A RESEARCH REVIEW™ SPEAKER SERIES

New Treatments in Psoriasis: Secukinumab, IL-17 and Chronic Plaque Psoriasis

Making Education Easy 2018

Dr John Sullivan
MBBS (Hons), MSc (Pharm), FACD

Dr Sullivan has experience as a consultant dermatologist and senior staff specialist at two teaching hospitals, Westmead Hospital and Liverpool Hospital, at the latter he was appointed as head of the Dermatology Department. He has also been a consultant with the Skin & Cancer Foundation Australia where he was director of product testing and involved in research and clinical trials. Dr Sullivan has held a conjoint appointment as a senior lecturer at the University of New South Wales and spent considerable time in private practice in Sydney.

John has authored several textbook chapters and has published a number of editorials and articles in medical journals including the British Medical Journal and British Journal of Dermatology. He is involved in teaching and training dermatology registrars and speaks regularly at national and international meetings.

Abbreviations used in this review

ACR = American College of Rheumatology
DLQI = Dermatology Life Quality Index
IBD = inflammatory bowel disease
IGA = Investigator's Global Assessment
IL = interleukin
IV = intravenous
MS = multiple sclerosis
NAPSI = Nail Psoriasis Severity Index
PASI = Psoriasis Area and Severity Index
ppIGA = palmoplantar psoriasis Investigator's Global Assessment
PSSI = Psoriasis Scalp Severity Index
QoL = quality of life
SC = subcutaneous
TNF = tumour necrosis factor

RESEARCH REVIEW™
Making Education Easy — Since 2006
www.researchreview.co.nz

This review is a summary of a presentation on secukinumab [Cosentyx®] in the treatment of psoriasis, by Australian Dermatologist Dr John Sullivan, who spoke at the 2018 New Zealand Dermatological Society Annual Conference, held in Auckland in August.

IL-17 and psoriasis

The aetiology of psoriasis is multifactorial and includes a complex interplay between the adaptive and innate immune systems.[1] Interleukin (IL)-23 and the IL-23/T-helper (Th)17 immune axis are both involved in the pathogenesis of psoriasis.[1] IL-23 is an upstream regulatory cytokine that acts early in the inflammatory cascade and is critical in the production of downstream effector cytokines, such as IL-17A, IL-17F and TNF. The IL-17 family has six members, IL-17A, B, C, D, E (IL-25) and F. These cytokines are secreted by T cells (Th17) and innate immune cells (e.g. macrophages, dendritic cells, natural killer cells and mast cells).[1] IL-17A has been identified as a key cytokine, involved in keratinocyte activation and proliferation, and the recruitment of inflammatory cells into psoriatic plaques.[1]

Antibodies directed against IL-17A include secukinumab [Cosentyx®] and ixekizumab[2,3] It is also possible to block the action of IL-17A by targeting the IL-17A receptor with brodalumab.[4] The rationale behind IL-17 inhibition for the treatment of psoriasis was based on biopsy findings showing enrichment of T cells and neutrophils containing IL-17 in psoriatic skin, the reduction of epidermal dysplasia with IL-17 neutralising antibodies in murine models of psoriasis, and a significant correlation between IL-17A serum levels and psoriatic disease severity.[5-7]

Neutrophils appear to be an early target of IL-17 inhibition in psoriasis, with an initial phase II study of IV secukinumab showing rapid disappearance of Munro's microabscesses (collections of neutrophils in the stratum corneum) and spongiform pustules (collections of neutrophils in the spinosum layer) within 2 weeks of treatment initiation.[8] These changes were correlated with a rapid drop in Psoriasis Area and Severity Index (PASI) score. Based on their findings, the study authors proposed a new model of psoriasis immunopathogenesis with a bimodal action, in which the early effects of anti-IL-17A antibodies involve a direct or indirect effect on the neutrophil-keratinocyte axis (innate immunity), leading to reduced neutrophil chemoattraction and epidermal recovery, and a subsequent inhibition of the dendritic cell-T cell axis (adaptive immunity) resulting in long-term clinical response.

Secukinumab

Secukinumab is a fully human IgG1 antibody that largely targets IL-17A and blocks its interaction with the IL-17 receptor, without neutralising IL-17F or directly affecting other Th17 functions or the Th1 pathway.[2]

Dosing: The recommended dosing of SC secukinumab for the treatment of moderate-to-severe plaque psoriasis is 300 mg administered as two separate SC injections of 150 mg, with initial dosing at weeks 0,1,2,3, and 4, followed by monthly maintenance dosing.[9] Peak secukinumab concentration is achieved between 5-6 days post injection, steady state is reached after 20 weeks with monthly dosing, and the estimated mean elimination half-life is approximately 27 days.[9]

Clinical efficacy

Secukinumab has demonstrated very good clinical efficacy in clinical trials for the treatment of moderate-to-severe plaque psoriasis. Dr Sullivan pointed out that in most of these trials, disease severity was defined as PASI ≥12, Investigator's Global Assessment (IGA) ≥3 and body surface area involvement of ≥10%, however, such parameters were often well above these values in individual patients.

In the trials, treatment with secukinumab was not associated with disease rebound on cessation of treatment and the mean time to loss of >50% of maximum PASI improvement was 24 weeks. Furthermore, retreatment saw good re-establishment of response, something that is not always seen with anti-TNFs. Challenging forms of psoriasis investigated in the secukinumab trials included moderate-to-severe palmoplantar psoriasis, moderate-to-severe nail psoriasis and moderate-to-severe scalp psoriasis. The findings of the individual studies are discussed below.

The ERASURE and FIXTURE trials – plaque psoriasis

The phase 3 ERASURE (n = 738) and FIXTURE (n = 1306) 52-week randomised, double-blind, placebo-controlled trials investigated the efficacy and safety of SC secukinumab versus placebo for the treatment of moderate-to-severe plaque psoriasis.[10] The FIXTURE trial also compared the efficacy of secukinumab with that of SC etanercept. The two trials had a similar design, with participants receiving induction with secukinumab 300 mg or 150 mg, or placebo weekly, at weeks 0, 1, 2, 3, and 4, then every 4 weeks until week 48. Etanercept recipients received 50 mg of the agent twice weekly from week 0-12 then once weekly through week 51. In both trials, at week 12, non-responding placebo recipients crossed over to secukinumab 150 mg or 300 mg.

The co-primary endpoints, reduction of 75% or more from baseline in the PASI score (PASI 75) and a score of 0 (clear) or 1 (almost clear) on a 5-point modified IGA at week 12, were achieved by significantly more patients receiving secukinumab than those receiving placebo or etanercept (**Figure 1**).[10] Improvements with secukinumab were rapid, with a 50% reduction in mean PASI score seen after a median of 3 weeks and 3.9 weeks in secukinumab 300 mg and 150 mg recipients, respectively, compared with 7 weeks with etanercept.[10] PASI 75 and IGA 0/1 responses with secukinumab were maintained over 52 weeks, with peak benefits seen at 16 weeks in both trials; 86.7% of secukinumab 300 mg recipients reached a PASI 75, 72.4% reached a PASI 90 and 36.8% reached a PASI 100 at 16 weeks in the FIXTURE trial.[10] Secukinumab at both doses showed superiority versus placebo and etanercept for DLQI 0/1 response at week 12.[10]

A pooled analysis of data from the ERASURE and FIXTURE trials revealed that significantly more PASI 90-100 than PASI 75-89 responders at week 12 had a DLQI 0/1 response (69.4% vs 47.1%; $p<0.001$) and sustained DLQI 0/1 response at week 52 (74.0% vs 56.7%; $p<0.001$).[11]

Investigating the long-term efficacy of secukinumab, an extension of the ERASURE and FIXTURE trials in which week 52 PASI 75 responders continued treatment with secukinumab 300 mg or 150 mg or were switched to placebo (treatment withdrawal), revealed that 88.2% of secukinumab 300 mg recipients and 75.5% of secukinumab 150 mg recipients reached week 104 without relapse (loss of >50% of the maximum PASI gain compared to the baseline of the core studies).[12] The majority of patients experienced relapses during treatment withdrawal; 136/181 (75.1%) secukinumab 300 mg recipients who had switched to placebo.[13] The median time to relapse was 28.0 weeks (95% CI 24.14-32.00) and the mean PASI at time of relapse was 15.6 (range 4.5-65.1).[13]

Strong recapture rates were observed in the ERASURE and FIXTURE trials, with PASI 75 response regained within 16 weeks (median time to response 4 weeks) by 94% of PASI 75 responders retreated after relapse.[13] Retreatment involved an induction phase of weekly dosing for 5 weeks, followed by 4-weekly maintenance dosing.

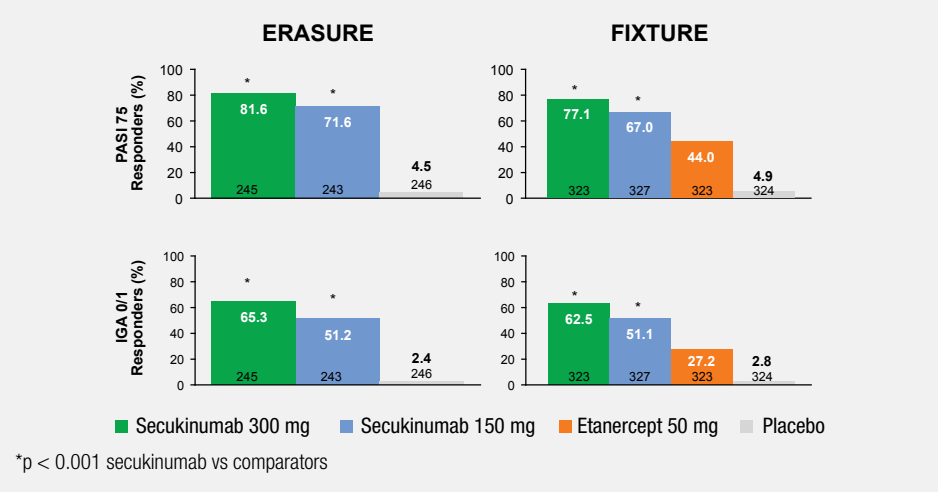


Figure 1. PASI 75 and IGA 0/1 responder rates at week 12 from the ERASURE and FIXTURE trials comparing secukinumab and placebo (ERASURE), and secukinumab, etanercept and placebo (FIXTURE). (*Adapted from Langley et al.*)[10]

The SCULPTURE Extension study – plaque psoriasis

The SCULPTURE Extension study assessed the efficacy and safety of SC secukinumab through 5 years of treatment for moderate-to-severe psoriasis and psoriatic arthritis.[14] In the core SCULPTURE study, PASI 75 responders at week 12 were randomised to fixed-interval secukinumab 300 mg or secukinumab 150 mg, or retreatment as needed. Patients who completed 52 weeks of treatment entered the blinded extension study, receiving maintenance therapy; the study became open-label from year 4.[14] Long-term findings from the secukinumab 300 mg arm of the study are shown in **Figure 2** and clearly demonstrate the high and long-lasting skin improvement seen with this agent over 5 years.[14]

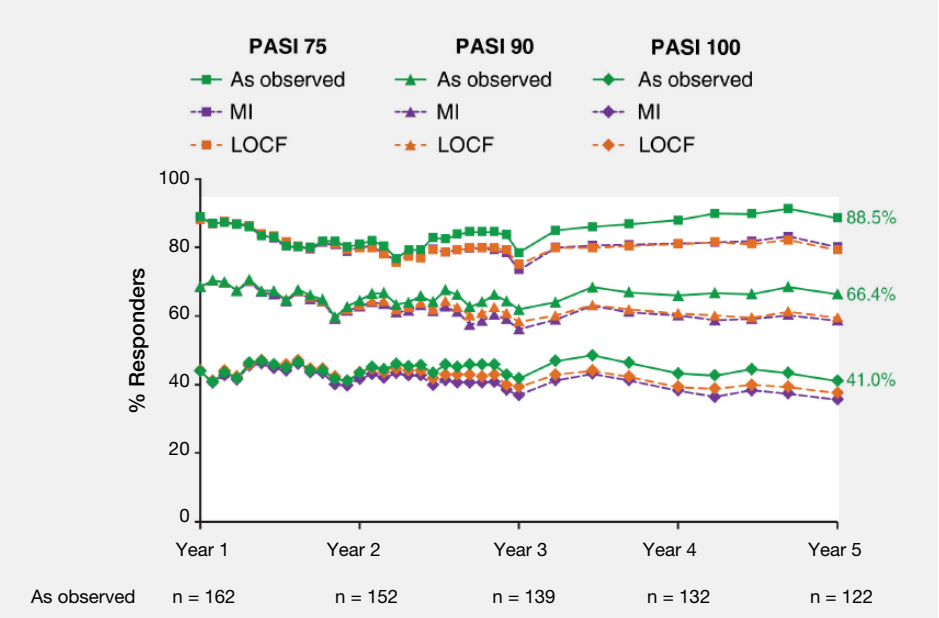


Figure 2. Percentage of secukinumab 300 mg recipients with PASI 75/90/100 response from year 1 to year 5 in the SCULPTURE study.[14]

Dr Sullivan explained that some patient's psoriatic symptoms tend to keep on improving over time with treatment and this is likely due to action through the adaptive immune system switching off the disease.

The CLEAR trial – plaque psoriasis

The CLEAR trial was a 52-week, phase 3b double-blind, head-to-head comparison of SC secukinumab versus ustekinumab in 676 patients with moderate-to-severe plaque psoriasis.[15] Secukinumab recipients received 300 mg of the agent at weeks 0, 1, 2, 3 and 4, then every 4 weeks from weeks 4 to 48, while ustekinumab was dosed per label (45 mg for subjects ≤100 kg at baseline; 90 mg for subjects >100 kg at baseline) at week 0 and week 4, then every 12 weeks from week 16 to week 40.[15]

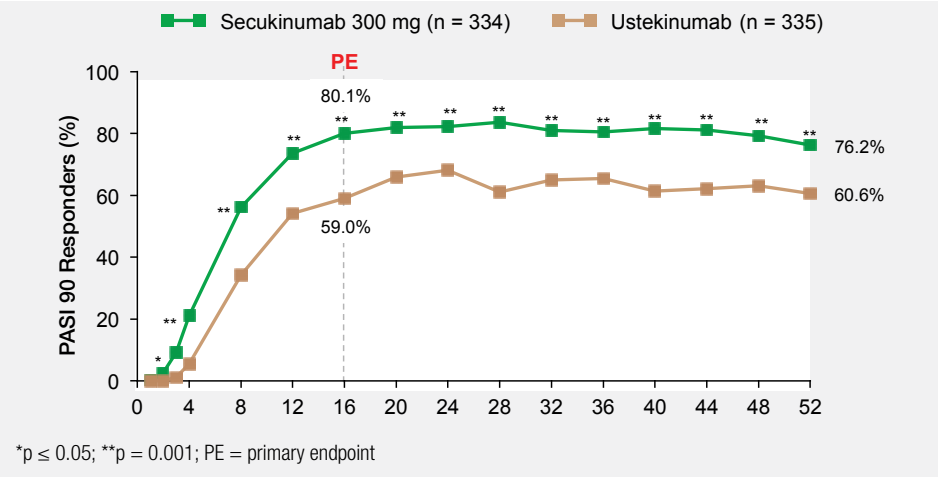
Secukinumab exhibited sustained superiority versus ustekinumab in PASI 90 response (primary endpoint) through week 52; PASI 90 response rate 76.2% versus 60.6% (**Figure 3**).[16] Secukinumab also demonstrated superiority over ustekinumab at week 52 for DLQI 0/1 (71.6% vs 59.2%, $p = 0.05$) and IGA clear/almost clear skin (80% vs 65%, $p < 0.0001$) responses.[16] A significantly greater proportion of patients with psoriatic arthritis achieved PASI 90 with secukinumab than with ustekinumab out to week 52.[16] The PASI 90 response rate for both secukinumab and ustekinumab was lower in biologic-experienced than biologic-naïve patients (79.3% vs 63.5%, $p = 0.0001$ and 58.7% vs 45.5%, $p = 0.3031$).[16]

According to Dr Sullivan, a striking finding in this study was the 50% PASI 75 response rate at week 4 observed in secukinumab recipients.[15]

Post-hoc pooled data analysis – plaque psoriasis

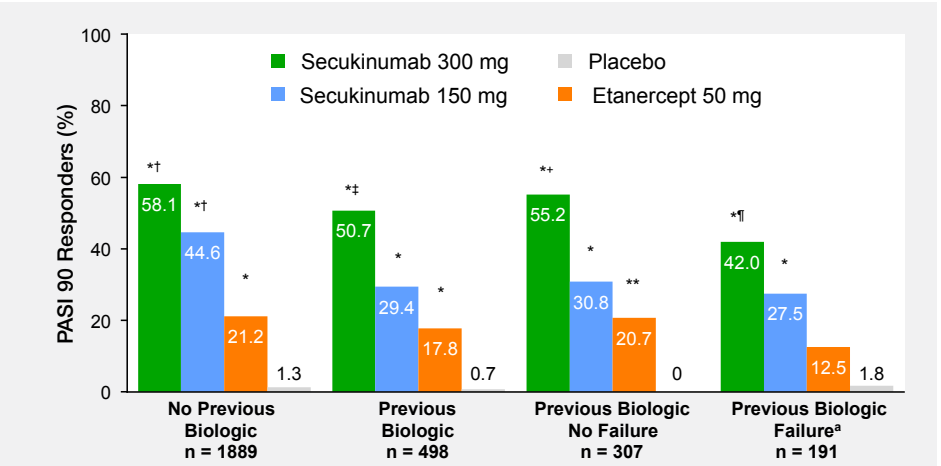
Post-hoc analysis of data from trials investigating secukinumab in plaque psoriasis has confirmed the benefits of treatment with this agent. Pooled data from the FIXTURE, ERASURE, FEATURE and JUNCTURE trials involving over 2000 plaque psoriasis patients receiving either secukinumab 300 mg, secukinumab 150 mg, etanercept or placebo, revealed low absolute PASI scores with both doses of secukinumab at week 12 regardless of baseline disease severity.[17] Analysis of pooled data from these trials also demonstrated the efficacy of secukinumab regardless of previous exposure to biologic therapy (**Figure 4**).[18,19] Dr Sullivan pointed out that while there appears to be a slightly lower response to secukinumab with previous biologic exposure, this is not as pronounced as the loss of response seen with some of the older biologic agents.

Secukinumab was also found to be efficacious regardless of age in a pooled analysis of data from the ERASURE, FIXTURE and CLEAR trials; PASI 90 response at week 52 was approximately 65% in both the 18-64 years and ≥65 years age groups.[20] Dr Sullivan explained that older psoriatic patients often do very well on biologics.



*p ≤ 0.05; **p = 0.001; PE = primary endpoint

Figure 3. PASI 90 response rates of secukinumab and ustekinumab to week 52 in the CLEAR trial.[16]



*p < 0.0001,**p < 0.001, †p < 0.01, ‡p < 0.05 vs. placebo; +p < 0.0001, ¶p <0.01 vs etanercept

[a]Experienced lack of primary or secondary efficacy or lack of tolerability

Figure 4. Pooled analysis from the FIXTURE, ERASURE, FEATURE and JUNCTURE trials demonstrating the efficacy (% of PASI 90 responders) of secukinumab according to previous biologic exposure.[19]

The GESTURE trial – palmoplantar psoriasis

The randomised, double-blind GESTURE trial examined the efficacy and safety of secukinumab in adult patients with moderate-to-severe palmoplantar psoriasis (ppIGA score of ≥3 on a 5-point scale) and at least one additional plaque outside of the palms and soles.[21] Patients received secukinumab 300 mg, secukinumab 150 mg or placebo every week from baseline to week 3 and then every 4 weeks from week 4 to 16; placebo recipients who were not ppIGA 0 or 1 responders at week 16 were re-randomised (1:1) to secukinumab 300 mg or 150 mg.

Sustained improvements in ppIGA 0/1 (clear or almost clear/minimal disease and a reduction of ≥2 points from baseline in the ppIGA scale) were observed through 2.5 years with both doses of secukinumab and the rate of response was seen to steadily increase over time (**Figure 5**).[22] One-third of patients achieved clear or almost clear palms and soles by week 16 and almost half achieved this by 1.5 years.[23] **Figure 6** shows representative images of improvements in palmoplantar psoriasis from subjects in the GESTURE trial. These improvements were associated with substantial improvements in patient-reported outcome data at 1.5 years across QoL tools, indicating clinically meaningful changes for many subjects compared with baseline.[23] The safety profile of secukinumab in this trial was consistent with that seen in other phase 3 trials.

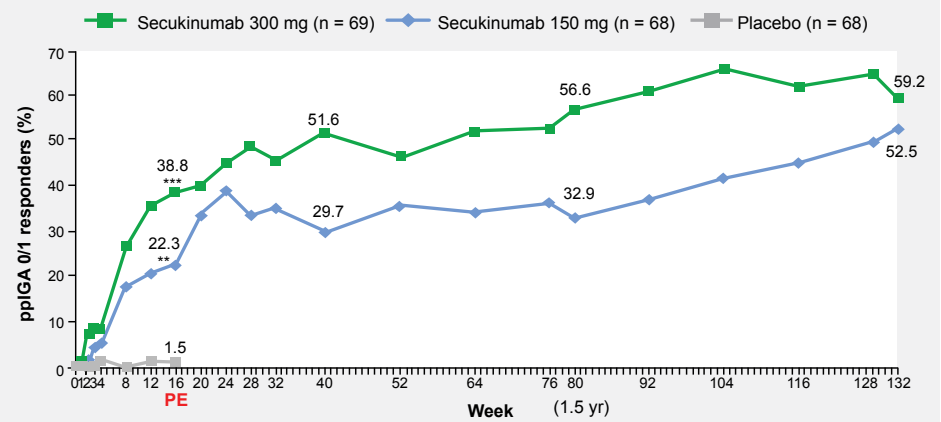
Dr Sullivan pointed out that palmoplantar psoriasis is sometimes slow to respond to treatment and if there is evidence of some improvement at week 16, it may be worthwhile persevering with treatment.

The TRANSFIGURE trial – nail psoriasis

Nail psoriasis is associated with decreased finger mobility, functional impairment, pain and reduced QoL.[24,25] Nails are affected in up to 50% of psoriatic patients and the lifetime incidence is as high as 90%.[24-27] Nail psoriasis, which is often resistant to available therapies, correlates with more severe psoriatic disease and is an important predictor of psoriatic arthritis.[24-27]

The randomised, double-blind, placebo-controlled phase 3b TRANSFIGURE trial evaluated the efficacy and safety of secukinumab 300 mg or secukinumab 150 mg in the treatment of moderate-to-severe nail psoriasis.[28] At week 16, placebo recipients crossed over to secukinumab 300 mg or secukinumab 150 mg.[28] The primary endpoint was superiority of secukinumab versus placebo by total fingernail Nail Psoriasis Severity Index (NAPSI) percentage change from baseline at week 16.

Figure 7 shows the mean change from baseline NAPSI with secukinumab 300 mg, secukinumab 150 mg and placebo to 32 weeks.[28] At 2.5-year follow-up, fingernail psoriasis had improved by 73.3% and 63.6% in secukinumab 300 mg and 150 mg recipients, respectivley.[29] Representative examples of the improvement in nails symptoms in the first 32 weeks of treatment are shown in **Figure 8**. Safety results in the TRANSFIGURE trial were consistent with previous studies, with no new or unexpected safety signals.[30]



**p < 0.001 vs placebo; ***p < 0.0001 vs placebo; PE = primary endpoint; ppIGA 0/1 (clear or almost clear/minimal disease and a reduction of ≥2 points from baseline in the ppIGA scale)

Figure 5. Sustained improvement in palmoplantar psoriasis IGA 0/1 through 2.5 years with secukinumab in the GESTURE trial.[22]

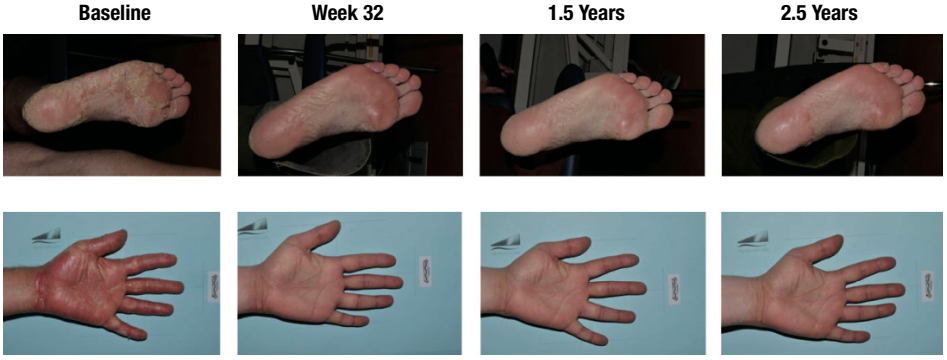
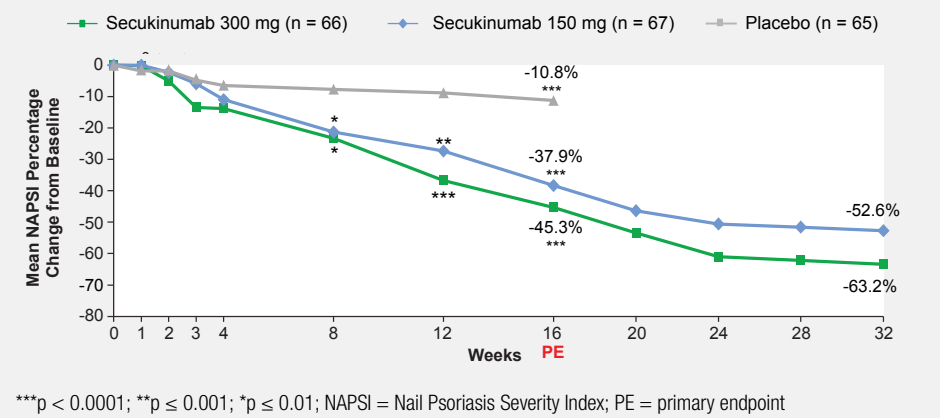


Figure 6. Images of improvements in palmoplantar psoriasis from subjects in the GESTURE trial.[22]



***p < 0.0001; **p ≤ 0.001; *p ≤ 0.01; NAPSI = Nail Psoriasis Severity Index; PE = primary endpoint

Figure 7. Mean change from baseline NAPSI in patients with moderate-to-severe nail psoriasis receiving secukinumab or placebo.[28]

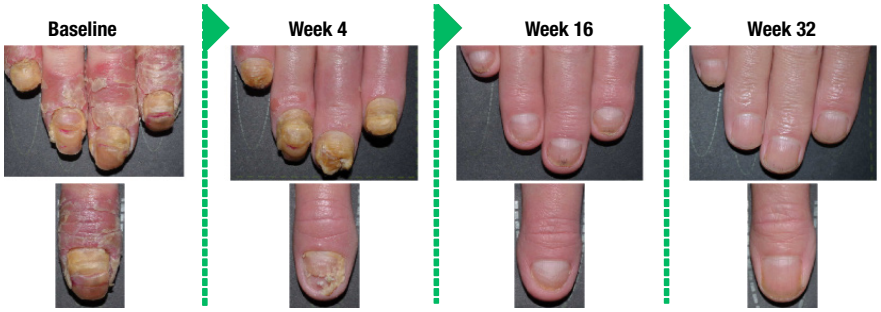
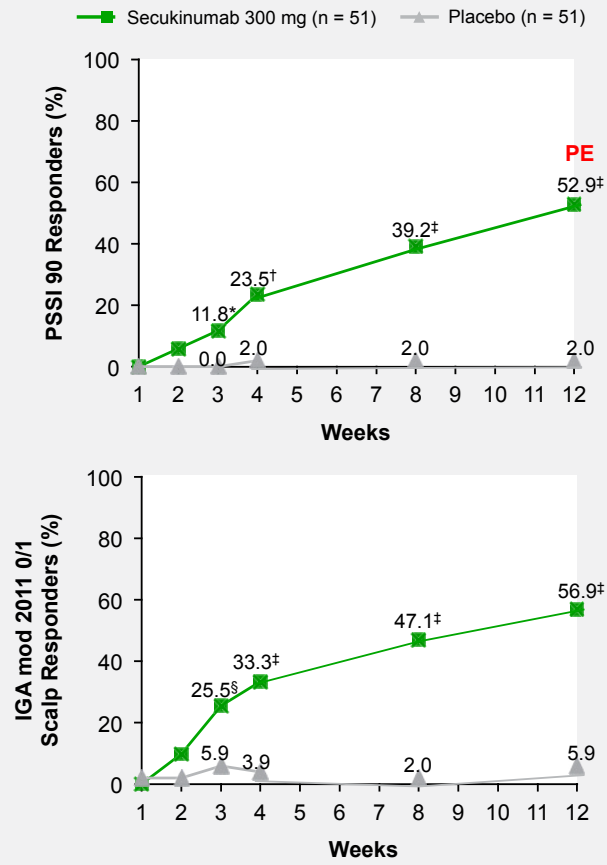


Figure 8. Representative images of improvements in nail psoriasis up to 32 weeks of treatment with secukimumab.[28]

The SCALP study – scalp psoriasis

Scalp involvement is present in 40-90% of psoriasis patients and significantly impairs QoL.[31,32] The randomised, placebo-controlled, phase 3 SCALP study is one of a few studies specifically designed to address patients suffering primarily from moderate-to-severe scalp psoriasis.[33] In contrast to other scalp psoriasis studies, this is the only trial that did not require specific severity of body psoriasis involvement. The study evaluated secukinumab 300 mg administered at weeks 0, 1, 2, 3 and 4, and then every 4 weeks to week 12 versus placebo in 102 patients with a ≥6 month history of moderate-to-severe scalp psoriasis.[33] Secukinumab demonstrated superiority over placebo in the primary endpoint, 90% improvement of Psoriasis Scalp Severity Index (PSSI 90) score from baseline to week 12, and also in the 2011 modified IGA scalp response 0/1 (**Figure 9**).[33]



*p = 0.011; †p = 0.001; ‡p < 0.001; §p = 0.005

IGA mod = Investigator's Global Assessment modified; PE = primary endpoint; PSSI = Psoriasis Scalp Severity Index

Figure 9. PSSI 90 and IGA 0/1 (scalp only) response up to 12 weeks in patients receiving secukinumab or placebo in the SCALP study.[33]

The FUTURE 2 trial – psoriatic arthritis

The randomised, double-blind, placebo-controlled, phase 3 FUTURE 2 trial of secukinumab in adults with active psoriatic arthritis, was undertaken at 76 centres in Asia, Australia, Canada, Europe, and the US.[34] Patients received secukinumab 300 mg (n = 100), secukinumab 150 mg (n = 100) or secukinumab 75 mg (n = 99) once a week from baseline and then every 4 weeks from week 4, or placebo (n = 98).[34] A significantly higher proportion of secukinumab 300 mg, secukinumab 150 mg and secukinumab 75 mg than placebo recipients achieved at least a 20% improvement in the American College of Rheumatology response criteria (ACR20) at week 24 (primary endpoint); 54.0%, 51.0% and 29.3% versus 15.3%, respectively.[34] Improvements in ACR20 with secukinumab were sustained through 2 years.[35] Dr Sullivan has observed an improvement in psoriatic arthritis symptoms in a number of patients treated with secukinumab.

Safety

Dr Sullivan explained that the secukinumab dosing regimen in psoriasis involves higher drug concentrations in the first 5 weeks than is normally used with adalimumab or etanercept, but the regimen appears remarkably safe.

Patients with psoriasis treated with biologics are often on these agents for many years. The long-term safety of such therapy is therefore of particular interest. In Dr Sullivan's experience, a subgroup of patients on secukinumab may have a higher risk of infection, including coughs and colds, and he instructs his patients to contact him if they become unwell in the initial few months of treatment. He stressed that upper respiratory tract infections tend to be a bit more persistent in patients treated with secukinumab and that it is important to check for candidiasis infections in patients receiving secukinumab, particularly in the induction period; in the FIXTURE trial, the rate of candidiasis was 4.7% in patients receiving secukinumab 300 mg.[10] These patients tend to respond well the correct candidiasis treatment. He added that there also needs to be an awareness of the possibility of inflammatory bowel disease (IBD), lupus-like reactions, autoimmune hepatitis, multiple sclerosis (MS) or tuberculosis reactivation in patients receiving biologics in general. There is however evidence that secukinumab is beneficial in patients with MS.[36]

Injection site reactions are rare with secukinumab as is malignancy. The Global Safety Database reporting pregnancies after exposure (maternal or paternal) to secukinumab shows no reports of congenital malformation to date.[37] According to pooled safety data from 10 pivotal phase II and III secukinumab trials in psoriasis, the most common adverse event from baseline through to week 52 is nasopharyngitis (20.0% of patients), followed by headache (8.16%), upper respiratory tract infections (6.65%), arthralgia (5.07%), hypertension (4.81%), diarrhoea (4.75%), back pain (4.26%), pruritus (3.94%), and cough (3.88%).[38] No concerning safety signals were identified in the safety analysis.

Take-home messages:

- Secukinumab targets IL-17 A
- Secukinumab is efficacious in the treatment of moderate-to-severe plaque psoriasis, moderate-to-severe palmoplantar psoriasis, moderate-to-severe nail psoriasis and moderate-to-severe scalp psoriasis
- Patients with psoriatic arthritis also benefit from treatment with secukinumab
- Secukinumab exhibits a good safety profile similar to other biologics
- Patients treated with secukinumab should be monitored for infections including candidiasis.

REFERENCES:

1. Girolomoni G et al. The role of IL-23 and the IL-23/TH 17 immune axis in the pathogenesis and treatment of psoriasis. J Eur Acad Dermatol Venereol. 2017;31(10):1616-26
2. Frieder J et al. Secukinumab: a review of the anti-IL-17A biologic for the treatment of psoriasis. Ther Adv Chronic Dis. 2018;9(1):5-21
3. Leonardi C et al. Anti-interleukin-17 monoclonal antibody ixekizumab in chronic plaque psoriasis. N Engl J Med. 2012;366(13):1190-9
4. Papp KA et al. Brodalumab, an anti-interleukin-17-receptor antibody for psoriasis. N Engl J Med. 2012;366(13):1181-9
5. Lin AM et al. Mast cells and neutrophils release IL-17 through extracellular trap formation in psoriasis. J Immunol. 2011;187(1):490-500
6. Nakajima K et al. Distinct roles of IL-23 and IL-17 in the development of psoriasis-like lesions in a mouse model. J Immunol. 2011;186(7):4481-9
7. Arican O et al. Serum levels of TNF-alpha, IFN-gamma, IL-6, IL-8, IL-12, IL-17, and IL-18 in patients with active psoriasis and correlation with disease severity. Mediators Inflamm. 2005;2005(5):273-9
8. Reich K et al. Evidence that a neutrophil-keratinocyte crosstalk is an early target of IL-17A inhibition in psoriasis. Exp Dermatol. 2015;24(7):529-35
9. Medsafe NZ. Cosentyx® data sheet. Available from: http://www.medsafe.govt.nz (Accessed Sept 2018)
10. Langley RG et al. Secukinumab in plaque psoriasis--results of two phase 3 trials. N Engl J Med. 2014;371(4):326-38
11. Elewski BE et al. Psoriasis patients with psoriasis Area and Severity Index (PASI) 90 response achieve greater health-related quality-of-life improvements than those with PASI 75-89 response: results from two phase 3 studies of secukinumab. J Dermatol Treat. 2017;28(6):492-99
12. Blauvelt A et al. Secukinumab maintains reductions in PASI through second year of treatment. A randomised extension of the ERASURE and FIXTURE studies in plaque psoriasis. J Am Acad Dermatol. 2016;74(5 Suppl 1);AB271
13. Blauvelt A et al. Secukinumab re-initiation achieves regain of high response levels in patients who interrupt treatment for moderate to severe plaque psoriasis. Br J Dermatol. 2017;177(3):879-81
14. Bissonnette R et al. Secukinumab demonstrates high sustained efficacy and a favourable safety profile in patients with moderate-to-severe psoriasis through 5 years of treatment (SCULPTURE Extension Study). J Eur Acad Dermatol Venereol. 2018; Feb 14 [Epub ahead of print]
15. Thaçi D et al. Secukinumab is superior to ustekinumab in clearing skin of subjects with moderate to severe plaque psoriasis: CLEAR, a randomized controlled trial. J Am Acad Dermatol. 2015;73(3):400-9
16. Blauvelt A et al. Secukinumab is superior to ustekinumab in clearing skin of subjects with moderate-to-severe plaque psoriasis up to 1 year: Results from the CLEAR study. J Am Acad Dermatol. 2017;76(1):60-9
17. Spelman LJ et al. Secukinumab demonstrates sustained efficacy in moderate to severe plaque psoriasis across disease severity subgroups. J Am Acad Dermatol. 2015;72(5; Suppl 1):AB248
18. Griffiths CEM et al. Secukinumab shows efficacy in subjects regardless of previous exposure to biologic therapy: A pooled sub-analysis from four phase 3 clinical trials in psoriasis. J Am Acad Dermatol. 2015;72(5; Suppl 1): AB251
19. Griffiths CEM et al. Secukinumab shows efficacy in subjects regardless of previous exposure to biologic therapy: A pooled subanalysis from four phase 3 clinical trials in psoriasis. Presented at: The 73rd Annual Meeting of the American Academy of Dermatology, on 20th March, 2015, San Francisco, USA (ePoster 1730)
20. Körber A et al. Efficacy and safety of secukinumab in elderly subjects with moderate to severe plaque psoriasis: A pooled analysis of phase III studies. Drugs Aging 2018;35(2):135-44
21. Gottlieb A et al. Secukinumab shows significant efficacy in palmoplantar psoriasis: Results from GESTURE, a randomized controlled trial. J Am Acad Dermatol. 2017;76(1):70-80
22. Gottlieb A et al. Secukinumab shows high and sustained efficacy in subjects with moderate to severe palmoplantar psoriasis: 2.5-year results from the GESTURE study. PGC. 2017; London, UK (Poster 026)
23. Gottlieb A et al. EADV. Secukinumab is effective in subjects with moderate to severe palmoplantar psoriasis: 1.5-Year results From the GESTURE Study 25th Annual Congress of the European Academy of Dermatology and Venereology, Vienna, Austria; September 28-October 2, 2016 (Poster 2076)
24. Radtke MA et al. Nail psoriasis - a treatment challenge. J Dtsch Dermatol Ges. 2013;11(3):203-19
25. Thaçi D et al. Adalimumab for the treatment of moderate to severe psoriasis: sub analysis of effects on scalp and nails in the BELIEVE study. J Eur Acad Dermatol Venereol. 2015;29(2):353-60
26. Baran R et al. The burden of nail psoriasis: an introduction. Dermatology 2010;221 (Suppl 1):1-5
27. Langenbruch A et al. Nail involvement as a predictor of concomitant psoriatic arthritis in patients with psoriasis. Br J Dermatol. 2014;171(5):1123-8
28. Reich K et al. Secukinumab shows significant efficacy in nail psoriasis: Week 32 results from the Transfigure study. Ann Rheum Dis. 2016;75(1):Suppl 2
29. Reich K et al Secukinumab and the neutrophil–keratinocyte axis: A potential early target for the anti–IL-17A antibody. Presented at: Psoriasis from Gene to Clinic 7th International Congress – 11-13 December 2014, London, UK (Poster 021)
30. Reich K et al. Secukinumab is effective in subjects with nail psoriasis: 16 week results from the TRANSFIGURE study. Presented at: 23rd World Congress of Dermatology, 8th-13th June, 2015, Vancouver, Canada (Poster 3086561)
31. Crowley J et al. Scalp psoriasis: an overview of the disease and available therapies. J Drugs Dermatol. 2010;9(8):912-8
32. Zampieron A et al. Quality of life in patients with scalp psoriasis. G Ital Dermatol Venereol. 2015;150(3):309-16
33. Bagel J et al. The effect of secukinumab on moderate-to-severe scalp psoriasis: Results of a 24-week, randomized, double-blind, placebo-controlled phase 3b study. J Am Acad Dermatol. 2017;77(4):667-74
34. McInnes IB et al. Secukinumab, a human anti-interleukin-17A monoclonal antibody, in patients with psoriatic arthritis (FUTURE 2): a randomised, double-blind, placebo-controlled, phase 3 trial. Lancet 2015;386(9999):1137-46
35. McInnes IB et al. Secukinumab provides sustained improvements in the signs and symptoms of active psoriatic arthritis: 104 weeks results from a phase 3 trial [abstract]. Arthritis Rheumatol. 2016; 68 (Suppl 10)
36. Havrdová E et al. Activity of secukinumab, an anti-IL-17A antibody, on brain lesions in RRMS: results from a randomized, proof-of-concept study. J Neurol. 2016;263(7):1287-95
37. Sullivan J et al. Secukinumab in pregnancy: Outcomes from the Global Safety Database. ACD 2017; Poster 167. Available from: http://acd2017asm.paperlessevents.com.au/presenters/399 - cbp=/more-info/11551/ (Accessed Sept 2018)
38. van de Kerkhof PC et al. Secukinumab long-term safety experience: A pooled analysis of 10 phase II and III clinical studies in patients with moderate to severe plaque psoriasis. J Am Acad Dermtaol. 2016;75(1):83-98

ABOUT RESEARCH REVIEW

A Research Review Speaker Series is a summary of a speaking engagement by a medical expert. It is made available to health professionals via e-mail or web-site download to Research Review subscribers or by physical distribution by Research Review or third parties. Research Review has no control over the content of this presentation, which has been developed and presented by the featured expert. Research Review is not responsible for any inaccuracies or errors of fact made by, or opinions of, the speaker.

Privacy Policy: Research Review will record your email details on a secure database and will not release them to anyone without your prior approval. Research Review and you have the right to inspect, update or delete your details at any time.

Research Review subscribers can claim CPD/CME points for time spent reading our reviews from a wide range of Australasian medical and nursing colleges. Find out more on our **CPD page**.

NOVARTIS

Publication of this article was supported by an educational grant from Novartis. The content or opinions expressed in this publication may not reflect the views of Novartis. Before prescribing any of the prescription medications mentioned in this article please consult the full Product Information. These are available at www.medsafe.govt.nz. Treatment decisions based on these data are the full responsibility of the prescribing physician.