Tswana’s ways: a resident’s rememberance

A resident’s four-week elective in Botswana as part of an AAD Resident International Grant, prepares her for a career in the world of dermatology

As a third-year dermatology resident about to graduate, I embarked on a four-week elective in Botswana that would prove to be one of the most educational and enriching experiences of my life. I had worked in underserved inner-city clinics in the United States as well as rural villages in Latin America, but I had never been to Africa. I had no idea how much I would learn — about dermatology, about global health, and about myself.

During my month in Gaborone, Botswana in 2014, I treated more than 20 patients a day. I saw adults and children, inpatients and outpatients. Many patients presented with mundane diagnoses — atopic dermatitis, folliculitis, etc., but several others suffered from conditions that were much more difficult to diagnose and/or manage. The high rate of HIV and dearth of resources added extra elements of complexity. Initially, functioning as the only dermatologist there felt a bit unnerving. What if I didn’t know something? What if I made a mistake? These fears quickly transformed into appreciation for both my training up to that point as well as for the opportunity to have full responsibility for my patients. This autonomy inspired me to study vigorously, talk to colleagues in other specialties, read slides, and submit teledermatology consults for difficult cases.

I saw patients I will never forget. The resident who rotated before me biopsied a growing mass on a patient who had been diagnosed with dermatomyositis; the result was subcutaneous T cell lymphoma. With the help of Carrie Kovarik, MD, director of the dermatology program at Prince Marina Hospital in Botswana, we arranged for the patient — who was initially very angry and upset with us for making this grave diagnosis — to get the appropriate workup and treatment with oncology. On one of my consults, I evaluated a pregnant woman with HIV and eclampsia on her way to an emergent C-section. She had noticed pink papules scattered all over her body. My differential was wide and included varicella and gonococccemia. While awaiting biopsy and culture results, she was placed on isolation precautions. After only a few days, her baby girl died due to necrotizing enterocolitis; because the patient was in isolation, she never even got to hold her. Another memorable patient was a 10-year-old boy with flat warts all over his face and scalp. He had been tested for HIV but never received the result. It was positive, but his mother refused to allow us to inform him or arrange counseling. These heartbreaking cases and the ethical issues they raised forced me to examine my own ideas of morality and cultural biases.

Of course there were many inherent challenges and frustrations. The pace of work in the hospital was slower than we expect in the United States. Many areas of the hospital lacked soap and running water. Charts were usually illegible and often lacked important information. Rarely did the dispensary have all the medications on formulary. We worked closely with one of the pharmacists for several months just to obtain liquid nitrogen. Treatments and tests that we take for granted in the United States were a luxury in Botswana. These difficulties reminded me how fortunate we are to live and practice in the United States, and they also helped outline some of the potential areas that could be targeted to improve care.

In addition to seeing patients, I tried to teach as much as possible. I precepted students and medical officers in clinic. I participated in morning report cases, and I gave talks at Princess Marina Hospital and at our outreach sites about various dermatologic topics. I spent evenings with residents from other specialties discussing their experiences in global health and their perceptions of the healthcare system in Botswana. I travelled to beautiful parks and natural wonders I have ever seen. I made friends with some of the locals and learned about their lives and culture.

The University of Pennsylvania-Botswana partnership program has created a unique
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Amgen is a proud sponsor of the American Academy of Dermatology's Directions in Residency
opportunity for dermatology residents to learn about dermatologic diseases they might never otherwise encounter, as well as the challenges and benefits of establishing and providing meaningful, long-lasting care in a developing country or underserved area. I will be forever grateful to the AAD and the UPenn partnership for allowing me to participate in this incredible rotation. I hope that by sharing my experience, I will inspire my peers to see the value in contributing to or developing similar programs.

To learn more about the AAD’s Resident International Grants go to: www.aad.org/residentgrant.

Rachel Schleichert, MD, is a procedural dermatology fellow at the Skin Institute of South Florida and cosmetic dermatologic surgery fellow at Hollywood Dermatology.

Resident Jeopardy 2015: “this is their moment”

Are you ready for Resident Jeopardy 2015? It’s happening Saturday, March 21, from 2-5 pm in Room 303 of the Moscone Center in San Francisco. Resident Jeopardy entertains while also providing you with the ability to assess core competencies and identify knowledge gaps to better prepare you for the board exam and life after residency.

“Our residents take such pride in being the best of the best,” said David Peng, MD, this year’s director of the session. “They were each top of their class in medical school, they study their rear ends off as residents. Resident Jeopardy is a great chance for them to be recognized. It gives them a chance to shine and represent not only their respective residency programs, but also our field. We have the best and brightest. And this is their moment.”

And now (cue dramatic music), here are your 2015 teams!

<table>
<thead>
<tr>
<th>Team Name</th>
<th>Institution</th>
<th>Team Members</th>
</tr>
</thead>
<tbody>
<tr>
<td>EB Ac-quiz-ita</td>
<td>University of Minnesota</td>
<td>Kevin Gaddis, MD, PGY3 &amp; Ronnie Hamrick, MD, PGY4</td>
</tr>
<tr>
<td>Miracle College of Georgia</td>
<td>Medical College of Georgia</td>
<td>Morgan Wenner, MD, PGY3 &amp; Jessica Burgy, MD, PGY3</td>
</tr>
<tr>
<td>Rash Decisions</td>
<td>McLaren Oakland Hospital</td>
<td>Ivy DeRosa, MD, PGY3 &amp; Cynthia Chen-Lazzaro, MD, PGY3</td>
</tr>
<tr>
<td>Chili Buns</td>
<td>Wake Forest Baptist Health</td>
<td>Jacqueline DeLuca, MD, PGY4 &amp; Robin Lewallen, MD, PGY3</td>
</tr>
<tr>
<td>East Carolina University</td>
<td>Brody School of Medicine,</td>
<td>Tanner Parrent, MD, PGY4 &amp; Ryan Harris, MD, PGY4</td>
</tr>
<tr>
<td>The Woolly Bears</td>
<td>UCSF Dermatology Residency</td>
<td>Roberto Ricardo-Gonzalez, MD, PGY4 &amp; Iris Achronowitz, MD, PGY4</td>
</tr>
<tr>
<td>The Dermcats</td>
<td>University of Arizona</td>
<td>Drew Kurtzman, MD, PGY4 &amp; Christie Alexander, MD, PGY2</td>
</tr>
<tr>
<td>Made in Detroit</td>
<td>Henry Ford</td>
<td>Alison Tisack, MD, PGY2 &amp; Swati Kannan, MD, PGY3</td>
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A strategy for winning

Directions asked Erik Domingues, MD (left), who along with Vincent Criscione, MD (right) was one half of the winning University of Massachusetts team in last year’s Resident Jeopardy competition in Denver, what contributed to their success in 2014. He offered these pearls.

1. Turn your board studying into Jeopardy studying.
2. Make Sima Jain, MD, your best friend.
3. Stay calm and Jeopardy on.
4. Forget about everything else in the room and focus on the task at hand.
5. Most importantly, have fun!
### Disorders of Hypopigmentation

**by Gina Chacon, MD, and Meredith Hancock, MD**

#### Vitiligo
- Heterogeneous group of disorders with various genetic backgrounds, exogenous and intrinsic triggers
- Functional melanocytes absent
- Clinical: amelanotic macules and patches with surrounding ni skin. Discrete margins, convex borders. Usually asymptomatic.
- Precipitation for face (periorificial), dorsal hands, nipples, axillae, umbilicus, sacral, anogenital areas. +/− scaly/smooth Koebner phenomenon.
  - Localized:
    - Focal: not clearly segmental
    - Unilateral/segmental: do not cross midpoint, children (15-30%) > adults (5-10%)
    - Mucosal: only mucous membranes
  - Generalized: (90% of cases)
    - Vulgaris/non-segmental: widely-distributed, scattered
    - Acrofacial
    - Mixed
  - Universal: complete depigmentation
- Associated (adults): adult-onset AI-DM, AI-thyroid, LE, RA, Addison’s disease, pernicious anemia; +/- halo nevi, AA, lichen sclerosus
- APCED (autoimmune polyendocrinopathy candidiasis ectodermal dystrophy) syndrome: AR, ARE gene, predisposed to vitiligo and AI destruction of endocrine cells. Failure to delete autoreactive T cells → AI disease

#### MEREDITARY HYPOMELANOSIS

<table>
<thead>
<tr>
<th>Disease</th>
<th>Genetics / Pathophysiology / Histology</th>
<th>Clinical Features</th>
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<tbody>
<tr>
<td><strong>Oculocutaneous Albinism (OCA)</strong></td>
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<tr>
<td>Diffuse pigmentary dilution</td>
<td>OCA1A AR, TYR gene no tyrosine activity (tyrosinase negative)</td>
<td>At birth white hair, milky white skin, blue-gray eyes, with age hair may yellow slightly, skin remains white, melanocytic nevi remain amelanotic</td>
</tr>
<tr>
<td>40% of cases are OCA1A (A + B)</td>
<td>OCA1B AR, TYR gene ↓ tyrosine activity (tyrosinase positive)</td>
<td>Yellow OCA: little to no pigment at birth. Hair becomes yellow with age (pheomelanin requires less tyrosine)</td>
</tr>
<tr>
<td>50% of cases are OCA2</td>
<td></td>
<td>Minimal pigment OCA, platinum OCA, temperature-sensitive OCA: little to no pigment at birth. Develop some pigment in by 2nd decade. Blind without tanning. Amelanotic or pigmented melanocytic nevi possible. Temperature-sensitive: in puberty, arm hairs → light reddish-brown, leg hairs → dark-brown. Mutated tyrosinase loses activity at 35˚C (like Senease cat)</td>
</tr>
<tr>
<td>Melanocytes present</td>
<td>OCA2 AR, OCA2 gene ( prev P) Transmembrane transporter, role in organelle pH regulation, tyrosinase trafficking/processing tyrosinase positive</td>
<td>Broad clinical spectrum. Mild to mod pigmentary dilution. Little to no ability to tan. Pigmented melanocytic nevi/leukocytes develop in sun-exposed skin. Brown OCA phenotype: African descent, light brown hair and skin, irides gray to tan at birth, do not sunburn. 1% of pts with PWS/AS also have OCA2: del + mutation 15q (with OCA2/P gene)</td>
</tr>
<tr>
<td>OCA3 AR, TYRP1 gene (melanocyte specific) Eumelanin synthesis via tyrosinase stabilization</td>
<td>Rufous OCA: skin type III-V, red-bronze skin, ginger-red hair, blue/brown irides</td>
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<tr>
<td>OCA4 SLC45A2 gene (prev MATP) Transmembrane transporter role in tyrosinase processing and intracellular trafficking of proetins to melanosome</td>
<td>Most common in Japanese (25% of pts), China (10-20%), India (~10%), &lt; 5 % in Caucasians</td>
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<td></td>
<td>OCA4 Variable phenotype; hair from white to yellow-brown, ± increased pigment in hair/skin with age</td>
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<tr>
<td>OCA1</td>
<td>OCA1</td>
<td>Severe decrease in visual acuity, hypopigmentation of the retina, +macromelanosomes in the eyes and skin; skin is normally pigmented overall</td>
</tr>
<tr>
<td><strong>Piebaldism</strong></td>
<td>AD, K7 proto-oncoprogene → tyrosine kinase transmembrane receptors on melanocytes, functional receptor required for ni melanocyte migration (from neural crest and postnatally); Hist: no melanocytes in amelanotic skin/hair follicles; hyperpigmented areas with abundance of melanomas</td>
<td>Present at birth. Leukodema favors central anterior trunk, mid-extremities, central forehead, midfrontal scalp (white forelock in up to 90% pts; absence does not exclude dx); distinctly irregular in shape, well-circumscribed, milk-white; ni pigment and hyperpigmented areas within leukodema. Poliosis of eyebrows, eyelashes common. Must exclude Waardenburg syndrome via eye exam, hearing exam</td>
</tr>
<tr>
<td><strong>Waardenburg Syndrome (WS)</strong></td>
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<tr>
<td>Absence or minimal numbers of melanocytes</td>
<td>WS1 AD, PAX3 gene</td>
<td>White forelock is most frequent pigment abnormality (20-60% pts) Leukodema similar to Piebaldism Leukodema index (partial/extorsional or complete) in ≥ 20% Synophrys (confluent, bushy eyebrows), broad nasal root Dystopia canthorum. Hearing impairment in up to 37% (unilateral)</td>
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<td></td>
<td>WS2 AD, MITF gene (also SOX10)</td>
<td>Similar to WS1 (except no dystopia canthorum), deafness more common</td>
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<td></td>
<td>WS3 (Klein-Waardenburg) AD, PAX3 gene</td>
<td>Similar to WS1, with upper limb abnormalities (hypoplasia, syndactyly) Dystopia canthorum</td>
</tr>
<tr>
<td></td>
<td>WS4 (Shah-Waardenburg) AD, EOMAB or SOX10 gene</td>
<td>Similar to WS1 (except no dystopia canthorum or broad nasal root) Hirschsprung disease Rarely CNS dysfunction, neonatal hypotonia, arthrogryposis (related to SOX10)</td>
</tr>
<tr>
<td><strong>Tiez Syndrome</strong></td>
<td>AD, MITF gene (alistic to WS2)</td>
<td>Diffuse pigmentary dilution of skin/hair, deafness, hypoplasia of eyebrows. Blue eyes without nystagmus or photophobia</td>
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</table>

#### DISORDERS OF MELANOSOME BIOGENESIS

<table>
<thead>
<tr>
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<tr>
<td><strong>Hermansky Pudlak Syndrome (HPS)</strong></td>
<td>AR, 9 subtypes Most subtypes have mutations in genes encoding biogenesis of lysosome-related organelles complexes 1-3 (BLOC1/2/3 genes) → involved in lysosome-related organelles (LRO), eg. melanosomes formation.</td>
<td>Pigmentary dilution of the skin, hair, eyes; variable, depending on mutation and ethnicity (HPS1 and 3 in Puerto Ricans). ↓ pigmentation with age. Nystagmus and reduced visual acuity similar to albinism, NMSC. Systemic symptoms due to organelle dysfunction: - Bleeding tendency (on EM: absence of dense bodies in platelets) - Intestinal pulmonary fibrosis → cause of early death (30-50 y) - granulomatous colitis (ceroid lipofuscin accumulation in lysosomes) - renal failure, cardiomyopathy, arthrogryposis (related to SOX10)</td>
</tr>
<tr>
<td></td>
<td>Exception is HPS2, mutation in AP3B1 gene → involved in protein sorting to lysosomes and LRO, associated with antigen presentation, secretion of lytic granules in cytotoxic T cells</td>
<td>HPS2: recurrent bacterial infection due to neutropenia, abnl cytotoxic T-Cell function</td>
</tr>
</tbody>
</table>
 Disorders of Hypopigmentation (cont.)

by Gina Chacon, MD, and Meredith Hancock, MD

Dermis non-caseating granulomas; melanocytes
Initially, skin erythematous to red-brown color with marked desquamation

Characteristic leukoderma (sclerotic and non-sclerotic skin) is circumscribed
Diffuse pigmentary dilution

Severe protein deficiency

NUTRITIONAL HYPOMELANOSIS

Kwashiorkor
Severe protein deficiency

Initially, skin erythematosus to red-brown color with marked desquamation → hypomelanosis and/or patchy postinflammatory hyperpigmentation. Hypomelanosis starts on the face; hair is dry, lusterless, +/− red-brown color

Copper Deficiency
Tyrosinase requires copper

Diffuse pigmentary dilution

POSTINFLAMMATORY HYPOMELANOSIS

Secondary to Psoriasis, seborrheic dermatitis, atopic dermatitis, sarcoidosis, lichen sclerosis, lichen striatus, PLC, MF, LE

Multiple factors/mediators affect skin color; dysregulation caused by inflammation can alter melanosomes biogenesis, melanin production, melanosome transport/transfer to keratinocytes. Severe inflammation can lead to functional loss of melanocytes, even cell death.

Pityriasis Alba
Reduced numbers of active melanocytes, decrease in # and size of melanosomes in affected skin

Slightly scaly, round/oval, ill-defined hypopigmented macules/patches. Children/adolescents > adults, more noticeable in darker skin, summer. Early lesions pink → white/dry/powdery with pink border

Face most common site. Wood’s lamp accentuates hypopigmentation

Sarcoidosis
Dermal non-caseating granulomas; melanocytes may or may not appear normal. Melanosomes appear normal but ↓ # in keratinocytes.

Circumscribed, poorly marginated papules/plaques +/− induration. Dermal nodules surrounded by hypomelanosis. Most common on extremities, asymptomatic, no secondary changes.

Histology consistent with MF; decreased melanosomes in keratinocytes. Ni # of melanosomes in melanocytes (↓: defect in transfer)

Variant of early stage MF; most frequently seen in people with darker pigmentation. Hypomelanotic lesions on trunk/extremities, +/− pruritus. Erythema and infiltration usually present.

Lichen Sclerosis (LS)
Unknown

Genital and extragenital lesions are hypomelanotic. +/− epidermal atrophy, follicular plugging, and purpura (anogenital). Extragenital LS may be guttate.

Lupus Erythematosus (DLE, SLE)
↓ # of melanocytes, epidermal atrophy, vascular changes, pigment incontinence

Hypomelanosis/amelanosis associated with cutaneous atrophy/scarring. Hypomelanosis at lesion center often with a rim of hyperpigmentation. Pigment loss can be permanent in chronic scarring DLE, reversible in SLE.

Abbreviations:


References:

Race for the Case
By Robyn Marszalek, MD

A 65-year-old African-American male presents with a several month history of extremely pruritic, circular, clustered plaques on the lower extremities with firmly adherent, depressed, concentric circular crusts and surrounding mild erythema. His past medical history is significant for end stage renal disease on hemodialysis x 2 years, type II diabetes mellitus requiring insulin, COPD, severe coronary artery and peripheral artery disease, rheumatoid arthritis managed on prednisone 15mg daily, and recurrent Clostridium difficile colitis s/p bowel resection.

1. This condition is classified under an “umbrella” category which encompasses four clinical variants. What is the name of this “umbrella” category?
2. What element(s) in this patient’s past medical history may predispose to this condition?
3. What are histopathologic findings that all four clinical variants share?
4. For each clinical variant, what is the primary unique substance seen on histopathology that clinches the diagnosis?
5. What are felt to be cutaneous triggers of this eruption?

Answers to Winter 2014 Race for the Case

1.) What is the name of this condition?  
Cutaneous larva migrans (larva migrans cutanea)

2.) What is the most likely culprit causing this condition?  
Larva of the animal hookworm Ancylostoma braziliense, which is the most common hookworm in the Caribbean and the most common to affect humans

3.) What is a skin biopsy of this lesion most likely to show?  
Histopathology on H&E shows a predominantly spongotic dermatitis with eosinophils, often with eosinophilic vesicles.

4.) What is the preferred treatment?  
An anti-helminthic medication like oral or topical thiabendazole, oral mebendazole, oral albendazole, or oral ivermectin

Visit the Residents and Fellows Resource Center Online at www.aad.org/members/residents-fellows-resource-center
Not to be missed! Events for residents in San Francisco

Make the most out of your time at Annual Meeting and be sure to check out the great courses and events for residents, all happening at the Moscone Center in San Francisco.

**Practice Management Symposium for Residents**
**Thursday, March 19, 7 am - 5:30 pm, Room 3022**
Broaden your practice management knowledge as you learn the ins and outs of working in a group, academic, or private setting from well-known, successful dermatologists. Also, be sure to attend a special reception, immediately following the symposium at 5:30 in the 3rd floor foyer of Moscone West. Network with your peers and symposium faculty. Drinks and hors d’oeuvres will be served!

**Board Prep for Residents C005 (NEW)**
**Friday, March 20, 9 am - 5 pm, Room 3001**
Gain hands on experience by taking a “mock certification exam” in the morning and review your results in the afternoon.

**AAD Career Networking Event**
**Friday, March 20, 5 pm - 7 pm, Golden Gate Ballroom A**
Make connections and explore different practice setting opportunities.

**Resident Reception**
**Friday, March 20 5 p.m. - 6:30 p.m., Golden Gate Ballroom B**
Network with fellow residents and meet up with old friends.

**Resident Jeopardy S024**
**Saturday, March 22, 2 pm – 5 pm, Room 303**
Engage in a friendly competition and test your knowledge (see page 3).

**Residents and Fellows Symposium S025**
**Sunday, March 22 11 am – 2 pm, Room 122**
Latest research discoveries!

**Boards and Beyond F083**
**Sunday, March 22 1 – 3 pm, Room 3010 (West Bldg.)**
Hear from successful dermatologists about the boards and life after boards.

**High Yield “Power Hour” for Residents F097**
**Sunday, March 22 3:30 pm – 5:30 pm, Room 3005 (West Bldg.)**
Examine pearls from topics encountered during training.

**Young Physicians Pearls and Pitfalls F103**
**Sunday, March 22 3:30 pm – 5:30 pm, Room 2008 (West Bldg.)**
Receive lessons learned in the first 10 years out from other young physicians.

**Board Blitz S039**
**Monday, March 23, 9 am – 12 pm, Room 103 (South Bldg.)**
Discover key points and tips in preparing for the certification exam.

**Advanced Self-Assessment of Dermatopathology C025**
**Monday, March 23, 9 am – 5 pm, Room 3001 (West Bldg.)**
Analyze 50 complex dermatopathology cases.

For a complete listing of all the sessions taking place, be sure to check out the Program online at aad.org.

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**Move over Zagat! The Sbicca List for San Francisco**

A West Coast dermatology resident and foodie provides personal picks and options for residents attending the AAD Annual Meeting 2015.

*by Jennifer Sbicca, MD*

I’ve lived all over the San Francisco Bay Area: San Francisco (mission district), Oakland, Berkeley, Santa Clara, and Campbell. When Directions in Residency asked for my top ten restaurant suggestions, I put down my fork, picked up my pen and scribbled down my thoughts on local dining.

1. **Best and Closest to Moscone Center** – Trou Normand and Super Duper Burgers
2. **Best Neighborhood Nooks** – Frances and Nopa.
3. **Best Mexican** – Cancun, El Farolito, and Papalote.
4. **Best Coffee** – Blue Bottle and Tartine
5. **Best Asian** – Burma Superstar and House of Nanking.
6. **Best places to impress** – Gary Danko and The Slanted Door.
7. **Best Vegetarian** – Greens and Verbena.
8. **Most starred** – Benu and Saison.
9. **Best Dim Sum** – Yank Sing.
10. **Best Brunch** – Foreign Cinema and Mr. Benjamin.

For more on these restaurant picks, be sure to read the extended story in Annual Meeting News, onsite and in your email, during the Annual Meeting in San Francisco.

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“Strive not to be a success, but rather to be of value.” –Albert Einstein

Beyond relativity, it seems that Einstein understood emotional intelligence. Value in healthcare has been previously defined as the health outcomes achieved per dollar spent. Value, value, value…say it three times fast, and you will catch a glimpse of the genie of Medicare reform. In January, the US Department of Health and Human Services announced that by 2018, 90 percent of payments will be tied to delivering value! As perplexing a measurement as the latter sounds, perhaps receiving a medical infusion of value is not necessarily a bad thing. In business [ie. healthcare], value is one characteristic that sets apart two otherwise identical products or services [ie. physicians/hospitals]. As residents, we should strive to become experts in delivering value to our patients sooner rather than later. From our vantage point — as dermatologists — this should be an easy application: ensure patients feel comfortable, sit down face-to-face during clinic visits, listen without interrupting, use patient friendly language, perform timely call-backs, answer questions honestly, etc. I bet many of you practice in this fashion on a daily basis! What about your attendings?

It seems Einstein was onto something: success is measured at the individual level, while value is projected for others’ benefit. The AAD is equally interested in demonstrating value by addressing the diverse needs of residents/fellows. I can attest that the AAD is honest, sincere, and steadfast in its mission to young physicians. In what remains an enigma, the current retention rate of AAD memberships of graduating residents is approximately 80 percent. The trend three to four years out slowly recovers to 93 percent retention of young physicians, which is impressive. However, this statistic reflects lost time for young physicians to benefit from the panoply of AAD resources at perhaps the most crucial time. The specialty misses out on some of the bright minds, which are desperately needed for dermatology to navigate future hurdles. To the 2015 graduates: please take advantage of the fleeting months of residency, trust in your training, and keep the AAD a central part of your career. It needn’t be lonely in the trenches. Get involved in the AAD and AADA. The value-added service that you will provide as a board certified dermatologist is unmatched!

It has been an honor and pleasure serving as chair of the Resident/Fellows Committee, and indubitably, you’re in store for yet another valuable addition — to this committee — with Nathan Miletta, MD, taking the chair this month.

Amgen proudly supports the American Academy of Dermatology and the Directions in Residency newsletter.