

PGA×BSA: A Measure of Psoriasis Severity Tested in Patients with Active Psoriatic Arthritis and Treated with Certolizumab Pegol

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ABSTRACT. *Objective.* The product of physician's global assessment and body surface area (PGA×BSA) to assess psoriasis severity has previously been investigated in patients with psoriasis, with the aim of assessing PGA×BSA as an alternative to the time-consuming Psoriasis Area and Severity Index (PASI). Here, we investigate PGA×BSA as an alternative to PASI in patients with psoriatic arthritis (PsA).

Methods. Analyses used data from the double-blind, placebo-controlled, RAPID-PsA trial (NCT01087788) that investigated the efficacy of certolizumab pegol (CZP) in patients with PsA. Outcomes assessed whether the PGA×BSA and PASI results were comparable, and whether these outcomes correlated with one another or with the Dermatology Life Quality Index (DLQI).

Results. For CZP-treated patients, both PGA×BSA and PASI demonstrated similar sensitivities to treatment between baseline and Week 24, with mean improvements of 77.4% and 69.0%, respectively. Similar improvements were also seen with placebo (PGA×BSA: 3.2%, PASI: 6.1%). Achievement of 75% response criterion in PGA×BSA and PASI was attained by similar proportions of patients with CZP (PGA×BSA75: 59.0%, PASI75: 61.4%) and placebo (PGA × BSA75: 15.1%, PASI75: 15.1%). Cross tabulations showed high concordance between achievement of response outcomes in PGA×BSA and PASI (79.6–95.2%). Spearman correlations revealed strong correlations between PGA×BSA and PASI at baseline ($r = 0.78$; $n = 225$) and percentage improvement to Week 24 ($r = 0.85$; $n = 186$). Both outcomes were only moderately correlated with DLQI ($r = 0.41$ – 0.50 ; $n = 179$ – 249).

Conclusion. PGA×BSA is sensitive to changes in skin manifestations in patients with PsA treated with CZP. Further, PGA×BSA correlates strongly with PASI, and achievement of 75% improvement was similar for PGA×BSA and PASI. (J Rheumatol First Release May 1 2018; doi:10.3899/jrheum.170244)

Key Indexing Terms:
PSORIASIS

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Psoriasis is a genetic, immune-mediated disease, affecting 1–3% of the US population. About 30% of patients with psoriasis are also affected by psoriatic arthritis (PsA)¹, which is associated with a wide variety of additional symptoms that contribute to the disease burden. Factors such as joint pain, erosive joint damage, enthesitis, and dactylitis, as well as psoriasis of the skin and nails further increase the longterm

effect on patients' quality of life, physical function, and ability to work^{2,3}.

The wide variety of symptoms makes evaluation of overall disease activity and response to therapy difficult in PsA; however, accurate assessment is essential for the clinician to determine the most appropriate treatment. These assessment difficulties are particularly true of skin disease because of limitations of the currently used outcome measures.

The Psoriasis Area and Severity Index (PASI) is the most widely used tool for the measurement of skin involvement and is considered the "gold standard" for clinical trials^{4,5}. However, PASI assessments are complex and time-consuming, and can be insensitive in patients with milder forms of psoriasis^{5,6}. The US Food and Drug Administration (FDA) does not accept PASI as a standalone efficacy endpoint in clinical trials for psoriasis. Rather, the FDA often requires the inclusion of a physician's global assessment (PGA) of psoriasis as a coprimary efficacy endpoint^{7,8}. However, PGA instruments do not consider the amount of coverage of skin involvement; they assess only the plaque qualities⁹. Therefore, 2 patients with similar plaque morphology but

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with different amounts of surface involvement (e.g., 30% vs 3%) could have the same PGA score.

To address these limitations, an alternative outcome measure has been proposed: the product of PGA and body surface area (PGA×BSA), which has the advantages of being simpler than the PASI and giving meaningful scores regardless of psoriasis severity¹⁰. The PGA×BSA tool is simpler than the PASI because skin disease severity and psoriasis BSA are scored only once for the entire body, rather than 4 times for each of the 4 body regions measured by the PASI⁹. Additionally, the PGA×BSA calculation is straightforward, whereas PASI scoring requires multiple mathematical computations. The Group for Research and Assessment of Psoriasis and Psoriatic Arthritis has recently highlighted the need for measures that are both simple and valid to assess clinical responses of psoriasis. These measures must be accessible to healthcare providers, patients, regulators, and payers. Importantly, these easily understood measures also have the potential to enhance the dialogue between physician and patient, and improve the measurement of disease activity over time¹¹.

Although simpler than PASI, it must be demonstrated that the PGA×BSA is a comparable outcome measure. In patients with psoriasis, PGA×BSA has been shown to correlate strongly with PASI¹⁰, with recent findings suggesting this simpler alternative tool may be used to assess disease severity and response to therapy in patients with moderate to severe psoriasis¹².

The analyses reported here aimed to investigate whether PGA×BSA can be used as an alternative to PASI for measuring psoriasis severity and response to therapy in patients with PsA using data from the RAPID-PsA trial.

MATERIALS AND METHODS

Patients. Analyses were carried out using data from the phase III RAPID-PsA multicenter clinical trial in Europe, North America, and Latin America (NCT01087788)¹³ that investigated the efficacy of certolizumab pegol (CZP), a PEGylated, Fc-free anti-tumor necrosis factor (TNF) agent, compared to placebo in patients with active PsA. The study was approved by a national, regional, or independent ethics committee or institutional review board at participating sites (ethical approval on behalf of the first author of this publication was provided by The University of Utah Institutional Review Board). The study was conducted in accordance with applicable regulatory and International Conference on Harmonisation Good Clinical Practice requirements, based on the Declaration of Helsinki and local laws. All patients provided written informed consent prior to any protocol-specific procedures being performed. Data from 73 sites were included in this posthoc analysis, including 26 from North America, 22 from Central/Eastern Europe, 14 from Western Europe, and 11 from Latin America. The study was double-blind to Week 24, dose-blind to Week 48, then open-label to Week 216. At Week 0, patients were randomized 1:1:1 to placebo, CZP 200 mg every 2 weeks (Q2W) or CZP 400 mg every 4 weeks (Q4W), administered subcutaneously. Placebo patients who did not achieve a 10% improvement from baseline in both swollen and tender joints at Weeks 14 and 16 underwent mandatory escape and were rerandomized at Week 16 to 1 of the 2 active treatment arms, in a blinded manner. The analyses presented here used data from the initial 24-week double-blind, placebo-controlled, period of RAPID-PsA.

To participate in RAPID-PsA, patients were required to fulfill the CIASsification criteria for Psoriatic ARthritis¹⁴ and to have active disease,

defined as ≥ 3 tender joints and ≥ 3 swollen joints, and either erythrocyte sedimentation rate ≥ 28 mm/h or C-reactive protein > 7.9 mg/l, with duration ≥ 6 months (full eligibility criteria have been previously published)¹³. Patients must have failed treatment with, or have been intolerant to, ≥ 1 disease-modifying antirheumatic drug. The primary clinical¹³ and radiographic¹⁵ endpoints of RAPID-PsA have been reported elsewhere.

Outcomes assessed. Skin outcomes included in these analyses were assessed in all patients with $\geq 3\%$ BSA affected by psoriasis at baseline. Outcomes assessed included the PASI (scored on a 0–72 scale), PGA (scored on a 0–5 scale, where 0 = clear, 1 = almost clear, 2 = mild, 3 = moderate, 4 = moderate to severe, and 5 = severe), BSA (scored as the percentage body area affected by psoriasis; 0–100%), the Dermatology Life Quality Index (DLQI; scored on a 0–30 scale), and the PGA×BSA [0–500; derived posthoc by multiplying patients' PGA scores (0–5) by patients' psoriasis BSA (0–100%)]. Achievement of $\geq 75\%$ response (improvement from baseline) was also considered for PASI (PASI75) and PGA×BSA (PGA×BSA75).

Statistical analyses. Data are reported for patients randomized to CZP (combined dose regimens: 200 mg Q2W and 400 mg Q4W) or placebo. Outcomes are also reported separated by the severity of patients' skin involvement at baseline (PASI ≥ 10 vs PASI < 10 , with PASI ≥ 10 indicating severe skin involvement).

Data are reported as observed case or with imputation. Last observation carried forward imputation was used for continuous outcomes measures and nonresponder imputation for dichotomous outcomes. For placebo patients who underwent mandatory escape at Week 16 to active treatment, their Week 16 response was carried forward to Week 24.

Responsiveness to change was measured between baseline and Week 24 with ANCOVA-adjusted standardized effect sizes, based on the difference in change from baseline between treatments divided by the residual SD, with treatment groups as an explanatory factor and baseline values as a covariate.

The level of agreement between PASI75 and PGA×BSA75 responses at Week 24 was assessed using cross tabulations. Similar analyses were performed for PASI90 and PGA×BSA90, and PASI100 and PGA×BSA100 responses.

Spearman correlation coefficients were calculated to compare PGA×BSA, PASI, and DLQI.

RESULTS

Baseline characteristics. Of 409 patients randomized in RAPID-PsA, 252 had $\geq 3\%$ BSA at baseline (166 received CZP and 86 placebo). Of these patients, 98 had PASI ≥ 10 at baseline (70 received CZP and 28 placebo) and 153 PASI < 10 (95 received CZP and 58 placebo). There were more males in the PASI ≥ 10 group (60.2%) compared to the PASI < 10 group (36.6%). Joint inflammation was similar in patients with PASI ≥ 10 and PASI < 10 at baseline, though all measures of psoriatic skin involvement were higher in patients with PASI ≥ 10 (Table 1).

PASI and PGA×BSA responses following anti-TNF treatment. At Week 24, the mean percentage improvement from baseline was similar for PGA×BSA and PASI in patients receiving CZP (PGA×BSA: 77.4%; PASI: 69.0%) or placebo (PGA×BSA 3.2%; PASI: 6.1%; Figure 1A). The standardized effect sizes, adjusted for baseline values, of the CZP treatment group versus the placebo group at Week 24 were also similar for the 2 measures: -1.0 (95% CI -1.28 to -0.73) for PASI and -1.05 (-1.36 to -0.74) for PGA×BSA. Greater responses were observed in patients with PASI ≥ 10 than PASI < 10 at baseline (Figure 1B). However, within both

Table 1. Baseline characteristics for patients from the RAPID-PsA trial with $\geq 3\%$ psoriasis BSA at baseline and PASI ≥ 10 or < 10 . Values are mean \pm SD unless otherwise specified.

Characteristics	$\geq 3\%$ Psoriasis BSA at Baseline, n = 252	$\geq 3\%$ Psoriasis BSA and PASI ≥ 10 at Baseline, n = 98	$\geq 3\%$ Psoriasis BSA and PASI < 10 at Baseline, n = 153
Demographics			
Age, yrs	47.8 \pm 11.5	48.1 \pm 11.7	47.6 \pm 11.4
Female, %	54.0	39.8	63.4
Weight, kg	85.7 \pm 18.8	86.6 \pm 19.4	84.9 \pm 18.4
Prior medication, %			
1 prior nonbiologic DMARD	61.9	59.2	63.4
≥ 2 prior nonbiologic DMARD	36.1	37.8	35.3
Prior anti-TNF	22.2	27.6	19.0
Disease characteristics			
Tender joint count, 0–68	21.6 \pm 15.1	23.4 \pm 15.4	20.6 \pm 14.9
Swollen joint count, 0–66	11.2 \pm 8.3	12.7 \pm 9.8	10.3 \pm 7.0
HAQ-DI, range 0–3	1.4 \pm 0.6	1.4 \pm 0.6	1.4 \pm 0.6
Psoriasis BSA, %	23.4 \pm 22.2	39.9 \pm 23.7	12.9 \pm 13.0
PASI	11.7 \pm 11.9, n = 251	22.8 \pm 12.1, n = 98	4.5 \pm 2.6, n = 153
PGA \times BSA	79.4 \pm 92.3, n = 226	150.6 \pm 105.2, n = 88	34.1 \pm 40.2, n = 137
DLQI	10.8 \pm 7.3, n = 250	14.5 \pm 7.3, n = 98	8.4 \pm 6.2, n = 151
mNAPSI ¹	3.6 \pm 2.1, n = 202	3.9 \pm 2.1, n = 83	3.4 \pm 2.1, n = 119

¹For patients with nail disease at baseline. PsA: psoriatic arthritis; BSA: body surface area; PASI: Psoriasis Area and Severity Index; DMARD: disease-modifying antirheumatic drugs; TNF: tumor necrosis factor; HAQ-DI: Health Assessment Questionnaire–Disability Index; PGA: physician’s global assessment; DLQI: Dermatology Life Quality Index; mNAPSI: modified Nail Psoriasis Area Severity Index.

subgroups (PASI ≥ 10 and PASI < 10), the responses were similar between the 2 outcome measures. At Week 24, CZP-treated patients with PASI ≥ 10 at baseline showed mean percentage improvements of 88.5% (PGA \times BSA), and 82.2% (PASI), while patients with PASI < 10 at baseline showed improvements of 68.7% (PGA \times BSA) and 60.0% (PASI). This was also reflected in the effect sizes: in the subgroup of patients with milder psoriasis at baseline (PASI < 10), adjusted standardized effect sizes were -0.69 (95% CI -1.03 to -0.35) for PASI and -0.72 (95% CI -1.09 to -0.34) for PGA \times BSA. In the subgroup of patients with PASI ≥ 10 , adjusted standardized effect sizes were -1.64 (95% CI -2.13 to -1.14) for PASI and -1.76 (95% CI -2.34 to -1.18) for PGA \times BSA.

At Week 24, a comparable proportion of CZP-treated patients achieved a PGA \times BSA75 and a PASI75 response (59.0% and 61.4%, respectively; Figure 2A). Similarly, patients receiving placebo achieved PASI75 and PGA \times BSA75 responses in comparable proportions (both 15.1%).

As expected, a greater proportion of CZP-treated patients with PASI ≥ 10 at baseline achieved PGA \times BSA75 and PASI75 at Week 24 than those with PASI < 10 at baseline. Again, however, within subgroups, similar proportions of CZP-treated patients achieved 75% response criteria in PGA \times BSA and PASI (PGA \times BSA75 achieved by 68.6% of patients with PASI ≥ 10 , compared to 52.6% of patients with PASI < 10 ; PASI75 achieved by 77.5% of patients with PASI ≥ 10 , compared to 49.5% of patients with PASI < 10). Response rates for the 2 outcome measures were also

comparable in placebo-treated patients (Figure 2B).

Concordance of PGA \times BSA and PASI responses. Further analyses considered whether patients achieving/not achieving a PGA \times BSA75 response also achieved/did not achieve a PASI75 response. The majority of patients (83.9%) had agreement in their responses to both outcomes (Table 2A).

The number of patients achieving a 75% response in 1 outcome but not the other was similar between the 2 outcomes, with 9.7% of patients achieving a PGA \times BSA75 response but not a PASI75 response, and 6.5% of patients achieving PASI75 but not PGA \times BSA75. This demonstrates the comparability of the outcomes. High correlations between the outcomes were seen when cross tabulations considered 90% or 100% responses (Table 2B and Table 2C).

Spearman correlations (Figure 3) revealed strong correlations between actual values PGA \times BSA and PASI at baseline (0.78), and percentage changes from baseline to Week 24 (0.85), which were similar across patients from North America, Latin America, Western Europe, and Eastern Europe (data not shown).

In terms of percentage change from baseline, PGA \times BSA also correlated more strongly with PASI than did BSA alone (PASI correlation with BSA: 0.77), and slightly more strongly than with PGA alone (PASI correlation with PGA: 0.83). However, both PGA \times BSA and PASI were only moderately correlated with DLQI at baseline (0.46 and 0.50, respectively) and at Week 24 (percentage change from baseline, both 0.41).

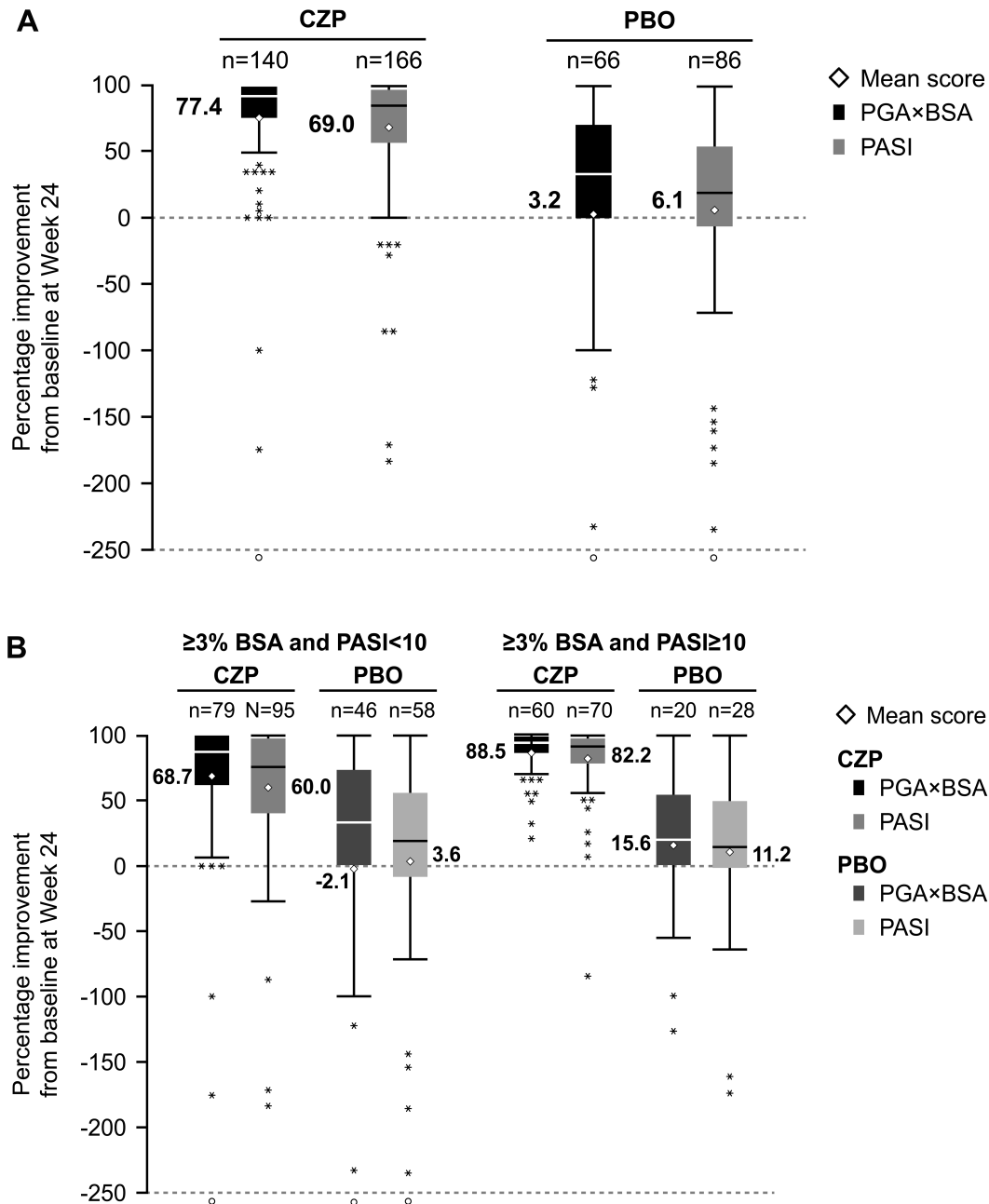


Figure 1. Percentage improvements from baseline to Week 24 in PGA×BSA and PASI (LOCF). **A.** All patients with $\geq 3\%$ psoriasis BSA at baseline. **B.** Patients with $\geq 3\%$ psoriasis BSA at baseline and PASI ≥ 10 or PASI < 10 . Extreme observations not shown in the following: PGA×BSA (CZP –300, PBO –1233); PASI (PBO –331). CZP: certolizumab pegol; PBO: placebo; PGA×BSA: physician's global assessment \times body surface area; PASI: Psoriasis Area and Severity Index; LOCF: last observation carried forward.

DISCUSSION

We demonstrated that the PGA×BSA can be used as an alternative outcome measure to PASI for measuring disease activity and treatment response in skin manifestations in patients with PsA. PGA×BSA is an outcome measure comparable to PASI and is sensitive to change in skin manifestations of PsA with varying levels of skin severity at baseline (PASI ≥ 10 and PASI < 10). Achievement of 75% improvement in

patients with PsA at Week 24 was comparable for PGA×BSA and PASI, with similar effect sizes observed for the 2 measures, when adjusting for baseline values.

Although PGA×BSA was found to correlate strongly with PASI, both outcomes were only moderately correlated with DLQI. This may reflect the fact that both PGA×BSA and PASI measure only plaque quality and surface area and do not account for other factors affecting cutaneous psoriasis

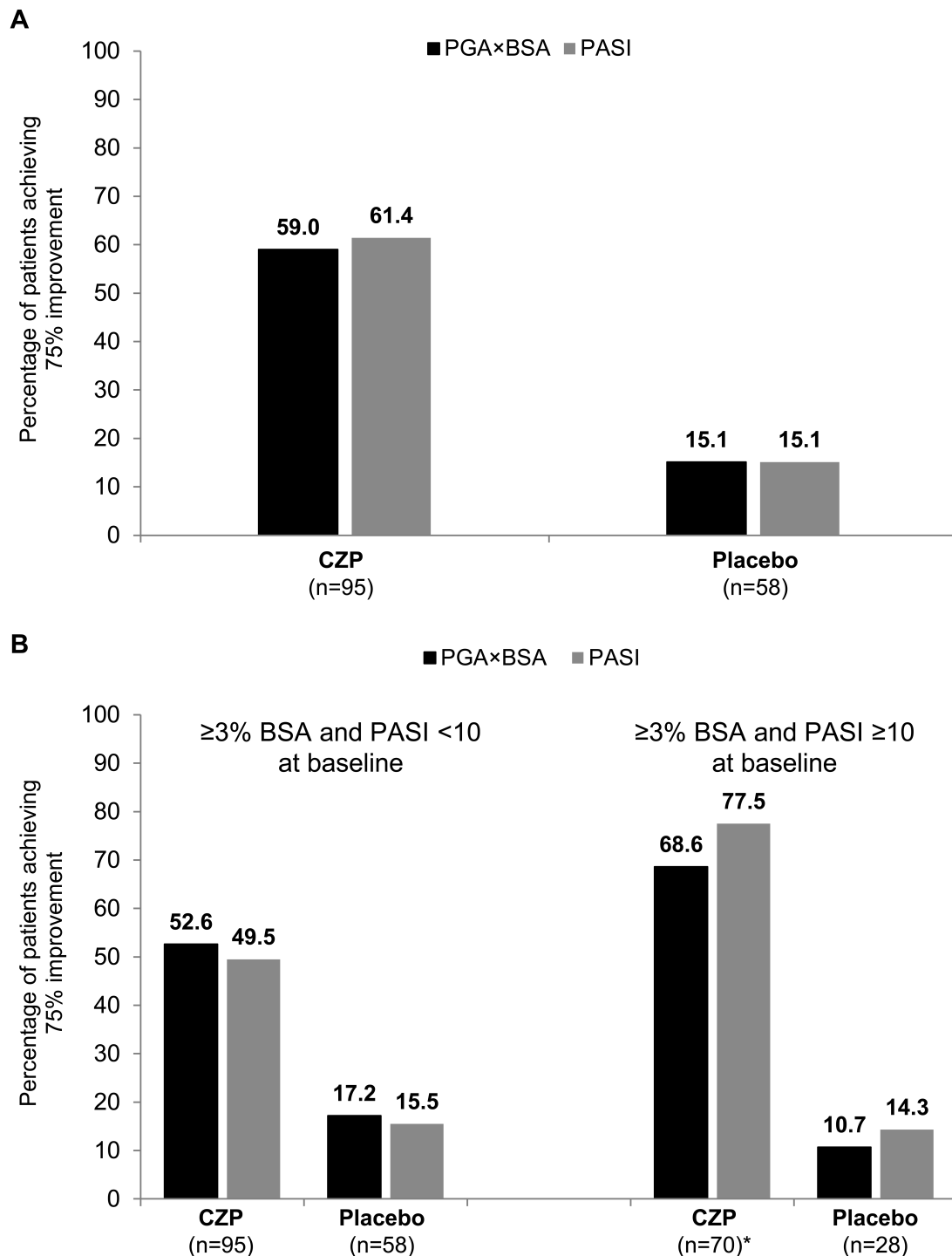


Figure 2. Percentage of patients achieving a 75% response in PASI (PASI75) or PGA×BSA (PGA×BSA75) at Week 24 (NRI). A. All patients with $\geq 3\%$ psoriasis BSA at baseline. B. Patients with $\geq 3\%$ psoriasis BSA at baseline and PASI ≥ 10 or PASI < 10 . *For CZP patients with PASI responses and severe skin involvement at baseline, $n = 71$. For placebo patients who escaped early, their response at Week 16 was used from the time the escape medication was initiated. PASI: Psoriasis Area and Severity Index; PGA×BSA: physician's global assessment \times body surface area; NRI: nonresponder imputation.

disease severity, such as itch, visibility, or discomfort, which are important factors affecting patients' quality of life.

The PGA×BSA has previously been evaluated in patients

with psoriasis, both through retrospective analyses of registry data^{10,16} and recently in a posthoc analysis using data from the ESTEEM 1 and ESTEEM 2 trials¹². Consistent with our

Table 2A. Cross-tabulations of PGA×BSA 75% and PASI 75 response achievement at Week 24 (observed case data; all patients with $\geq 3\%$ psoriasis BSA at baseline).

		PGA×BSA75		Total
		Yes	No	
PASI75	Yes	102 (54.8)	12 (6.5)	114 (61.3)
	No	18 (9.7)	54 (29.0)	72 (38.7)
	Total	120 (64.5)	66 (35.5)	

Shaded numbers = agreement in responses: 83.8%. Bold face numbers = disagreement in responses: 16.2%. Sensitivity of PGA×BSA75 relative to PASI75 = 89.5%, specificity = 75.0%, diagnostic OR = 25.5.

Table 2B. Cross-tabulations of PGA×BSA 95% and PASI 90 response achievement at Week 24 (observed case data; all patients with $\geq 3\%$ psoriasis BSA at baseline).

		PGA×BSA90		Total
		Yes	No	
PASI90	Yes	63 (33.9)	11 (5.9)	74 (39.8)
	No	27 (14.5)	85 (45.7)	112 (60.2)
	Total	90 (48.4)	96 (51.6)	

Shaded numbers = agreement in responses: 79.6%. Bold face numbers = disagreement in responses: 20.4%. Sensitivity of PGA×BSA90 relative to PASI90 = 85.1%, specificity = 75.9%, diagnostic OR = 18.0.

Table 2C. Cross-tabulations of PGA×BSA 100% and PASI 100 response achievement at Week 24 (observed case data; all patients with $\geq 3\%$ psoriasis BSA at baseline)

		PGA×BSA100		Total
		Yes	No	
PASI100	Yes	36 (19.4)	3 (1.6)	39 (21.0)
	No	6 (3.2)	141 (75.8)	147 (79.0)
	Total	42 (22.6)	144 (77.4)	

Shaded numbers = agreement in responses: 95.2%. Bold face numbers = disagreement in responses: 4.8%. Sensitivity of PGA×BSA100 relative to PASI100 = 92.3%, specificity = 95.9%, diagnostic OR = 282.0. PGA×BSA: physician's global assessment \times body surface area; PASI: Psoriasis Area and Severity Index.

data, the ESTEEM analyses showed that PGA×BSA and PASI were highly correlated with each other both at baseline and at Week 16 of treatment. While there were some differences between the patient populations included in the ESTEEM trials and our analysis, notably with a greater psoriasis severity in ESTEEM (PASI ≥ 12 , BSA $\geq 10\%$, PGA ≥ 3 at baseline), the similar findings suggest PGA×BSA to be a consistent and simple measure for the assessment of psoriasis across different conditions and at varying levels of disease severity.

Here, the product of PGA×BSA correlated more strongly with PASI than either component alone, although for PGA this was a fairly small incremental improvement. Despite this relative similarity at a population level, within individual patients PGA×BSA can provide a more representative overview of their disease by taking into account both the plaque qualities and extent of psoriasis. Use of either component alone could lead to misclassification, for example, by categorizing a patient with a PGA of 5 and a

BSA of 0.2% as having more severe disease than a patient with a PGA of 2 and a BSA of 50%.

One limitation of our study is that both the PGA×BSA and PASI were assessed at the same visits by the same physician. The physician's scoring of these outcomes may therefore not be completely independent of the other and thus these analyses may overestimate the agreement between the 2 outcomes. It should also be noted that we used PGA scored on a 0–5 scale; however, other available versions of this instrument use different scales and may affect the reproducibility of the results seen here. In the ESTEEM analyses, the correlations between PASI and PGA×BSA were lower than those seen here, which may be in part because of the different PGA scale used. Additionally, standardized effect sizes should be interpreted with caution because both PASI and PGA×BSA scores after treatment are skewed toward the lower end of the respective scales, which may affect calculations of effect size. Further research is therefore required from prospective studies to further validate this tool.

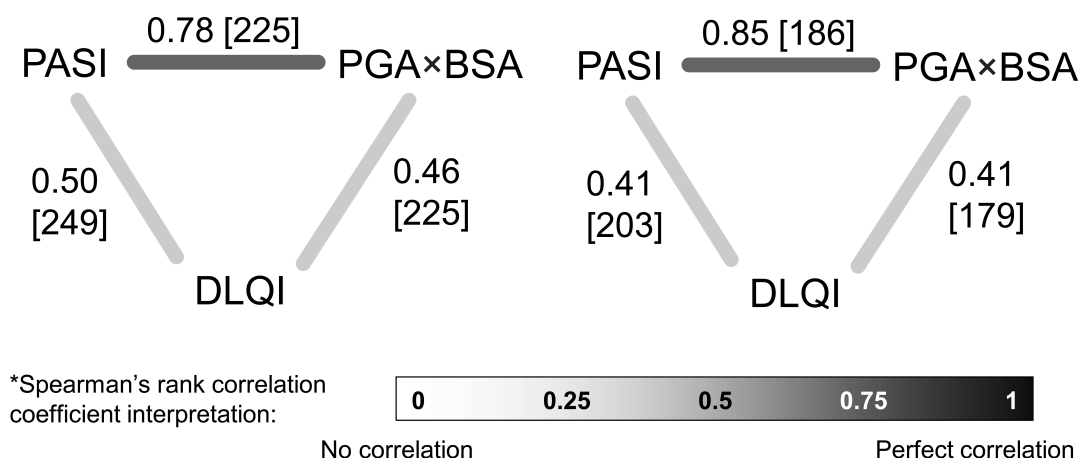


Figure 3. Spearman correlations between PGA×BSA, PASI, and DLQI (observed case data; all patients with $\geq 3\%$ psoriasis BSA at baseline). Numbers shown are Spearman's rank correlation (no. patients). PASI: Psoriasis Area and Severity Index; PGA×BSA: physician's global assessment \times body surface area; DLQI: Dermatology Life Quality Index.

In patients with PsA, PGA×BSA may be considered a practical alternative to PASI for the measurement of psoriasis severity and response to therapy.

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