Direct Comparison of Real-world Effectiveness of Biologics for Psoriasis using Absolute and Relative Psoriasis Area and Severity Index Scores in a Prospective Multicentre Cohort

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Real-world evidence, directly comparing the effectiveness of interleukin (IL17)-inhibitors, IL23-inhibitors, tumour necrosis factor alpha (TNF-α)-inhibitors and an IL12/23-inhibitor in psoriasis, is scarce. The aim of this study was to directly compare the first-year effectiveness of biologic therapies for psoriasis, corrected for confounders. This prospective, multicentre cohort study assessed BioCAPTURE data on etanercept, adalimumab, ustekinumab, secukinumab, ixekizumab, and guselkumab in 1,080 treatment episodes of 700 patients with psoriasis. The course of the mean absolute Psoriasis Area and Severity Index (PASI) and the proportion of patients who achieved PASI90/PASI175 were compared using linear mixed models and mixed logistic regression models respectively, corrected for baseline PASI, biologic naïveté, and weight. Patients treated with adalimumab, ustekinumab, secukinumab, ixekizumab, or guselkumab all had a significantly lower mean PASI after 12 months compared with etanercept, and significantly higher overall odds of reaching PASI90 than those treated with etanercept. Patients treated with ixekizumab or guselkumab also had higher probabilities of reaching PASI90 than adalimumab, ustekinumab, and secukinumab. Relative to randomized controlled trials, the proportions of patients who reached PASI90/75 were lower in this real-world study.

Key words: psoriasis; biologics; PASI; real-world evidence; effectiveness; observational studies.

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The development of biologic therapies has led to a breakthrough in psoriasis treatment. In network meta-analyses, the comparative efficacy of biologics has been outlined, based on data from randomized controlled trials (RCTs) (1–4). However, in RCTs, the “real-life” patient is not always adequately represented, due to, for example, the exclusion of patients with comorbidities or those above a certain age limit (5). Comparative effectiveness in daily practice has often been studied for tumour necrosis factor (TNF)-α-inhibitors and ustekinumab (6–8), but such research is limited for newer generation biologics. Real-world data, directly comparing the effectiveness of IL17-inhibitors, IL23-inhibitors, ustekinumab (IL12/23-inhibitor) and TNF-α-inhibitors, are needed to supplement current evidence.

In RCTs, which typically include patients with high baseline Psoriasis Area and Severity Index (PASI) scores, efficacy outcomes are mostly reported as relative PASI-reduction compared with baseline (e.g. 75% and 90% PASI-reduction; PASI75 and PASI90). In a systematic review on short-term efficacy (10–16 weeks) of biologics, the proportions of PASI75 and PASI90 responders ranged up to 94.6% and 91.9%, respectively (9). Since reaching a relative PASI target is highly dependent on the baseline value, patients with low PASI scores at baseline may not achieve this, even though absolute treatment response may be excellent. In daily practice, baseline PASIs can be low, especially in patients switching thera-
pies. Furthermore, patients may switch between biologics for reasons other than ineffectiveness (e.g. side-effects or trying to conceive). In addition to the relatively low baseline PASI, treatment results in “real life” may be less favourable than in trials, due to the less controlled setting and the inclusion of patients with comorbidities and co-medication (10). Altogether, this may impede the achievement of PASI75/90 in daily practice.

This study analysed and directly compared the effectiveness of TNF-α-inhibitors (etanercept and adalimumab), an IL12/23-inhibitor (ustekinumab), IL17-inhibitors (secukinumab and ixekizumab), and an IL23-inhibitor (guselkumab) in patients with psoriasis from a prospective multicentre cohort, by reporting absolute and relative PASI measures, with confounder-correction.

MATERIALS AND METHODS

BioCAPTURE database

Data were extracted from the prospective, multicentre, Continuous Assessment of Psoriasis Treatment Use Registry with Biologics (BioCAPTURE-registry, www.biocapture.nl). This study contains data on patients with psoriasis treated with biological therapy in 3 academic and 14 non-academic centres in the Netherlands (2005 to 2021). According to our regional medical ethics committee, ethical approval was not necessary for this non-interventional study.

Data collection

A treatment episode (TE) represents the period of time in which a patient is continuously treated with a certain biologic. When treatment is discontinued or interrupted for ≥ 90 days, the current TE ends. Therefore, a single patient can have multiple TEs. Biologics for which <50 TEs were not included in this study. For biologics with ≥ 50 TEs in the registry, TEs without a baseline PASI or a single follow-up PASI score within the first year of treatment were excluded. Baseline PASI was defined as the PASI score at the start of a TE, allowing a time window of 90 days prior, and up to 7 days after the initiation of treatment. Baseline patient characteristics were collected and calculated for every TE.

In the first year of a new treatment episode, patients generally visit at baseline, week 6, week 12, and every 3 months thereafter. PASIs are measured at each visit. Since scheduling visits at exact time-points is not feasible in a clinical setting, linear interpolation was used to estimate PASIs at 5 time-points: weeks 6, 12, 26, 39 and 52. PASIs measured in a range of 120 days from the intended time-points were used for interpolation. Interpolated PASIs were used to calculate PASI 0, ≤1, ≤3, ≤5, and PASI90, PASI75 or PASI50 at each time-point.

Statistical analysis

Statistical analyses were performed in SPSS version 25.0 (IBM, Armonk, NY, USA) or SAS 9.4 (SAS Institute, Inc., Cary, NC, USA). A p-value < 0.05 was considered significant. Baseline patient and treatment characteristics for the first TE per patient, and per biologic were displayed using descriptive statistics (mean ± standard deviation (SD), median and interquartile range (IQR), n (%)). Continuous variables were compared between treatment groups with one-way analysis of variance (ANOVA) in case of a parametric, and Kruskal–Wallis tests in case of a non-parametric distribution, respectively. For categorical variables, Pearson’s χ² test was used for comparisons.

Proportion achieving PASI75 and PASI90

Data on PASI90 and PASI75 were analysed using a mixed logistic regression model (MLR), built in SAS. In this model, the dependent variable was dichotomous (PASI90 or PASI75 yes/no). Baseline characteristics that were identified as confounders in the LMM were also set as fixed effects in the MLR. The MLR allowed for pairwise comparisons for each treatment with regards to reaching PASI90/PASI75, and calculation of odds ratios (OR). PASI90 and PASI75 analyses were repeated with imputing missing PASIs using LOCF.

RESULTS

At data lock, 1,472 TEs of 871 patients (etanercept 340, adalimumab 485, ustekinumab 392, secukinumab 111, ixekizumab 76, guselkumab 68) were included. Infliximab, certolizumab-pegol, brodalumab, risankizumab and tildrakizumab were excluded, as < 50 TEs were available. After eliminating TEs without a baseline or single follow-up PASI, 1,080 TEs from 700 patients (etanercept 287 TEs (26.6%), adalimumab 343 TEs (31.8%), ustekinumab 276 TES (25.6%), secukinumab 75 TEs (6.9%), ixekizumab 55 TES (5.1%), guselkumab 44 TEs (4.1%)) were included for analyses. Baseline patient characteristics, at the start of the first TE included (n = 700), are shown in Table I. Table II reports baseline patient characteristics per biologic. After 1 year of treatment, 817 TEs (75.6%) were still ongoing, 127 TEs (11.8%) were discontinued due to ineffectiveness, 61 TEs (5.6%) due to adverse events, 24 TEs (2.2%) due to a combination of...
Proportions of PASI75 and PASI90

To correct for confounders (baseline PASI, weight, and biologic naïvety), proportions of TEs achieving PASI75 and PASI90 were analysed with a MLR model. The interaction term between time and type of biologic did not

Table II. Baseline and treatment characteristics of the included treatment episodes (TEs) per biologic

| Baseline TE characteristics | Etanercept n = 287 TEs | Adalimumab n = 343 TEs | Ustekinumab n = 276 TEs | Secukinumab n = 75 TEs | Ixekizumab n = 55 TEs | Guselkumab n = 44 TEs | p-value |
|-----------------------------|------------------------|------------------------|------------------------|------------------------|------------------------|------------------------|---------|
| Age at start of biologic, years, mean ± SD | 47.7 ± 13.0 Missing: 0 | 49.4 ± 13.6 Missing: 0 | 48.5 ± 13.6 Missing: 0 | 50.2 ± 12.1 Missing: 0 | 49.9 ± 11.7 Missing: 0 | 50.5 ± 14.6 Missing: 0 | 0.443d |
| Sex (male), n (%) | Missing: 0 | Missing: 0 | Missing: 0 | Missing: 0 | Missing: 0 | Missing: 0 | 0.009c |
| Height, cm, median [IQR] | 174.0 [12.0] Missing: 31 | 175.2 [14.0] Missing: 31 | 176.0 [15.0] Missing: 31 | 174.0 [14.4] Missing: 31 | 176.8 [13.8] Missing: 10 | 178.0 [11.5] Missing: 5 | 0.208c |
| Weight, kg, median [IQR] | 85.8 [22.8] Missing: 31 | 87.0 [23.3] Missing: 31 | 88.5 [22.8] Missing: 31 | 88.9 [25.9] Missing: 16 | 94.3 [25.0] Missing: 16 | 89.5 [29.0] Missing: 5 | 0.007c |
| BMI, kg/m², median [IQR] | 27.8 [7.3] Missing: 28 | 28.4 [7.2] Missing: 14 | 28.3 [7.6] Missing: 16 | 29.0 [7.5] Missing: 14 | 30.5 [9.4] Missing: 14 | 27.9 [8.1] Missing: 14 | 0.089c |
| Positive family history of psoriasis, yes, n (%) | 190 (67.1) Missing: 4 | 212 (65.4) Missing: 4 | 184 (70.2) Missing: 14 | 44 (62.9) Missing: 4 | 32 (62.7) Missing: 4 | 24 (63.1) Missing: 4 | 0.736b |
| Psoriatic arthritis, yes, n (%) | 83 (30.9) Missing: 4 | 96 (32.0) Missing: 4 | 72 (29.8) Missing: 14 | 27 (38.0) Missing: 4 | 33 (51.9) Missing: 4 | 11 (32.4) Missing: 4 | 0.274b |
| Duration of psoriasis until start of biologic, years, median [IQR] | 20.8 [16.9] Missing: 18 | 19.6 [19.1] Missing: 18 | 18.6 [15.3] Missing: 12 | 22.8 [12.3] Missing: 2 | 21.6 [17.9] Missing: 2 | 21.2 [12.2] Missing: 2 | <0.001b |
| Biologic naïve, yes, n (%) | 164 (57.1) Missing: 1 | 163 (47.5) Missing: 1 | 93 (33.7) Missing: 0 | 5 (6.7) Missing: 0 | 4 (7.3) Missing: 0 | 4 (9.1) Missing: 0 | <0.001b |
| Baseline PASI score, median [IQR] | 12.0 [8.4] Missing: 1 | 10.1 [8.1] Missing: 1 | 11.1 [10.3] Missing: 0 | 9.0 [6.5] Missing: 0 | 6.8 [5.9] Missing: 0 | 8.5 [8.8] Missing: 0 | <0.001c |

aOne-way analysis of variance (ANOVA); bχ² test; cKruskal–Wallis test.
SD: standard deviation; IQR: interquartile range; BMI: body mass index; PASI: Psoriasis Area and Severity Index.
significantly contribute to the model \((p=0.11)\) and was therefore not incorporated. As a result, overall odds ratios (ORs) are presented instead of ORs for each time-point. Overall, adalimumab (OR 3.2 \((95\% \text{ CI } 2.2–4.7)\) \(p<0.001\)), ustekinumab (OR 4.2 \((2.7–6.3)\) \(p<0.001\)), secukinumab (OR 5.3 \((2.8–6.3)\) \(p<0.001\)), ixekizumab (OR 20.3 \((10.7–38.5)\) \(p<0.001\)), and guselkumab (OR 12.9 \((6.2–27.0)\) \(p<0.001\)) all had a significantly higher probability of reaching PASI90 than etanercept. Furthermore, ixekizumab and guselkumab both had a higher probability of reaching PASI90 than adalimumab (OR 6.3 \((3.6–11.3)\) \(p<0.001\) and OR 4.0 \((2.0–8.1)\) \(p<0.001\), respectively), ustekinumab (OR 4.9 \((2.8–6.6)\) \(p<0.001\) and OR 3.1 \((1.6–6.1)\) \(p=0.001\), respectively) and secukinumab (OR 3.8 \((1.9–7.7)\) \(p<0.001\) and OR 2.4 \((1.1–5.3)\) \(p=0.028\), respectively). Table SIII provides information on model output. Quantitative interpretation of ORs is difficult and therefore, the proportions of patients achieving PASI90/PASI75 were calculated based on the model and visualized for patients with average characteristics (e.g. baseline PASI 12, weight 90 kg). Split for biologic naivety, the proportion of TEs reaching PASI90 within the first year of treatment according to MLR is shown in Fig. 3. Visualizations for PASI75 are shown in Fig. S2. For ixekizumab, the odds of reaching PASI75 (OR 2.7 \((1.3–5.3)\) \(p=0.005\)), but not PASI90 (OR 1.57 \((0.7–3.4)\) \(p=0.261\)), were significantly higher compared with guselkumab. Analyses were repeated using the LOCF method, which showed similar results compared to the original analyses.

Data on LOCF analyses for both LMM and MLR are available on request.

**DISCUSSION**

This prospective study compared the effectiveness of 6 frequently used biologics: etanercept, adalimumab,
ustekinumab, secukinumab, ixekizumab and guselkumab (TNF-α-, IL12/23-, IL17-, and IL23-inhibitors). For all TEs, 9.3% (n=56) was free from psoriasis (PASI100) after 1 year. Absolute PASI ≤ 1, ≤ 3 and ≤ 5 was reached in 21.3% (n=128), 51.8% (n=312), and 72.4% (n=436), and PASI90, PASI75 and PASI50 was reached by 21.8% (n=131), 46.2% (n=278) and 77.2% (n=465) of TEs, respectively. Adjusted for baseline PASI, weight and biologic naïvety, patients on adalimumab, ustekinumab, secukinumab, ixekizumab and guselkumab all had significantly lower PASI-scores after 1 year compared with etanercept. Furthermore, patients on ixekizumab had a significantly lower mean PASI after 1 year compared with adalimumab, ustekinumab and secukinumab. This was also the case for guselkumab compared with adalimumab and secukinumab. The overall, adjusted probability of reaching PASI90 was significantly higher for patients on adalimumab, ustekinumab, secukinumab, ixekizumab and guselkumab compared with patients on etanercept. Patients on ixekizumab and guselkumab also had a higher probability of reaching PASI90 than patients on adalimumab, ustekinumab, and secukinumab.

In systematic reviews and meta-analyses based on RCT data, higher success rates have been reported for IL17- and IL23-inhibitors compared with TNF-α-inhibitors and ustekinumab, which is roughly in line with the current results (3, 4, 13, 14). In real-world effectiveness studies, different biologics have been compared directly, but mostly from 1 or 2 different classes (e.g. TNF-α-inhibitors and ustekinumab) only (6–8, 15–18). Real-world effectiveness studies on long-term (40–76 weeks) effectiveness of IL17- and IL23-inhibitors is accumulating (17, 19–28). In most real-world effectiveness studies, however, relative PASI-targets were used, and the composition of the investigated population differed, e.g. regarding the proportion of biologic-naïve patients, which can explain differences with the current results. Yiu et al. (17) reported higher probabilities of reaching PASI≤2 for secukinumab than for ustekinumab. Schwarz et al. (26) reported proportions of patients reaching PASI≤2 for adalimumab, etanercept, secukinumab and ustekinumab between 51.8–89.6%, which was comparable with reaching PASI≤5 in our cohort (60.1–80.0%). For all biologics in our RWE-cohort, relative PASI targets, such as PASI90, were reached less often than in a Danish and Italian cohort (estimated PASI90 responses for etanercept,
ustekinumab, adalimumab, secukinumab and ixekizumab ranged between 31.7% (etanercept) and 81.0% (ixekizumab). However, baseline PASI scores were not reported (28), or different timeframes for reaching PASI-targets were used (27), hampering direct comparisons.

In another study from the British Association of Dermatologists Biologics and Immunomodulators Register (BADBIR) it has been demonstrated that, in 90% of cases, absolute PASI ≤ 2 was in concordance with reaching PASI90 (29). In the current cohort, we observed that the proportion of patients who reached PASI90 was very similar to the proportion of patients who reached an absolute PASI ≤ 1 at each time-point, although no formal correlation analyses were performed. Discrepancies between these 2 studies are partly due to the difference in mean baseline PASI (15.4 in BADBIR vs 12.0 in our cohort), as it is more difficult to reach a relative PASI improvement in case of low baseline scores. Therefore, patients with low PASI scores at baseline may not achieve relative PASI outcomes, even though absolute treatment response may be excellent in practice. In most RCTs, a fixed percentage in PASI-reduction (e.g. PASI90) is still the primary outcome measure, although some studies have also reported absolute PASI outcomes (30–32). For instance, in the IXORA-S trial, the percentage of patients who reached an absolute PASI ≤ 5 on ixekizumab (88.2%, n = 120) was similar to our results (85.7%, n = 24), whilst the proportion of patients who reached PASI90 was markedly higher (76.5%, n = 105) than in the current study (21.4%, n = 6) at week 52. Displaying absolute PASIs in addition to relative PASIs could lead to more robust comparisons between studies with different designs, either real-world effectiveness studies or RCTs.

A strength of this study is the high external validity due to the real-world practice environment and multicentre, prospective design of BioCAPTURE. LOCF analyses were performed, and led to very similar results compared with the as-treated analyses, showing robustness of the current results. A limitation was that fewer PASIs were available for ixekizumab and guselkumab, due to more recent regulatory approval, and fewer clinical visits due to COVID-19 restrictions. Due to a low number of patients on brodalumab (IL-17 inhibitor), risankizumab and tildrakizumab (IL-23 inhibitors), these relatively newer biologics could not be included in the current analyses. Furthermore, although we performed confounder-correction for baseline PASI, body weight and biologic naivety, residual confounding may still be present due to unmeasured factors.

This prospective, real-world study analysed the comparative effectiveness of the biologics etanercept, adalimumab, ustekinumab, secukinumab, ixekizumab, and guselkumab in patients with moderate to severe psoriasis from the BioCAPTURE registry. Except for a nearly significant difference between the estimated mean PASI scores after 12 months in guselkumab vs ustekinumab (p = 0.062), ixekizumab and guselkumab showed better results compared with the other biologics for both absolute and relative PASI outcomes. However, the proportion of patients who reached PASI90 was relatively low for all biologics, compared with what has been reported in RCTs and other real-world studies. As the therapeutic options for psoriasis continue to expand, ongoing comparative, real-world effectiveness research remains important. Absolute PASI could serve as a more robust outcome measure to compare outcomes of RCTs with real-world effectiveness studies. Replication by other large prospective daily practice cohorts will be key in verifying the current results.

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REFERENCES

1. Bai F, Li GG, Liu Q, Niu X, Li R, Ma H. Short-term efficacy and safety of IL-17, IL-12/23, and IL-23 inhibitors brodalumab, secukinumab, ixekizumab, ustekinumab, guselkumab, tildrakizumab, and risankizumab for the treatment of moderate to severe plaque psoriasis: a systematic review and network meta-analysis of randomized controlled trials. J Immunol Res 2019; 2019: 2546161.
2. Jabbar-Lopez ZK, Yiuz ZZN, Ward V, Exton LS, Mohd Mustapa MF, Samarakere E, et al. Quantitative evaluation of biologic therapy options for psoriasis: a systematic review and network meta-analysis. J Invest Dermatol 2017; 137: 1646–1654.

3. Loos AM, Liu S, Segel C, Ollendorf DA, Pearson SD, Linder JA. Comparative effectiveness of targeted immunomodulators for the treatment of moderate-to-severe plaque psoriasis: a systematic review and network meta-analysis. J Am Acad Dermatol 2019; 81: 135–144.

4. Sawyer LM, Malottki K, Sabry-Grant C, Yasmeen N, Wright MA, et al. Systemic pharmacological treatments for chronic plaque psoriasis: a one-year real-life study in the Latino region, Italy. Expert Opin Biol Ther 2021; 21: 1299–1310.

5. Garcia-Doval J, Venkatachari V, Kimball AB, Naldi L, Arancio L, Gaiani F, et al. Comparative effectiveness of targeted immunomodulators: a 1- and 5-year effectiveness of adalimumab, etanercept and ustekinumab in patients with psoriasis in daily clinical practice: results from the prospective BioCAPTURE registry. Br J Dermatol 2017; 176: 1001–1009.

6. Carrera CG, Dapavo P, Malagoli P, Naldi L, Arancio L, Gaiani F, et al. Real-world experience of adalimumab, etanercept and ustekinumab in moderate-to-severe plaque psoriasis: a network meta-analysis. J Invest Dermatol 2018; 139: 179–187.

7. Strober BE, Bissonnette R, Fiorentino D, Kimball AB, Naldi L, Shear NH, et al. Comparative effectiveness of biological agents for the treatment of psoriasis in a real-world setting: results from a large, prospective, observational study (Psoriasis Longitudinal Assessment and Registry [PSOLAR]). J Am Acad Dermatol 2016; 74: 851–861.e4.

8. Carrera CG, Papavo P, Malagoli P, Naldi L, Arancio L, Gaieni F, et al. PACE study: real-life Psoriasis Area and Severity Index (PASI) 100 response with biological agents in moderate-to-severe psoriasis. J Dermatol Treat 2018; 29: 481–486.

9. Tada Y, Watanabe R, Noma H, Kanai Y, Nomura T, Kaneko K. Short-term effectiveness of biologics in patients with moderate-to-severe plaque psoriasis: a systematic review and network meta-analysis. J Dermatol Sci 2020; 99: 53–61.

10. Cinelli E, Fabbrocini G, Megna M. Efficacy and safety of guselkumab in psoriasis patients who failed ustekinumab and/or anti-interleukin-17 treatment: a real-life 52-week retrospective study. Dermatol Ther 2022; 35: e107–e108.

11. Berglind ND, Hjartardottir A, Öhman A, et al. Comparing the 1- and 5-year effectiveness of adalimumab, etanercept and ustekinumab in moderate-to-severe plaque psoriasis: a network meta-analysis. J Dtsch Dermatol Ges 2021; 19: 47–56.

12. van Lümig PPM, Driessen RJB, Kievit W, Boezeman JBM, van de Kerkhof PCM, Driessen RJ, et al. Comparison of the 1- and 5-year effectiveness of adalimumab, etanercept and ustekinumab in patients with psoriasis in daily clinical practice: results from the prospective BioCAPTURE registry. Br J Dermatol 2017; 176: 1001–1009.

13. Strober BE, Bissonnette R, Fiorentino D, Kimball AB, Naldi L, Shear NH, et al. Comparative effectiveness of biological agents for the treatment of psoriasis in a real-world setting: results from a large, prospective, observational study (Psoriasis Longitudinal Assessment and Registry [PSOLAR]). J Am Acad Dermatol 2016; 74: 851–861.e4.

14. Carrera CG, Papavo P, Malagoli P, Naldi L, Arancio L, Gaieni F, et al. PACE study: real-life Psoriasis Area and Severity Index (PASI) 100 response with biological agents in moderate-to-severe psoriasis. J Dermatol Treat 2018; 29: 481–486.

15. Tada Y, Watanabe R, Noma H, Kanai Y, Nomura T, Kaneko K. Short-term effectiveness of biologics in patients with moderate-to-severe plaque psoriasis: a systematic review and network meta-analysis. J Dermatol Sci 2020; 99: 53–61.

16. Cinelli E, Fabbrocini G, Megna M. Efficacy and safety of guselkumab in psoriasis patients who failed ustekinumab and/or anti-interleukin-17 treatment: a real-life 52-week retrospective study. Dermatol Ther 2022; 35: e107–e108.

17. van Lümig PPM, Driessen RJB, Kievit W, Boezeman JBM, van de Kerkhof PCM, Driessen RJ, et al. Comparison of the 1- and 5-year effectiveness of adalimumab, etanercept and ustekinumab in moderate-to-severe plaque psoriasis: a network meta-analysis. J Dtsch Dermatol Ges 2021; 19: 47–56.

18. Ohata C, Ohyama B, Katayama E, Nakama T. Real-world efficacy and safety of interleukin-17 inhibitors for psoriasis: a single-center experience. J Dermatol 2020; 47: 405–408.

19. Richter L, Vujic Z, Sesti A, Monshi B, Sanlorenzo M, Posch C, et al. Etanercept, adalimumab, and ustekinumab in psoriasis: analysis of 209 treatment series in Austria. J Dtsch Dermatol Ges 2017; 15: 309–317.

20. Yiuz ZZN, Mason KJ, Hampton PJ, Reynolds NJ, Smith CH, Lunt M, et al. Randomized trial replication using observational data for comparative effectiveness of secukinumab and ustekinumab in psoriasis: a study from the British Association of Dermatologists Biologics and Immunomodulators Register. JAMA Dermatol 2021; 157: 66–73.