

Impact of early treatment of psoriasis on disease recurrence—Results from the STEPIn study

Lars Iversen, MD, DMSc,^a Richard G. Langley, MD,^b Johann E. Gudjonsson, MD, PhD,^c Liv Eidsmo, MD, PhD,^{d,e} Joanna Narbutt, MD,^f Rafal Czajkowski, MD, PhD,^g Andreas Pinter, MD,^h Andrew E. Pink, MD, PhD,ⁱ Raquel Rivera-Diaz, MD,^j Dheeraj A. Gianchandani, MS,^k Frank Kolbinger, PhD,^l Weibin Bao, MS,^m Corine Gaillez, MD,ⁿ Piotr Jagiello, MD,ⁿ and Curdin Conrad, MD^o

Background: Benefits of early intervention with secukinumab in patients with new-onset plaque psoriasis (disease duration: ≤ 1 year) were investigated in STEPIn study (NCT03020199).

Objective: To evaluate the proportion of patients maintaining sustained skin clearance in the treatment-free follow-up period up to week (W) 104 following withdrawal of secukinumab and narrow band ultraviolet B at W52.

Methods: Wald test was used for analysis of proportion of patients achieving PASI 90 (key secondary endpoint) from W52 through W104. Missing values were imputed using nonresponder imputation approach.

Results: Overall, 93.5% (72/77) of patients completed 52 weeks in the secukinumab arm. At W104, 20.8% of patients who previously received secukinumab sustained PASI 90 responses. At W104, 11.7%, 22.1%, and 24.7% of patients previously treated with secukinumab sustained a PASI 100 response, IGA mod 2011 0/1 and PASI < 3 . Among secukinumab treated patients, 44% (29/66) who entered the treatment-free follow-up period at W52 did not relapse (loss of 50% of the maximum improvement in PASI following treatment cessation) until W104.

Limitation: Absence of blinding due to open-label nature of the study.

From the Department of Dermatology and Venereology, Aarhus University Hospital, Aarhus, Denmark^a; Division of Dermatology, Department of Medicine, Dalhousie University, Halifax, Nova Scotia, Canada^b; Department of Dermatology, University of Michigan, Ann Arbor, Michigan^c; Department of Medicine Solna, Karolinska Institutet, Stockholm, Sweden^d; Gastro-/HUD/Rheuma, Karolinska University Hospital, Stockholm, Sweden^e; Dermoklinika Medical Centre, Lodz, Poland^f; DermoDent Centrum Medyczne Aldona Czajkowska Rafał Czajkowski s.c., Osielsko, Poland^g; Department of Dermatology, Venereology and Allergology, Goethe-Universität Frankfurt am Main, Frankfurt, Germany^h; St John's Institute of Dermatology, Guy's and St Thomas' NHS Foundation Trust and King's College London, London, UKⁱ; Department of Dermatology, Hospital Universitario 12 de Octubre, Madrid, Spain^j; Novartis Healthcare Private Limited, Hyderabad, India^k; Department of Immunology, Novartis Biomedical Research, Basel, Switzerland^l; Novartis Pharmaceuticals Corporation, East Hanover, New Jersey^m; Novartis IMI AG, Basel, Switzerlandⁿ; and Department of Dermatology, CHUV Lausanne University Hospital and University of Lausanne (UNIL), Lausanne, Switzerland.^o

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regulations. The study protocol was approved by the independent ethics committee or institutional review board of each participating center. A written informed consent was obtained from each study participant before enrollment in the study.

Data availability: Novartis is committed to sharing with qualified external researchers, access to patient-level data, and supporting clinical documents from eligible studies. These requests are reviewed and approved by an independent review panel on the basis of scientific merit. All data provided are anonymized to respect the privacy of patients who have participated in the trial in line with the applicable laws and regulations. This trial data availability is according to the criteria and process described on www.clinicalstudydatarequest.com.

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Correspondence to: Prof Curdin Conrad, MD, Department of Dermatology, CHUV Lausanne University Hospital and University of Lausanne (UNIL), Rue de Bugnon 21, CH-1011 Lausanne, Switzerland. E-mail: Curdin.Conrad@chuv.ch.

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Conclusions: Early intervention with secukinumab maintains efficacy in a subset of patients with new-onset moderate to severe psoriasis, 1-year following treatment withdrawal. (J Am Acad Dermatol <https://doi.org/10.1016/j.jaad.2026.03.050>.)

Key words: disease modification; early intervention; IGA mod 2011 0/1; new-onset; PASI 90; phototherapy; plaque psoriasis; secukinumab; STEPIn; treatment withdrawal.

INTRODUCTION

The treatment landscape of psoriasis (PsO) has significantly improved with the widespread adoption of biologics. Evidence indicates that biologics for PsO are both effective and safer compared with conventional therapies.^{1,2} Despite advances in understanding immunopathogenesis and availability of multiple treatments, nearly half of the patients with moderate to severe PsO face delays of >3 years in using systemic agents, including biologics.^{3,4}

Biologic treatment of patients with plaque PsO has been studied predominantly in patients with long-standing disease and with a history of prior systemic treatment. In pivotal secukinumab phase 3 trials, ERASURE and FIXTURE, the mean disease duration in patients with moderate to severe plaque PsO was ~17 years⁵ and these patients were more likely to have 2 to 4 comorbidities at the time of diagnosis⁶ underscoring the importance of early intervention for optimal disease management.

Early intervention reduces cutaneous symptoms and may decrease the risk of developing long-term adverse outcomes such as cardiovascular disease.^{3,7,8} An inverse relationship between time to relapse and shorter disease duration further emphasizes the positive impact of early intervention in improving the long-term disease outcomes.⁹ Early intervention is beneficial in rheumatoid arthritis and Crohn disease, resulting in improved long-term clinical outcomes.^{10,11} However, the evidence supporting early intervention in PsO still remains limited and inconsistent.^{3,12,13}

Secukinumab is a recombinant, high-affinity, fully human immunoglobulin G1κ monoclonal antibody that selectively binds and neutralizes IL-17A,¹⁴ with a proven efficacy and favorable safety in the treatment of multiple immune-mediated inflammatory diseases.¹⁵⁻¹⁹

STEPIn (NCT03020199) demonstrated early intervention and blockade of IL-17A with secukinumab provided high and sustained skin clearance versus narrow band ultraviolet B (nb-UVB) following 52 weeks of treatment in patients with new-onset

CAPSULE SUMMARY

- Early intervention with secukinumab maintains efficacy in subset of patients with new-onset moderate to severe psoriasis, 1-year after treatment withdrawal.
- STEPIn included patients with disease duration of ≤1 year. The results indicate potential of early intervention to improve primary response, long-term outcomes, and reduce disease burden.

(disease duration of ≤1 year) moderate to severe plaque PsO.²⁰ Here, we evaluated the proportion of patients who maintained sustained skin clearance off-treatment with secukinumab and nb-UVB from week 52 through week 104.

MATERIALS AND METHODS

Study design and patients

STEPIn main study was a randomized, open-label, multicenter study to investigate the early intervention with secukinumab 300 mg via subcutaneous (SC) injection versus treatment with nb-UVB over 52 weeks in patients with new-onset (disease duration of ≤1 year) moderate to severe plaque PsO. The study design and treatment regimen have been published previously.^{20,21} Following 52 weeks of treatment, patients who achieved ≥50% improvement in Psoriasis Area and Severity Index (PASI) in both treatment arms did not receive any further study treatment and were followed up for disease activity up to week 104.

All patients in the study had new-onset moderate to severe plaque psoriasis, defined at screening and baseline by all the following: PASI ≥10; body surface area (BSA) ≥10%; and Investigator's Global Assessment modified 2011 (IGA mod 2011) ≥3, with appearance of the first PsO plaques within the 12 months before randomization. Patients were naïve to any previous systemic treatment and phototherapy. Here, we present the results of the treatment-free follow-up period of up to week 104 (after 1 year of treatment withdrawal).

Outcomes and assessments

At Week 52, a high dropout rate (40.8%) was observed in the nb-UVB arm versus secukinumab arm (7.0%), due to the lack of efficacy or other reasons (Supplementary Fig 1, available via Mendeley at <https://data.mendeley.com/datasets/8fkgxty3v4/1>). This led to a selection bias of the nb-UVB-treated patients entering the treatment-free follow-up period. Therefore, the efficacy results for

Abbreviations used:

CI:	confidence interval
DLQI:	Dermatology Life Quality Index
IGA mod 2011 0/1:	Investigator's Global Assessment 2011 modified version score of 0 or 1
LOCF:	last observation carried forward
nb-UVB:	narrow-band ultraviolet B
NRI:	nonresponder imputation
OR:	odds ratio
PASI:	Psoriasis Area and Severity Index
PsO:	psoriasis
SAPIS:	subject's assessment of pain, itching and scaling
SEC:	secukinumab
SGA:	subject's global assessment of psoriatic disease response

the secukinumab arm during the off-treatment follow-up period are presented without nb-UVB control.

Efficacy assessments included the proportion of all randomized patients who achieved PASI 90 (key secondary endpoint) and PASI 100 responses at week 104. IGA mod 2011 0/1 was measured from baseline through week 104. Exploratory endpoints included time to relapse (defined as the loss of 50% of the maximum improvement in PASI following treatment cessation according to the EMA guideline),²² absolute PASI response (<3, <1) from the baseline through week 104.

Patient-reported outcomes, collected through week 104 were as follows:

- Dermatology Life Quality Index (DLQI): 10 questions rated from 0 (not at all) to 3 (very much) and a total score (0 to 30); higher total score indicated a greater impairment
- Subject's Assessment of Pain, Itching and Scaling (SAPIS): 11-point numeric rating questionnaire (0: absence of pain/itching/scaling over the past 24 hours; 10: pain/itching/scaling as bad as it could be over the past 24 hours)
- Subject's Global Assessment (SGA) of psoriatic disease during the previous week was measured on a 100-mm visual analogue scale (0: not severe; 100: very severe)

Statistical analyses

For efficacy endpoints, NRI (nonresponder imputation) and LOCF (last observation carry forward) analyses were presented for dichotomous and continuous variables, respectively. All analyses were based on the modified full analysis set

(including patients who were randomized and took at least 1 dose of study treatment). Wald test was used for treatment comparisons for PASI 90 at week 104.

Ethical guidance

This study was designed and conducted in accordance with the ICH Harmonized Tripartite Guidelines for Good Clinical Practice and the Declaration of Helsinki, with applicable local regulations. The study protocol was approved by the independent ethics committee or institutional review board of each participating center. A written informed consent was obtained from each study participant before enrollment in the study.

RESULTS

Patient disposition

Among the randomized patients who received ≥ 1 dose of study treatment, 72 of 77 (93.5%) versus 45 of 76 patients (59.2%) in the secukinumab and nb-UVB arm completed 52 weeks, respectively. Of those who completed week 52 treatment, 66 patients from the secukinumab and 35 patients from the nb-UVB arm who had $\geq 50\%$ of improvement in PASI at week 52 entered the treatment-free follow-up period and had at least 1 PASI assessment after week 52. Of all the patients who received ≥ 1 dose of study treatment, 32 of 77 (41.5%) and 20 of 76 patients (26.3%) completed week 104 from secukinumab and nb-UVB arm, respectively. Reasons for the discontinuation are shown in Supplementary Fig 1, available via Mendeley at <https://data.mendeley.com/datasets/8fkgxty3v4/1>.

Patient demographics and baseline characteristics of patients entering the treatment-free follow-up period

Demographics and baseline clinical characteristics of the patients have been previously reported and were similar across both treatment arms.²⁰ Supplementary Table I, available via Mendeley at <https://data.mendeley.com/datasets/8fkgxty3v4/1> presents the clinical characteristics of patients who entered treatment-free follow-up period at week 52.

Efficacy outcomes

The PASI 90 response (key secondary endpoint) was achieved by 20.8% (16 of 77) at week 104 (Fig 1). IGA mod 2011 0/1 response was achieved by 22.1% (17 of 77) of patients previously treated with secukinumab at week 104 (Fig 2).

At week 104, 11.7% (9 of 77) of patients previously treated with secukinumab maintained a PASI 100 response (Supplementary Fig 2, available via Mendeley at <https://data.mendeley.com/datasets/>

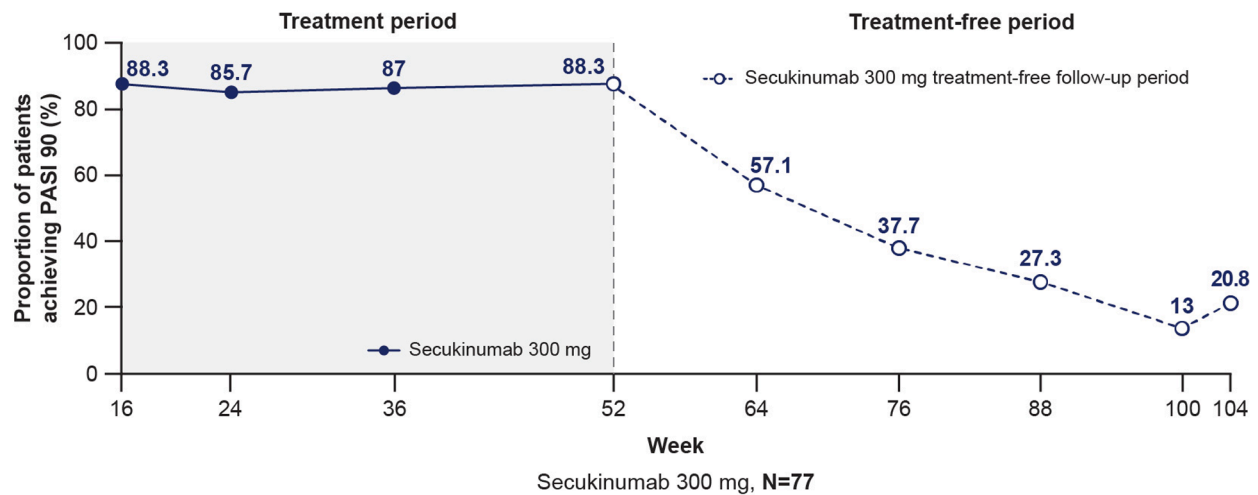


Fig 1. Proportion of patients with PASI 90 response through week 104 (NRI). Missing values are imputed via nonresponder imputation (NRI). Modified full analysis set: all subjects who are randomized and took at least 1 dose of study treatment. *CI*, Confidence interval; *m*, number of subjects evaluable; *n*, number of subjects with observed PASI 90 response; *NRI*, non-responder imputation; *OR*, odds ratio; *PASI*, Psoriasis Area and Severity Index.

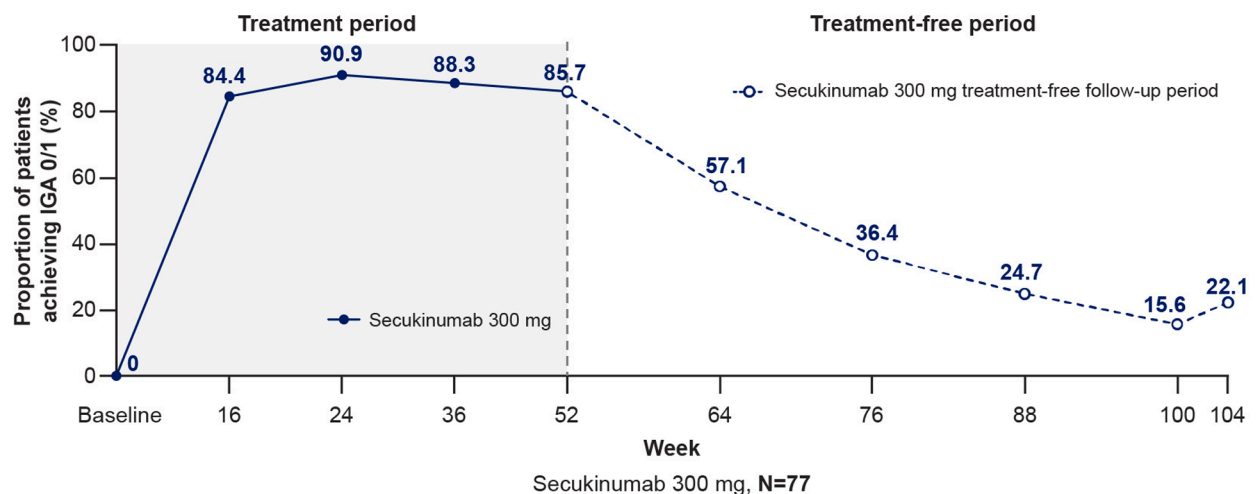


Fig 2. Proportion of patients with IGA mod 2011 0/1 response from baseline to week 104 (NRI). Missing values are imputed via nonresponder imputation (NRI). Modified full analysis set: all subjects who are randomized and took at least 1 dose of study treatment. IGA mod 2011 0/1, Investigator's Global Assessment, 2011 modified version score of 0 or 1.

8fkgxty3v4/1). The proportion of patients achieving a PASI <3 or PASI <1 at week 104 was 24.7% and 18.2% in patients previously treated with secukinumab, respectively (Fig 3). The median PASI score (Q1, Q3) at baseline, 15.6 (12.8, 18.8) reduced following treatment with secukinumab with complete disease control through week 64 (median PASI 0) and low disease activity at week 104 (3.0 [0.2-9.5]) (Supplementary Fig 3, available via Mendeley at <https://data.mendeley.com/datasets/8fkgxty3v4/1>).

In a subgroup of patients stratified by disease duration (<6 months [*N* = 35] and 6-12 months [*N* = 42]), over 85% of patients previously treated with secukinumab rapidly achieved and sustained PASI 90 response till week 52 in both subgroups. The PASI 90 response at week 104 was numerically higher but statistically not significant in patients of the <6 months versus 6 to 12 months disease duration group (28.6% versus 14.3%) (Supplementary Fig 4, A, available via Mendeley at <https://data.mendeley.com/datasets/8fkgxty3v4/1>).

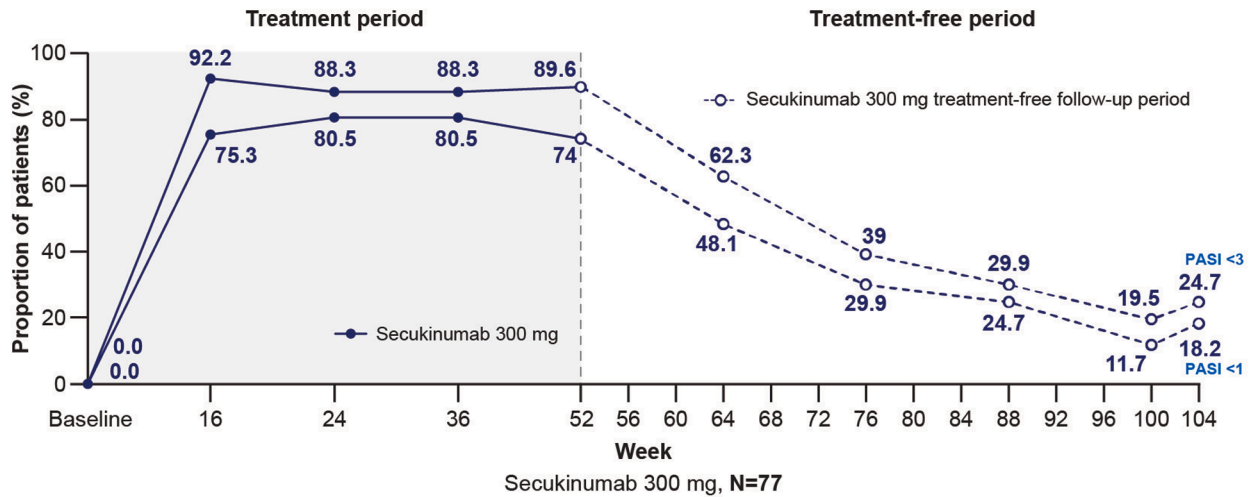


Fig 3. Proportion of patients with PASI score <3 and PASI score <1 from baseline to week 104 (NRI). Missing values are imputed via nonresponder imputation (NRI). Modified full analysis set: all subjects who are randomized and took at least 1 dose of study treatment. *PASI*, Psoriasis Area and Severity Index.

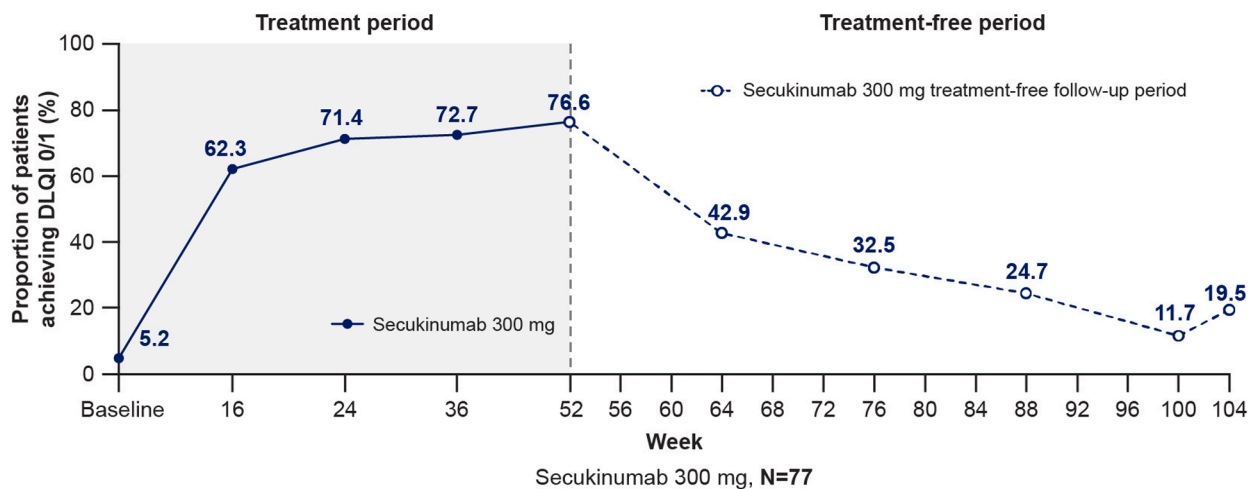


Fig 4. Proportion of patients with DLQI 0/1 response up to week 104 (NRI) Modified full analysis set: all subjects who are randomized and took at least 1 dose of study treatment. Wald test by visit till week 52 (missing values are replaced by a nonresponder imputation method). *DLQI*, Dermatology Life Quality Index.

[com/datasets/8fkgxty3v4/1](https://data.mendeley.com/datasets/8fkgxty3v4/1)) with a similar trend for PASI 100, PASI <1 and IGA mod 2011 0/1 response highlighting a higher response in very early disease (Supplementary Fig 4, B-D, available via Mendeley at <https://data.mendeley.com/datasets/8fkgxty3v4/1>).

Among the patients who entered the treatment-free follow-up period, 29 of 66 patients (44%) in the secukinumab arm did not relapse between week 52 and week 104; among those who relapsed, the mean time to relapse was 32 weeks after treatment withdrawal in the secukinumab arm (Supplementary Table

II, available via Mendeley at <https://data.mendeley.com/datasets/8fkgxty3v4/1>).

The proportion of patients achieving a DLQI of 0 or 1 (little to no impact on quality of life) was 19.5% at 1 year after secukinumab treatment withdrawal (Fig 4). The mean change in SGA from baseline to week 104 was -33.6 in patients previously treated with secukinumab (Fig 5). Patients treated with secukinumab in the first 52 weeks had a reduction in pain (1.7) as measured by SAPIS score at week 104. A reduction in pain, itching, and scaling using SAPIS score was also observed from baseline to week 104

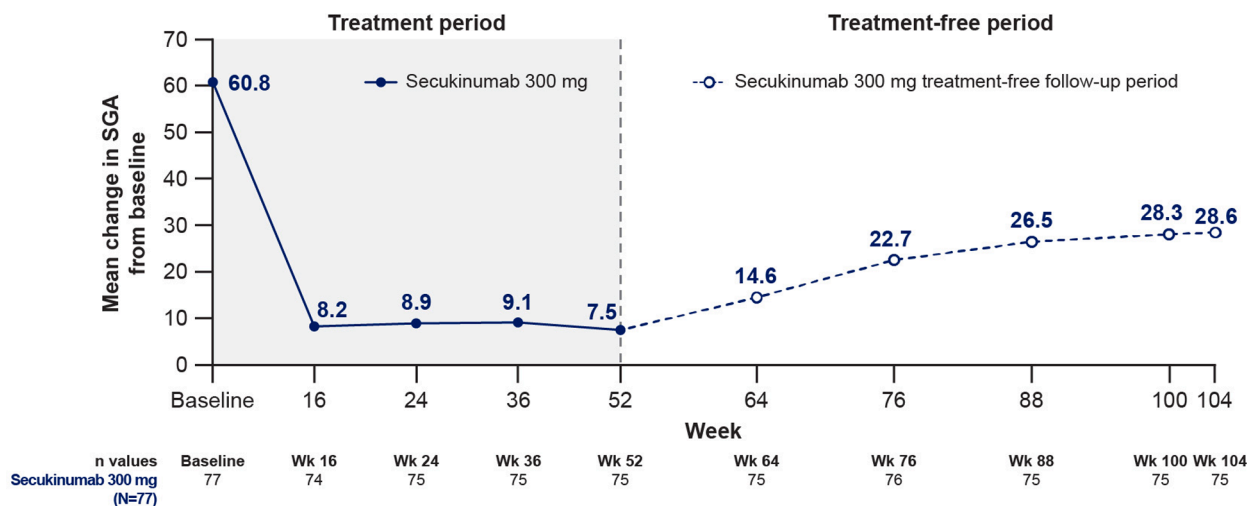


Fig 5. Mean values for subject's global assessment from baseline up to week 104 (LOCF). Modified full analysis set: all subjects who are randomized and took at least 1 dose of study treatment. LOCF analysis used. Subject's global assessment of psoriasis was captured using a 100-mm visual analogue scale, where 0 = "Not severe" and 100 = "Very severe." LOCF, Last observation carried forward; *n*, patients with a value at both baseline and the respective post-baseline visit; SGA, subject's global assessment of psoriatic disease response.

(Supplementary Fig 5, A-C, available via Mendeley at <https://data.mendeley.com/datasets/8fkgxty3v4/1>).

Supplementary Table III, available via Mendeley at <https://data.mendeley.com/datasets/8fkgxty3v4/1> presents the data for efficacy endpoints in the patients previously treated with nb-UVB.

DISCUSSION

STEPIn investigated the effect of early intervention with secukinumab on sustained skin clearance in systemic treatment-naïve patients with new-onset PsO (≤ 1 year since diagnosis). Following a year after treatment withdrawal, responses were maintained in a subset of patients with moderate to severe PsO as indicated by PASI 90 response, PASI score < 1 and PASI < 3 .

When compared with results from the ERASURE and FIXTURE trials, which were performed in patients with long-standing PsO, early treatment with secukinumab was associated with better clinical outcomes in terms of efficacy. In the STEPIn study, 91.1% of patients with a disease duration of < 1 year achieved PASI 90 response at week 52 versus 72.8% of patients with long-standing PsO (disease duration ~ 17 years)⁵ in the ERASURE and FIXTURE studies.²³ These results are consistent with the data from early intervention with the IL-23 blocker, guselkumab demonstrating the association of shorter disease duration with improved clinical outcomes.²⁴ Patients with shorter disease duration (< 1 year) demonstrated markedly higher PASI 90 response rates with secukinumab compared to

those with long-standing disease, reinforcing the clinical value of initiating therapy early.

Besides improved clinical efficacy under treatment, patients with shorter disease duration and lower baseline PASI may remain relapse-free for longer following treatment discontinuation with secukinumab.⁹ This observation is further supported by our data, a higher percentage of patients in STEPIn maintained PASI 90 at week 52 (91.1% versus 72.8%)^{20,23} and remained relapse-free (44% versus 20.8%),⁹ 1 year after withdrawal of secukinumab treatment versus long-standing PsO patients in ERASURE and FIXTURE studies indicating potential of secukinumab to modify the natural disease course. However, in the ERASURE/FIXTURE study the patients had more severe baseline disease (baseline PASI = 22.8) compared to STEPIn (baseline PASI = 18.0).

Further, in a subgroup of patients stratified by disease duration, PASI 90, PASI 100, PASI < 1 and IGA mod 2011 0/1 response was numerically even higher in patients of the < 6 months versus 6 to 12 months disease duration group highlighting a greater treatment response in very early disease.

The recurrence of psoriatic lesions following treatment withdrawal with biologics remains one of the challenges in the management of PsO.²⁵ Therefore, treatment discontinuation is usually not advised in patients with moderate to severe PsO.⁹ This study demonstrates maintenance of remission occurred in few patients even in absence of active treatment and prolonged relapse-free interval in a proportion of

patients. This may be helpful for patients who temporarily discontinue treatment due to surgeries, vaccinations, or pregnancy. The reason some patients maintain efficacy after treatment withdrawal, while the disease recurs in others, remains unclear. The gradual loss of response seen in patients previously treated with secukinumab following treatment withdrawal may be explained by a washout of the drug over 3 half-lives.²⁶ However, even 1 year after drug withdrawal, sustained PASI 90/100 responses to secukinumab were still maintained in 20.8% and 11.7% of the patients, respectively. Thus, the maintenance of efficacy upon treatment cessation in these patients cannot be explained by residual drug levels but might rather be mediated through modulation of disease memory and may represent disease modification in plaque PsO. This hypothesis is further supported by molecular data from the STEPIn mechanistic Sub study revealing that secukinumab treatment normalized the DNA methylation differences observed at baseline in lesional skin of patients with new-onset PsO to levels observed in non-lesional skin. In contrast, a residual difference in DNA methylation remained after 52 weeks of treatment in patients with long-standing PsO (disease duration of ≥ 5 years), which may represent a “molecular scar” at the epigenetic level. The absence of this molecular scar in the patients with new-onset PsO indicates a positive clinical and molecular impact on the disease progression and long-term tissue damage with an early intervention.²⁷

Several cells have been implicated in the mechanism of PsO relapse. Transient production of type 1 IFN by plasmacytoid dendritic cells is a key event in early disease.²⁸ Skin resident memory T cells producing IL-17, which persist in resolved psoriatic lesions,²⁹ and their crosstalk with IL-23-producing dendritic cells are critical for disease recurrence and maintaining chronic skin inflammation.³⁰ Moreover, innate immune cells and keratinocyte stem cells retain an inflammatory memory and might be relevant drivers of relapse. It remains to be shown which of these cells are involved and how they can potentially be modified through therapeutic intervention. UVB phototherapy depletes T cells from the skin and to reduce type 1 IFN production.³¹ These mechanisms could provide an explanation for the extended time to relapse upon withdrawal of phototherapy,³² which was reported in a selected subset of patients in the nb-UVB treatment arm of the current study.

Strengths and limitations

The strengths of the study include its unique and novel design of early intervention followed by a withdrawal period in newly diagnosed patients with PsO. However, the absence of blinding between nb-

UVB and secukinumab arm represents a limitation of the study. A high proportion of patients who received nb-UVB treatment dropped out due to the lack of efficacy or other reasons during the first year. Therefore, only a few patients receiving nb-UVB remained in the treatment-free follow-up period representing a relevant selection bias. This is consistent with the previous nb-UVB trials highlighting a subpopulation of complete responders who continue to maintain a response even after treatment withdrawal.^{32,33} This indicates that phototherapy selects responders early on, who then continue to maintain the response even after treatment withdrawal.

Hence, the efficacy results of the treatment withdrawal period focused on secukinumab data without side-by-side comparison with nb-UVB to prevent any potential bias of interpretation across the 2 treatment groups. It is important to note that no definitive conclusions regarding the maintenance of response for nb-UVB versus secukinumab can be drawn from this study, given the presence of selection bias.

CONCLUSION

Sustained skin responses were maintained in few patients with moderate to severe new-onset PsO, 1 year after treatment withdrawal. This indicates that early intervention with secukinumab in newly diagnosed PsO may lead to disease modification in few patients with better long-term improvement and reduced disease burden. These findings, together with the observation that IL-17 targeting biologics are more effective if used early in the disease course, support a change in the management of patients with moderate to severe plaque PsO towards proactive early intervention.

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Conflicts of interest

Iversen has served as a consultant and/or paid speaker for and/or participated in clinical trials sponsored by AbbVie, Amgen, Astra Zeneca, BMS, Boehringer Ingelheim, Celgene, Centocor, Eli Lilly, Janssen Cilag, Kyowa, Leo Pharma, MSD, Novartis, Pfizer, Regranion, Samsung, Union Therapeutics, UCB. LI is also employed by

MC2 Therapeutics A/S. Langley has served as principal investigator for and is a paid member of the scientific advisory board or served as a speaker for AbbVie, Amgen, Celgene, Janssen, Leo, Lilly, Merck, Novartis, Pfizer, UCB, Union, and Boehringer Ingelheim. Gudjonsson reports grants from Almirall, Eli Lilly, Celgene, AbbVie, and personal fees from Almirall, Anaptys Bio, Eli Lilly, Novartis, and AbbVie, outside the submitted work; and is partly supported by the National Institutes of Health (P30-AR075043 and R01-AR069071), the National Psoriasis Foundation, and by the Taubman Medical Research Institute. Eidsmo has received consultancy fees from Galderma, Leo Pharma, Novartis, and Pfizer and investigator-initiated research grants from MSD, Novartis, and Pfizer. Narbutt has served as a consultant and/or paid speaker for and/or participated in clinical trials sponsored by AbbVie, Almirall, Amgen, BMS, Boehringer Ingelheim, Celgene, Eli Lilly, Janssen Cilag, Sanofi, Regeneron, Sandoz, Leo Pharma, MSD, Novartis, Pfizer, Samsung, Union Therapeutics, and UCB. Czajkowski has served as a consultant and/or paid speaker for and/or participated in clinical trials sponsored by AbbVie, BMS, Eli Lilly, Janssen Cilag, Regeneron, Leo Pharma, MSD, Novartis, Pfizer, Samsung, and UCB. Pinter has worked as an investigator and/or speaker and/or advisor for AbbVie, Almirall-Hermal, Amgen, Biogen Idec, Boehringer Ingelheim, Celgene, GSK, Eli Lilly, Galderma, Hexal, Janssen, LEO Pharma, MC2, Medac, Merck Serono, Mitsubishi, MSD, Novartis, Pascoe, Pfizer, Tigercat Pharma, Regeneron, Roche, Sandoz Biopharmaceuticals, Sanofi-Genzyme, Schering-Plough, and UCB. Pink has acted as an advisor, speaker, investigator, or received research or educational support from Sanofi, AbbVie, Eli Lilly, Pfizer, Galderma, BI, Novartis, Almirall, La-Roche Posay, LEO, UCB, BMS, Amgen, Celgene, Novartis, and Janssen. Rivera-Diaz has worked as an investigator/speaker and/or advisor for Abbvie, Almirall, Amgen, Boehringer Ingelheim, BMS, Johnson & Johnson, Leo Pharma, Eli Lilly, Pfizer, Sanofi, Novartis, and UCB. Gianchandani is an employee of Novartis. Jagiello was an employee of Novartis during the conduct of the study. Kolbinger and Bao are employees and own stocks of Novartis. C. Gaillez is an employee of Novartis and owns stocks of Novartis and BMS. Conrad served as a scientific advisor and/or clinical study investigator and/or paid speaker for AbbVie, Actelion, Almirall, Amgen, Boehringer Ingelheim, BMS, Celgene, Galderma, Incyte, Janssen, LEO Pharma, Eli Lilly, MSD, Novartis, Pfizer, Samsung, Sanofi, and UCB.

REFERENCES

1. Armstrong AW, Puig L, Joshi A, et al. Comparison of biologics and oral treatments for plaque psoriasis: a meta-analysis. *JAMA Dermatol.* 2020;156(3):258-269.
2. Thatiparthi A, Martin A, Liu J, Egeberg A, Wu JJ. Biologic treatment algorithms for moderate-to-severe psoriasis with comorbid conditions and special populations: a review. *Am J Clin Dermatol.* 2021;22(4):425-442.
3. Ben Abdallah H, Emmanuel T, Bregnhøj A, Johansen C, Iversen L. Early intervention and disease memory in psoriasis: a literature review. *JEADV Clin Pract.* 2022;1(4):307-316.
4. Maza A, Richard MA, Aubin F, et al. Significant delay in the introduction of systemic treatment of moderate to severe psoriasis: a prospective multicentre observational study in outpatients from hospital dermatology departments in France. *Br J Dermatol.* 2012;167(3):643-648.
5. Langley RG, Elewski BE, Lebwohl M, et al. Secukinumab in plaque psoriasis—results of two phase 3 trials. *N Engl J Med.* 2014;371(4):326-338.
6. Buja A, Miatton A, Cozzolino C, et al. The prevalent comorbidity at the onset of psoriasis diagnosis. *Dermatol Ther (Heidelb).* 2023;13:2093-2105.
7. Takeshita J, Grewal S, Langan SM, et al. Psoriasis and comorbid diseases: epidemiology. *J Am Acad Dermatol.* 2017;76(3):377-390.
8. Bellinato F, Chiricozzi A, Piaserico S, Targher G, Gisondi P. Could targeted pharmacotherapies exert a "disease modification effect" in patients with chronic plaque psoriasis? *Int J Mol Sci.* 2022;23(21):12849.
9. Lebwohl M, Iversen L, Eidsmo L, et al. Investigation of plaque psoriasis relapse after secukinumab withdrawal in patients from two phase III studies. *Clin Exp Dermatol.* 2024;49(8):793-800.
10. Berg DR, Colombel JF, Ungaro R. The role of early biologic therapy in inflammatory bowel disease. *Inflamm Bowel Dis.* 2019;25(12):1896-1905.
11. Deane KD, Holers VM. Rheumatoid arthritis pathogenesis, prediction, and prevention: an emerging paradigm shift. *Arthritis Rheumatol.* 2021;73(2):181-193.
12. Girolomoni G, Griffiths CEM, Krueger J, et al. Early intervention in psoriasis and immune-mediated inflammatory diseases: a hypothesis paper. *J Dermatol Treat.* 2015;26(2):103-112.
13. Felix PAO, Sampaio AL, Silva BL, Viana ALP. Early intervention in psoriasis: where do we go from here? *Front Med (Lausanne).* 2022;9:1027347.
14. Kolbinger F, Di Padova F, Deodhar A, et al. Secukinumab for the treatment of psoriasis, psoriatic arthritis, and axial spondyloarthritis: physical and pharmacological properties underlie the observed clinical efficacy and safety. *Pharmacol Ther.* 2022;229:107925.
15. Garnock-Jones KP. Secukinumab: a review in moderate to severe plaque psoriasis. *Am J Clin Dermatol.* 2015;16(4):323-330.
16. Blair HA. Secukinumab: a review in psoriatic arthritis. *Drugs.* 2021;81(4):483-494.
17. Blair HA, Dhillon S. Secukinumab: a review in ankylosing spondylitis. *Drugs.* 2016;76(10):1023-1030.
18. Brunner HI, Foeldvari I, Alexeeva E, et al. *Ann Rheum Dis.* 2023; 82:154-160.
19. Kimball AB, Jemec GBE, Alavi A, et al. Secukinumab in moderate-to-severe hidradenitis suppurativa (SUNSHINE and SUNRISE): week 16 and week 52 results of two identical, multicentre, randomised, placebo-controlled, double-blind phase 3 trials. *Lancet.* 2023;401(10378):747-761.
20. Iversen L, Conrad C, Eidsmo L, et al. Secukinumab demonstrates superiority over narrow-band ultraviolet B phototherapy in new-onset moderate to severe plaque psoriasis patients: week 52 results from the STEPIn study. *J Eur Acad Dermatol Venereol.* 2023;37(5):1004-1016.
21. Iversen L, Eidsmo L, Austad J, et al. Secukinumab treatment in new-onset psoriasis: aiming to understand the potential for disease modification - rationale and design of the randomized, multicenter STEPIn study. *J Eur Acad Dermatol Venereol.* 2018; 32(11):1930-1939.
22. Guideline on clinical investigation of medicinal products indicated for the treatment of psoriasis. European Medicines Agency. Committee for medicinal products for human use (CHMP). CHMP/EWP/2454/02 corr, 2004. Accessed February 3, 2026.

- https://www.ema.europa.eu/en/documents/scientific-guideline/guideline-clinical-investigation-medicinal-products-indicated-treatment-psoriasis_en.pdf
23. Langley RG, Sofen H, Dei-Cas I, et al. Secukinumab long-term efficacy and safety in psoriasis through to year 5 of treatment: results of a randomized extension of the phase III ERASURE and FIXTURE trials. *Br J Dermatol*. 2023;188(2):198-207.
 24. Schäkel K, Reich K, Asadullah K, et al. Early disease intervention with guselkumab in psoriasis leads to a higher rate of stable complete skin clearance ('clinical super response'): week 28 results from the ongoing phase IIIb randomized, double-blind, parallel-group, GUIDE study. *J Eur Acad Dermatol Venereol*. 2023;37(10):2016-2027.
 25. Masson Regnault M, Shourick J, Jendoubi F, Tauber M, Paul C. Time to relapse after discontinuing systemic treatment for psoriasis: a systematic review. *Am J Clin Dermatol*. 2022;23(4):433-447.
 26. Bruin G, Loesche C, Nyirady J, Sander O. Population pharmacokinetic modeling of secukinumab in patients with moderate to severe psoriasis. *J Clin Pharmacol*. 2017;57(7):876-885.
 27. Conrad C, Gaulis S, Bier K, et al. (Poster 828). Early intervention with secukinumab may affect the establishment of tissue memory in psoriasis: results from a DNA methylation analysis. *J Invest Dermatol*. 2023;143(5):S142.
 28. Conrad C, Gilliet M. Psoriasis: from pathogenesis to targeted therapies. *Clin Rev Allergy Immunol*. 2018;54(1):102-113.
 29. Matos TR, O'Malley JT, Lowry EL, et al. Clinically resolved psoriatic lesions contain psoriasis-specific IL-17-producing $\alpha\beta$ T cell clones. *J Clin Invest*. 2017;127:4031-4041.
 30. Sortebeck D, Schoenfeldt T, Duvetorp A, Agerholm-Nielsen R, Eidsmo L. Skin-resident T cells contribute to the dynamic disease manifestations of psoriasis. *J Immunol*. 2024;213(9):1267-1277.
 31. Rác E, Prens EP, Kurek D, et al. Effective treatment of psoriasis with narrow-band UVB phototherapy is linked to suppression of the IFN and Th17 pathways. *J Invest Dermatol*. 2011;131(7):1547-1558.
 32. Yones SS, Palmer RA, Garibaldinos TT, Hawk JLM. Randomized double-blind trial of the treatment of chronic plaque psoriasis: efficacy of psoralen-UV-A therapy vs narrowband UV-B therapy. *Arch Dermatol*. 2006;142(7):836-842.
 33. Archier E, Devaux S, Castela E, et al. Efficacy of psoralen UV-A therapy vs. narrowband UV-B therapy in chronic plaque psoriasis: a systematic literature review. *J Eur Acad Dermatol Venereol*. 2012;26(Suppl 3):11-21.