in UC[2]

Symptomatic remission



Endoscopic remission (normalisation)

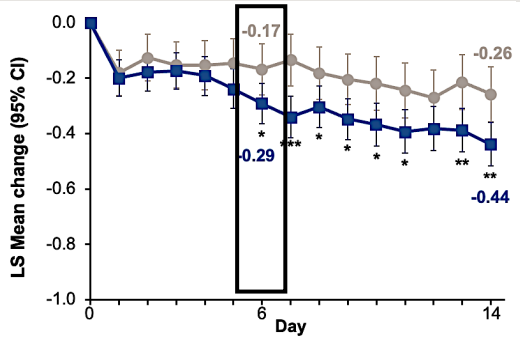


GUS 100 mg SC q8w (AO) | GUS 200 mg SC q4w (AO)

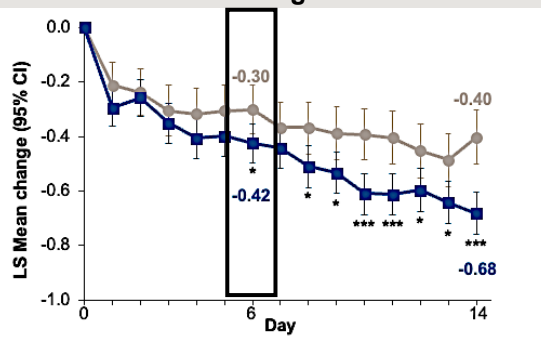
- Both GUS maintenance dose regimens demonstrated sustained clinical, endoscopic and histologic efficacy in participants with UC through Week 140 of the LTE
- Although NRI results were numerically lower than AO results, the overall trends were consistent due to the high retention rate throughout the LTE

QUASAR: Early symptomatic response in UC[3]

Stool frequency subscore



Rectal bleeding subscore



PBO IV | GUS 200 mg IV

Nominal *p<0.05, **p<0.01, ***p<0.001 vs placebo

- GUS demonstrated very early symptomatic improvement in participants with moderately to severely active UC
- This early improvement is associated with long-term outcomes

Endoscopic remission: MES of 0. **Symptomatic remission:** SFS of 0 or 1 and not increased from induction baseline, RBS of 0

AO, as observed; CD, Crohn's disease; CI, confidence interval; EIM, extraintestinal manifestation; GUS, guselkumab; IV, intravenous; LTE, long-term extension; MES, Mayo Endoscopic Score; NRI, non-responder imputation; PBO, placebo; qXw, every X weeks; RBS, rectal bleeding subscore; SC, subcutaneous; SFS, stool frequency subscore; UC, ulcerative colitis.

1. Danese S, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. DOP001; 2. Peyrin-Biroulet L, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. DOP104; 3. Dignass A, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. OP10. Full prescribing information: www.swissmedicinfo-pro.ch.

Contents (1/3)

All data | Key data only | Abstract text only

Guselkumab

Crohn's disease

GALAXI	Extraintestinal manifestations in participants with moderately to severely active Crohn's disease: Results from the phase 3 GALAXI 2 & 3 studies	Danese, et al.	All data
	Efficacy and safety of guselkumab in participants with moderately to severely active Crohn's disease who had maintenance dose adjustment: Results from the phase 3 GALAXI 2 & 3 long-term extension	Panaccione, et al.	All data
	Impact of disease duration on clinical and endoscopic responses at 1 year in patients with Crohn's disease treated with guselkumab: Pooled analysis of the GALAXI 2 & 3 studies	Ferrante, et al.	All data
GALAXI-GRAVITI	Efficacy of intravenous and subcutaneous guselkumab induction by weight and body mass index in patients with Crohn's disease: Results from the phase 3 GALAXI and GRAVITI studies	Deepak, et al.	All data
	Unsupervised machine learning to identify distinct response patterns to guselkumab in participants with Crohn's disease: *Post-hoc* analysis of the GRAVITI and GALAXI 2/3 studies	Schreiber, et al	Abstract text only

Ulcerative colitis

QUASAR	Efficacy and safety of guselkumab for ulcerative colitis through week 140 of the QUASAR long-term extension study	Peyrin-Biroulet, et al.	All data
	Symptomatic improvement with intravenous guselkumab induction therapy is observed early in patients with moderately to severely active ulcerative colitis: *Post-hoc* analysis of QUASAR	Dignass, et al.	All data
	Predictors of endoscopic remission at 1 year in patients with ulcerative colitis treated with guselkumab: Post-hoc analyses of the QUASAR trial	Rubin, et al.	All data
	Association of endoscopic, histologic, and composite outcomes with long-term guselkumab efficacy in ulcerative colitis: 2-year results from the QUASAR long-term extension	Magro, et al.	Key data only

*Denotes encore.

Contents (2/3)

All data | Key data only | Abstract text only

Guselkumab

*Denotes encore.

BMI, body mass index; CD, Crohn's disease; IBD, inflammatory bowel disease; IL, interleukin; PsA, psoriatic arthritis; PsO, psoriasis; UC, ulcerative colitis.

Contents (3/3)

All data | Key data only | Abstract text only

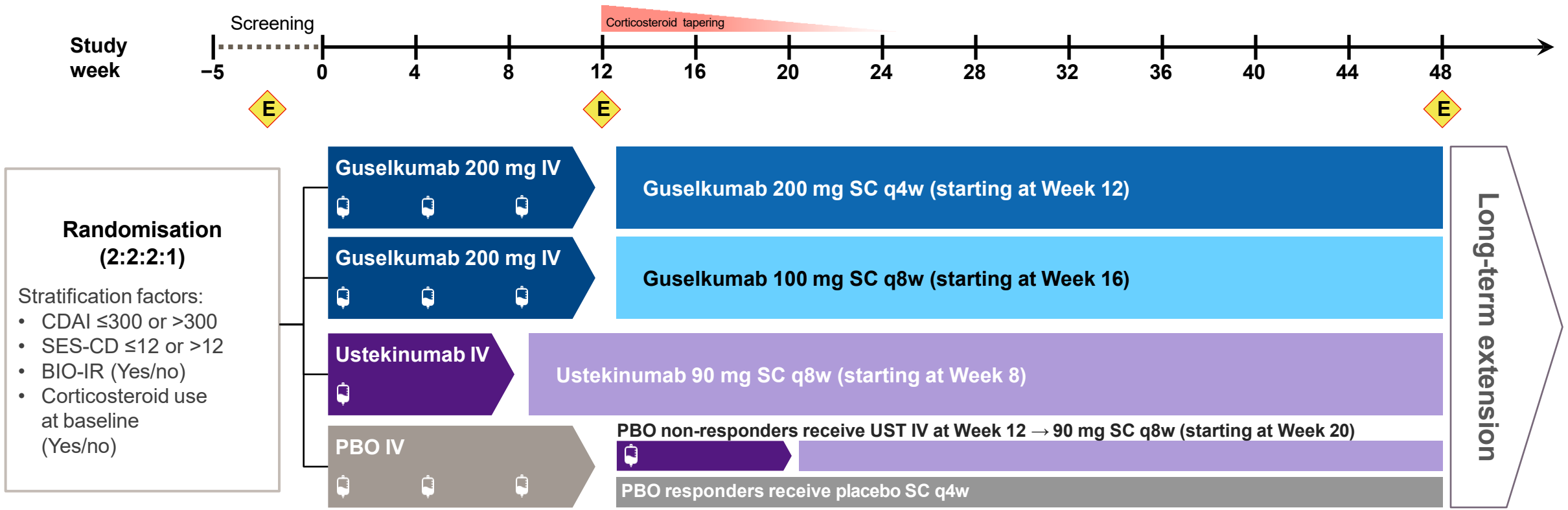
IBD, inflammatory bowel disease; PCDAI, Paediatric Crohn's Disease Activity Index.

Guselkumab in Crohn’s disease

GALAXI

Immunology

GALAXI 2 and 3 study design



Key eligibility criteria

- Moderately to severely active Crohn's disease (CDAI score 220–450 + mean daily SF count >3 or AP score >1) and SES-CD score ≥0.6 (or ≥0.4 for isolated ileal disease)
- Inadequate response/intolerance to oral corticosteroids or 6-MP/AZA/MTX or biologic therapies, or naïve to biologics

To maintain treatment masking, all participants received active and/or placebo IV q4w through Week 12 and active and/or placebo SC q4w through Week 48.
6-MP, 6-mercaptopurine; AP, abdominal pain; AZA, azathioprine; BIO-IR, inadequate response/intolerance to biologic therapy; CD, Crohn's disease; CDAI, Crohn's Disease Activity Index; E, endoscopy; GUS, guselkumab; IL, interleukin; IV, intravenous; MOA, mechanism of action; MTX, methotrexate; PBO, placebo; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous; SES-CD, Simple Endoscopic Score for Crohn's Disease; SF, stool frequency; UST, ustekinumab.
Danese S, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. DOP001. Full prescribing information: www.swissmedicinfo-pro.ch.

DOP001

Extraintestinal manifestations in participants with moderately to severely active Crohn's disease: Results from the Phase 3 GALAXI 2 & 3 studies

Danese S,[1] Hisamatsu T,[2] Rampelbergh R Van,[3] Duijnhoven W Van,[3] Scapini G,[4] Adsul S,[4] Rubin DT[5]

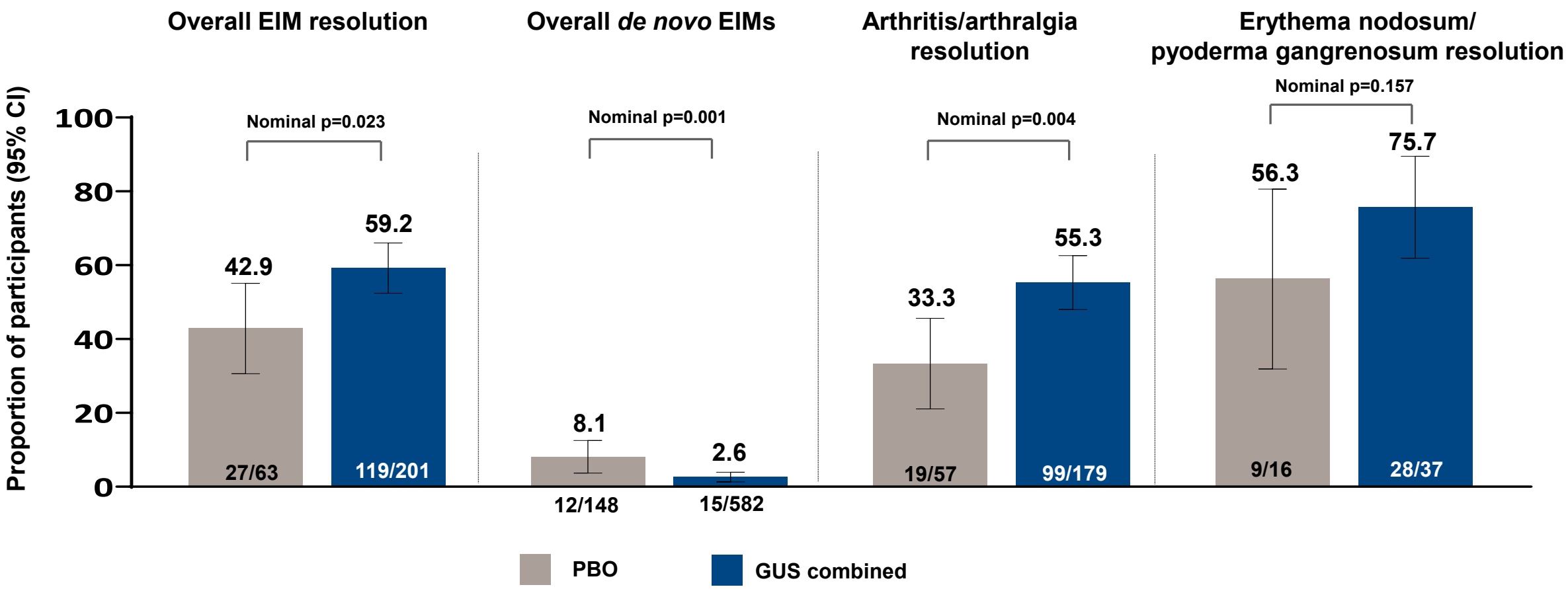
[1]IRCCS Ospedale San Raffaele and University Vita-Salute San Raffaele, Milano, Italy; [2]Department of Gastroenterology and Hepatology, Kyorin University, Tokyo, Japan; [3]Johnson & Johnson, Antwerp, Belgium; [4]Johnson & Johnson, Horsham, PA, USA; [5]University of Chicago Medicine Inflammatory Bowel Disease Center, Chicago, IL, USA

Pooled baseline demographics and disease characteristics

	PBO	GUS 200 mg IV q4w→ 100 mg SC q8w	GUS 200 mg IV q4w→ 200 mg SC q4w	GUS combined
Primary analysis set, N	**148**	**286**	**296**	**582**
Number of participants with EIMs at baseline, n (%)	63 (42.6)	115 (40.2)	86 (29.1)	201 (34.5)
Demographics				
Age, years, mean (SD)	37.7 (13.16)	37.2 (12.11)	39.1 (13.81)	38.0 (12.86)
Men, n (%)	36 (57.1)	57 (49.6)	44 (51.2)	101 (50.2)
CD duration, years, mean (SD)	6.9 (7.34)	7.4 (7.01)	7.9 (8.23)	7.6 (7.54)
Characteristics				
CDAI score at baseline, mean (SD)	291.6 (52.46)	302.8 (55.24)	302.1 (51.77)	302.5 (53.65)
Involved GI areas (as assessed by central reader), n (%)				
Ileum only	17 (27.0)	30 (26.1)	32 (37.2)	62 (30.8)
Colon only	23 (36.5)	40 (34.8)	26 (30.2)	66 (32.8)
Ileum and colon	23 (36.5)	45 (39.1)	28 (32.6)	73 (36.3)
EIMs, n (%)*				
Arthritis/arthralgia	57 (90.5)	102 (88.7)	77 (89.5)	179 (89.1)
Erythema nodosum/pyoderma gangrenosum	16 (25.4)	23 (20.0)	14 (16.3)	37 (18.4)
Iritis/uveitis	2 (3.2)	5 (4.3)	6 (7.0)	11 (5.5)
Corticosteroid use, n (%)				
Oral corticosteroids	16 (25.4)	37 (32.2)	23 (26.7)	60 (29.9)
Budesonide	12 (20.6)	17 (14.8)	13 (15.1)	30 (14.9)
Prior use of biologics, n (%)				
Adalimumab	19 (30.2)	45 (39.1)	29 (33.7)	74 (36.8)
Infliximab	22 (34.9)	37 (32.2)	27 (31.4)	64 (31.8)
Vedolizumab	6 (9.5)	13 (11.3)	6 (7.0)	19 (9.5)
Certolizumab pegol	1 (1.6)	4 (3.5)	3 (3.5)	7 (3.5)

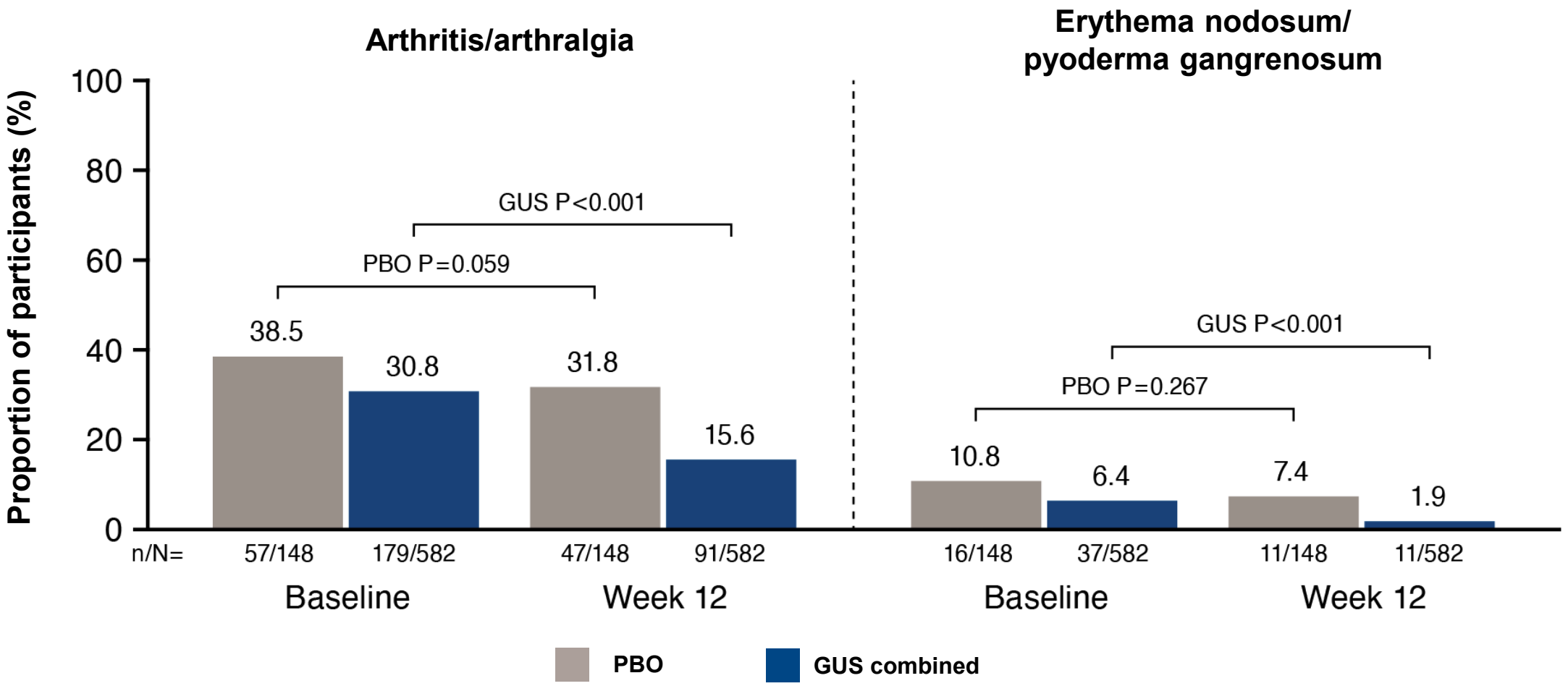
CD, Crohn's disease; CDAI, Crohn's Disease Activity Index; EIM, extraintestinal manifestation; GI, gastrointestinal; GUS, guselkumab, IL, interleukin; IV, intravenous; MOA, mechanism of action; PBO, placebo; qXw, every X weeks;
RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous; SD, standard deviation.
Danese S, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. DOP001. Full prescribing information: www.swissmedicinfo-pro.ch.

Week 12 EIM outcomes among participants with EIMs at baseline



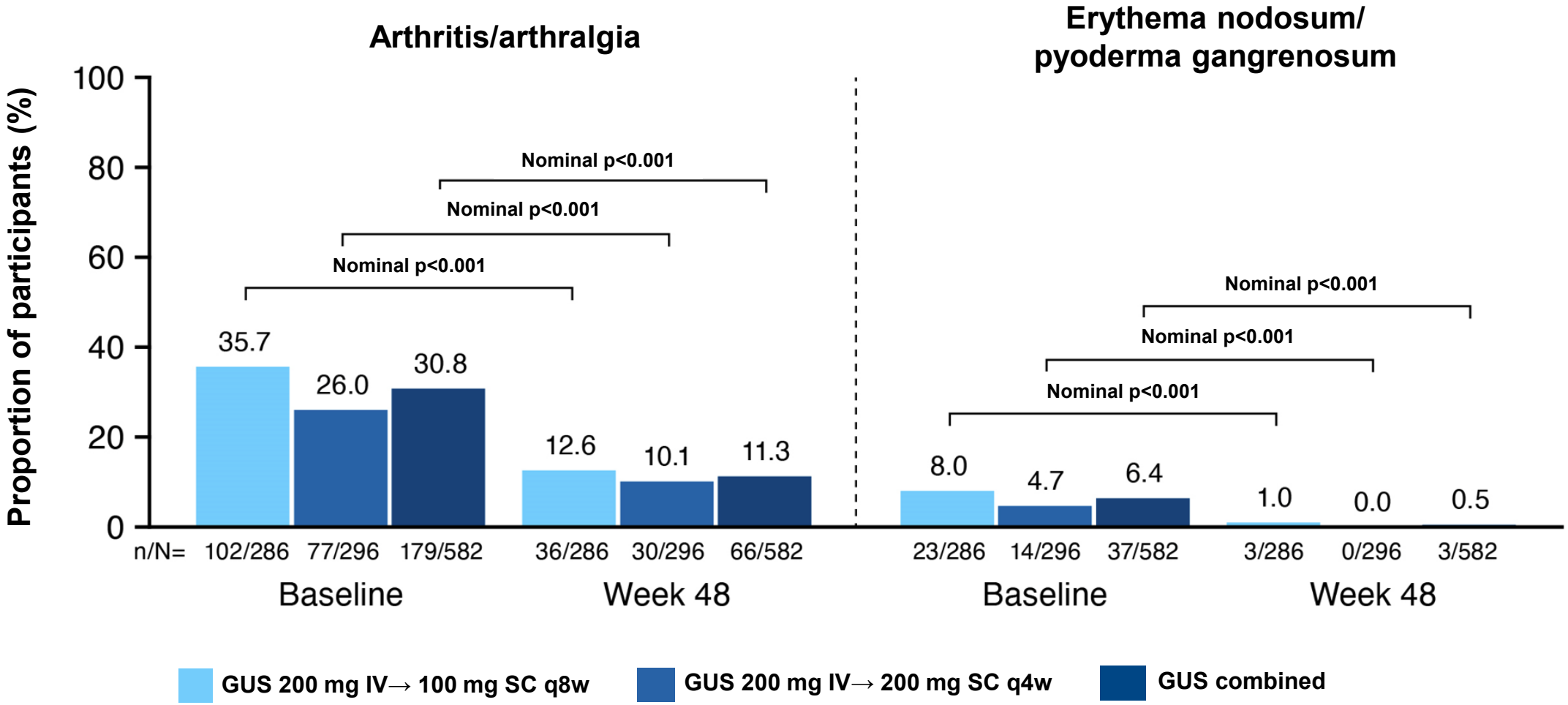
The CIs for the proportion of subjects meeting the endpoint in each treatment group were based on the normal approximation confidence limits. In cases of rare events, the exact confidence limits were provided.
A single participant may have more than one individual EIM.
The nominal p-values are based on the chi-square test.
CD, Crohn's disease; CI, confidence interval; EIM, extraintestinal manifestation; GUS, guselkumab, IL, interleukin; MOA, mechanism of action; PBO, placebo; RDBPC, randomised, double-blind, placebo-controlled.
Danese S, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. DOP001. Full prescribing information: www.swissmedicinfo-pro.ch.

Week 12 EIMs after induction



The nominal p-values are based on the McNemar's test comparing the prevalence of EIMs at Week 12 to baseline.
CD, Crohn's disease; EIM, extraintestinal manifestation; GUS, guselkumab, IL, interleukin; MOA, mechanism of action; PBO, placebo; RDBPC, randomised, double-blind, placebo-controlled.
Danese S, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. DOP001. Full prescribing information: www.swissmedicinfo-pro.ch.

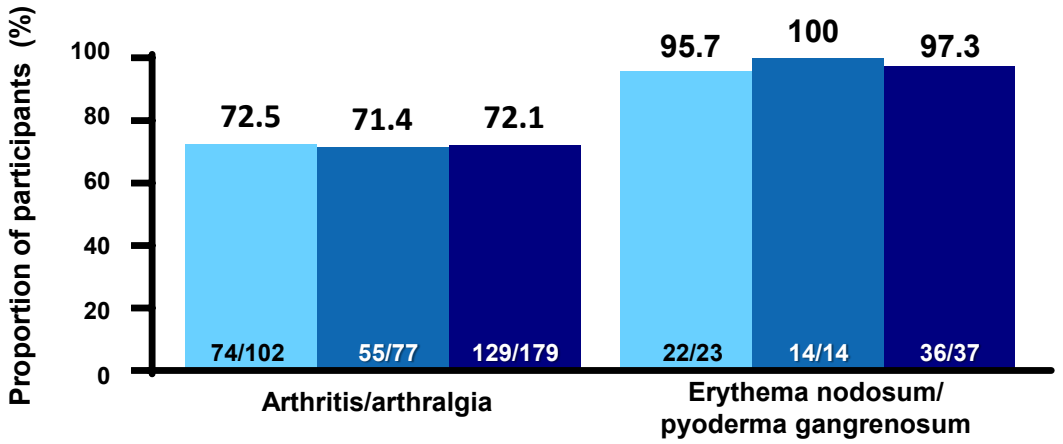
Week 48 EIMs



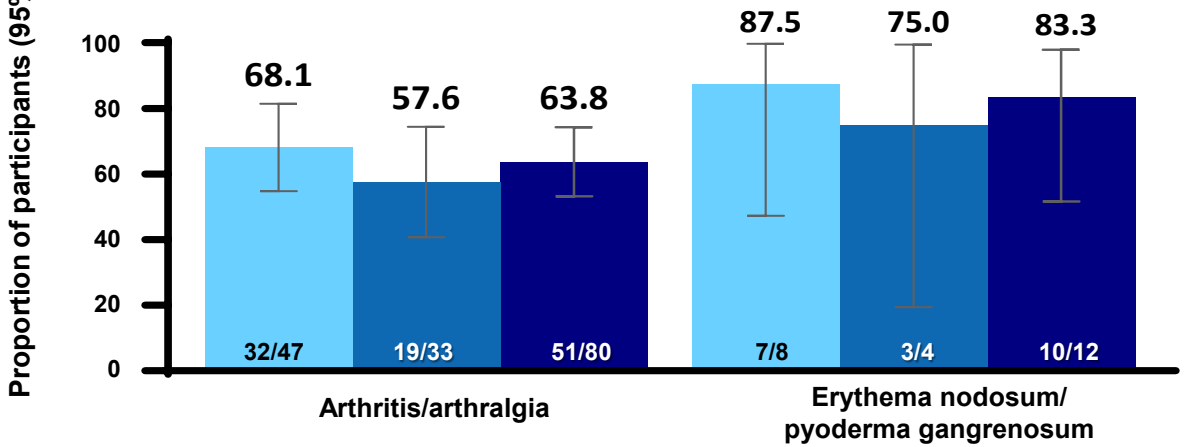
The nominal p-values are based on the McNemar's test comparing prevalence of EIMs at Week 48 to baseline.
CD, Crohn's disease; EIM, extraintestinal manifestation; GUS, guselkumab, IL, interleukin; IV, intravenous; MOA, mechanism of action; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous.
Danese S, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. DOP001. Full prescribing information: www.swissmedicinfo-pro.ch.

Corticosteroid-free EIM resolution

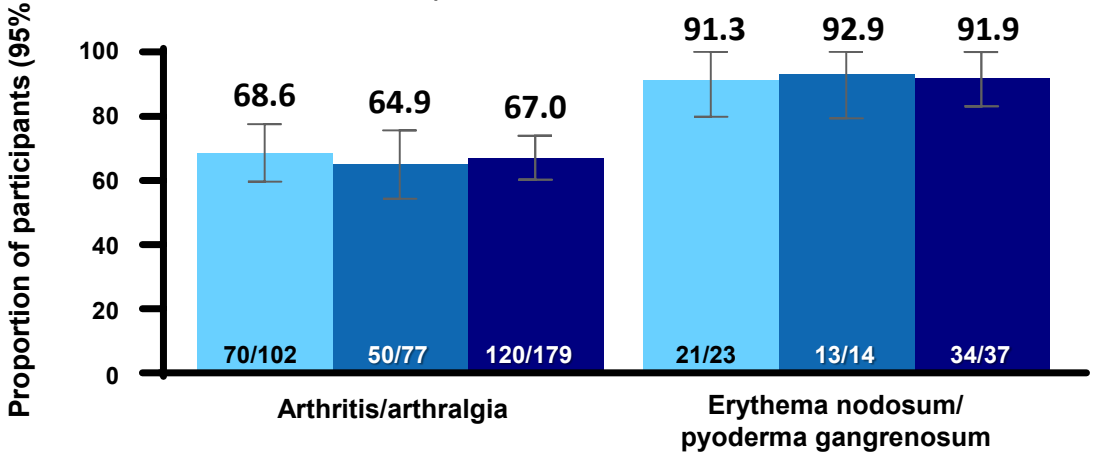
EIM resolution at Week 48
Participants with EIMs at baseline



90-day corticosteroid-free EIM resolution at Week 48
Participants with EIMs who were receiving corticosteroids at baseline



90-day corticosteroid-free EIM resolution at Week 48
Participants with EIMs at baseline



GUS 200 mg IV→ 100 mg SC q8w

GUS 200 mg IV→ 200 mg SC q4w

GUS combined

Most participants with EIM resolution at Week 48 were corticosteroid-free

CD, Crohn's disease; CI, confidence interval; EIM, extraintestinal manifestation; GUS, guselkumab, IL, interleukin; IV, intravenous; MOA, mechanism of action; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous.

Danese S, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. DOP001. Full prescribing information: www.swissmedicinfo-pro.ch.

Conclusions

- GUS-treated participants with CD had greater EIM resolution and lower rates of *de novo* EIMs at Week 12 vs placebo
- EIM resolution continued through Week 48 and was not dependent on corticosteroid use
- These results suggest GUS may improve and prevent EIMs in patients with CD

CD, Crohn's disease; EIM, extraintestinal manifestation; GUS, guselkumab, IL, interleukin; MOA, mechanism of action; RDBPC, randomised, double-blind, placebo-controlled.
Danese S, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. DOP001. Full prescribing information: www.swissmedicinfo-pro.ch.

P0596

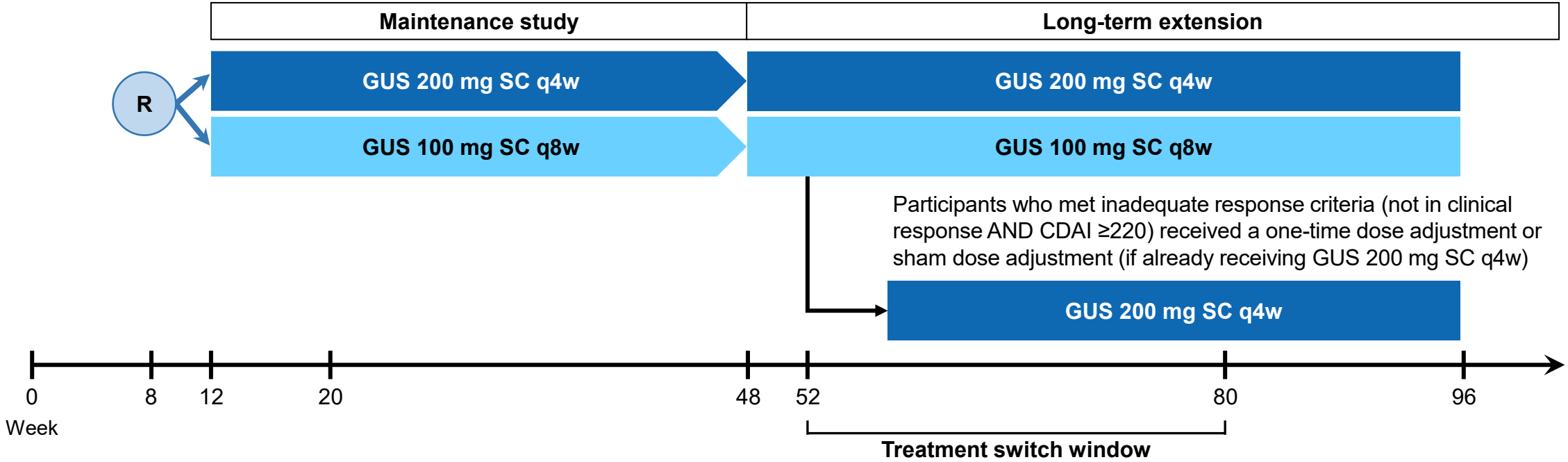
Efficacy and safety of guselkumab in participants with moderately to severely active Crohn's disease who had maintenance dose adjustment: Results from the Phase 3 GALAXI 2 & 3 long-term extension

Panaccione R,[1] Hisamatsu T,[2] Afzali A,[3] Rampelbergh R Van,[4] Yee J,[5] Duijnhoven W Van,[6] Corbett C,[7] Sands BE,[8] Danese S[9]

[1]University of Calgary, Inflammatory Bowel Disease Unit, Division of Gastroenterology and Hepatology, Calgary, AB, Canada; [2]Kyorin University School of Medicine, Department of Gastroenterology and Hepatology, Tokyo, Japan; [3]University of Cincinnati College of Medicine, Division of Gastroenterology and Hepatology, Cincinnati, OH, USA; [4]Johnson & Johnson, Clinical Development–Immunology, Antwerp, Belgium; [5]Johnson & Johnson, Clinical Science–Immunology, Raritan, NJ, USA; [6]Johnson & Johnson, Statistics & Decision Sciences, Antwerp, Belgium; [7]Johnson & Johnson, Statistics & Decision Sciences, High Wycombe, UK; [8]Icahn School of Medicine at Mount Sinai, Dr. Henry D. Janowitz Division of Gastroenterology, NY, USA; [9]IRCCS San Raffaele Hospital and Vita-Salute San Raffaele University, Gastroenterology and Endoscopy, Milan, Italy.

GALAXI 2 and 3 LTE study design

Aim: to assess the efficacy and safety through Week 96 of GUS in participants who dose adjusted after an inadequate response to their randomised GUS maintenance regimen during Weeks 52–80 in the LTE of the GALAXI 2 & 3 studies



- Treatment allocation in the LTE remained blinded until all participants had completed treatment through Week 48, the Week 48 database was locked, and the Week 48 analysis was finalised
- Per the prespecified analysis plan, participants undergoing dose adjustment in the LTE were evaluated as follows:
  - Clinical response and clinical remission were assessed 16 weeks after dose adjustment and at Week 96
  - Endoscopic response and endoscopic remission were assessed at Weeks 48 and 96
  - Safety was assessed through Week 96

Non-responder imputation and handling of missing data: participants who experienced events indicative of a lack of efficacy were considered non-responders at all analysis time points after experiencing the event. Participants who discontinued study agent due to COVID-19-related reasons (excluding COVID-19 infection) or regional crisis had their observed data used, if available, to determine responder and non-responder status at the analysis time point. Participants who were missing endpoint scoring data at the analysis time point were considered not to have achieved the endpoint at the analysis time point.

CD, Crohn's disease; CDAI, Crohn's Disease Activity Index; IL, interleukin; GUS, guselkumab; LTE, long-term extension; MOA, mechanism of action; qXw, every X weeks; R, randomised; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous.

Panaccione R, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. Poster P0596. Full prescribing information: www.swissmedicinfo-pro.ch.

Disease characteristics of GALAXI 2 & 3 participants who did and did not receive dose adjustment

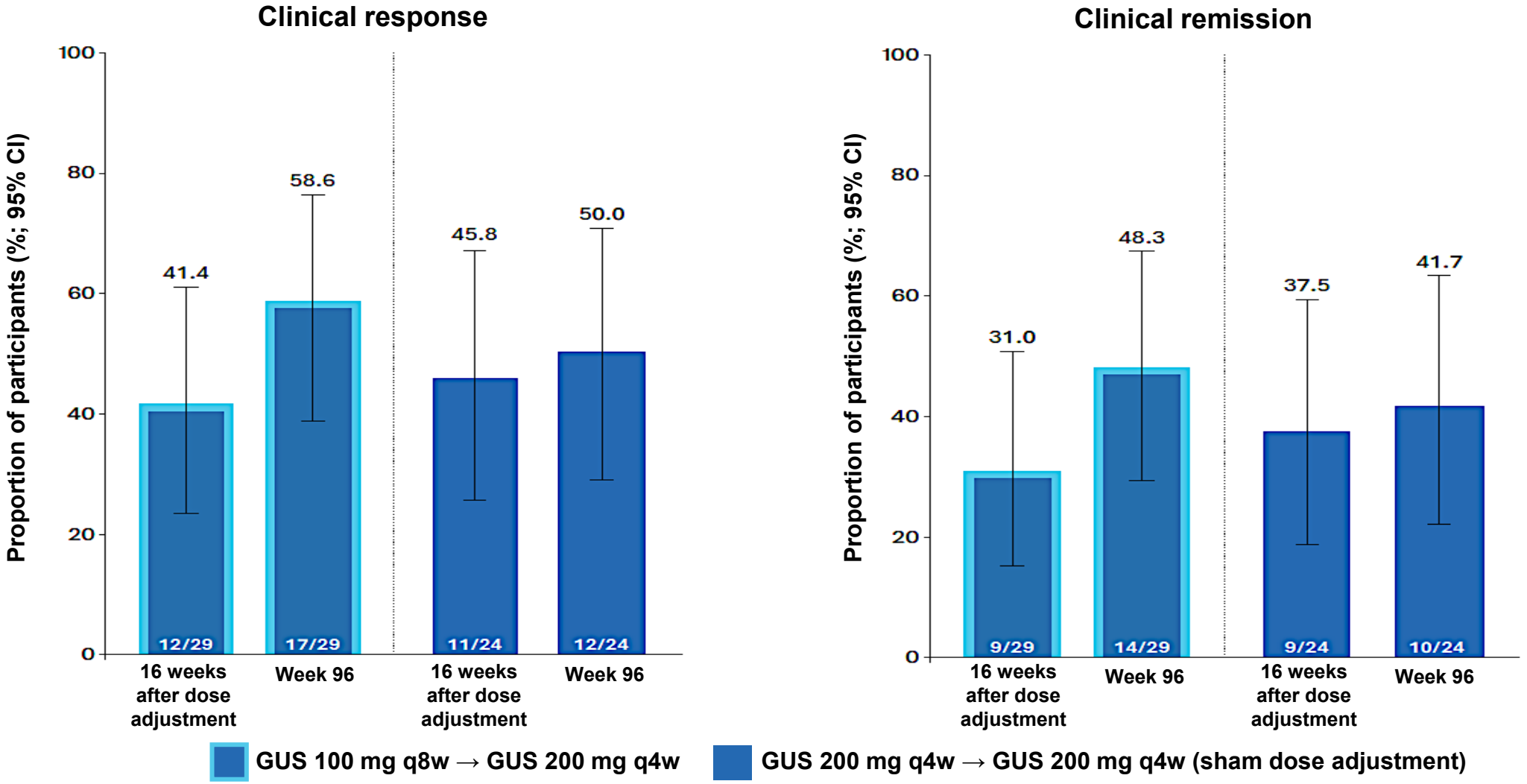
	GUS 100 mg q8w*		GUS 200 mg q4w*	
	Did NOT receive dose adjustment	Received dose adjustment	Did NOT receive dose adjustment	Received sham dose adjustment
N†	208	29	218	24
CHARACTERISTICS AT BASELINE (WEEK 0)				
Crohn's disease duration, years, median (IQR)	4.9 (1.8; 10.2)	8.2 (5.3; 14.5)	4.1 (1.6; 9.0)	6.3 (3.0; 14.3)
History of BIO-IR, n (%)	102 (49.0)	17 (58.6)	98 (45.0)	14 (58.3)
CDAI score, median (IQR)	280.0 (248.0; 329.0)	294.0 (263.0; 319.0)	283.5 (252.0; 334.0)	289.0 (264.0; 328.5)
SES-CD, median (IQR)	11.0 (7.0; 17.0)	13.0 (7.0; 19.0)	11.0 (7.0; 17.0)	13.0 (9.5; 18.0)
Endoscope disease severity (per SES-CD)				
Moderate (7–16), n (%)	116 (55.8)	14 (48.3)	106 (48.6)	11 (45.8)
Severe (>16), n (%)	55 (26.4)	12 (41.4)	61 (28.0)	9 (37.5)
Involved GI areas by central reader				
Ileum only, n (%)	41 (19.7)	6 (20.7)	56 (25.7)	6 (25.0)
Colon only, n (%)	80 (38.5)	13 (44.8)	87 (39.9)	6 (25.0)
Ileum and colon, n (%)	87 (41.8)	10 (34.5)	75 (34.4)	12 (50.0)
C-reactive protein, mg/L, median (IQR)	7.2 (2.1; 19.4)	8.5 (2.9; 36.2)	6.5 (2.8; 19.8)	6.1 (2.1; 31.1)
Faecal calprotectin, µg/g, median (IQR)‡	903.0 (390.0; 1892.0)	848.5 (403.5; 2116.5)	982.0 (319.0; 1936.5)	1354.5 (658.5; 1747.5)
CHARACTERISTICS AT WEEK 48				
CDAI score, median (IQR)	69.5 (30.0; 128.5)	155.0 (81.0; 232.0)	68.5 (32.0; 112.0)	138.5 (72.0; 206.5)
SES-CD, median (IQR)§	4.0 (0.0; 7.0)	7.0 (4.0;15.0)	3.0 (1.0; 6.0)	6.0 (3.5; 10.5)
In endoscopic response, n (%)	128 (61.5)	9 (31.0)	149 (68.3)	8 (33.3)
In endoscopic remission, n (%)	91 (43.8)	4 (13.8)	106 (48.6)	5 (20.8)
Endoscopic disease severity (per SES-CD)§				
Moderate (7–16), n (%)	49 (24.0)	10 (34.5)	36 (16.7)	8 (33.3)
Severe (>16), n (%)	5 (2.5)	7 (24.1)	6 (2.8)	2 (8.3)
C-reactive protein, mg/L, median (IQR)¶	1.8 (0.8; 5.8)	5.9 (0.8; 12.7)	2.2 (0.9; 4.6)	4.0 (0.6; 8.3)
Faecal calprotectin, µg/g, median (IQR)**	173.5 (65.0; 555.0)	591.0 (115.0; 1739.0)	121.0 (47.0; 408.0)	160.0 (49.0; 366.0)

*Treatment group at the start of the LTE period. Participants who had a dose adjustment between Week 52 and Week 80 received GUS 200 mg SC q4w. Participants who were already receiving the GUS 200 mg SC q4w maintenance dose and met the inadequate response criteria received a "sham" dose adjustment; †Participants with a Crohn's disease-related surgery (with the exception of minor procedures) prior to Week 48 or a prohibited change in Crohn's disease medications prior to Week 48 who remained on treatment and subsequently entered the LTE are excluded; ‡N=206 (GUS 100 non-adjusters), 28 (GUS 100→200), 216 (GUS 200 non-adjusters), and 24 (GUS 200→200) participants with evaluable samples at baseline; §N=204 (GUS 100 non-adjusters), 29 (GUS 100→200), 216 (GUS 200 non-adjusters) and 24 (GUS 200→200) participants with SES-CD data at Week 48; ¶N=205 (GUS 100 non-adjusters), 27 (GUS 100→200), 213 (GUS 200 non-adjusters) and 24 (GUS 200→200) participants with evaluable samples at Week 48; **N=198 (GUS 100 non-adjusters), 27 (GUS 100→200), 206 (GUS 200 non-adjusters) and 23 (GUS 200→200) participants with evaluable samples at Week 48.

BIO-IR, inadequate response/intolerance to biologic therapy; CD, Crohn's disease; CDAI, Crohn's Disease Activity Index; GI, gastrointestinal; GUS, guselkumab; IL, interleukin; IQR, interquartile range; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous; SES-CD, Simple Endoscopic Score for Crohn's Disease.

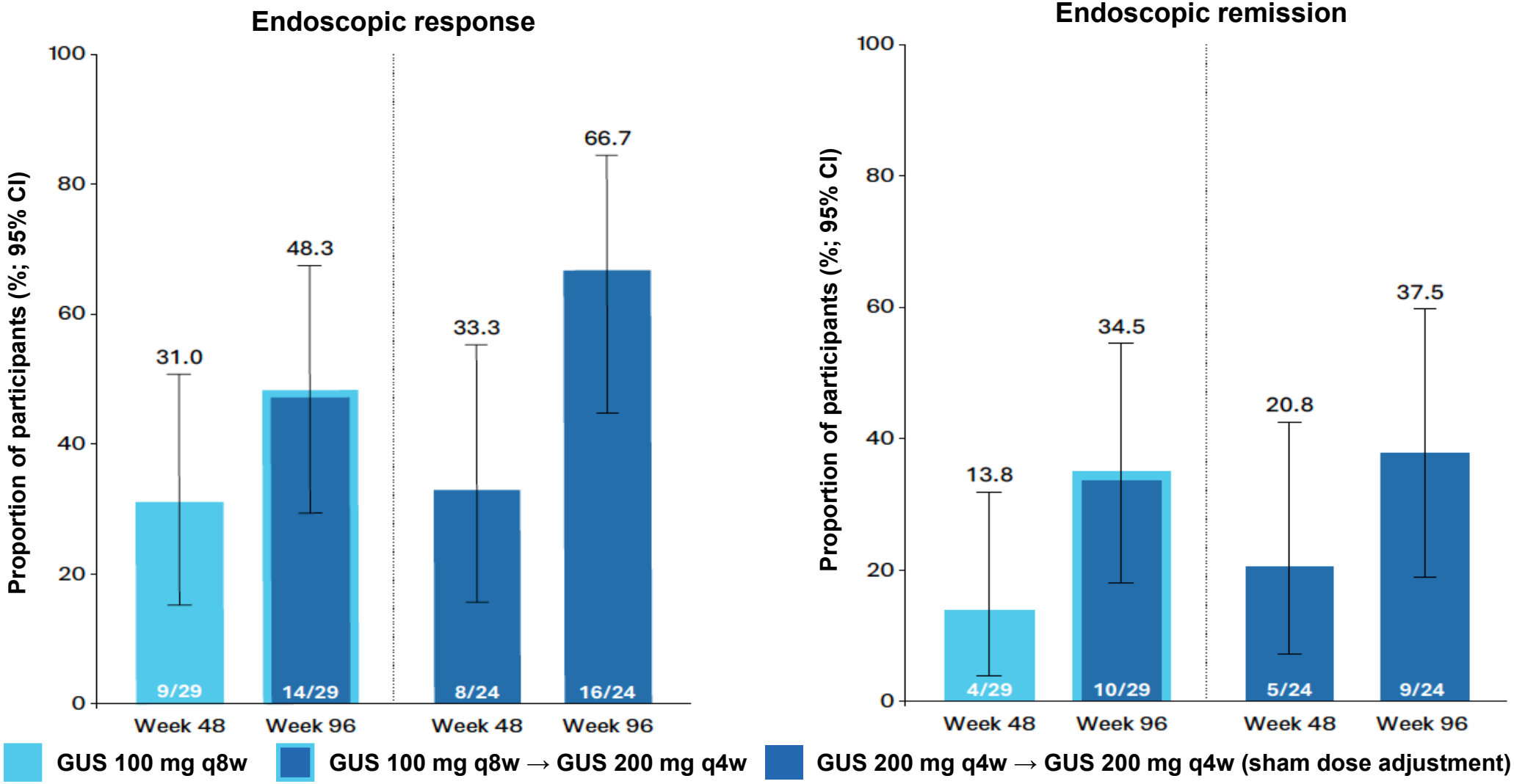
Panaccione R, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. Poster P0596. Full prescribing information: www.swissmedicinfo-pro.ch.

Over 40% of participants achieved clinical response 16 weeks after dose adjustment, and the percentage increased further by Week 96



No participant was in clinical response or clinical remission at the time of dose adjustment per the criteria for dose adjustment (not in clinical response AND CDAI ≥220).
Clinical remission: CDAI score <150. **Clinical response:** ≥100-point reduction in CDAI score from the time of dose adjustment or CDAI score <150.
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; RDBPC, randomised, double-blind, placebo-controlled.
Panaccione R, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. Poster P0596. Full prescribing information: www.swissmedicinfo-pro.ch.

Participants showed improvement in endoscopic outcomes after receiving dose adjustment



Endoscopic remission: SES-CD ≤4 and ≥2-point reduction from Week 0 and no subscore >1 in any individual component. **Endoscopic response:** ≥50% improvement from Week 0 in SES-CD or SES-CD ≤2.
CD, Crohn's disease; CI, confidence interval; GUS, guselkumab; IL, interleukin; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; SES-CD, Simple Endoscopic Score for Crohn's Disease.
Panaccione R, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. Poster P0596. Full prescribing information: www.swissmedicinfo-pro.ch.

Adverse event rates during the LTE were similar before and after dose adjustment

	Randomised treatment and up to dose adjustment*	After dose adjustment†	Randomised treatment and up to dose adjustment*	After sham dose adjustment†
	GUS 100 mg q8w	**GUS 100 mg q8w →200 mg q4w**	**GUS 200 mg q4w**	**GUS 200 mg q4w →200 mg q4w**
N‡	36	36	28	28
Average duration of follow-up, weeks	12.1	33.6	12.9	33.1
Participant-years (P-Y) of follow-up	8.3	23.2	6.9	17.7
Participants with ≥1 AE, n (%)	19 (52.8)	24 (66.7)	14 (50.0)	15 (53.6)
Events/100 P-Y (95% CI)§	360.7 (243.4, 514.9)	341.1 (270.0, 425.1)	519.1 (363.6, 718.7)	484.6 (387.6, 598.5)
Participants with ≥1 SAE, n (%)	0	3 (8.3)	1 (3.6)	1 (3.6)
Events/100 P-Y (95% CI)§	0.0 (0.0, 36.0)	13.0 (2.7, 37.9)	14.4 (0.4, 80.3)	5.6 (0.1, 31.4)
Participants with ≥1 AE leading to discontinuation, n (%)	0	2 (5.6%)	1 (3.6%)	0
Events/100 P-Y (95% CI)§	0.0 (0.0, 36.0)	17.3 (4.7, 44.2)	14.4 (0.4 80.3)	0.0 (0.0, 16.9)
Participants with ≥1 infection, n (%)	7 (19.4)	12 (33.3)	4 (14.3)	9 (32.1)
Events/100 P-Y (95% CI)§	120.2 (57.7, 221.1)	77.7 (46.1, 122.8)	72.1 (23.4, 168.3)	84.5 (47.3, 139.4)
Participants with ≥1 serious infection, n (%)	0	1 (2.8)	0	0
Events/100 P-Y (95% CI)§	0.0 (0.0, 36.0)	4.3 (0.1, 24.1)	0.0 (0.0, 43.2)	0.0 (0.0, 16.9)
Participants who died, n (%)	0	0	0	0

*Treatment group at the start of the LTE. Includes events for participants who received a dose adjustment from Week 48 up to the time point of dose adjustment; †Only events after dose adjustment (including "sham") are included in this column. Participants receiving GUS 100 mg SC q8w who met inadequate response criteria between Week 52 and Week 80 had a dose adjustment to GUS 200 mg SC q4w. Participants receiving GUS 200 mg SC q4w who met the inadequate response criteria between Week 52 and Week 80 received "sham" dose adjustment; ‡All participants who met criteria for dose adjustment are included; §CI based on an exact method assuming that the observed number of events follows a Poisson distribution. Note: participants are counted only once for any given event, regardless of the number of times they actually experienced the event. Adverse events are coded using MeDRA version 27.0.

AE, adverse event; CD, Crohn's disease; CI, confidence interval; GUS, guselkumab; IL, interleukin; LTE, long-term extension; P-Y, participant years; qXw, every X weeks; RDBPC, randomised, double blind, placebo-controlled; SAE, serious adverse event; SC, subcutaneous.

Panaccione R, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. Poster P0596. Full prescribing information: www.swissmedicinfo-pro.ch.

Conclusions

- Maintenance dose adjustment to GUS 200 mg q4w may benefit patients with CD who experience inadequate response to GUS 100 mg q8w
- Over 40% of GUS 100 mg q8w participants receiving an adjustment to GUS 200 mg q4w achieved clinical remission or endoscopic response up to Week 96
- Efficacy outcomes in GUS 200 mg q4w participants who received a sham dose adjustment were generally similar to those who switched from GUS 100 mg q8w to 200 mg q4w
- Safety outcomes after dose adjustment were consistent with the established safety profile of GUS in approved indications
- Interpretation is affected by small sample sizes

CD, Crohn's disease; GUS, guselkumab, IL, interleukin; MOA, mechanism of action; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled.
Panaccione R, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. Poster P0596. Full prescribing information: www.swissmedicinfo-pro.ch.

P1102

Impact of disease duration on clinical and endoscopic responses at 1 year in patients with Crohn's disease treated with guselkumab: Pooled analysis of the GALAXI 2 & 3 studies

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Baseline characteristics were generally well balanced across disease duration groups

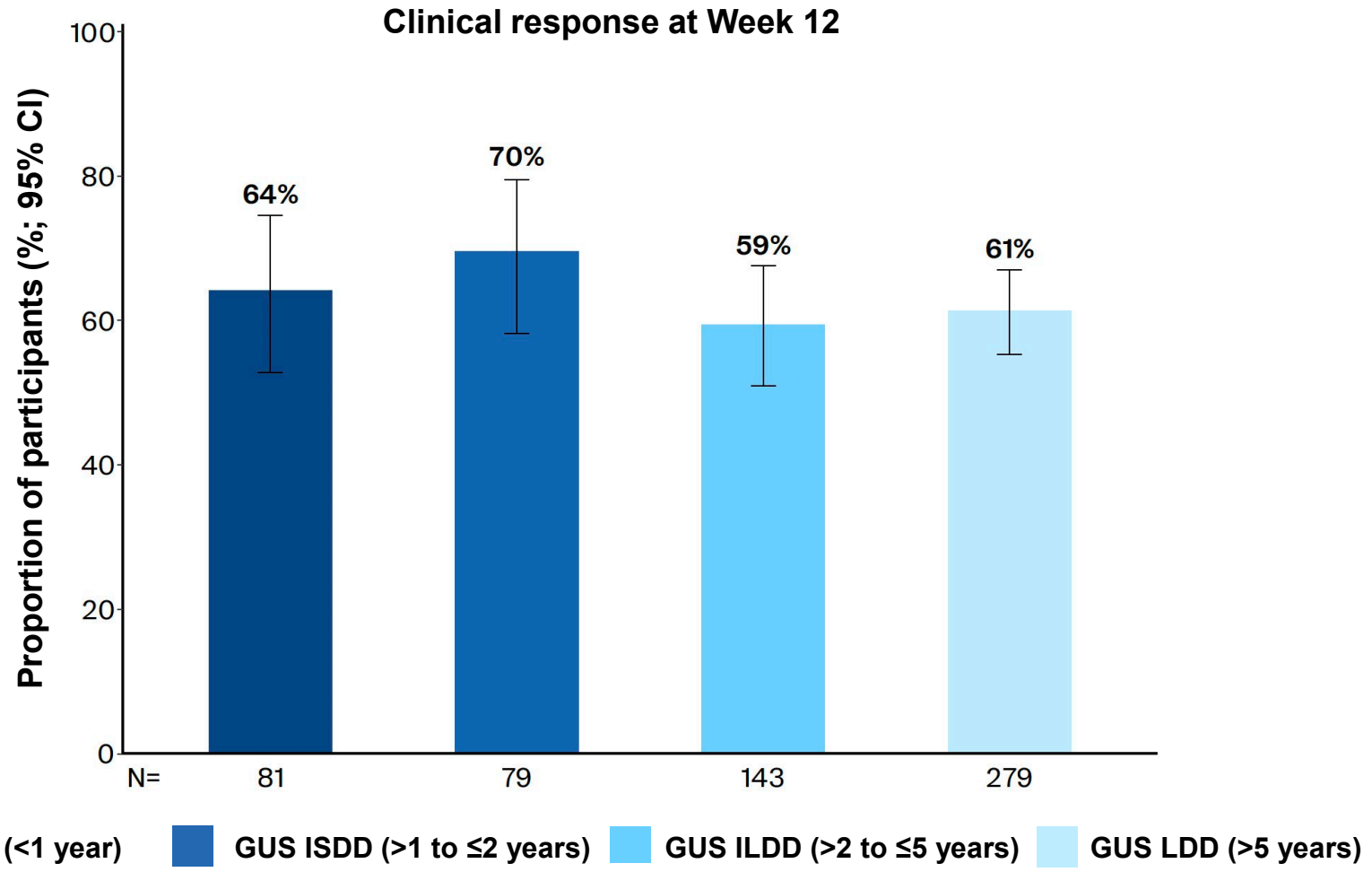
Baseline characteristics	USDD (≤1 year) (n=81)	ISDD (>1 to ≤2 years) (n=79)	ILDD (>2 to ≤5 years) (n=143)	LDD (>5 years) (n=279)
Demographics				
Age, years, mean (SD)	34.6 (12.8)	34.0 (14.7)	34.7 (12.5)	38.6 (12.0)
Male, %	52	53	64	56
Race, Asian/Black/White, %	22/0/78	27/0/72	23/2/71	20/1/77
Disease characteristics				
CDAI, mean (SD)	298.0 (54.3)	293.0 (53.2)	298.7 (55.6)	295.0 (52.4)
SES-CD, mean (SD)	11.6 (7.4)	14.3 (8.3)	11.8 (6.2)	13.4 (7.5)
Involved GI areas*, %				
Ileum only	23	27	31	19
Colon only	28	23	27	24
Ileum and colon	47	51	41	57
Proximal small intestine	14†	4	13	11
≥1 open or draining fistula, %	14	8	11	13
≥1 open or draining perianal fistula, %	11†	8	9	11
≥1 open or draining perirectal fistula, %	1	0	0	<1
≥1 extra-intestinal manifestation, %	20	15	24	24
CRP, mg/L, mean (SD)	12.9 (17.4)	18.1 (22.7)	15.1 (18.9)	16.5 (23.6)
Faecal calprotectin, µg/g, mean (SD)	1460.0 (1548.7)	1865.8 (2225.3)‡	1570.3 (1724.5)§	1974.1 (3844.8)¶
History of intolerance/inadequate response to biologic therapy, %	19	49	53	63
Concomitant medication use				
Azathioprine, mercaptopurine or methotrexate, %	35	29	30	32
Oral corticosteroids, %	49	32	33	37

*Assessed by central reader; †N=80; ‡N=78; §N=141; ¶N=277.

CD, Crohn's disease; CDAI, Crohn's Disease Activity Index; CRP, C-reactive protein; GI, gastrointestinal; GUS, guselkumab, IL, interleukin; ILDD, intermediate-long disease duration; ISDD, intermediate-short disease duration; LDD, long disease duration; MOA, mechanism of action; RDBPC, randomised, double-blind, placebo-controlled; SD, standard deviation; SES-CD, Simple Endoscopic Score for Crohn's Disease; USDD, ultra-short disease duration.

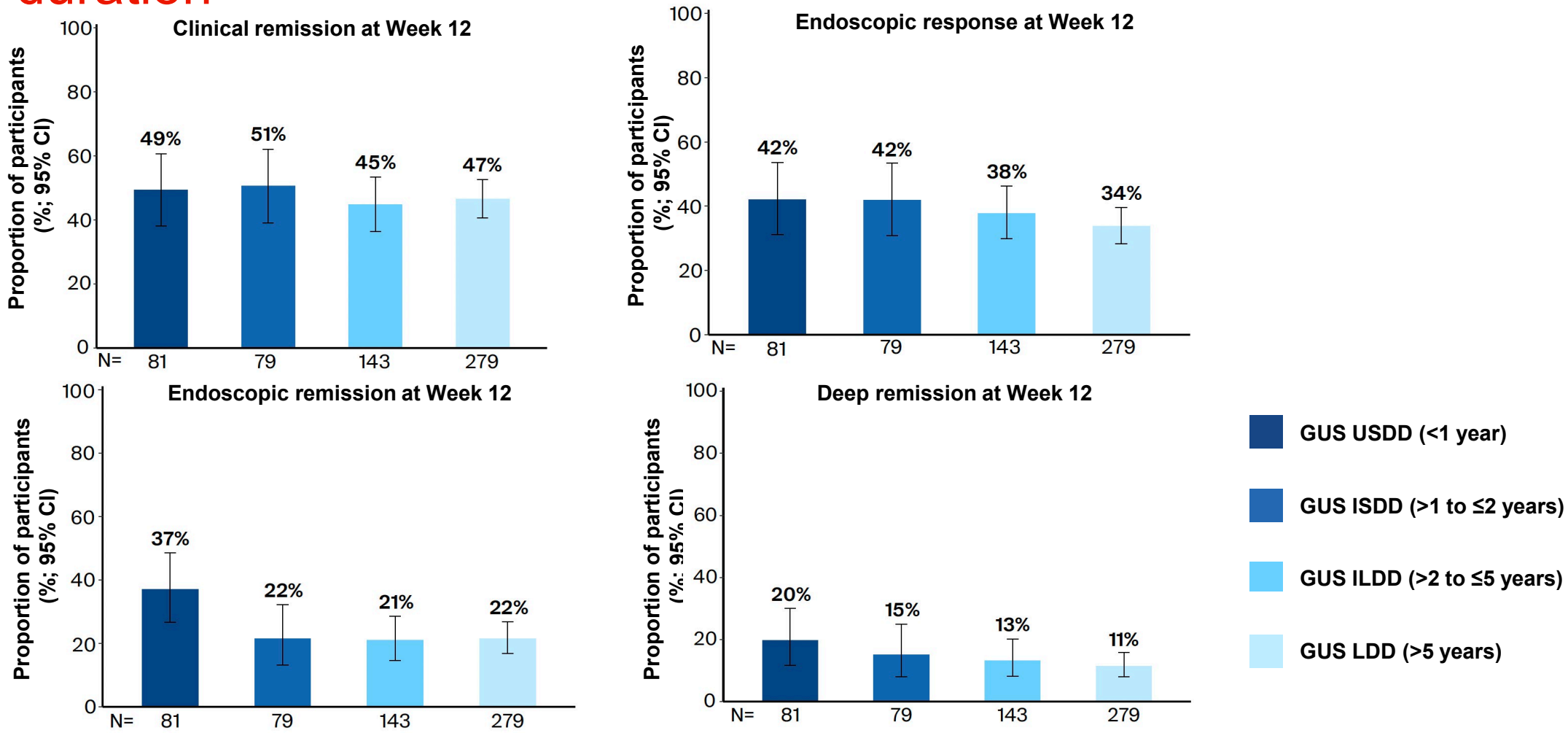
Ferrante M, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. Poster P1102. Full prescribing information: www.swissmedicinfo-pro.ch.

Approximately 60–70% of participants achieved clinical response with GUS induction across disease duration groups at Week 12



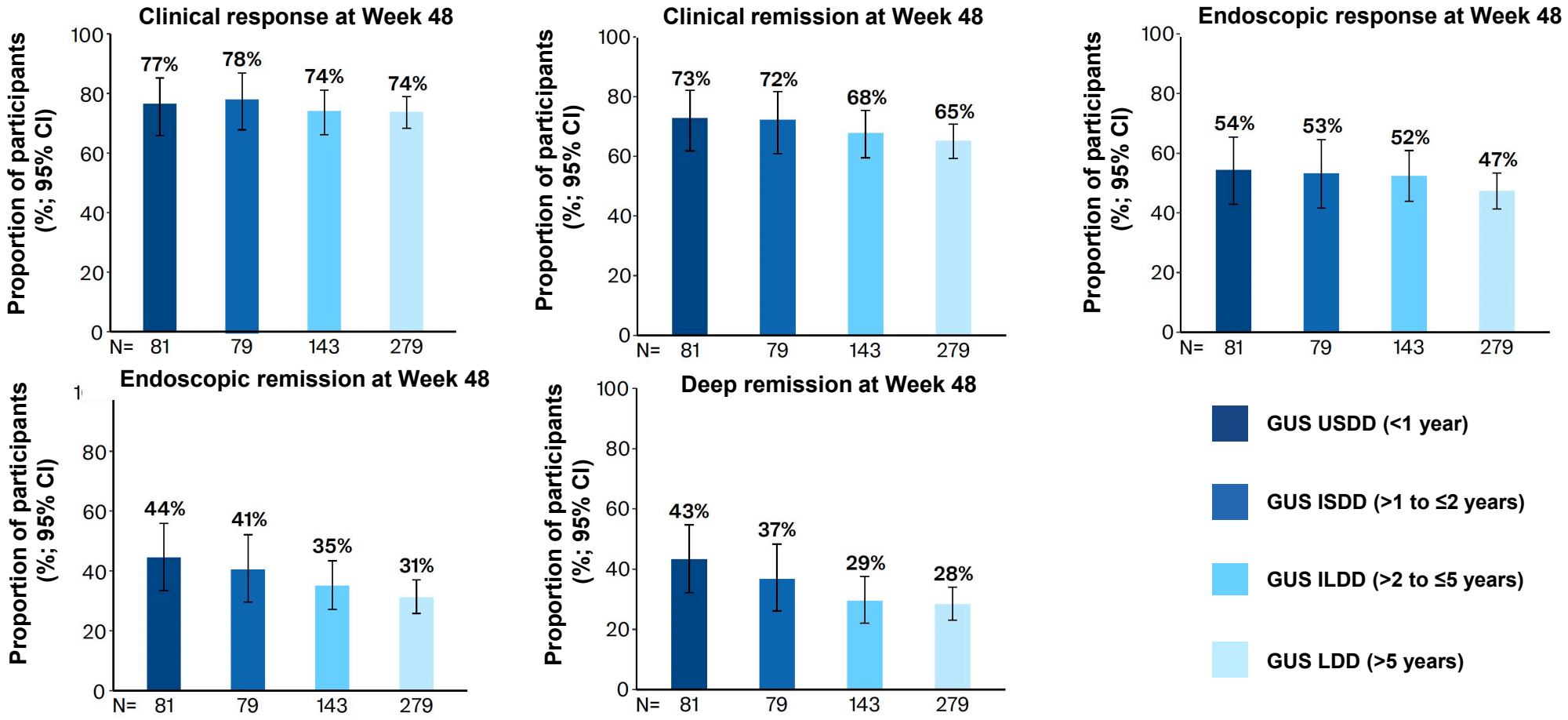
Endpoints were evaluated among pooled GUS-randomised (100 mg q8w, 200 mg q4w) patients. Non-responder imputation was applied for missing data.
Clinical response: 100-point decrease from baseline in CDAI or CDAI <150.
CD, Crohn's disease; CDAI, Crohn's Disease Activity Index; CI, confidence interval; GUS, guselkumab, IL, interleukin; ILDD, intermediate-long disease duration; ISDD, intermediate-short disease duration; LDD, long disease duration; MOA, mechanism of action; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; SD, standard deviation; USDD, ultra-short disease duration.
Ferrante M, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. Poster P1102 . Full prescribing information: www.swissmedicinfo-pro.ch.

At Week 12, GUS induction therapy was effective irrespective of disease duration



Endpoints were evaluated among pooled GUS-randomised (100 mg q8w, 200 mg q4w) patients. Non-responder imputation was applied for missing data.
Clinical remission: CDAI <150. **Endoscopic response:** ≥50% improvement from baseline in SES-CD or SES-CD ≤2. **Endoscopic remission:** SES-CD ≤4, ≥2-point reduction from baseline in SES-CD and no SES-CD sub-score >1. **Deep remission:** clinical and endoscopic remission.
CD, Crohn's disease; CDAI, Crohn's Disease Activity Index; CI, confidence interval; GUS, guselkumab, IL, interleukin; ILDD, intermediate-long disease duration; ISDD, intermediate-short disease duration; LDD, long disease duration; MOA, mechanism of action; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; SD, standard deviation; SES-CD, Simple Endoscopic Score for Crohn's Disease; USDD, ultra-short disease duration.
Ferrante M, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. Poster P1102. Full prescribing information: www.swissmedicinfo-pro.ch.

GUS showed increased rates across clinical and endoscopic outcomes from Week 12 through Week 48 across disease duration groups



Clinically relevant decreases in endoscopic and deep remission rates with GUS were observed at Week 48 with longer disease duration

Endpoints were evaluated among pooled GUS-randomised (100 mg q8w, 200 mg q4w) patients. Non-responder imputation was applied for missing data.
Clinical remission: CDAI <150. **Clinical response:** 100-point decrease from baseline in CDAI or CDAI <150. **Endoscopic response:** ≥50% improvement from baseline in SES-CD or SES-CD ≤2. **Endoscopic remission:** SES-CD ≤4, ≥2-point reduction from baseline in SES-CD and no SES-CD subscore >1. **Deep remission:** clinical and endoscopic remission.
CD, Crohn's disease; CDAI, Crohn's Disease Activity Index; CI, confidence interval; GUS, guselkumab, IL, interleukin; ILDD, intermediate-long disease duration; ISDD, intermediate-short disease duration; LDD, long disease duration; MOA, mechanism of action; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; SES-CD, Simple Endoscopic Score for Crohn's Disease; USDD, ultra-short disease duration.
Ferrante M, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. Poster P1102. Full prescribing information: www.swissmedicinfo-pro.ch.

Conclusions

Among GUS-randomised adults with moderately to severely active CD pooled from the Phase 3 GALAXI 2 & 3 trials:

- GUS induction therapy was effective irrespective of disease duration, with Week 12 outcomes appearing similar across groups
- GUS demonstrated clinical and endoscopic benefits through 1 year across disease duration groups, with lower endoscopic and deep remission rates in patients with LDD
- Differences emerging at Week 48 may be partly driven by the more treatment-refractory profile of longer duration groups, including a higher incidence of prior biologic failure

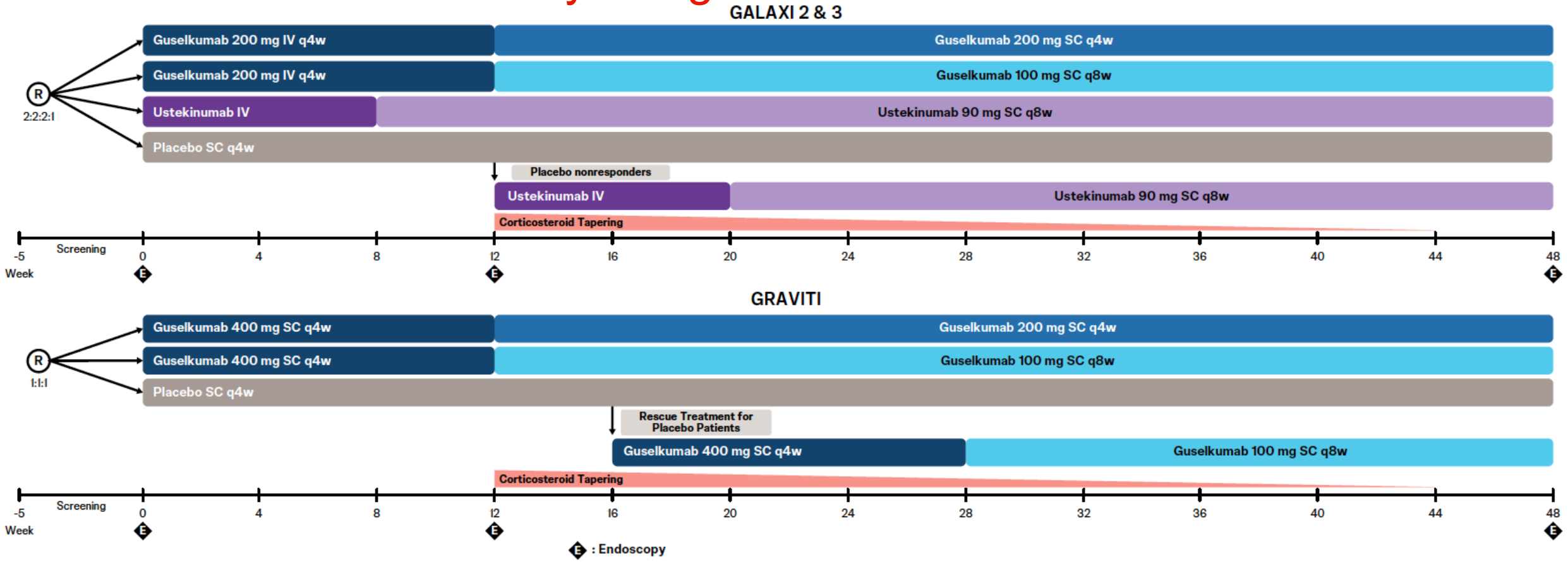
Findings support improved therapeutic outcomes following earlier initiation of GUS treatment in CD

CD, Crohn's disease; GUS, guselkumab, IL, interleukin; LDD, long disease duration; MOA, mechanism of action; RDBPC, randomised, double-blind, placebo-controlled.
Ferrante M, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. Poster P1102. Full prescribing information: www.swissmedicinfo-pro.ch.

GALAXI/GRAVITI

Immunology

GALAXI and GRAVITI study designs



Randomisation stratified by:

- CDAI score (≤300 or >300)
- SES-CD (≤12 or >12)
- Prior BIO-failure status
- Corticosteroid use at baseline (GALAXI only)

Key eligibility criteria:

- Moderately to severely active CD (CDAI score 220–450 AND either mean daily SF count ≥4 OR AP score ≥2) and SES-CD score ≥6 (or ≥4 for isolated ileal disease)
- Inadequate response/intolerance to oral corticosteroids, 6-MP/AZA/MTX or biologic therapies*

Note: only the study designs through Week 48 are shown. GRAVITI included data handling rules for rescue treatment at Week 16 in participants meeting prespecified criteria; these data handling rules were suspended for the Week 48 analyses shown below. *Biologic therapies: TNF antagonists or vedolizumab.

6-MP, mercaptopurine; AP, abdominal pain; AZA, azathioprine; BIO, biologic; CD, Crohn's disease; CDAI, Crohn's Disease Activity Index; GUS, guselkumab; IL, interleukin; IV, intravenous; 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.

Deepak P, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. Poster P1110. Full prescribing information: www.swissmedicinfo.ch

P1110

Efficacy of intravenous and subcutaneous guselkumab induction by weight and body mass index in patients with Crohn's disease: Results from the Phase 3 GALAXI and GRAVITI studies

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Baseline demographics and disease characteristics

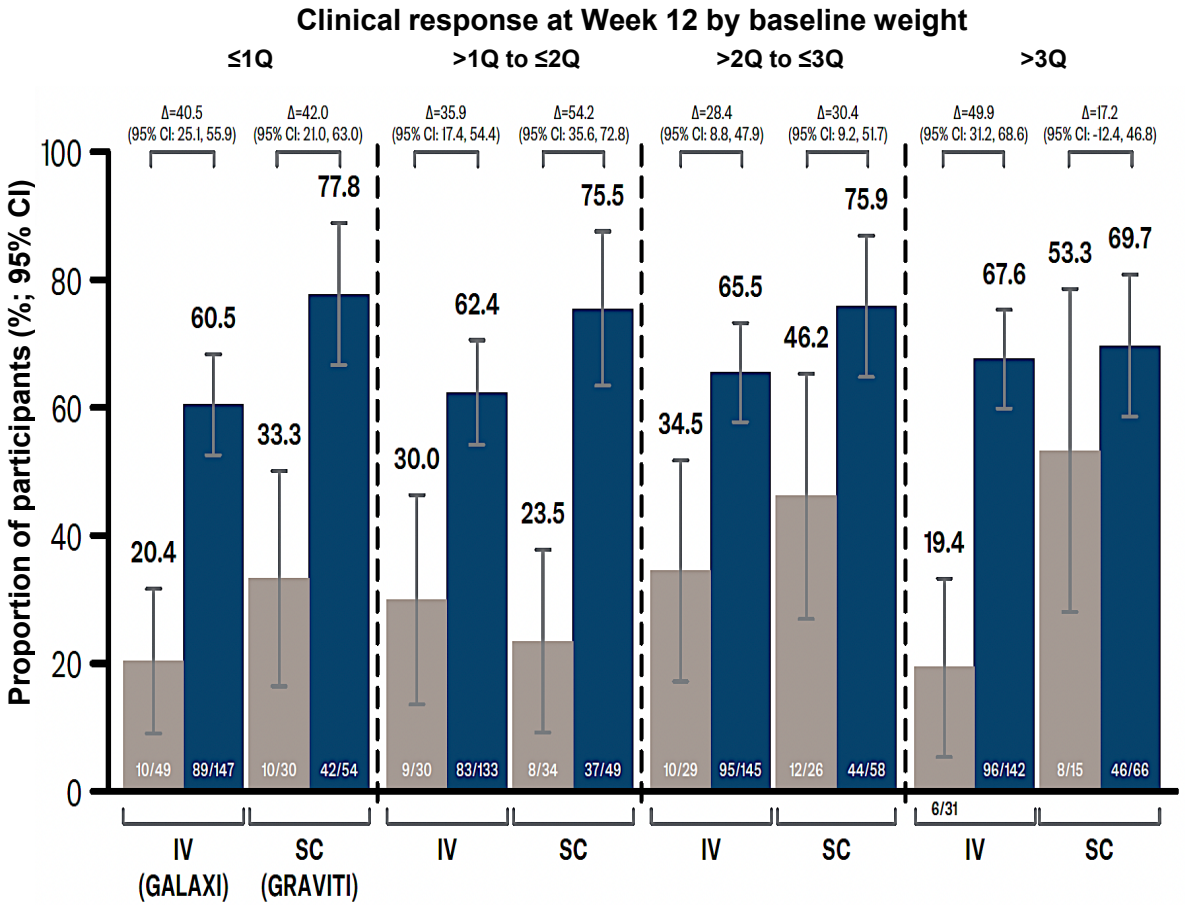
Full analysis set	GALAXI 2 & 3				GRAVITI			
	PBO IV (n=148)	GUS 200 mg IV → 100 mg SC q8w (n=286)	GUS 200 mg IV → 200 mg SC q4w (n=296)	GUS combined (n=582)	PBO SC (n=117)	GUS 400 mg SC → 100 mg SC q8w (n=115)	GUS 400 mg SC → 200 mg SC q4w (n=115)	GUS combined (n=230)
Demographics								
Weight in kg, mean (SD)	67.12 (18.14)	67.36 (16.52)	68.21 (16.36)	67.79 (16.43)	68.13 (16.20)	70.88 (19.00)	72.88 (19.28)	71.88 (19.13)
Body mass index, kg/m^2, mean (SD)	22.77 (5.28)	23.08 (4.60)	23.23 (4.77)	23.15 (4.68)	23.85 (4.88)	24.28 (5.62)	24.84 (6.27)	24.56 (5.95)
Age, years, mean (SD)	34.8 (12.15)	36.0 (12.24)	36.9 (13.27)	36.5 (12.77)	36.0 (12.71)	37.4 (13.32)	39.1 (12.56)	38.2 (12.95)
Male, n (%)	88 (59.5)	154 (53.8)	178 (60.1)	332 (57.0)	67 (57.3)	66 (57.4)	70 (60.9)	136 (59.1)
Characteristics								
CD duration, years, mean (SD)	7.15 (7.53)	7.12 (6.72)	7.07 (7.24)	7.09 (6.98)	6.96 (7.75)	9.17 (9.08)	7.89 (7.13)	8.53 (8.17)
CDAI score, mean (SD)	293.4 (52.72)	296.3 (54.27)	295.9 (52.74)	296.1 (53.45)	293.0 (49.09)	300.4 (54.32)	297.3 (54.69)	298.8 (54.41)
SES-CD score, mean (SD)	13.3 (7.55)	13.2 (7.43)	12.5 (7.24)	12.8 (7.34)	12.0 (6.89)	12.2 (6.85)	11.8 (7.12)	12.0 (6.97)
Endoscopic disease severity (SES-CD score), n (%)								
Moderate (7–16)	77 (52.0)	164 (57.3)	147 (49.7)	311 (53.4)	61 (52.1)	64 (55.7)	49 (42.6)	113 (49.1)
Severe (>16)	43 (29.1)	81 (28.3)	79 (26.7)	160 (27.5)	25 (21.4)	26 (22.6)	27 (23.5)	53 (23.0)
Involved disease location by central reader, n (%)								
Colon only	62 (41.9)	113 (39.5)	112 (37.8)	225 (38.7)	40 (34.2)	41 (35.7)	40 (34.8)	81 (35.2)
Ileum only	31 (20.9)	59 (20.6)	80 (27.0)	139 (23.9)	22 (18.8)	25 (21.7)	27 (23.5)	52 (22.6)
Ileum and colon	55 (37.2)	114 (39.9)	104 (35.1)	218 (37.5)	55 (47.0)	49 (42.6)	48 (41.7)	97 (42.2)
Biomarkers								
C-reactive protein, mg/L, median (IQR)	5.1 (1.5; 15.8)	7.7 (2.6; 21.8)	6.2 (2.7; 21.3)	6.7 (2.6; 21.8)	79 (2.1; 14.7)	5.2 (1.7; 13.3)	5.7 (1.7; 16.1)	5.5 (1.7; 14.9)
Faecal calprotectin, µg/g,* median (IQR)	962.0 (255.0; 2595.0)	969.0 (404.0; 2085.0)	1045.5 (323.0; 2006.0)	1010.0 (372.0; 2054.0)	712.0 (243.0; 1724.0)	610.0 (226.0; 1554.0)	600.5 (235.0; 1650.0)	610.0 (228.0; 1608.0)
Biologic medication history, n (%)								
BIO-naïve	61 (41.2)	116 (40.6)	128 (43.2)	244 (41.9)	56 (47.9)	53 (46.1)	52 (45.2)	105 (45.7)
History of inadequate response/ intolerance to biologic therapy,† n (%)	78 (52.7)	153 (53.5)	147 (49.7)	300 (51.5)	53 (45.3)	55 (47.8)	53 (46.1)	108 (47.0)
Concomitant CD medications at baseline, n (%)								
6-MP/AZA	36 (24.3)	85 (29.7)	89 (30.1)	174 (29.9)	32 (27.4)	28 (24.3)	36 (31.3)	64 (27.8)
MTX	4 (2.7)	2 (0.7)	7 (2.4)	9 (1.5)	1 (0.9)	1 (0.9)	1 (0.9)	2 (0.9)
Oral corticosteroids	51 (34.5)	109 (38.1)	106 (35.8)	215 (36.9)	33 (28.2)	32 (27.8)	38 (33.0)	70 (30.4)

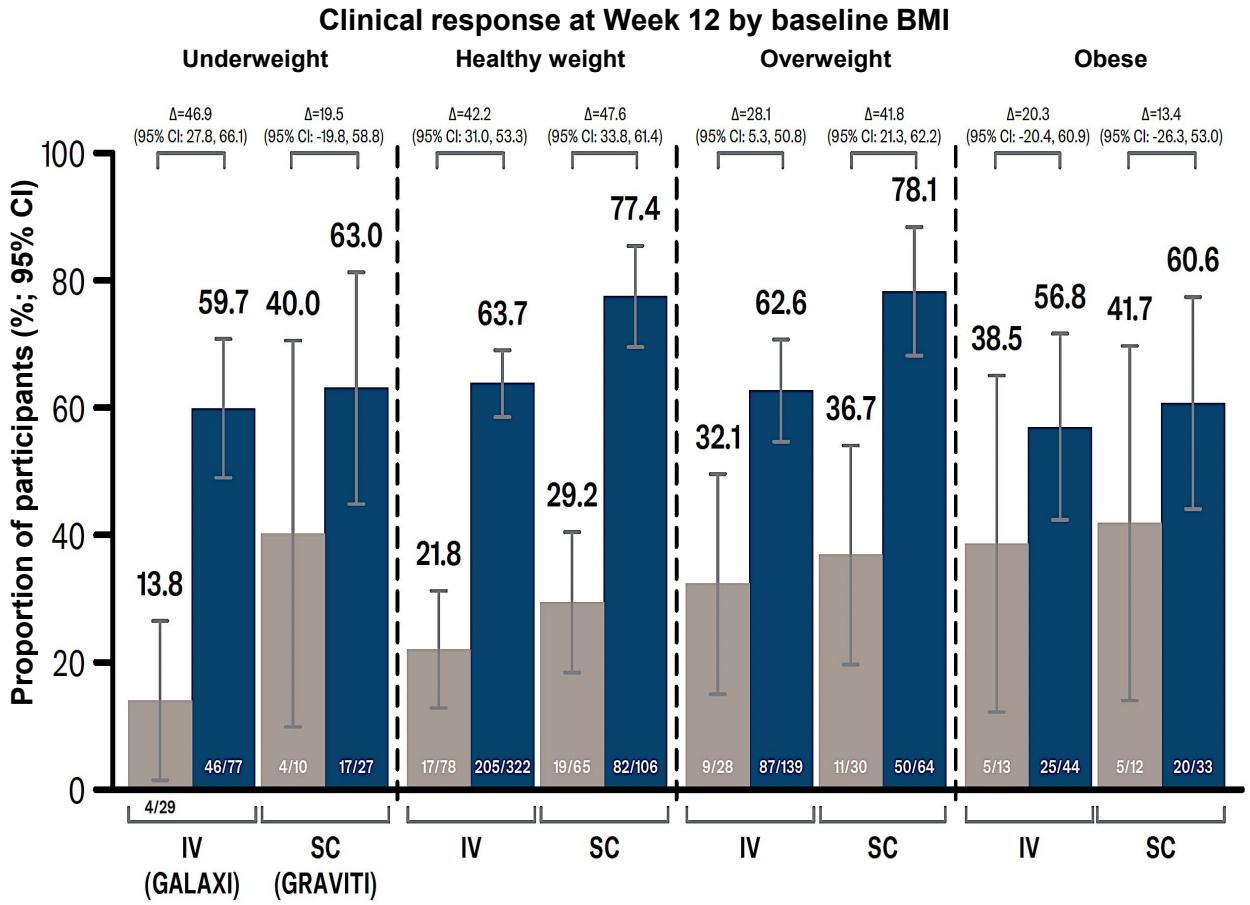
*Based on n=147 for placebo, n=283 for GUS 100 mg, n=294 for GUS 200 mg and n=577 for GUS combined in GALAXI; and n=117 for placebo, n=115 for GUS 100 mg, n=114 for GUS 200 mg and n=229 for GUS combined in GRAVITI; †Primary non-response, secondary non-response or intolerance.

6-MP, mercaptopurine; AP, abdominal pain; AZA, azathioprine; BIO, biologic; 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; PBO, placebo; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous; SD standard deviation; SES-CD, Simple Endoscopic Score for Crohn's Disease; TNF, tumour necrosis factor.

Deepak P, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. Poster P1110. Full prescribing information: www.swissmedicinfo-pro.ch.

Clinical response at Week 12 by baseline weight and BMI





PBO (IV or SC)

GUS induction (200 mg IV or 400 mg SC)

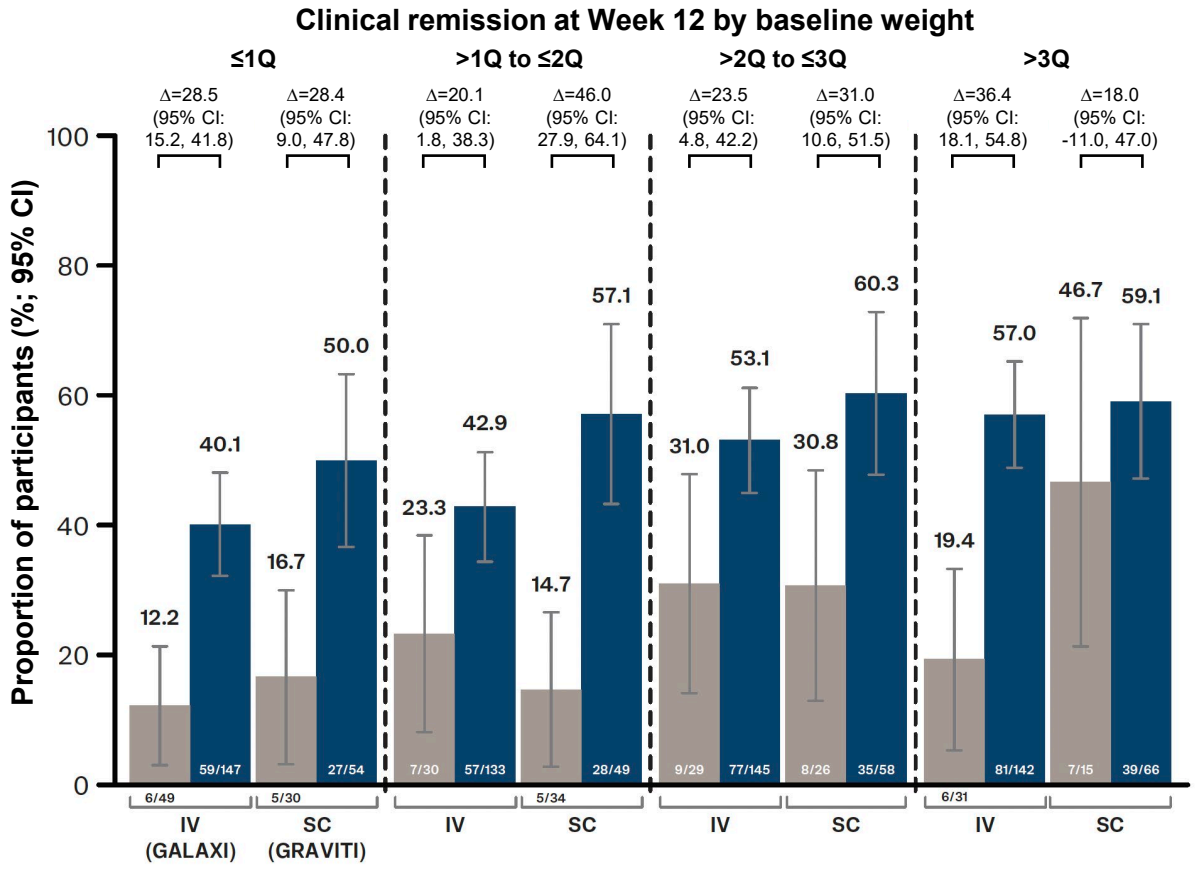
Clinical response: ≥100-point reduction from baseline in CDAI score or CDAI score <150.

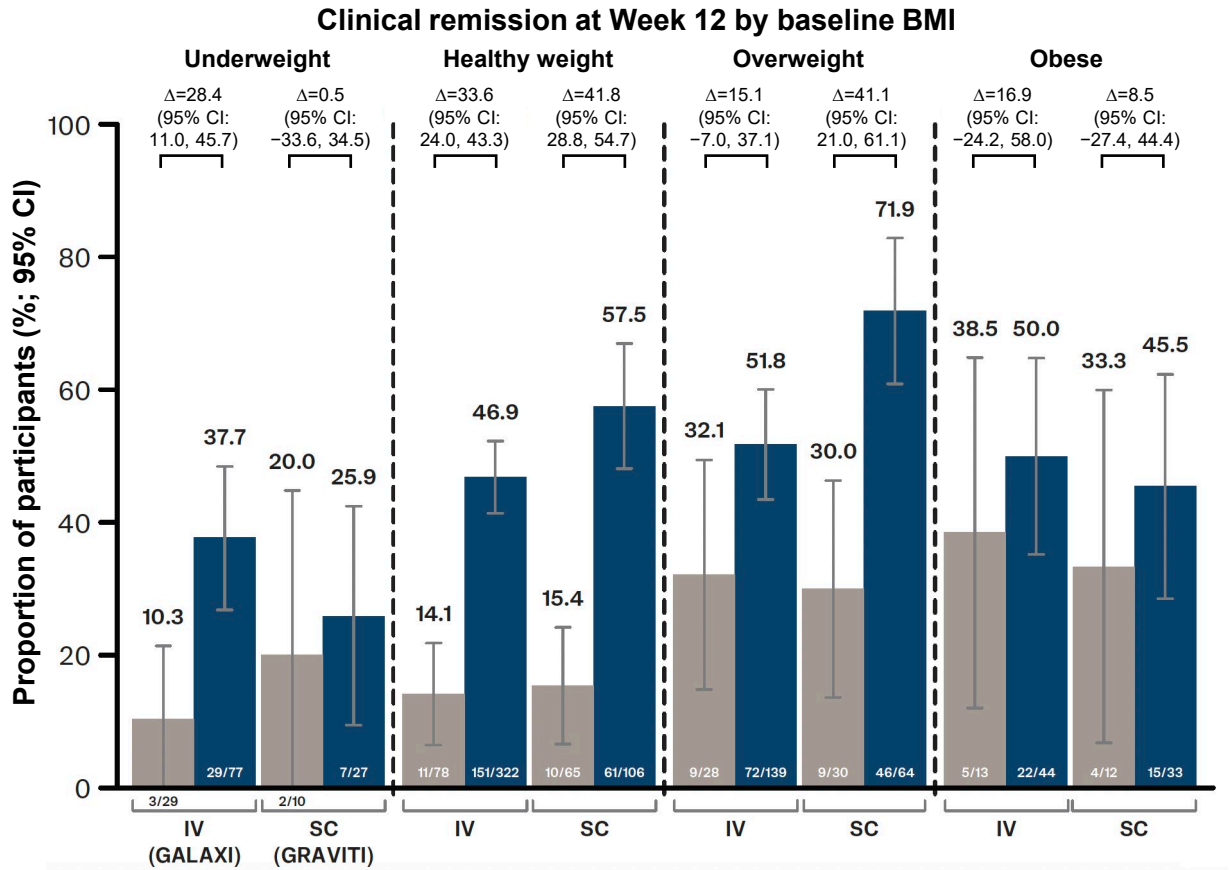
Standard BMI subgroups were used (i.e. underweight [<18 kg/m^2], healthy weight [≥18 to <25 kg/m^2], overweight [≥25 to <30 kg/m^2] and obese [≥30 kg/m^2]). Weight quartiles were calculated for each time point using only the treatment arms assessed at that time point (i.e. placebo and GUS for Week 12 and only GUS for Week 48). Weight quartiles for Week 12 analyses were based on participants in the primary analysis set as follows:
GALAXI—57 kg (1Q), 67.1 kg (2Q), 79 kg (3Q); and GRAVITI—59 kg (1Q), 69.5 kg (2Q), 81 kg (3Q).

BMI, body mass index; CD, Crohn's disease; CDAI, Crohn's Disease Activity Index; CI, confidence interval; GUS, guselkumab; IL, interleukin; IV, intravenous; MOA, mechanism of action; Q, quartile; PBO, placebo; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous.

Deepak P, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. Poster P1110. Full prescribing information: www.swissmedicinfo-pro.ch.

Clinical remission at Week 12 by baseline weight and BMI





PBO (IV or SC) | GUS induction (200 mg IV or 400 mg SC)

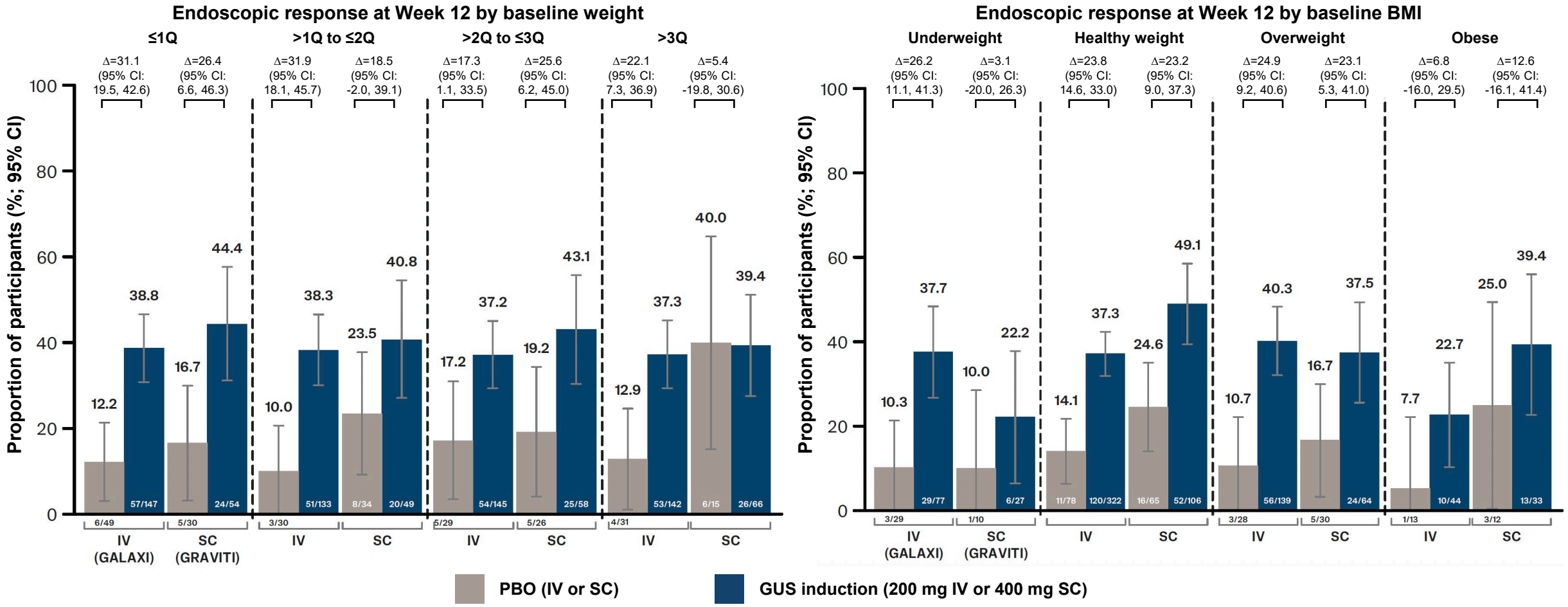
Clinical remission: CDAI <150.

Standard BMI subgroups were used (i.e. underweight [<18 kg/m^2], healthy weight [≥18 to <25 kg/m^2], overweight [≥25 to <30 kg/m^2] and obese [≥30 kg/m^2]). Weight quartiles were calculated for each time point using only the treatment arms assessed at that time point (i.e. placebo and GUS for Week 12 and only GUS for Week 48). Weight quartiles for Week 12 analyses were based on participants in the primary analysis set as follows: GALAXI—57 kg (1Q), 67.1 kg (2Q), 79 kg (3Q); and GRAVITI—59 kg (1Q), 69.5 kg (2Q), 81 kg (3Q).

BMI, body mass index; CD, Crohn's disease; CDAI, Crohn's Disease Activity Index; CI, confidence interval; GUS, guselkumab; IL, interleukin; IV, intravenous; MOA, mechanism of action; Q, quartile; PBO, placebo; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous.

Deepak P, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. Poster P1110. Full prescribing information: www.swissmedicinfo-pro.ch.

Endoscopic response at Week 12 by baseline weight and BMI



Endoscopic response: ≥50% improvement from baseline in SES-CD.

Standard BMI subgroups were used (i.e. underweight [<18 kg/m^2], healthy weight [≥18 to <25 kg/m^2], overweight [≥25 to <30 kg/m^2] and obese [≥30 kg/m^2]). Weight quartiles were calculated for each time point using only the treatment arms assessed at that time point (i.e. placebo and GUS for Week 12 and only GUS for Week 48). Weight quartiles for Week 12 analyses were based on participants in the primary analysis set as follows:
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BMI, body mass index; CD, Crohn's disease; CI, confidence interval; GUS, guselkumab; IL, interleukin; IV, intravenous; MOA, mechanism of action; Q, quartile; PBO, placebo; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous; SES-CD, Simple Endoscopic Score for Crohn's Disease.

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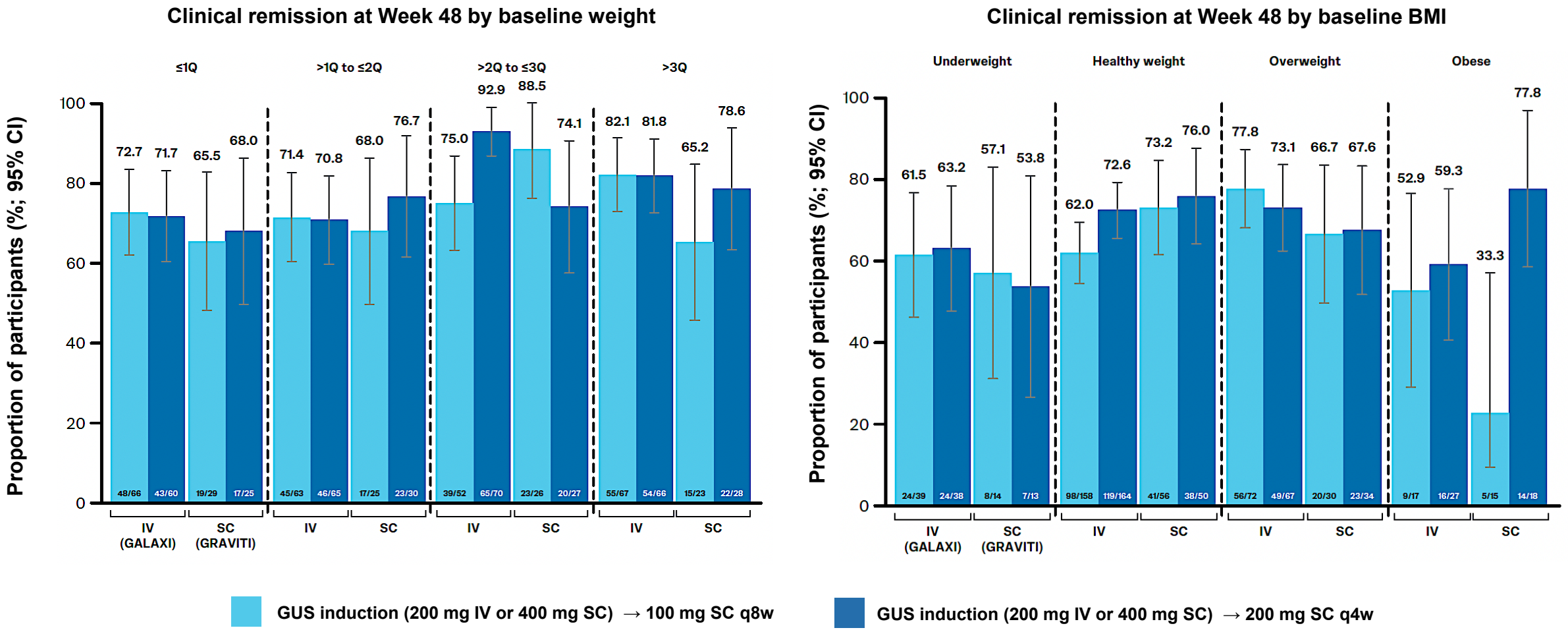
Clinical remission at Week 48 by baseline weight and BMI



Clinical remission: CDAI <150.

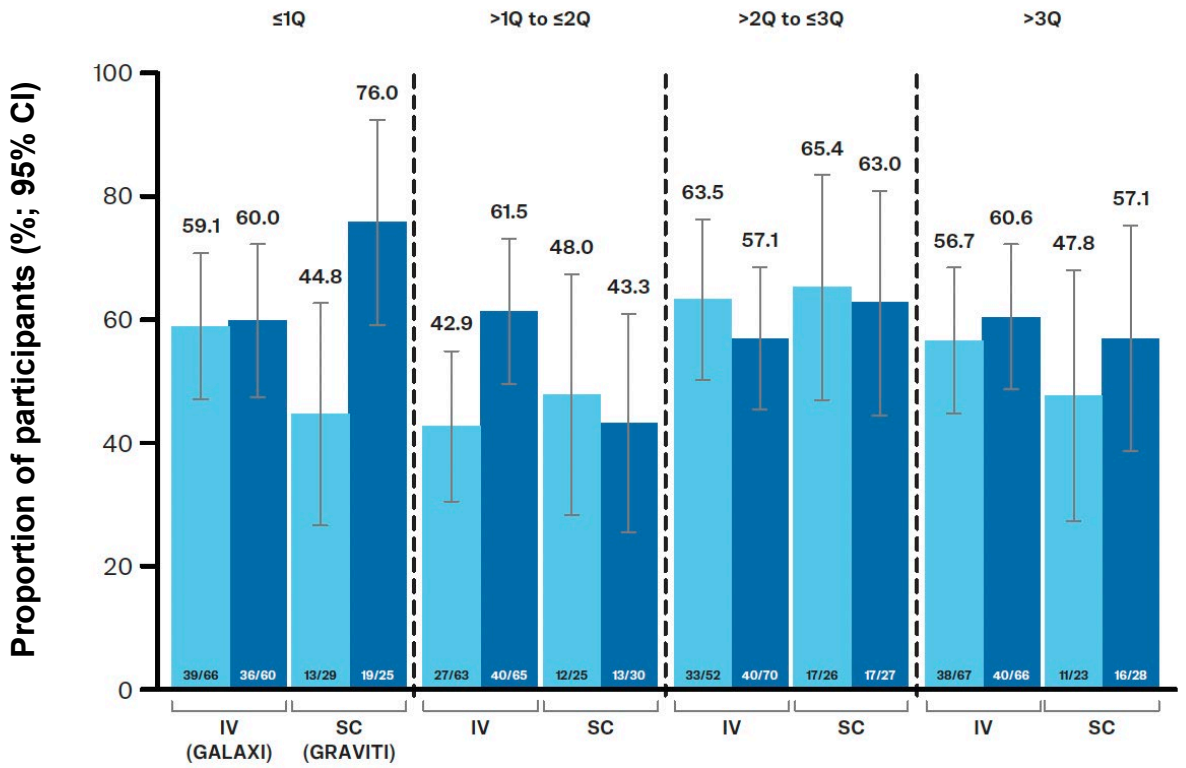
Standard BMI subgroups were used (i.e. underweight [<18 kg/m^2], healthy weight [≥18 to <25 kg/m^2], overweight [≥25 to <30 kg/m^2] and obese [≥30 kg/m^2]). Weight quartiles were calculated for each time point using only the treatment arms assessed at that time point (i.e. placebo and GUS for Week 12 and only GUS for Week 48). Weight quartiles for Week 48 analyses were based on participants in the primary analysis set as follows: GALAXI—58.6 kg (1Q), 70 kg (2Q), 81 kg (3Q); and GRAVITI—62 kg (1Q), 73 kg (2Q), 85 kg (3Q).

BMI, body mass index; CD, Crohn's disease; CDAI, Crohn's Disease Activity Index; CI, confidence interval; GUS, guselkumab; IL, interleukin; IV, intravenous; MOA, mechanism of action; Q, quartile; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous.

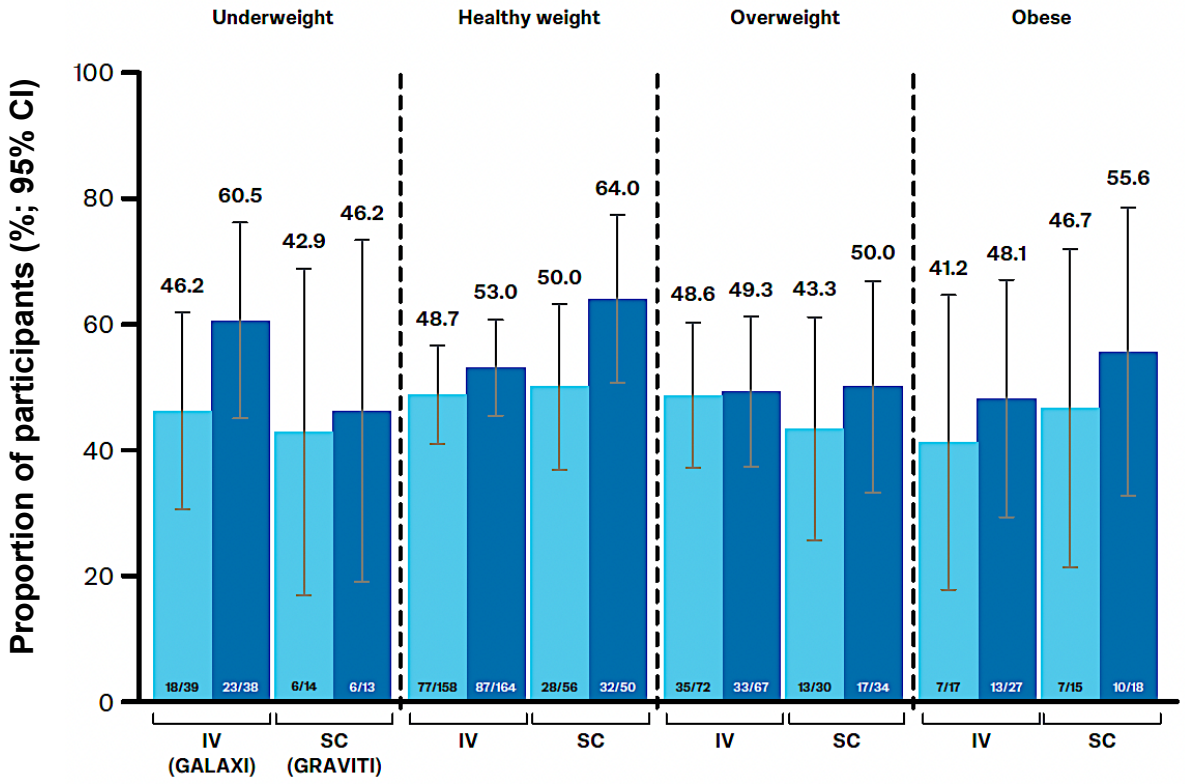
Deepak P, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. Poster P1110. Full prescribing information: www.swissmedicinfo-pro.ch.

Endoscopic response at Week 48 by baseline weight and BMI

Endoscopic response at Week 48 by baseline weight



Endoscopic response at Week 48 by baseline BMI



GUS induction (200 mg IV or 400 mg SC) → 100 mg SC q8w

GUS induction (200 mg IV or 400 mg SC) → 200 mg SC q4w

Endoscopic response: ≥50% improvement from baseline in SES-CD.
Standard BMI subgroups were used (i.e. underweight [<18 kg/m^2], healthy weight [≥18 to <25 kg/m^2], overweight [≥25 to <30 kg/m^2] and obese [≥30 kg/m^2]). Weight quartiles were calculated for each time point using only the treatment arms assessed at that time point (i.e. placebo and GUS for Week 12 and only GUS for Week 48). Weight quartiles for Week 48 analyses were based on participants in the primary analysis set as follows: GALAXI—58.6 kg (1Q), 70 kg (2Q), 81 kg (3Q); and GRAVITI—62 kg (1Q), 73 kg (2Q), 85 kg (3Q).
BMI, body mass index; CD, Crohn's disease; CI, confidence interval; GUS, guselkumab; IL, interleukin; IV, intravenous; MOA, mechanism of action; Q, quartile; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous; SES-CD, Simple Endoscopic Score for Crohn's Disease.
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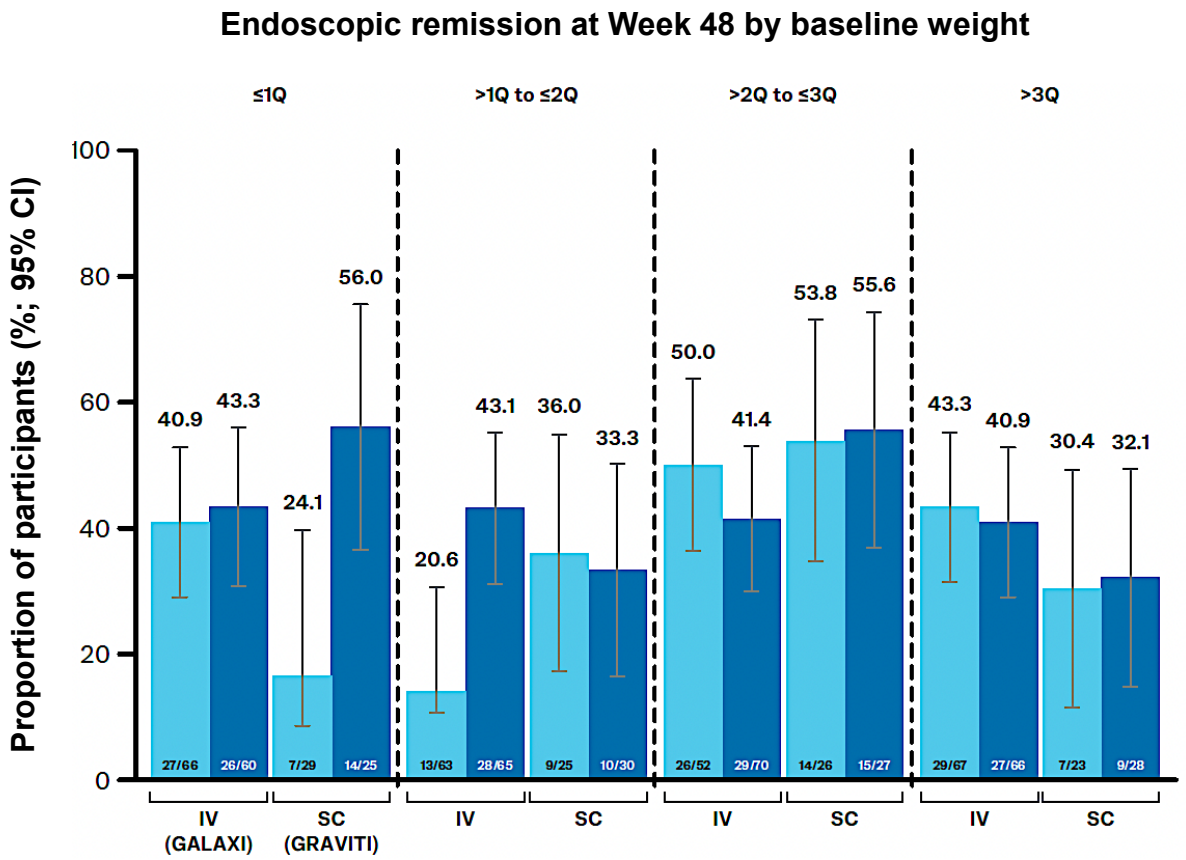
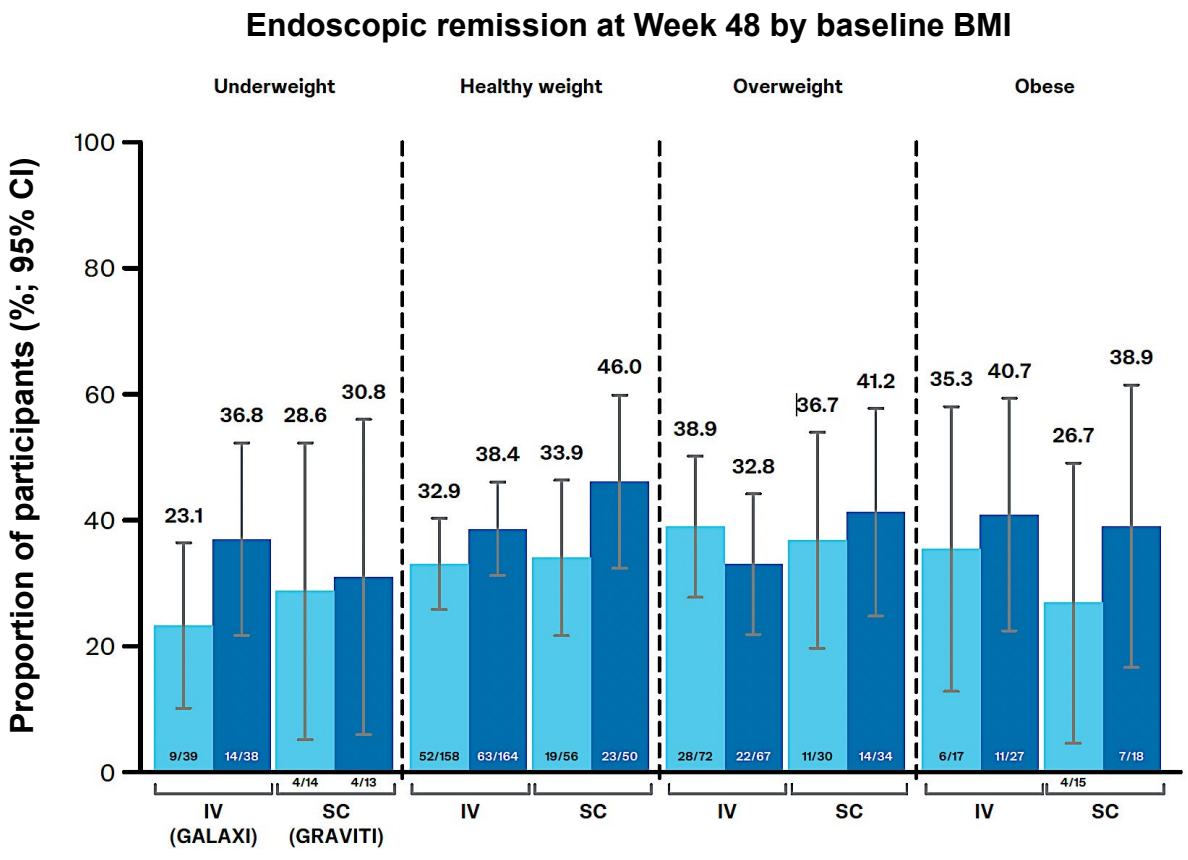
Endoscopic remission at Week 48 by baseline weight and BMI



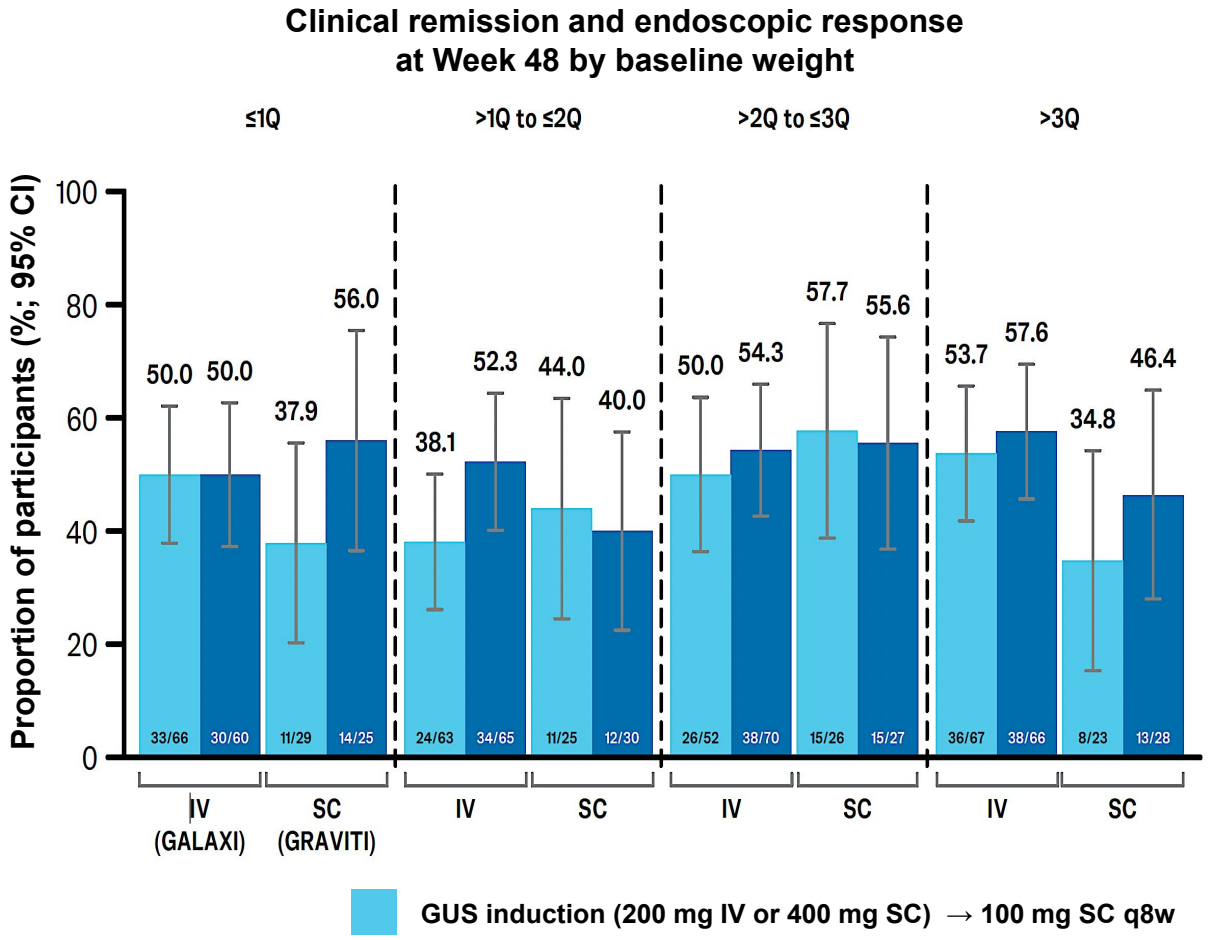


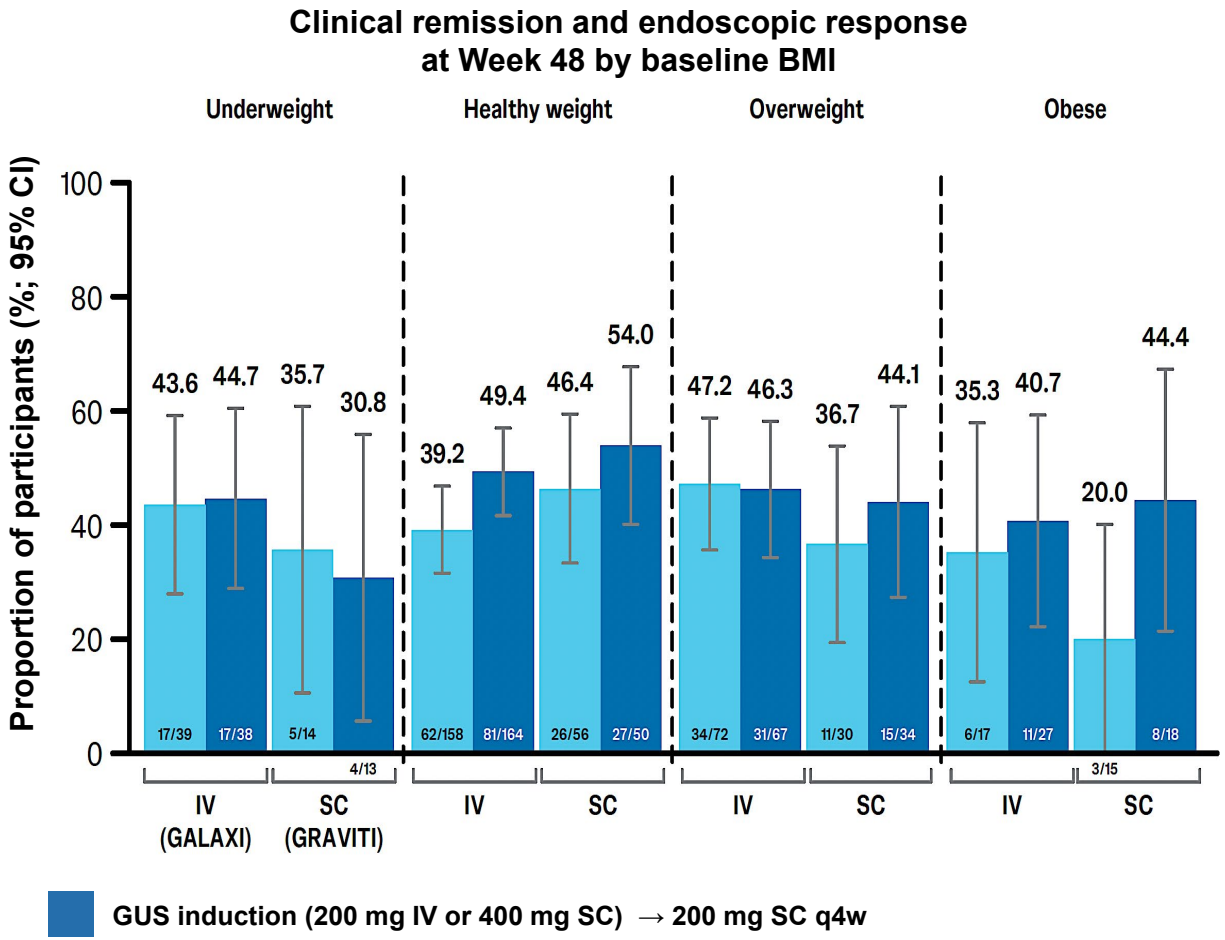
GUS induction (200 mg IV or 400 mg SC) → 100 mg SC q8w

GUS induction (200 mg IV or 400 mg SC) → 200 mg SC q4w

Endoscopic remission: SES-CD ≤4, ≥2-point reduction from baseline in SES-CD and no SES-CD subscore >1.
Standard BMI subgroups were used (i.e. underweight [<18 kg/m^2], healthy weight [≥18 to <25 kg/m^2], overweight [≥25 to <30 kg/m^2] and obese [≥30 kg/m^2]). Weight quartiles were calculated for each time point using only the treatment arms assessed at that time point (i.e. placebo and GUS for Week 12 and only GUS for Week 48). Weight quartiles for Week 48 analyses were based on participants in the primary analysis set as follows:
GALAXI—58.6 kg (1Q), 70 kg (2Q), 81 kg (3Q); and GRAVITI—62 kg (1Q), 73 kg (2Q), 85 kg (3Q).
BMI, body mass index; CD, Crohn's disease; CI, confidence interval; GUS, guselkumab; IL, interleukin; IV, intravenous; MOA, mechanism of action; Q, quartile; qXw, every X weeks;
RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous; SES-CD, Simple Endoscopic Score for Crohn's Disease.
Deepak P, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. Poster P1110. Full prescribing information: www.swissmedicinfo-pro.ch.

Clinical remission and endoscopic response at Week 48 by baseline weight and BMI





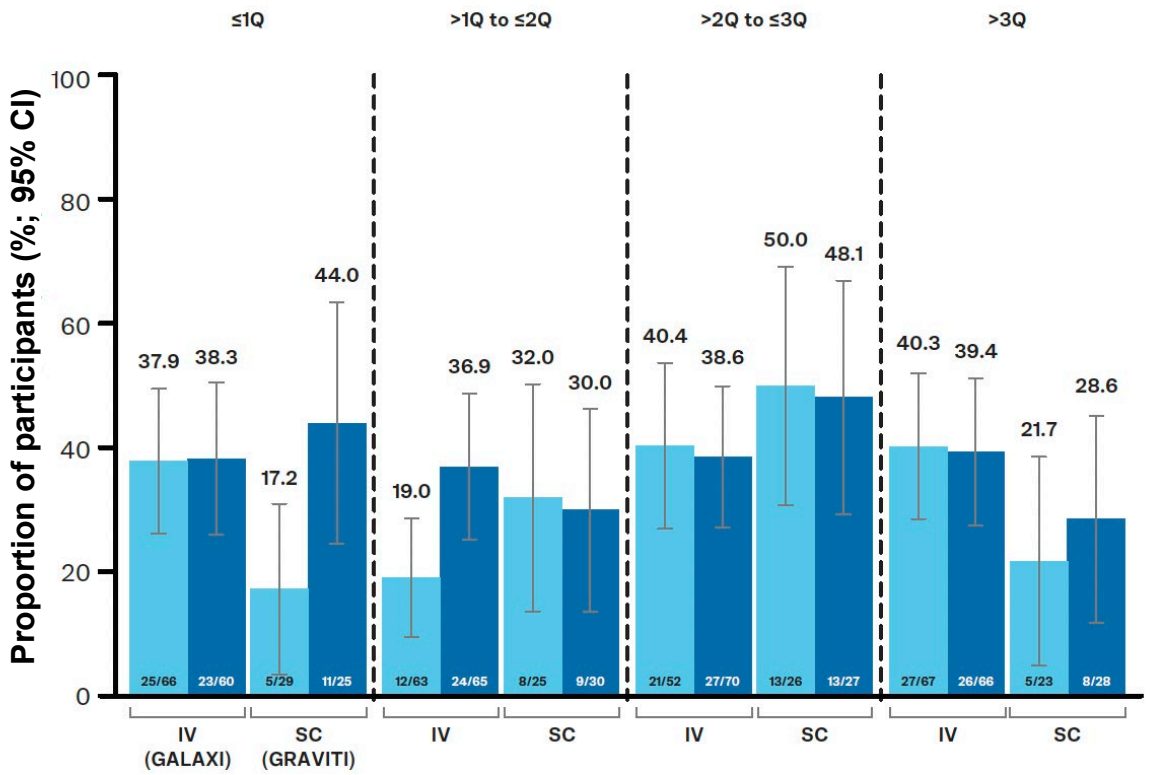
GUS induction (200 mg IV or 400 mg SC) → 100 mg SC q8w

GUS induction (200 mg IV or 400 mg SC) → 200 mg SC q4w

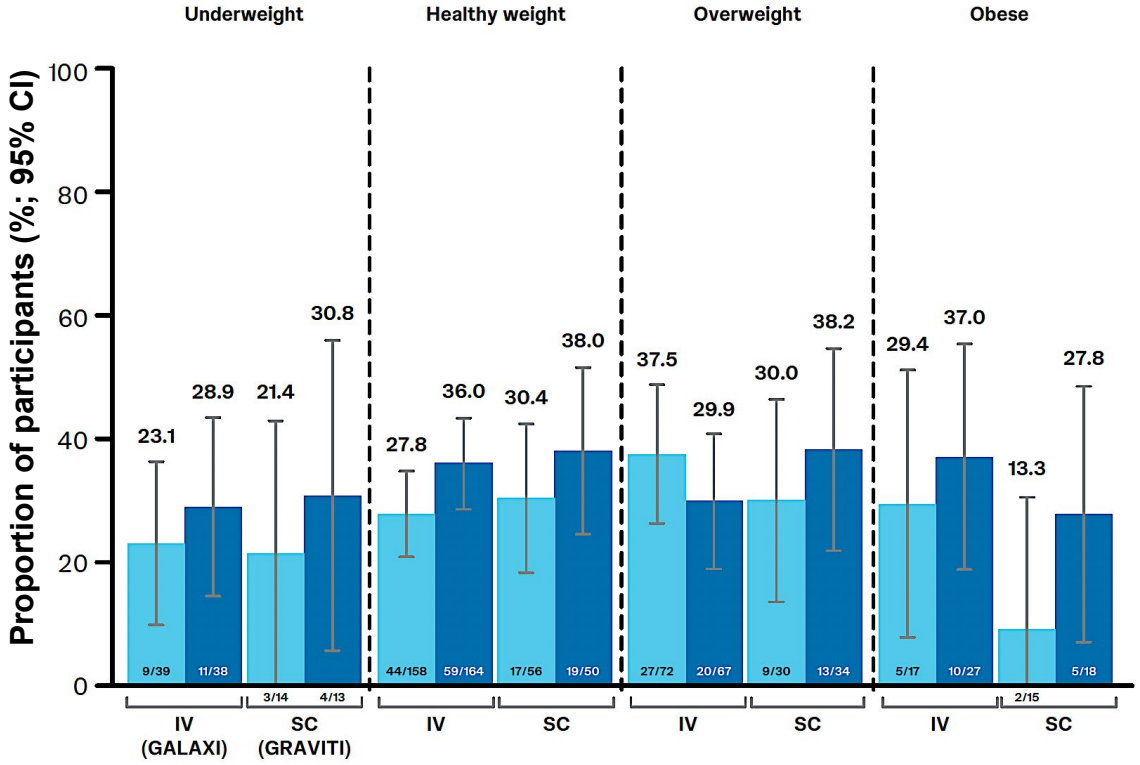
Clinical remission: CDAI <150. **Endoscopic response:** ≥50% improvement from baseline in SES-CD.
Standard BMI subgroups were used (i.e. underweight [<18 kg/m^2], healthy weight [≥18 to <25 kg/m^2], overweight [≥25 to <30 kg/m^2] and obese [≥30 kg/m^2]). Weight quartiles were calculated for each time point using only the treatment arms assessed at that time point (i.e. placebo and GUS for Week 12 and only GUS for Week 48). Weight quartiles for Week 48 analyses were based on participants in the primary analysis set as follows: GALAXI—58.6 kg (1Q), 70 kg (2Q), 81 kg (3Q); and GRAVITI—62 kg (1Q), 73 kg (2Q), 85 kg (3Q).
BMI, body mass index; CD, Crohn's disease; CDAI, Crohn's Disease Activity Index; CI, confidence interval; GUS, guselkumab; IL, interleukin; IV, intravenous; MOA, mechanism of action; Q, quartile; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous; SES-CD, Simple Endoscopic Score for Crohn's Disease.
Deepak P, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. Poster P1110. Full prescribing information: www.swissmedicinfo-pro.ch.

Deep remission at Week 48 by baseline weight and BMI

Deep remission at Week 48 by baseline weight



Deep remission at Week 48 by baseline BMI



GUS induction (200 mg IV or 400 mg SC) → 100 mg SC q8w

GUS induction (200 mg IV or 400 mg SC) → 200 mg SC q4w

Clinical remission: CDAI <150. **Endoscopic remission:** SES-CD ≤4, ≥2-point reduction from baseline in SES-CD and no SES-CD subscore >1. **Deep remission:** clinical remission and endoscopic remission.
Standard BMI subgroups were used (i.e. underweight [<18 kg/m^2], healthy weight [≥18 to <25 kg/m^2], overweight [≥25 to <30 kg/m^2] and obese [≥30 kg/m^2]). Weight quartiles were calculated for each time point using only the treatment arms assessed at that time point (i.e. placebo and GUS for Week 12 and only GUS for Week 48). Weight quartiles for Week 48 analyses were based on participants in the primary analysis set as follows: GALAXI—58.6 kg (1Q), 70 kg (2Q), 81 kg (3Q); and GRAVITI—62 kg (1Q), 73 kg (2Q), 85 kg (3Q).
BMI, body mass index; CD, Crohn's disease; CDAI, Crohn's Disease Activity Index; CI, confidence interval; GUS, guselkumab, IL, interleukin; IV, intravenous; MOA, mechanism of action; Q, quartile; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous; SES-CD, Simple Endoscopic Score for Crohn's Disease.
Deepak P, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. Poster P1110. Full prescribing information: www.swissmedicinfo-pro.ch.

Conclusions

- GUS IV and SC induction were similarly effective in participants with moderately to severely active CD compared with placebo at Week 12, regardless of participants' baseline body weight or BMI
- Similar efficacy was observed at Week 48 between participants treated with either GUS IV or SC induction, regardless of participants' baseline body weight or BMI
- Efficacy of GUS does not appear to be impacted by body weight or BMI, irrespective of route of induction treatment administration

BMI, body mass index; CD, Crohn's disease; GUS, guselkumab, IL, interleukin; IV, intravenous; MOA, mechanism of action; q4w/q8w, every 4 or 8 weeks; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous.

Deepak P, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. Poster P1110. Full prescribing information: www.swissmedicinfo-pro.ch.

P1043

Unsupervised machine learning to identify distinct response patterns to guselkumab in participants with Crohn's disease: Post hoc analysis of the GRAVITI and GALAXI 2/3 studies

Schreiber S,[1] D'Haens G,[2] Reinisch W,[3] Hart A,[4] Rani S,[5] Olurinde M,[5] Rampelbergh R Van,[6] Gao L-L,[5] Yang Z,[5] Hoops T,[7] Valiathan C,[8] Dulai PS[9]

[1]Department of Internal Medicine I, Christian-Albrechts-University and University Hospital Schleswig-Holstein, Kiel, Germany; [2]Department of Gastroenterology, Amsterdam University Medical Centers, Amsterdam, The Netherlands; [3]Division of Gastroenterology & Hepatology, Medical University of Vienna, Vienna, Austria; [4]London North-West University Healthcare NHS Trust, London, United Kingdom; [5]Johnson & Johnson, Spring House, PA, USA; [6]Johnson & Johnson, Antwerp, Belgium; [7]Johnson & Johnson, Horsham, PA, USA; [8]Johnson & Johnson, La Jolla, CA, USA; [9]Division of Gastroenterology & Hepatology, Northwestern University, Chicago, IL, USA

Unsupervised machine learning to identify distinct response patterns to GUS in participants with CD: *post hoc* analysis of the GRAVITI and GALAXI 2/3 studies

Background

Crohn's disease (CD) trials often report efficacy using only binary endpoints (clinical remission/endoscopic response), aggregating outcomes of all participants at specified timepoints. Individual longitudinal efficacy trends using participant-level clinical data helps to understand response dynamics in relationship to outcome, and may help optimize personalized IBD treatment. AI, such as machine learning, may identify patterns/predictors of individual treatment response in participants with CD. GUS is an IL-23p19 subunit antagonist. Unsupervised machine learning was used to identify clusters of GUS-treated participants by individual Crohn's Disease Activity Index (CDAI)-based response trajectories.

Methods

Unsupervised machine learning approach with latent class trajectory modelling was applied using CDAI scores from Weeks 0–48 of individual GUS-treated participants with moderate to severely active CD from three Phase 3, randomized, double-blind, treat-through trials: GRAVITI (GV; SC induction & maintenance), GALAXI 2 (G2) & 3 (G3) (both IV induction, SC maintenance). Participants could be separated into distinct clusters using dynamics of longitudinal CDAI score responses. GUS-treated participants with all CDAI data were included. The optimal cluster number was determined using multiple clustering metrics. A model was first created using GV CDAI trajectories and applied to pooled G2/3. Participant characteristics at baseline/Week 4/Week 12 were evaluated for association with response patterns. CDAI was visualised by cluster as a heatmap using participant-level data. Participants in endoscopic remission at Weeks 12&48 were assessed. All post hoc analyses are descriptive.

Results

In GV, 5 distinct participant groups were detected representing clusters with different CDAI response dynamics & arranged by greatest to partial response: A (22% of participants), B (20%), C (21%), D (23%), & E (15%). CDAI improvements occurred in all clusters. Applying clustering to G2/3 data confirmed similar trajectories. Heatmap analysis of GV participant-level CDAI scores through Week 48 enabled visualisation of response profiles. In G2/3, median CD duration & CDAI scores at baseline, and CRP & fecal calprotectin at Weeks 4&12 had trends indicative of response trajectories. Similar response patterns were observed across clusters for participants in endoscopic remission at Week 12 (A:33.8%; E:14.3%) & Week 48 (A:46.8%; E:21.4%).

Conclusion

Unsupervised machine learning using CDAI scores of GUS-treated pts detected 5 distinct response clusters in GRAVITI and GALAXI. Through Week 48, distinct participant groups were separated by degrees/dynamics of clinical response, which were similar with SC and IV induction. Further analyses of participant characteristics related to these response patterns may direct long-term therapies and aid in an individualized treatment approach to IBD.

CD, Crohn's disease; CDAI, Crohn's Disease Activity Index; G, GALAXI; GUS, guselkumab; GV, GRAVITI; IBD, inflammatory bowel disease; IL, interleukin; IV, intravenous; MOA, mechanism of action; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous.

Schreiber S, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. Poster P1043. Full prescribing information: www.swissmedicinfo-pro.ch.

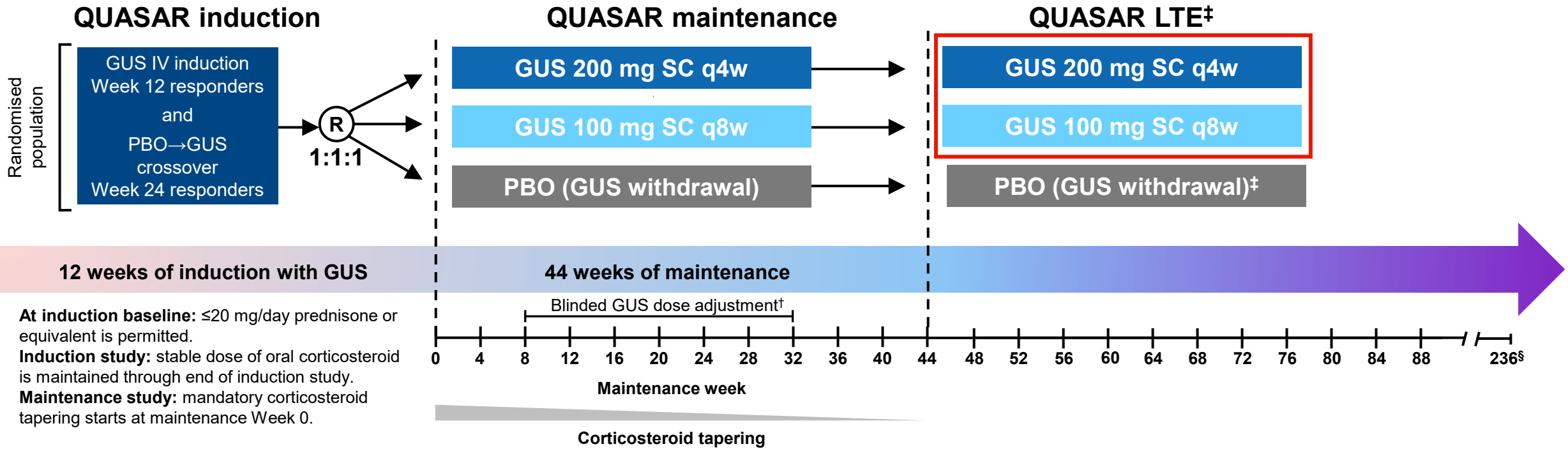
Guselkumab in ulcerative colitis

QUASAR

Immunology

QUASAR study design

Population
- Adults with moderately to severely active UC* who were in clinical response 12 weeks following GUS IV induction



At induction baseline: ≤20 mg/day prednisone or equivalent is permitted.
Induction study: stable dose of oral corticosteroid is maintained through end of induction study.
Maintenance study: mandatory corticosteroid tapering starts at maintenance Week 0.

Overall, 87% of participants randomised to GUS at maintenance Week 0 entered the LTE, and approximately 89% of the LTE-randomised GUS-treated population completed treatment through LTE Week 140
- Reasons for discontinuation were AE, lack of efficacy, lost to follow-up, death, withdrawal by participant, physician decision, pregnancy and other

*Defined as induction baseline modified Mayo score of 5 to 9 with a Mayo RBS ≥1 and an MES ≥2 based on central review; †Between maintenance Weeks 8 and 32, 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: PBO 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); ‡The study blind was maintained during the LTE until the last participant in the maintenance study completed the maintenance Week 44 visit. After the maintenance study was unblinded to the investigative sites, participants receiving PBO were terminated from study participation; §Week 236 is the final efficacy visit; the final safety visit is 12 weeks from the last GUS dose (approximately Week 244).
AE, adverse event; GUS, guselkumab; IV, intravenous; LTE, long-term extension; MES, Mayo endoscopy score; MOA, mechanism of action; PBO, placebo; qXw, every X weeks; R, randomisation; RBS, rectal bleeding subscore; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous; UC, ulcerative colitis.
Peyrin-Biroulet L, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. DOP104. Full prescribing information: www.swissmedicinfo-pro.ch.

DOP104

Efficacy and safety of guselkumab for ulcerative colitis through Week 140 of the QUASAR long-term extension study

Peyrin-Biroulet L,[1] Bressler B,[2] Dignass A,[3] Hisamatsu T,[4] Sands BE,[5] Alvarez Y,[6] Baker T,[6] Shipitofsky N,[6] Miao Y,[6] Zhang H,[6] Lichtenstein GR,[7] Rubin DT,[8] Allegretti JR[9]

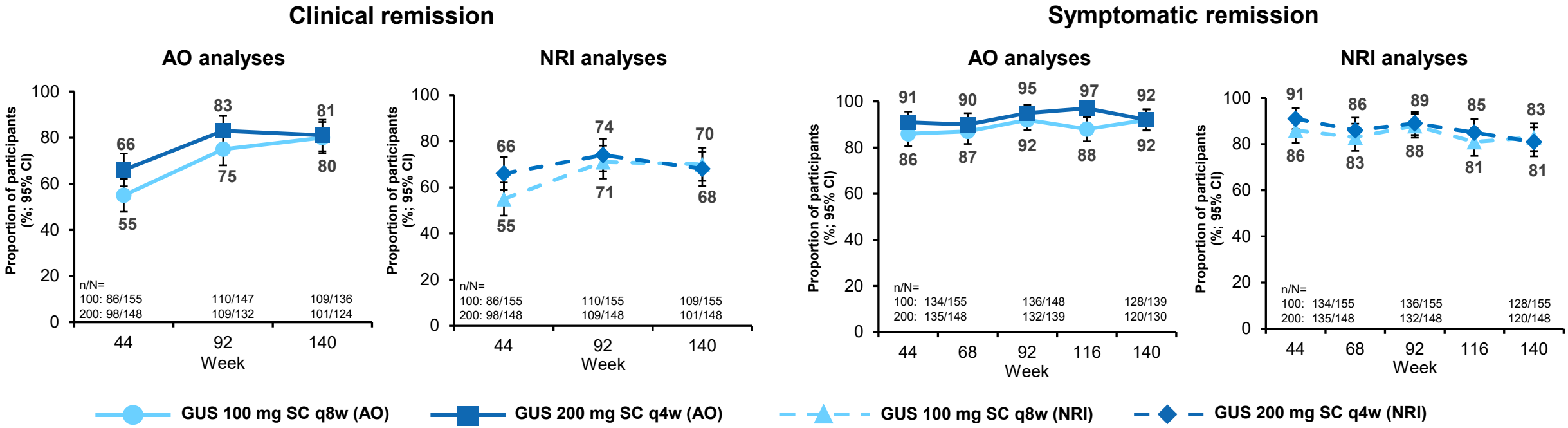
[1]Department of Gastroenterology, CHRU Nancy, INSERM NGERE, Université de Lorraine, F-54000 Nancy, France; [2]University of British Columbia, Vancouver, BC, Canada; [3]Department of Medicine I, Agaplesion Markus Hospital, Goethe University, Frankfurt, Germany; [4]Kyorin University School of Medicine, Tokyo, Japan; [5]Icahn School of Medicine at Mount Sinai, New York, NY, USA; [6]Johnson & Johnson, Spring House, PA, USA; [7]University of Pennsylvania School of Medicine, Philadelphia, PA, USA; [8]University of Chicago Medicine Inflammatory Bowel Disease Center, Chicago, IL, USA; [9]Division of Gastroenterology, Hepatology and Endoscopy, Brigham and Women's Hospital, Harvard Medical School, Boston, MA, USA

Demographics and disease characteristics at induction baseline among participants who entered the LTE

	GUS 100 mg SC q8w (n=155)	GUS 200 mg SC q4w (n=148)
Demographics		
Age, years	40.2 (12.8)	40.6 (15.1)
Male, %	54	51
Disease characteristics		
UC disease duration, years	8.2 (9.0)	8.2 (8.5)
Modified Mayo score (0–9)	6.8 (1.2)	6.9 (1.1)
Modified Mayo score 7–9 (severe), %	61	66
Mayo endoscopic subscore of 3 (severe), %	66	64
Extensive UC, %	43	47
CRP, 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)
Oral corticosteroid use, %	36	36
Immunomodulatory drug use,‡ %	25	24
UC-related biologic/JAK inhibitor medication history§		
Biologic and JAK inhibitor naïve,¶ %	58	55
History of inadequate response or intolerance to biologic and/or JAK inhibitor therapy, %	39	42

Data shown are mean (SD) unless otherwise noted. Includes only participants with a modified Mayo score 5–9 at induction baseline who were in clinical response to GUS IV induction and randomised to receive GUS maintenance treatment and did not experience a dose adjustment from Week 8 through Week 32.
*Based on N=153 for GUS SC 100 mg q8w and N=145 for GUS SC 200 mg q4w; †Based on N=133 for GUS SC 100 mg q8w and N=134 for GUS SC 200 mg q4w; ‡6-mercaptopurine, azathioprine; or methotrexate; §Includes TNF-α antagonists, vedolizumab and/or tofacitinib. ¶3% of participants in each group were biologic/JAK inhibitor-experienced without documented inadequate response/intolerance.
CRP, C-reactive protein; GUS, guselkumab; IL, interleukin; IQR, interquartile range; IV, intravenous; JAK, Janus kinase; LTE, long-term extension; MOA, mechanism of action; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous; SD, standard deviation; TNF, tumour necrosis factor; UC, ulcerative colitis.
Peyrin-Biroulet L, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. DOP104. Full prescribing information: www.swissmedicinfo-pro.ch.

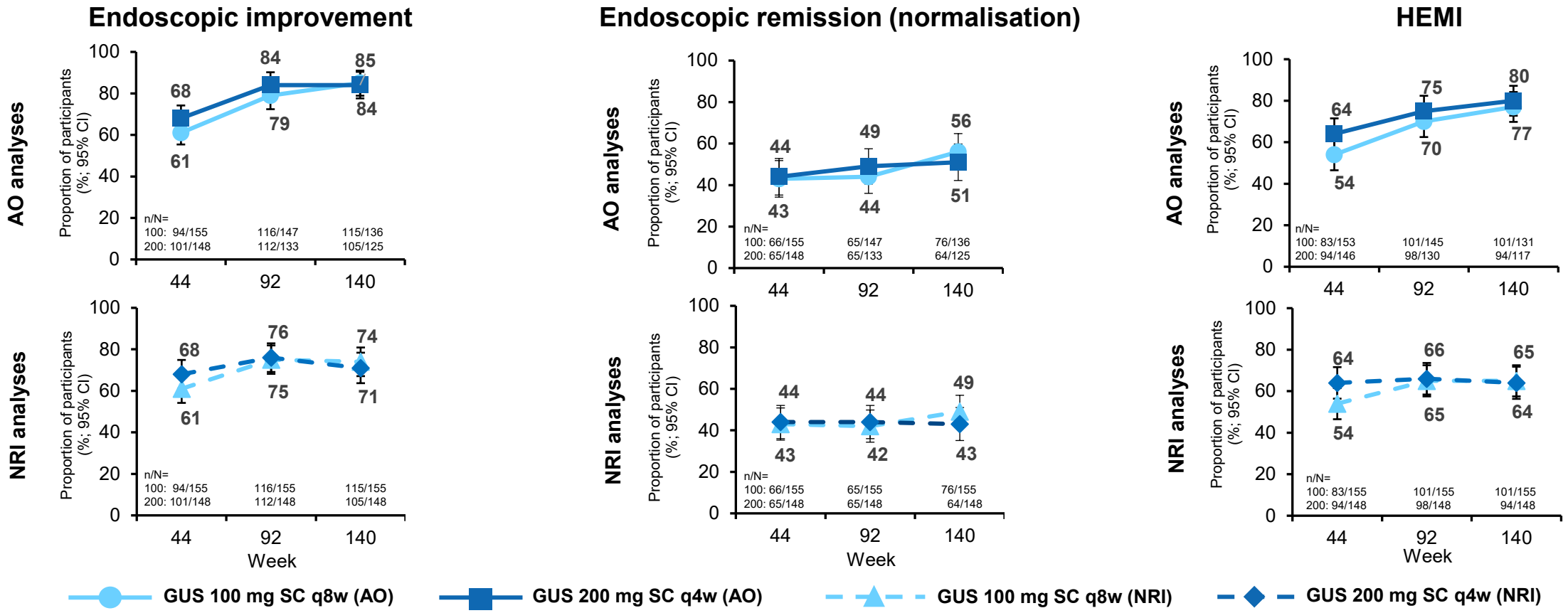
Clinical and symptomatic remission rates were sustained from Week 44 through Week 140 among LTE participants



- Overall, 205 of 210 participants (98%) in clinical remission at Week 140 were corticosteroid-free for ≥8 weeks before Week 140

Clinical remission: SFS of 0 or 1 and not increased from induction baseline, RBS of 0, MES of 0 or 1. **Symptomatic remission:** SFS of 0 or 1 and not increased from induction baseline, RBS of 0.
Includes only participants with a modified Mayo score 5–9 at induction baseline in clinical response to GUS IV induction and randomised to receive GUS maintenance treatment and did not experience a dose adjustment from maintenance Weeks 8 through 32 and continued to receive GUS treatment in the LTE (LTE randomised GUS-treated population). "As observed" analyses were based on data available at the analysis time point. For NRI analyses, participants who had an ostomy or colectomy or discontinued study agent due to lack of therapeutic effect or due to an AE of worsening of UC before a time point were considered not to have achieved binary endpoints, and for participants who discontinued study agent due to COVID-19-related reasons (excluding infection), regional crises or reasons other than those previously stated, observed values (if available) were used; after accounting for the above rules, participants missing ≥1 of the components pertaining to an endpoint were considered not to have achieved that endpoint.
AE, adverse event; AO, as observed; CI, confidence interval; GUS, guselkumab; IL, interleukin; IV, intravenous; LTE, long-term extension; MES, Mayo Endoscopic Score; MOA, mechanism of action; NRI, non-responder imputation; qXw, every X weeks; RBS, rectal bleeding subscore; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous; SFS, stool frequency subscore; UC, ulcerative colitis.
Peyrin-Biroulet L, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. DOP104. Full prescribing information: www.swissmedicinfo-pro.ch.

Endoscopic and histologic outcomes were sustained from Week 44 through Week 140 among LTE participants



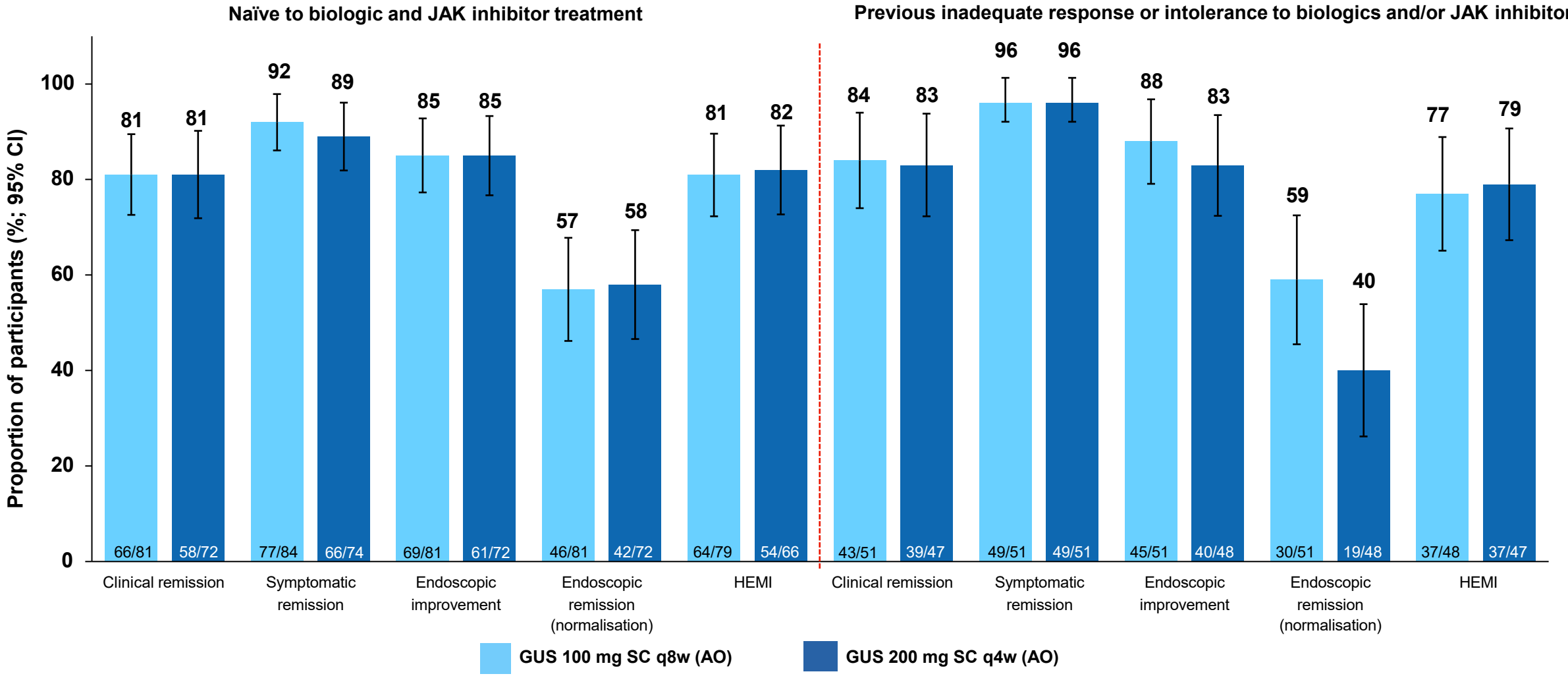
Endoscopic improvement: MES of 0 or 1. **Endoscopic remission (normalisation):** MES of 0. **HEMI:** combination of histologic improvement (neutrophil infiltration in <5% of crypts, no crypt destruction and no erosions, ulcerations or granulation tissue according to the Geboes grading system) and endoscopic improvement.

Includes only participants with a modified Mayo score 5–9 at induction baseline in clinical response to GUS IV induction and randomised to receive GUS maintenance treatment and did not experience a dose adjustment from maintenance Weeks 8 through 32 and continued to receive GUS treatment in the LTE (LTE randomised GUS-treated population). "As observed" analyses were based on data available at the analysis time point. For NRI analyses, participants who had an ostomy or colectomy or discontinued study agent due to lack of therapeutic effect or due to an AE of worsening of UC before a time point were considered not to have achieved binary endpoints, and for participants who discontinued study agent due to COVID-19-related reasons (excluding infection), regional crises or reasons other than those previously stated, observed values (if available) were used; after accounting for the above rules, participants missing ≥1 of the components pertaining to an endpoint were considered not to have achieved that endpoint.

AE, adverse event; AO, as observed; CI, confidence interval; GUS, guselkumab; HEMI, histo-endoscopic mucosal improvement; IL, interleukin; LTE, long-term extension; MES, Mayo Endoscopic Score; MOA, mechanism of action; NRI, non-responder imputation; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous; UC, ulcerative colitis.

Peyrin-Biroulet L, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. DOP104. Full prescribing information: www.swissmedicinfo-pro.ch.

Outcomes at LTE Week 140 were consistent in subpopulations with a history of treatment with a biologic and JAK inhibitor (as observed)



Includes only participants with a modified Mayo score 5–9 at induction baseline in clinical response to GUS IV induction and randomised to receive GUS maintenance treatment, and did not experience a dose adjustment from maintenance Weeks 8 through 32, and continued to receive GUS treatment in the LTE (LTE randomised GUS-treated population). "As observed" analyses were based on data available at the analysis time point.
AO, as observed; CI, confidence interval; GUS, guselkumab; HEMI, histo-endoscopic mucosal improvement; IL, interleukin; IV, intravenous; JAK, Janus kinase; LTE, long-term extension; MOA, mechanism of action; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous; UC, ulcerative colitis.

Peyrin-Biroulet L, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. DOP104. Full prescribing information: www.swissmedicinfo-pro.ch.

AEs from Week 44 through Week 140

	PBO* (n=178)	GUS 100 mg SC q8w† (n=155)	GUS 200 mg SC q4w‡ (n=332)	GUS combined (n=487)
Mean weeks of follow-up	55.0	91.3	88.8	89.6
Deaths, n (%)	1 (0.5)§	0	1 (0.3)¶	1 (0.2)
Participants with events/100 patient-years (95% CI)				
AEs	66.1 (55.0, 78.8)	45.0 (37.3, 53.7)	48.0 (42.4, 54.0)	47.0 (42.5, 51.9)
SAEs	10.7 (6.5, 16.5)	4.1 (2.0, 7.2)	6.6 (4.6, 9.0)	5.7 (4.2, 7.6)
AEs leading to discontinuation of study agent	12.8 (8.2, 19.0)	2.2 (0.8, 4.8)	4.1 (2.6, 6.1)	3.5 (2.3, 5.0)
Infection**	35.2 (27.2, 44.8)	25.1 (19.5, 31.8)	29.7 (25.4, 34.6)	28.2 (24.7, 32.1)
Serious infection**	1.1 (0.1, 3.8)	0.7 (0.1, 2.7)	1.4 (0.6, 2.8)	1.2 (0.6, 2.2)

Among GUS-treated participants:
- One death (aortic dissection in a participant with a history of hypertension and ischaemia, deemed unrelated to GUS) was reported
- No active tuberculosis, opportunistic infection, anaphylaxis, serum sickness or Hy's law cases were reported

Safety was evaluated only among participants with a modified Mayo score of 5 to 9 at induction baseline.
*Participants in clinical response to IV GUS induction randomised to PBO and did not have a dose adjustment and participants in clinical response to PBO induction and received PBO in the maintenance study; †Participants in clinical response to IV GUS induction randomised to SC GUS 100 mg q8w and did not have a dose adjustment; ‡Participants in clinical response to IV GUS induction randomised to SC GUS 200 mg q4w, participants randomised to PBO or SC GUS 100 mg q8w who had a dose adjustment, participants not in clinical response to IV GUS induction at induction Week 12 but in clinical response at induction Week 24 after receiving SC GUS. Data were summarised based on the study treatment participants were receiving upon entering the LTE; §No further information; ¶Aortic dissection; **Infections were defined as any AE coded to the MedDRA system organ class 'Infections and infestations'.
AE, adverse event; CI, confidence interval; GUS, guselkumab; IL, interleukin; IV, intravenous; LTE, long term extension; MedDRA, Medical Dictionary for Regulatory Activities; MOA, mechanism of action; PBO, placebo; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; SAE, serious adverse event; SC, subcutaneous; UC, ulcerative colitis.
Peyrin-Biroulet L, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. DOP104. Full prescribing information: www.swissmedicinfo-pro.ch.

Conclusions

- Both GUS maintenance dose regimens demonstrated sustained clinical, endoscopic and histologic efficacy in participants with UC through Week 140 of the LTE
- Although NRI results were numerically lower than as observed results, the overall trends were consistent due to the high retention rate throughout the LTE
- Efficacy was sustained regardless of biologic and/or JAK inhibitor treatment history
- Results show a favourable long-term benefit–risk profile
- No new safety concerns were observed

GUS, guselkumab; IL, interleukin; JAK, Janus kinase; LTE, long-term extension; MOA, mechanism of action; NRI, non-responder imputation; RDBPC, randomised, double-blind, placebo-controlled; UC, ulcerative colitis.
Peyrin-Biroulet L, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. DOP104. Full prescribing information: www.swissmedicinfo-pro.ch.

OP10

Symptomatic improvement with intravenous guselkumab induction therapy is observed early in patients with moderately to severely active ulcerative colitis: *Post-hoc* analysis of QUASAR

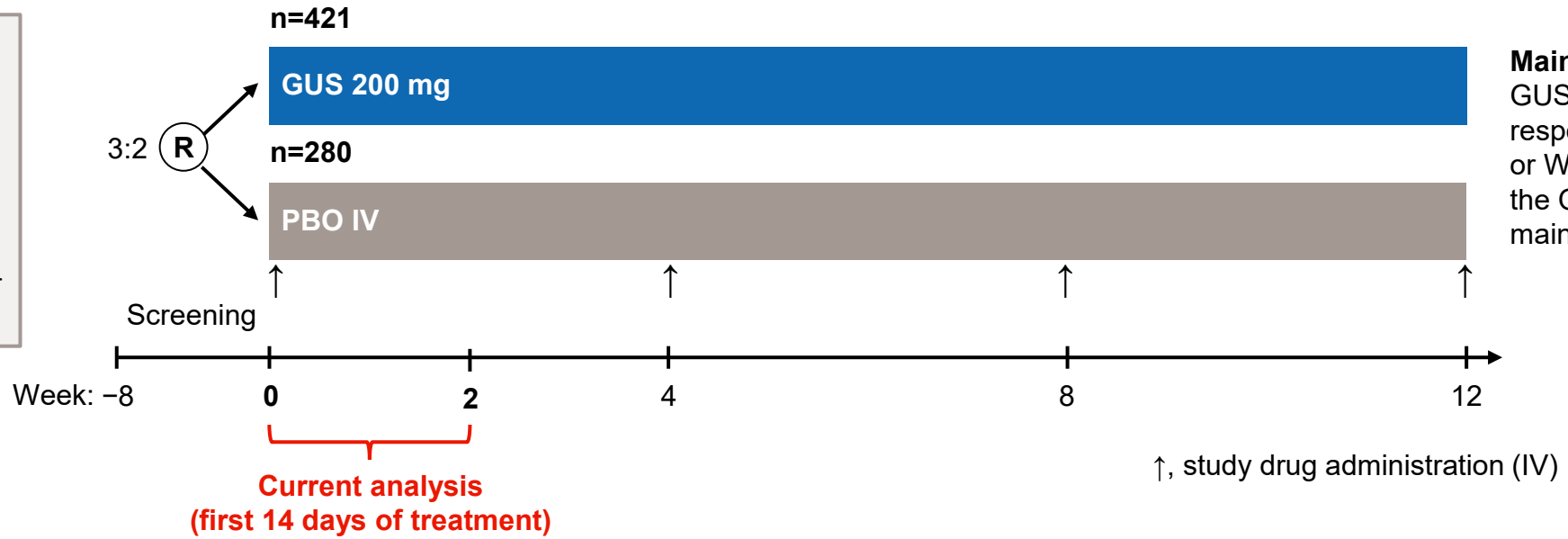
Dignass A,[1] Hirai F,[2] Saruta M,[3] Sasaki A,[4] Yoshigoe S,[4] Zhuo J,[5] Yang Y,[5] Herr K,[6] Hisamatsu T[7]

[1]Department of Medicine, Agaplesion Markus Hospital, Goethe University, Frankfurt, Germany; [2]Fukuoka University Faculty of Medicine, Fukuoka, Japan; [3]The Jikei University School of Medicine, Tokyo, Japan; [4]Johnson & Johnson, Tokyo, Japan; [5]Johnson & Johson, Shanghai, China; [6]Johnson & Johnson, Singapore; [7]Kyorin University School of Medicine, Tokyo, Japan

QUASAR Phase 3 induction study design (through Week 12)[1]

Participants
- ≥18 years of age
- Moderately to severely active UC*

Stable doses of oral corticosteroids (≤20 mg/day prednisone or equivalent) were permitted



Maintenance study
GUS IV induction responders (Week 12 or Week 24) entered the QUASAR maintenance study

***Post hoc* evaluation of symptomatic efficacy during the first 14 days of IV induction treatment in QUASAR[2]**
- LS mean changes from baseline in Mayo stool frequency and rectal bleeding subscores per daily PRO-2 data
- Symptomatic response (decrease in symptomatic Mayo score [sum of stool frequency and rectal bleeding subscores] by ≥30% and ≥1 point)
- Symptomatic remission (stool frequency subscore of 0 or 1; rectal bleeding subscore of 0)

Associations between early symptomatic efficacy and long-term outcomes in QUASAR were also explored[2]

Rates of symptomatic response and remission at Week 2 and Week 4, respectively, with GUS 400 mg SC (at Weeks 0, 4 and 8) induction treatment are from the Phase 3 ASTRO study[3]

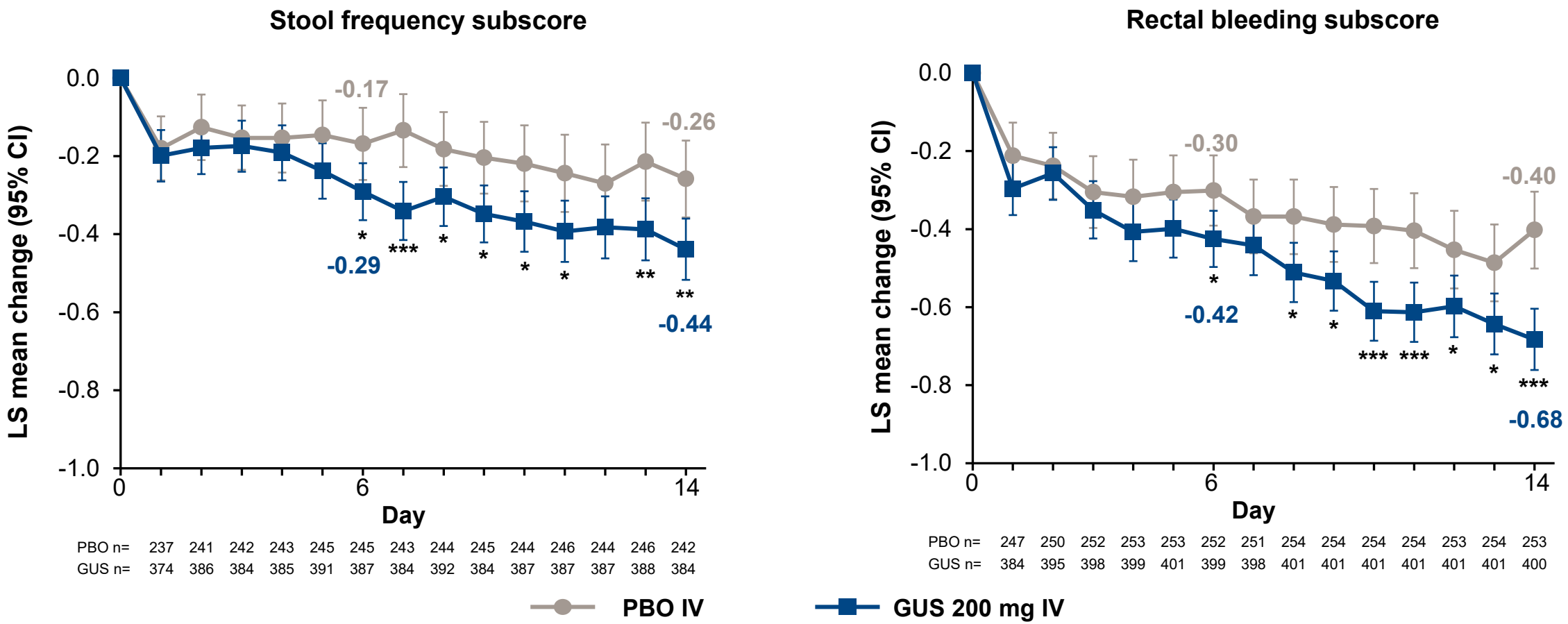
*Defined as a modified Mayo score of 5–9 with a Mayo rectal bleeding subscore ≥1 and a Mayo endoscopic subscore ≥2 based on central review.[2]
GUS, guselkumab; IL, interleukin; IV, intravenous; LS, least squares; MOA, mechanism of action; PBO, placebo; PRO-2, patient-reported outcome-2; R, randomisation; SC, subcutaneous; UC, ulcerative colitis.
1. Rubin DT, et al. *Lancet* 2025;405:33–49; 2. Dignass A, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. OP10; 3. Long M, et al. *Lancet Gastro Hep* 2026;S2468-1253(25)00322-X. Full prescribing information: www.swissmedicinfo-pro.ch.

Baseline characteristics at induction

	GUS 200 mg IV q4w (n=421)	PBO IV q4w (n=280)
Demographics		
Age, years	41.0 (13.9)	39.8 (13.4)
Male, %	57	58
Race, Asian/Black/White, %	21/1/72	22/1/73
Weight, kg	72.9 (16.7)	71.8 (17.0)
Disease characteristics		
UC disease duration, years	7.8 (7.7)	7.1 (6.5)
Modified Mayo score (0–9)	6.9 (1.1)	6.9 (1.1)
Modified Mayo score 7–9 (severe), %	65	64
Extensive UC, %	45	52
CRP, mg/L, median (IQR)	4.3 (1.5; 11.2)*	3.8 (1.6; 9.1)†
Faecal calprotectin, mg/kg, median (IQR)	1651 (647; 3479)‡	1606 (654; 3077)§
UC medication history, %		
Oral corticosteroid use at baseline	43	43
Immunosuppressant use at baseline	22	19
History of BIO/JAK-IR	49	49
No history of BIO/JAK-IR	51	51
Biologic and JAK inhibitor-naïve¶	95	95

Data shown are mean (SD) unless otherwise noted.
*n=416; †n=278; ‡n=370; §n=253; ¶Denominator is participants without a history of BIO/JAK-IR.
BIO/JAK-IR, inadequate response or intolerance to biologic and/or Janus kinase inhibitor; CRP, C-reactive protein; GUS, guselkumab; IL, interleukin; IV, intravenous; IQR, interquartile range; JAK, Janus kinase; MOA, mechanism of action; PBO, placebo; qXw, every X weeks; SD, standard deviation; UC, ulcerative colitis.
Dignass A, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. OP10. Full prescribing information: www.swissmedicinfo-pro.ch.

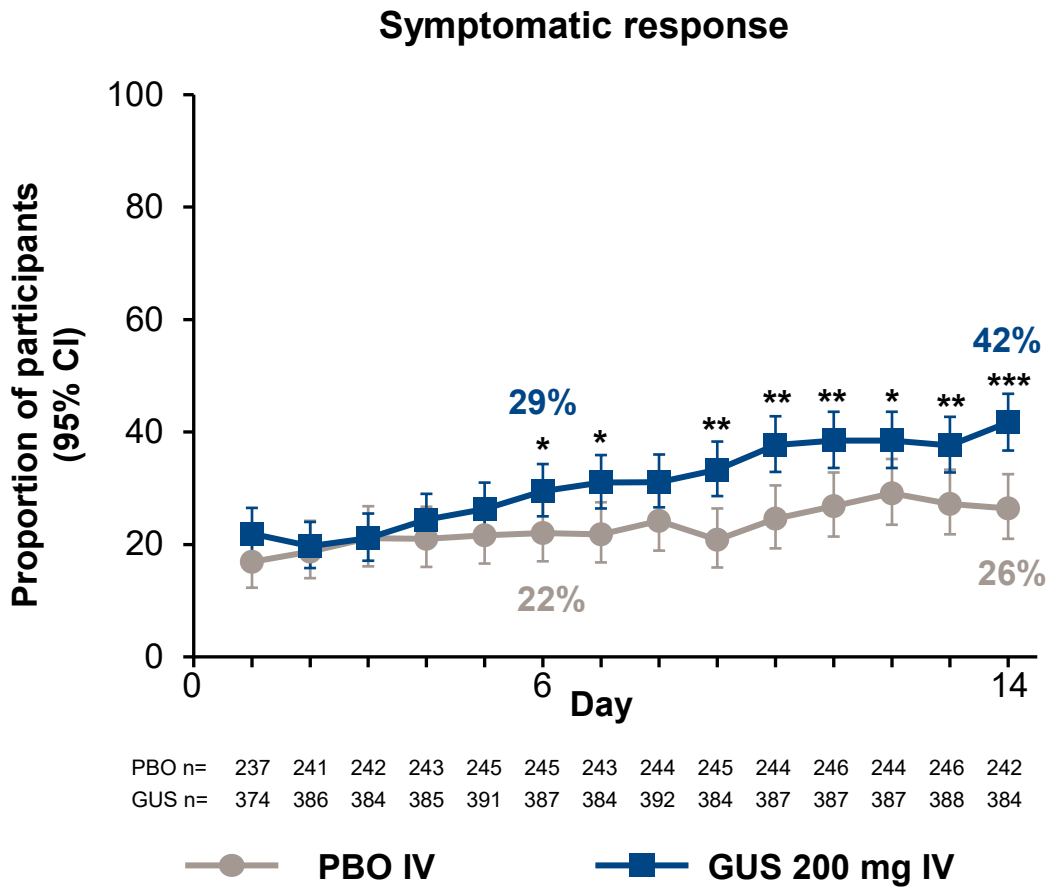
Stool frequency and rectal bleeding subscore changes from baseline



By Day 6, participants receiving GUS had greater improvements in stool frequency and rectal bleeding compared with PBO

Nominal *p<0.05; **p<0.01; ***p<0.001 vs PBO, based on MMRM.
MMRM based on observed case data was used to estimate changes in stool frequency and rectal bleeding subscores within the first 14 days. Explanatory variables of the MMRM include respective baseline scores, treatment groups, two randomisation factors (advanced therapy failure status [Yes/No] and concomitant use of corticosteroids at baseline [Yes/No]), visit (day), and an interaction term of visit with treatment group as fixed effects, and participant differences as a random effect. The within-participant covariance between visit (day) was estimated via an unstructured variance-covariance matrix.
CI, confidence interval; GUS, guselkumab; IL, interleukin; IV, intravenous; LS, least squares; MMRM, mixed-effect model for repeated measures; MOA, mechanism of action; PBO, placebo; UC, ulcerative colitis.
Dignass A, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. OP10. Full prescribing information: www.swissmedicinfo-pro.ch.

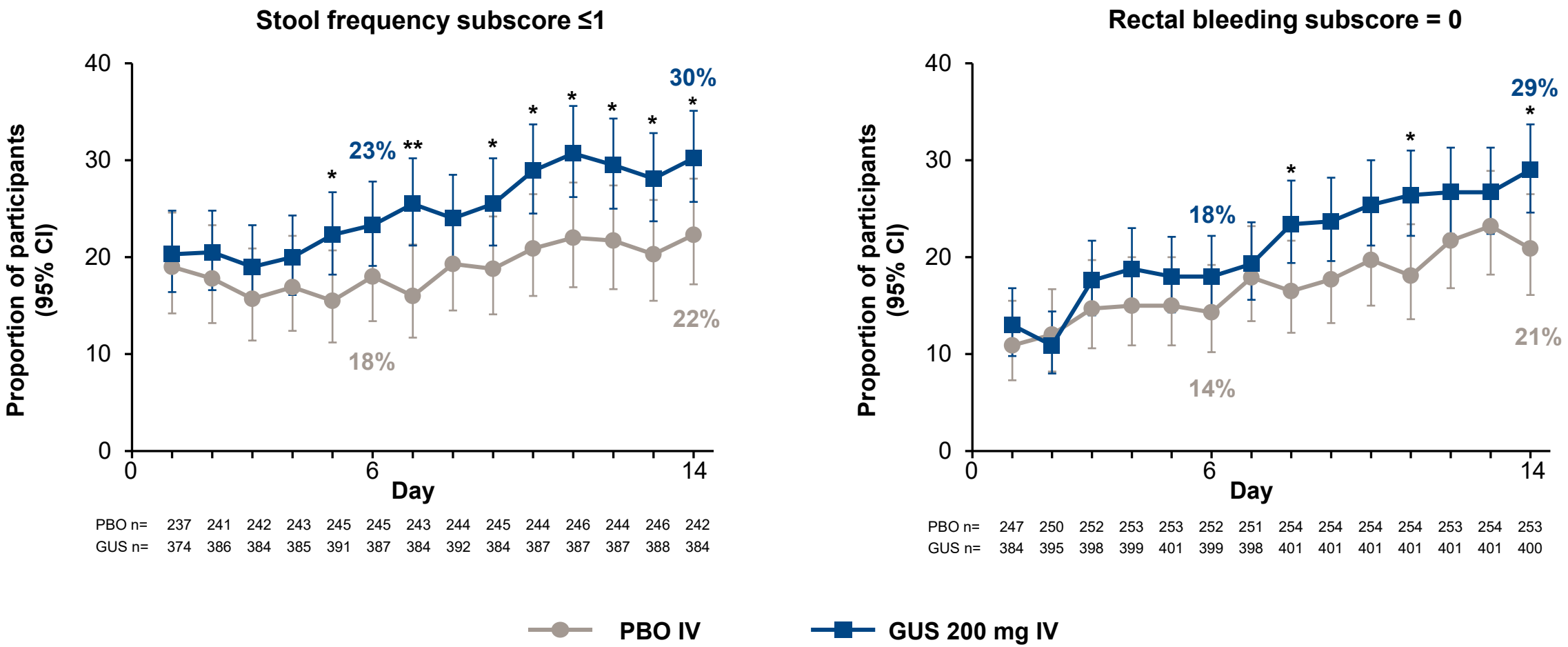
Symptomatic response



By Day 6, significantly greater proportions of participants receiving GUS vs PBO achieved a symptomatic response

Nominal *p<0.05; **p<0.01; ***p<0.001 vs PBO, based on CMH.
Observed case analysis. Nominal p-values based on the generalised CMH test, adjusting for two randomisation factors (advanced therapy failure status [Yes/No] and concomitant use of corticosteroids at baseline [Yes/No]).
Symptomatic response: reduction from induction baseline in symptomatic Mayo score (sum of stool frequency and rectal bleeding subscores) by ≥30% and ≥1 point.
CI, confidence interval; CMH, Cochran–Mantel–Haenszel; GUS, guselkumab; IL, interleukin; IV, intravenous; MOA, mechanism of action; PBO, placebo; UC, ulcerative colitis.
Dignass A, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. OP10. Full prescribing information: www.swissmedicinfo-pro.ch.

Stool frequency and rectal bleeding normalisation



Nominal *p<0.05; **p<0.01; vs PBO, based on CMH.
Observed case analysis. Nominal p-values based on the generalised CMH test, adjusting for two randomisation factors (advanced therapy failure status [Yes/No] and concomitant use of corticosteroids at baseline [Yes/No]).
CI, confidence interval; CMH, Cochran–Mantel–Haenszel; GUS, guselkumab; IL, interleukin; IV, intravenous; MOA, mechanism of action; PBO, placebo; UC, ulcerative colitis.
Dignass A, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. OP10. Full prescribing information: www.swissmedicinfo-pro.ch.

Early symptomatic improvements and clinical, endoscopic and QoL outcomes at Week 12 and Week 44

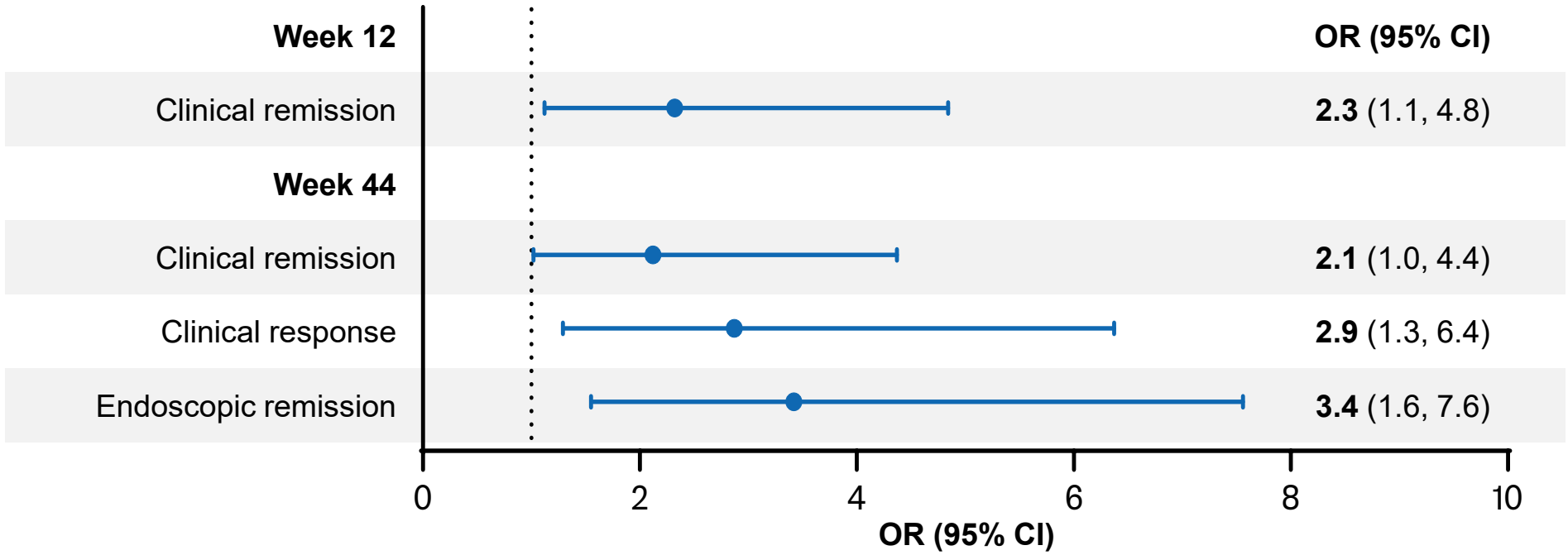
	PPV for Week 12 endpoint		PPV for Week 44 endpoint				
	Clinical response	Clinical remission	Clinical response	Clinical remission	Endoscopic response	Endoscopic remission	IBDQ remission
Symptomatic remission, %							
Within Day 1–14*	77	35	73	40	41	36	71
On Day 14	86	46	77	50	52	40	71
Symptomatic response, %							
Within Day 1–14*	67	26	72	41	42	31	62
On Day 14	74	31	69	42	44	32	59

Day 14 symptomatic response and remission predict Week 12 clinical response with high accuracy

Data are reported for participants in the GUS group only. **Symptomatic response:** reduction from induction baseline in symptomatic Mayo score (sum of stool frequency and rectal bleeding subscores) by ≥30% and ≥1 point.
Symptomatic remission: stool frequency subscore=0 or 1 and rectal bleeding subscore=0. PPV indicates the probability of achieving both symptom remission/response and the indicated Week 44 endpoint.
*Includes participants with ≥1 symptomatic remission/response within the first 14 days.
GUS, guselkumab; IBDQ, Inflammatory Bowel Disease Questionnaire; IL, interleukin; MOA, mechanism of action; PPV, positive predictive value; QoL, quality of life; UC, ulcerative colitis.
Dignass A, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. OP10. Full prescribing information: www.swissmedicinfo-pro.ch.

Exploratory analysis of stool frequency normalisation at Day 3 and clinical and endoscopic outcomes at Week 12 and Week 44

ORs of achieving clinical and endoscopic endpoints at Week 12 or Week 44 by Day 3 stool frequency subscore ≤1



Day 3 normalisation of stool frequency (stool frequency subscore ≤1) is associated with achieving long-term outcomes

Exploratory sensitivity analysis of participants in the GUS treatment group. Multivariable logistic regression analysis was used to assess potential associations. Models were adjusted for baseline stool frequency/rectal bleeding subscores and randomisation factors (advanced therapy failure status [Yes/No] and concomitant use of corticosteroids at baseline [Yes/No]). For participants with a non-integer number of stools in 24 hours when in remission or prior to UC, the daily number of stools was rounded up to the nearest whole number. ORs were calculated using the reference group with a stool frequency subscore >1.
CI, confidence interval; GUS, guselkumab; IL, interleukin; MOA, mechanism of action; OR, odds ratio; UC, ulcerative colitis.
Dignass A, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. OP10. Full prescribing information: www.swissmedicinfo-pro.ch.

Symptomatic outcomes at Week 2 following one dose of IV or SC GUS induction[1]

Proportion of participants at Week 2 (NRI)	IV induction (QUASAR)[2*]		SC induction (ASTRO)[3†]	
	GUS 200 mg IV (n=421)	PBO IV (n=280)	GUS 400 mg SC (n=279)	PBO SC (n=139)
Symptomatic response, %	34	24	36	26
Symptomatic remission, %	12	9	12	8

At Week 2, rates of symptomatic response and remission were similar with IV (QUASAR) and SC (ASTRO) GUS induction

Symptomatic response: reduction from induction baseline in symptomatic Mayo score (sum of stool frequency and rectal bleeding subscores) by ≥30% and ≥1 point. **Symptomatic remission:** stool frequency subscore=0 or 1 and rectal bleeding subscore=0.

*Week 2 symptomatic response endpoint was not multiplicity controlled; †Week 2 symptomatic response and remission endpoints are *post hoc*; values were based on the most recent 3 consecutive days within the 7 days prior.

GUS, guselkumab; IL, interleukin; IV, intravenous; MOA, mechanism of action; NRI, non-responder imputation; IV, intravenous; PBO, placebo; SC, subcutaneous; UC, ulcerative colitis.

1. Dignass A, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. OP10; 2. Lichtenstein GR, et al. Presented at ACG, Vancouver, Canada, 20–25 October 2023. Abstract 34; 3. Long M, et al. *Lancet Gastro Hep* 2026;S2468–1253(25)00322-X. Full prescribing information: www.swissmedicinfo-pro.ch.

Conclusions

- GUS shows very early symptomatic improvement in participants with moderately to severely active UC
- This early improvement is associated with long-term outcomes
- These findings underscore the clinical relevance of early symptom control and align with STRIDE-II short-term treatment targets
- Proportions of participants with symptomatic improvements continued to increase after Week 2 with continued GUS induction therapy

GUS, guselkumab; IL, interleukin; MOA, mechanism of action; UC, ulcerative colitis.

Dignass A, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. OP10. Full prescribing information: www.swissmedicinfo-pro.ch.

P0805

Predictors of endoscopic remission at 1 year in patients with ulcerative colitis treated with guselkumab: *Post-hoc* analyses of the QUASAR trial

Rubin DT,[1] Fumery M,[2] Armuzzi A,[3,4] Ferrante M,[5] Baker T,[6] Alvarez Y,[6] Bravatà I,[7] Nazar,[8] Denderen J Van,[9] McCaffrey V,[10] Atreya R[11]

[1]Inflammatory Bowel Disease Center, University of Chicago Medicine, Chicago, IL, USA; [2]Gastroenterology Unit, Amiens University Hospital, Amiens, France; [3]IBD Center, IRCCS Humanitas Research Hospital, Rozzano, Milan, Italy; [4]Department of Biomedical Sciences, Humanitas University, Pieve Emanuele, Milan, Italy; [5]Department of Gastroenterology and Hepatology, University Hospitals Leuven, Leuven, Belgium; [6]Johnson & Johnson, Spring House, PA, USA; [7]Johnson & Johnson, Milan, Italy; [8]Johnson & Johnson, Warsaw, Poland; [9]Johnson & Johnson, Breda, the Netherlands; [10]Johnson & Johnson, Buckinghamshire, UK; [11]Medical Department 1, University Hospital Erlangen, Friedrich-Alexander-University of Erlangen-Nuremberg, Erlangen, Germany.

Induction baseline demographics and disease characteristics

Induction baseline characteristics of GUS responders re-randomised to SC GUS q8w or q4w in the QUASAR maintenance study	Patients with ER at maintenance Week 44 (n=129)	Patients without ER at maintenance Week 44 (n=249)
Demographics		
Age, years	38.8 (13.9)	41.3 (13.8)
Female sex	57%	41%
Race, Asian/Black/White	20%/1%/77%	22%/1%/70%
Disease characteristics		
Disease duration, years	7.8 (9.2)	8.2 (8.0)
UC type, %		
Extensive	53	38
Limited to left side of colon	47	62
Endoscopic Mayo Score, %		
Moderate (endoscopy subscore=2)	37	33
Severe (endoscopy subscore=3)	63	67
CRP, %		
≤3 mg/L	37*	48†
Faecal calprotectin, %		
≤250 µg/g	15‡	10§
Medication use		
Prior ADT failure, %	35	47
Concomitant use, %		
Oral corticosteroids	42	39
Immunomodulators	24	21
Oral aminosalicylates	84	74

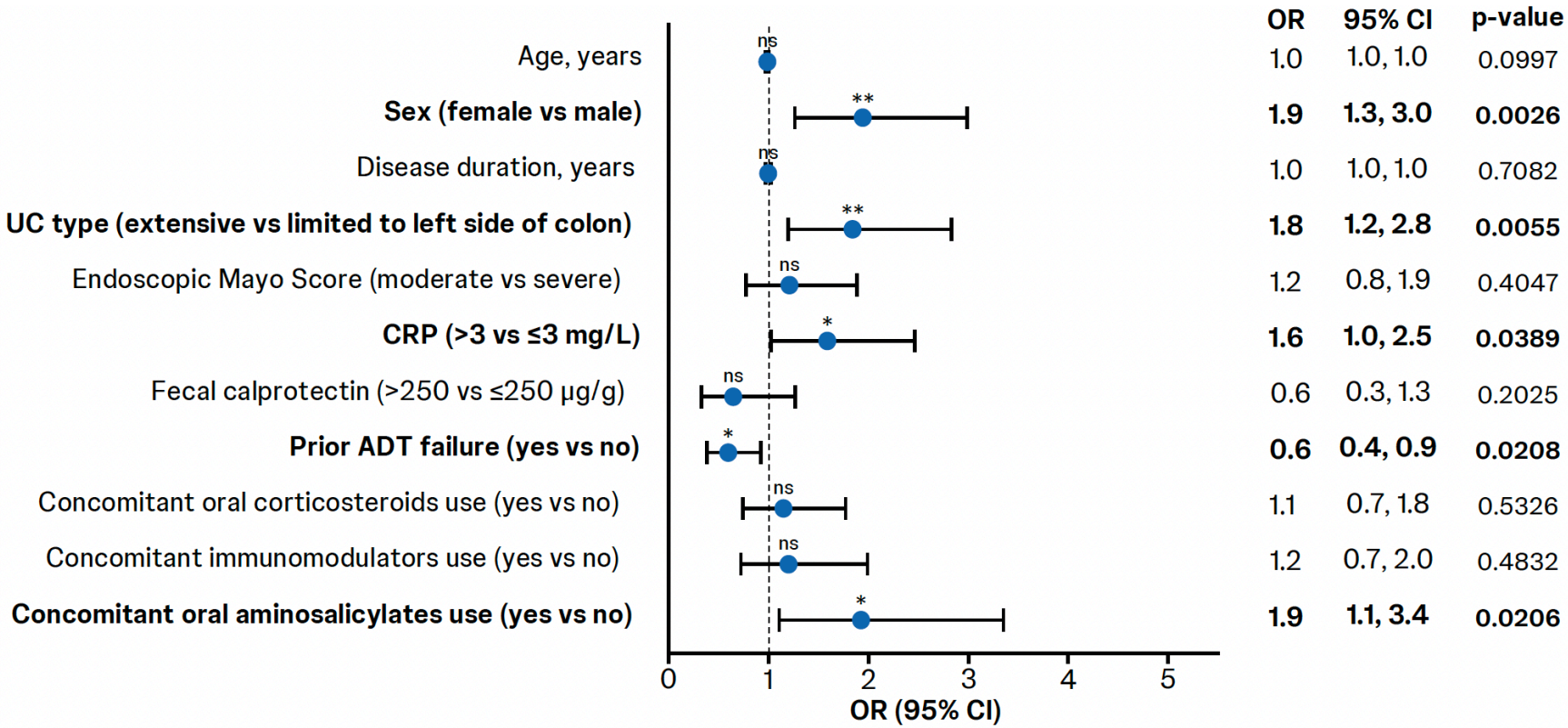
Participants achieving ER at maintenance Week 44 of GUS treatment were slightly younger, more often female and more likely to have extensive UC and concomitant oral aminosalicylate use, with higher CRP at induction baseline

Data are % for categorical variables and mean (SD) for continuous variables. *n=128; †n=244; ‡n=111; §n=220.
ADT, advanced drug therapy; CRP, C-reactive protein; ER, endoscopic remission; GUS, guselkumab; IL, interleukin; MOA, mechanism of action; qXw, every X weeks; SC, subcutaneous; SD, standard deviation; UC, ulcerative colitis.
Rubin D, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. Poster 0805. Full prescribing information: www.swissmedicinfo-pro.ch.

Induction baseline predictors of Week 44 endoscopic remission in unadjusted models

Unadjusted models:

Induction baseline predictors of Week 44 endoscopic remission†

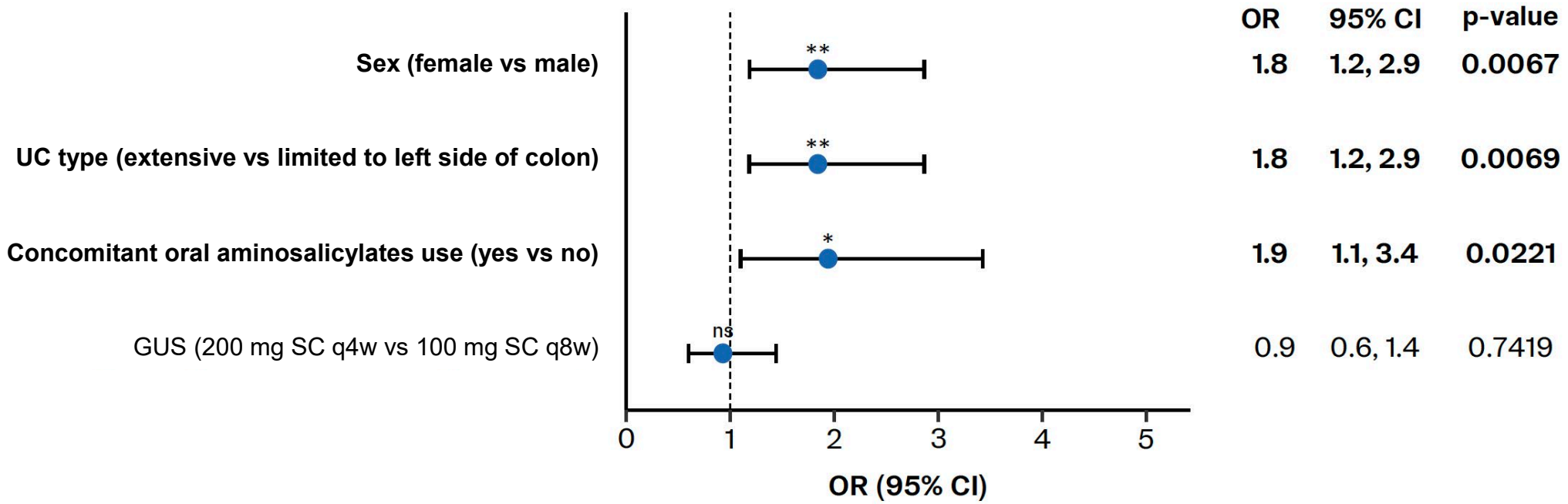


Induction baseline predictors of endoscopic remission at maintenance Week 44 of GUS treatment were female sex, extensive UC, elevated CRP levels, concomitant oral aminosalicylate use and no prior ADT failure

Nominal *p<0.05, **p<0.01. †Univariate models were adjusted for GUS dosing regimen in the maintenance study (GUS 200 mg SC q4w vs GUS 100 mg SC q8w).
ADT, advanced drug therapy; CI, confidence interval; CRP, C-reactive protein; ER, endoscopic remission; GUS, guselkumab; IL, interleukin; MOA, mechanism of action; ns, not significant; OR, odds ratio; qXw, every X weeks; SC, subcutaneous; UC, ulcerative colitis.
Rubin D, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. Poster 0805. Full prescribing information: www.swissmedicinfo-pro.ch.

Induction baseline predictors of Week 44 endoscopic remission in adjusted models

Adjusted models:
Induction baseline predictors of Week 44 endoscopic remission†



Female sex, extensive UC and concomitant oral aminosalicylate use at induction baseline were independent predictors of endoscopic remission at maintenance Week 44 of GUS treatment, irrespective of dosing regimen
GUS regimen was not a baseline predictor of ER achievement at maintenance Week 44

Nominal *p<0.05, **p<0.01. †Backward selection multivariate models included induction baseline variables with a significance level of p<0.10 in univariate models and GUS dosing regimen.
CI, confidence interval; ER, endoscopic remission; GUS, guselkumab; IL, interleukin; MOA, mechanism of action; ns, not significant; OR, odds ratio; qXw, every X weeks; SC, subcutaneous; UC, ulcerative colitis.
Rubin D, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. Poster 0805. Full prescribing information: www.swissmedicinfo-pro.ch.

Maintenance baseline characteristics of GUS responders re-randomised to SC GUS q8w or q4w in the QUASAR maintenance study

	Participants with ER at maintenance Week 44 (n=129)	Participants without ER at maintenance Week 44 (n=249)
Disease characteristics, %		
CRP normalisation*	86†	75‡
Faecal calprotectin normalisation§	78¶	66**
Endoscopic healing††	58	32
GUS dosing regimen, %		
200 mg SC q4w	50	51
100 mg SC q8w	50	49

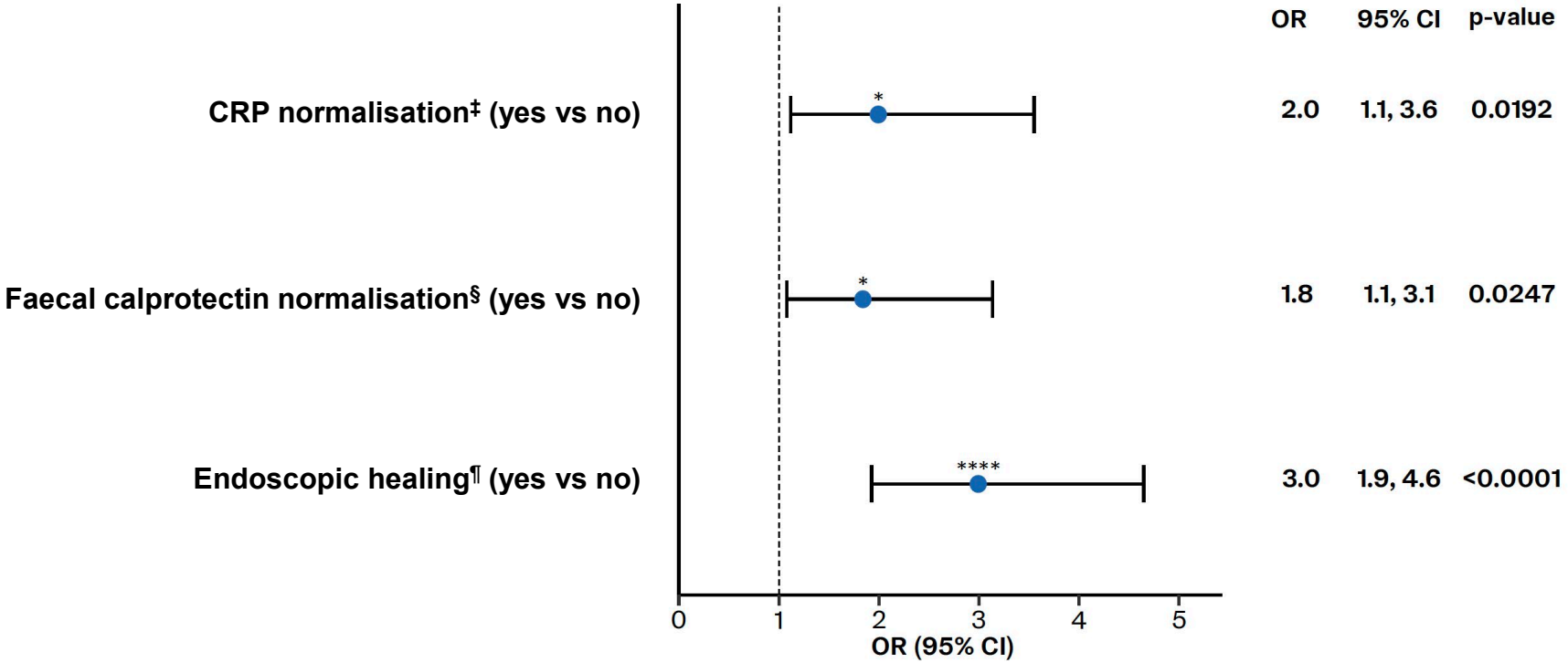
Patients achieving endoscopic remission at maintenance Week 44 of GUS treatment had lower UC disease burden at maintenance baseline
Rates of endoscopic remission achievement at maintenance Week 44 did not differ across GUS regimens

*Defined as ≤3 mg/L or 50% reduction from induction baseline; †n=128; ‡n=244; §Defined as ≤250 µg/g or 50% reduction from induction baseline; ¶n=110; **n=218; ††Defined as Endoscopic Mayo subscore of 0 or 1 and no friability.
CRP, C-reactive protein; ER, endoscopic remission; GUS, guselkumab; IL, interleukin; MOA, mechanism of action; qXw, every X weeks; SC, subcutaneous; UC, ulcerative colitis.
Rubin D, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. Poster 0805. Full prescribing information: www.swissmedicinfo-pro.ch.

Maintenance baseline predictors of Week 44 endoscopic remission in unadjusted models

Unadjusted models:

Maintenance baseline predictors of Week 44 endoscopic remission†

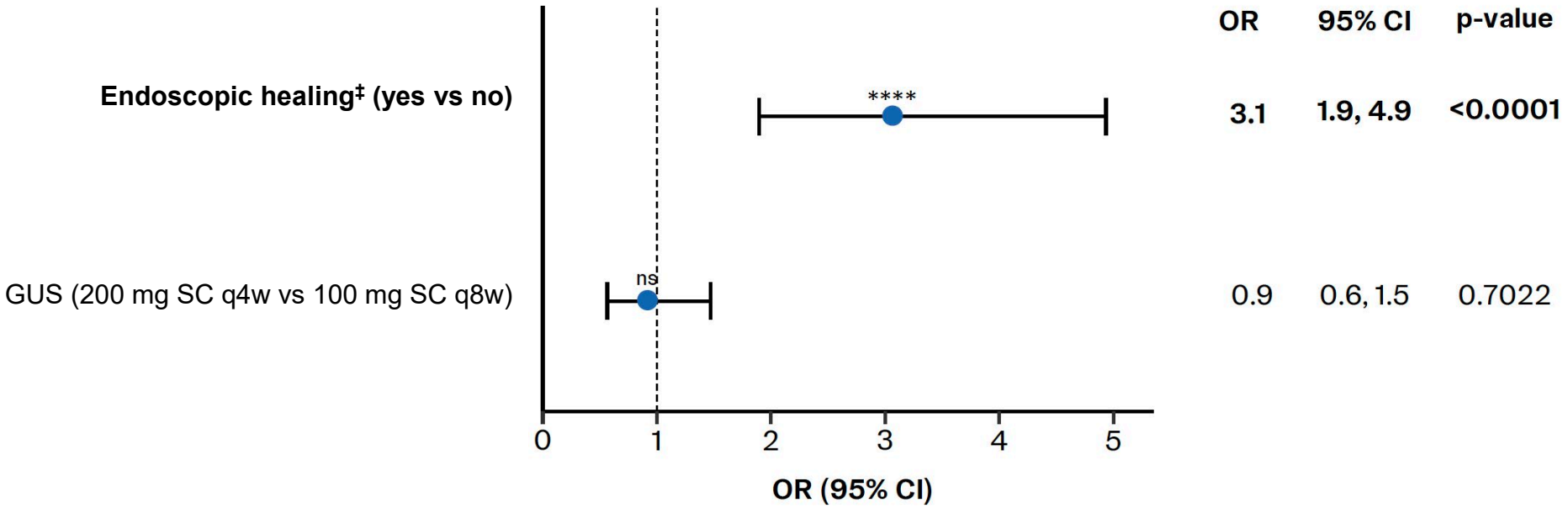


Maintenance baseline predictors of endoscopic remission at maintenance Week 44 of GUS treatment were CRP normalisation, faecal calprotectin normalisation and endoscopic healing

Nominal *p<0.05, ****p<0.0001. †Univariate models were adjusted for GUS dosing regimen in the maintenance study (GUS 200 mg SC q4w vs GUS 100 mg SC q8w); ‡Defined as ≤3 mg/L or 50% reduction from induction baseline; §Defined as ≤250 µg/g or 50% reduction from induction baseline; ¶Defined as Endoscopic Mayo subscore of 0 or 1 and no friability.
CI, confidence interval; CRP, C-reactive protein; GUS, guselkumab; IL, interleukin; MOA, mechanism of action; OR, odds ratio; qXw, every X weeks; SC, subcutaneous; UC, ulcerative colitis.
Rubin D, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. Poster 0805. Full prescribing information: www.swissmedicinfo-pro.ch.

Maintenance baseline predictors of Week 44 endoscopic remission in adjusted models

Adjusted models:
Maintenance baseline predictors of Week 44 endoscopic remission†



Achievement of endoscopic healing at maintenance baseline was an independent predictor of endoscopic remission at maintenance Week 44 of GUS treatment, irrespective of dosing regimen
GUS regimen was not a baseline predictor of ER achievement at maintenance Week 44

Nominal ****p<0.0001. †Backward selection multivariate models included maintenance baseline variables with a significance level of p<0.10 in univariate models and GUS dosing regimen; ‡Defined as Endoscopic Mayo subscore of 0 or 1 and no friability.
CI, confidence interval; ER, endoscopic remission; GUS, guselkumab; IL, interleukin; MOA, mechanism of action; ns, not significant; OR, odds ratio; qXw, every X weeks; SC, subcutaneous; UC, ulcerative colitis.
Rubin D, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. Poster 0805. Full prescribing information: www.swissmedicinfo-pro.ch.

Conclusions

Among patients with moderately to severely active UC who responded to GUS induction and were re-randomised to GUS in the QUASAR maintenance study:

- Female sex, extensive UC and oral aminosalicylate use at induction baseline were independently associated with ER at 1 year, irrespective of GUS dosing regimen
- Endoscopic healing at maintenance baseline (i.e., after induction) was strongly associated with a higher likelihood of achieving ER at 1 year, regardless of GUS dosing regimen

Collectively, these findings may provide insight into the long-term endoscopic benefits of GUS treatment in UC

ER, endoscopic remission; GUS, guselkumab; IL, interleukin; MOA, mechanism of action; UC, ulcerative colitis.
Rubin D, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. Poster 0805. Full prescribing information: www.swissmedicinfo-pro.ch.

P0980

Association of endoscopic, histologic, and composite outcomes with long-term guselkumab efficacy in ulcerative colitis: 2-year results from the QUASAR long-term extension

Magro F,[1] Rubin DT,[2] Lichtenstein GR,[3] Alvarez Y,[4] Baker T,[4] Miao Y,[4] Peyrin-Biroulet L[5]

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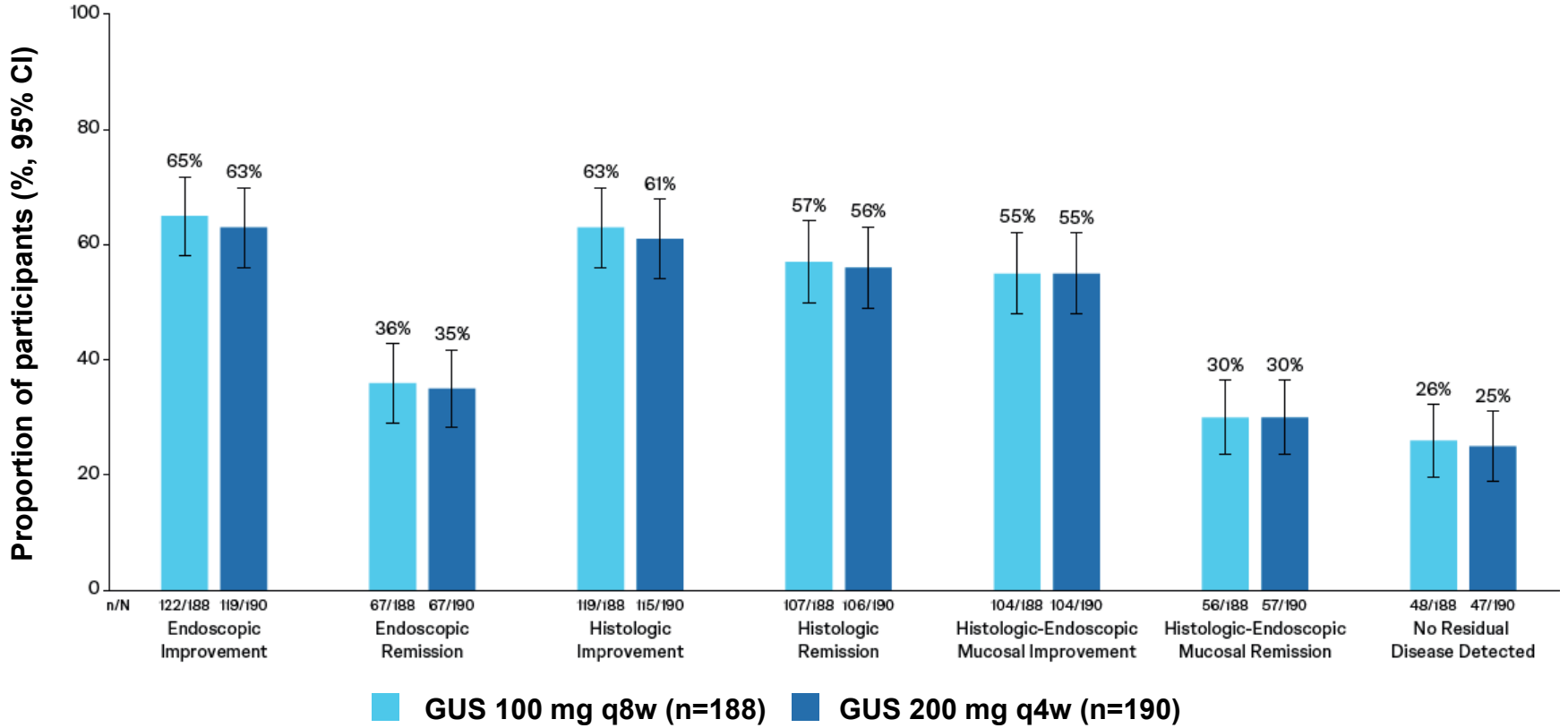
Immunology

Aim: to explore whether achievement of select efficacy outcomes at QUASAR maintenance baseline (Week M-0) or at maintenance Week 44 was associated with achievement of CS-free clinical remission at Week 92 in the QUASAR LTE

Achievement of endoscopic, histologic and composite outcomes at LTE Week 92 among participants randomised to GUS (NRI analysis)



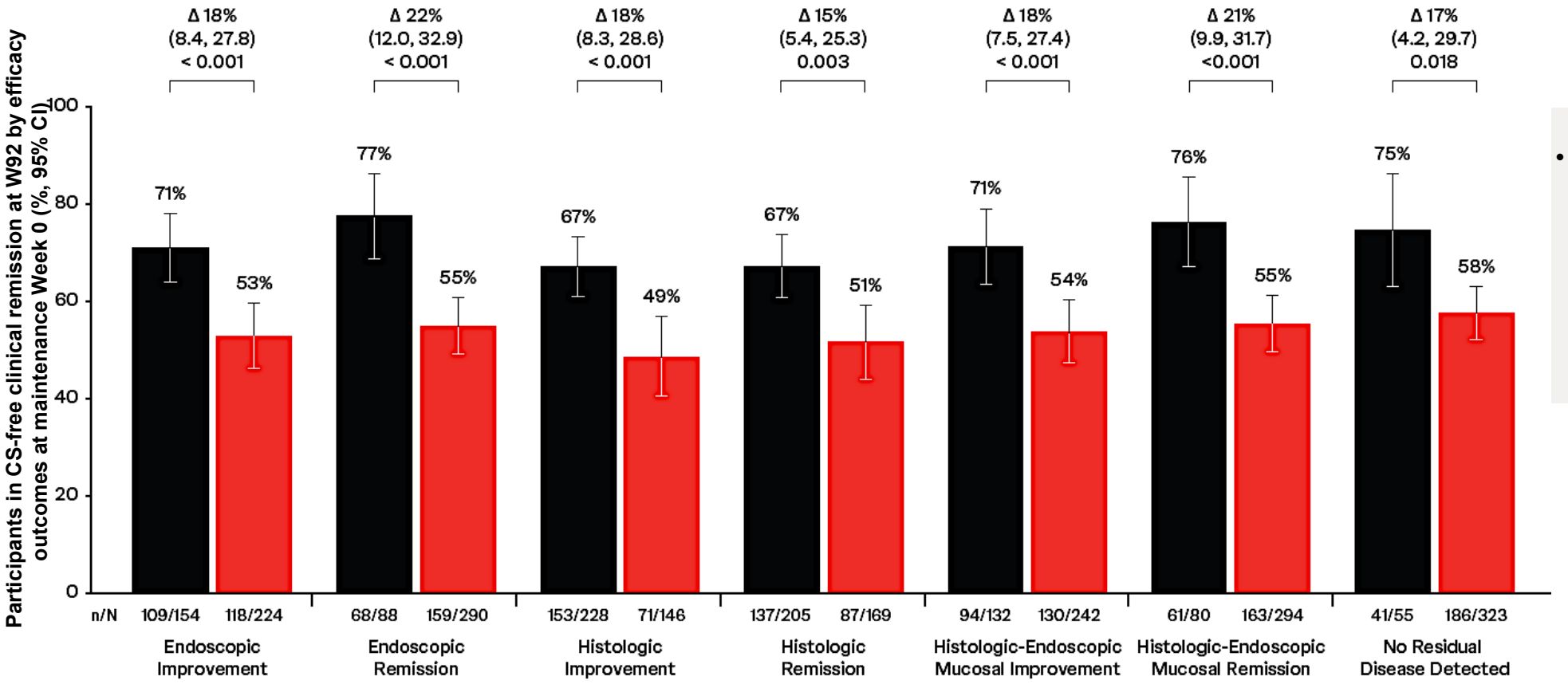
Includes participants with modified Mayo score 5–9 at induction baseline who were in clinical response to GUS IV induction and randomised to receive GUS 100 mg SC q8w or GUS 200 mg SC q4w at maintenance study entry.

CS-free clinical remission: Mayo stool frequency subscore of 0 or 1 and not increased from induction baseline, Mayo rectal bleeding subscore of 0 and Mayo endoscopic subscore of 0 or 1 without the use of corticosteroids for ≥8 weeks prior to Week 92 of the LTE. **Endoscopic improvement:** Mayo endoscopic subscore of 0 or 1. **Endoscopic remission:** Mayo endoscopic subscore of 0. **Histologic improvement:** neutrophil infiltration in <5% of crypts, no crypt destruction and no erosions, ulcerations or granulation tissue according to the Geboes grading system. **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. **Histologic-endoscopic mucosal improvement:** combination of histologic improvement and endoscopic improvement. **Histologic-endoscopic mucosal remission:** combination of histologic remission and endoscopic remission. **No residual disease detected:** combination of symptomatic remission (stool frequency subscore of 0 or 1 and not increased from induction baseline and a rectal bleeding score of 0), histologic remission, endoscopic remission and faecal calprotectin ≤250 mg/kg.

CI, confidence interval; CS, corticosteroid; GUS, guselkumab; IL, interleukin; IV, intravenous; LTE, long-term extension; MOA, mechanism of action; M, maintenance; NRI, non-responder imputation; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous; UC, ulcerative colitis.

Magro F, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. Poster P0980. Full prescribing information: www.swissmedicinfo-pro.ch.

Among GUS-randomised participants, regardless of whether they entered the LTE, achievement of efficacy outcomes at maintenance Week 0 was associated with greater achievement of CS-free clinical remission at LTE Week 92*



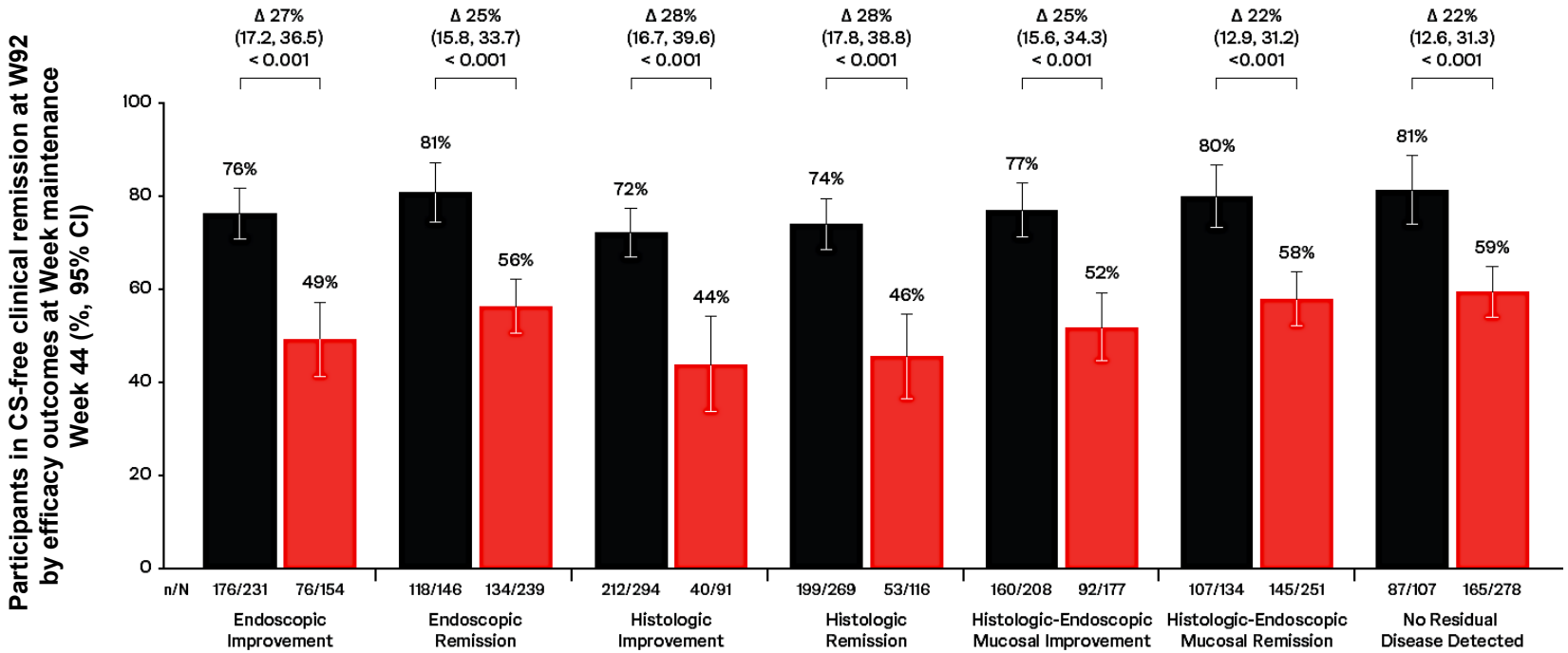
- Across outcomes, 67–77% of participants who achieved an outcome at maintenance Week 0 achieved CS-free clinical remission at LTE Week 92 vs 49–58% of participants who did not (15–22% difference; all nominal p<0.05)

*Based on NRI analyses. All p-values are nominal. Includes participants with modified Mayo score 5–9 at induction baseline who were in clinical response to GUS IV induction and randomised to receive GUS 100 mg SC q8w or GUS 200 mg SC q4w at maintenance study entry regardless of whether they entered the LTE. Participants who did not enter the LTE were considered CS-free clinical remission non-responders at LTE Week 92. **CS-free clinical remission:** Mayo stool frequency subscore of 0 or 1 and not increased from induction baseline, Mayo rectal bleeding subscore of 0 and Mayo endoscopic subscore of 0 or 1 without the use of corticosteroids for ≥8 weeks prior to Week 92 of the LTE. **Endoscopic improvement:** Mayo endoscopic subscore of 0 or 1. **Endoscopic remission:** Mayo endoscopic subscore of 0. **Histologic improvement:** neutrophil infiltration in <5% of crypts, no crypt destruction and no erosions, ulcerations or granulation tissue according to the Geboes grading system. **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. **Histologic-endoscopic mucosal improvement:** combination of histologic improvement and endoscopic improvement. **Histologic-endoscopic mucosal remission:** combination of histologic remission and endoscopic remission. **No residual disease detected:** combination of symptomatic remission (stool frequency subscore of 0 or 1 and not increased from induction baseline and a rectal bleeding score of 0), histologic remission, endoscopic remission and faecal calprotectin ≤250 mg/kg.

CI, confidence interval; CS, corticosteroid; GUS, guselkumab; IL, interleukin; IV, intravenous; LTE, long-term extension; MOA, mechanism of action; NRI, non-responder imputation; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; UC, ulcerative colitis.

Magro F, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. Poster P0980. Full prescribing information: www.swissmedicinfo-pro.ch.

Among GUS-treated participants who entered the LTE, achievement of efficacy outcomes at maintenance Week 44 was associated with greater achievement of CS-free clinical remission at LTE Week 92*



- Across outcomes, 72–81% of participants who achieved an outcome at maintenance Week 44 achieved CS-free clinical remission at LTE Week 92 versus 44–59% of participants who did not (22–28% difference; all nominal p<0.001)

Achieved at maintenance Week 0

Not achieved at maintenance Week 0

Conclusions

- In QUASAR, achievement of endoscopic, histologic and composite outcomes following induction and 1 year of maintenance therapy was associated with achievement of CS-free clinical remission at LTE Week 92
- Composite outcomes did not appear to be more strongly associated with CS-free clinical remission than endoscopic or histologic outcomes alone
- These findings support endoscopic and histologic outcomes as sensible shorter-term targets associated with longer-term clinical outcomes in moderately to severely active UC

*Based on NRI analyses. All p-values are nominal. Includes participants with modified Mayo score 5–9 at induction baseline who 1) were in clinical response to GUS IV induction and randomised to receive GUS 100 mg SC q8w or GUS 200 mg SC q4w at maintenance study entry who entered the LTE; and 2) participants in clinical response to GUS IV induction randomised to PBO at maintenance study entry who experienced a dose adjustment to GUS 200 mg SC q4w during the maintenance study and who entered the LTE.

CS-free clinical remission: Mayo stool frequency subscore of 0 or 1 and not increased from induction baseline, Mayo rectal bleeding subscore of 0 and Mayo endoscopic subscore of 0 or 1 without the use of corticosteroids for ≥8 weeks prior to Week 92 of the LTE. **Endoscopic improvement:** Mayo endoscopic subscore of 0 or 1. **Endoscopic remission:** Mayo endoscopic subscore of 0. **Histologic improvement:** neutrophil infiltration in <5% of crypts, no crypt destruction and no erosions, ulcerations or granulation tissue according to the Geboes grading system. **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. **Histologic-endoscopic mucosal improvement:** combination of histologic improvement and endoscopic improvement. **Histologic-endoscopic mucosal remission:** combination of histologic remission and endoscopic remission. **No residual disease detected:** combination of symptomatic remission (stool frequency subscore of 0 or 1 and not increased from induction baseline and a rectal bleeding score of 0), histologic remission, endoscopic remission and faecal calprotectin ≤250 mg/kg.

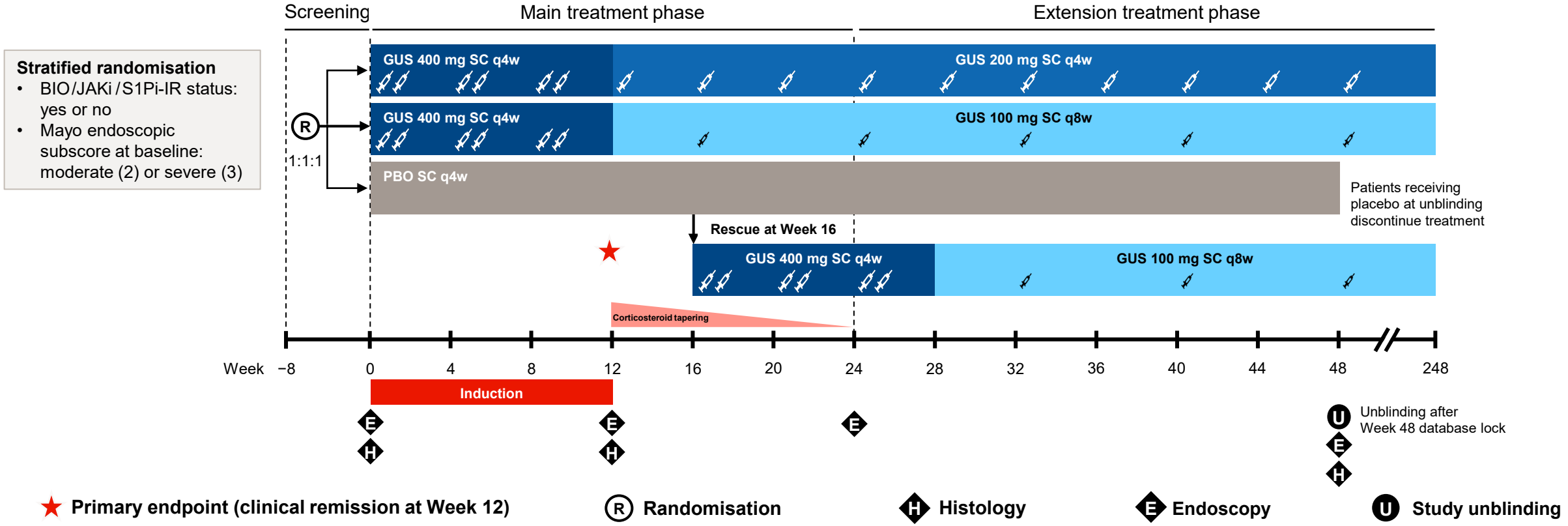
CI, confidence interval; CS, corticosteroid; GUS, guselkumab; IL, interleukin; IV, intravenous; LTE, long-term extension; MOA, mechanism of action; NRI, non-responder imputation; PBO, placebo; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous; UC, ulcerative colitis.

Magro F, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. Poster P0980. Full prescribing information: www.swissmedicinfo-pro.ch.

ASTRO

Immunology

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



Key eligibility criteria

- Baseline (Week 0) modified Mayo score of 5–9, inclusive
- Baseline Mayo rectal bleeding subscore ≥1, Mayo endoscopic subscore ≥2 (centrally reviewed)
- IR to TNFα blocker, vedolizumab, JAK inhibitor or S1Pi (BIO-/JAKi-/S1Pi-IR) **OR** naïve to BIO/JAKi/S1Pi (or exposed to BIO/JAKi/S1Pi without IR) and IR to corticosteroids, 6-MP or AZA

6-MP, 6-mercaptopurine; AZA, azathioprine; BIO, biologic; GUS, guselkumab; IL, interleukin; IR, inadequate response; JAKi, Janus kinase inhibitor; MOA, mechanism of action; PBO, placebo; qXw, every X weeks; R, randomised; RDBPC, randomised, double-blind, placebo-controlled; S1Pi, sphingosine-1-phosphate inhibitor; SC, subcutaneous; TNFα, tumour necrosis factor alpha; UC, ulcerative colitis.

Danese S, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. DOP105. Full prescribing information: www.swissmedicinfo-pro.ch.

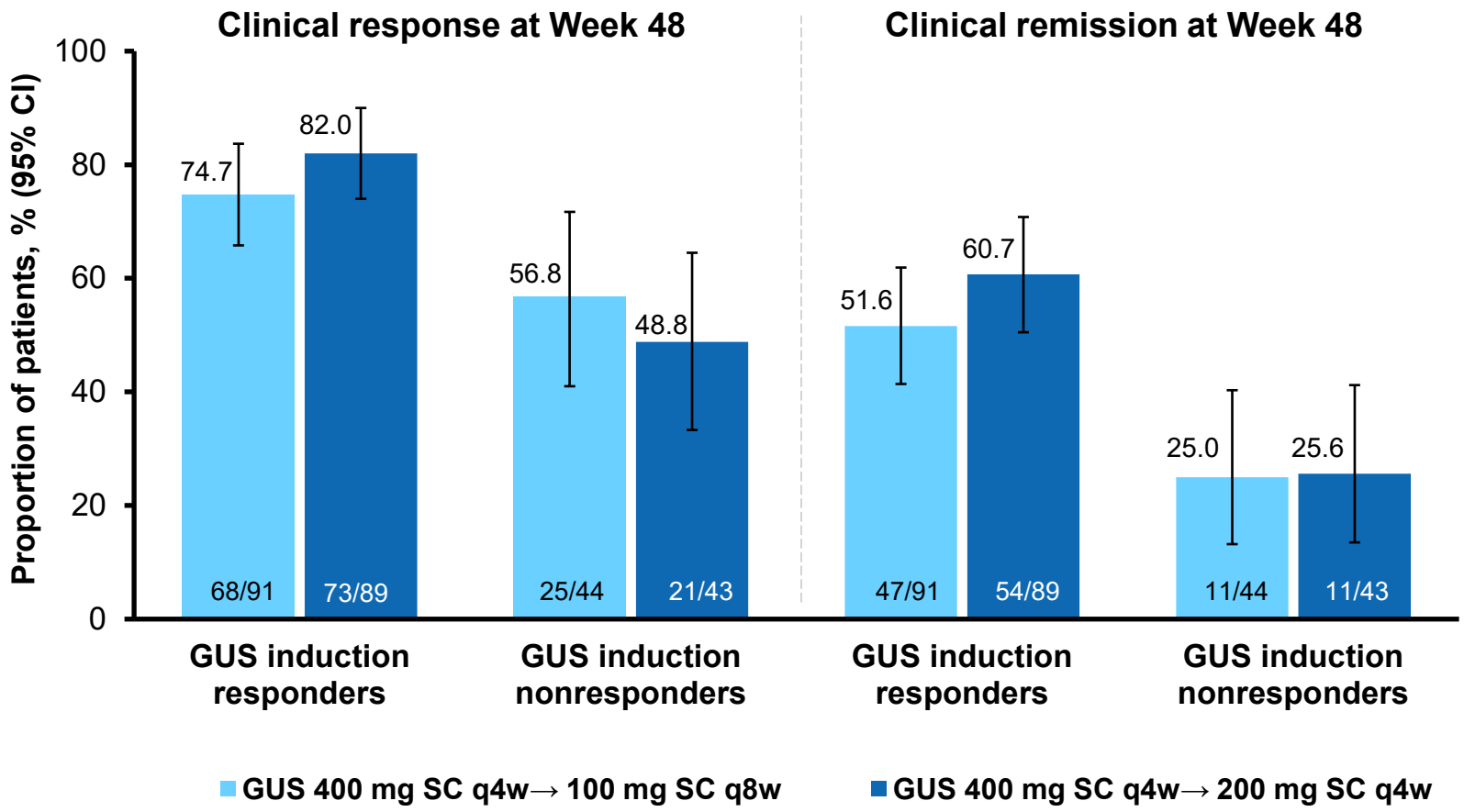
DOP105

Efficacy of subcutaneous guselkumab in moderately to severely active ulcerative colitis by induction Week 12 clinical response status: Week 48 results from the Phase 3 ASTRO study

Danese S,[1] Long M,[2] Peyrin-Biroulet L,[3] Hisamatsu T,[4] Baker T,[5] Alvarez Y,[5] Jiang L,[6] Zhang H,[6] Rubin DT,[7] Allegretti JR[8]

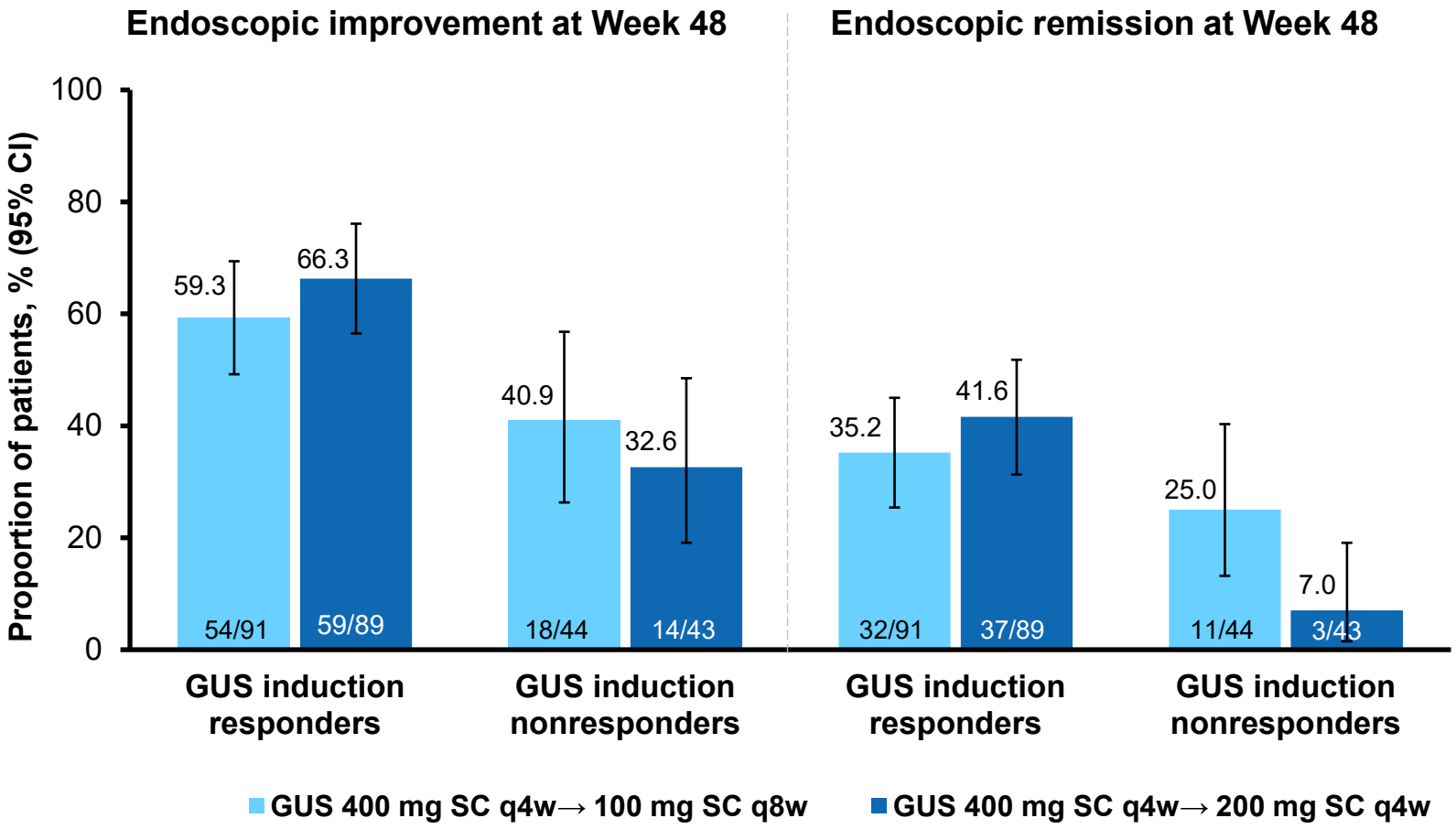
[1]San Raffaele and University Vita-Salute San Raffaele, Gastroenterology and Endoscopy, Milan, Italy; [2]University of North Carolina at Chapel Hill, Department of Gastroenterology, Chapel Hill, NC, USA; [3]Hopitaux De Brabois, Department of Gastroenterology and Inserm U954, France Vandœuvre-lès-Nancy, France; [4]Kyorin University School of Medicine, Department of Gastroenterology and Hepatology, Tokyo, Japan; [5]Johnson & Johnson, Immunology, Spring House, PA, USA; [6]Johnson & Johnson, Statistics and Decision Sciences, Spring House, PA, USA; [7]University of Chicago Medicine Inflammatory Bowel Disease Center, Gastroenterology-Hepatology & Nutrition, Chicago, IL, USA; [8]Brigham and Women's Hospital, Division of Gastroenterology-Hepatology and Endoscopy, Harvard Medical School, Boston, MA, USA

Week 48 clinical outcomes by induction response status at Week 12



Clinical response: a decrease in mMayo score from baseline by ≥30% and ≥2 points, with either a ≥1-point decrease from baseline in the rectal bleeding subscore or achieving a rectal bleeding subscore of 0 or 1.
Clinical remission: a Mayo stool frequency subscore of 0 or 1 and no increase from baseline, a Mayo rectal bleeding subscore of 0 and a Mayo endoscopy subscore of 0 or 1 with no friability.
CI, confidence interval; GUS, guselkumab; IL, interleukin; mMayo, modified Mayo; MOA, mechanism of action; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous; UC, ulcerative colitis.
Danese S, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. DOP105. Full prescribing information: www.swissmedicinfo-pro.ch.

Week 48 endoscopic outcomes by induction response status at Week 12



Endoscopic improvement: a Mayo endoscopy subscore of 0 or 1 with no friability. **Endoscopic remission:** a Mayo endoscopy subscore of 0.
CI, confidence interval; GUS, guselkumab; IL, interleukin; MOA, mechanism of action; qXw, every X weeks; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous; UC, ulcerative colitis.
Danese S, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. DOP105. Full prescribing information: www.swissmedicinfo-pro.ch.

Baseline characteristics and medication history of GUS induction Week 12 responders and non-responders

	GUS induction Week 12 responders (n=180)	GUS induction Week 12 non-responders (n=87)
Demographics		
Age in years, mean (SD)	41.6 (14.0)	44.9 (15.1)
Male, %	56.7	66.7
Disease characteristics		
UC duration in years, mean (SD)	8.09 (7.02)	8.21 (6.72)
mMayo score (0–9), mean (SD)	6.7 (1.1)	6.7 (1.2)
mMayo score of 7–9 (severe), %	57.2	67.8
Mayo endoscopy subscore of 3 (severe), %	50.0	65.5
Extensive UC, %	48.9	62.1
UC-related concomitant medications, %		
Oral corticosteroid use	30.0	36.8
6-MP/AZA use	22.2	16.1
Oral 5-ASA compound use	78.9	75.9
BIO/JAKi/S1Pi naïve, n (%)	115 (63.9)	43 (49.4)
BIO/JAKi/S1Pi-IR, n (%)	63 (35.0)	43 (49.4)
One class*	47 (74.6)	29 (67.4)
≥2 classes*	16 (25.4)	14 (32.6)

*Denominator is participants who were BIO/JAKi/S1Pi-IR.
5-ASA, 5-aminosalicylic acid; 6-MP, 6-mercaptopurine; AZA, azathioprine; BIO, biologic; GUS, guselkumab; IL, interleukin; IR, inadequate response; JAKi, Janus kinase inhibitor; mMayo, modified Mayo; MOA, mechanism of action; RDBPC, randomised, double-blind, placebo-controlled; S1Pi, sphingosine-1-phosphate inhibitor; SD, standard deviation; UC, ulcerative colitis.
Danese S, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. DOP105. Full prescribing information: www.swissmedicinfo-pro.ch.

Baseline characteristics and medication history of GUS induction Week 12 non-responders by remission status at Week 48

	GUS induction Week 12 non-responders	
	Achieved clinical remission at Week 48 (n=22)	Did not achieve clinical remission at Week 48 (n=65)
Demographics		
Age, years, mean (SD)	43.1 (16.4)	45.5 (14.8)
Male, %	68.2	66.2
Disease characteristics		
UC duration, years, mean (SD)	6.71 (5.93)	8.72 (6.93)
mMayo score (0–9), mean (SD)	6.9 (1.6)	6.6 (1.1)
mMayo score of 7–9 (severe), %	77.3	64.6
Mayo endoscopy subscore of 3 (severe), %	72.7	63.1
Extensive UC, %	54.5	64.6
UC-related concomitant medications, %		
Oral corticosteroid use	31.8	38.5
6-MP/AZA use	36.4	9.2
Oral 5-ASA compound use	90.9	70.8
BIO/JAKi/S1Pi naïve, n (%)	15 (68.2)	28 (43.1)
BIO/JAKi/S1Pi-IR, n (%)	7 (31.8)	36 (55.4)
One class*	5 (71.4)	24 (66.7)
≥2 classes*	2 (28.6)	12 (33.3)

*Denominator is participants who were BIO/JAKi/S1Pi-IR.
5-ASA, 5-aminosalicylic acid; 6-MP, 6-mercaptopurine; AZA, azathioprine; BIO, biologic; GUS, guselkumab; IL, interleukin; IR, inadequate response; JAKi, Janus kinase inhibitor; mMayo, modified Mayo; MOA, mechanism of action; RDBPC, randomised, double-blind, placebo-controlled; S1Pi, sphingosine-1-phosphate inhibitor; SD, standard deviation; UC, ulcerative colitis.
Danese S, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. DOP105. Full prescribing information: www.swissmedicinfo-pro.ch.

Conclusions

Among patients with moderately to severely active UC in the ASTRO study:

- Participants who achieved clinical response after GUS SC induction treatment had better clinical and endoscopic outcomes at Week 48 than those who did not achieve clinical response after induction
- A subset of participants who were not in clinical response after induction, but continued GUS SC maintenance, achieved clinical and endoscopic endpoints at Week 48

Overall, these results suggest a benefit of continued GUS treatment after Week 12 regardless of induction clinical response status

GUS, guselkumab; IL, interleukin; MOA, mechanism of action; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous; UC, ulcerative colitis.
Danese S, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. DOP105. Full prescribing information: www.swissmedicinfo-pro.ch.

P1155

Evaluation of complete bowel symptomatic remission (CBSR) in patients with moderately to severely active ulcerative colitis

Higgins P,[1] Alvarez Y,[2] Baker T,[2] Germinaro M,[2] Fieo R,[2] Kato K,[2] Han C[3]

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Aim: to validate a novel PRO composite measure that includes AP, BU, RB and SF using daily diary data from the UC-PRO/SS instrument

Correlations between UC bowel symptom composite scores and relevant IBDQ or PROMIS-29 components were moderate to strong

UC-PRO/SS bowel symptom scores

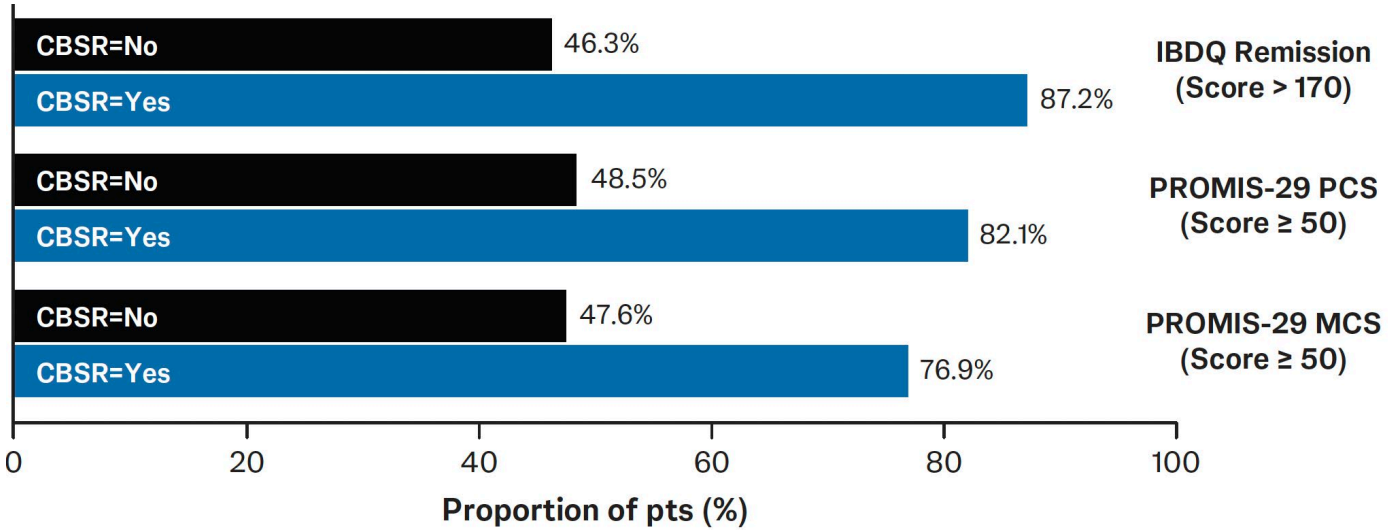
	AP	BU	RB	SF	Composite
IBDQ					
Total score	−0.54	−0.58	−0.52	−0.55	**−0.66**
Domain scores					
Bowel	−0.59	−0.63	−0.58	−0.58	**−0.71**
Emotional	−0.48	−0.51	−0.46	−0.47	**−0.57**
Systematic	−0.49	−0.45	−0.40	−0.45	**−0.53**
Social function	−0.43	−0.54	−0.44	−0.54	**−0.59**
PROMIS-29					
Physical health summary score	−0.44	−0.46	−0.37	−0.46	**−0.52**
Mental health summary score	−0.51	−0.46	−0.38	−0.45	**−0.53**
Item scores					
Physical function	−0.32	−0.36	−0.29	−0.35	**−0.40**
Anxiety	0.42	0.37	0.33	0.32	**0.43**
Depression	0.36	0.28	0.27	0.26	**0.33**
Fatigue	0.40	0.36	0.30	0.36	**0.42**
Sleep disturbance	0.37	0.30	0.21	0.31	**0.36**
Social participation/daily activity	−0.41	−0.48	−0.39	−0.47	**−0.52**
Pain interference	0.55	0.46	0.37	0.45	**0.53**
Pain intensity NRS	0.60	0.44	0.36	0.39	**0.52**

Data presented are Spearman's correlation coefficients; all p-values <0.0001. Higher scores in IBDQ domains indicate better outcomes. Higher PROMIS-29 domain scores indicate more severe symptoms or higher functioning. Higher UC-PRO/SS symptom scores indicate more severe symptoms.

AP, abdominal pain; BU, bowel urgency; GUS, guselkumab; IBDQ, Inflammatory Bowel Disease Questionnaire; IL, interleukin; MOA, mechanism of action; NRS, numeric rating scale; PRO, patient-reported outcome; PROMIS-29, Patient-Reported Outcomes Measurement Information System; RB, rectal bleeding; RDBPC, randomised, double-blind, placebo-controlled; SF, stool frequency; UC, ulcerative colitis; UC-PRO/SS, Ulcerative Colitis Patient-Reported Outcomes Signs and Symptoms.

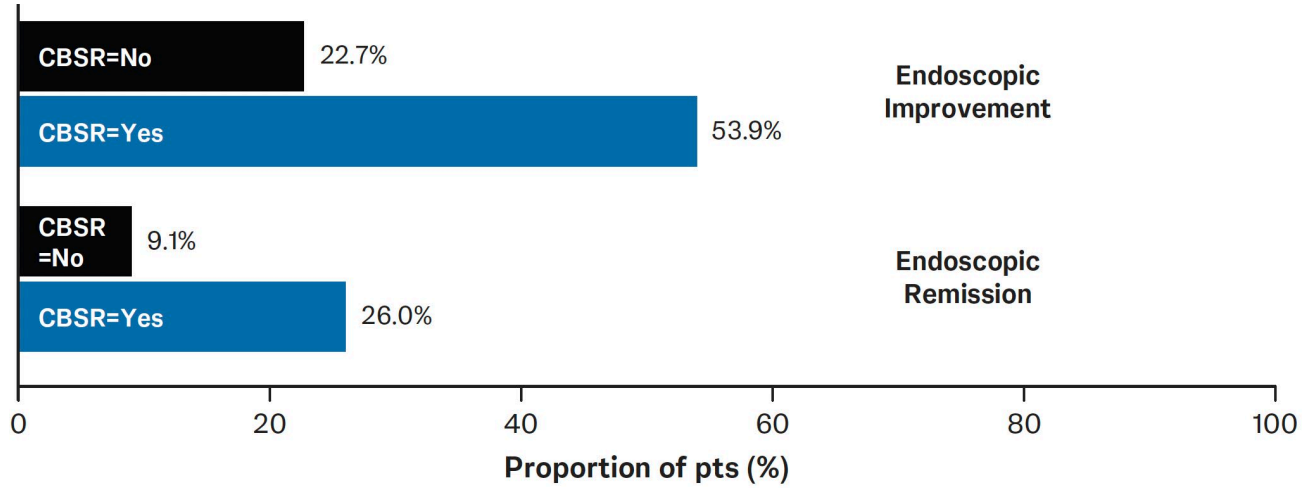
Higgins P, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. Poster P1155. Full prescribing information: www.swissmedicinfo-pro.ch.

Participants with normalised HRQoL by CBSR (yes [n=78] or no [n=379]) at Week 12



- More participants with CBSR achieved HRQoL normalisation vs those without CBSR

Participants with endoscopic improvement and endoscopic remission by CBSR (yes [n=77] or no [n=308]) at Week 12



- More participants with CBSR achieved endoscopic improvement and endoscopic remission vs those without CBSR

Conclusions

- The UC bowel symptom composite score is a validated assessment of bowel symptom severity in participants with UC
- CBSR was associated with better endoscopic outcomes and normalised HRQoL
- Using CBSR to assess efficacy in UC clinical trials may be a more comprehensive, stringent and differential approach than the current convention (i.e., based solely on rectal bleeding and stool frequency from Mayo score components)

Complete bowel symptomatic remission: complete resolution of AP, BU and RB (rounded weekly average score of '0') with SF score ≤1 (i.e. ≤4 times per day), based on UC-PRO/SS. **Normalised HRQoL**: disease-specific IBDQ total score >170; generic PROMIS-29 PCS or MCS T-score ≥50. Raw scores (observed) were used without adjustment. Data are for all ASTRO treatment groups (i.e., GUS and PBO) combined.

AP, abdominal pain; BU, bowel urgency; CBSR, complete bowel symptomatic remission; GUS, guselkumab; HRQoL, health-related quality of life; IBDQ, Inflammatory Bowel Disease Questionnaire; IL, interleukin; MCS, mental health component score; MOA, mechanism of action; PBO, placebo; PCS, physical health component score; PROMIS-29, Patient-Reported Outcomes Measurement Information System; pts, patients; RB, rectal bleeding; RDBPC, randomised, double-blind, placebo-controlled; SF, stool frequency; UC, ulcerative colitis; UC-PRO/SS, Ulcerative Colitis Patient-Reported Outcomes Signs and Symptoms.

Higgins P, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. Poster P1155. Full prescribing information: www.swissmedicinfo-pro.ch.

QUASAR/ASTRO

DOP103

Intravenous and subcutaneous guselkumab induction are similarly efficacious in patients with ulcerative colitis across weight quartile and BMI subgroups: Week 12 results from the Phase 3 QUASAR and ASTRO studies

Yarur AJ,[1] Deepak P,[2] Hisamatsu T,[3] Alvarez Y,[4] Baker T,[4] Adsul S,[5] Piscitelli D,[5] Miao Y,[4] Rubin DT,[6] Dignass A[7]

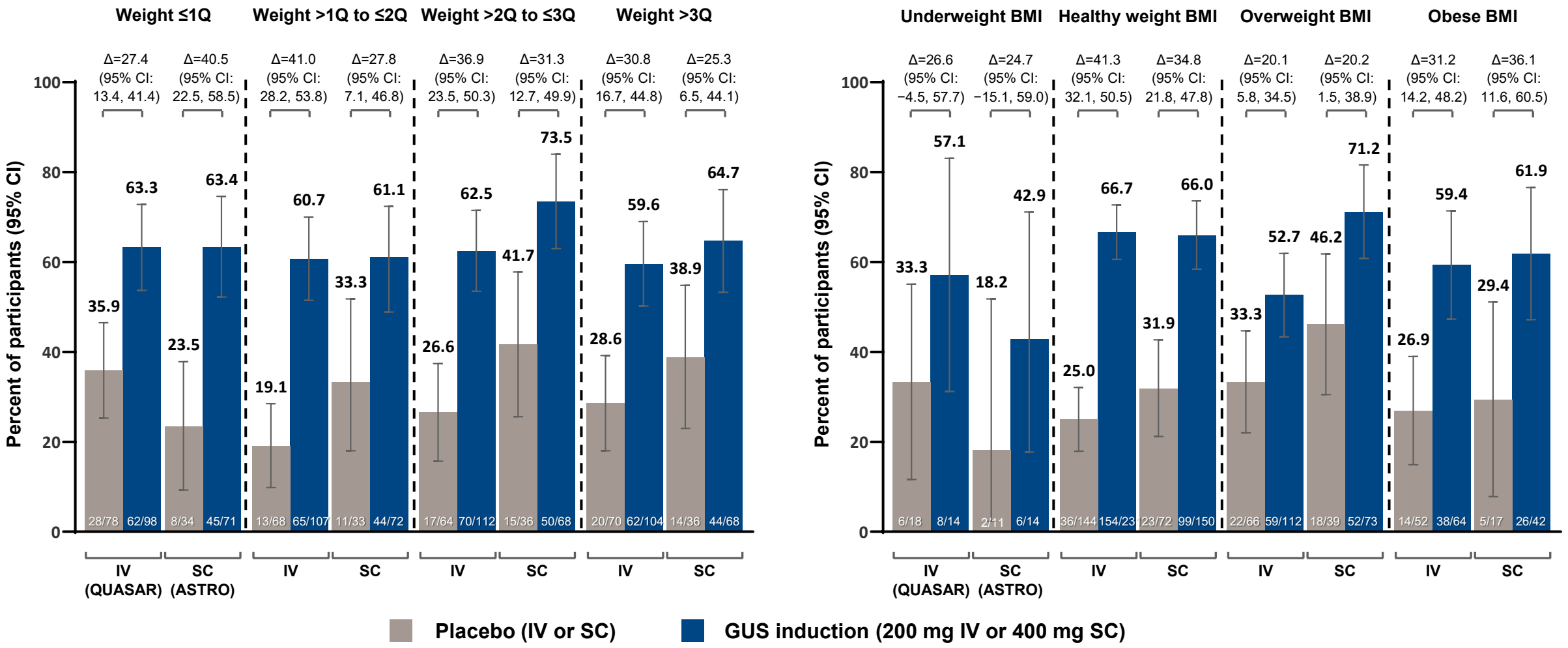
[1]Cedars-Sinai Inflammatory Bowel Disease Center, Los Angeles, CA, USA; [2]Washington University, St. Louis, MO, USA; [3]Kyorin University, Tokyo, Japan; [4]Johnson & Johnson, Spring House, PA, USA; [5]Johnson & Johnson, Horsham, PA, USA; [6]University of Chicago Medicine Inflammatory Bowel Disease Center, Chicago, IL, USA; [7]Department of Medicine I, Agaplesion Markus Hospital, Goethe University, Frankfurt, Germany

Baseline demographics and disease characteristics

Primary analysis set	QUASAR*	ASTRO
	Total N=701	Total N=418
Demographics		
Weight in kg, mean (SD)	72.45 (16.84)	71.40 (16.45)
Median (range)	70.6 (38.0; 127.1)	69.0 (37.4; 121.6)
Age in years, mean (SD)	40.5 (13.72)	41.7 (14.22)
Male, n (%)	399 (56.9)	256 (61.2)
Characteristics		
UC disease duration in years, mean (SD)	7.52 (7.282)	7.56 (6.674)
Modified Mayo score (0–9),† mean (SD)	6.9 (1.10)	6.7 (1.15)
Modified Mayo score of 7–9 (severe),† n (%)	452 (64.5)	259 (62.1)
Mayo endoscopic subscore of 3 (severe), n (%)	476 (67.9)	234 (56.0)
Extensive UC, n (%)	335 (47.8)	224 (53.6)
Biomarkers		
C-reactive protein in mg/L,‡ median (IQR)	4.2 (1.5; 10.1)	4.1 (1.4; 8.9)
Faecal calprotectin in µg/g,§ median (IQR)	1641.0 (647.0; 3304.0)	1566.0 (641.0; 2964.0)
Concomitant UC medications at baseline, n (%)		
6-MP/AZA	138 (19.7)	82 (19.6)
MTX	8 (1.1)	2 (0.5)
Oral corticosteroids	302 (43.1)	137 (32.8)
BIO/JAK inhibitor therapy history, n (%)		
BIO/JAK inhibitor naïve	339 (48.4)	243 (58.1)
History of inadequate response/intolerance to BIO/JAK inhibitor	344 (49.1)	168 (40.2)

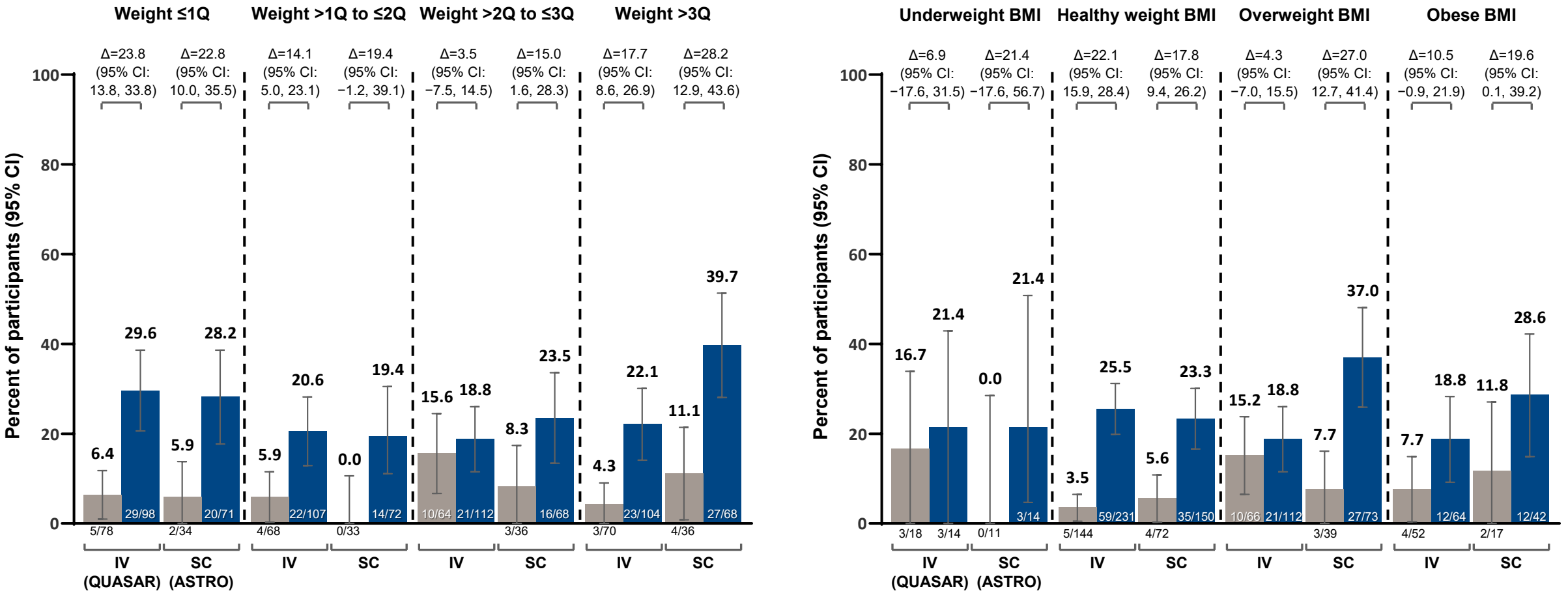
*Includes all participants in QUASAR induction study 2; †Based on N=701 for QUASAR and N=417 for ASTRO; ‡Based on n=694 for QUASAR and n=414 for ASTRO; §Based on n=623 for QUASAR and n=385 for ASTRO.
6-MP, 6-mercaptopurine; AZA, azathioprine; BIO, biologic; GUS, guselkumab; IL, interleukin; IQR, interquartile range; JAK, Janus kinase; MOA, mechanism of action; MTX, methotrexate; SD, standard deviation; UC, ulcerative colitis.
Yarur A, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. DOP103. Full prescribing information: www.swissmedicinfo-pro.ch.

Clinical response at Week 12 by baseline weight quartile and BMI subgroups



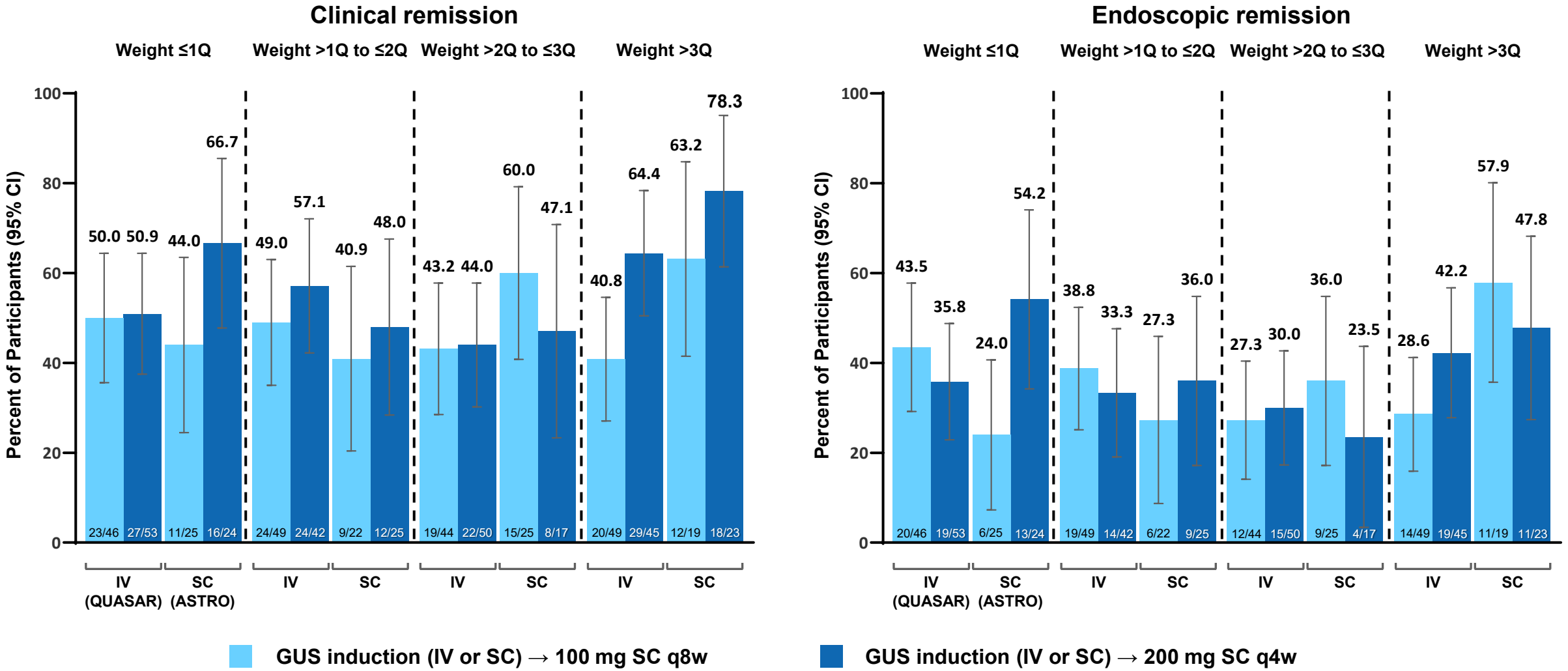
Post hoc analysis.
CI, confidence interval; BMI, body mass index; GUS, guselkumab; IL, interleukin; IV, intravenous; MOA, mechanism of action; Q, quartile; SC, subcutaneous; UC, ulcerative colitis.
Yarur A, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. DOP103. Full prescribing information: www.swissmedicinfo-pro.ch.

Clinical remission at Week 12 by baseline weight quartile and BMI subgroups



Post hoc analysis.
CI, confidence interval; BMI, body mass index; GUS, guselkumab; IL, interleukin; IV, intravenous; MOA, mechanism of action; Q, quartile; SC, subcutaneous; UC, ulcerative colitis.
Yarur A, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. DOP103. Full prescribing information: www.swissmedicinfo-pro.ch.

Efficacy at Week 44/48 by baseline weight quartile



Post hoc analysis.
CI, confidence interval; GUS, guselkumab; IL, interleukin; IV, intravenous; MOA, mechanism of action; Q, quartile; qXw, every X weeks; SC, subcutaneous; UC, ulcerative colitis.
Yarur A, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. DOP103. Full prescribing information: www.swissmedicinfo-pro.ch.

Conclusions

- Guselkumab IV and SC induction were similarly effective in participants with moderately to severely active UC compared with placebo at Week 12 regardless of participants' baseline body weight or BMI
- Among Week 12 clinical responders, similar efficacy was observed at 1 year (Week 44 and 48 for QUASAR and ASTRO, respectively) between participants treated with either guselkumab IV or SC induction regardless of participants' baseline body weight
- Efficacy of guselkumab does not appear to be impacted by body weight or BMI, irrespective of route of induction treatment administration

BMI, body mass index; GUS, guselkumab; IL, interleukin; IV, intravenous; MOA, mechanism of action; RDBPC, randomised, double-blind, placebo-controlled; SC, subcutaneous; UC, ulcerative colitis.
Yarur A, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. DOP103. Full prescribing information: www.swissmedicinfo-pro.ch.

P0925

Pharmacokinetics and exposure-response relationships of guselkumab intravenous or subcutaneous induction in participants with ulcerative colitis

Peyrin-Biroulet L,[1] Xu Z,[2] Shao J,[2] Hisamatsu T,[3] Long M,[4] Danese S,[5] Germinaro M,[2] Vetter ML,[2] Yarandi S,[2] Baker T,[2] Allegretti JR,[6] Rubin DT[7]

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Pharmacokinetics and exposure-response relationships of GUS intravenous or subcutaneous induction in participants with UC

Background

GUS a dual-acting IL-23p19 subunit inhibitor approved for the treatment of UC after IV or SC induction, followed by SC maintenance therapy. Here, we evaluated the PK and exposure-response of IV and SC GUS induction.

Methods

The GUS clinical development programme in participants with moderately to severely active UC consisted of the Phase 2b/3 QUASAR studies of IV induction (200 mg q4w x3) and SC maintenance (100 mg q8w or 200 mg q4w) and the Phase 3 ASTRO study of SC induction (400 mg q4w x3) and SC maintenance (same as QUASAR). All studies had randomised, double-blind, placebo-controlled, parallel group designs. To compare GUS PK exposure after 200 mg IV vs 400 mg SC induction through Week 12, individual *post-hoc* PK parameter values (estimated with the established QUASAR 2-compartment linear popPK model with first-order absorption and elimination) and participant dosing information from QUASAR and ASTRO were used to simulate concentration–time profiles and calculate individual induction exposure metrics. Comparative graphical E-R analysis (QUASAR vs ASTRO) was conducted for key Week 12 efficacy outcomes (clinical remission, clinical response, endoscopic improvement and histologic-endoscopic mucosal improvement) using the overall exposure during induction ($C_{ave,\ Week\ 0–12}$) and associated exposure quartiles from the combined study populations.

Results

Consistent with model predictions, SC induction resulted in similar average concentrations (Week 0–Week 12), similar area under the concentration–time curves (Week 0–Week 12), lower peak concentrations (at Week 8) and higher trough concentrations (at Week 12) compared with the PK profile of IV induction. GUS steady-state concentration was reached by Week 24, and popPK model-based simulations showed that serum GUS concentrations were comparable by Week 24 after the same maintenance dose regimen, regardless of induction administration route. Key efficacy outcomes at Week 12 were comparable within the same GUS concentration quartiles following IV vs SC induction. Similar positive E-R trends were observed after IV or SC induction.

Conclusion

Consistent average serum GUS concentrations and E-R patterns after IV and SC induction underscore the observed clinical efficacy and support the use of either induction administration route in patients with UC.

E-R, exposure-response; GUS, guselkumab; IL, interleukin; IV, intravenous; MOA, mechanism of action; PK, pharmacokinetics; popPK, population pharmacokinetics; qXw, every X weeks; SC, subcutaneous; UC, ulcerative colitis.

Peyrin-Biroulet L, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. Poster P0295. Full prescribing information: www.swissmedicinfo-pro.ch.

Guselkumab in IBD

P1042

Pregnancy outcomes in maternal exposure to guselkumab: Review of cases reported to the company's global safety database

Mahadevan U,[1] Long M,[2] Julsgaard M,[3] Lin C,[4] Geldhof A,[5] Ballina MR,[6] Li H,[7] Gisbert JP,[8] Chaparro M[8]

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Objective and methods

Objective

This analysis reported data on pregnancy cases with known outcomes in women exposed to GUS during pregnancy

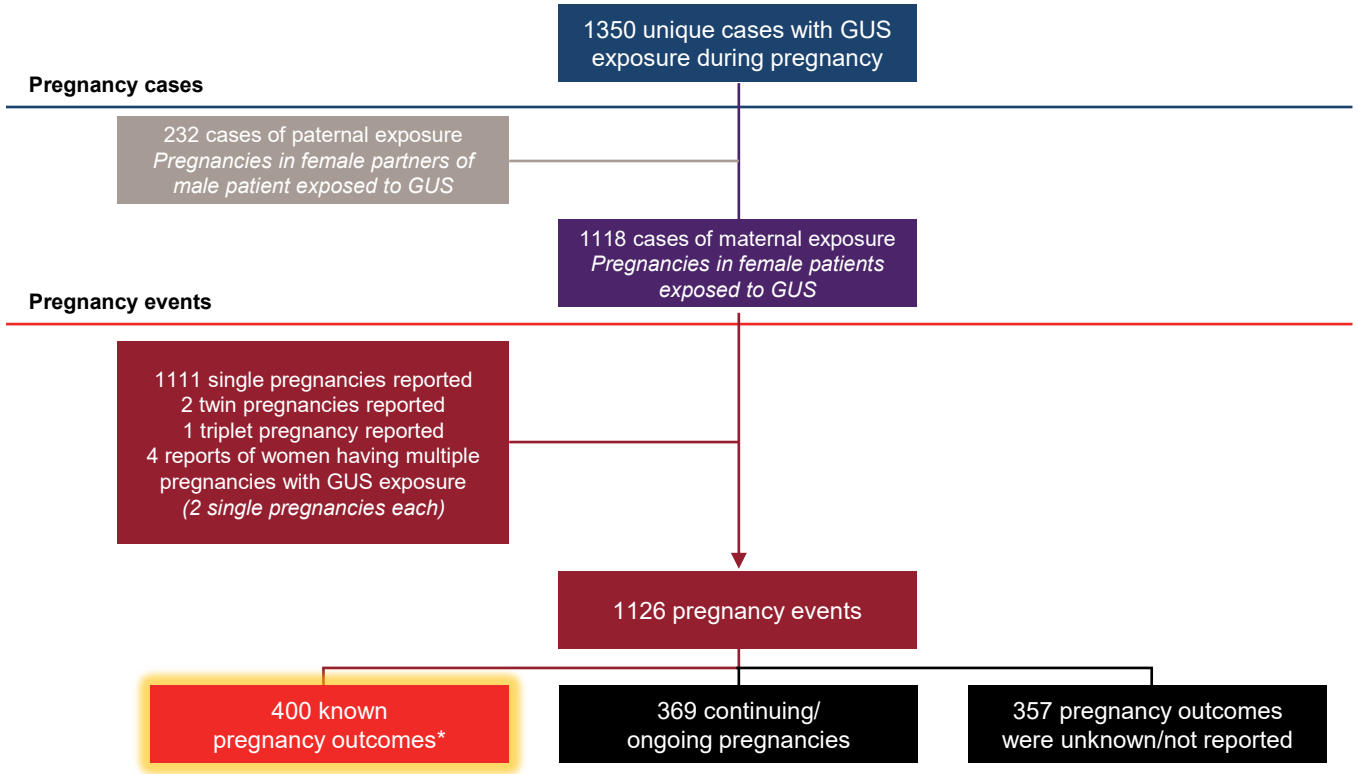
Methods

- **Pregnancy cases were reported to the Company Global Safety Database through 12 July 2025**
  - Cases with known outcomes were reported from clinical studies and spontaneous sources
- **Data were summarised descriptively for pregnancies reported prospectively and retrospectively**
- **GUS therapeutic indications in pregnancy cases were:**
  - Psoriatic disease
  - CD
  - UC
  - Other/not reported
- **Maternal GUS exposure was categorised as follows:**
  - Before conception (within 3 months prior to conception)
  - During the first trimester (T1)
  - After the first trimester only (T2, T3)
  - Throughout pregnancy
  - Not reported
- **Pregnancy outcomes were classified as:**
  - Live births (with or without congenital anomalies)
  - Spontaneous abortions
  - Elective terminations (with or without foetal defects, or unknown)
  - Ectopic pregnancies
  - Stillbirths (with or without foetal defects)
  - Unspecified abortions (with or without foetal defects, or unknown)

CD, Crohn's disease; GUS, guselkumab; IL, interleukin; MOA, mechanism of action; T1, first trimester; T2, second trimester; T3, third trimester; TA, therapy area; UC, ulcerative colitis.
Mahadevan U, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. P1042. Full prescribing information: www.swissmedicinfo-pro.ch.

Among 396 women, 400 pregnancy events with known outcomes occurred; the majority of women received GUS treatment for psoriatic disease

Pregnancy cases and pregnancy events among 396 women



- Maternal age was reported for 264/396 women (67%); mean maternal age was **32 years**

Proportions of women exposed to GUS during pregnancy by GUS therapeutic indication (N=396)

	n	Percentage
Psoriatic disease†	**289**	**73.0%**
Not reported	**70**	**17.7%**
Inflammatory bowel disease	**25**	**6.3%**
CD	15	3.8%
UC	10	2.5%
Other‡	12	3.0%

Proportions of prospectively and retrospectively reported pregnancy events (N=400)

70% **Retrospective events**

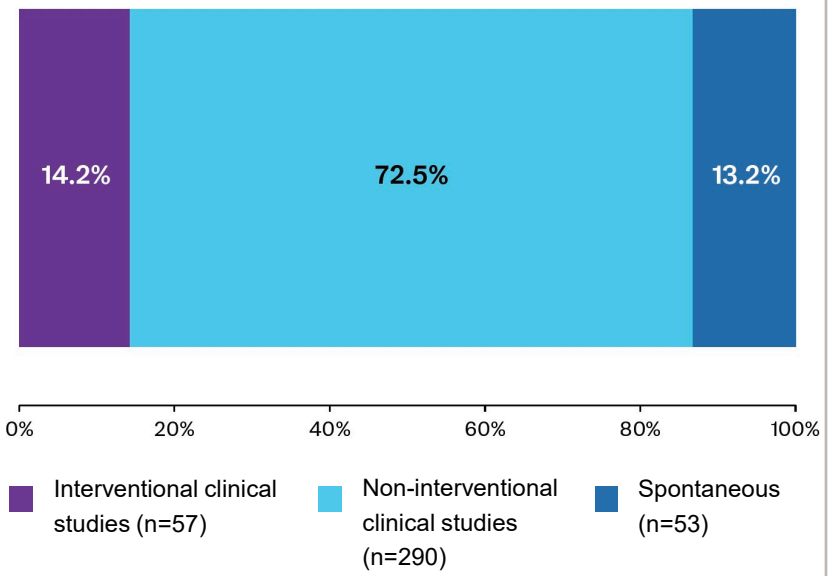
vs

30% **Prospective events**

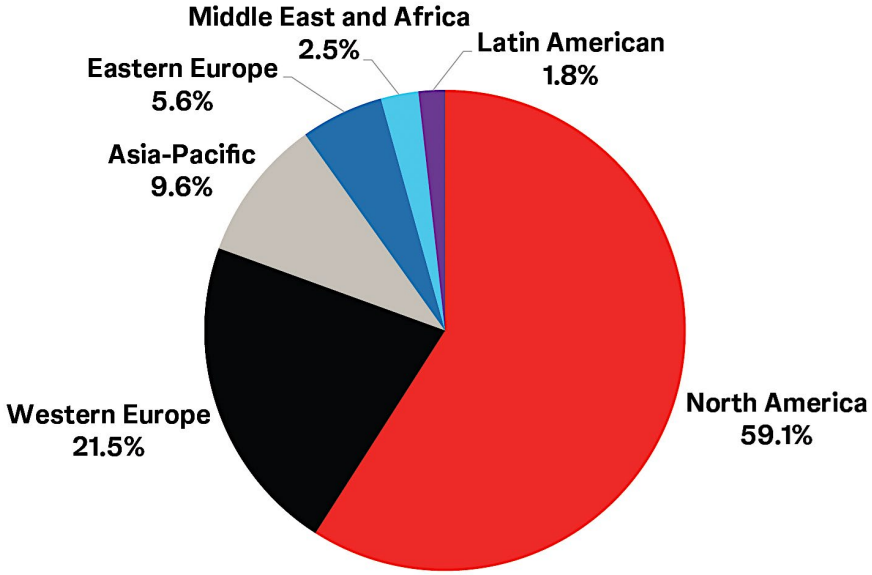
*Includes 287 medically confirmed pregnancies and 109 medically unconfirmed pregnancies; †Includes cases reported as psoriasis, psoriatic arthritis and psoriasis + psoriatic arthritis; ‡Other indications included hidradenitis suppurativa, palmoplantar pustulosis, guttate psoriasis, pityriasis rubra pilaris, rheumatoid arthritis and healthy individuals from Phase 1 studies.
CD, Crohn's disease; GUS, guselkumab; IL, interleukin; MOA, mechanism of action; TA, therapy area; UC, ulcerative colitis.
Mahadevan U, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. P1042. Full prescribing information: www.swissmedicinfo-pro.ch.

Proportions of pregnancy cases/events during GUS treatment reported by reporting source, geographic region and timing of exposure

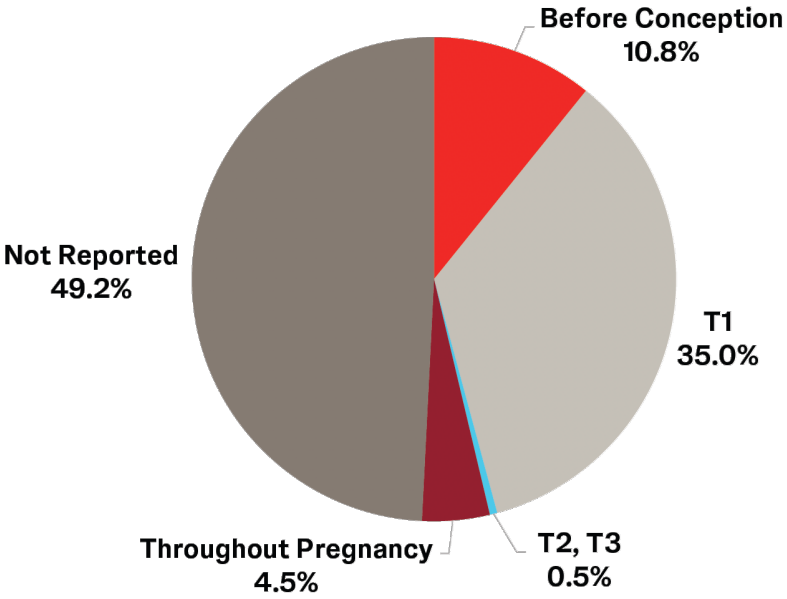
Proportions of pregnancy events by reporting source (N=400)



Proportions of pregnancy cases reported by geographic region (N=396)

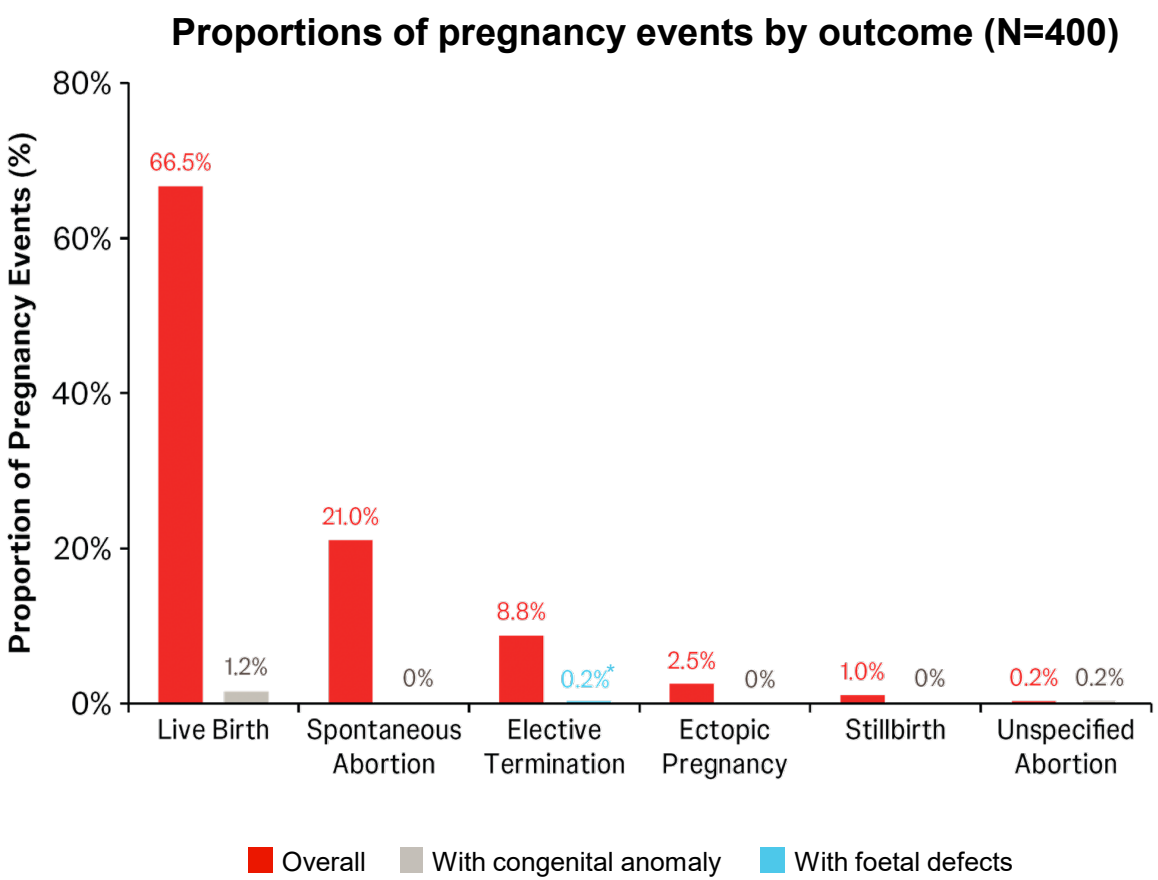


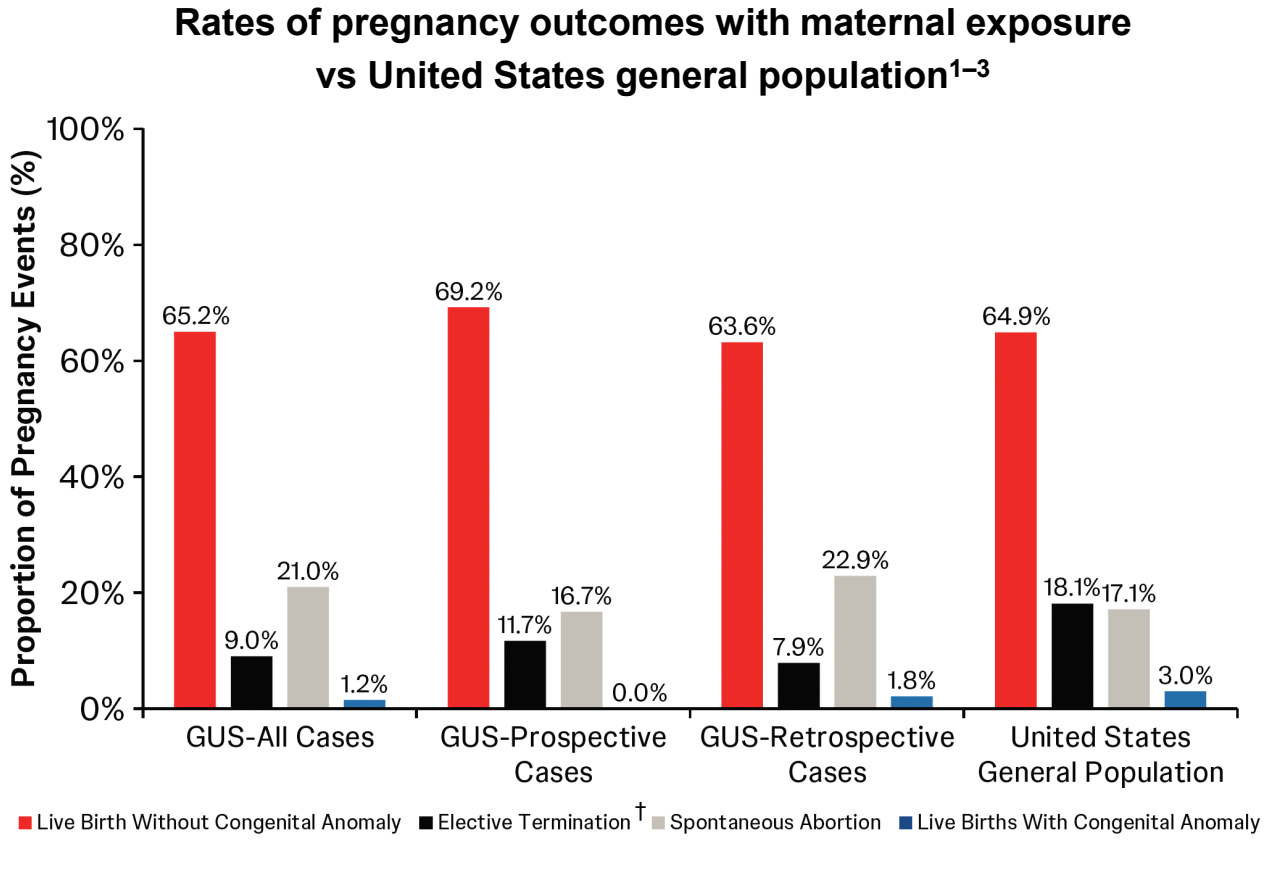
Proportions of pregnancy cases reported by timing of exposure (N=400)



GUS, guselkumab; IL, interleukin; MOA, mechanism of action; T1, first trimester; T2, second trimester; T3, third trimester; TA, therapy area.
Mahadevan U, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. P1042. Full prescribing information: www.swissmedicinfo-pro.ch.

Pregnancy outcomes





Rates of pregnancy outcomes in women exposed to GUS were consistent with those of the general US population[1–3]

*One case reported baby adverse event of foetal disorder with no further information; conservatively, it was categorised as a foetal defect; †Included cases reporting unspecified abortions.
GUS, guselkumab; IL, interleukin; MOA, mechanism of action; TA, therapy area.

1. Mahadevan U, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. P1042; 2. Curtin SC, et al. *NCHS Data Brief*. 2013;(136):1-8; 3. About Birth Defects. U.S. Centers for Disease Control and Prevention. Available at: https://www.cdc.gov/birth-defects/index.html. Accessed February 2026. Full prescribing information: www.swissmedicinfo-pro.ch.

Congenital anomalies by pregnancy outcome and timing of exposure

Congenital anomalies by pregnancy outcome and timing of exposure

Events of interest†	Number of events	Pregnancy outcomes	Timing of maternal exposure	Classification‡
Trisomy 13§	1	Live birth	Throughout pregnancy	Major anomaly Chromosomal
Congenital heart disease	1	Live birth	Not reported	Major anomaly Non-chromosomal
Oesophageal atresia‖	1	Live birth	Not reported	Major anomaly Non-chromosomal
Cerebral ventricle dilation	1	Live birth	During the first trimester	Major anomaly Non-chromosomal
Single umbilical artery	1	Live birth	During the first trimester	Major anomaly Non-chromosomal
Foetal malformation	1	Unspecified abortion	During the first trimester	Termination of pregnancy due to foetal anomaly

Of the 400 pregnancy events with known outcomes, 6 (1.5%) pregnancies were associated with congenital anomalies

†Medical Dictionary for Regulatory Activities (MedDRA, version 28.0) was used to identify adverse events based on the System Organ Class of congenital, familial or genetic disorders, which is a sub-search of the Standardized MedDRA Query of pregnancy and neonatal topics; ‡Major and chromosomal congenital anomalies per EUROCAT classification are reported unless otherwise specified; §Pre-term delivery at less than 37 weeks; baby died due to Trisomy 13; ‖Baby adverse event of tracheomalacia was reported.
GUS, guselkumab; IL, interleukin; MOA, mechanism of action; TA, therapy area.
Mahadevan U, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. P1042. Full prescribing information: www.swissmedicinfo-pro.ch.

Conclusions

- These findings suggest no apparent impact of GUS on pregnancy outcomes; however, they should be interpreted cautiously, given data limitations.
- Further studies are warranted to confirm these observations and to better characterise the safety profile of GUS exposure during pregnancy.

The limitations of these findings include, but are not limited to, the lack of study control, a small sample size of pregnancies with known outcomes, and reported data with varying degrees of missing information.
GUS, guselkumab; IL, interleukin; MOA, mechanism of action; TA, therapy area.
Mhadevan U, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. P1042. Full prescribing information: www.swissmedicinfo-pro.ch.

P1069

Safety of guselkumab in patients aged ≥60 years with immune-mediated inflammatory diseases: A pooled analysis of registrational trials in UC, CD, PsA and PsO

Faye AS,[1] Sebastian S,[2] McCaffrey V,[3] Bravatà I,[4] Nazar M,[5] Piscitelli D,[6] Chakravarty SD,[6,7] Adsul S,[6] Yee J,[3] Baker T,[3] Sands BE[8]

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Objective and methods

Objective

Evaluate the safety of GUS up to 1 year in participants aged ≥60 years pooled from 14 Phase 2/3 RCTs

Methods

Analysis cohort

- Data were pooled from 14 Phase 2/3 RCTs of GUS in PsA, PsO, UC and CD
- Adults who received ≥1 dose of study treatment were included
- Non-biologic immunomodulators (including disease-modifying anti-rheumatic drugs) and corticosteroids were permitted in IBD (UC and CD) and PsA RCTs, but not in PsO RCTs

Outcomes and analyses

- Pooled safety data from RCTs were analysed through up to 1 year
- Participants were grouped by age: **≥60 years of age** and **overall population**
- Age groups were further subdivided by indication: **IBD** and **combined indications**
- Exposure-adjusted rates of TEAEs were reported per 100 patient-years with 95% confidence intervals

Pooled data from 14 Phase 2/3 RCTs of GUS in PsA, PsO, UC and CD

RCT ID	RCT name	Indication
NCT03162796	DISCOVER-1	PsA
NCT03158285	DISCOVER-2	PsA
NCT02319759	PsA Phase 2	PsA
NCT02203032	NAVIGATE	PsO
NCT02207231	VOYAGE 1	PsO
NCT02207244	VOYAGE 2	PsO
NCT01483599	X-PLORE	PsO
NCT05528510	ASTRO	UC
NCT04033445	QUASAR	UC
NCT03662542	VEGA	UC
NCT03466411	GALAXI 1	CD
NCT03466411	GALAXI 2	CD
NCT03466411	GALAXI 3	CD
NCT05197049	GRAVITI	CD

CD, Crohn's disease; GUS, guselkumab; IBD, inflammatory bowel disease; IL, interleukin; MOA, mechanism of action; PsA, psoriatic arthritis; PsO, psoriasis; RCT, randomised controlled trial; TA, therapy area; TEAE, treatment-emergent adverse event; UC, ulcerative colitis.

Faye AS, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. P1069. Full prescribing information: www.swissmedicinfo-pro.ch.

Among participants with IBD aged ≥60 years, 238 received GUS and 98 received PBO, contributing 175.5 and 47.9 PY of follow-up, respectively, across up to 1 year in 14 RCTs

Exposure and follow-up metrics by age group and indication*

	Patients aged ≥60 years				Overall population			
	IBD†		Combined indications†‡		IBD†		Combined indications†‡	
	GUS§ (n=238)	PBO‖ (n=98)	GUS§ (n=618)	PBO‖ (n=212)	GUS§ (n=2057)	PBO‖ (n=886)	GUS§ (n=5048)	PBO‖ (n=1771)
Mean follow-up, weeks¶	38.5	25.5	41.9	22.3	41.4	25.0	42.9	22.3
Mean treatment, weeks	31.1	22.4	32.8	19.2	33.8	21.6	33.5	19.1
Total PY of follow-up	175.5	47.9	496.8	90.5	1630.1	424.3	4152.8	758.3

In participants aged ≥60 years across combined indications, 618 received GUS and 212 received PBO, contributing 496.8 and 90.5 PY of follow-up, respectively, across up to 1 year

Mean follow-up and treatment duration were comparable within treatment groups for participants aged ≥60 years compared with the overall population

*Safety events reported throughout the reporting period to approximately 1 year; SCS all treated; †UC: CNTO1959UCO3001 (to Week 20, 32 or 44 depending on entry or treatment status); CNTO1959UCO2002 (to Week 38, monotherapy arm only). CD: CNTO1959CRD3001 GALAXI 1, GALAXI 2 and GALAXI 3 and CNTO1959CRD3004 GRAVITI (to Week 48); ‡Includes IBD (CD and UC), PsO and PsA. UC: CNTO1959UCO3001 (to Week 20, 32 or 44 depending on entry or treatment status); CNTO1959UCO2002 (to Week 38, monotherapy arm only). CD: CNTO1959CRD3001 GALAXI 1, GALAXI 2 and GALAXI 3 and CNTO1959CRD3004 GRAVITI (to Week 48). PsO: CNTO1959PSO3001, CNTO1959PSO3002, CNTO1959PSO3003 and CNTO1959PSO2001 (to Week 52). PsA: CNTO1959PSA3001 (to Week 60), CNTO1959PSA3002 (to Week 52), CNTO1959PSA2001 (to Week 56); §UC: all GUS data and up to 12 weeks post-induction for PBO in maintenance study. CD, PsO, PsA: data from first GUS dose for early escape/crossover; ‖UC: data to first GUS dose (PBO) or ≥12 weeks after last induction (re-randomised to PBO), until dose adjustment. CD, PsO, PsA: data to early escape/rescue/crossover; ¶Cumulative treatment duration for each study agent was calculated as the time from first to last dose across all relevant periods.

CD, Crohn's disease; GUS, guselkumab; IBD, inflammatory bowel disease; IL, interleukin; MOA, mechanism of action; PBO, placebo; PsA, psoriatic arthritis; PsO, psoriasis; PY, patient-years; RCT, randomised controlled trial; SCS, summary of clinical safety; TA, therapy area; UC, ulcerative colitis.

Faye AS, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. P1069. Full prescribing information: www.swissmedicinfo-pro.ch.

Among participants with IBD aged ≥60 years, exposure-adjusted rates of TEAEs, SAEs and infections were numerically lower with GUS than with PBO across both the IBD and combined indications groups

TEAEs by age group and indication*

	Participants aged ≥60 years				Overall population			
	IBD†		Combined indications†‡		IBD†		Combined indications†‡	
	GUS§ (n=238)	PBO‖ (n=98)	GUS§ (n=618)	PBO‖ (n=212)	GUS§ (n=2057)	PBO‖ (n=886)	GUS§ (n=5048)	PBO‖ (n=1771)
Patients with AEs, n (%)	164 (68.9)	63 (64.3)	418 (67.6)	125 (59.0)	1502 (73.0)	558 (63.0)	3349 (66.3)	973 (54.9)
TEAE/100 PY (95% CI)¶								
Any TEAE**	93.5 (79.7, 108.9)	131.5 (101.0, 168.2)	84.1 (76.3, 92.6)	138.1 (115.0, 164.6)	92.1 (87.5, 96.9)	131.5 (120.8, 142.9)	80.6 (77.9, 83.4)	128.3 (120.4, 136.6)
SAE	11.4 (7.0, 17.6)	23.0 (11.5, 41.4)	9.3 (6.8, 12.4)	16.6 (9.3, 27.3)	8.8 (7.5, 10.4)	17.9 (14.1, 22.4)	6.5 (5.8, 7.3)	12.7 (10.3, 15.5)
Infection	39.9 (31.1, 50.4)	56.4 (37.1, 82.0)	42.3 (36.7, 48.4)	55.3 (41.0, 72.8)	47.5 (44.2, 51.0)	53.0 (46.3, 60.4)	44.7 (42.7, 46.8)	53.0 (48.0, 58.5)
Serious infection	2.3 (0.6, 5.8)	2.1 (0.1, 11.6)	1.8 (0.8, 3.4)	1.1 (0.0, 6.2)	1.8 (1.2, 2.6)	1.7 (0.7, 3.4)	1.4 (1.0, 1.8)	1.2 (0.5, 2.3)
Death	0.0	4.2 (0.5, 15.1)	0.0	2.2 (0.3, 8.0)	0.1 (<0.1, 0.4)	0.5 (0.1, 1.7)	0.1 (0.0, 0.2)	0.5 (0.1, 1.4)

- No deaths were reported with GUS in either the IBD or combined indication groups among participants aged ≥60 years
- Similar findings were observed in the overall population

*Safety events reported throughout the reporting period to approximately 1 year; SCS all treated; †UC: CNTO1959UCO3001 (to Week 20, 32 or 44 depending on entry or treatment status); CNTO1959UCO2002 (to Week 38, monotherapy arm only). CD: CNTO1959CRD3001 GALAXI 1, GALAXI 2 and GALAXI 3 and CNTO1959CRD3004 GRAVITI (to Week 48); ‡Includes IBD (CD and UC), PsO and PsA. UC: CNTO1959UCO3001 (to Week 20, 32 or 44 depending on entry or treatment status); CNTO1959UCO2002 (to Week 38, monotherapy arm only). CD: CNTO1959CRD3001 GALAXI 1, GALAXI 2 and GALAXI 3 and CNTO1959CRD3004 GRAVITI (to Week 48). PsO: CNTO1959PSO3001, CNTO1959PSO3002, CNTO1959PSO3003 and CNTO1959PSO2001 (to Week 52). PsA: CNTO1959PSA3001 (to Week 60), CNTO1959PSA3002 (to Week 52), CNTO1959PSA2001 (to Week 56); §UC: all GUS data and up to 12 weeks post-induction for PBO in maintenance study. CD, PsO, PsA: data from first GUS dose for early escape/crossover; ‖UC: data to first GUS dose (PBO) or ≥12 weeks after last induction (re-randomised to PBO), until dose adjustment. CD, PsO, PsA: data to early escape/rescue/crossover; ¶Cumulative treatment duration for each study agent was calculated as the time from first to last dose across all relevant periods; **Confidence interval based on an exact method assuming that the observed number of subjects follows a Poisson distribution.
AE, adverse event; CD, Crohn's disease; CI, confidence interval; GUS, guselkumab; IBD, inflammatory bowel disease; IL, interleukin; MOA, mechanism of action; PBO, placebo; PsA, psoriatic arthritis; PsO, psoriasis; PY, patient-years; RCT, randomised controlled trial; SAE, serious adverse event; SCS, summary of clinical safety; TA, therapy area; TEAE, treatment-emergent adverse event; UC, ulcerative colitis.
Faye AS, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. P1069. Full prescribing information: www.swissmedicinfo-pro.ch.

Among participants with IBD aged ≥60 years, exposure-adjusted rates of opportunistic infections, MACEs and VTEs were numerically lower with GUS than with PBO across both the IBD and combined indications groups

Targeted TEAEs by age group and indication*

	Participants aged ≥60 years				Overall population			
	IBD†		Combined indications†‡		IBD†		Combined indications†‡	
	GUS§ (n=238)	PBO‖ (n=98)	GUS§ (n=618)	PBO‖ (n=212)	GUS§ (n=2057)	PBO‖ (n=886)	GUS§ (n=5048)	PBO‖ (n=1771)
TEAE/100 PY (95% CI)¶								
Active tuberculosis**	0.0	0.0	0.0	0.0	0.1 (0.0, 0.3)	0.0 (0.0, 0.7)	0.0 (0.0, 0.1)	0.0 (0.0, 0.4)
Malignancy††,‡‡	4.6 (2.0, 9.0)	4.2 (0.5, 15.1)	4.0 (2.5, 6.2)	2.2 (0.3, 8.0)	0.7 (0.4, 1.3)	0.9 (0.3, 2.4)	0.7 (0.5, 1.0)	0.7 (0.2, 1.5)
Opportunistic infection§§	1.1 (0.1, 4.1)	2.1 (0.1, 11.6)	0.6 (0.1, 1.8)	1.1 (<0.1, 6.2)	0.3 (0.1, 0.6)	0.7 (0.2, 2.1)	0.1 (0.1, 0.3)	0.4 (0.1, 1.2)
MACE‖‖	1.1 (0.1, 4.1)	4.2 (0.5, 15.1)	1.2 (0.4, 2.6)	2.2 (0.3, 8.0)	0.3 (0.1, 0.7)	0.5 (0.1, 1.7)	0.4 (0.2, 0.6)	0.4 (0.1, 1.2)
VTE¶¶	0.6 (<0.1, 3.20	2.1 (0.1, 11.6)	0.4 (0.1, 1.5)	1.1 (<0.1, 6.2)	0.4 (0.2, 0.9)	0.0 (0.0, 0.7)	0.3 (0.2, 0.6)	0.0 (0.0, 0.4)

- Among participants aged ≥60 years, exposure-adjusted malignancy rates (including non-melanoma skin cancer) were comparable between GUS and PBO in the IBD group, with broadly overlapping 95% CIs in the combined indications group indicating similar rates
- No cases of active tuberculosis were reported with GUS in either the IBD or combined indications groups among participants aged ≥60 years
- Similar findings were observed in the overall population

*Safety events reported throughout the reporting period to approximately 1 year; SCS all treated; †UC: CNTO1959UCO3001 (to Week 20, 32 or 44 depending on entry or treatment status); CNTO1959UCO2002 (to Week 38, monotherapy arm only). CD: CNTO1959CRD3001 GALAXI 1, GALAXI 2 and GALAXI 3 and CNTO1959CRD3004 GRAVITI (to Week 48); ‡Includes IBD (CD and UC), PsO and PsA. UC: CNTO1959UCO3001 (to Week 20, 32 or 44 depending on entry or treatment status); CNTO1959UCO2002 (to Week 38, monotherapy arm only). CD: CNTO1959CRD3001 GALAXI 1, GALAXI 2 and GALAXI 3 and CNTO1959CRD3004 GRAVITI (to Week 48). PsO: CNTO1959PSO3001, CNTO1959PSO3002, CNTO1959PSO3003 and CNTO1959PSO2001 (to Week 52). PsA: CNTO1959PSA3001 (to Week 60), CNTO1959PSA3002 (to Week 52), CNTO1959PSA2001 (to Week 56); §UC: all GUS data and up to 12 weeks post-induction for PBO in maintenance study. CD, PsO, PsA: data from first GUS dose for early escape/crossover; ‖UC: data to first GUS dose (PBO) or ≥12 weeks after last induction (re-randomised to PBO), until dose adjustment. CD, PsO, PsA: data to early escape/rescue/crossover; ¶Cumulative treatment duration for each study agent was calculated as the time from first to last dose across all relevant periods; **Active tuberculosis events are identified by the MedDRA HLT of 'Tuberculous infections' excluding the preferred term of 'Latent tuberculosis'; ††Malignancies are defined as the narrow terms in the MedDRA SMQ of 'Malignant Tumours'; ‡‡The majority of malignancies reported in patients aged ≥60 years were non-melanoma skin cancers, primarily basal cell carcinoma and squamous cell carcinoma; §§Opportunistic infections are defined as the narrow terms in the MedDRA SMQ of 'Opportunistic Infections'; ‖‖MACE were identified by clinical review; ¶¶VTE terms are based on customised MedDRA query.

CD, Crohn's disease; CI, confidence interval; GUS, guselkumab; HLT, high-level terms; IBD, inflammatory bowel disease; IL, interleukin; MACE, major adverse cardiovascular event; MedDRA, Medical Dictionary for Regulatory Activities; MOA, mechanism of action; PBO, placebo; PsA, psoriatic arthritis; PsO, psoriasis; PY, patient-year; RCT, randomised controlled trial; SCS, summary of clinical safety; SMQ, standardised MedDRA Queries; TA, therapy area; TEAE, treatment-emergent adverse event; VTE, venous thromboembolic event; UC, ulcerative colitis.

Faye AS, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. P1069. Full prescribing information: www.swissmedicinfo-pro.ch.

Conclusions

- Among adults aged ≥60 years with CD, UC, PsO or PsA pooled from 14 RCTs, GUS demonstrated safety outcomes that were similar to, or lower than, those observed with placebo
  - Exposure-adjusted rates of TEAEs, SAEs, infections, MACEs and VTEs were numerically lower with GUS compared with placebo
  - No deaths or active tuberculosis cases were reported with GUS
  - Findings were generally consistent with those observed in the overall study population
- Overall, GUS showed a favourable safety profile in adults aged ≥60 years, aligned with its established safety profile

CD, Crohn's disease; GUS, guselkumab; IL, interleukin; MACE, major adverse cardiovascular event; MOA, mechanism of action; PsA, psoriatic arthritis; PsO, psoriasis; SAE, serious adverse event; RCT, randomised controlled trial; TA, therapy area; TEAE, treatment-emergent adverse event; UC, ulcerative colitis; VTE, venous thromboembolic event.

Faye AS, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. P1069. Full prescribing information: www.swissmedicinfo-pro.ch.

Paediatric IBD

Safety and efficacy of UST in paediatric UC: Results from the Phase 3 UNIFI Jr study[1,2]

Background

The UNIFI Jr study (NCT04630028) evaluated efficacy and safety of UST in paediatric participants with moderately-to-severely-active UC.

Methods

112 participants (2 to <18 years; weight ≥10kg); moderately-to-severely active UC (baseline Mayo score ≥6, Mayo endoscopy subscore ≥2 and inadequate response or intolerant to conventional/biologic therapy or corticosteroid-dependent) received a single open-label, IV ustekinumab induction dose. At Week 8, 109 participants were randomised in a 1:1 ratio stratified by weight (<40kg/≥40kg) and Week 8 clinical response status (decrease from BL in Modified Mayo score ≥30% and ≥2 points with decrease from BL rectal bleeding subscore ≥1 or rectal bleeding subscore ≤1) to receive blinded SC UST maintenance therapy q8w/q12w for 44-weeks. UST dosing was based on body-surface-area (<40kg) or weight-tier (≥40kg). Primary endpoints were clinical remission (Mayo subscores: stool ≤1 without an increase from BL, rectal bleeding 0, endoscopy 0–1 with no friability present) at Week 8 and at Week 52 in those with induction response at Week 8.

Results

Among 112 participants (median [IQR] age 14.0 [11.0–15.5] years; 54.5% female; 60.7% biologic-naive); median (IQR) PUCAI score 55.0 (45.0–60.0), 91.7% moderate UC, median (IQR) Mayo score 8.0 (7.0–9.0), and 67.0% extensive UC. At Week 8, 79 participants achieved clinical response (induction responders). At Week 52, 32 of 79 (40.5% [95% CI: 30.4%–51.5%]) clinical responders achieved clinical remission, 52 (65.8%) achieved symptomatic remission, 51 (64.6%) were in clinical remission by PUCAI (score <10), 32 (40.5%) achieved endoscopic improvement, 32 (40.5%) were corticosteroid-free for ≥90 days, and 29 (36.7%) had histologic-endoscopic mucosal improvement. Remission rate was higher in participants without prior biologic failures (25 [47.2%; 95% CI: 34.4%–60.3%]) compared to those with biologic failure (7 [26.9%; 95% CI: 13.7%–46.1%]); not having previously failed biologic therapy was associated with higher Week 52 remission rates. Remission rates were similar from Week 8 to Week 52 in all weight subgroups. Both q8w and q12w maintenance regimens were efficacious. During maintenance therapy, SAEs occurred in 6.4% (7/109) of participants, most commonly reported SAEs were GI disorders (UC). AE rates were similar between q8w/q12w groups and treatment-emergent SAEs occurred in 9.3% (5/54) and 3.6% (2/55) of participants in q8w/q12w groups, respectively.

Conclusion

UST induction and maintenance therapy was effective in treating paediatric participants aged 2 to <18 years with moderate-to-severe paediatric UC. UST was well-tolerated with no new safety signals.

AE, adverse event; BL, baseline; CI, confidence interval; GI, gastrointestinal; IL, interleukin; IQR, interquartile range; IV, intravenous; MOA, mechanism of action; OL, open-label; PUCAI, Paediatric Ulcerative Colitis Activity Index; qXw, every X weeks; RDB, randomised double blind; SAE, serious adverse event; SC, subcutaneous; UC, ulcerative colitis; UST, ustekinumab.

1. De Greef E, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. P1154; 2. De Greef E, et al. *J Crohns Colitis* 2026;20:Supplement 1; jjaf231-1335. Ustekinumab is not approved for pediatric patients in the indication IBD by Swissmedic. Detailed information: www.swissmedicinfo-pro.ch

Exposure optimisation substudy (EOS) of ustekinumab in paediatric ulcerative colitis (UC): Q4W results from the Phase 3 UNIFI Jr study[1,2]

Background

To examine efficacy, safety, and PK of UST in paediatric patients with moderate-to-severe UC in the UNIFI Jr (NCT04630028) EOS.

Methods

Participants (N=112; 2 to <18 years; weight ≥10kg); moderate-to-severe UC, inadequate response/intolerant to prior treatment or corticosteroid-dependent) received one open-label IV UST induction dose in UNIFI Jr. At Week 8, participants were randomised 1:1 to blinded maintenance SC UST q8w or q12w for 44-weeks. Participants who lost response after Week 16 or were Week 16 induction nonresponders with low steady-state trough UST concentrations (<1.4µg/mL) were eligible for entry in an optional EOS (≥16 weeks) with q4w dosing. Participants were evaluated at Week 16 for clinical response (partial Mayo score) and clinical remission (PUCAI score).

Results

Of 109 participants randomised to maintenance, 97 were responders. 21 (19.3%) went to a q4w dosing in the EOS (4/109 [3.7%] were induction non-responders by Week 16; 17/97 [17.5%] had confirmed loss of response and low UST levels during maintenance period). Key baseline demographics of the 21 participants treated in the EOS: median [IQR] age was 14.0 [9.0; 15.0] years; 12/21 (57.1%) had prior biologic failure; all participants had extensive colitis; 12/21 (57.1%) and 16/21 (76.2%) had elevated CRP and Fcal, respectively. 16 of 21 participants (76.2%) completed the 16-week EOS and 11/13 (84.6%) achieved clinical response (by partial Mayo score); 9/16 (56.3%) achieved clinical remission (by PUCAI score) at EOS Week 16. Average PUCAI scores and partial Mayo scores from initiation of EOS decreased over time. Before administration of UST q4w, median (mean) serum UST concentration was 0.38 (0.97) µg/mL. Following q4w dosing, UST concentrations in paediatric participants increased to within the observed range of adults with UC receiving 90 mg q8w (serum trough UST concentrations ranged from 0.86 to 7.18 µg/mL at substudy Week 12 in UNIFI Jr when this approximated steady-state). AE(s) were reported in 81.0% of participants (57.1% ≥1 infection, none serious) and 9.5% had SAE(s). Safety with SC UST q4w dosing was generally consistent with the main study; no new or unexpected safety concerns were identified.

Conclusion

Participants enrolled in EOS responded favourably to UST q4w dosing leading to both increased clinical response and remission associated with higher serum drug levels. Overall safety was comparable to that previously reported with UST. Trough levels of UST in participants treated in the EOS were within the range of those in adults receiving q8w dosing.

AE, adverse event; CRP, C-reactive protein; EOS, Exposure Optimisation Substudy; Fcal, faecal calprotectin; IL, interleukin; IQR, interquartile range; IV, intravenous; MOA, mechanism of action; OL, open-label; PK, pharmacokinetics; PUCAI, Paediatric Ulcerative Colitis Activity Index, qXw, every X weeks; RDB, randomised double blind; SAE, serious adverse event; SC, subcutaneous; UC, ulcerative colitis; UST, ustekinumab.

1. De Greef E, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. P1010; 2. De Greef E, et al. *J Crohns Colitis* 2026;20:Supplement 1;jjaf231-1191. Ustekinumab is not approved for pediatric patients in the indication IBD by Swissmedic. Detailed information: www.swissmedicinfo-pro.ch

CH_CP-568830 02/26

Dose escalation in participants with primary/secondary loss of response to conventional dosing of UST in CD (UNITI Jr study)[1,2]

Background

Limited data exist on the safety and efficacy of shortening UST maintenance intervals in paediatric patients with CD who experience an initial lack of response or subsequent loss of response. This analysis examines UST efficacy, safety, and PK in paediatric participants from the UNITI Jr (NCT04673357) EOS.

Methods

Participants (n=101; ≥2–<18 years; PCDAI >30; inadequate response/intolerant to prior treatment or corticosteroid-dependent) received 1 open-label IV dose of UST and were randomised 1:1 at Week 8 to blinded maintenance SC UST q8w or q12w for 44-weeks. Those who had low steady-state trough UST concentrations (<1.4µg/mL) and who either were Week 16 induction non-responders or lost response after Week 16 were eligible for entry to an optional 16-week EOS with q4w dosing.

Results

Of randomised participants in the main study (n=97), 26 (26.8%) enrolled in the EOS with median age (IQR) 14.0 (12.0; 16.0) years; 21/26 (80.8%) had prior biologic failure; 14/26 (53.8%) had PCDAI >40 at baseline of the EOS. While 21 (80.8%) completed ≥16-weeks of EOS treatment, 20 completed the sPCDAI assessment at Week 16 of the EOS. 10/20 (50.0%) and 19/20 (95.0%) achieved clinical remission and response, respectively; at Week 16 of the EOS and the median/mean (range) sPCDAI score was 10.0/12.5 (0; 45); change-from-baseline was −40.0/−37.5 (−70; 5). Laboratory parameters of inflammation and faecal lactoferrin improved from EOS baseline to Week 16 and were similar to Week 52 of the main study. Median (IQR) UST concentration increased from 0.71µg/mL (0.37; 1.02) at the start of the EOS to 3.92µg/mL (2.08; 4.74) at Week 16, falling within the observed Week 16 range in adults with CD receiving a q8w regimen (median 2.05µg/mL [1.00; 3.70]). AEs were recorded in 73.1% of participants (of those, 38.5% were GI-related) and 11.5% had SAEs. Safety was generally consistent with the main UNITI Jr study; no new safety issues were identified.

Conclusion

Paediatric participants who were non-responders or who lost response to UST in the UNITI Jr study responded favourably to switching to qw4 dosing with an increased proportion of participants achieving clinical response and remission, plus improvements in inflammatory markers after 16 weeks. Q4w dosing resulted in higher serum concentrations that were within the exposure range observed in adult CD UST studies. The safety profile of UST in these participants was similar to that in the overall UNITI Jr study and the known profile in adults.

AE, adverse event; CD, Crohn's disease; EOS, Exposure Optimisation Substudy; GI, gastrointestinal; IL, interleukin; IQR, interquartile range; IV, intravenous; MOA, mechanism of action; OL, open-label; PCDAI, Paediatric Crohn's Disease Activity Index; PK, pharmacokinetics; qXw, every X weeks; RDB, randomised double blind; SAE, serious adverse event; SC, subcutaneous; sPCDAI, short PCDAI; UST, ustekinumab.

1. Russell R, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. P0849; 2. Russell R, et al. J *Crohns Colitis* 2026;20:Supplement 1; jjaf231.1030. Ustekinumab is not approved for pediatric patients in the indication IBD by Swissmedic. Detailed information: www.swissmedicinfo-pro.ch

CH_CP-568830 02/26

The UNITI Jr Study: Safety and efficacy results of ustekinumab in paediatric patients with Crohn's disease[1,2]

Background

UST therapy induced and maintained response and remission in adult patients with moderate-to-severe CD in the IM-UNITI program. The UNITI Jr study (NCT04673357) evaluated efficacy and safety of UST in paediatric participants with moderately-to-severely-active CD.

Methods

101 participants (≥2–<18 years; PCDAI score >30; inadequate response/intolerant to conventional/biologic therapy or corticosteroid-dependent) received 1 open-label IV UST (induction) dose. At Week 8, 97 participants were randomised (1:1) stratified by baseline body weight (<40kg/≥40kg) and response status (response: PCDAI decline ≥12.5 points with a total PCDAI score not >30/non-responder PCDAI decline <12.5) to receive blinded SC UST maintenance therapy every 8- or 12-weeks (q8w/q12w) for 44-weeks. UST dosing was based on body-surface-area (for participants <40kg) or weight-tiered (for participants ≥40kg). Primary endpoints were clinical remission (PCDAI ≤10) at Week 8, and at Week 52 among randomised, induction responders. Analysis was modified intent-to-treat (only includes responders at Week 8 before randomisation).

Results

In UNITI Jr, 101 participants were enrolled (59.4% male; median [IQR] age 14.0 [12.0–15.0] years; 42.6% biologic-naive; median [IQR] PCDAI score 40.0 [35.0–45.0]); at Week 8, 47 (46.5%) participants achieved clinical remission. Among participants who were in clinical remission at Week 8, 32/47 (68.1%) maintained clinical remission at Week 52. At Week 8, 85 (84.2%) achieved clinical response, of whom at Week 52, 46 (54.1% [95% CI: 43.6%–64.3%]) achieved clinical remission and 45 (52.9% [95% CI: 42.4%–63.2%]) achieved corticosteroid-free clinical remission. Not having previously failed biologic therapy was associated with higher Week 52 remission rates. Remission rates were similar from Week 8 to Week 52 in all weight subgroups. During maintenance, the efficacy was similar between q8w and q12w groups. AE/SAE rates were similar between q8w/q12w groups. SAEs occurred in 16.8% (17/101) of participants, of which CD exacerbation was most frequent (5/17); serious infections occurred in 5.9%.

Conclusion

UST was well-tolerated with no new safety signals. Induction and maintenance therapy with UST is effective in treating moderate-to-severe paediatric CD.

AE, adverse event; CD, Crohn's disease; CI, confidence interval; EOS, end of study; IL, interleukin; IQR, interquartile range; IV, intravenous; MOA, mechanism of action; OL, open-label; PCDAI, Paediatric Crohn's Disease Activity Index; qXw, every X weeks; RDB, randomised double blind; SAE, serious adverse event; SC, subcutaneous; UST, ustekinumab.

1. Turner D, et al. Presented at ECCO, Stockholm, Sweden, 18–21 February 2026. OP18; 2. Turner D, et al. *J Crohns Colitis* 2026;20:Supplement 1; jjaf231.018. Ustekinumab is not approved for pediatric patients in the indication IBD by Swissmedic. Detailed information: www.swissmedicinfo-pro.ch

Tremfya® Abbreviated Information for Professionals

TREMFYA® (Guselkumab, human IgG1λ mAb) solution for injection for subcutaneous or intravenous use.
I: Indicated for the treatment of adult patients with moderate to severe chronic plaque psoriasis (PsO) who are candidates for systemic therapy, active psoriatic arthritis (PsA) alone or in combination with methotrexate, who have an inadequate response or have been intolerant to prior disease-modifying antirheumatic drug (DMARD) therapy, and for the treatment of moderate to severe active ulcerative colitis (UC) or Crohn's disease (CD) who have an inadequate response, lost response, or were intolerant to either conventional therapy or a biologic treatment. **D:** Administration under the supervision and guidance of a physician experienced in the treatment of these diseases. Self-injection is possible after training. For psoriasis and psoriatic arthritis, the recommended dose is 100 mg as a s.c. injection at week 0 and 4, followed by every 8 weeks (q8w). For ulcerative colitis or Crohn's disease, the induction dose is 200 mg i.v. or alternatively 400 mg s.c. at week 0, 4 and 8, followed by a maintenance dose of 100 mg s.c. from week 16 onwards q8w. Alternatively, a maintenance dose of 200 mg as a s.c. injection every 4 weeks (q4w) may be considered at the physician's discretion from week 12. If there is no response after 12 weeks (UC and CD), 16 weeks (PsO) or 24 weeks (PsA), consider discontinuation of therapy. **CI:** Severe hypersensitivity to active substance/ excipients. Clinically relevant active infections (e.g. active tuberculosis). **VM:** In case of severe infections: monitor the patient closely and discontinue treatment until the infection resolves. No live vaccines during therapy. TB screening before therapy, if necessary, prophylactic treatment for latent TB. Discontinue therapy in case of severe hypersensitivity reactions. **AEs:** Very common: respiratory tract infections; common: herpes simplex infections, tinea infections, gastroenteritis, headache, diarrhea, increased transaminases, arthralgia, injection site reactions, hypersensitivity, urticaria, rashes. See product information for further AEs. **IA:** No clinically relevant interactions observed to date. **Packaging:** solution for injection in pre-filled syringe, pen, or pen - PushPen (100 mg/ 1 ml or 200 mg/ 2 ml); concentrate for solution for infusion in vial (200 mg/ 20 ml). **Tariff category:** B.

Detailed information: www.swissmedic.ch or www.swissmedicinfo.ch

Marketing Authorization Holder: Janssen-Cilag AG, Gubelstrasse 34, 6300 Zug (CP-564852 02/26)

Ulcerative colitis at 40x magnification

Stelara® Abbreviated Information for Professionals

Stelara® (ustekinumab, human monoclonal IgG1κ-antibody**) I: Plaque psoriasis:** Adults and children and adolescents from 6 years of age and older with moderate to severe plaque psoriasis who did not respond to other systemic therapies or PUVA or for whom these were contraindicated or not tolerated. **Psoriatic arthritis:** Adults with active psoriatic arthritis, as monotherapy or in combination with methotrexate when the response to a previous therapy with DMARDs has been insufficient. Stelara® improves physical functional ability in patients with psoriatic arthritis. **Crohn's disease:** Adults with moderate to severe active Crohn's disease whose response to conventional therapies or treatment with a TNFα antagonist was inadequate, or who are no longer responding to treatment, for whom such therapy is contraindicated or was not tolerated. **Ulcerative colitis:** Adults with moderate to severe active ulcerative colitis whose response to conventional therapies or treatment with a biologic was inadequate, or who are no longer responding to treatment, for whom such treatment is contraindicated or was not tolerated. **D:** Use under the direction and supervision of a physician experienced in the diagnosis and treatment of this therapeutic area; after proper training, also s.c. self-administration. Psoriasis (adult): 45 mg s.c. at weeks 0, 4, then every 12 weeks; patients > 100 kg: 90 mg; no response after 28 weeks: discontinue therapy. Psoriasis (children and adolescents ≥ 6 years of age): s.c. according to body weight: 0.75 mg/kg (< 60 kg), 45 mg (60-100 kg), 90 mg (> 100 kg) at weeks 0, 4, then every 12 weeks; no response after 28 weeks: discontinue therapy. Psoriatic arthritis (adults): 45 mg s.c. at weeks 0, 4, then every 12 weeks; with insufficient response: 90 mg; no response after 28 weeks: discontinue therapy. Crohn's and colitis (adult): i.v. induction according to body weight: 260 mg (≤ 55 kg), 390 mg (56-85 kg) or 520 mg (> 85 kg). 90 mg s.c. after 8 weeks, then every 12 or 8 weeks; no response after 16 weeks under dosage every 8 weeks: consider discontinuing. **CI:** Severe hypersensitivity to the active substance or any of the excipients. Clinically relevant, active infection. **SP:** Test for tuberculosis infection before starting therapy and initiate anti-tuberculosis therapy first in case of latent TB. Do not administer any live vaccines during treatment. Combination with immunosuppressants only partially studied and not studied with phototherapy. Avoid intensive sunlight exposure. Caution with regard to malignant tumours, hypersensitivity reactions, allergen specific immunotherapy, reversible posterior leukoencephalopathy syndrome. Watch for symptoms of erythrodermic psoriasis or exfoliative dermatitis when conducting psoriasis follow-up examinations; if a drug reaction is suspected, discontinue Stelara®. **ADR:** *Very common:* none. *Common:* infection of the upper respiratory tract, nasopharyngitis, sinusitis, dizziness, headache, pain in the oropharynx, diarrhoea, nausea, vomiting, pruritus, back pain, muscle pain, arthralgia, exhaustion, erythema and/or pain at the injection site. For further ADRs, see Compendium. **IA:** No interaction studies conducted. **P/L:** Use in pregnant women only when clearly necessary. In the decision about stopping breastfeeding or discontinuing Stelara® therapy, the benefit of breastfeeding for the child and the benefit of Stelara® therapy for the mother should be considered. **Pack sizes:** Solution for injection in vial (45 mg in 0.5 mL), ready-to-use syringe or pre-filled pen (45 mg in 0.5 mL or 90 mg in 1 mL). Concentrate for solution for infusion (130 mg in 26 mL). **Subject to reimbursement by health insurance funds. Dispensing category:** B. **Detailed information:** www.swissmedic.ch or www.swissmedicinfo.ch

Marketing Authorisation Holder: Janssen-Cilag AG, a Johnson & Johnson company, Gubelstrasse 34, 6300 Zug (CH-CP-432851_12/23)

References can be obtained from the licence holder Janssen-Cilag AG.

Ulcerative colitis at 40x magnification