Guselkumab Efficacy on Improvements in ACR Response Components in Participants With Active Psoriatic Arthritis and Inadequate Response to One Prior TNFi

Philip J Mease[1,2], Alice B Gottlieb[3], Jose U Scher[4], Joseph F Merola[5], Kimberly Parnell Lafferty[6], Soumya D Chakravarty[6,7], Yanli Wang[6], Fowzia Ibrahim[8], Yevgeniy Krol[6], Stacey Fitch[6], Anthony Todd[6], Alexis Ogdie[9], Christopher T Ritchlin[10]

[1]Rheumatology Research, Providence Swedish Medical Center, Seattle, WA, USA; [2]University of Washington School of Medicine, Seattle, WA, USA; [3]UT Southwestern Medical Center, Department of Dermatology, Dallas, TX, USA; [4]New York University Grossman School of Medicine, New York, NY, USA; [5]UT Southwestern Medical Center and O'Donnell School of Public Health, Department of Dermatology and Department of Medicine, Division of Rheumatology, Dallas, TX, USA; [6]Johnson & Johnson, Horsham, PA, USA; [7]Drexel University College of Medicine, Philadelphia, PA, USA; [8]Johnson & Johnson, High Wycombe, UK; [9]University of Pennsylvania School of Medicine, Philadelphia, PA, USA; [10]University of Rochester Medical Center, Rochester, NY, USA

Scan the QR code.
The QR code is intended to provide scientific information for individual reference, and the information should not be altered or reproduced in any way.

Background

- Guselkumab (GUS), a fully human, dual-acting,[1] monoclonal antibody inhibiting the interleukin (IL)-23p19 subunit, has demonstrated multidomain efficacy in participants (pts) with active psoriatic arthritis (PsA)[2]
- SOLSTICE is a phase 3b, multicenter, randomized, placebo (PBO)-controlled study evaluating GUS 100 mg every 4 weeks (Q4W) and Q8W in pts with active PsA and inadequate response (IR; inadequate efficacy or intolerance) to 1 prior tumor necrosis factor inhibitor (TNFi)[3]
  - At W24, GUS-treated pts had significantly greater improvements in PsA signs and symptoms vs PBO

Objective

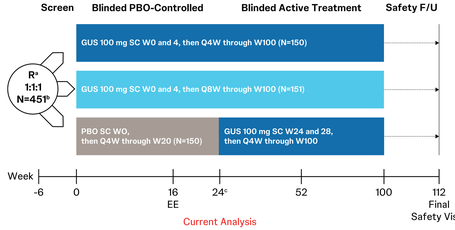
To evaluate the efficacy of GUS in improving the individual American College of Rheumatology (ACR) response components through W24 of SOLSTICE

Methods

Key inclusion criteria:

- Age ≥18 years
- Active PsA (≥3 SJC; ≥3 TJC; CRP ≥0.3 mg/dL); CASPAR criteria met
- **Inadequate efficacy and/or intolerance with 1 prior TNFi**
- History of or active PsO (≥1 plaque ≥2 cm and/or nail PsO)

SOLSTICE Study Design



Endpoints

- ACR20
- ACR50
- ACR70
- Proportions achieving ≥20%/≥50%/≥70% improvements in ACR components
  - SJC
  - TJC
  - Pt Pain
  - PtGA
  - PGA
  - HAQ-DI
  - CRP

Statistical Analyses

- **Pts were considered nonresponders** through W24 if they discontinued study agent for any reason other than ND/MD, increased dose/initiated csDMARDs or oral corticosteroids, or initiated protocol-prohibited PsA therapies
- **Data impacted by ND/MD were imputed using MI; other missing data were imputed using NRI.** Response rates through W24 shown are the average proportion achieving response, over 200 MI datasets
- ACR20 at W24 was multiplicity-controlled; ACR20 at W16, ACR50 at W16 and 24, and ACR70 at W24 were weakly controlled (not nominal). Response rates for improvement in individual ACR components were determined post hoc; p values are nominal.

[a]Randomization was stratified by baseline use of csDMARDs. [b]Total number randomized=453, the full analysis set of 451 excludes 1 pt who was double randomized. [c]Crossover. [d]Final safety F/U at W112 is 12W after final study agent administration. **ACR20/50/70**=≥20%/≥50%/≥70% in ACR response criteria, **CASPAR**=CIASsification criteria for Psoriatic ARthritis, **CRP**=C-reactive protein, **csDMARDs**=conventional synthetic disease-modifying antirheumatic drugs, **EE**=early escape, **F/U**=follow-up, **HAQ-DI**=health assessment questionnaire disability index, **MD**=major disruption: involving Ukraine and neighboring countries/territories beginning 24 February 2022, **MI**=multiple imputation, **ND**=natural disaster: site closure, site access restrictions, or lockdowns due to the COVID-19 pandemic, **NRI**=nonresponder imputation, **PGA**=Physician's global assessment, **PtGA**=patient's global assessment (arthritis), **R**=randomization, **SC**=subcutaneous, **SJC**=Swollen joint count, **TJC**=Tender joint count.

Key Takeaways

- **In SOLSTICE, GUS demonstrated improvement after 1 or 2 doses in both clinician-assessed and patient-reported outcomes across all ACR response components**
- **Similar efficacy through W24 was observed between the 2 GUS dosing regimens in TNFi-IR pts**

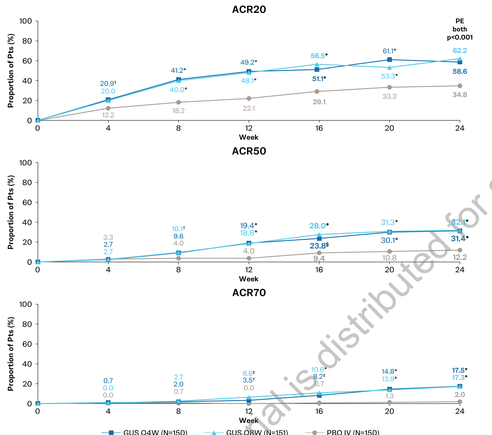
Results

Baseline demographics, disease characteristics, and concomitant medications were well balanced among treatment groups

		GUS 100 mg Q4W	GUS 100 mg Q8W	PBO
Pts, N		150	151	150
Demographics				
	Age, years	50.6 (13.3)	51.9 (12.9)	49.2 (12.6)
	Female	50.0%	51.0%	56.7%
	Weight, kg	86.5 (20.8)	89.5 (19.8)	85.9 (21.1)
ACR Components				
	PsA disease duration, years	8.8 (8.3)	8.3 (7.5)	7.0 (6.6)
	SJC [0-66]	10.7 (7.8)	10.3 (6.6)	10.2 (6.4)
	TJC [0-68]	18.1 (12.6)	17.1 (11.2)	16.8 (11.6)
	Pt Assessment of Pain [VAS; 0-10cm]	6.1 (2.0)	6.2 (2.0)	6.2 (2.0)
	PtGA (arthritis) [VAS; 0-10cm]	6.1 (2.1)	6.2 (2.1)	6.2 (1.9)
	PGA [VAS; 0-10cm]	6.5 (1.5)	6.7 (1.6)	6.5 (1.6)
	HAQ-DI [0-3]	1.3 (0.6)	1.3 (0.6)	1.3 (0.6)
	CRP, mg/dL	1.2 (1.6)	1.3 (1.7)	1.4 (1.9)
Concomitant medications				
	csDMARDs	58.7%	57.0%	56.0%
	Methotrexate	50.0%	45.7%	48.0%

Data reported as mean (standard deviation) unless otherwise noted. **VAS**=Visual analog scale.

At W24, significantly greater proportions of pts in the GUS Q4W and Q8W groups vs PBO achieved ACR20, ACR50, and ACR70[‡]



p<0.001, †p=0.001, ‡p<0.01 †p<0.05 vs PBO. ACR20 at W24 was multiplicity-controlled; ACR20 at W16, ACR50 at W16 and 24, and ACR70 at W24 were weakly controlled (not nominal). All other p-values are nominal. **PE**=primary endpoint.

Across all ACR components, response rates for achieving ≥20%, ≥50%, and ≥70% improvement were greater for GUS Q4W/Q8W-treated pts vs PBO at W24

- Separation from PBO (nominal p<0.05) was observed early as W4-8 (≥20% and ≥50% improvements), and W8-12 (≥70% improvement)

≥20% Improvement in ACR Components

GUS Q4W						
Week	4	8	12	16	20	24
SJC	61.7†	75.9†	79.8†	81.1*	83.1*	83.9*
TJC	48.9	69.5*	75.2*	76.0*	79.5*	79.6*
Pt Pain	41.5†	56.1*	55.9†	58.9*	64.2*	62.1†
PtGA	44.9†	53.8*	58.8*	61.5*	59.3†	62.0†
PGA	49.8†	65.2*	74.4*	79.8*	82.0*	78.6*
HAQ-DI	39.4‡	48.5*	48.6	51.3†	58.4†	56.9†
CRP	40.5	47.7†	45.1†	49.2†	52.4*	48.1‡

GUS Q8W						
Week	4	8	12	16	20	24
SJC	57.0	75.5*	78.5*	89.1*	86.0*	87.1*
TJC	48.2	67.0*	72.8*	80.5*	83.9*	82.6*
Pt Pain	36.8‡	50.1*	51.9†	60.3*	57.0†	60.6†
PtGA	36.4	52.6*	50.5†	60.6*	58.7*	64.4*
PGA	47.4‡	69.7*	71.8*	80.3*	81.1*	83.8*
HAQ-DI	32.9	42.8‡	42.6	51.0	51.9‡	54.0†
CRP	49.6†	53.4*	60.3*	55.4*	47.8‡	51.7†

PBO						
Week	4	8	12	16	20	24
SJC	49.7	59.2	63.6	63.4	64.9	65.1
TJC	40.3	47.0	52.9	54.2	55.3	58.6
Pt Pain	23.5	22.1	36.5	38.4	40.4	44.6
PtGA	26.8	24.9	33.2	38.5	40.9	44.6
PGA	35.7	37.8	46.9	47.1	54.2	55.5
HAQ-DI	27.6	28.9	39.2	39.8	39.6	38.4
CRP	35.3	30.6	32.8	30.1	29.1	33.9

nominal *p<0.001, †p<0.01, ‡p<0.05

≥50% Improvement in ACR Components

GUS Q4W						
Week	4	8	12	16	20	24
SJC	39	54.4†	62.8†	69.3*	75.1*	77.3*
TJC	19.1	40.5‡	52.7*	54.0*	63.9*	67.3*
Pt Pain	9.5	19.0*	26.3*	29.1†	36.0*	36.8†
PtGA	11.5‡	18.9†	28.0*	28.4‡	38.0†	40.0*
PGA	20.3	38.4*	49.6*	53.8*	55.0*	57.1*
HAQ-DI	14.8	23.9†	22.9‡	28.3†	32.3†	30.4
CRP	24.9	26.8	29.8‡	33.1†	33.5*	31.7‡

GUS Q8W						
Week	4	8	12	16	20	24
SJC	33.3	49.5‡	62.2‡	74.6*	77.3*	76.5*
TJC	18.5	40.9‡	51.1*	61.3*	67.3*	72.7*
Pt Pain	12.2	22.1*	26.4*	29.1†	33.2*	33.4†
PtGA	8.9	20.0†	25.0†	30.4‡	33.1†	34.7*
PGA	19.7	35.8*	42.0*	55.3*	61.5*	65.6*
HAQ-DI	11.6	15.6	20.7	25.4	30.3†	33.7†
CRP	28.5‡	33.3†	35.9*	38.9*	31.4*	36.6†

PBO						
Week	4	8	12	16	20	24
SJC	30.0	35.4	44.4	48.3	48.0	51.6
TJC	17.8	27.2	30.4	32.7	33.7	37.1
Pt Pain	6.2	4.2	10.0	15.0	16.6	22.8
PtGA	4.8	8.2	11.1	17.1	20.6	21.5
PGA	15.0	13.6	22.0	26.5	29.6	36.3
HAQ-DI	8.9	10.3	13.1	17.7	19.2	23.3
CRP	17.9	18.4	17.3	18.7	14.7	20.0

nominal *p<0.001, †p<0.01, ‡p<0.05

≥70% Improvement in ACR Components

GUS Q4W						
Week	4	8	12	16	20	24
SJC	20.2	33.0	39.5	49.5†	57.2*	62.7*
TJC	6.2	16.6	28.2	31.0	46.0*	50.6*
Pt Pain	3.4	9.0†	11.4*	16.3†	22.8*	27.0*
PtGA	2.0	7.7‡	9.1†	15.8‡	24.9*	24.3*
PGA	7.4	16.3*	21.6†	28.0*	37.6*	37.7*
HAQ-DI	7.4	10.2‡	12.6†	15.1‡	16.7‡	20.0‡
CRP	10.9	18.9	21.4‡	22.7‡	21.5†	20.2‡

GUS Q8W						
Week	4	8	12	16	20	24
SJC	19.1	32.2	47.0†	58.7*	63.5*	62.1*
TJC	13.0	21.8‡	31.5‡	40.4†	46.3*	49.0*
Pt Pain	4.7	7.6†	12.9*	16.7†	17.4†	22.3*
PtGA	2.7	5.5	10.3†	14.7†	19.4†	23.5*
PGA	8.1	17.2*	19.0‡	35.1*	39.4*	40.9*
HAQ-DI	4.0	7.4	6.9	12.0	16.0	18.7
CRP	15.2	18.7	22.3†	22.0‡	21.8†	24.9†

PBO						
Week	4	8	12	16	20	24
SJC	17.7	24.4	31.5	33.2	36.2	41.7
TJC	8.9	11.7	21.2	23.7	20.6	25.3
Pt Pain	1.4	2.0	1.5	4.8	5.6	7.7
PtGA	0.7	2.1	1.5	6.8	6.9	9.0
PGA	7.5	3.4	8.8	10.8	13.7	19.1
HAQ-DI	3.4	3.4	4.2	6.1	8.9	11.6
CRP	9.3	11.4	9.9	11.4	9.7	11.0

nominal *p<0.001, †p<0.01, ‡p<0.05

PRESENTED AT: CGR-East; April 30–May 3, 2026; Destin, Florida, USA. **PRESENTER:** Anthony Todd; employee of Johnson & Johnson and owns stock in Johnson & Johnson. **REFERENCES: 1.** Sachen KL. *Front Immunol.* 2025;16:1532852. **2.** Tremfya: Package insert. Horsham, PA: Janssen Biotech, Inc.; 2025. **3.** Ogdie A. *Arthritis Rheumatol.* 2026. doi: 10.1002/art.70092. **ACKNOWLEDGMENTS:** Medical writing support was provided by Teresa Tartaglione, PharmD, of Certara, Radnor, PA under the direction of the authors in accordance with Good Publication Practice guidelines (*Ann Intern Med.* 2022;175:1298–1304). Sponsored by Johnson & Johnson. **DISCLOSURES: PJM** received consulting fees from AbbVie, Amgen, Bristol Myers Squibb, Century, Cullinan, Eli Lilly, Immagene, Johnson & Johnson, Merck, Moonlake, Novartis, Pfizer, Spyre, SUN Pharma, Takeda, and UCB; grants from AbbVie, Amgen, Bristol Myers Squibb, Eli Lilly, Johnson & Johnson, Moonlake, Novartis, Sana, Takeda, and UCB; and speaker fees from AbbVie, Amgen, Eli Lilly, Johnson & Johnson, Novartis, and UCB. **ABG** received research/educational grants from Bristol Myers Squibb, Johnson & Johnson, Moonlake, and UCB (all paid to Mount Sinai School of Medicine until May 1, 2025); at UT Southwestern, Dr. Gottlieb is a sub-investigator on studies sponsored by Bristol Myers Squibb and Johnson & Johnson; and received honoraria as an advisory board member and consultant for AbbVie, Amgen, Bristol Myers Squibb, Eli Lilly, Johnson & Johnson, Novartis, Oruka, Sanofi, Sun Pharma, Takeda, Teva, and UCB. **JUS** received grant/research support from Johnson & Johnson and Pfizer and consulting fees from Bristol Myers Squibb, Johnson & Johnson, Novartis, Pfizer, and UCB. **JFM** is 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. **KPL, SDC, FI, YK, SF,** and **AT** are employees of Johnson & Johnson and own stock in Johnson & Johnson. **YW** is an employee of IQVIA providing statistical support (funded by Johnson & Johnson). **AO** is a consultant of AbbVie, Amgen, Bristol Myers Squibb, CorEvitas, Eli Lilly, Gilead, GlaxoSmithKline, Johnson & Johnson, Merck, Novartis, Oruka, Pfizer, Spyre, Takeda, and UCB; received advisory board fees from AbbVie, Amgen, Bristol Myers Squibb, Eli Lilly, Gilead, GlaxoSmithKline, Johnson & Johnson, Novartis, Pfizer, and UCB and grants from AbbVie, Pfizer, and Novartis (all to University of Pennsylvania) and Amgen (to Forward/NDB); and other funding from NIAMS, Rheumatology Research Foundation, National Psoriasis Foundation, University of Pennsylvania. **CTR** received grant/research support from AbbVie, Amgen, and UCB; and received consulting fees from AbbVie, Amgen, Eli Lilly, Gilead, Johnson & Johnson, Novartis, Pfizer, and UCB.

This material is distributed for scientific purposes on J&J Medical Connect, and is not for promotional use