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Biomarker-Driven Insights From the Phase 2a AFFINITY Study Evaluating Guselkumab + Golimumab Combination Therapy Versus Guselkumab Monotherapy in Psoriatic Arthritis

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Conflicts of Interest

IBM: Served as a consultant for AbbVie, Amgen, AstraZeneca, Bristol Myers Squibb, Cabaletta Bio, Compugen, Dextera Biosciences, Eli Lilly, Gilead, GlaxoSmithKline, Johnson & Johnson, Moonlake, Novartis, Pfizer, Roche, Sanofi, and UCB; received grant/research support from Amgen, AstraZeneca, Bristol Myers Squibb, Eli Lilly, GlaxoSmithKline, Johnson & Johnson, Novartis, Roche, and UCB; is a share-option holder of Cabaletta Bio, Causeway Therapeutics, Compugen, Dextera Biosciences, and Montai Therapeutics; and is a trustee of Arthritis UK.

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ERS: Served as a consultant for AbbVie, Johnson & Johnson, Novartis, and Roche; received grant/research support from AbbVie, Johnson & Johnson, Novartis, Pfizer, Roche, and UCB; and speaker fees from Amgen, Bristol Myers Squibb, Eli Lilly, Johnson & Johnson, Novartis, Pfizer, Roche, and UCB.

JFM: Served as a consultant and/or investigator for AbbVie, Amgen, AstraZeneca, Biogen, Boehringer Ingelheim, Bristol Myers Squibb , Dermavant, Eli Lilly, Galderma, Johnson & Johnson, Moonlake, Novartis, Oruka, Pfizer, Regeneron, Sanofi, Sun Pharma, and UCB.

PB: Served as a consultant for AbbVie, Amgen, AstraZeneca, Bristol Myers Squibb, Eli Lilly, Johnson & Johnson, Novartis, Pfizer, Sanofi-Regeneron, and UCB; received grant/research support: AbbVie, Amgen, AstraZeneca, Bristol Myers Squibb, Eli Lilly, Johnson & Johnson, Novartis, Pfizer, Sanofi-Regeneron, and UCB; speakers bureau for AbbVie, Eli Lilly, Gilead, Johnson & Johnson, Merck, Pfizer, and UCB.

VC: Received honoraria from AbbVie, Bristol Myers Squibb, Eli Lilly, Fresenius Kabi, Johnson & Johnson, Novartis, Takeda, and UCB; received grant/research support from AbbVie, Amgen, and Eli Lilly; spouse is an employee of AstraZeneca; and is supported by the Dr. Dafna D. Gladman Chair in Psoriatic Arthritis Research, a joint Hospital-University Named Chair between the University of Toronto, the University Health Network, and the UHN Foundation.

LSG: Served as a consultant for Altis Medicines, Eli Lilly, Johnson & Johnson, Novartis, and UCB; received grant/research support from UCB.

PJM: Received consulting fees from AbbVie, Amgen, Bristol Myers Squibb, Century, Cullinan, Eli Lilly, Inmagene, Johnson & Johnson, Merck, Moonlake, Novartis, Pfizer, Spyre, Sun Pharma, Takeda, and UCB; received grants from AbbVie, Amgen, Bristol Myers Squibb, Eli Lilly, Johnson & Johnson, Moonlake, Novartis, Sana, Takeda, and UCB; received speaker fees from AbbVie, Amgen, Eli Lilly, Johnson & Johnson, Novartis, and UCB.

JUS: Received grant/research support from Johnson & Johnson and Pfizer; received consulting fees from Bristol Myers Squibb, Johnson & Johnson, Novartis, Pfizer, and UCB.

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Background

Psoriatic arthritis (PsA) is a complex, multi-pathway inflammatory disease in which combination therapies may enhance efficacy by modulating complementary immune mechanisms[1,2]

Guselkumab (GUS)[3]

- Fully human, dual-acting monoclonal antibody that selectively inhibits interleukin (IL)-23 by targeting its p19 subunit and binding CD64 on IL-23-producing inflammatory monocytes[4]

Golimumab (GOL)[5]

- Fully human monoclonal antibody targeting tumor necrosis factor (TNF)

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[1] Cuchacovich R. *J Rheumatol.* 2012;39:187-193. [2] Scher JU. *Arthritis Rheumatol.* 2021;73:1574-1578. [3] Tremfya (guselkumab) [Prescribing Information]. Available from: https://www.accessdata.fda.gov/drugsatfda_docs/label/2025/761061s027lbl.pdf. Accessed March 2026. [4] Sachen K. *Front Immunol.* 2025;1532852. [5] Simponi (golimumab) [Prescribing Information]. Available from: https://www.accessdata.fda.gov/drugsatfda_docs/label/2011/125289s0064lbl.pdf. Accessed March 2026.

AFFINITY Study Design (NCT05071664)

Key Inclusion Criteria

- Adults ≥18 to ≤65 years
- TNFi-IR[a]
- PsA diagnosis for ≥6 months; CASPAR requirement met; ≥1 PsA subset class[b]
- Active PsA (≥3 TJC and ≥3 SJC)
- Active plaque PsO (≥1 plaque ≥2 cm in diameter or psoriatic nail changes)

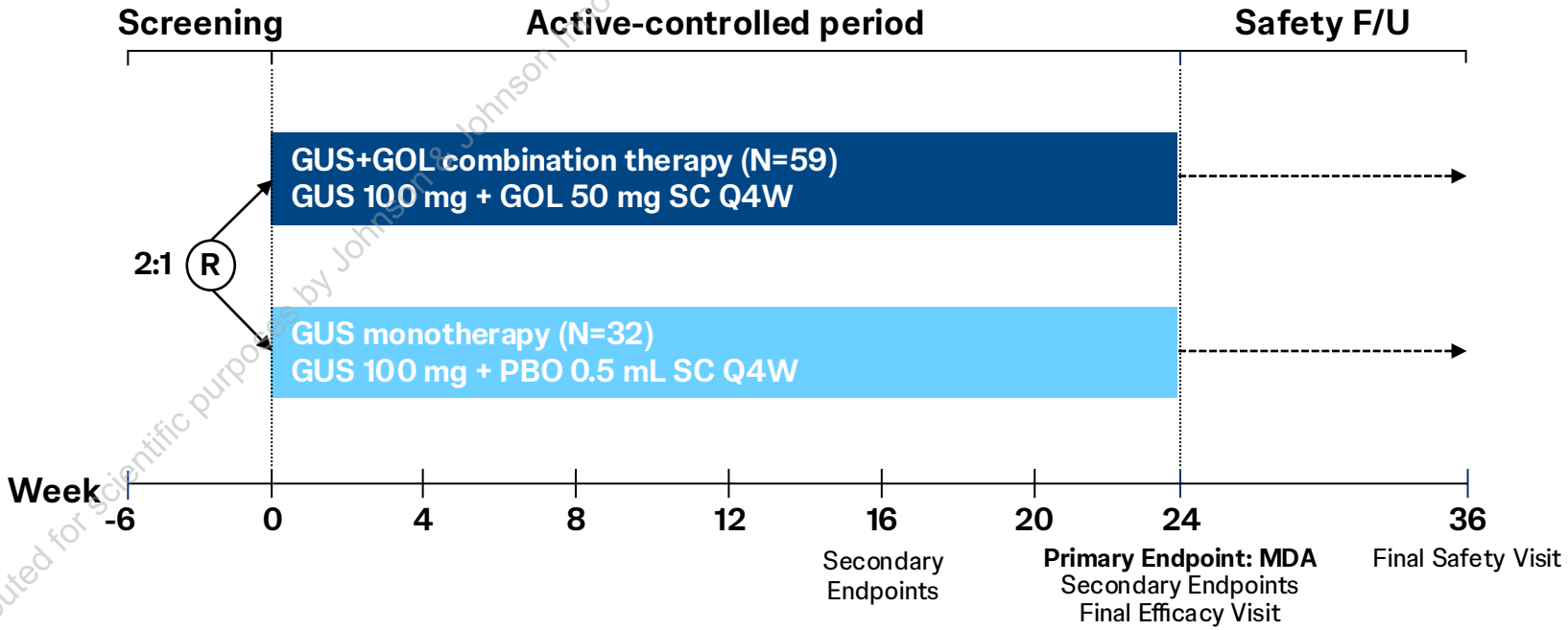
Protocol Amendment

Changes to inclusion criteria to mitigate significant impact of Ukraine/Russia crisis on study recruitment:

- Removed screening high sensitivity CRP ≥0.3 mg/dL criterion
- Expanded IR to prior TNFi from 1 to 2

Phase 2a, randomized, double-blind, active-controlled, proof-of-concept study

- Evaluated GUS+GOL combination vs GUS monotherapy in adults with active PsA and TNF inhibitor inadequate response (TNFi-IR)



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[a]TNFi-IR defined as lack of benefit in response after ≥12 weeks of etanercept, adalimumab, certolizumab pegol (or their biosimilars), or ≥14 weeks of infliximab/biosimilars, with last TNFi dose administered >5 half-lives prior to starting study treatment. [b]Subset classification including distal interphalangeal joint involvement, polyarticular arthritis without rheumatoid nodules, asymmetric peripheral arthritis, or spondylitis with peripheral arthritis. **CASPAR**=CIASsification criteria for Psoriatic ARthritis, **CRP**=C-reactive protein, **F/U**=follow up, **IR**=inadequate response, **MDA**=minimal disease activity, **PBO**=placebo, **Q4W**=every 4 weeks, **R**=randomization, **SC**=subcutaneous, **SJC**=swollen joint count, **TJC**=tender joint count, **TNFi**=tumor necrosis factor inhibitor, **W**=Week.

Baseline characteristics were generally well balanced across treatment groups

- Approximately 1/3 of participants had baseline CRP <0.3 mg/dL

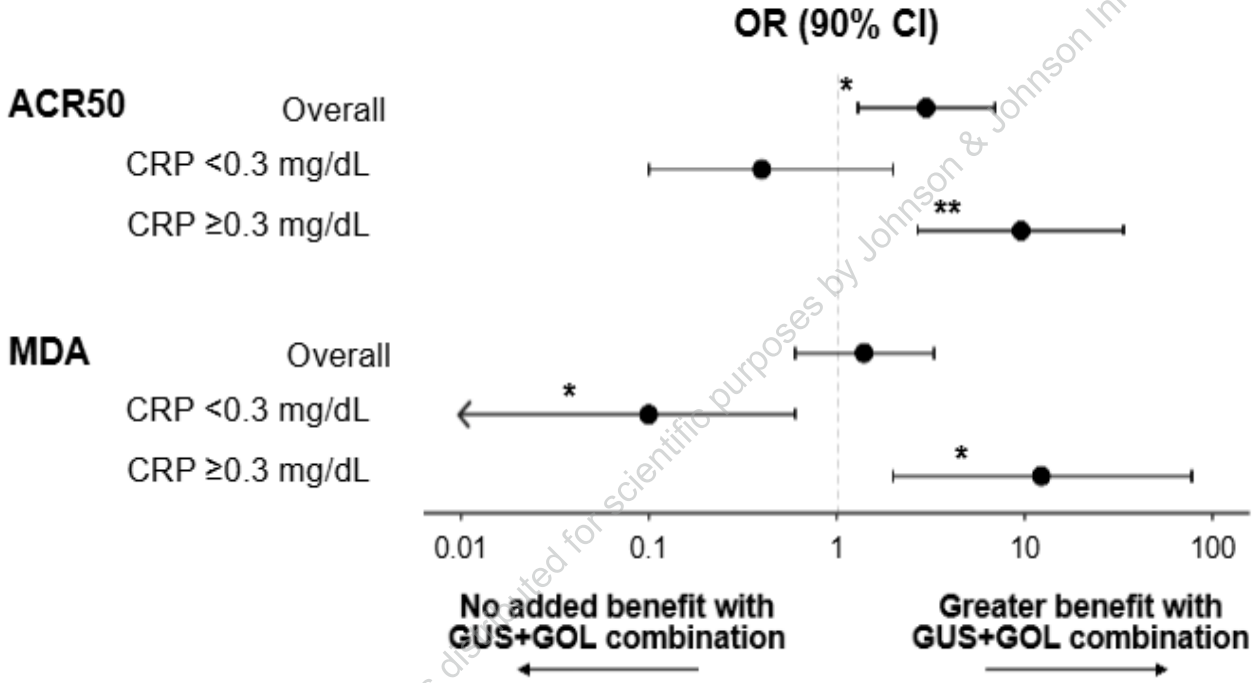
Baseline Characteristics	GUS+GOL combination (N=59)	GUS monotherapy (N=32)
Demographics		
Age, years	50.2 (10.5)	47.7 (10.3)
Female	61%	62%
Race, White/Not reported or unknown	97% / 3%	100% / 0%
BMI, kg/m^2	31.4 (6.8)	30.5 (6.4)[a]
Clinical Characteristics		
PsA duration, years	8.2 (7.0)	7.2 (7.0)
SJC (0-66), median (range)	7.0 (3; 50)	8.0 (3; 37)
TJC (0-68), median (range)	13.0 (3; 63)	12.0 (3; 66)
Enthesitis per LEI	59%	66%
LEI (1-6)[b]	2.8 (1.7)	2.4 (1.4)
Dactylitis per DSS	22%	25%
DSS (1-60)[c]	9.3 (12.5)	9.6 (14.3)
Screening CRP (Week -6)		
< / ≥ 0.3 mg/dL	32% / 68%	31% / 69%
Baseline CRP (Week 0)		
< / ≥ 0.3 mg/dL	29% / 71%	34% / 66%
CRP, mg/dL	0.8 (1.3)	1.4 (3.3)
PsO duration, years	13.4 (11.2)	10.0 (8.7)
≥3% psoriatic BSA and IGA ≥2	37%	41%
PASI[d] (0-72), median (range)	5.1 (0.8; 29.4)[e]	4.0 (0.5; 48.2)[f]

Data are mean (SD) unless noted otherwise. [a]N=31. [b]Among participants with available assessment and LEI >0. [c]Among participants with available assessment and DSS >0. [d]Among participants with ≥3% psoriatic BSA and IGA ≥2 at baseline. [e]N=22. [f]N=13. **BSA**=body surface area, **BMI**=body mass index, **CRP**=C-reactive protein, **DSS**=Dactylitis Severity Score, **GOL**=golimumab, **GUS**=guselkumab, **IGA**=Investigator's Global Assessment, **LEI**=Leeds Enthesitis Index, **PASI**=Psoriasis Area and Severity Index, **PsA**= psoriatic arthritis, **PsO**=psoriasis, **SJC**=swollen joint count, **TJC**=tender joint count.

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Clinical findings from the AFFINITY study

- **Primary analyses suggested that GUS+GOL combination may offer clinically meaningful benefits in joint disease activity and physical function, particularly among participants with elevated CRP[1]**
  - No additional benefits were observed in PsO/enthesitis/dactylitis; no new safety signals



	GUS monotherapy n/N (%)	GUS+GOL combination n/N (%)	Interaction *P* value[a]
ACR50 Overall	7/32 (21.9)	26/59 (44.1)	
CRP <0.3 mg/dL	4/10 (40.0)	4/19 (21.1)	0.007
CRP ≥0.3 mg/dL	3/22 (13.6)	22/40 (55.0)	
MDA Overall	7/32 (21.9)	17/59 (28.8)	
CRP <0.3 mg/dL	6/10 (60.0)	4/19 (21.1)	0.002
CRP ≥0.3 mg/dL	1/22 (4.5)	13/40 (32.5)	

[1]Scher JU. *Arthritis Rheumatol*. 2026; doi:10.1002/art.70152. *Nominal $p<0.05$; **$p\leq0.01$ versus GUS monotherapy. [a]*P* value for two-way interaction of treatment group with CRP level.
ACR50=≥50% improvement in American College of Rheumatology response criteria, **CI**=confidence interval, **CRP**=C-reactive protein, **GOL**=golimumab, **GUS**=guselkumab, **MDA**=minimal disease activity, **OR**=odds ratio, **PsO**=psoriasis.

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Objective



- The current analysis utilized the AFFINITY trial to assess:
  - Pharmacodynamic effects *and*
  - Biomarker relationships with clinical responses

  Among TNFi-IR participants with PsA treated with GUS+GOL combination therapy or GUS monotherapy

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GOL=golimumab, **GUS**=guselkumab, **PsA**=psoriatic arthritis, **TNFi-IR**=tumor necrosis factor inhibitor inadequate response.

Biomarker Assessments & Analyses

Assessments	Analyses

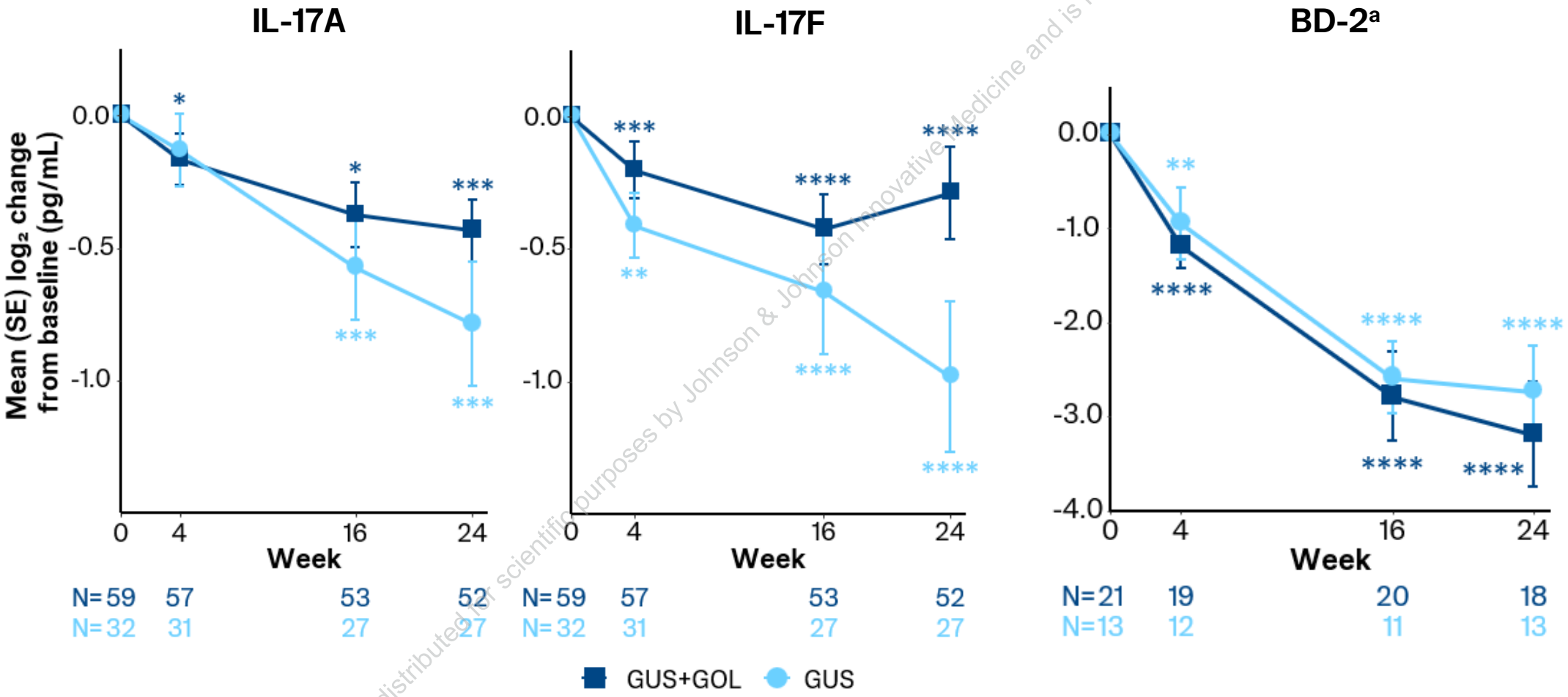
PD effects and disease marker levels evaluated at Weeks 0, 4, 16, and 24
• IL-17A, IL-17F (serum Th17 cytokines) by Single Molecule Counting immunoassays
• CRP, IL-6 (general inflammation markers) by MSD immunoassays
• BD-2 (PsO activity marker) by custom MSD immunoassay
• TNF by TNF activity score using whole blood RNA sequencing[1]
	Wilcoxon rank-sum exact test for changes from baseline in:
	• TNF activity (mean GSVA enrichment score):
	  • Within regimens
	  • For GUS+GOL vs GUS monotherapy
	• Other biomarkers levels (mean $\log_2$):
	  • Within regimens
	  • For GUS+GOL vs GUS monotherapy

Clinical response–biomarker relationships; statistically significant findings presented for: • MDA and ACR50 at Week 24 (clinical endpoints) • IL-17A, IL-17F, CRP, IL-6, BD-2 (serum biomarkers)	**Wilcoxon rank-sum exact test** for associations of clinical response with biomarker level: • At baseline ($\log_2$; within regimens) • Change from baseline (mean $\log_2$; within regimens)

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[1]Desai P. Presented at United European Gastroenterology (UEG) Week; October 8-11, 2022; Vienna, Austria.
ACR50=≥50% improvement in American College of Rheumatology response criteria, **BD-2**=Beta-Defensin-2, **CRP**=C-reactive protein, **GSVA**=gene set variation analysis, **MDA**=minimal disease activity, **MSD**=Meso-Scale Discovery, **PD**=pharmacodynamic **PsO**=psoriasis, **RNA**=ribonucleic acid, **Th17**=T helper 17 cells, **TNF**=tumor necrosis factor.

Both regimens significantly reduced serum IL-17A/F and BD-2 levels through W24

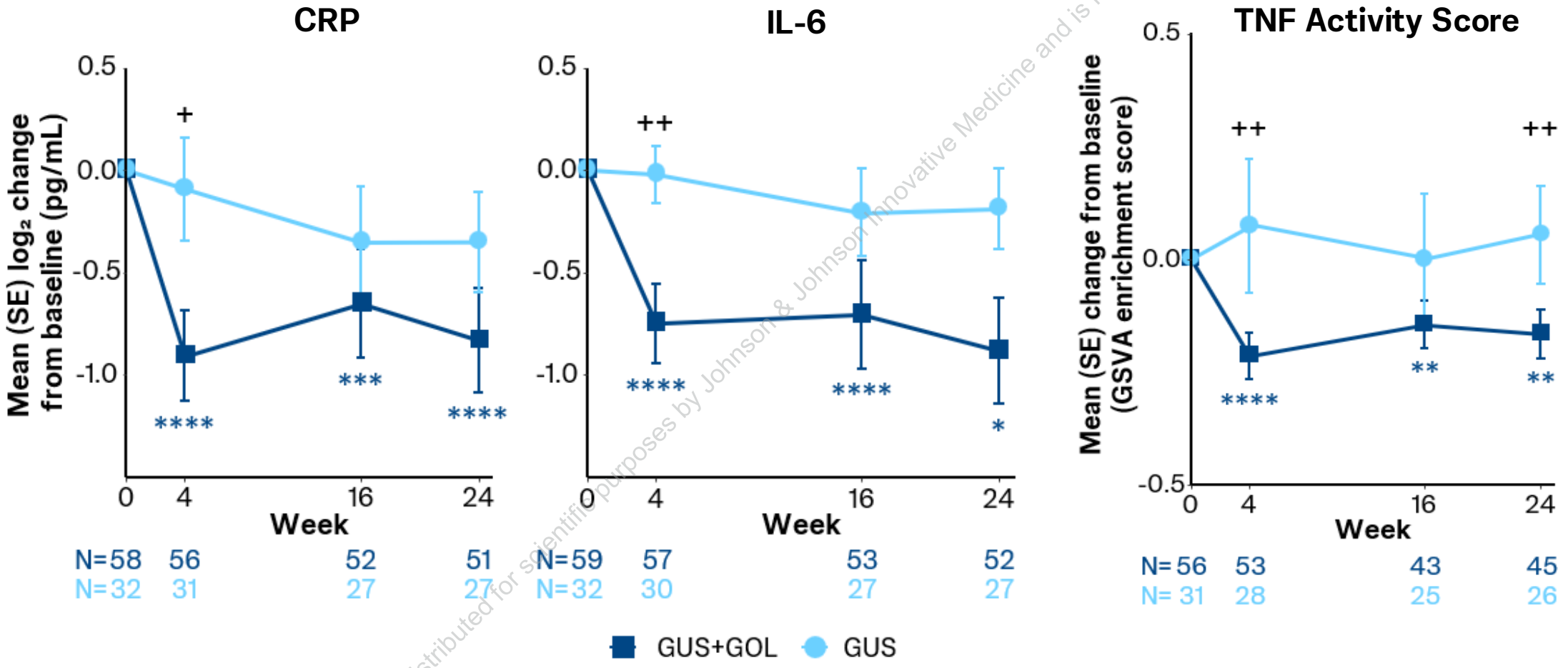


***p*≤0.05, ****p*≤0.01, *****p*≤0.001, ******p*≤0.0001 vs baseline within regimen.

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[a]Among participants with ≥3% BSA PsO involvement and an IGA score ≥2 at baseline. **BD-2**=Beta-Defensin-2, **BSA**=body surface area, **GOL**=golimumab, **GUS**=guselkumab, **IGA**=Investigator's Global Assessment, **IL**=interleukin, **PsO**=psoriasis, **SE**=standard error, **W**=Week.

GUS+GOL combination significantly reduced serum CRP and IL-6 levels, and TNF activity score from baseline as early as W4, through W24



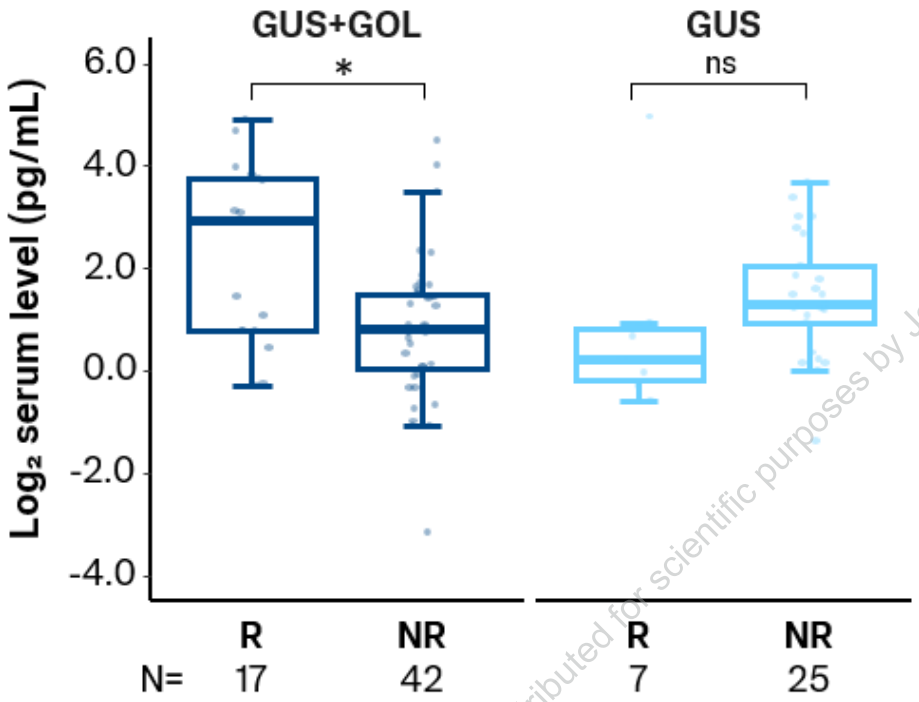
*$p \leq 0.05$, **$p \leq 0.01$, ***$p \leq 0.001$, ****$p \leq 0.0001$ vs baseline within regimen. +$p \leq 0.05$,++$p \leq 0.01$ for GUS+GOL combination vs GUS monotherapy.

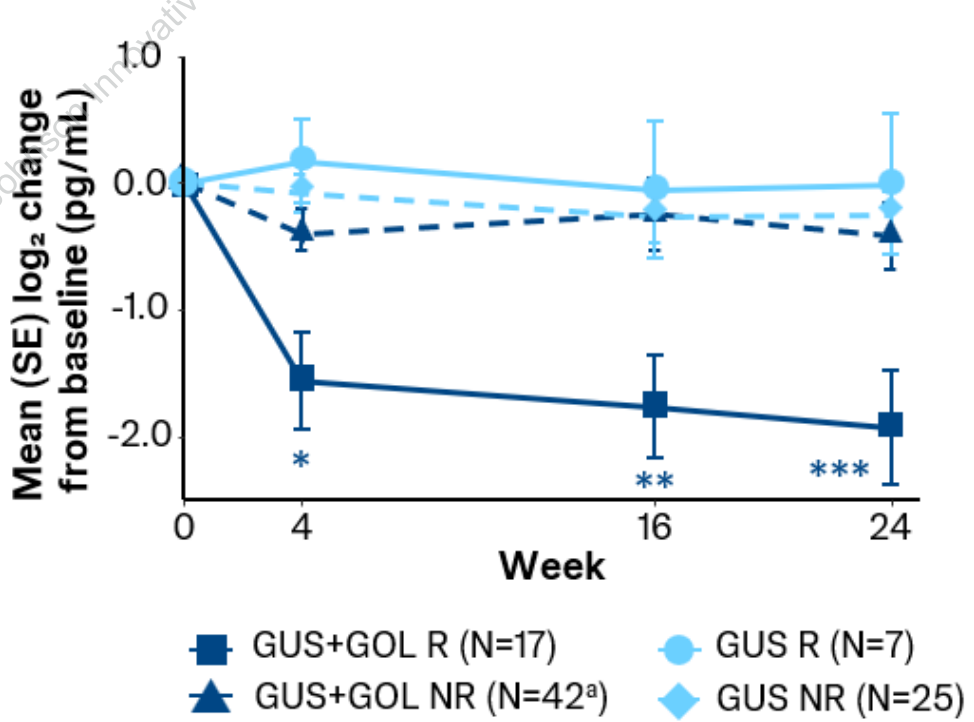
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CRP=C-reactive protein, **GOL**=golimumab, **GSVA**=gene set variation analysis, **GUS**=guselkumab, **IL**=interleukin, **SE**=standard error, **TNF**=tumor necrosis factor, **W**=Week.

In the GUS+GOL combination group, W24 MDA responders had higher baseline levels and greater reductions in serum IL-6 through W24 vs nonresponders

- In the GUS monotherapy group, no differences in baseline IL-6 levels or in IL-6 reduction from baseline through W24 were observed between W24 MDA responders vs nonresponders





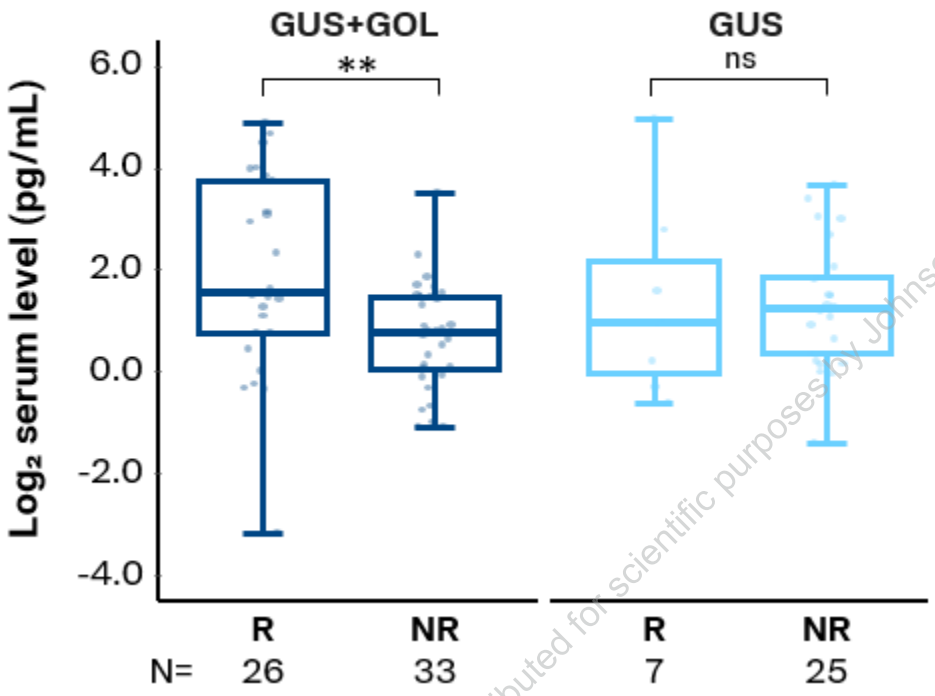
*$p \leq 0.05$, **$p \leq 0.01$, ***$p \leq 0.001$ for MDA R vs NR within regimen.

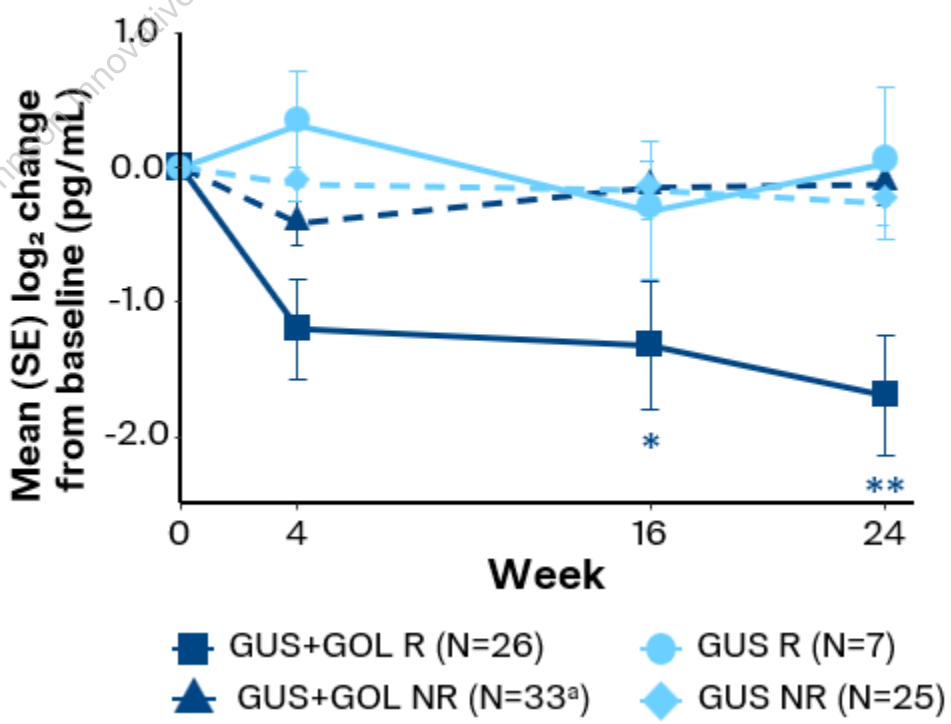
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[a]N=41 for W24 GUS+GOL NR. **GOL**=golimumab, **GUS**=guselkumab, **IL**=interleukin, **MDA**=minimal disease activity, **NR**=nonresponders, **ns**=not significant, **R**=responders, **SE**=standard error, **W**=Week.

In the GUS+GOL combination group, W24 ACR50 responders had higher baseline levels and greater reductions in serum IL-6 through W24 vs nonresponders

- In the GUS monotherapy group, no differences in baseline IL-6 levels or in IL-6 reductions from baseline through W24 were observed between W24 ACR50 responders vs nonresponders





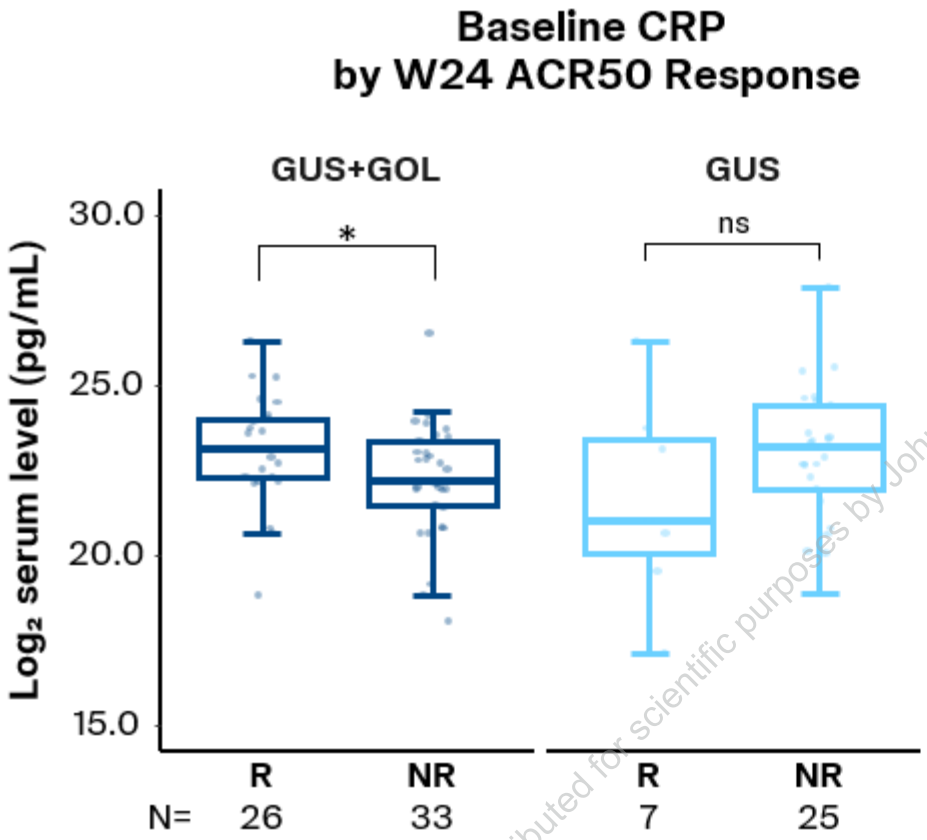
***p*≤0.05, ****p*≤0.01 for ACR50 R vs NR within regimen.**

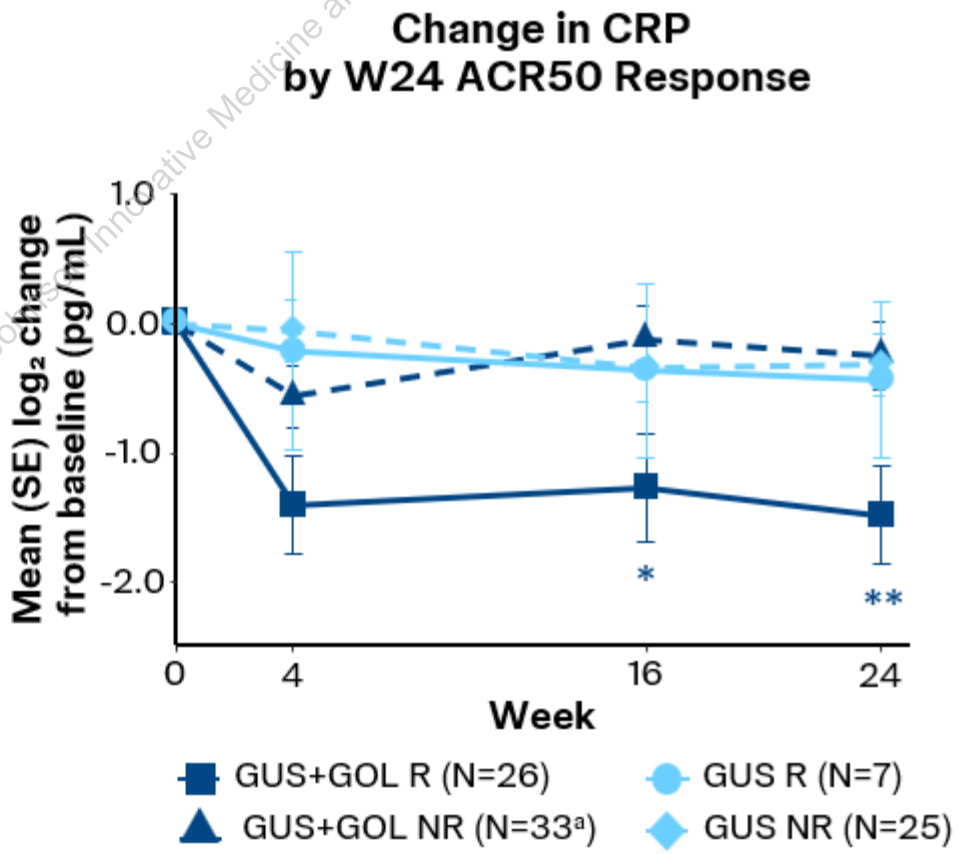
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[a]N=32 for W24 GUS+GOL NR. **ACR50**=≥50% improvement in American College of Rheumatology response criteria, **CRP**=C-reactive protein, **GOL**=golimumab, **GUS**=guselkumab, **IL**=interleukin, **NR**=nonresponders, **ns**=not significant, **R**=responders, **SE**=standard error, **W**=Week.

In the GUS+GOL combination group, W24 ACR50 responders had higher baseline levels and greater reductions in serum CRP through W24 vs nonresponders

- In the GUS monotherapy group, no differences in baseline CRP levels or in CRP reductions from baseline through W24 were observed between W24 ACR50 responders vs nonresponders





***p*≤0.05, ****p*≤0.01 for ACR50 R vs NR within regimen.**

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[a]N=32 for W24 GUS+GOL NR. **ACR50**=≥50% improvement in American College of Rheumatology response criteria, **CRP**=C Reactive Protein, **GOL**=golimumab, **GUS**=guselkumab, **NR**=nonresponders, **ns**=not significant, **R**=responder, **SE**=standard error, **W**=Week.

Key Takeaways

In the proof-of-concept, phase 2a AFFINITY study of TNFi-IR participants with active PsA:

- ✓ Consistent with clinical findings suggesting benefit in joint disease activity and physical function (particularly with elevated CRP),[1] GUS+GOL combo showed:
  - ✓ Significant reductions in serum CRP and IL-6 levels, and TNF activity scores through W24
  - ✓ W24 MDA and ACR50 responders had higher baseline levels and greater reductions in IL-6 and CRP
- ✓ GUS+GOL combo and GUS mono elicited similar reductions in Type 17 cytokines and BD-2, consistent with comparable improvements in psoriasis/enthesitis/dactylitis with the addition of a TNFi[1]

Participants with TNFi-IR PsA and elevated inflammatory biomarkers may derive additional joint-related benefits with GUS+GOL combination therapy

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[1] Scher JU. *Arthritis Rheumatol*. 2026; doi:10.1002/art.70152. **ACR50**=≥50% improvement in American College of Rheumatology response criteria, **BD-2**=Beta-Defensin-2, **CRP**=C-reactive protein, **IL**=interleukin, **GOL**=golimumab, **GUS**=guselkumab, **MDA**=minimal disease activity, **PsA**=psoriatic arthritis, **TNF**=tumor necrosis factor, **TNFi**=TNF inhibitor, **TNFi-IR**=TNFi inadequate response, **W**=Week.