Secukinumab provides sustained low rates of radiographic progression in psoriatic arthritis: 52-week results from a phase 3 study, FUTURE 5.

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Secukinumab provides sustained low rates of radiographic progression in psoriatic arthritis: 52-week results from a phase 3 study, FUTURE 5

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Abstract

Objective. To evaluate the effect of secukinumab on radiographic progression through 52 weeks in patients with PsA from the FUTURE 5 study.

Methods. Patients with active PsA, stratified by prior anti-TNF use (naïve or inadequate response), were randomized to s.c. secukinumab 300 mg load (300 mg), 150 mg load (150 mg), 150 mg no load regimens or placebo at baseline, at weeks 1, 2 and 3 and every 4 weeks starting at week 4. Radiographic progression was assessed by change in van der Heijde-modified total Sharp score (vdH-mTSS; mean of two readers). Statistical analysis used a linear mixed-effects model (random slope) at weeks 24 and 52, and observed data at week 52. Assessments at week 52 included additional efficacy endpoints (non-responders imputation and mixed-effects models for repeated measures) and safety.

Results. The majority (86.6%) of patients completed 52 weeks of treatment. The proportion of patients with no radiographic progression (change from baseline in vdh-mTSS ≤0.5) was 91.8, 85.2 and 87.2% in 300, 150 and 150 mg no load groups, respectively, at week 52. The change in vdh-mTSS from baseline to week 52 using random slope [mean change (S.E.)] was −0.18 (0.17), 0.11 (0.18) and −0.20 (0.18) in 300, 150 and 150 mg no load groups, respectively; the corresponding observed data [mean change (S.D.)] was −0.09 (1.02), 0.13 (1.39) and 0.21 (1.15). Clinical efficacy endpoints were sustained, and no new or unexpected safety signals were reported through 52 weeks.

Conclusion. Secukinumab 300 and 150 mg with or without s.c. loading regimen provided sustained low rates of radiographic progression through 52 weeks of treatment.

Trial registration. ClinicalTrials.gov, http://clinicaltrials.gov, NCT02404350.

Key words: spondylarthropathies (including psoriatic arthritis), biological therapies, immunotherapy, cytokines and inflammatory mediators, inflammation, pain assessment and management.
Introduction

PsA is a chronic inflammatory peripheral arthritis associated with axial disease, dactylitis, enthesisitis, skin and nail psoriasis, physical disability, and reduced health-related quality of life [1, 2]. More prominently, it is associated with structural changes to the joints (erosive or proliferative bone changes, erosive joint destruction, joint space narrowing, fluffy periostitis and pencil-in-cup deformities), which are detected and characterized radiographically [3–5]. Approximately 50% of PsA patients are diagnosed with erosive joint destruction within 2 years of disease onset [6]. Assessment of radiographic disease progression in PsA is an important outcome from a clinical perspective, to assess disease severity and to quantify the effect of treatment on disease progression [7]. IL-17A plays a crucial role in the pathogenesis of PsA; structural damage and radiographic disease progression are strongly correlated with the presence of IL-17A-producing immune cells at the inflamed joints [8–10].

Secukinumab, a human monoclonal IgG1κ antibody that directly inhibits IL-17A, has demonstrated rapid and sustained efficacy in patients with PsA across Phase 3 FUTURE studies [11–14]. Secukinumab 300 and 150 mg s.c. doses have been approved for treatment of PsA. In the FUTURE 1 study, secukinumab 150 mg reported sustained clinical responses with a low rate of radiographic progression over 3 years [15]. In FUTURE 5, secukinumab 300 and 150 mg s.c. dosing regimens significantly lowered the rate of radiographic disease progression vs placebo at week 24 [12]. Herein, we present the effects of secukinumab 300 and 150 mg dosing regimens on radiographic disease progression through 52 weeks from the FUTURE 5 study.

Methods

Patients and study design

FUTURE 5 (NCT02404350) is an ongoing randomized placebo-controlled 2-year Phase 3 trial. The detailed inclusion and exclusion criteria and study design have been reported previously [12]. Patients ≥18 years of age, fulfilling the CASPAR (Classification criteria for Psoriatic Arthritis) criteria [16], with symptoms of active PsA for at least 6 months: three or more tender joints (TJC) and three or more swollen joints (SJC) despite treatment with NSAIDs, conventional synthetic DMARDs (csDMARDs) and/or anti-TNF agents. Concomitant use of glucocorticoids (≤10 mg/day), NSAIDs and MTX (≤25 mg/week) were allowed. Patients with an inadequate response (IR) or who stopped treatment due to safety or intolerance to an anti-TNF agent (hereafter collectively referred as anti-TNF-IR) were included in this study. Key exclusion criteria included active/history of ongoing infection, prior use of a biologic other than an anti-TNF agent, use of greater than three different anti-TNF agents and active inflammatory disease other than PsA.

Eligible patients were randomized (2:2:2:3) to one of four treatment groups: self-administered s.c. secukinumab 300 mg with loading dose (300 mg), 150 mg with loading dose (150 mg), 150 mg without loading dose (150 mg no load) or placebo. All patients received s.c. secukinumab 300 mg, 150 mg or placebo at baseline, at weeks 1, 2 and 3, and every 4 weeks starting at week 4. Patients, investigators and assessors remained blinded to the treatment assignment until all patients reached the week 52 visit. At week 16, non-responders (<20% reduction in TJC and/or SJC) in the placebo group were switched to s.c. secukinumab 300 or 150 mg and all remaining patients (responders) on placebo were switched at week 24. Patients were stratified according to prior use of anti-TNF therapy status [i.e. patients who were naïve to anti-TNF therapy (anti-TNF-naïve) vs those who were anti-TNF-IR]. The study was planned to enrol no more than 30% anti-TNF-IR patients.

The study was conducted in accordance with the Declaration of Helsinki [17] and was approved by institutional review boards or independent ethics committees at each participating centre. Written informed consent was obtained from all enrolled patients. Data were collected in accordance with Good Clinical Practice guidelines by the study investigators and were analysed by the sponsor. Data presented here are from the week 52 (1 year) analysis of the study.

Efficacy outcomes

Radiographic disease progression was assessed by change from baseline in van der Heijde-modified total Sharp score (vdH-mTSS; sum of bone erosion [0–5 in the hands and 0–10 in the feet] and joint space narrowing [0–4 scores] for PsA [18]). Radiographic scores were assessed from two reading sessions: reading session 1: baseline, weeks 16 and 24; and reading session 2: baseline, weeks 16, 24 and 52. The vdh-mTSS assessment was based on hands/wrists/feet radiographs obtained from reading session 1 for 24-week analysis and from reading session 2 for 52-week analysis. Mean scores were assessed by two blinded readers independently (if there was an adjudicator involved then three readers were used) who were blinded to all patient information, treatment allocation and order of radiographs. The total radiographic score (hands and feet combined) ranged from 0 to 528, with higher scores indicating more articular damage. Data are shown for weeks 24 and 52. As recommended by van der Heijde et al., a change from baseline in vdh-mTSS ≤0.5 was used to define no structural progression; the proportion of patients with no structural progression was determined [18] at week 52.

Other assessments at week 52 included the ACR 20, 50 and 70 responses, Psoriasis Area and Severity Index 75 and 90 responses, mean change from baseline in HAQ-Disability Index, 28-joint DAS using CRP (DAS28-CRP), and Short Form 36 Physical Component Summary Score (SF-36 PCS), and resolution of enthesitis and dactylitis [19–25].

Pre-specified subgroup analyses based on previous use of anti-TNF therapy status (naïve vs IR) on radiographic disease progression was also performed.
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Overall safety and tolerability of secukinumab over the 52 weeks was assessed by monitoring adverse events (AEs), serious AEs, laboratory assessments and vital signs.

Radiography study population

The 52-week analysis
At week 52, data are presented by linear mixed-effects model (random intercept and random slopes, which will be referred to as random slope hereafter) and as observed analysis. The random slope model at week 52 included patients with baseline and at least one post-baseline radiographic assessment (evaluable radiographs) from week 16, 24 or 52. Observed analysis included patients who had radiographic assessments at baseline and week 52.

The 24-week analysis
In addition to week 52, data at week 24 are presented using linear extrapolation and random slope models. Linear extrapolation and random slope analyses at week 24 included patients with baseline and at least one post-baseline radiographic assessment from week 16 or 24. This 24-week linear extrapolation was reanalysed after an incorrect mapping of the radiographic data was identified in the interim analysis.[12] It was confirmed that 10/936 (1.1%) patients with baseline and post-baseline radiographs were impacted by the incorrect mapping and were included in this 24-week linear extrapolation reanalysis.

Statistical analyses
Data are presented for patients originally randomized to secukinumab and placebo. In the linear mixed-effects model, the random slope and random intercept was used to calculate the changes in radiographic structural progression score over 52 weeks. The week 24 random slope excluded data after escape for placebo patients who received escape therapy at week 16. The model assumed approximately linear progression over time and estimated a difference in rates (slopes) of progression over 52 weeks to compare treatment arms. The model accounted for covariates of time, treatment, treatment-by-time interaction, baseline mTSS score, body weight and prior anti-TNF status.

The linear extrapolation approach used the change rate from baseline to week 16 to impute the missing change rate for week 24; that is, for patients who met the criteria for early escape at week 16 and did not have week 24 measurement on the same treatment. Linear extrapolation was used to impute the missing values at week 24 if at least two assessments existed before week 24 (including baseline assessment).

Cumulative probability plot was generated as described previously [26] to show the vdH-mTSS for all patients according to dose group from baseline to week 52.

Clinical efficacy analyses used logistic regression for binary variables, and mixed-effects models for repeated measures (MMRM) for continuous variables. All analyses included treatment and randomization stratum (anti-TNF status) as a factor (time, treatment-by-time and baseline-by-time interaction were also used for MMRM), and body weight and the corresponding baseline value as a covariate. Missing values and placebo patients rescued at week 16 were imputed as non-responders for binary variables (rescue penalty) and the missing at random assumption of the MMRM analysis was applied for continuous variables. Safety analysis included all patients who received one or more dose of study drug and were summarized descriptively. Patients were evaluated according to the treatment they received. All analyses were computed with SAS version 9.4.

Results
Of the 996 patients randomized at baseline, 86.6% (862/996) completed 52 weeks of treatment [300 mg: 91.9% (204/222), 150 mg: 91.4% (201/220), 150 mg no load: 86.9% (193/222) and placebo-secukinumab: 79.5% (264/332)]. A total of 108 (10.8%) patients discontinued before or at week 52. The detailed disposition up to week 52 including reasons for discontinuation is presented in Fig. 1. Demographics and baseline disease characteristics were balanced between treatment arms and have been reported previously [12]. At baseline, ~70% of patients were anti-TNF-naive and 50% were receiving concomitant MTX. The number of patients with evaluable radiographs at both baseline and week 52 were as follows: 207 (93.2%) in 300 mg, 203 (92.3%) in 150 mg and 195 (87.8%) in 150 mg no load. The number of patients included in the linear extrapolation and random slope analyses at week 24 were: 217 (97.7%) in the 300 mg, 213 (96.8%) in 150 mg, 210 (94.6%) in 150 mg no load and 296 (89.2%) in the placebo group.

Efficacy
Radiographic progression
In the overall population, the radiographic progression rate was low at week 52 across all treatment groups. Mean changes from baseline in vdH-mTSS using observed data and random slope analysis at week 52 are presented in Fig. 2A. Random slope data (s.e.) showed numerically lower progression in the 300 mg [−0.18 (0.17)] and 150 mg no load [−0.20 (0.18)] groups compared with the 150 mg [0.11 (0.18)] group. Observed data (s.d.) showed lower [−0.09 (1.02)] progression in the 300 mg group compared with the 150 [0.13 (1.39)] and 150 mg no load [0.21 (1.15)] groups.

Mean changes (s.d.) from baseline in vdH-mTSS by linear extrapolation were 0.02 (1.34; P < 0.001), 0.13 (1.22; P < 0.01) and -0.10 (2.87; P < 0.01) in the 300, 150 and 150 mg no load groups, respectively, vs 0.50 (1.71) in the placebo group at week 24 (Fig. 2B). Corresponding mean changes (s.e.) from baseline by random slope analysis were very similar, with 0.03 (0.13; P < 0.01), 0.14 (0.13; P < 0.05) and -0.10 (0.13; P < 0.001), respectively, vs 0.51 (0.11) at week 24 (Fig. 2B).
In patients with prior anti-TNF therapy status, the mean changes from baseline in vdH-mTSS by random slope, observed data and linear extrapolation analyses followed a similar trend of low radiographic progression to that in the overall population (Fig. 2A and B). The anti-TNF-naive group showed lower progression than the anti-TNF-IR group.

Additionally, the mean changes from baseline at week 24 in vdH-mTSS by linear extrapolation from patients with baseline and at least one post-baseline radiographic
The proportion of patients with no radiographic progression (change from baseline in vdH-mTSS \( \leq 0.5 \)) with secukinumab at week 52 were 91.8\% (\( n = 207; 300 \) mg), 85.2\% (\( n = 203; 150 \) mg) and 87.2\% (\( n = 195; 150 \) mg no load). Cumulative probability in each dose group in the range of 0 to 100\%. The lower score indicates more inhibition achieved. \( N \): total number of patients randomized; \( n \): number of evaluable patients; vdH-mTSS: van der Heijde-modified total Sharp score.

FIG. 3 Cumulative probability plot in vdH-mTSS at week 52

The proportion of patients with no radiographic progression (change from baseline in vdH-mTSS \( \leq 0.5 \)) with secukinumab at week 52 were 91.8\% (\( n = 207; 300 \) mg), 85.2\% (\( n = 203; 150 \) mg) and 87.2\% (\( n = 195; 150 \) mg no load). Cumulative probability plots in vdH-mTSS change from baseline to week 52 showed the scores of the individual patients in the different secukinumab doses over 52 weeks of therapy (Fig. 3). The findings in the pre-specified subgroup of patients by anti-TNF therapy status through 52 weeks were similar. The proportion of anti-TNF-naïve patients with no radiographic progression at week 52 were: 94.6\% (300 mg), 85.7\% (150 mg) and 88.7\% (150 mg no load), and in anti-TNF-IR patients were: 85.0\% (300 mg), 83.9\% (150 mg) and 83.3\% (150 mg no load).

Clinical efficacy
The ACR20 response rates reported at week 16 [12] were sustained through 52 weeks of secukinumab treatment. At week 52, the ACR20 response was 68.9, 64.1 and 65.8\% in secukinumab 300, 150 and 150 mg no load groups, respectively (Table 1). At week 52, the ACR20 response rate in anti-TNF-naïve patients was 72.7, 74.2 and 69.6\% in the secukinumab 300 mg, 150 mg and 150 mg no load groups, respectively; in comparison, a numerically lower response (60.3, 40.0 and 56.3\%) was observed in anti-TNF-IR patients (Table 1). In the overall population, the least squares mean change (S.E.) from baseline in DAS28-CRP score was -1.92 (0.07), -1.72 (0.07) and -1.83 (0.07) in the 300, 150 and 150 mg no load groups, respectively, at week 52. Corresponding mean change in SF-36 PCS score was 7.16 (0.46), 6.31 (0.46) and 7.03 (0.47), respectively, at week 52. Improvements reported across all other efficacy endpoints at week 16 were also sustained at week 52 (Table 1).

Safety
The safety data were consistent with that reported previously [12]. Over the entire treatment period, mean secukinumab exposure was 309.0 days (patient-years: 815.5). The most commonly reported treatment-emergent AEs were nasopharyngitis and upper respiratory tract infection (Table 2). Most serious AEs represented single events with no discernible pattern. Serious AE rates were low and comparable in both any secukinumab 300 mg (8.1\%) and 150 mg (8.3\%) dose groups. The rate of discontinuation due to any AE was also low and comparable between any secukinumab 300 mg (2.4\%) and 150 mg (3.4\%) dose groups. The rate of selected AEs of interest are shown in Table 2. No new or unexpected safety signals, no tuberculosis infections and no deaths were reported.

Discussion
This randomized placebo-controlled large Phase 3 study of s.c. secukinumab 300 mg and 150 mg, with or without...
| Efficacy endpoints | Overall population | Anti-TNF-naïve | Anti-TNF-IR |
|-------------------|--------------------|---------------|------------|
|                   | 300 mg (N=222) | 150 mg (N=220) | 150 mg no load (N=222) | 300 mg (N=154) | 150 mg (N=155) | 150 mg no load (N=158) | 300 mg (N=68) | 150 mg (N=65) | 150 mg no load (N=64) |
| ACR20 response, % | 68.9 | 64.1 | 65.8 | 72.7 | 74.2 | 69.6 | 60.3 | 40.0 | 56.3 |
| ACR50 response, % | 46.8 | 41.4 | 43.2 | 53.2 | 49.7 | 48.1 | 32.4 | 21.5 | 31.3 |
| ACR70 response, % | 28.8 | 25.9 | 24.8 | 33.1 | 30.3 | 26.6 | 19.1 | 15.4 | 20.3 |
| PASI 75 response (BL psoriasis ≥3% body surface area), % | (n = 110) 74.5 | (n = 125) 63.2 | (n = 117) 59.8 | (n = 75) 77.3 | (n = 95) 69.5 | (n = 92) 63.0 | (n = 35) 68.6 | (n = 30) 43.3 | (n = 25) 48.0 |
| PASI 90 response (BL psoriasis ≥3% body surface area), % | (n = 110) 57.3 | (n = 125) 47.2 | (n = 117) 41.0 | (n = 75) 56.0 | (n = 95) 54.7 | (n = 92) 43.5 | (n = 35) 60.0 | (n = 30) 23.3 | (n = 25) 32.0 |
| HAQ-DI score, LS mean change from BL (s.e.) | −0.56 (0.04) | −0.50 (0.04) | −0.52 (0.04) | −0.59 (0.04) | −0.57 (0.04) | −0.57 (0.04) | −0.57 (0.08) | −0.40 (0.08) | −0.46 (0.08) |
| Enthesitis resolution (enthesitis subset), % | (n = 140) 70.7 | (n = 141) 63.1 | (n = 129) 61.2 | (n = 97) 76.3 | (n = 93) 69.9 | (n = 87) 64.4 | (n = 43) 58.1 | (n = 48) 50.0 | (n = 42) 54.8 |
| Dactylitis resolution (dactylitis subset), % | (n = 82) 69.5 | (n = 80) 72.5 | (n = 103) 68.9 | (n = 55) 80.0 | (n = 52) 80.8 | (n = 79) 72.2 | (n = 27) 48.1 | (n = 28) 57.1 | (n = 24) 58.3 |

Missing values were imputed as non-response for binary variables and MMRM analysis for continuous variables. BL: baseline; DAS28-CRP: 28-joint DAS using CRP; HAQ-DI: HAQ-Disability Index; LS: least squares; PASI: Psoriasis Area and Severity Index; MMRM: mixed-effect model repeated measure; N: total number of patients; n: number of patients with that symptom at baseline.
loading regimen, demonstrated sustained low rates of radiographic disease progression, with consistent clinical efficacy and a favourable safety profile, in patients with active PsA over 52 weeks of treatment. The majority (87%) of patients enrolled at baseline remained in the study for 52 weeks, reflecting a high retention rate. The results reported in this paper further substantiate previously published data from the FUTURE 1 and 5 studies that demonstrated significant and sustained inhibition of radiographic progression in patients with PsA [12, 15]. Furthermore, this study validates the treatment effect of secukinumab on radiographic disease progression by utilizing different statistical methodologies to analyse the radiographic data, and obtaining very similar results from each of these methods. The radiographic assessment in this study used random slope, evaluable radiograph and linear extrapolation. The random slope analysis accounted for more baseline covariates and handled statistical uncertainty more robustly than single imputation, which may be a preferred way of handing missing data. The linear extrapolation approach handled inter-current events (i.e. placebo escape patients’ data) and used linearity to model vdH-mTSS over time. This model assumes a linear rate of progression for all patients and uses single imputation. However, the uncertainty surrounding the missing data may not be sufficiently accounted for due to slightly larger CIs.

The majority (85–92%) of secukinumab-treated patients in the overall population showed no radiographic progression at week 52. The vdH-mTSS were slightly different between random slope and observed analyses presented at week 52, with random slope analysis showing lower radiographic progression scores across all secukinumab doses compared with observed data. At week 24, the vdH-mTSS showed significant reduction of radiographic progression in secukinumab-treated patients compared with placebo; the scores were almost identical across all dose groups as measured by linear extrapolation and random slope.

Similar to the overall population, low rates of radiographic progression were also observed in secukinumab-treated patients regardless of prior anti-TNF therapy status. In the subgroup of TNF-naïve patients, there was a significant reduction of radiographic progression in the secukinumab-treated patients at week 24 compared with placebo, which was lower through 52 weeks of treatment. The majority (85–92%) of secukinumab-treated patients in the overall population showed no radiographic progression at week 52. The vdH-mTSS were slightly different between random slope and observed analyses presented at week 52, with random slope analysis showing lower radiographic progression scores across all secukinumab doses compared with observed data. At week 24, the vdH-mTSS showed significant reduction of radiographic progression in secukinumab-treated patients compared with placebo; the scores were almost identical across all dose groups as measured by linear extrapolation and random slope.

### Table 2 Clinical safety with secukinumab during the entire treatment period through week 52

| Variable | Any secukinumab 300 mg (N=371) | Any secukinumab 150 mg (N=593) |
|----------|-------------------------------|-------------------------------|
| Total exposure |                               |                               |
| Mean (s.d.) | 298.5 (83.22) | 315.5 (83.21) |
| Patient-years | 303.2 | 512.2 |
| Any AE, n (%) | 259 (69.8) | 436 (73.5) |
| Any SAE, n (%) | 30 (8.1) | 49 (8.3) |
| Discontinuation due to AE, n (%) | 9 (2.4) | 20 (3.4) |
| Death, n (%) | 0 | 0 |
| Most frequent AEs, n (EAIR per 100 patient-years) | | |
| Nasopharyngitis | 41 (14.4) | 67 (14.1) |
| Upper respiratory tract infection | 24 (8.2) | 55 (11.4) |
| Hypertension | 14 (4.7) | 28 (5.6) |
| Diarrhoea | 17 (5.8) | 24 (4.8) |
| Influenza | 16 (5.4) | 15 (3.0) |
| Headache | 10 (3.4) | 25 (5.0) |
| Selected AEs of interest, n (EAIR per 100 patient-years) | | |
| Serious infections | 5 (1.7) | 8 (1.6) |
| Fungal infection | 0 (0.0) | 2 (0.4) |
| Candida infection (HLT) | 9 (3.0) | 9 (1.8) |
| Oral candidiasis | 6 (2.0) | 3 (0.6) |
| Ulcerative colitis | 0 (0.0) | 1 (0.2) |
| Crohn’s disease | 0 (0.0) | 2 (0.4) |
| MACE | 1 (0.3) | 1 (0.2) |
| Malignant/unspecified tumours | 2 (0.7) | 3 (0.6) |
| Neutropenia | 3 (1.0) | 8 (1.6) |

*The entire treatment period for safety data was from baseline through to the week 52 visit of each patient enrolled in this study. *a*Any secukinumab group represents each originally randomized secukinumab patient plus placebo patients who switched to active treatment at week 16 or 24. *b*AEs that occurred with an EAIR of at least 5.0 cases per 100 patient-years in either any secukinumab 300 or 150 mg group over the entire treatment period. Events listed according to preferred term in the Medical Dictionary for Regulatory Activities (MedDRA version 20.1), sorted in descending order of EAIR. AE: adverse event; EAIR: exposure-adjusted incidence rate; HLT: high-level term; MACE: major adverse cardiovascular event; N: total number of patients; SAE: serious adverse event.
secukinumab treatment. In the (smaller) group of anti-TNF-IR patients, higher progression was observed with secukinumab compared with those observed in TNF-naïve patients. The lower response in the anti-TNF-IR group may be attributed to a relatively small number of patients and the heterogeneity of the anti-TNF subgroup, which included patients with treatment failure due to lack of efficacy, IR, intolerance or safety issues [27] with anti-TNF therapies as specified in the inclusion criteria of the current study. An additional reason may be that these patients are harder to treat. Previous studies confirm the inhibition of radiographic disease progression in PsA patients treated with anti-TNF agents [28–32]. The radiographic findings from the current study are aligned with those reported in the anti-TNF trials and further establish the therapeutic effect of secukinumab in patients with PsA.

Sustained improvement was observed in secukinumab-treated patients across all efficacy endpoints including disease activity and physical function through week 52 in the overall population. Clinical improvements observed at week 16 were sustained through 52 weeks of secukinumab treatment, with numerically greater efficacy in anti-TNF-naïve vs -IR patients. The safety profile up to 52 weeks was consistent with that reported in prior secukinumab trials [11–14], with no new or unexpected safety signals reported.

Limitations included that (i) this study was not designed to identify a difference between treatment regimens (load vs no load) or to assess treatment differences in response to previous anti-TNF therapy status and (ii) the data presented in the anti-TNF-IR group should be interpreted with caution due the small patient population.

In conclusion, this study demonstrated that secukinumab 300 and 150 mg provided significant inhibition of radiographic disease progression at week 24, with sustained low rates of progression at week 52. This study confirms the robustness of the radiographic data as there is very low or minimal impact on the outcome irrespective of the statistical methodology used. These results extend previous findings confirming sustained low rates of progression, clinical efficacy and safety of secukinumab for the treatment of patients with active PsA [11–14].

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Supplementary data

Supplementary data are available at Rheumatology online.
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