# Secukinumab for moderate-to-severe palmoplantar pustular psoriasis: Results of the 2PRECISE study

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***Background:*** Palmoplantar pustular psoriasis (PPP) is a debilitating disease of the palms and/or soles that is resistant to treatment. Secukinumab, an anti–interleukin 17A monoclonal antibody, is highly efficacious in the treatment of moderate-to-severe psoriasis.

***Objective:*** The primary objective was to determine the rate of achievement of a 75% improvement from baseline in Palmoplantar Psoriasis Area and Severity Index (PPPASI75) with secukinumab at week 16 versus with placebo (at a 2.5% significance level).

***Methods:*** 2PRECISE was a phase 3b multicenter, randomized, double-blind, placebo-controlled, parallel-group study comparing treatment with 300 mg of secukinumab (n = 79), 150 mg of secukinumab (n = 80), and placebo (n = 78) in subjects with moderate-to-severe PPP over a period of 52 weeks.

***Results:*** The primary end point was not met. At week 16, 26.6% of subjects treated with 300 mg of secukinumab achieved PPPASI75 versus 14.1% of those who received placebo ($P$ = .0411) (odds ratio, 2.62; 95% confidence interval, 1.04-6.60). At week 52, 41.8% of subjects treated with 300 mg of secukinumab had achieved ppPASI75. More Dermatology Life Quality Index responses of 0 or 1 were achieved with 300 mg of secukinumab (13.0%) than with placebo (4.3%) at week 16. At week 52, 43.1% of subjects receiving 300 mg of secukinumab had a Dermatology Life Quality Index response of 0 or 1. No unexpected adverse events were observed.

***Limitations:*** Small sample size and characteristics of the PPP disease course.

***Conclusion:*** Patients with PPP who were treated with secukinumab, 300 mg, showed benefit in terms of PPPASI75 responses over 52 weeks and improved quality of life. ( J Am Acad Dermatol 2019;80:1344-52.)

***Key words:*** palmoplantar pustular psoriasis; PPPASI75; quality of life; secukinumab; randomized controlled trial.

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Palmoplantar pustular psoriasis (PPP) is a debilitating disease of the palms and/or soles that is resistant to treatment.[1] PPP causes a high disease burden and working disability, and it has a high impact on health-related quality of life.[2]

Whether PPP is a unique disease entity or a subphenotype of psoriasis is debated.[3] Both conditions display dysregulated inflammatory pathways, 20% of subjects with PPP have concomitant plaque psoriasis, and the 2 conditions have shared histologic features.[4]

**CAPSULE SUMMARY**

- Palmoplantar pustular psoriasis is chronic, disabling, and resistant to treatment.
- The 2PRECISE randomized controlled trial shows potential benefits of secukinumab in the treatment of palmoplantar pustular psoriasis over 52 weeks, with improved quality of life and no unexpected adverse events.

The pathogenesis of PPP remains unclear, but the skin's innate immune system may be important. The level of neutrophil-attracting chemokine interleukin 8 (IL-8) is increased in PPP.[5] The antimicrobial peptide LL-37 and its precursors are implicated in pathogenesis of PPP and up-regulate the cytokines IL-1, IL-8, IL-23, and IL-17.[6,7] PPP can associate with *HLA-B27* gene but not with the major plaque psoriasis gene *HLA-Cw6*.[8] IL-17A–targeted genes are also up-regulated in PPP, indicating a potential role of IL-17A in its pathophysiology.[9] Recent studies identified 3 distinct subsets of PPP subjects with interleukin 36 receptor agonist gene (*IL36RN*), adaptor related protein complex 1 subunit sigma 3 gene (*AP1S3*), and caspase recruitment domain family member 14 gene (*CARD14*) mutations, which up-regulated the IL-36 pathway, favoring the differentiation of type 17 helper T cells.[10-12]

PPP is resistant to treatment, with high rates of recurrence. Drugs tested in PPP include colchicine, itraconazole, alitretinoin, and biologics (including ustekinumab and guselkumab).[13-15] There are limited guidelines for treatment of PPP, but management algorithms have been proposed.[3]

Secukinumab, which is a fully human monoclonal antibody that selectively targets IL-17A, is highly efficacious in the treatment of moderate-to-severe plaque psoriasis, with a sustained effect and a favorable safety profile.[16,17] Secukinumab is effective in generalized pustular psoriasis, which shares characteristics with PPP, and also in nonpustular palmoplantar psoriasis.[18-21] Epidermal neutrophils disappear shortly after initiation of secukinumab treatment.[22] Therefore, targeting IL-17A with secukinumab may be beneficial for PPP.

The 2PRECISE study aimed to investigate the efficacy and safety of secukinumab in treatment-refractory PPP in a multinational, randomized, placebo-controlled clinical trial.

## METHODS

### Study design and participants

2PRECISE (CAIN457A3301; NCT02008890 registration on December 11, 2013; European Clinical Trials database No. 2013-003086-34) was a phase 3b multicenter, randomized, double-blind, placebo-controlled, parallel-group study comparing 2 doses of secukinumab with placebo in subjects with moderate-to-severe PPP (Fig 1). Subjects were randomized 1:1:1 to 300 mg of secukinumab, 150 mg of secukinumab, or placebo for 16 weeks (treatment period 1 [TP1]). Randomization was carried out by using interactive response technology. Subjects who completed TP1 continued to week 52 in treatment period 2 (TP2), wherein nonresponders to placebo were rerandomized to secukinumab, 300 mg, or secukinumab, 150 mg (Fig 1). Patients who completed TP2 were offered participation in an extension study from week 52 to week 148 (those in the group with a response to placebo were not eligible). Blinding of investigators and subjects was maintained to week 52.

The study protocol was reviewed by the independent ethics committee/institutional review board for each center, and the trial was conducted according to the Declaration of Helsinki. Written informed consent was obtained from all subjects.

Subjects aged 18 years or older with moderate-to-severe chronic PPP (a baseline Palmoplantar Psoriasis Area and Severity Index [PPPASI][23] of ≥12 and a Dermatology Life Quality Index [DLQI] of ≥10) were enrolled. The PPPASI is a modification of the PASI with evaluation of erythema, vesicles/pustules, and scaling/desquamation. Subjects had to have a diagnosis of PPP for at least 6 months before randomization, with confirmed presence of pustules in the past 6 months if not present at screening. Subjects who had erythrodermic or guttate psoriasis or had generalized pustular psoriasis were excluded. All subjects were candidates for systemic therapy (PPP inadequately controlled by topical treatment, phototherapy, and/or previous systemic therapy). Subjects with concomitant plaque psoriasis were

*Abbreviations used:*

| | |
|---|---|
| AE: | adverse event |
| DLQI: | Dermatology Life Quality Index |
| IL: | interleukin |
| OR: | odds ratio |
| PPP: | palmoplantar pustular psoriasis |
| PPPASI75: | 75% improvement from baseline in palmoplantar Psoriasis Area and Severity Index score |
| TP1: | treatment period 1 |
| TP2: | treatment period 2 |
| WPAI-PSO: | Work Productivity and Activity Impairment Questionnaire-Psoriasis |

permitted. No prior use of secukinumab or IL-17–targeting biologics was permitted. Use of other systemic treatments or ultraviolet therapy was not permitted during the study; the washout period was between 2 weeks and 6 months depending on the therapy.

### Procedures

In TP1, secukinumab, 300 mg or 150 mg, or placebo was administered at baseline; at weeks 1, 2, 3, and 4; and then at 4-week intervals. In TP2, placebo subjects who were rerandomized to secukinumab treatment received weekly injections of secukinumab, 300 mg or 150 mg, for 5 weeks followed by administration every 4 weeks. Secukinumab was self-administered subcutaneously with use of a prefilled syringe.[24]

### Study objectives

The primary objective of the 2PRECISE study was to demonstrate the superiority of secukinumab, 300 mg and/or 150 mg, compared with placebo in subjects with moderate-to-severe chronic PPP at week 16, as measured by difference in the rate of achievement of a 75% improvement from baseline in palmoplantar Psoriasis Area and Severity Index (PPPASI75).

The main secondary end point was the mean change from baseline PPPASI with secukinumab, 300 mg or 150 mg, compared with placebo at week 16. Other secondary end points were the time course of PPPASI response rates and the safety and tolerability of secukinumab.

Exploratory outcomes included PPPASI response in subjects with or without concomitant plaque psoriasis or previous systemic or biologic therapy. Biopsies were not performed in this study. Patient-reported outcomes were assessed by using the DLQI,[25] the Palmar-Pustular Quality of Life Index, and the Work Productivity and Activity Impairment Questionnaire-Psoriasis (WPAI-PSO).

### Statistical analysis

For a PPPASI75 response rate of 47% with secukinumab, 300 mg, and 20% response with placebo, 70 subjects per arm would result in 90% power to detect a significant difference.

Efficacy analyses were based on the full analysis set, comprising all subjects to whom study treatment was assigned. Safety analyses were performed on all subjects who received at least 1 dose of study treatment.

The primary analysis was conducted on the full analysis set via logistic regression that included treatment and presence versus absence of plaque-type psoriasis as factors. Subjects with missing PPPASI assessments at week 16 were considered responders if they met the response criteria by the time of dropout; otherwise, they were considered nonresponders (last observation carried forward). To address multiplicity, the 2 primary hypotheses were tested by using a Bonferroni-Holm procedure: if the smaller of the 2 *P* values obtained from the logistic regression model was less than or equal to 2.5%, then the comparison was regarded as significant. Multiple and nonresponder imputation sensitivity analyses were also performed.

Absolute change from baseline to week 16 of the PPPASI was analyzed by using analysis of covariance. A further logistic regression model was used to compare PPPASI response over time for secukinumab-treated and placebo-treated subjects.

Absolute change from baseline of DLQI total score during TP1 was analyzed by analysis of covariance for all pairwise comparisons between the secukinumab treatment groups and the placebo group.

## RESULTS

### Subject disposition

Data were collected between December 26, 2013 (the date of first patient's first visit), and January 20, 2016 (the data cutoff date). Overall, 237 subjects from 61 centers were enrolled in the study. In TP1, 79 were randomized to receive secukinumab 300 mg, 80 to receive secukinumab 150 mg, and 78 to receive placebo (Fig 1). Baseline characteristics were balanced between treatment groups (Table I). A total of 195 subjects (82.3%) completed 16 weeks of treatment (Fig 1). AEs were the most common reason for discontinuation between week 1 and week 16 (Fig 1).

In TP2, 92 subjects in each group received secukinumab, 300 mg, and secukinumab, 150 mg (including placebo nonresponders [Fig 1]). There were 65 (33.5%) discontinuations between weeks 16

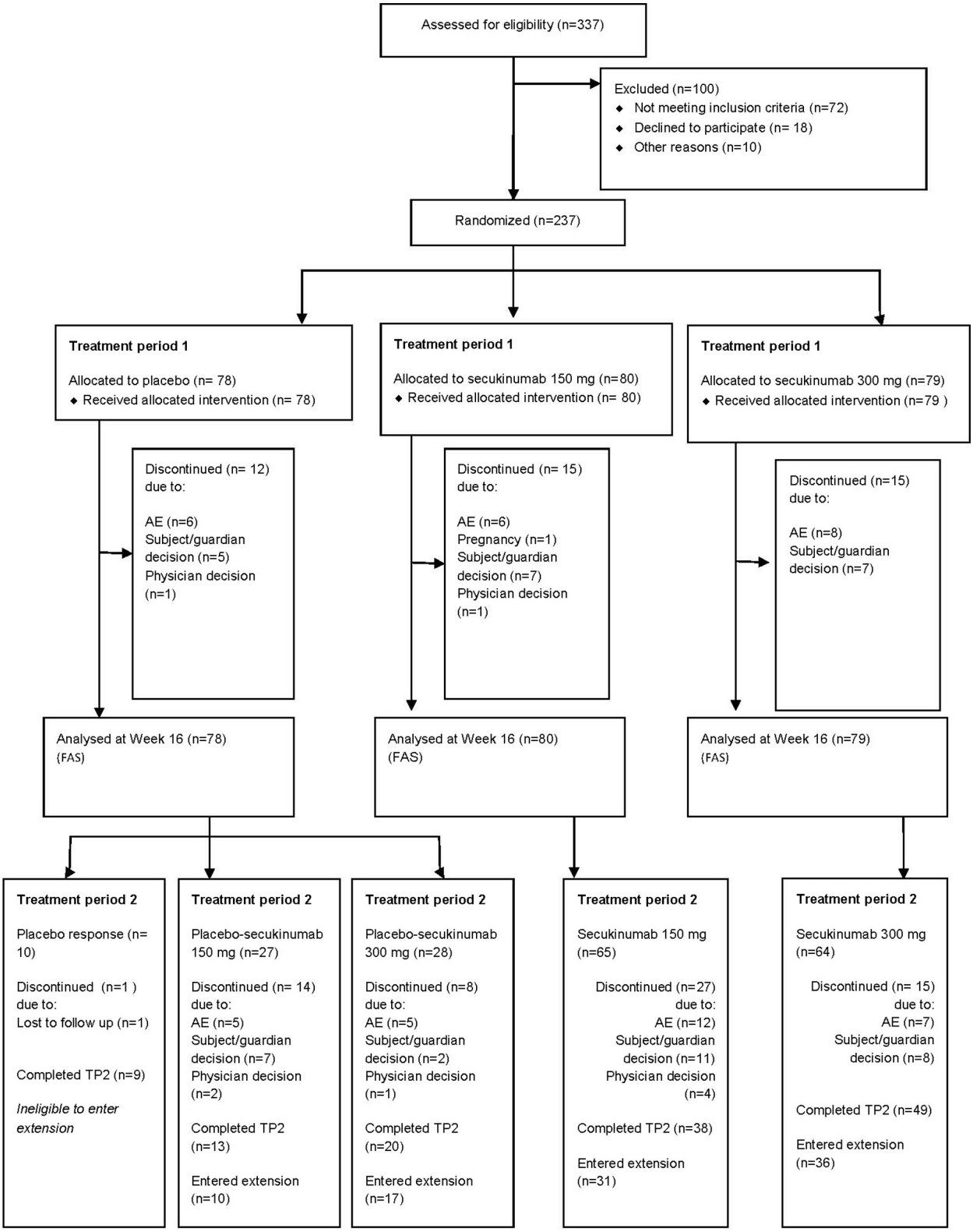


**Fig 1.** 2PRECISE study design and patient disposition. This was a phase 3b multicenter, randomized, double-blind, placebo-controlled, parallel-group study. *AE*, Adverse event; *TP2*, treatment period 2.

**Table I.** Baseline characteristics

| Characteristic | Secukinumab, 300 mg (n = 79) | Secukinumab, 150 mg (n = 80) | Placebo, (n = 78) |
|---|---|---|---|
| Sex, n (%) | | | |
| Female | 64 (81.0) | 63 (78.8) | 59 (75.6) |
| Male | 15 (19.0) | 17 (21.3) | 19 (24.4) |
| Mean age ± SD, y | 50.6 ± 14.8 | 50.7 ± 13.7 | 52.9 ± 11.3 |
| Race, n (%) | | | |
| Black | 0 (0.0) | 1 (1.3) | 1 (1.3) |
| White | 78 (98.7) | 78 (97.5) | 76 (97.4) |
| Missing | 0 (0.0) | 1 (1.3) | 1 (1.3) |
| Other | 1 (1.3) | 0 (0.0) | 0 (0.0) |
| Weight | | | |
| Mean ± SD, kg | 79.0 ± 18.0 | 79.1 ± 16.6 | 78.2 ± 17.7 |
| <90 kg, n (%) | 62 (78.5) | 62 (77.5) | 62 (79.5) |
| ≥90 kg, n (%) | 17 (21.5) | 18 (22.5) | 16 (20.5) |
| Smoking status, n (%) | | | |
| Current | 48 (60.8) | 48 (60.0) | 45 (57.7) |
| Former | 21 (26.6) | 22 (27.5) | 26 (33.3) |
| Never | 10 (12.7) | 10 (12.5) | 7 (9.0) |
| Mean baseline PPPASI ± SD | 23.0 ± 10.3 | 23.1 ± 9.9 | 23.6 ± 10.3 |
| Plaque-type psoriasis, n (%) | | | |
| Yes | 34 (43.0) | 38 (47.5) | 36 (46.2) |
| No | 45 (57.0) | 42 (52.5) | 42 (53.8) |
| Mean baseline PASI score for subjects with plaque-type psoriasis ± SD | 4.4 ± 4.7 | 4.0 ± 3.6 | 4.1 ± 4.7 |
| Mean baseline DLQI ± SD | 16.2 ± 5.1 | 17.1 ± 5.4 | 16.8 ± 5.7 |
| Mean baseline SGA ± SD | 31.2 ± 21.9 | 32.7 ± 22.7 | 30.9 ± 20.7 |
| Mean time since occurrence of PPP ± SD (calculated as years between start date and date of screening visit) | 8.0 ± 8.8 | 9.5 ± 9.5 | 10.3 ± 12.0 |

*DLQI*, Dermatology Life Quality Index; *PASI*, Psoriasis Area and Severity Index; *PPP*, palmoplantar pustular psoriasis; *PPPASI*, 75% improvement from baseline in palmoplantar Psoriasis Area and Severity Index; *SD*, standard deviation score; *SGA*, Subject's Global Assessment.

and 52. Withdrawal rates were higher in the 150-mg group than in the 300-mg group, with the most common reasons for withdrawal being adverse events (AEs) or subject decision (Fig 1).

### PPPASI75 response rates to week 16

At week 16, a PPPASI75 response was achieved in 26.6% of subjects treated with 300 mg of secukinumab (21 of 79) versus in 14.1% who received placebo (11 of 78) ($P$ = .0411) and in 17.5% of those treated with 150 mg of secukinumab (14 of 80) ($P$ = .5722) (Fig 2). The superiority of secukinumab, 300 mg, over placebo did not reach the 2.5% significance threshold; however, an odds ratio (OR) of 2.62 (95% confidence interval, 1.04-6.60) favoring 300 mg of secukinumab over placebo was calculated.

Similar results were seen with use of multiple and nonresponder imputation. The multiple response imputation results for missing PPPASI values were an OR of 2.42 and a $P$ value of .0634, and the last observation carried forward (obtained when data were analyzed by nonresponder imputation) results were an OR of 2.72 with a 95% confidence interval of 1.06 to 7.00.

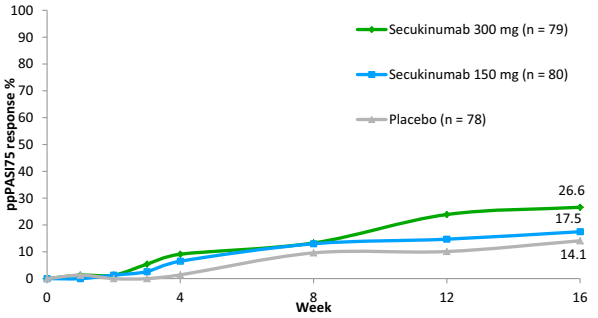


**Fig 2.** Proportion of subjects (full analysis set 1, all patients, last observation carried forward) achieving a 75% improvement from baseline in the Palmoplantar Psoriasis Area Severity Index score (PPPASI75) through week 16. The primary end point was not met, but clinical benefit was observed with secukinumab, 300 mg, with 26.6% of subjects achieving PPPASI75 at week 16.

Excluding subjects from the efficacy analysis by prespecified criteria (where visit 9 was not at week 16, with missing PPPASI at visit 9, and with any

missed doses during TP1) gave an OR between 300 mg of secukinumab and placebo of 3.69 and a *P* value of 0.0150.

A 50% improvement from baseline in PPPASI was achieved by 52.2% of subjects treated with secukinumab, 300 mg, at week 16 (36 of 69) versus by 32.9% of those receiving placebo (23 of 70) (*P* = .0159).

### Mean change from baseline of PPPASI to week 16

The mean change in PPPASI from baseline to week 16 was −30.2% for secukinumab, 300 mg, versus −26.7% for placebo (−9.74 vs −6.73) (*P* = .1576).

### Time course of PPPASI response

The proportion of responders in the group treated with secukinumab, 300 mg, increased between week 8 (13.3% [10 of 75]) and week 16 (26.6% [21 of 79]) (Fig 2, *A*).

At week 52, a PPPASI75 response was achieved in 41.8% of subjects treated with 300 mg of secukinumab (33 of 79) versus in 35.0% of subjects treated with 150 mg of secukinumab (28 of 80) (Fig 3).

### PPPASI response in subjects with plaque psoriasis and previous therapy

At week 16, a PPPASI75 response was achieved in 22.2% of subjects without concomitant plaque psoriasis who received secukinumab, 300 mg, versus in 9.5% of those in the placebo group. In subjects with concomitant plaque psoriasis, the PPPASI75 response rates were 32.4% in those treated with secukinumab, 300 mg, versus in 19.4% of those who received placebo.

Among subjects who had received previous systemic therapy for PPP, 28.6% of those in the group treated with secukinumab, 300 mg, and 14.8% of those in the placebo group achieved a PPPASI75 response at week 16. In comparison, among subjects without prior systemic therapy, a PPPASI75 response at week 16 was achieved by 21.7% of those in the group treated with secukinumab, 300 mg, versus by 12.5% of those in the placebo group.

The rate of ppPASI75 response was also analyzed for subjects who had failure of a previous biologic therapy. In these subjects, PPPASI75 response was higher at week 16 in those subjects receiving 300 mg of secukinumab (42.9%) than in those receiving placebo (14.3%).

### Subject-reported outcomes

More scores of DLQI 0 or 1 were achieved with 300 mg of secukinumab (13.0%) than with placebo (4.3%) at week 16. At week 52, the proportion of subjects with a DLQI of 0 or 1 was considerably higher for those treated with secukinumab, 300 mg, than for those in all the other groups.

Subjects receiving secukinumab, 300 mg, showed consistently greater reductions in WPAI-PSO components (presenteeism, work productivity loss, and total activity impairment) at week 16 (compared with placebo) and at week 52.

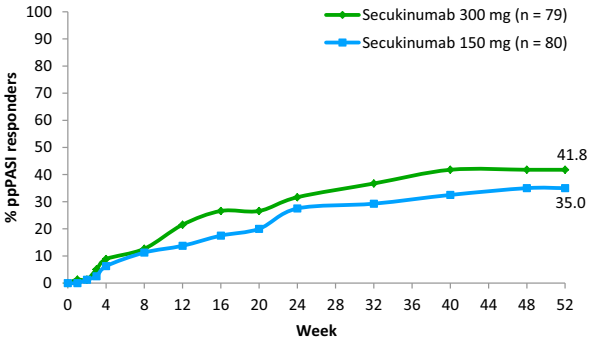


**Fig 3.** Improvements in the rate of achievement of a 75% improvement from baseline in the Palmoplantar Psoriasis Area Severity Index (PPPASI75) with secukinumab were observed to continue to week 52.

### Safety

The safety of secukinumab has been well established in phase 3 clinical trials in plaque psoriasis, with no appreciable differences between the 150-mg and 300-mg doses; the most frequent AEs were nasopharyngitis and upper respiratory tract infections, with a low incidence of AEs of special interest (ie, serious infections, malignancy, major adverse cardiovascular events, and Crohn's disease).[26] The safety profile of secukinumab was consistent with that in previous phase 3 trials, and no new or unexpected safety signals were observed. There were no deaths but 14 serious AEs (8 in subjects receiving 300 mg of secukinumab, 1 in a subject receiving 150 mg of secukinumab, and 5 in those given placebo) (Table II). The most frequent AEs were nasopharyngitis, pustular psoriasis, headache, and pruritus (the incidence rates per 100 patient-years for subjects receiving secukinumab, 300 mg, were 50.3, 30.5, 16.1, and 15.1, respectively) (Table II).

Tonsil cancer was reported in 1 subject who received placebo but was switched to secukinumab, 300 mg, and cerebrovascular accident was reported 1 subject who received placebo during TP1. Neither event was suspected of being related to the study drug.

**Table II.** Summary of AEs through week 52 (the entire treatment period)

| AE | Secukinumab, 300 mg (n = 79) | Secukinumab, 150 mg (n = 80) | Placebo (n = 78) | All secukinumab, 300 mg (n = 107) | All secukinumab, 150 mg (n = 107) |
|---|---|---|---|---|---|
| Any AE, n (%) | 74 (93.7) | 76 (95.0) | 59 (75.6) | 95 (88.8) | 96 (89.7) |
| SAE, n (%) | 10 (12.7) | 4 (5.0) | 5 (6.4) | 13 (12.1) | 5 (4.7) |
| Death | 0 | 0 | 0 | 0 | 0 |
| Most frequent treatment-emergent adverse events by term, IR per 100 patient-years | | | | | |
| Nasopharyngitis | 55.9 | 27.7 | 28.1 | 50.3 | 24.5 |
| Pustular psoriasis | 32.6 | 37.1 | 10.7 | 30.5 | 32.0 |
| Headache | 19.7 | 10.9 | 23.2 | 16.1 | 9.3 |
| Pruritus | 16.9 | 10.8 | 7.9 | 15.1 | 7.8 |
| Psoriasis | 11.5 | 10.6 | 10.0 | 8.5 | 10.3 |
| Bronchitis | 9.8 | 7.0 | 2.0 | 11.0 | 6.3 |
| Cough | 11.5 | 8.9 | 0.0 | 6.4 | 9.6 |
| Folliculitis | 11.8 | 7.1 | 1.9 | 9.9 | 5.1 |
| Eczema | 11.4 | 3.4 | 5.8 | 8.5 | 5.1 |
| Pain in extremity | 11.5 | 1.7 | 11.7 | 8.6 | 3.8 |
| Back pain | 9.9 | 5.3 | 7.7 | 7.4 | 3.8 |
| Diarrhea | 9.8 | 5.2 | 7.7 | 7.4 | 3.8 |
| Nausea | 8.3 | 3.5 | 0.0 | 7.3 | 3.8 |
| Urinary tract infection | 8.1 | 3.5 | 5.8 | 6.0 | 5.1 |
| Arthralgia | 4.8 | 5.2 | 9.7 | 3.6 | 5.2 |

*AE*, Adverse event; *IR*, incidence rate; *SAE*, serious adverse event.

## DISCUSSION

As PPP is a chronic debilitating condition, there is a high unmet need for long-term therapeutic options.

After 16 weeks of treatment there was clinical improvement of PPP in the group treated with secukinumab, 300 mg; however, the significance threshold was not reached. The placebo response rate in 2PRECISE was relatively high for a clinical trial in psoriasis; however, a high proportion of patients with PPP appeared to experience spontaneous remissions and the natural course of the disease is not fully understood.[27] To our knowledge, efficacy similar to that seen at 52 weeks has not been reported in this patient population for any other treatment used in PPP to date.[14,28] Secukinumab was well tolerated, with no new or cumulative treatment-emergent AEs. The slightly higher discontinuation rate as compared with that in psoriasis trials with biologics, including secukinumab, may be due to the disease characteristics of PPP; however, because of a lack of similar clinical trials in PPP, there is no direct comparison.

Response to secukinumab, 300 mg, was higher in subjects with concomitant plaque psoriasis than in those without. In subjects who received secukinumab throughout the 52-week period, response was separated between those with or without concomitant plaque psoriasis. There may be 2 distinct nosologic entities, which may explain why the response of PPP to secukinumab is different than that of plaque psoriasis. Genetic studies suggest that PPP is distinct from plaque psoriasis; for instance, PPP is associated with *HLA-B27* but not with the major plaque psoriasis gene *HLA-Cw6*.[8] Subtypes of PPP may also exist—as evidenced by mutations detected in 3 innate immunity genes, *IL36RN*, *AP1S3*, and *CARD14*[10-12]—and these could respond differently to treatment. The pronounced effect of secukinumab on the signs and symptoms PPP after 52 weeks argues for a pathogenetic role of IL-17A in PPP, which is supported by evidence that type 17 helper T cells and IL-17 cytokines play an important role in PPP.[9,29-31]

Published guidance on treatment of PPP highlights the importance of long-term disease control.[3] Therefore, the efficacy of secukinumab after 52 weeks of treatment may constitute a more meaningful time point. The difference between secukinumab and placebo at week 16 was small; this difference may also have arisen from variation in the natural course of PPP, which is not fully understood. The week 52 response to secukinumab, 300 mg, is relevant to clinical practice in this treatment-refractory condition. This is supported by a substantial improvement in health-related quality of life, particularly in the context of the mean

baseline DLQI of 16 to 17.[32] Furthermore, the assessment of the WPAI-PSO showed major improvements in presenteeism, work productivity loss, and total activity impairment.

A secondary objective was to evaluate the time course of PPPASI response rates between baseline and week 16, and between week 16 and week 52. As PPP is known to show fluctuations, the observations for the placebo group may have arisen from the natural course of the disease.

The 2PRECISE study was limited by its sample size, which although large for PPP, was not comparable to that in larger trials in plaque psoriasis. The primary end point at week 16 may not fully match the characteristics of the PPP disease course after initiation of any treatment. Clinical experience shows that clearing of PPP lesions may take longer than clearing of psoriatic lesions elsewhere on the body. The statistical analysis plan using a Bonferroni-Holm strategy missed the stringent statistical significance threshold after 16 weeks of secukinumab therapy despite the clinical efficacy of the dose of 300 mg of secukinumab. Additionally, use of the stringent primary end point of achievement of more than a 75% improvement of PPPASI as compared with baseline may not have been appropriate in a highly treatment-resistant condition such as PPP.

A criterion standard of treatment for PPP does not exist. Many drugs have shown no or limited benefits in small sample sizes or subject case reports.[13,14] In the latest Cochrane Review of treatments for PPP, only a few high-quality studies were identified, and few of them used patient-reported outcomes.[28] Therefore, any new therapeutic option suitable for long-term control of PPP is of great interest.

The primary end point of secukinumab superiority to placebo at week 16 in patients with PPP was not met in 2PRECISE. However, the reduction of PPPASI by 75% in more than 41% of subjects and the achievement of a DLQI of 0 or 1 in 43% of subjects treated with secukinumab, 300 mg, over 52 weeks in 2PRECISE represents potential benefit in a difficult-to-treat condition.

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