VISIBLE COHORT B: GUSELKUMAB SCALP CLEARANCE AND LONG-TERM PATIENT-REPORTED OUTCOMES THROUGH WEEK 100 IN PARTICIPANTS WITH MODERATE-TO-SEVERE SCALP PSORIASIS ACROSS ALL SKIN TONES

A. McMichael,[1] T. Bhutani,[2] C. Kindred,[3,4] S. Smith,[5] T. Alkousakis,[6] O. Choi,[6] T. Ma,[7] R. Radusky,[8] J. Yeung,[9,10] G. Han,[11] S.C. Taylor[12]

[1]Wake Forest University School of Medicine, Winston-Salem, NC, USA; [2]Synergy Dermatology, San Francisco, CA, USA; [3]Kindred Hair & Skin Center Marriottsville, MD, USA; [4]Howard University College of Medicine, Washington, DC, USA; [5]California Dermatology & Clinical Research Institute, Encinitas, CA, USA; [6]Johnson & Johnson, Horsham, PA, USA; [7]Johnson & Johnson, Spring House, PA, USA; [8]Dermatology Treatment and Research Center, Dallas, TX, USA; [9]University of Toronto, Toronto, ON, CA; [10]Probity Medical Research, Waterloo, ON, CA; [11]Icahn School of Medicine at Mount Sinai, New York, NY, USA; [12]University of Pennsylvania, Philadelphia, PA, USA

Scan the QR code for the full digital poster

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

VISIBLE, a phase 3b, multicenter, randomized, double-blind, placebo (PBO)-controlled clinical trial, evaluated the efficacy and safety of guselkumab (GUS) in participants (pts) with moderate-to-severe plaque psoriasis (PsO) across a broad range of self-identified racial/ethnic groups and objectively measured phototypes

VISIBLE is comprised of 2 cohorts:

- **Cohort A:** pts with moderate-to-severe plaque PsO
- **Cohort B:** pts with moderate-to-severe scalp PsO

Scalp is the most commonly involved high-impact site in patients with moderate-to-severe plaque PsO, especially among those with skin of color (SOC)

Patients with PsO and SOC have reported a disproportionately greater impact of PsO on their health-related quality of life (HRQoL) compared to White pts[1,2]

OBJECTIVE/METHODS

Objective

Evaluate efficacy, safety, and impact of GUS treatment on pt-reported PsO symptoms and HRQoL in Cohort B through 2 years

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

Cohort A: 103 pts with moderate-to-severe plaque PsO

BSA ≥10%, PASI ≥12, IGA ≥3

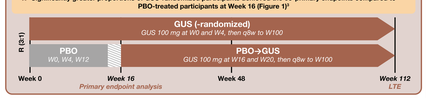
Cohort B: 108 pts with moderate-to-severe scalp PsO

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

Summary of VISIBLE Study

Coprimary Endpoints: ss-IGA 0/1 and PSSI 90 (Cohort B) at Week 16

☆ **Significantly greater proportions of GUS-randomized participants achieved the co-primary endpoints compared to PBO-treated participants at Week 16 (Figure 1)[3]**



Efficacy Outcomes Presented Through Week 100

(Response Rates Assessed From Weeks 0-100, Safety Assessments Evaluated From Weeks 0-112)

- **scalp-specific Investigator's Global Assessment (ss-IGA)**
  - ss-IGA 0/1 (clear/minimal)
  - ss-IGA 0 (clear)
- **Psoriasis Scalp Severity Index (PSSI)**
  - PSSI 90
  - PSSI 100
  - Mean percent improvement from baseline in PSSI
- **Scalp surface area (SSA)**
  - Mean percent improvement from baseline in SSA
- **Safety**
  - AEs and SAEs

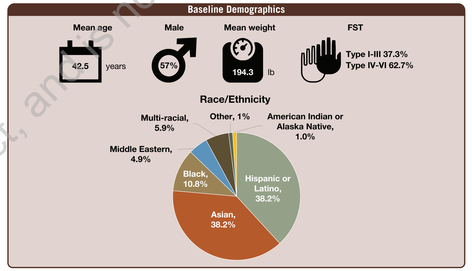
HRQoL Outcomes Presented Through Week 100

- **Dermatology Life Quality Index (DLQI)**
  - DLQI 0/1 (no effect)
- **Scalp Itch Numerical Rating Scale (NRS)**
  - Mean NRS scores over time
- **Psoriatic Arthritis Impact of Disease-12 (PsAID-12)**
  - Mean PsAID-12 scores over time
- **Psoriasis Symptoms and Signs Diary (PSSD)**
  - Mean symptoms scores over time
  - Symptoms score of 0

AE=Adverse event; BSA=Body surface area; IGA=Investigator's Global Assessment; PASI=Psoriasis Area and Severity Index; q8w=Every 8 weeks; SAE=Serious adverse event; W=Week.

COHORT B BASELINE DEMOGRAPHICS AND DISEASE CHARACTERISTICS (N=102)

Baseline Demographics

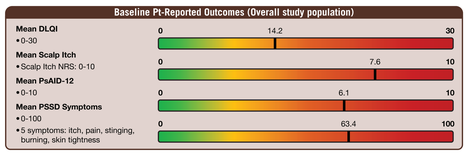


FST=Fitzpatrick Skin Type.

Baseline Disease Characteristics

	PBO (N=26)	GUS (N=76)
PsO Duration, y	11.3 (12.8)	11.3 (9.8)
ss-IGA, n (%)		
Moderate [3]	20 (76.9)	64 (84.2)
Severe [4]	6 (23.1)	12 (15.8)
PSSI (0-72)	34.0 (11.8)	34.4 (13.7)
SSA, %	56.6 (22.4)	60.8 (27.1)

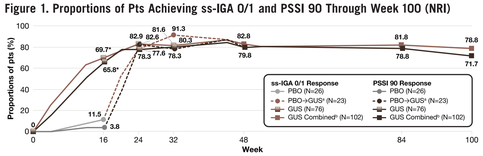
Baseline Pt-Reported Outcomes (Overall study population)



RESULTS

- Week 16 ss-IGA 0/1 and PSSI 90 response rates were significantly greater for the GUS-randomized group vs the PBO group, and improved to >70% for both the GUS and PBO→GUS groups through Week 100 (**Figure 1**)
- Significantly greater proportions of GUS-randomized pts (>59%) achieved scalp clearance (ss-IGA 0 and PSSI 100) compared to PBO-treated pts at Week 16, with response rates generally improving to >66% for both the GUS and PBO→GUS groups through Week 100 (NRI) (data not shown)

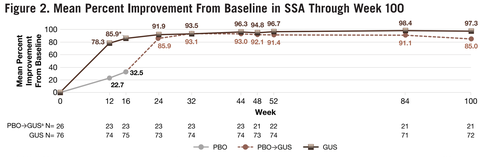
Figure 1. Proportions of Pts Achieving ss-IGA 0/1 and PSSI 90 Through Week 100 (NRI)



**p<0.001 vs PBO; p-values were based on the Cochran-Mantel-Haenszel (CMH) test stratified by FST (I-II/III-VI). [a]For pts who were randomized to PBO at Week 0, only those pts who crossed over to GUS at or after Week 16 were included in Weeks 20–48. [b]Includes pts randomized to GUS at baseline and pts randomized to PBO at baseline who then crossed over to receive GUS at or after Week 16. Pts who discontinued study agent due to lack of efficacy, worsening PsO, or use of a prohibited PsO treatment prior to designated visit were considered non-responders from that point forward. Pts with missing data were considered non-responders at that time point. NRI=Non-responder imputation.*

Mean percent improvements in SSA (**Figure 2**) and PSSI (data not shown) at Week 16 for the GUS-randomized group were >85%, and improved to ~90% for both the GUS and PBO→GUS groups through Week 100

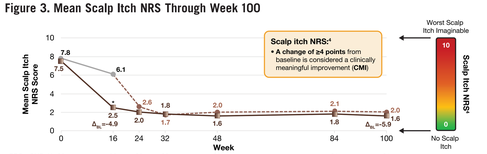
Figure 2. Mean Percent Improvement From Baseline in SSA Through Week 100



**p<0.001 vs PBO; p-values were based on the mixed model for repeated measures; explanatory variables included treatment group, visit, baseline score, FST (I-II/III-VI), an interaction term of visit with treatment group, and an interaction term of visit with baseline score. [a]For pts who were randomized to PBO at Week 0, only those who crossed over to GUS at or after Week 16 were included. Zero change from baseline was assigned if pts discontinued due to lack of efficacy or worsening PsO or used a prohibited PsO treatment (intercurrent events).*

By Week 16, meaningful improvement in mean scalp itch NRS was achieved in the GUS-randomized group and was sustained through Week 100 (**Figure 3**). The proportion of pts achieving ≥4-point reduction was 69.4% at Week 16 improving to 77.8% at Week 100.

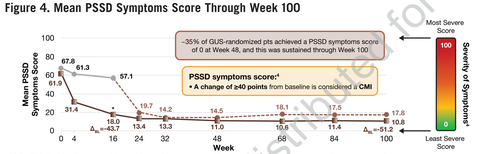
Figure 3. Mean Scalp Itch NRS Through Week 100



**nominal p<0.001 vs PBO; p-values were based on the mixed model for repeated measures; explanatory variables included treatment group, visit, baseline score, FST (I-II/III-VI), an interaction term of visit with treatment group, and an interaction term of visit with baseline score. [a]For pts who were randomized to PBO at Week 0, only those who crossed over to GUS at or after Week 16 were included. Baseline values were assigned if pts discontinued due to lack of efficacy or worsening PsO or used a prohibited PsO treatment. ΔBL=Mean improvement from baseline; zero change from baseline was assigned if pts discontinued due to lack of efficacy or worsening PsO or used a prohibited PsO treatment (intercurrent events).*

On average, pts in the GUS-randomized group had meaningful improvements in PsO symptoms by Week 16; improvements were sustained through Week 100 (**Figure 4**)

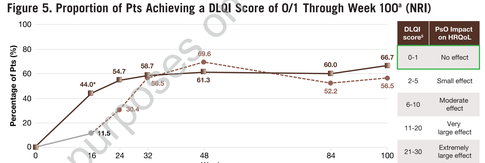
Figure 4. Mean PSSD Symptoms Score Through Week 100



**nominal p<0.001 vs PBO; p-values were based on the mixed model for repeated measures; explanatory variables included treatment group, visit, baseline score, FST (I-II/III-VI), an interaction term of visit with treatment group, and an interaction term of visit with baseline score. [a]For pts who were randomized to PBO at Week 0, only those pts who crossed over to GUS at or after Week 16 were included. Baseline values were assigned if pts discontinued due to lack of efficacy or worsening PsO or used a prohibited PsO treatment. ΔBL=Mean improvement from baseline; zero change from baseline was assigned if pts discontinued due to lack of efficacy or worsening PsO or used a prohibited PsO treatment (intercurrent events).*

Approximately 45% of GUS-randomized pts reported no effect of PsO on their QoL by Week 16, with over 60% achieving and sustaining this endpoint through Week 100 (**Figure 5**)

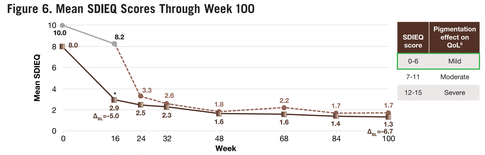
Figure 5. Proportion of Pts Achieving a DLQI Score of 0/1 Through Week 100[a] (NRI)



**nominal p=0.003 vs PBO; p-values were based on the Cochran-Mantel-Haenszel χ² test stratified by FST (I-II/III-VI). [a]Among pts with baseline DLQI >1. [b]For pts who were randomized to PBO at Week 0, only those who crossed over to GUS at or after Week 16 were included. Pts who discontinued due to lack of efficacy or worsening PsO or used a prohibited PsO treatment prior to a designated study visit were considered not to have achieved the binary endpoint, and NRI was used for missing data.*

Mean SDIEQ score, reflecting the effect of pigmentation on QoL, improved from the moderate to the mild range by Week 16 in the GUS-randomized group; improvements in mean SDIEQ continued and were sustained through Week 100 (**Figure 6**)

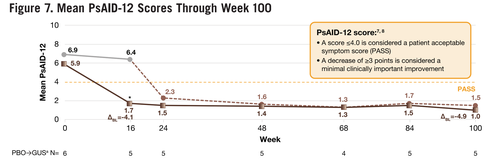
Figure 6. Mean SDIEQ Scores Through Week 100



**nominal p<0.001 vs PBO; p-values are based on the mixed model for repeated measures; explanatory variables treatment group, visit, baseline score, FST (I-II/III-VI), an interaction term of visit with treatment group, and an interaction term of visit with baseline score. [a]For pts who were randomized to PBO at Week 0, only those pts who crossed over to GUS at or after Week 16 were included. Baseline values were assigned if pts discontinued due to lack of efficacy or worsening PsO or used a prohibited PsO treatment (intercurrent events). QoL=Quality of life.*

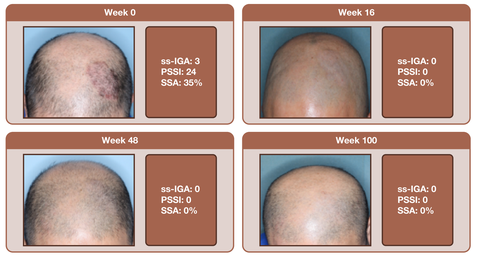
By Week 16, mean PsAID-12 score, reflecting impact of PsA, improved to a patient acceptable level in the GUS-randomized group, and improvements were sustained through Week 100 (**Figure 7**)

Figure 7. Mean PsAID-12 Scores Through Week 100



**nominal p<0.001 vs PBO; p-values were based on the mixed model for repeated measures; explanatory variables included treatment group, visit, baseline score, FST (I-II/III-VI), an interaction term of visit with treatment group, and an interaction term of visit with baseline score. [a]For pts who were randomized to PBO at Week 0, only those who crossed over to GUS at or after Week 16 were included. Baseline values were assigned if pts discontinued due to lack of efficacy or worsening PsO or used a prohibited PsO treatment. ΔBL=Mean improvement from baseline; zero change from baseline was assigned if pts discontinued due to lack of efficacy or worsening PsO or used a prohibited PsO treatment (intercurrent events).*

Figure 8. Participant Who Achieved Complete Scalp Clearance (ss-IGA 0 and PSSI 100) by Week 16 and sustained response through Week 100



- Safety findings were consistent with the established GUS safety profile, with no new safety signals identified through Week 112
- Through Week 112, there were no events of death, cancer, active tuberculosis, MACE, IBD, or serum-like sickness/anaphylaxis

Table 1. Key Safety Information and AEs of Interest Through Week 112

	PBO (N=27) Weeks 0-16	GUS (N=81) Weeks 0-16	PBO→GUS[a] (N=24) Weeks 16-112	GUS (N=81) Weeks 0-112
Mean weeks of follow-up	15.4	16.2	88.8	108.3
Pts with ≥1 AE	3 (11.1)	29 (35.8)	11 (45.8)	63 (77.8)
AEs leading to discontinuation of study agent	0	0	0	0
Serious AEs[b]	1 (3.7)	0	0	3 (3.7)
AEs of interest				
Infections[c]	1 (3.7)	12 (14.8)	8 (33.3)	42 (51.9)
Serious infections	0	0	0	1 (1.2)[c]
Clinically important hepatic disorder[d]	0	0	0	0
MACE[e]	0	0	0	0
Malignancy	0	0	0	0
Venous thromboembolism	0	0	0	0
Serum-like sickness anaphylaxis	0	0	0	0
Tuberculosis	0	0	0	0
IBD[e]	0	0	0	0

Data shown are n (%), unless otherwise indicated. IBD=Inflammatory bowel disease; MACE=Major adverse cardiovascular events. [a]Includes only PBO pts who crossed over to receive GUS. [b]Weeks 0-16: 1 PBO pt had a viral rash; Weeks 0-112: 1 GUS pt had angina pectoris, 1 GUS pt had pancreatitis, and 1 GUS pt had right lower lobe pneumonia. [c]The most common infections for all GUS-treated pts (>5%) included upper respiratory tract infections (24.7%), COVID-19 (16.0%), and nasopharyngitis (7.4%). [d]One GUS pt had pneumonia. [e]No clinically important hepatic disorder AEs were based on a narrow Hepatic Disorders Standardised MedDRA Queries search and recorded on the case report form as serious or leading to study treatment discontinuation. [f]MACE includes sudden cardiac death, nonfatal myocardial infarction, and nonfatal stroke. [g]IBD includes preferred terms of Crohn's disease, ulcerative colitis, and IBD. Pts were counted only once for any given event, regardless of the number of times they experienced the event. AEs were coded using MedDRA version 27.1.

CONCLUSIONS

Through Year 2, VISIBLE Cohort B study results showed:

- ~70% of all GUS-treated pts achieved clear/almost clear scalp skin (ss-IGA 0/1 and PSSI 90)
- ~90% mean % improvement from baseline in SSA and PSSI among all GUS-treated pts
- >66% of all GUS-treated pts achieved complete scalp clearance
- CMIs in HRQoL, PsO symptoms (e.g., scalp itch), and PsA were generally sustained or improved

Clinical responses achieved at Week 16 were maintained or improved through Week 100 with continuous GUS treatment, demonstrating high efficacy and durable responses in diverse pts across all objectively measured skin phototypes

References: 1. Alexis AF & Blackcloud P. *J Clin Aesthet Dermatol.* 2014;7:16–24. **2.** Takeshita J, et al. *J Invest Dermatol.* 2022;142:2528–2531.e3. **3.** McMichael A, et al. *JAMA Dermatol.* 2025;161:912–922. **4.** Armstrong A, et al. *J Dermatolog Treat.* 2019;30:27–34. **5.** Hongbo Y, et al. *J Invest Dermatol.* 2005;125:659–664. **6.** Maymone MBC, et al. *J Dermatol.* 2018;45:361–362. **7.** Gossec L, et al. *Ann Rheum Dis.* 2014;73:1012–1019. **8.** Holland R, et al. *J Psoriasis Psoriatic Arthritis.* 2022;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;175:1298–1304). This poster was supported by Johnson & Johnson, Horsham, PA, USA. **Disclosures: A McMichael** has received grants/research funding from Concert, Incyte, Procter and Gamble, and Revian; consulting fees from AbbVie, Almirall, Apogee, Arcutis, Beiersdorf, Bristol Myers Squibb, Canfield, Concert, Dermavant, Eli Lilly, Galderma, Incyte, Johnson & Johnson, Kenvue, LEO, L'Oréal, Nutrafol, Pelage, Pfizer, Procter and Gamble, Revian, Sanofi-Genzyme, Sun Pharma, and UCB; and royalties from Informa/Taylor and Francis, McGraw Hill, and UpToDate. **T Bhutani** is currently a principal investigator for studies being sponsored by AbbVie, Castle, CorEvitas, Dermavant, Galderma, Mindera, and Pfizer; has additional research funding from Novartis and Regeneron, and has served as an advisor for AbbVie, Arcutis, Boehringer-Ingelheim, Bristol Myers Squibb, Eli Lilly, Johnson & Johnson, LEO, Pfizer, Novartis, Sun, and UCB. **C Kindred** has served as a consultant, advisory board member, and/or speaker for AbbVie, Eli Lilly, Johnson & Johnson, Novartis, Pfizer, Regeneron, Sanofi, Sun, and UCB; has served as an investigator and/or medical board member for AbbVie, Aerolase, Eli Lilly, Pfizer, and Selphyl; and is a journal editor for Cutis. **S Smith** receives honoraria or research grants from AbbVie, Aclaris, Almirall, Alumis, Amgen, Arcutis, Caliway, Candesant, Eli Lilly, Endo Pharmaceuticals, Galderma, Johnson & Johnson, Moberg, Nielsen, Pfizer, Sun, and Texoxane. **O Choi** was an employee of Johnson & Johnson at the time this work was conducted and is a shareholder of Johnson & Johnson; she is currently an employee of Apogee Therapeutics. **T Alkousakis** and **T Ma** are employees of Johnson & Johnson; employees may own stock/stock options in Johnson & Johnson. **R Radusky** is a principal investigator for AbbVie, Amgen, Eli Lilly, Incyte, Jasper Pharmaceutical, Johnson & Johnson, Pfizer, and Sanofi. **J Yeung** has served as a speaker/consultant/honoraria/trialist for AbbVie, Amgen, Anacor, Arcutis, Astella, Bausch, Baxalta, Boehringer Ingelheim, Bristol Myers Squibb, Celgene, Centocor, Coherus, Dermira, Eli Lilly, Forward, Galderma, Johnson & Johnson, LEO, Medimmune, Novartis, Pfizer, Regeneron, Roche, Sanofi-Genzyme, Sun, Takeda, UCB, and Xenon. **G Han** is a consultant, speaker, or received research support from AbbVie, Amgen, Athenex, Beiersdorf, Bristol Myers Squibb, Boehringer Ingelheim, Bond Avillion, Castle Biosciences, Celgene, CeraVe, Dermavant, Dermtech, Eli Lilly, Johnson & Johnson, LEO, MC2, MedX, Novartis, Ortho Dermatologics, Pfizer, Regeneron/Sanofi, Sun, Takeda, and UCB. **SC Taylor** has received honoraria/stock options serving as an advisor/consultant and/or speaker for AbbVie, Arcutis, Armis, Avita, Beiersdorf, Biorez, Bristol Myers Squibb, Cara, Dior, Eli Lilly, EPI, Evolus, Galderma, GloGetter, Hugel America, Johnson & Johnson, L'Oréal, Medscape/WebMD, MJH LifeSciences, Piction Health, Sanofi-Regeneron, Scientis US, UCB, and Vichy; has received honoraria/Board of Directors from Mercer Strategies; served as an author/received royalties from McGraw-Hill; served on the editorial board for Archives in Dermatologic Research, British Journal of Dermatology (peer reviewer), Cutis, and Practical Dermatology; and served as an investigator for Concert Pharmaceuticals, Croma-Pharma, Eli Lilly, and Pfizer.

Presented at Maui Derm Hawaii; January 25-29, 2026; Maui, Hawaii.