Readers of Directions are always looking for improved methods of dermatology residency study. After completing his residency in 2012, Jules Lipoff, MD, went on to create and publish a new review book, Dermatology Simplified: Outlines and Mnemonics. The title intrigued us, so we talked to Dr. Lipoff about how he learned to study, and how these methods worked for him.

Where did you do your residency and why?

My residency was at Einstein-Montefiore Medical Center in the Bronx, New York. I was there for residency from 2008 to 2012, and I also was there for medical school from 2003 to 2008. I chose Einstein because of its strength and dedication in teaching and my familiarity and comfort with the program.

Were you familiar with Michael Fisher, MD — the founding chairman at the time — and the training?

Yes. The training was clinically focused with a strong emphasis on medical dermatology. Michael Fisher, MD, was the founding chairman who stepped down several years ago into an emeritus role. His influence upon the Einstein program’s style of teaching cannot be overemphasized.

What about Dr. Fisher’s process made an impact on you?

When Dr. Fisher runs grand rounds, residents examine patients without any history and record their differential diagnoses on paper to turn in to him. They are not to use any books or Internet, nor discuss with colleagues. In conference, Dr. Fisher calls on residents to explain their thoughts in front of the group. He will also anonymously read all of the thoughts of the residents not called on, and finally asks for pathology to be revealed if available for discussion.

So it’s a Socratic Method?

Yes, Dr. Fisher’s style of Socratic teaching emphasizes the importance of morphology and building a differential diagnosis — having the right method is more important than finding the one right answer. It’s this art of the practice of medicine that lured me into dermatology in the first place as a medical student.

Dr. Fisher emphasized “reaction patterns.” Can you explain what this means, and how it differs from any other methods/approaches?

Dr. Fisher’s reaction patterns are the five basic morphologic categories of skin diseases: papulosquamous, eczematous, vesiculobullous, vascular, and dermal. It’s a system that teaches you to allow the morphology to dictate your differential diagnosis, instead of a random approach of naming different diseases that come to mind for various reasons. Many diseases do not cleanly fit into this scheme, but it is an organized approach that allows you to focus your learning and build upon it as your knowledge base grows. In my book, I sought to share the value of Dr. Fisher’s system, but also expand upon it to include every dermatology disease into as few categories as possible (13 total categories, including the original five), while still enumerating each disease’s high-yield facts, buzzwords, and pearls.

In your book, Dermatology Simplified, the medical dermatology section is organized by clinical differential diagnosis or by pathophysiology. What is the value of studying this way?

I think it’s important to learn multiple approaches to categorizing and learning dermatology diseases: by morphology, by anatomic location, by clinical context, by molecular pathways, etc. These different ways complement each other and reinforce underlying themes.

Can you give an example of how this method of study can be effective?

Here’s one example: let’s say a patient presents with an eruption on the bilateral legs. With that information alone, there is a differential. If instead there’s a purpuric eruption, that’s another differential. If the patient has a combination, a purpuric...
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REACTION PATTERNS from p. 1

eruption on the bilateral legs, the two approaches, one from distribution and one from morphology, complement each other and help you narrow your focus.

Further, it can help expand ideas: what clinical context could predispose red blood cells to escape the vessels — inflammation like vasculitis or pigmented purpuric dermatosis; thrombocytopenia; weakened collagen from inherited disease (Ehlers-Danlos); medication (prednisone); deposition (amyloidosis); nutritional deficiency (scurvy); etc. etc.

**What are your thoughts on study habits? Can they be developed, improved?**

I think studying is very person-specific. Some people need to work alone; others thrive in groups. Some people read a book and mark it up meticulously; others read books quickly but repeat with multiple passes. Mostly, I think people shouldn’t reinvent the wheel — trust what has worked for you in the past, and try to make it fun! For me, I tried to make studying for boards into a game — questions were designed to test and trick me, and I had to make sure I could win. In general, don’t be afraid to get things wrong. If anything, try to fail as early and as often as possible, because you learn the most from your mistakes (I never forgot anything I got wrong that Dr. Fisher asked me). If you are asked a question and get it right, kudos for you! If you get it wrong, then that’s also great, because you’ve just figured out something you didn’t know.

**What do you believe is the hardest part?**

Getting over your ego and directly tackling your biggest gaps in knowledge and weaknesses — you can’t worry that you don’t know enough or that others know more. I’m learning new things every day; it’s a constant journey. Don’t beat yourself up when you find things you don’t know despite studying hard — you have to see these discoveries for the gifts that they are, since identifying specifically what you don’t understand is the biggest challenge. It’s difficult to see mistakes as a positive, but they really are!

**What do you remember about your boards?**

I was surprised at how anticlimactic taking the boards was. In the end, it’s just a test, and there’s a limit to what it can measure. It can’t tell if you are going to be a good dermatologist. The boards felt just like an in-training exam to me, only harder and longer (not as many obvious questions as I anticipated). After boards came and went, it didn’t feel satisfying, as though I had not gotten the chance to show for all the hard work and studying I had done. But it taught me that we can’t view residency as a journey to do well on a test; the test is an important motivation for learning facts, but we must always balance board studying with a focus on the right approach and methods to being a good dermatologist, and tests don’t necessarily measure that.

**What prompted you to work on and publish Dermatology Simplified? What gap does it fill?**

I wanted to create a text that would, as simply as possible, compile all the nuggets and facts I have learned in one place. My goal is that it’s not only a review book, but also a book that presents its own way of organizing the facts and dermatology residency curriculum. This is the book I wish I had when I was a resident — it would have saved me so much time figuring out what I needed to know!

(And I still use it as an external brain for the things I can’t remember.)

**When did you start?**

I began my first drafts of the book during residency by writing outlines since I had trouble consolidating the different ways certain subjects were taught by teachers, books, and my hands-on experience. Writing the book gave me purpose in studying to always have my ears open to facts and details that seemed important. My hope is that the book approaches an organized compilation of all the facts any dermatology resident could be expected to know.

**Do you have any thoughts on mentorship?**

William James, MD, and Carrie Kovarik, MD, have primarily been my mentors since I’ve been an attending at Penn. They are both excellent mentors and have supported me in different ways. Dr. Kovarik has supported my interests in teledermatology and global health — both as a resident from afar (since I was at a different program) — and now. Dr. James is a great mentor for supporting my general career goals and teaching development. I am completely humbled by both of them; they are amazing people. I think it is essential not only to seek mentors to find guidance for your career, but also to share your insights and ideas from the very beginning with your colleagues, and seek to be a mentor yourself.
### Granulomas

**by Amanda Laska, MD and Danielle Neal, DO**

<table>
<thead>
<tr>
<th>Disease</th>
<th>Epidemiology</th>
<th>Pathogenesis</th>
<th>Clinical features</th>
<th>Histopathology</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Sarcoidosis</strong></td>
<td>Bimodal: ages 25-35 and 45-65; more often in African-Americans, esp. women; children may develop before age 4 or at ages 8-14</td>
<td>Th1 CD4+ pattern upregulated following antigen stimulation; unknown antigen (perhaps infection due to seasonality); HLA-DRB1, DQB1 good prognosis</td>
<td>25% with skin involvement; red-brown papules/plaques on head, neck, upper trunk/arms; hypopyonlgmentation, nodules, alopecia; erythema nodosum a/w good prognosis; may koebnerize with trauma</td>
<td>Superficial and deep collections of epithelioid histocytes with sparse lymphocytic infiltrate; Langhans giant cells possibly containing asteroid/Schaumann bodies</td>
<td>Coriosteurocytes (topical, IL systemic) Antiinflammatory Tetracyclines PUVA Methotrexate TNF-alpha inhibitors</td>
</tr>
<tr>
<td><strong>Granuloma annulare</strong></td>
<td>2-1 female to male affected; 2/3 younger than 30 years old, no racial predilection; Classic: children, young adults; Generalized: middle-aged females; SubQ: children (boys &gt;girls) &lt; 8 years old</td>
<td>Unknown: possibly incited by infection, trauma, UV light; Th1-type inflammation; can exhibit Koebner response; possible relationship to HLABw35</td>
<td>Classic: annular plaque on dorsal hands/feet, arms, legs and trunk. Generalized: 10-100s small coalescing papules on trunk/symmetric extremities, a/w lipid abnormalities; Perforating: papules with umbilication; SubQ: deep nodules common on dorsal foot</td>
<td>Two patterns: 1. Palsading histocytes + lymphocytes around central altered collagen in superficial and deep dermis; mucin present 2. Intestinal: histocytes, monocytes + mucin amongst altered collagen</td>
<td>Observation Topical/IL steroids Topical calcineurin inhibitors Cure surgery PUVA/UV-A IL IFN-gamma For systemic: Nicotinamide Itraconozol Triple antibiotics with rifampin, ofloxacin, minocycline</td>
</tr>
<tr>
<td><strong>Necrobiotic lipiodica</strong></td>
<td>&gt;50% of patients have diabetes/glucose intolerance; 3:1 female to male ratio</td>
<td>Unknown: possibly vascular disease resulting from immunoreactants or microangiopathic change seen in glucose intolerance</td>
<td>Red-brown papules that coalesce and become yellow, atrophic plaques with elevated border usually in prebital region; rarely a/w squamous cell carcinoma, ulceration</td>
<td>Square punch with pali- saded alternating tiers of epithelioid histocytes and degenerated collagen; superficial and deep perivascular mixed infiltrate with plasma cells; mucin rare</td>
<td>First-line: Topical/IL/oral steroids Second-line: Pentoxifylline ASA + dipyridamole Nicotinamide PUVA/UV-A Thalidomide Surgery for severe lesions</td>
</tr>
<tr>
<td><strong>Anular elasto- lytic giant cell granuloma (Miescher’s granuloma, actinic granuloma of O’Brien)</strong></td>
<td>Uncommon: middle-aged women (&gt;40); Uncommon: middle-aged women; children; children also be affected</td>
<td>Unknown: may be variant of GA; possible cell-mediated response to antigen on actinically-damaged elastic fibers</td>
<td>Sun-exposed sites (head, neck, upper extremities): annular plaques with atrophic center and raised, erythema- tous border; multiple small papules usually &lt;10mm and fewer than 10 lesions that coalesce on sun-exposed skin</td>
<td>Upper-mid dermis with histocytes, giant cells; epithelioid histocytes with occasional pali- sading and no altered collagen; giant cells engulf elastin (elastophagocytosis) and stain positive with elastin stains; lack of elastin within granulomatous regions characteristic; no mucin</td>
<td>Difficult to treat; responds poorly to: Topical/IL steroids PUVA Antimalarials Retinoids Arecoloidal reports: Cyclosporine Chloroquine</td>
</tr>
<tr>
<td><strong>Cutaneous Crohn’s disease (metastatic Crohn’s forms non-casing granulomas while other cutaneous findings do not necessarily)</strong></td>
<td>20-45% of patients with Crohn’s will develop cutaneous Crohn’s; 2/3 are female</td>
<td>Th-1, Th-17 cytokines elevated; thought to be immunologic response to enteric bacteria</td>
<td>Genital lesions include labial/scrotal swelling, perianal lesions (fistulas, ulcers); non-genital lesions include oral/leg ulcers, non-descript erythematous papules/nodules in other locations</td>
<td>Epithelioid granulomas with surrounding lymphocytes, non-casing, superficial and deep dermis involved</td>
<td>Severely unrelated to intestinal Crohn’s Metronidazole Topical steroids Treat underlying Crohn’s</td>
</tr>
<tr>
<td><strong>Foreign body reaction</strong></td>
<td>Non-biologic foreign bodies include: tattoo, paraffin, silicone, silica, aluminum, beryllium, talc</td>
<td>First have infiltrate of neutrophils followed by macrophages that engulf foreign material; then may form multinucleated giant cells</td>
<td>Acute erythema/inflammation initially followed by chronic inflammation manifested most commonly as red-brown papules or nodules or plaques all site of injury</td>
<td>Several patterns possible: lichenoid, pseudolymphomatous and granulomatous; in latter, may have predominance of either epithelioid histocytes or Langhans-type giant cells that may contain inclusions of a cytoplasm</td>
<td>Depends on inciting agent: Tattoo reaction: IL/topical steroids, surgical excision, lasers Other non-biologic agents: excision Fillers reaction: hyaluroni- dase/IL steroids</td>
</tr>
<tr>
<td><strong>Necrobiotic xan- thogranuloma</strong></td>
<td>Rare condition affecting men and women equally; average age is sixth decade</td>
<td>Strongly associated with mononuclear granulopathy (fgGK) and lymphoproliferative disorders (usually not aggressive); may elicit giant cell granulomatos response</td>
<td>Cutaneous findings include: yellow periiorificial papules and plaques; trunk may form red-yellow annular plaques with atrophic center</td>
<td>In mid-dermis or subcu- tis, palisading granulomas composed of histiocytes, foam cells, giant cells surrounding zone of altered collagen; cholest erol clefts present</td>
<td>Treatment of underlying paraproteinaemia: Chlorambucil, melphalan or cyclophosphamide Systemic corticosteroids Radiation CO2 laser Plasmapheresis</td>
</tr>
<tr>
<td><strong>Rheumatoid nodule</strong></td>
<td>20% of rheumatoid arthritis patients affected; associated with moderate to high titer RF</td>
<td>Interspary of genetic and environmental factors; link to HLA-DR4; aggregates of immune complexes consisting of RF may contribute</td>
<td>Skin colored, nontender nodules millimeters to centimeters in size over extensor joints, commonly elbows and dorsal hands; rapid appearance of multiple nodules a/w metho- trexate/TNF inhibitors</td>
<td>In deep dermis/subcutis are palpated histocytes around fibrin; no mucin is present</td>
<td>Excision (often recur) Other non-biologic steroids can reduce size RA treatment usually has no effect</td>
</tr>
</tbody>
</table>
Granulomas (cont.)

by Amanda Laska, MD and Danielle Neal, DO

<table>
<thead>
<tr>
<th>Disease</th>
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<tbody>
<tr>
<td>Primary inoculation tuberculosis (cutaneous primary complex)</td>
<td>Worldwide distribution, but commonly seen in developing and impoverished populations; less than 10% of infection leads to clinical disease</td>
<td>M. tuberculosis infection and interaction with T lymphocytes/mycobacterial antigens increased MHC II antigens and IL-2 macrophages accumulate and granulomas are formed. (patient with no immunity to bacteria)</td>
<td>Inoculation into skin/mucosa is painless, firm, red-brown papule develops 2-4 weeks after inoculation erodes to sharply demarcated ulcer or spontaneous healing in 3-12 months with residual atrophic scar</td>
<td>Initial lesions may have a suppurative mixed dermal infiltrate (neutrophils, lymphocytes, plasma cells) and subsequently become granulomatous with necrosis, ulceration and caseation (weeks); AFB may be isolated</td>
<td>First line: Rifampin + isoniazid + pyrazinamide + ethambutol, Streptomycin Second line: Thiacetazole, Streptomyacin, Amikacin Quinolones</td>
</tr>
<tr>
<td>Tuberculids</td>
<td>Similar geographic distribution as primary inoculation tuberculosis</td>
<td>Immune reaction in skin due to hematogenous dissemination of M. tuberculosis antigens from an internal focus; (patient with high cell-mediated immunity to bacteria)</td>
<td>1. Erythema induratum: subcutaneous, erythematous nodules on bilateral calves involving creating ulcers that heal with scarring</td>
<td>1. Erythema induratum: tuberculous panniculitis, may see extension of tuberculid granulomas into lower dermis</td>
<td>First line: Rifampin + isoniazid + pyrazinamide + ethambutol, Streptomycin Second line: Thiacetazole, Streptomyacin, Amikacin Quinolones</td>
</tr>
<tr>
<td>Leprosy (tuberculid leprosy-  TT, borderline tuberculid- BT, borderline- BB)</td>
<td>Prevalent in tropical environments, including India, Asia, Central Africa, Central and South America</td>
<td>Immune reaction in skin due to hematogenous dissemination of M. tuberculosis antigens from an internal focus; (patient with high cell-mediated immunity to bacteria)</td>
<td>Immune reaction in skin due to hematogenous dissemination of M. tuberculosis antigens from an internal focus; (patient with high cell-mediated immunity to bacteria)</td>
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</tr>
<tr>
<td>Late syphilis (tertiary syphilis)</td>
<td>Worldwide distribution, higher rate in homosexual men, seen in 1/3 of untreated individuals months to years after initial infection</td>
<td>Incubation period from months to years; bacilli affects peripheral nerves, salivary glands, bones and viscera</td>
<td>Clinical presentation highly dependent on immunologic status of infected patient</td>
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</tr>
<tr>
<td>Cutaneous leishmaniasis</td>
<td>Seen in 1/3 of untreated individuals months to years after initial infection</td>
<td>Clinical presentation highly dependent on immunologic status of infected patient</td>
<td>Acute lesions: papules that become nodular and ulcerate over time, leaving a scar</td>
<td>Acute lesions: papules that become nodular and ulcerate over time, leaving a scar</td>
<td>Acute lesions: papules that become nodular and ulcerate over time, leaving a scar</td>
</tr>
<tr>
<td>Granulomatous Rosacea</td>
<td>Far-skinned individuals, reported in both adults and children; also in association with HIV</td>
<td>Known: granuloma formation may be in response to Demodex</td>
<td>Persistent erythema and telangiectasia of blanching cheeks, less often chin, nose, forehead; +/- papules, pustules, rhinophyma</td>
<td>Infiltrate of lymphocytes, histiocytes, plasma cells and giant cells arranged into tuberculoid granulomas; granulomas may be centered around ruptured hair follicles; necrosis only noted in 11% of cases</td>
<td>Topical: metronidazole, azelaic acid, tretinoin Oral: tetracyclines, TMP/SMX, isotretonin</td>
</tr>
<tr>
<td>Periarticular dermatitis</td>
<td>Young females, also reported in children</td>
<td>Erythematous papules, pus-tules and occasionally vesicles arranged symmetrically around mouth, chin, nose and nasolabial folds; characteristic sparing of immediate perianal area</td>
<td>Stalk parakeratosis surrounding follicular ostia, spongiosis and acanthosis characterizes the epidemis, associated perivas-cular lymphohistiocytic infiltrate, occasional tuberculoid granuloma noted in several cases</td>
<td>Stalk parakeratosis surrounding follicular ostia, spongiosis and acanthosis characterizes the epidemis, associated perivas-cular lymphohistiocytic infiltrate, occasional tuberculoid granuloma noted in several cases</td>
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</tr>
<tr>
<td>Lupus miliaris disseminated faciei</td>
<td>Males and females equally affected</td>
<td>Known: may be related to rosacea</td>
<td>Discrete red to yellow/brown papules localized over central face and periorbital region; lesions may last for months, then heal with scarring</td>
<td>Demarcated area of dermal caseation necrosis surrounded by multinucleated giant cells and lymphocytes; more often than not associated with ruptured pilar follicles, granulomas indicative of established lesions</td>
<td>Topical: metronidazole, azelaic acid, tretinoin Oral: tetracyclines, TMP/SMX, isotretonin</td>
</tr>
</tbody>
</table>

References:
Race for the Case: Spring 2016

By Emily de Golian, MD

A 69-year-old Caucasian male presented for treatment evaluation for a 4.3 x 3.7 cm left hip plaque, which was present for 10 years prior to recent biopsy by an outside physician. Firm palpable nodules were present within this asymptomatic, growing lesion. His medical history is otherwise non-contributory.

1. What translocation is most likely present within this lesion?
2. What are the histopathologic findings?
3. Identify the immunohistochemical pattern classic to this diagnosis.
4. What is the recommended standard treatment option with the highest cure rate without recurrence?
5. What treatment is recommended for patients with recurrent or metastatic lesions?

Respond online with the correct answers at www.aad.org/RaceForTheCase for the opportunity to win a Starbucks gift card! If you win, we will also publish your mug (face), and if you have an interesting story to tell residents, we might share it (see our current winner profile to the right). Good luck!

Answers to Winter 2015 Race for the Case

Winter 2015 RFTC was submitted by Travis Morrell, MD, MPH — a resident physician at Loma Linda University Dermatology.

A 55-year-old female was admitted for pneumonia complicated by sepsis. On hospital day three, she was noted to have six asymptomatic, non-tender, edematous papules and plaques scattered on her neck, upper arms, and left hand. Her medical history including a hematologic malignancy, for which she received her first course of chemotherapy two weeks prior. On biopsy, the inflammatory infiltrate was centered on sweat ducts.

1. What is the name of the disorder? Neutrophilic Eccrine Hidradenitis
2. This was first characterized in association with what malignancy? Acute myelogenous leukemia
3. What is the most common chemotherapy association? Cytarabine
4. Classic finding on pathology? Peri-eccrine neutrophilic inflammation

Winter 2015 Race for the Case Winner Profile: Winter 2015

Congratulations to Nicole Harter, MD — a second-year resident at the University of Southern California in Los Angeles, California. She is originally from Hilo, Hawaii, but grew up in the charming, small-town of Prescott, Arizona. Nicole is pursuing a fellowship in pediatric dermatology and plans to continue a career in academic medicine, with particular interest in pediatric dermatologic surgery. When she is not with her USC residency-family, she loves to be active by running, hiking, biking, and enjoying the year-round beauty of Pasadena, California with her adventurous husband and adorable pup. Together they love to explore and live to travel! Nicole loves to cook, and her specialty is fun-flavored cupcakes — best when shared among family and co-residents!
Welcome Faranak Kamangar, MD: RFC incoming Chair!

Faranak Kamangar, MD, begins her term this month as chair of the Residents / Fellows Committee (RFC). Dr. Kamangar completed an Internal Medicine internship at the California Pacific Medical Center, and completed her medical education at the University of California, Davis School of Medicine. During that time, she also accomplished a two-year research and clinical fellowship at the University of California, San Francisco department of dermatology under the mentorship of John Koo, MD, and focused on psoriasis and complex medical therapeutics. She also completed her undergraduate with a biotechnology bachelor’s degree at the University of California, Davis.

Her areas of interest include health care policy and advocacy within dermatology; community and international outreach; and clinical research and therapeutic innovation. She has been involved in clinical research for over 11 years now, and has authored over 20 publications and book chapters. During her residency at UC Davis, her clinical research was funded by the ASDS Cutting Edge Research Grant (CERG).

In her personal time, Dr. Kamangar enjoys spending time with her family and enjoying all that California has to offer — from surfing to hiking, to maintaining a peaceful balance through meditation and yoga.

Join the Camp Discovery Residents Challenge!

Engage in a fun and friendly competition with fellow dermatology residents, and give kids with chronic skin conditions the chance to laugh, play, and enjoy the magic of summer at the American Academy of Dermatology’s Camp Discovery.

It’s an experience like no other, letting kids swim, fish, go horseback riding, hike and make friends. At camp, they’re just kids — not their skin conditions.

To take part, just put together a team and raise funds however you choose. The team that raises the most funds will be featured in AAD’s Aspire, and the top five teams will be included in Directions in Residency. Plus, the winning team will receive an award that can be displayed in their program’s offices, and each winning team member will receive a gift card!

For more information on how to sign up, go to: www.events.aad.org/residents.

Apply now! Grants available for residents

The AAD knows that residents are always looking to broaden their horizons and gain experience outside the residency program, so they’re offering two exciting opportunities to travel and provide care for underserved communities. Deadlines are fast approaching!

Resident International Grant
This is an opportunity for 15 U.S. and Canadian senior dermatology residents to participate in a four- to six-week elective in Gaborone, Botswana. Residents will rotate between the Princess Marina Hospital (in conjunction with the Botswana-UPenn Partnership) and the Baylor International Pediatric AIDS Initiative (BIPAI) to provide dermatologic HIV care for both children and adults. Residents will also be expected to prepare lectures/presentations, submit teledermatology consults, and develop a database of photos, as well as present their activities to the Academy and their home programs.

Applications for the January–June (2017) travel rotation must be submitted by April 1, 2016, while applications for the July–December (2017) travel rotation may be submitted until September 30, 2016. For more information, visit www.aad.org/international, or contact Janine Mueller at jmueller@aad.org.

Native American Health Service Resident Rotation Program
The Education and Volunteers Abroad Committee at the AAD is providing four grants to second- and third-year U.S. dermatology residents to participate in a rural health elective in Chinle, Arizona for one to two weeks. Residents will provide dermatologic care to the Navajo Nation population and will assist primary health care providers with diagnoses and disease management. Residents will be expected to keep records of consults, prepare lectures, and submit an evaluation of activities to the Academy within one month of the rotation.

Applications must be submitted by April 1, 2016 for the 2016-2017 rotations: November 2016; March 2017; May 2017; and August 2017. Applications submitted after April 1 will be considered for the 2017-2018 rotation series. For more information, please visit www.aad.org/nativeamerican, or email Janine Mueller at jmueller@aad.org.
In my final message, I would like to briefly discuss a topic even more anxiety-provoking than the board exam: financial planning.

Given declining/stagnant reimbursements, swelling administrative costs, and an average debt of $170,000 per medical student, you can see the importance of addressing financial planning early. In fact, despite relatively high incomes, physicians represent a disproportionately low percentage of the total wealth in America.

Getting started can be overwhelming. For that reason, I recommend The White Coat Investor: A Doctor’s Guide to Personal Finance, by James M. Dahle, MD. It provides a simplified blueprint for financial planning throughout the career of a physician, and highlights a number of pitfalls to avoid along the way.

As one example, look at the important student loan transitions for the average resident/fellow and several of the available options in the chart below.*

I hope you take this opportunity to seize control of your financial future and serve as a leader for others in your respective programs. As health care delivery in America evolves, financial stability will put you in the best position to serve your patients moving forward.

With the conclusion of the 74th Annual Meeting, I am happy to announce that Faranak Kamangar, MD, will be transitioning into the role of chair of the Residents and Fellows Committee (see page 7). I would like to thank our terrific staff, including Carrie Gremer, Cindy Kuhn, Jessica TenBusch, and Dean Monti for their tireless effort and commitment to dermatology. It has been an absolute pleasure to serve our residents and fellows. I thank you for this opportunity, and look forward to continued advocacy in dermatology and medicine.

AAD partners with DRB Student Loan
The AAD recently partnered with trusted lending source DRB Student Loan to provide residents with a way to consolidate and refinance loans to a much lower interest rate. Visit student.drbank.com/aad for more information.

### Table: Transition Options

<table>
<thead>
<tr>
<th>Transition</th>
<th>Program</th>
<th>Overview</th>
</tr>
</thead>
<tbody>
<tr>
<td>Medical Student to Intern/Resident</td>
<td>Direct Loan Consolidation</td>
<td>Consolidates federal education loans. May apply for one of several income-related repayment options during internship/residency. May help make monthly payments manageable during residency. NOTE: you may want to pick the option that qualifies for the public student loan forgiveness program.</td>
</tr>
<tr>
<td>Public Student Loan Forgiveness Program (PSLF)</td>
<td>If you are employed by the government or a not-for-profit employer (most residents), you may qualify to have your loans discharged after 120 consecutive monthly payments. Requires continued employment for gov’t or NPO as staff.</td>
<td></td>
</tr>
<tr>
<td>Resident/Fellow to Staff Dermatologist</td>
<td>Private Student Loan Refinancing</td>
<td>If taking a position that will not qualify for PSLF, or if you would prefer to expedite paying off loans, consider private refinancing. In particular, the AAD has partnered with DRB Student Loans to offer an additional discount on your rate.</td>
</tr>
</tbody>
</table>

**Private Refinancing Example:** $170,000 of Student Loan Debt:
- Five-year repayment at 3.59%; monthly payment = $3.579; total = $214,740
- Ten-year repayment at 6.9%; monthly payment = $2.555; total = $306,600
- Paying an extra $1,000/mo as staff will allow you to pay off loans in 5 years and save approximately $92,000

*Note: This example may not apply to everyone. Please explore all options available to you and your particular situation.

ZO Skin Health Inc. by Zein Obagi, MD, proudly supports the American Academy of Dermatology and the Directions in Residency newsletter.

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