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IL-23 Inhibitors in Psoriasis: A Canadian Dermatology Roundtable

Simon Nigen, MD, FRCPC
Ajith Cy, MD, FRCPC

Moderated By: Vimal Prajapati, MD, FRCPC

ABOUT THE SPEAKERS

Simon Nigen, MD, FRCPC

Dr. Simon Nigen obtained his Doctor of Medicine degree from the Université de Montréal, having previously completed a Bachelor of Pharmacy in 1993. He subsequently completed his residency in dermatology at Université Laval in Québec and his post-doctoral training (fellowship) at the University of Toronto in drug reactions and clinical pharmacology. He currently practises at Verdun Hospital and directs the Sima Recherche clinical research dermatology clinic at that institution. Dr. Nigen is an Assistant Clinical Professor in the Faculty of Medicine at the Université de Montréal and is Chief of the Dermatology Service at Verdun Hospital. He also works at the Montreal General Hospital and directs the contact dermatitis clinic at that institution. He is also an Assistant Clinical Professor at McGill University. He is a member of the board of directors of the Canadian Eczema Society. He also served as Secretary-Treasurer of the Dermatology Society of Montreal, Director on the board of directors of the Canadian Dermatology Association, member of the examination committee for the Royal College of Physicians and Surgeons of Canada, Director of the Skin Allergy Clinic (epicutaneous testing) at Maisonneuve-Rosemont Hospital, and a member of numerous learned societies. He has also worked as a researcher and investigator since 2003 on numerous research projects. He has over 50 publications to his credit. His primary areas of interest are psoriasis, atopic eczema, urticaria, skin allergy, cutaneous oncology, acne, contact eczema, and photodynamic therapy. He has led numerous clinical studies in various areas of dermatology, including psoriasis, urticaria, atopic dermatitis, alopecia areata, acne vulgaris, skin aging, scar treatment, seborrhoeic dermatitis, onychomycosis, and photosensitivity.

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Ajith Cy, MD, FRCPC

Dr. Ajith Cy is Dermatologist practising full-time Medical Dermatology in Waterloo, Ontario, since 2015. He completed his Fellowship in Pediatric Dermatology and Residency in Dermatology at the University of Toronto. Dr Cy's special interests in dermatology are related to inflammatory skin diseases, treatment of skin cancers, vitiligo & alopecia areata. He is also interested in upcoming medical treatments for inflammatory skin diseases, and is actively involved in clinical research.

Affiliations: None declared.

Moderated By: Vimal Prajapati, MD, FRCPC

Dr. Vimal H. Prajapati is a Clinical Assistant Professor at the University of Calgary, co-creator of The Dermatology Philosophy, as well as co-founder and co-director of the Skin Health & Wellness Centre, Dermphi Centre, Dermatology Learning Institute, Dermatology Research Institute, Dermphi Therapeutics, and D&P Commercial Group. Additionally, he has started several subspecialty initiatives, including multidisciplinary clinics for pediatric morphea, pediatric scleroderma, and pediatric psoriasis in Calgary, rapid access clinics for psoriasis and eczema in Calgary, as well as rural outreach clinics for psoriasis and eczema in Medicine Hat.

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Moderated By: Vimal Prajapati, MD, FRCPC

The three approved IL-23 inhibitors — guselkumab, risankizumab, and tildrakizumab — have transformed the management of moderate-to-severe plaque psoriasis. Canadian Dermatology Today brought together Dr. Simon Nigen and Dr. Ajith Cy, moderated by Dr. Vimal Prajapati, to discuss practical strategies for managing difficult-to-treat psoriasis sites including the scalp, nails, and genitals; long-term outcomes and drug survival in real-world practice; and the emerging role of GLP-1 receptor agonists in addressing cardiometabolic comorbidities in patients with plaque psoriasis.

Vimal Prajapati (VP): What factors do you consider when a patient presents with plaque psoriasis at difficult-to-treat sites such as the scalp, nails, or genitals, and do you use site-specific assessment tools in routine practice?

Simon Nigen (SN): The most important considerations are the extent and severity of involvement at these sites and the patient's quality of life. Nail psoriasis, for example, can be profoundly embarrassing, and that burden must be taken into account. I also consider the patient's treatment history and the duration of disease. In clinical trials, we use site-specific instruments such as NAPSI for nails or PSSI for the scalp, but in real-world practice I take a more general approach — a physician's global assessment of each site — combined with a careful discussion about how these areas are affecting the patient's life. I find photography particularly useful for tracking nail changes over time, since improvements are gradual and both patients and physicians tend to forget what things looked like at baseline.

Ajith Cy (AC): Everything depends on how much the problem is impacting the patient. For scalp psoriasis, pruritus can be so severe that patients can't sleep at night. The primary question is always: how much is this bothering you? I also assess the extent of psoriasis at other body sites and always screen for psoriatic arthritis, which can change the therapeutic approach entirely. In routine practice, I use the overall PASI score and document special site involvement. For nail or hand disease, I always add a note about the impact on the patient's ability to work.

VP: Photography is an excellent tool for tracking nails precisely because patients — and physicians — often forget what the nails looked like before treatment began. Having that visual reference helps reinforce the benefit when improvement is otherwise gradual and subtle.

VP: From your clinical experience, have you observed meaningful differences between guselkumab, risankizumab, and tildrakizumab when treating patients with scalp, nail, or other difficult-to-treat sites?

AC: All three are excellent therapies for psoriasis, including at special sites. If pressed to create a hierarchy, I'd say risankizumab and tildrakizumab perform comparably in my experience. My clinical impression is that guselkumab may be somewhat slower to show results at difficult sites — particularly nails, palms, and soles — within the first six months. That said,

all three ultimately respond well, and I want to be clear that this is based on real-world impression rather than head-to-head data. All three have clinical trial evidence supporting their use at special sites **(Figures 1-4)**.

SN: I agree there is no major difference between the three. Patient-to-patient variability is probably greater than any difference between the agents themselves. Dosing frequency is one practical differentiator — one agent is administered every two months, another every three — but the clinical efficacy profiles are quite similar. If a specific agent has compelling site-specific data, that might influence me slightly, but overall, all three are very good choices.

VP: As longer-term data continues to emerge, how does durability of response influence your confidence and treatment selection for patients with scalp-predominant plaque psoriasis?

AC: Durability is a major factor. Scalp psoriasis can take longer to achieve full clearance than body plaques, so once we achieve a response, we want it sustained. If real-world evidence shows superior drug survival for a particular IL-23 inhibitor in scalp psoriasis, that would be an important consideration in my selection — though it's one factor among many, including the overall safety profile, dosing schedule, and patient preference.

VP: Scalp psoriasis can be particularly challenging, and there is also basic science research demonstrating that the immunologic milieu in the scalp is distinct from other sites — which likely explains why the scalp is often the last area to clear and can be the most recalcitrant to treatment.

Where do IL-23 inhibitors fit in the treatment sequencing for patients with scalp psoriasis who are struggling with topical therapy burden or experiencing significant psychosocial impact from visible scalp disease?

SN: In my practice, IL-23 inhibitors are among the best options for scalp psoriasis. Tildrakizumab, for example, has demonstrated sustained responses at 52 weeks, and the safety profile is excellent. Beyond the clinical outcomes, the quality-of-life improvement for patients with chronic, visible scalp disease is substantial. Patients who have been managing scalp symptoms for years and finally achieve good control report meaningful changes in self-confidence and daily functioning. I consider IL-23 inhibitors a very appropriate early choice in the biologic treatment pathway for these patients.

Which nail psoriasis patient profiles most benefit from early consideration of an IL-23 inhibitor, and how do you decide when to escalate to a biologic for nail involvement?

AC: The key differentiator is whether psoriatic arthritis is present. If there is significant psoriatic arthritis, I lean toward an IL-17 inhibitor where I have greater real-world comfort. For patients with nail psoriasis without meaningful arthritis, IL-23 inhibitors are my preference, given both their efficacy at this site and their more favourable safety profile. If nail psoriasis is significantly impacting quality of life or ability to work, I am very willing to initiate a biologic — nails are visible to the world, and that visibility carries real psychological and functional weight. I wouldn't wait for the situation to become more severe.

SN: The decision to escalate depends on the degree of nail involvement and the patient's occupation and quality of life. Mild pitting is different from severe onycholysis with pain. For a musician, waiter, or anyone who works with their hands in a public-facing role, the burden of nail psoriasis can be disproportionate to the clinical score. Once the decision is made to treat,

biologics — particularly IL-23 inhibitors — are the most effective option, with solid evidence from multiple clinical trials and a reassuring long-term safety profile.

Given the slow growth rate of nails, how do you set patient expectations about timelines for improvement when using an IL-23 inhibitor?

AC: I tell patients not to expect any visible improvement for at least six months. Some IL-23 inhibitors may begin to show early benefit, but meaningful nail improvement typically takes longer. I counsel patients that we need the treatment for a minimum of two years before drawing conclusions about efficacy, and I see nail patients at the six-month mark after starting a biologic. I also let patients know that dose optimization may be considered at six months if the response is insufficient. The message is: this takes time, and we need to commit to the treatment.

SN: My framing is similar — six months to one year, with one year being my primary goal. I deliberately avoid creating high expectations too early, even though I have occasionally seen encouraging responses at three months. I prefer to set a conservative timeline and be pleasantly surprised, rather than have patients become discouraged. I wait at least six to twelve months before concluding whether a biologic has been sufficiently effective for nail disease.

Beyond efficacy, what practical considerations most influence long-term satisfaction in patients with scalp and nail psoriasis?

SN: Safety and tolerability are the next most important factors after efficacy. IL-23 inhibitors require fewer monitoring obligations compared to IL-17 or TNF inhibitors, which simplifies management. Some agents also offer the convenience of room-temperature storage, which matters for patients who travel. All of these factors contribute to treatment adherence over the long term.

AC: For scalp psoriasis specifically, I emphasize early pruritus relief. Scalp psoriasis tends to be more pruritic than other sites, and patients often notice that itching improves earlier than visible clearance. I use this as a positive early signal to help patients maintain confidence in the treatment while waiting for full clearance — similar to how we counsel patients with atopic dermatitis. Knowing the itch is coming down is meaningful reassurance that the medication is beginning to work.

How do IL-23 inhibitors align with your definition of long-term success in plaque psoriasis management compared to IL-17 and TNF inhibitors, and what factors most influence the ability to maintain long-term disease control?

AC: Drug survival is my primary metric for long-term success. IL-23 inhibitors, as a class, tend to have higher drug survival rates compared to IL-17 and TNF inhibitors, particularly in patients without significant psoriatic arthritis — which is the population for whom I typically select them. Patient visits are also shorter and simpler for those on IL-23 inhibitors long term. Regarding factors that influence disease control over time, if a patient develops de novo psoriatic arthritis while on an IL-23, that changes the treatment calculus. But the long-term safety and convenience of this class make it a compelling default in the right patient.

SN: In terms of predicting who will maintain long-term control, younger patients and those with earlier disease often respond better — though this is a generalization. Beyond that, specific agents do differ in their long-term efficacy data, and I pay attention to those differences. Overall, long-term outcomes with this class are very encouraging: patients are generally very satisfied, efficacy is sustained, and tolerability is not an issue.

VP: Studies consistently show that IL-23 inhibitors perform well as a class when it comes to drug survival, which makes them a natural anchor for long-term plaque psoriasis management in appropriate patients.

Are there real-world patient experiences that stand out in illustrating the long-term impact of IL-23 inhibitors on quality of life and treatment adherence?

AC: Elderly patients stand out in particular — not just those in their sixties, but patients well into their seventies and eighties. The safety profile of IL-23 inhibitors is especially reassuring in this population, where comorbidities amplify concern about systemic risk. Just recently, I saw an 86-year-old woman who had suffered with psoriasis for years and had never wanted to try a systemic treatment. Her daughter finally convinced her to try a biologic, and she came to her next appointment telling me it was the first time she had slept well in years. Her hesitation had been rooted in fear of the unknown — she didn't know what the medication might do to her. She is now grateful she made the decision. These moments are a reminder of what's possible when we find the right treatment for the right patient at the right time.

With the rising use of GLP-1 receptor agonists for weight management and cardiovascular risk reduction, how are these agents beginning to influence your approach to plaque psoriasis patients with cardiometabolic comorbidities?

SN: This is a very interesting and evolving area. There is emerging evidence suggesting that IL-23 inhibitors may help protect against the development of psoriatic arthritis — an important dimension that warrants further study. On the GLP-1 side, there are data in psoriatic arthritis and hidradenitis suppurativa showing that adding a GLP-1 receptor agonist to biologic therapy can enhance treatment response, and studies specifically in psoriasis are beginning to appear. In the coming years, I expect GLP-1 receptor agonists to become increasingly relevant to dermatologists managing patients with inflammatory skin disease and metabolic comorbidities.

AC: When psoriasis is well-controlled on an IL-23 inhibitor alone, I don't necessarily make a GLP-1 recommendation. But if control is suboptimal and I identify two or more cardiometabolic comorbidities — typically obesity combined with diabetes or hypertension — I include a comment in my referral note to the family physician suggesting consideration of a GLP-1 receptor agonist. I haven't yet started prescribing GLP-1 agents myself, but I anticipate that changing.

Do you see dermatologists taking a more active role in prescribing GLP-1 receptor agonists for patients with psoriasis, similar to how we have taken the lead in managing psoriatic arthritis?

AC: I think we should, and the analogy to psoriatic arthritis is apt. Collaboration with primary care for cardiometabolic disease can be complicated — family physicians manage many competing priorities and may not fully appreciate the systemic inflammatory burden of psoriasis as an independent cardiovascular risk factor. They understand obesity, hypertension, and diabetes, but may be less convinced that psoriasis itself adds cardiovascular risk. Just as we took the lead in identifying and co-managing psoriatic arthritis, I believe dermatologists should be more proactive in initiating GLP-1 receptor agonists for our psoriasis patients with metabolic comorbidities. I would like to see that become standard practice.

SN: I agree. There will be a place for closer collaboration with endocrinologists and internal medicine specialists, just as we collaborate with rheumatologists for psoriatic arthritis. But there is also a real gap — endocrinologists may not be accessible for all of our psoriasis patients. As dermatologists, we may need to become more comfortable prescribing GLP-1 agents ourselves as the evidence matures and access becomes a concern.

VP: The GLP-1 story is one I would not have found particularly interesting five years ago, but the growing body of evidence for both weight-loss-dependent and weight-loss-independent benefits in dermatology — including ongoing studies in psoriasis and hidradenitis suppurativa — makes it an area that will genuinely change how we manage patients in the coming years.

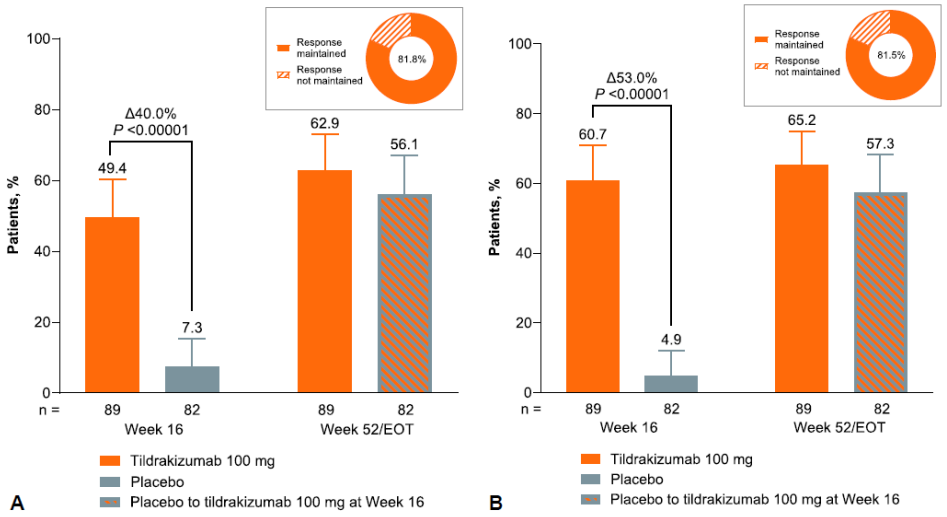


Figure 1. (A) Maintenance of IGA mod 2011 (scalp) and (B) PSSI 90 responses from week 16 to week 52. IGA mod 2011 (scalp) response and PSSI 90 response were analyzed in the mITT analysis set with NRI. Treatment difference is shown only at week 16. The percentage of week 16 responders who maintained a response at week 52 is calculated using N as the denominator, where N is the number of IGA mod 2011 (scalp) or PSSI 90 responders to tildrakizumab at week16 in the mITT population; *Sofen et al., JAM ACAD DERMATOL VOLUME 92, NUMBER 4*

Abbreviations: EOT: End of treatment; **IGA mod 2011 (scalp) response:** Investigator Global Assessment modified 2011 score of 0 or 1 with a ≥2-point reduction from baseline for the scalpt; **mITT:** modified intention-to-treat; **NRI:** nonresponder imputation; **PSSI 90 response:** ≥90% improvement in the Psoriasis Scalp Severity Index.

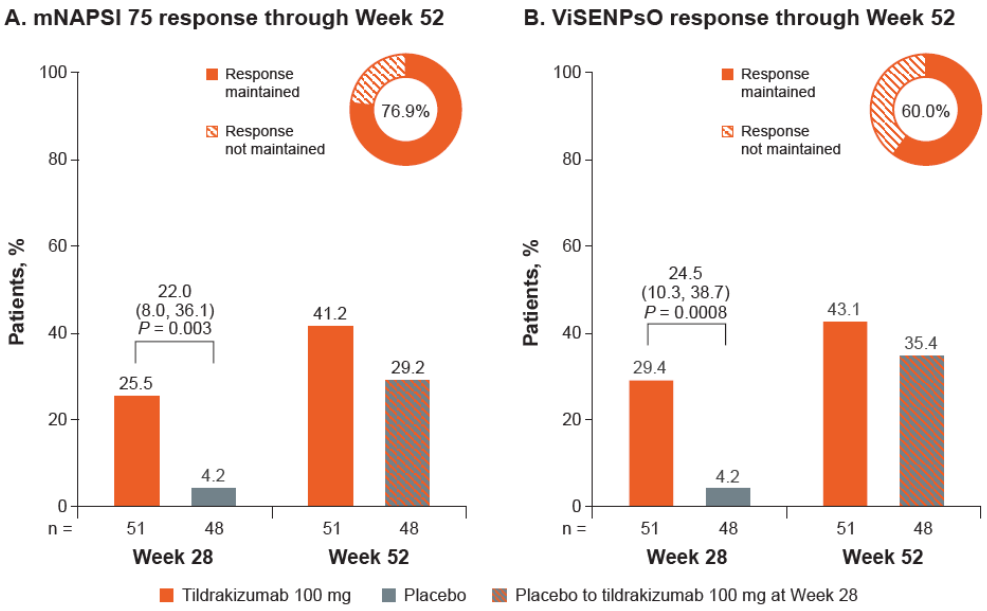
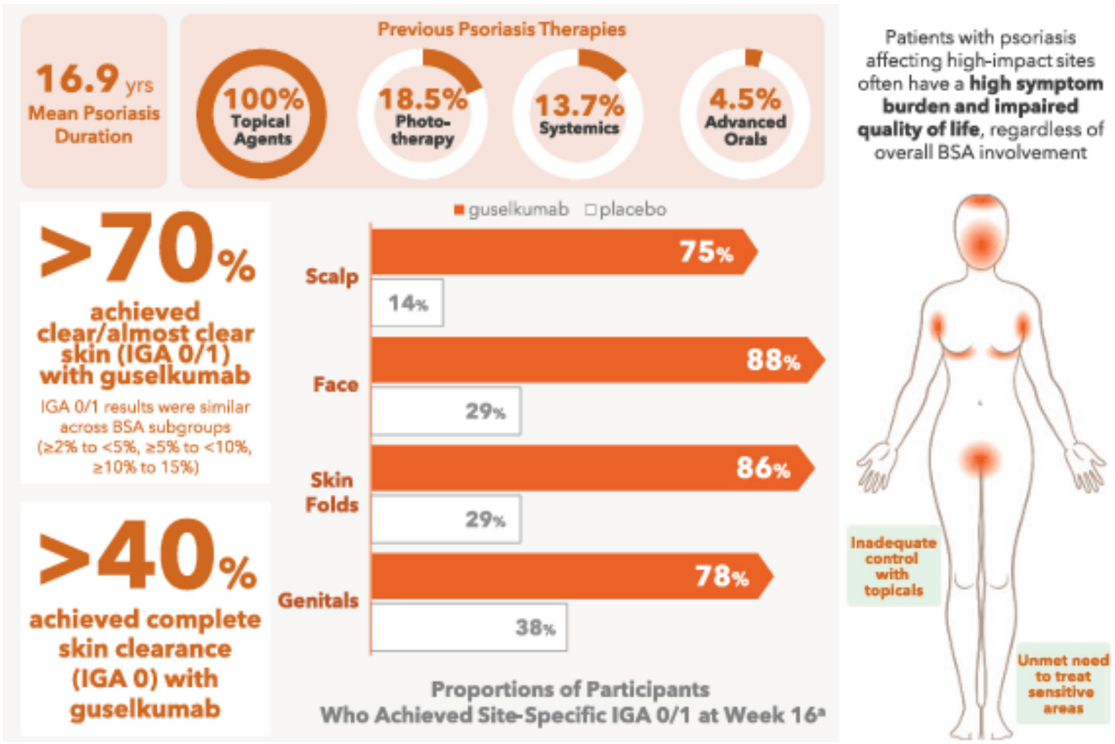


Figure 2. Comparisons at Week 28 are shown as treatment difference (95% confidence interval). mNAPSI 75 and ViSENPsO responses were analyzed using a Cochran-Mantel-Haenszel test in the ITT population (all patients randomized and dispensed study treatment). Missing data were handled using NRI; *Yamauchi et al, 2026 Winter Clinical Conference Poster Presentation*

Abbreviations: ITT: intention-to-treat; **mNAPSI:** modified Nail Psoriasis Severity Index; **mNAPSI 75 response:** ≥75% improvement from baseline in mNAPSI; **NRI:** nonresponder imputation; **ViSENPsO:** Visual Medical Scale to Evaluate Nail Psoriasis Severity; **ViSENPsO response:** ViSENPsO score of 0 (normal) or 1 (minimal) with a ≥2-point decrease from baseline.



*P<0.001 for guselkumab vs. placebo at all high-impact sites

Figure 3. Guselkumba for low BSA, moderate psoriasis affecting high-impact sites, results at week 16; *Gold et al, Br J Dermatol. 2026 Jan 6;194(1):25-36. doi: 10.1093/bjd/ljaf327.*

Abbreviations: BSA: body surface area; **IGA:** Investigator's Global Assessment.

Timepoint	Scalp Involvement	Palmoplantar Involvement	Nail Involvement
6 months	72.1% (*n* = 154)	50.0% (*n* = 50)	60.0% (*n* = 94)
1 year	79.1% (*n* = 153)	53.9% (*n* = 49)	68.4% (*n* = 92)
2 years	72.5% (*n* = 129)	52.2% (*n* = 40)	64.7% (*n* = 76)

Figure 4. Complete clearance of high-impact sites with risankizumab at multiple timepoints; *Contente, M., et al., (2026). Journal of Dermatological Treatment, 37(1). https://doi.org/10.1080/09546634.2026.2668213*

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