# VISIBLE: GUSELKUMAB IMPACT ON PSORIATIC ARTHRITIS THROUGH WEEK 100 IN PARTICIPANTS WITH MODERATE-TO-SEVERE PSORIASIS AND UNCONTROLLED PSORIATIC ARTHRITIS ACROSS ALL SKIN TONES

Joseph F. Merola,[1] Richard G. Langley,[2] Maryam Shayesteh-Alam,[3] Andrea Nguyen,[4] Theodore Alkousakis,[5] Sarah Ofori,[5] Elizabeth Adamson,[5] Tony Ma,[5] Joe K. Tung,[6] Alice B. Gottlieb[1]

[1]UT Southwestern Medical Center, Dallas, TX, USA; [2]Dalhousie University, Halifax, Nova Scotia, Canada; [3]SimcoDerm Medical and Surgical Dermatology Centre, Barrie, Ontario, Canada; [4]First OC Dermatology, Fountain Valley, CA, USA; [5]Johnson & Johnson, Horsham and Spring House, PA, USA; [6]University of Pittsburgh Medical Center, Pittsburgh, PA, USA

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## BACKGROUND

- **Psoriasis** (PsO) remains **frequently misdiagnosed and undertreated** among individuals with **skin of color** (SoC)[1,2]
- Scalp PsO **is a risk factor** for development of **psoriatic arthritis (PsA)**
- **VISIBLE** is a Phase 3b study that enrolled participants who self-identified as **non-white** and have **moderate-to-severe plaque (Cohort A) or scalp (Cohort B) PsO**
- VISIBLE participants were evaluated for **PsA at screening** based on
  - **Rheumatologist-confirmed diagnosis of PsA**
  - **Psoriasis Epidemiology Screening Tool (PEST) score ≥3**
- Participants were considered to have **uncontrolled** PsA at baseline if they had a **12-item Psoriatic Arthritis Impact of Disease questionnaire (PsAID-12) score of >4.0**
  - The PsAID-12 patient-acceptable symptom state [PASS] is defined as a score of ≤4.0[3]

## OBJECTIVES/METHODS

**This post-hoc analysis reports PsAID-12 results through Week 100 for participants with uncontrolled PsA at baseline[a]**

### VISIBLE included participants who self-identified as non-white, across all skin tones

**Cohort A: 103 participants with moderate-to-severe plaque PsO**

BSA ≥10%, PASI ≥12, IGA ≥3

**Cohort B: 102 participants with moderate-to-severe scalp PsO**

SSA ≥30%, PSSI ≥12, ss-IGA ≥3, and ≥1 plaque outside of the scalp

### Study Design


### PsA Assessments

- **PsAID-12** was used for evaluating participants identified as having PsA at screening
- Self-reported assessment of physical, social, and psychological impact of PsA (score range, 0-10)[3,4]
  - **PASS** = score of ≤4.0
  - **MCII** = reduction of ≥3.0 points

[a]Efficacy analysis set: VISIBLE Cohorts A and B, 22.9% (n=47) had uncontrolled PsA, PsAID ≥4 at baseline. **BSA**=Body surface area; **GUS**=Guselkumab; **IGA**=Investigator's Global Assessment; **MCII**=Minimal clinically important improvement; **PASI**=Psoriasis Area and Severity Index; **PASS**=Patient Acceptable Symptom Score; **PBO**=Placebo; **PSSI**=Psoriasis Scalp Severity Index; **SSA**=Scalp surface area; **ss-IGA**=Scalp-specific IGA; **W**=Week.

## RESULTS

**At baseline, 29.8% (61/205) of VISIBLE Cohort A and B participants had PsA (full efficacy analysis set). Of these, 47 participants had either a history of rheumatologist-diagnosed PsA (n=19 [40.4%]) or PEST ≥3 at screening (n=28 [59.6%] with previously undiagnosed PsA) AND a PsAID-12 score >4.0 at baseline.**

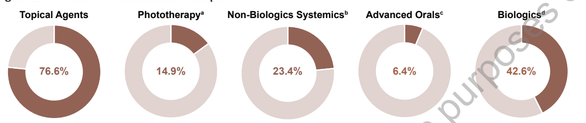
Baseline demographics and disease characteristics were generally comparable between PsA patients diagnosed by a rheumatologist and those with PsA identified by PEST screening (**Table 1**) and between GUS and PBO treatment groups (data not shown)

| Table 1. Baseline Characteristics | Rheumatologist-Confirmed Diagnosis of PsA (N=19) | Screening PEST Score ≥3 (N=28) |
|---|---|---|
| **Demographics** | | |
| **Age,** yrs | 45.6 (13.5) | 41.9 (12.5) |
| **Male** | 63% | 68% |
| **Race** | | |
| Hispanic | 32% | 54% |
| Black | 32% | 7% |
| Asian | 16% | 29% |
| Middle Eastern | 10% | 4% |
| Multi-racial | 10% | 4% |
| Pacific Islander or Native Hawaiian | 0% | 4% |
| **BMI,** kg/m$^2$ | 32.7 (9.3) | 34.8 (10.2) |
| **Fitzpatrick skin type** | | |
| I-III | 16% | 14% |
| IV-VI | 84% | 86% |
| **Disease Characteristics** | | |
| **PsO disease duration,** yrs | 12.6 (10.5) | 11.1 (8.3) |
| **IGA,** moderate (3) | 74% | 86% |
| **BSA,** % | 23.4 (17.6) | 23.8 (15.3) |
| **PASI (0–72)** | 18.7 (9.4) | 20.8 (8.1) |
| **PSSI (0-72)** | 31.4 (19.0) | 25.7 (13.2) |
| **PsAID-12 score** | 7.0 (1.4) | 7.9 (1.7) |

*Data shown are mean (SD), unless otherwise indicated.*
***BMI**=body mass index; **SD**=standard deviation.*

- Among the 47 participants with uncontrolled PsA at baseline, prior treatments included topicals (76.6%), phototherapy (14.9%), methotrexate (21.3%), apremilast (6.4%), and biologics (42.6% any biologic; tumor necrosis factor 21.3%, interleukin (IL)-17 19.1%, and IL-12/23 inhibitors 19.1%) (**Figure 1**)

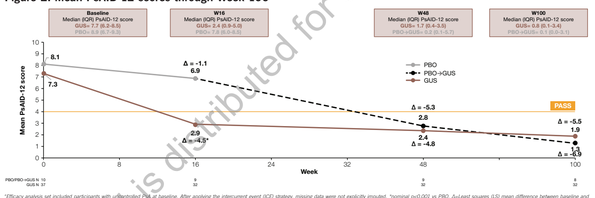
**Figure 1. Previous PsO Medications/Therapies**




*[a]Includes PUVA or UVB. [b]Includes PUVA, methotrexate, cyclosporine, acitretin. [c]Includes apremilast. [d]Includes etanercept, infliximab, adalimumab, certolizumab, brodalumab, ixekizumab, secukinumab, ustekinumab.*
***PUVA**=Psoralen plus ultraviolet A; **UVB**=Ultraviolet B.*

**The GUS-randomized group with uncontrolled PsA at baseline had significantly decreased (improved) mean PsAID-12 score at Week 16 compared to the PBO-randomized group; mean PsAID-12 scores for the GUS and PBO→GUS subgroups continued to improve through Week 100 (Figure 2)**

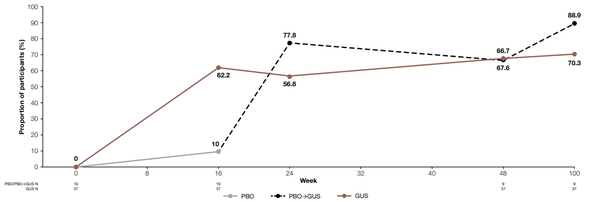
**Figure 2. Mean PsAID-12 scores through Week 100[a]**




*[a]Efficacy analysis set included participants with uncontrolled PsA at baseline. After applying the intercurrent event (ICE) strategy, missing data were not explicitly imputed. *nominal p<0.001 vs PBO. Δ=Least squares (LS) mean difference between baseline and Week 16 among participants with data at both timepoints. LS mean differences and p-values are based on an analysis of covariance model, with treatment group, baseline PsAID-12 score, and IGT (I-III or IV-VI) as covariates; all p-values are nominal as this is a post hoc analysis and also sample size is small (n=47) in PBO group. Participants who met treatment failure rules (discontinued study agent due to lack of efficacy, had worsening PsO, or initiated a prohibited psoriasis treatment prior to Week 16) were assigned a change from baseline=0. Missing data were not imputed.*

**At Week 100, more than 70% of GUS-treated participants with uncontrolled PsA at baseline achieved PASS (PsAID-12 score ≤4.0; Figure 3)**

**Figure 3. Achievement of PASS Through Week 100[a]**




*[a]Efficacy analysis set included participants with uncontrolled PsA at baseline. After applying the ICE strategy, missing data were not explicitly imputed. For participants who were randomized to PBO at Week 0, only those participants who crossed over to GUS at or after Week 16 were included.*

**Figure 4. Participant Who Achieved IGA 0/1, PASI 90, and PsAID-12=0 at Week 16 and maintained responses through Week 100**


**Figure 5. Participant Who Achieved IGA 0, PASI 100 (Complete Clearance), and PsAID-12=0 at Week 16 and Maintained Clear Skin Through Week 100**


## CONCLUSIONS

- At baseline, approximately one quarter of VISIBLE participants had uncontrolled PsA (based on having a PsAID-12 score above the PASS threshold of 4.0), indicating lack of awareness of the need for routine PsA screening by dermatologists, including patients across all skin tones
- Specifically, PsA screening with the PEST in patients with PsO, especially those with risk factors, should be considered to enable early detection, initiation of appropriate treatment, and improved outcomes in PsA
- In participants with uncontrolled and often undiagnosed PsA at baseline, treatment with GUS provided clinically meaningful improvements in the physical and psychological impact of joint disease, based on mean PsAID-12 scores decreasing below the PASS at Week 16
- Improvements in PsAID-12 scores continued and were maintained through 100 weeks of GUS treatment



**References: 1.** Alexis A, et al *J Drugs Dermatol.* 2022;21:1054–1060. **2.** Gottlieb AB, Merola JF. *J Dermatolog Treat.* 2022;33:1907–1915. **3.** Gossec, L, et al. *Ann Rheum Dis.* 2014;73:1012-9. 4. Holland R, et al. *J Psoriasis Psoriatic Arthritis.* 2020;5:12–22. **Acknowledgements:** 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;175:1298–304). Presented at The Masterclasses in Dermatology Annual Conference at the Ritz Carlton in Sarasota, FL on February 19-22, 2026. This poster was supported by Johnson & Johnson, Horsham, PA, USA. **Disclosures: JFM** is a consultant and/or investigator for AbbVie, Amgen, Astra-Zeneca, Biogen, Boehringer Ingelheim, Bristol Myers Squibb, Dermavant, Eli Lilly, Galderma, Johnson & Johnson, Moonlake, Novartis, Oruka, Pfizer, Regeneron, Sanofi, Sun Pharma, and UCB. **RGL** has received compensation in the form of grant funding and/or honoraria, as principal investigator for and has served on the scientific advisory board or served as a speaker for AbbVie, Amgen, Boehringer Ingelheim, Bristol Myers Squibb, Celgene, Eli Lilly, Johnson & Johnson, LEO Pharma, Merck, Novartis, Pfizer, SUN, Takeda, and UCB. **MS-A** has served or currently serving on the advisory boards, as a consultant, and/or speaker for AbbVie, Amgen, Bausch Health, Bristol Myers Squibb, Dermavant, Eli Lilly, Galderma, Incyte, Johnson & Johnson, Novartis, Pfizer, Sanofi/Regeneron, and UCB; serves as a principal investigator for clinical trials sponsored/conducted in collaboration with AbbVie, Aclaris, Alumis, Amgen, Avalo Therapeutic, Bristol Meyers Squibb, Citryl, Concert Pharmaceutical, Eli Lilly, Galderma, Incyte, Insmed, Johnson & Johnson, Moonlake Therapeutics, Novartis, Pfizer, Sanofi/Regeneron, Takeda, and UCB; and receives institutional research funding through SimcoDerm Medical & Surgical Dermatology Centre for industry-sponsored clinical trials. **AN** is a speaker, investigator, and/or consultant for AbbVie, Almirall, Amgen, Arcutis, ASLAN, Beiersdorf, Boehringer Ingelheim, Bristol Meyers Squibb, Dermira, Dermavant, EPI, Galderma, Incyte, ISDIN, Johnson & Johnson, Eli Lilly, Leo Pharma, Mayne, Novan, Novartis Pharmaceuticals, Ortho Dermatologics, Pfizer, Regeneron, Sanofi Genzyme, Sun Pharma, and UCB. **TA, SO, EA,** and **TM** are employees and stockholders of Johnson & Johnson. **JKT** is or has been a clinical trials investigator, consultant, or speaker for AbbVie, Amgen, Arcutis, Boehringer Ingelheim, Bristol Myers Squibb, Castle Biosciences, Eli Lilly, Galderma, Incyte, Johnson & Johnson, Novartis, Pfizer, Regeneron, Sanofi, Sun, Takeda, and UCB. **ABG** has 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 has 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.