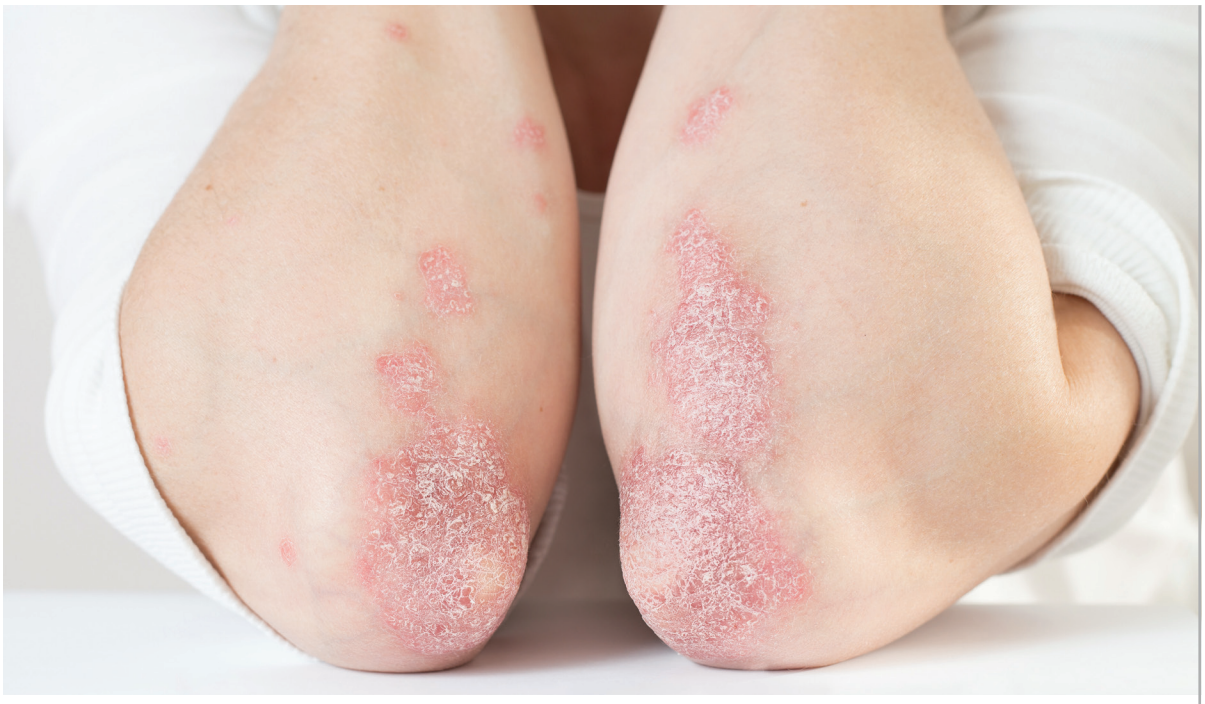
AAD
# DermWorld
directions in residency
A Publication of the American Academy of Dermatology | Association
Winter 2022



# What residents need to know about psoriasis

By Brad Glick, DO, MPH, FAAD, and George Han, MD, PhD, FAAD

*For over 20 years, the National Psoriasis Foundation (NPF) has hosted residents from across the country to participate in a one-and-a-half day program focused on psoriasis. The latest NPF Resident Meeting was held last October in Nashville featuring an all-star faculty lineup including AAD President Mark Kaufman, MD, FAAD. George Han, MD, PhD, FAAD, and Brad Glick, DO, MPH, FAAD, were co-chairs of the meeting. On the heels of their presentation,* Directions *asked Drs. Han and Glick to provide an overview of psoriasis essentials for residents. We have amalgamated their responses here for our resident audience.*

## How should a resident prepare for a course of study on something as vast and important as psoriasis?

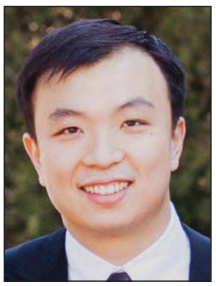
Our understanding of psoriasis has evolved greatly in recent years, and older textbooks are quickly becoming outdated with regard to pathophysiology, treatments, and comorbidities. These are all important topics that directly impact our patients. Thankfully, there have been numerous supplements and review papers in the dermatology literature, such as in the *Journal of the American Academy of Dermatology* where a collection on psoriasis contains well over 500 manuscripts from within the past five years. While the literature is broad, there do exist numerous forums online for a more guided discussion about advances in psoriasis, including a panel we had developed for *HCP Live* titled, Advances in the Management of Plaque Psoriasis. Importantly, even guidelines for treatment are changing, with new guidance from the International Psoriasis Council on appropriate patients for systemic therapy.

## What are some basic topics residents should know about psoriasis by the time they complete their residency?

**Immunology:** While we are still searching for specific triggers of psoriasis and a cure, it has become

see **PSORIASIS** on p. 3

**Brad Glick, DO, MPH, FAAD,** is residency program director at Larkin Palm Springs Hospital in Florida and also a member of the AAD Board of Directors.



**George Han, MD, PhD, FAAD,** is an associate professor in the department of dermatology at the Donald and Barbara Zucker School of Medicine at Hofstra / Northwell in New York, and also a member of the medical board of the National Psoriasis Foundation.

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**PSORIASIS** from p. 1

clearer that the development of psoriasis involves aspects of autoimmune pathways. Inflammatory, and in some cases (such as in pustular psoriasis) autoinflammatory pathways are involved, as well as T-cell immune-mediated pathways. This inflammatory drive centers around the TH17 cells involving the proinflammatory cytokines IL-23 and IL-17, but further nuances — such as the role of different types of IL-17 (i.e., IL-17A and IL-17F) and the involvement of IL-36 in generalized pustular psoriasis — are being elucidated.

**Comorbidities:** Psoriasis is becoming more recognized as a disease state that may involve underlying inflammation. Understanding the importance of comorbidities associated with psoriatic disease, not only including prototypical-related conditions such as psoriatic arthritis (PsA), but also cardiovascular disease, metabolic syndrome, and inflammatory bowel disease, is important in counseling patients in a holistic manner that takes into account both their existing comorbidities as well as their risk of developing these conditions.

**Diverse presentations of psoriasis and differential diagnosis:** While many psoriasis patients present with typical findings of scaly, well-demarcated erythematous plaques on extensor surfaces, there is actually great variety in the manifestations of psoriasis, spanning inverse psoriasis, scalp psoriasis, guttate psoriasis, annular psoriasis, and nail psoriasis. In many cases, these types of psoriasis will present without the typical lesions that we are trained to look for, so a thorough history and physical is necessary along with considering important differential diagnoses such as atopic dermatitis (especially on the hands and feet), cutaneous T-cell lymphoma, pityriasis rubra pilaris, and tinea (dermatophyte) infections. Often, subtle nuances, such as whether the erythema is within the body folds or spares the folds, can help cinch a diagnosis of psoriasis rather than tinea cruris. Additionally, it is important to recognize manifestations of psoriasis in patients with skin of color, as it is often either misdiagnosed or the severity of psoriasis is underestimated in darker skin tones.

**Understanding, utilizing, and selecting appropriate treatments:** With 11 biologics approved for psoriasis and both new oral and topical entries into the market, it is challenging to keep track of all the treatment options in psoriasis. When our patients come to us, they expect that we are familiar with these options, and patients are often reassured when we can discuss them fluidly. While we have many topical steroids to choose from, combination agents and new topicals featuring novel mechanisms (tapinarof, an aryl hydrocarbon agonist, and roflumilast, a phosphodiesterase inhibitor) have expanded our topical armamentarium, so understanding the features of these medications is important to be able to use them appropriately. Being able to detail, review, and describe the mechanism of action of oral therapeutic agents such as methotrexate, cyclosporine, acitretin, and apremilast, is also critical, as is knowing when to reach for them. Newer oral agents such as deucravacitinib have opened up completely new pathways and a novel treatment class for psoriasis. We should also not forget phototherapy, which remains a useful treatment for psoriasis and other skin diseases. Lastly, biologic therapies have revolutionized management of individuals with psoriatic skin and joint disease and have allowed us to look at specific patient characteristics and comorbidities to help select an appropriate therapy.

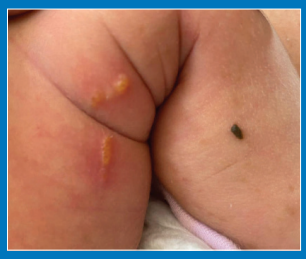
*Part two of our in-depth look at psoriasis will appear in the spring issue of* Directions. DR

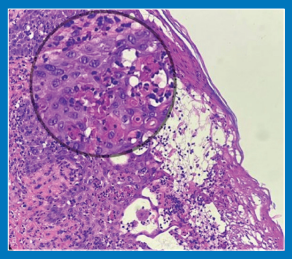
**Eduardo Michelen Gómez, MD,** is a PGY-2 dermatology resident at the University of Puerto Rico.

**Marely Santiago-Vázquez, MD, FAAD,** is a board-certified dermatologist practicing in San Juan, Puerto Rico.

## Race for the Case

By Eduardo Michelen Gómez, MD, and Marely Santiago-Vázquez, MD, FAAD





*A 23-day-old baby girl was born at term via spontaneous vaginal delivery to a healthy G1P1A0 mother. The mother consulted to dermatology services for evaluation of linear vesicular eruption in left inner thigh and left mid calf. The mother noticed the lesions shortly after birth as solitary vesicular lesions involving the left medial inner thigh, with eventual progression to linear vesicular eruption. HSV PCR, IgG, and IgM were unremarkable. The mother denied any personal or known history of similar eruptions, painful vesicular eruption, previous history of STDs, or family history of similar eruptions. She also does not report any history of hypopigmentation, hair loss, or tooth loss at early age, and no prenatal or intrapartum complications. Mother received adequate prenatal care.*

1. **What is the most likely diagnosis and most common mutation?**
2. **Describe the clinical and pathologic findings of the different stages of this genodermatoses.**
3. **Which genetic condition is associated with the typical clinical presentation of this condition in male patients?**
4. **What are the most common extracutaneous findings associated with this genodermatoses?**

Respond with the correct answers at **www.aad.org/RaceForTheCase** for the opportunity to win a Starbucks gift card!

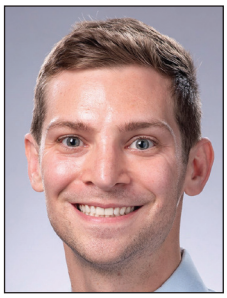
### Race for the Case winner (Fall 2022)

Congrats go out to **Courtney A. Cook, DO**, a PGY-3 resident in the department of dermatology at the University of Oklahoma, who correctly identified extragenital lichen sclerosus and gave the most comprehensive answers to the questions in the quickest time. You can read more about this case online at **www.aad.org/race-case-answers**. And if you can solve the latest case, there may be a Starbucks gift card in your future, plus you'll be invited to contribute to our Resident Life section. The race begins!

***Note:*** *We are now accepting submissions for new cases for 2023! If we accept your Race for the Case submission, we'll throw in a Starbucks gift card!*

# Procedural modalities for scar revision

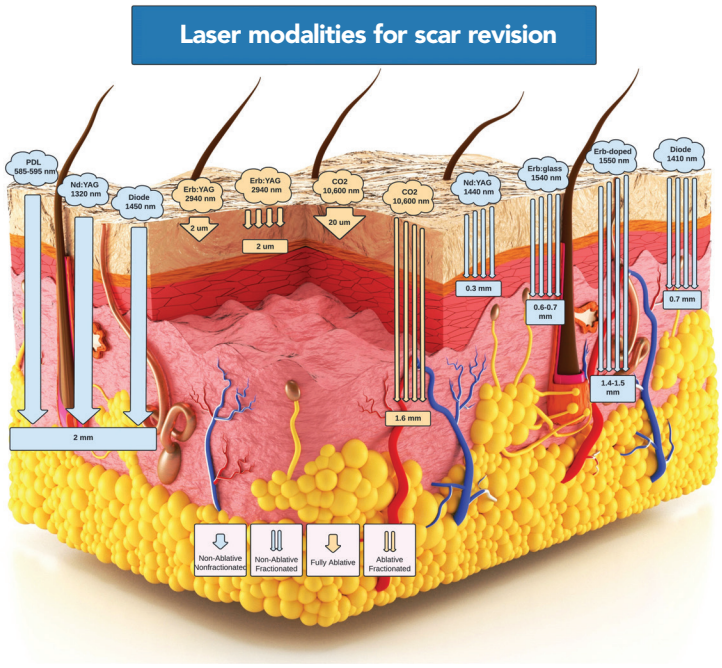
By Michael J. Visconti, DO, Emily R. Davis, DO, and Kent J. Krach, MD, FAAD



**Michael J. Visconti, DO,** is a PGY-3 dermatology resident at St. Joseph Mercy Ann Arbor.

**Emily R. Davis, DO,** is a PGY-5 micrographic surgery and dermatologic oncology fellow at St. Joseph Mercy Ann Arbor under the training of Dr. Kent J. Krach.

**Kent J. Krach, MD, FAAD,** is a board-certified dermatologist, fellowship-trained Mohs surgeon, Fellow of the American College of Mohs Surgery (FACMS), and the Program Director for the Micrographic Surgery and Dermatologic Oncology (MSDO) Fellowship at St. Joseph Mercy Ann Arbor.




## Laser modalities for scar revision

| Pulsed dye Llaser | Nd: YAG | Diode | Erbium: glass | Erbium doped | Erbium: YAG | $CO_2$ |
|---|---|---|---|---|---|---|
| **Wavelength (nm)** | | | | | | |
| 585-595 | 1320 or 1440 | 1410 or 1450 | 1540 | 1550 | 2940 | 10600 |
| **Fractionated or unfractionated** | | | | | | |
| Unfractionated | Either | | Fractionated | | Either | |
| **Ablative or nonablative** | | | | | | |
| Nonablative | | | | | Either | |
| **Primary chromophore** | | | | | | |
| Hemoglobin (**Oxy**-) | Water | | | | | |
| **Scar candidate(s)** | | | | | | |
| Pigmented scars Hypertrophic scars/keloids | Atrophic scars Superficial facial scars | | Atrophic or hypopigmented scars Moderately/severely hypertrophic scars | | | |
| **Complications** | | | | | | |
| Purpura Pigmentary change | Pain Edema Blistering | Hypopigmentation (Fitzpatrick IV-VI) | Moderate pain (>> PDL) | Scabbing Crusting Pruritus | Elevated bleed risk | Longer down-time |

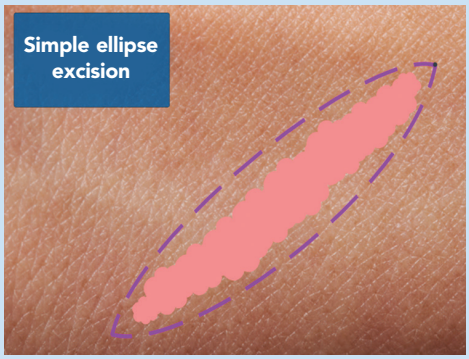
# Procedural modalities for scar revision

By Michael J. Visconti, DO, Emily R. Davis, DO, and Kent J. Krach, MD, FAAD

## Surgical modalities for scar revision

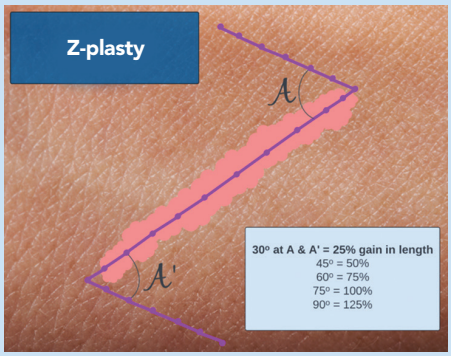
### Subcision

- **Candidates**: depressed scars of small area (i.e., facial scars)
- **Technique**:
  - Insert 20g tribeveled hypodermic needle
  - Direct tip to the deep dermis and/or subcutaneous tissue, then maintain parallel direction to skin surface while moving forward and back, which utilizes sharp edges to release fibrotic tissue



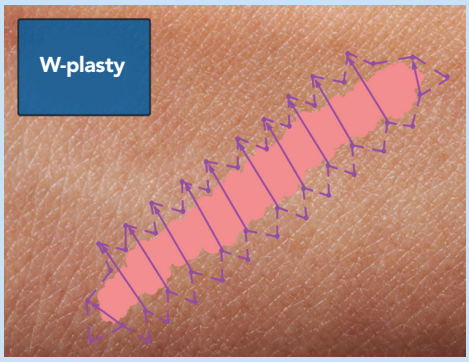

### Simple ellipse excision

- **Candidates:** spread or depressed scars (poor wound eversion), mature scars (increased laxity of neighboring skin compared to the high tension of newly formed scars)




### Z-plasty

- **Candidates:** deformations involving free margin (i.e., elevated, or depressed oral commissure)
- Serves multiple benefits: creation of irregular lines, redirection of scars parallel to RSTLs, lengthening of contracted scars




### W-plasty

- **Candidates:** shorter scars (longer → geometric broken-line closure)


### Geometric broken line closure

- More extensive W-plasty
- **Candidates:** longer scars (shorter → W-plasty)

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## More boards!

There are more new charts online! A complementary chart on **non-surgical scar revision** by Michael J. Visconti, DO, Emily R. Davis, DO, and Kent J. Krach, MD, FAAD is now available online. We also have new charts on **systemic antifungal agents** and **systemic antiviral agents** by Sujitha Yadlapati, MD, and Leah Shama-Brown, DO. Check out the full archives at **www.aad.org/boardsfodder**.

**Raj Chovatiya MD, PhD, FAAD**, is assistant professor, director for the center for eczema and itch, and medical director of clinical trials in the department of dermatology at Northwestern University Feinberg School of Medicine. He is also a member of the AAD Young Physician's Committee.

## Clinical Pearls

*Clinical Pearls help prepare residents for the future by providing them with top tips from experts about what they should know about specific, key subject areas by the time they complete their residency.*

# Atopic dermatitis

By Raj Chovatiya MD, PhD, FAAD

**1. Be purposeful with your language.** Atopic dermatitis (AD), atopic eczema (AE), dermatitis, and eczema are all often used interchangeably to describe the same entity: a clinically diagnosed, pruritic rash consisting of inflammatory changes (e.g., redness, swelling, scaling) in a characteristic distribution. Much of the confusion in terminology stems from historical precedent, changing of descriptors based on evolution of diagnostic criteria over the past century, geographic differences, and patient vs. physician preference. Currently, eczema is thought of as a group of conditions with shared signs and symptoms, of which AD (or AE) is the most common form worldwide. Given that patients and non-dermatologist clinicians often imprecisely refer to AD as eczema, dermatologists have an important educational opportunity for this disease state. This becomes especially critical when using ICD-10-CM coding, as most of the newer, targeted therapies are specifically indicated for AD.[1]

**2. Get comfortable with diagnostic criteria.** We often treat AD as a "gut" diagnosis — you know it when you see it. However, with the proliferation of so many new, targeted therapies (and many more to come) it's imperative to understand the essentials of how to make the diagnosis to connect the right patients to the right therapy. This is often easier said than done given several different accepted diagnostic criteria, many of which are not commonly used in the clinic setting. Most criteria are based upon the gold standard Hanifin and Rajka guidelines, first published in 1980, which require the presence of at least three major and three minor features from a list containing multiple options. Several groups have attempted to simplify these for routine clinical practice over the years, and in the last set of guidelines published by the AAD in 2014, only two essential features are needed for diagnosis: pruritus and eczema (consisting of typical morphology, age-specific patterns, and/or chronic or relapsing history). No matter the choice of diagnostic criteria, all agree that AD is a clinical diagnosis.[2]

**3. Don't get fooled when searching for a "classic" presentation.** Historically, AD was described as disease of childhood consisting of pruritic, red patches localized to the flexures that generally resolved by adulthood. Though this description is still commonly associated with the disease, our knowledge of AD presentation has evolved dramatically. AD is now better understood to be an extremely heterogeneous disease consisting of various cutaneous morphologies (e.g., nummular eczema, prurigo nodules, lichen simplex chronicus, lichenoid lesions, follicular papules, dyshidrosis) and topographies (head/neck, eyelids, lips, nipples, hands/feet, genitals, widespread erythroderma), with further differences in the presentation of skin inflammation (erythema, edema, lichenification, excoriation, xerosis, etc.) across diverse skin types. Looking beyond skin signs, symptoms (including pruritus, skin pain, sleep disturbance, and mental health symptoms), comorbidities, longitudinal patterns, and effects on quality of life also vary dramatically between patients.[3]

**4. Keep a broad differential in mind before locking in the diagnosis.** AD has such a high prevalence (approximately 10% in the United States) that it can be tempting to diagnose and treat any pruritic rash with characteristic eczematous features as AD. Remember: There are other subtypes of eczema along with AD, and each of these can occur in the absence or presence of AD: seborrheic dermatitis, stasis dermatitis, nummular eczema, dyshidrotic eczema, neurodermatitis/lichen simplex chronicus, and contact dermatitis. Additionally, in reflection of its heterogeneity, AD can present similarly to a variety of other non-eczematous conditions, including psoriasis (and other papulosquamous disorders), immunobullous disease, lichenoid dermatoses, tinea infections, scabies infestation, immunodeficiencies and other genodermatoses, nutritional deficiencies, and cutaneous lymphomas. Though AD is a clinical diagnosis, biopsy and/or lab workup can be especially useful in these circumstances.[4]

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# Freedom Ink restores dignity to community lives

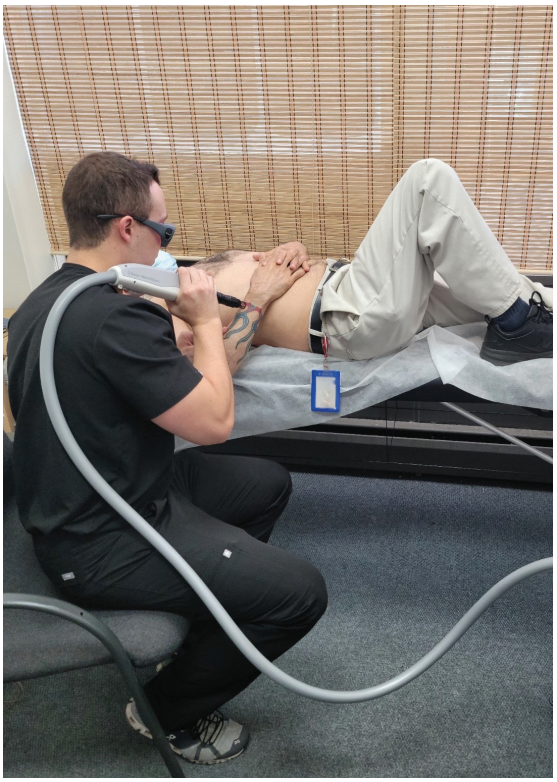
By Aili Swan, DO

On a biweekly basis, the Beaumont Farmington Hills dermatology residents volunteer our time and expertise at a tattoo removal clinic, aptly named "Freedom Ink." The program is a collaboration between the Detroit Hispanic Development Corporation and our residency.

Freedom Ink was created 16 years ago by Eric Seiger, DO, FAAD, one of our Beaumont dermatology attendings, as a community project associated with the Landmark Forum Leadership program. It provides an opportunity for people who have been trafficked and branded, incarcerated, or in gangs to have their tattoos removed at no cost. Gang-related tattoos, tattoos that would be considered offensive, and those on the face, neck, and hands can limit the opportunities of individuals who are trying to find employment and restart their lives. Freedom Ink draws people from all over Michigan as well as patients who come from other states, seeking help. To date, hundreds of patients have been helped, with more than 15,000 treatments provided.

Over the course of treatment, which can take anywhere from 5-15 visits depending on the tattoo, we develop a rapport with these individuals. We get to know them, hear their stories, and listen to the wisdom they wish to pass on. Some of them even open up about their life's journey.

Unwanted tattoos can be a perpetual reminder of a life one would choose to leave behind. We want to empower people who have had the courage to change and help them feel confident in their new path. While we hear how grateful they are for what we provide them, we are likewise grateful to them for letting us be a part of their process toward healing. DR



Trevor Nessel, DO, with a tattoo removal patient.

Left to right: Freedom Ink volunteers Trevor Nessel, DO, Brittany Valk, DO, Nate Hansen, DO, Aili Swan, DO, Ned Ivanov, DO, and Zach Burr, DO.

Drs. Valk and Burr examine a recent Freedom Ink patient.

**Aili Swan, DO,** is a PGY-3 resident at Beaumont Farmington Hills Dermatology.

Is something interesting happening in your residency program? We'd like to feature it in *Directions*.

Send your ideas to **dmonti@aad.org**.

# Inside this Issue

**Naomi Briones, MD,** is a PGY-4 dermatology resident at University of Michigan in Ann Arbor, and a member of the AAD Resident and Fellows Committee.

Our cover story is on psoriasis, one of the first topics covered during the year. The sheer volume of material to study can be overwhelming. As an enjoyable distraction to memorizing mediators and pathways, I find it fascinating to learn about the rich history of dermatologic diseases and treatments, including their origins and evolution over time. I recently had the chance to sit down with Charles Ellis, MD, FAAD, an illustrious attending in my department, who is one of the most cited dermatologists in the world and was pivotal in transforming the therapeutic approach to psoriasis through his groundbreaking publications on acitretin, cyclosporine, and the earliest biologic therapies. I'm thrilled to share some highlights from our conversation:

**Dr. Briones:** I would love to hear your reflections on the remarkable evolution of psoriasis therapies. From your perspective, what were the most exciting developments in psoriasis medications in the past few decades?

**Dr. Ellis:** One of the biggest was with cyclosporine. Most people thought psoriasis was a keratinocyte-driven disease. When we showed just how effective cyclosporine was, that tipped the field away from keratinocytes and toward psoriasis being immunologically driven. And that led to the realization that methotrexate's beneficial effect was more due to dampening the immune system than reducing keratinocyte proliferation.

**NB:** Modified Goeckerman Therapy (MGT), a resource we are grateful to offer at University of Michigan, is no longer commonly used across the U.S. and may not be well-known to some. Can you offer us some history?

**CE:** When I started in dermatology, our two main treatments for psoriasis were methotrexate and Goeckerman therapy. Dr. William Goeckerman developed it in the 1920s, but there are stories of similar techniques used by ancient Egyptians, who would go into the sun after applying paste to their skin. Originally, Goeckerman therapy utilized black tar, applied throughout the day, which was washed off before phototherapy. I remember you could smell the tar as soon as you walked onto the psoriasis unit, it was so pungent. We had to get brown pajamas and sheets manufactured because the tar had ruined so much of the hospital's regular linens! With modified Goeckerman therapy (MGT), topical steroids were added. (*Ed note: The brown pajamas have stood the test of time and are still used for our MGT patients.*)

**NB:**Where do you see the future of psoriasis? What are the most exciting potential therapies on the horizon?

**CE:** Psoriasis therapy has become increasingly targeted, and it's possible we'll find a therapy that is even more specific than those we have today. Also, for years there has been hope for "vaccines" for psoriasis. Currently, we are blocking various parts of the immune system, but perhaps eventually we can remodel the immune system and 'turn off' the pathways that induce the disease. That is the next thing that would revolutionize psoriasis therapy. DR



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**Winter 2022**

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