Barrier basics. First, McGrath presented an overview of the healthy barrier’s structural and immune components and some of the more important proteins involved. Above the basement membrane, he pointed to the basal cell layer (and keratins 5, 14, 15), the spinous layer (keratins 1 and 10 and transglutaminases 1 and 5), the granular layer (and keratin 2, profilaggrin, and transglutaminases 1, 3, and 5), and the squames—the complex cornified layer—on top (with loricrin, involucrin, trichohyalin, S100 proteins, and small proline-rich proteins).

Squames are keratinocytes that have terminally differentiated and collapsed—becoming 95% keratin filaments—to maximize their protective capability. This flattening is accomplished by filaggrin (filament aggregating protein), which is then broken down to produce NMF (natural moisturizing factor). McGrath spoke of the other proteins—spotlighting the cross-linking enzyme family of transglutaminases—and the lipids involved in building and maintaining the skin barrier, and the ways in which a dysfunctional barrier compromises its physical and immune protective capabilities.

Reductionism and personalized medicine. “The story here is about the skin barrier, and going from rare diseases to thinking about common ones.” Identifying the genes in uncommon monogenetic skin barrier diseases advances cutaneous science as well as our understanding—and ultimately the treatment—of the genetically complex diseases we see frequently. They also enable us to be “splitters rather than lumpers,” providing the molecular basis for separating similar clinical entities that have been inappropriately bundled together under the same descriptive term. This approach also permits personalized treatment.

Atopic Dermatitis—The Genetics of Skin as a Barrier
John A. McGrath, MD, FRCP

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Monogenetic diseases of the skin barrier. McGrath preceded current insights about atopic dermatitis (AD) by sketching many of the more than 40 distinct genodermatoses due to a mutation in a structural or biochemical component of the cornified cell envelope. His list included the umbrella classification ARCI (autosomal recessive congenital ichthyosis) and the respective genes involved in its different manifestations. He noted monogenetic diseases of lipids and of lipid transport, of cross-linking enzymes, of protease inhibitors, of loricrin, and of keratin.

“We are starting to see how a particular drug might be best targeted to particular patients when we know their molecular pathology.” Liarozole, e.g., the new P450 cytochrome inhibitor for ichthyosis that increases levels of keratin 2 and TNF-α, works better in patients with mutations in ichthyin rather than in transglutaminase 1. “Personalized medicine will be appearing for the ichthyoses over the next several years.”

Clues to AD. “Can any of these rare monogenic skin barrier disorders provide clues to genetic risk factors for what we call AD?” Netherton syndrome, a protease inhibitor disease, provides part of the story for 1–2% of patients. Elevated IgE levels are common in Netherton syndrome and in many patients with AD. Netherton syndrome involves mutations in the SPINK5 gene, which encodes the serine protease inhibitor LEKTI. One LEKTI mutation in particular—if inherited from the mother—quadruples the risk of developing atopy.

Dermatologists know that ichthyosis vulgaris (IV) is the disease most likely to provide insight into AD. “We are all familiar with patients who have both—so we know that the gene for IV could provide fascinating clues.” IV, a semi-dominant condition, is caused by mutations in exon 3 of the filaggrin gene. Filaggrin gene mutations “are incredibly common. We see them every day, because 10% of Europeans and white Americans have at least one allele that makes no functional protein.” In addition to the more than 30 filaggrin mutations so far identified, certain units of this exon repeat in a very individualized fashion. Both the unit repeated and the number of repeats vary considerably, and each pattern has a different effect. In general, the more filaggrin repeat units one has, the more moist the skin—and vice versa. The interaction of mutations and repeats creates a very broad clinical spectrum.

Filaggrin mutations are now a known risk factor for AD. “Up to 50% of one’s patients with moderate to severe disease have a filaggrin gene mutation.” Different genetic and environmental factors contribute to the pathogenesis in the remaining 50%. Filaggrin mutations are also associated with increased severity of atopic asthma and the development of systemic allergies, and can modify other diseases. (They are not associated with psoriasis or non-atopic asthma.)

Conclusions. Maintaining the skin barrier early in life in vulnerable children may reduce the atopic march (i.e., the progression from mild atopic eczema to severe atopic dermatitis associated with asthma). Efforts are underway to find therapeutics that will enhance filaggrin production. Aminoglycoside antibiotics do this by overcoming nonsense mutations (which stop RNA translation prematurely, producing a truncated, non-functional protein) in the filaggrin gene, although their use is not problem-free. There is interesting evidence that Lactobacillus helveticus, found in fermented milk, may do this. More desirable drugs for this are anticipated.
The Dermatology Foundation is pleased to recognize the dermatologists listed below who recently joined their colleagues in the Leaders Society. These forward-looking individuals made the wise decision to invest $1,500 annually to amplify the progress that advances patient care.

(As of July 27, 2010)

**MINI-SYMPOSIUM: SURGICAL MANAGEMENT & HUMAN APPEARANCE**

**Update on Pre-Op Evaluation: Antibiotics and Anticoagulation**

*Isaac M. Neuhaus, MD*

**Introduction.** The American Heart Association substantially restricted their recommendations for endocarditis prophylaxis in 2007. Neuhaus reviewed the revisions, assessed their implications for dermatologic surgical procedures, then discussed appropriate prophylaxis in two additional contexts. When relevant, he recommended appropriate antibiotics, dosage, and timing.

**Endocarditis prophylaxis.** The AHA recognized that endocarditis is much more likely to occur from random bacteremias than from surgery, and that antibiotic prophylaxis rarely—if ever—reduces the minimal surgical risk while posing significant risk (allergic reactions, drug reactions, drug resistance, high cost). The documented ~2% bacteremia rate during cutaneous surgery is equivalent to the spontaneous incidence of bacteremia. The AHA advises prophylaxis only within their 4 narrowly defined high-risk categories, and then only before biopsy or perforation of the oral mucosa, or procedures on infected skin.

Translating this to dermatology practice suggests prophylaxis prior to an invasive procedure (e.g., excision, Mohs, simple biopsy) only if (1) the skin is infected, in which case the patient would be receiving aggressive treatment anyway; or (2) the patient is in an AHA high-risk category and the procedure involves the oral mucosa (prophylaxis 30–60 minutes beforehand).

**Prophylaxis for prosthetic devices.** Most joint infections occur during the replacement procedure itself. Guidelines were co-developed by the American Academy of Orthopedic Surgeons and the American Dental Association, which Neuhaus extrapolated to dermatology practice. Keep in mind that: the bacteremia rates are 60–90% in oral surgery vs the 2% associated with dermatologic procedures; the flora of the mouth and skin are completely different; and there are no published reports of a prosthetic infection following cutaneous surgery. Neuhaus suggested prophylaxis about 60 minutes only before a perforating procedure involving the oral mucosa if the tissue is infected (actually a primary treatment) or if the patient is in a high-risk category.

**Surgical site infection prophylaxis.** With no clear guidelines, practice varies from those who use it without exception to those who rarely if ever use it because of dermatologic surgery’s low infection rate. Neuhaus used the 4-stage risk-based classification of wounds in the general surgical literature as a

<table>
<thead>
<tr>
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<tr>
<td>ALASKA</td>
<td>Joy C. Wu, DO</td>
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<td>ALABAMA</td>
<td>Dena J. Howell, MD</td>
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<tr>
<td>CALIFORNIA</td>
<td>David H. Chu, MD, PhD, Rebecca L. Fitzgerald, MD</td>
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<tr>
<td>DELAWARE</td>
<td>Marguerite D. Thew, MD</td>
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<tr>
<td>FLORIDA</td>
<td>Just Brahmatewari, MD, Anna F. Falabella, MD, Amy Ross, MD, Jon R. Ward, MD</td>
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<td>ILLINOIS</td>
<td>Kathleen J. Green, PhD</td>
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<tr>
<td>INDIANA</td>
<td>Mitchell L. Bressack, MD, Annette M. Dinneen, MD</td>
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<td>MASSACHUSETTS</td>
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<td>MINNESOTA</td>
<td>Daniel H. Kaplan, MD, PhD, Lynda S. Kauls, MD</td>
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<tr>
<td>NEW JERSEY</td>
<td>Adrian L. Connolly, MD, Eric S. Siegel, MD</td>
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<td>NEW YORK</td>
<td>John A. Carlson, MD, William E. Clack, MD</td>
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<td>Carrie Cusack, MD, Ellen J. Kim, MD</td>
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<td>TEXAS</td>
<td>Michael J. Majors, MD, Joseph R. Peterson, MD, Deborah K. Phillips, MD</td>
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<tr>
<td>VIRGINIA</td>
<td>William L. Coker, Jr., MD, Brett Krasner, MD</td>
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**Cardiac Conditions Warranting IE Prophylaxis for Oral Procedures**

- Prosthetic cardiac valve or prosthetic material used for cardiac valve repair
- Previous IE
- Congenital heart disease (CHD)
  - Unrepaired cyanotic CHD, including palliative shunts and conduits
  - Completely repaired congenital heart defect with prosthetic material or device, whether placed by surgery or by catheter intervention, during the first 6 months after the procedure
  - Repaired CHD with residual defects at the site or adjacent to the site of a prosthetic patch or prosthetic device (which inhibit endothelialization)
- Cardiac transplantation recipients who develop cardiac valvulopathy

*Wilson et al. Circulation. 2007*

**IE Prophylaxis Recommendations**

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<td>Yes</td>
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<tr>
<td>No antibiotic prophylaxis</td>
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<td>Procedure involving oral mucosa?</td>
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**Hematogenous Total Joint Infection Prophylaxis Recommendations**

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<td>No antibiotic prophylaxis</td>
<td>No</td>
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**Surgical Pearls: When Simple is Better**

**Tri H. Nguyen, MD**

**Introduction.** Nguyen is noted for his approach to challenging excisions and wounds. He prizes simplicity—"achieving more with much less"—and shared pearls (along with demonstration videos) to make procedures simpler and faster. He suggested that his interventions typically result in greater patient convenience, less morbidity and greater safety, with even better results.

**Dermal collagen matrices.** Nguyen uses these bovine collagen products; for faster second-intention healing—especially to minimize the risk of contour depression in vital anatomic areas; in lieu of split-thickness skin grafts to avoid the morbidity of a second donor site; and as optimal preparation for a delayed full-thickness skin graft. Three patients with challenging wounds highlighted the utility of these collagen products in achieving functional restoration and excellent cosmesis. Nguyen discussed PrMatrix and Puracol-Plus with detailed instructions for using them successfully.

**Suturing pearls.** “The most important” suture for tension reduction is SMAS imbrication. Nguyen showed a patient with a recurrent BCC (5.5 x 3 cm) on the postauricular neck. With minimal undermining, the first suture is at the wound base that contains the fascial envelope—rather than the edge—which folds the fascia over itself and reduces tension on the wound's leading edge. For dermal wound tension, two throws of double vertical mattress sutures act as a buried pulley to redistribute and reduce tension on the leading edge. For wound length discrepancies, Nguyen suggests a horizontal-to-vertical suture over standing-cone excision. He places the horizontal suture on the long side (limiting the bite width to 4–5 mm), and the vertical suture is placed on the shorter side directly across from the horizontal midpoint. Nguyen avoids knots with the Quill suture retention system. Bidirectional bars allow suture retention, provide tissue support, and prevent slippage. Applied to wounds on the scalp, trunk, or neck, Quill sutures rapidly close large wounds without knot tying. Nguyen uses a broken line approach to removing large lipomas. This creates an accordion effect that allows much greater exposure of the wound base compared to a straight line. He uses a W-plasty near the scalp to mimic the natural hairline. Elsewhere he uses an S-plasty with a 3-point suture (first at the wound base, then on either side of the free edge) to bring the deep tissue upward, eliminating potential cavitation and preventing unnecessary skin excision.

**Dermal Collagen Matrix Journey**

Mohs clearance SCC (A) with immediate collagen matrix repair (B); at 13 days post-surgery, healing with collagen matrix (C) with FTSG applied over it (D); at 20 days post-surgery (E); at 42 days post-surgery (F).
Hyperhidrosis Therapy: Using Botulinum Toxins
Dee Anna Glaser, MD

Introduction. Sweating maintains normal thermal regulation. The sweat glands, located primarily at the deep dermal–subcutaneous junction, are innervated by the sympathetic nervous system via acetylcholine, which is why botulinum toxin therapy for hyperhidrosis is so effective. Excessive sweating involves an abnormal neural signal. Generalized hyperhidrosis is almost always secondary to another condition—e.g., endocrine, infectious, malignant—that must be identified and treated. Most focal hyperhidrosis is idiopathic. This affects roughly 3% of the population and is what dermatologists treat. The treatment goal is to reduce sweating from unbearable to tolerable, not stop it completely. There are many treatments, with the focus here on botulinum toxin injections (type A is commonly used) and mostly off-label uses. Botox® is FDA-approved for treating axillary hyperhidrosis. Dysport’s recent approval was for cosmetic use and cervical dystonia. Type B is infrequently used because of systemic side effects.

Axillary hyperhidrosis. With Botox®, 50 units are used per underarm. Glaser begins with a starch-iodine test (a Betadine swab and cornstarch from the grocery store) on a very dry underarm. In 2–5 minutes, a blue-black color precisely identifies where the sweat is produced. Reconstitute the Botox® with 4 cc of normal saline. Dermal to subcutaneous injections are ~1.5–2 cm apart for effective diffusion, with the number of shots determined by the size of the area to be treated. If an effective starch-iodine test cannot be done, treat the hair-bearing area and explain that any missed areas will be treated in a couple of weeks. Treatment duration averages 7 months.

Non-axillary locations. Palms, soles, face, scalp, and groin, among other areas, are part of Glaser’s practice, with the palms most common after the underarms. Use 100 units for smaller hands and 200 units for larger ones; injections are every 1–15 cm. Adequate pain control is critical. Glaser relies primarily on cold and vibration. Ice should always be applied with pressure. (Despite the attempt to stay superficial when injecting the Botox®, muscle weakness occurs in ~20% of palmar treatments.) Glaser uses the same technique for feet, adding a vibrator for analgesia because pain is greater here. She is beginning to see many more faces and scalps in her practice, with sweating patterns that vary from primarily forehead to an ophiasis pattern to global to facial. (Botox® cannot treat all areas of the face because of the underlying musculature.) Dosing is unknown, but Glaser finds 2–5 units every 2 cm to be effective in these areas. She briefly discussed use of Dysport.

Conclusion. Glaser concluded with time-saving tips, which includes sending new patients to www.SweatHelp.org so they arrive at their first visit knowing the options.

Botulinum Toxin Therapy

- BTX A
  - Excellent efficacy and safety
  - Botox® FDA-approval July 19, 2004 for severe primary axillary hyperhidrosis
  - Dysport® FDA-approval for glabellar lines and cervical dystonia
- BTX B
  - Limited numbers of HH patients treated
  - Systemic side effects reported

Starch-Iodine Test

Dermatological Toxicities of Novel Cancer Therapies
Mario E. Lacouture, MD

Background. An estimated 1.5 million people in the U.S. were diagnosed with cancer in 2009, with approximately 650,000 receiving chemotherapy and 700,000 receiving radiotherapy. Over 41 therapeutic agents produce 52 different dermatologic toxicities or adverse effects, and many cancer survivors are left with “a constellation of findings once cancer treatment has been completed.” Skin irritation and dry skin are among the 3 symptoms that most impair quality of life (QOL), outranking constitutional and GI side effects. Up to 76% of patients required dose reduction of their
Dermatology Foundation Member Profile:
Dermatologic Surgeon Supports the Future of the Specialty

Isaac Neuhaus, MD, recognized early in his career that the work of the DF helps keep the specialty he loves vibrant and cutting-edge. That is why he made the decision to be a member of the DF’s Leaders Society following completion of his Mohs fellowship in 2005. “I have witnessed the benefits of DF support first-hand through colleagues who have earned DF research awards,” Dr. Neuhaus says. As an Assistant Professor at UCSF, he sees how their results are already expanding knowledge and expertise. He points to one of his associates who received a DF Career Development Award to study important aspects of Merkel cell carcinoma. Few people have studied this rare and serious type of skin cancer, and there are many critical questions about how to care for these patients effectively. “It would have been very difficult to study this independently,” Dr. Neuhaus observes. “With the help of a DF grant, this physician-scientist is quickly becoming one of the experts in this area.”

Dr. Neuhaus explains that this kind of progress is essential for the specialty. “Dermatologists are very fortunate. We love what we do, and I think we have one of the highest satisfaction levels of any medical specialty. But the only way we can continue to be experts for our patients—really deliver the care we enjoy so much—is to ensure that dermatology continues to move forward. We cannot all do research, but we can all contribute to it through the DF. It’s the one way for us to give back—and it will come back to us in multitudes.”

Hair abnormalities. Alopecia occurs in 65–100% of patients treated with agents affecting rapidly dividing cells, most frequently the taxanes, alkylating agents, topoisomerase inhibitors, and antimetabolites. The psychosocial impact of hair loss is substantial, and hair regrowth is sparser and with altered color and texture. A cumbersome scalp cooling device used during infusion can reduce complete alopecia by 81%, and the prophylactic application of minoxidil 2% shortens its duration. A nitroxide is in phase II trials for preventing radiation-induced alopecia. Hair abnormalities associated with targeted therapies are emerging now, especially trichomegaly and increased eyelash growth (potentially eye damage) with EGFRIs, and curly or depigmented hair.

EGFR-induced skin rash. This is “perhaps one of the most significant toxicities seen in patients with modern cancer therapy.” Red papular pustules develop on the face and upper body in ≥80% of treated patients. Up to 60% also experience pruritus and tenderness. The most commonly used EGFRIs—erlotinib (oral drug), and cetuximab and panitumumab (IV)—are FDA-approved for lung, colorectal, pancreatic, and head and neck cancers. Many of these patients have metastatic disease, yet rash severity requires dose interruption (70% of patients) or treatment discontinuation (30%) by oncologists. This rash severely impairs QOL (Skindex-16), and adds an average of $2,7000 to treatment costs. Some toxicities are unresponsive to standard treatment (antibiotics and topical steroids). And ≤38% of EGFRI-treated patients with rash develop skin infections—“a complication of a complication”—that worsen the dermatologic toxicity. Lacouture found that prophylactic treatment for the first 6 weeks of treatment in panitumumab-treated patients—doxycycline 100 mg twice daily, hydrocortisone cream, moisturizer and sunscreen when needed—reduced ≥grade 2 skin toxicity from 62% in reactively treated patients to only 29%. Numerous trials are in progress for managing the most common dermatologic side effects of EGFRIs.

Radiation—which commonly causes dermatitis, itself poorly managed—is used in ~50% of cancer patients and is now increasingly combined with EGFRIs. Lacouture’s meta-analysis of combined treatment trials found a more than double risk of severe radiation dermatitis.

Hand–foot syndrome and nail changes to taxanes. The hand–foot syndrome associated primarily with capcitabine or pegylated doxorubicin—AKA palmoplantar erythrodysesthesia—occurs in 6–42% of this patient group. It differs from the reaction associated with the taxanes, sorafenib and sunitinib—hand–foot syndrome associated with tenderness of hemorrhagic or air-filled blisters in areas of friction or pressure. Still different is the taxane-caused Pateo syndrome—periarticular thenar erythema with onycholysis. The erythema of the dorsal hands responds to high-potency topical steroids. Lacouture treats the onycholysis with a vinegar soak for hands and feet, adding culture-driven antibiotics. Prophylaxis to docetaxel-induced nail changes—which decreases nail toxicity incidence by >50%—involves placing hands and feet in frozen gloves and slippers during the infusion. To treat the challenging paronychia caused by EGFRIs, Lacouture uses weekly silver nitrate chemical cauterization in the lateral nail folds, eliminating the need for nail avulsions.
The dermatologist’s role. The dermatologist should routinely be involved in the care of oncology patients, beginning before therapy with counseling and available prophylaxis. Further, dermatology’s unique contributions include: (1) active pharmacovigilance, i.e., close observation of these patients “to recognize dermatologic side effects that would be unrecognized or misdiagnosed by those without dermatology training,” increasingly important as these drugs are approved in the adjuvant setting; (2) EGFRIs’ significant potential in the dermatologic setting for hyperproliferative skin diseases; (3) EGFR-induced rash severity correlates with therapeutic response, and it is likely that with other drugs as well, the induced skin effects can be identified as surrogate markers for therapeutic activity.

MINI-SYMPOSIUM: EMERGING DISEASES & THERAPIES

Emerging Infestations and Infections
Dirk M. Elston, MD

Setting the stage. The incidence of serious skin infections—those requiring hospitalization—went up by almost 1/3 between 2000–2004. Although MRSA is the primary factor, significant reemerging diseases—including atypical mycobacteria—and zoonotic diseases contribute as domestic vectors expand into new areas, global travel enables local microbes to cross oceans, and immunosuppressive drugs eliminate immune defenses.

Tick-borne diseases. The deer tick *Ixodes scapularis* carries Lyme disease, the malaria-like babesia, and human anaplasmosis (previously called human granulocytic ehrlichiosis). The incidence of anaplasmosis—and co-infection with these other organisms—is rising as the exploding deer population moves into new areas and outdoor recreation increases. Older individuals risk critical illness with anaplasmosis. Further, babesia—unlike all of the other tick-borne organisms—does not respond to tetracycline. Immediately look for babesia co-infection because the lab findings normalize after roughly one week. Confirm by blood smears and treat with clindamycin.

The tiny lone star tick (*Amblyomma americanum*) has expanded its range to the mid-Atlantic states and midwest. They carry RMSF, tularemia, human monocytic ehrlichiosis, *Ehrlichia ewingii* ehrlichiosis, and southern tick-associated rash illness. They attach in very large numbers, and are often unrecognizable as ticks until engorged.

Atypical mycobacteria. With chlorinated water, vulnerable organisms are giving way to chlorine-resistant microbes. This includes *Mycobacterium abscessus*, prevalent in foot-soak tubs in pedicure salons. It produces an abscess, so drainage is critical. Immediately look for babesia co-infection because the lab findings normalize after roughly one week. Confirm by blood smears and treat with clindamycin.

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Anaplasmosis

- Tick-borne *Ehrlichia* and *Anaplasma* are intracellular bacteria that infect wild and domestic mammals and man
- Human granulocytotropic anaplasmosis (HGA):
  - Acute, nonspecific febrile illness >3000 cases
  - Immune compromised may develop influenza-like illness with high fever, myalgias, headache
  - 2–4 fold increase in LFTs

(Continued on page 12)
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Hemangioma Research Gains Momentum With DF Support

The Foundation provides a variety of research funding opportunities for supporting progress and talented new teachers and investigators in all areas of dermatology. Ilona J. Frieden, MD, has seen how a modest investment by the DF can give back many times over. The Foundation’s early-career research support has helped four clinical pediatric dermatologists significantly increase the understanding of infantile hemangioma (IH), a common but poorly understood pediatric skin disease, as well as launch their academic careers, and help secure federal research funding.

Dr. Frieden, Director of Pediatric Dermatology at UC, San Francisco and at UCSF Children’s Hospital, founded the collaborative Hemangioma Investigators Group (HIG) in 1999 with a small number of like-minded pediatric dermatologists. Their mission is to advance knowledge about infantile hemangioma and improve outcomes for affected children through research, education, and advocacy. HIG’s research program was launched through the efforts of these four young pediatric dermatologists enabled by their research support from the DF.

To date they have generated 17 publications, secured research funds, facilitated an upcoming therapeutic trial, and made a significant impact on clinical care.

“Anita was eager to study IH,” Dr. Frieden recalls, “and she ably used her DF award to get the HIG research program off the ground.”

Dr. Haggstrom received one of the first Pediatric Dermatology Fellowships co-sponsored by the Society of Pediatric Dermatology and the DF in 2002, when she began her fellowship training with Dr. Frieden. The DF award freed 50% of her time for her pioneering Prospective Study of Hemangiomas of Infancy, allowing her to organize and evaluate the vast pool of data HIG had gathered on a cohort of more than 1,000 patients. Dr. Haggstrom’s study illuminated IH demographics and risk factors, generated eight important articles, initiated further groundbreaking research, and was key to garnering significant NIH funding.

Now Assistant Professor of Dermatology and Pediatrics at the University of Indiana, Dr. Haggstrom is extremely grateful for her DF support. “It strengthened my commitment to academic medicine.”

Dr. Chamlin and Dr. Haggstrom recently received an exceedingly competitive NIH Challenge grant for developing a quality-of-life and severity scale for children with IH and their families.

Dr. Chamlin—now Associate Professor of Pediatrics and Dermatology at Northwestern University Feinberg School of Medicine and at Children’s Memorial Hospital—had begun her research career with a three-year career development award in 2002 to develop the
quality-of-life Childhood Atopic Dermatitis Impact Scale (CADIS). “There are few sources of available support for junior faculty with research interest and expertise. In addition, the skills and expertise I acquired with this DF support are now directly applicable in HIG,” Dr. Chamlin states.

Dr. Drolet, HIG member at the Medical College of Wisconsin, recently won FDA funds for the first federally funded phase II clinical trial in IH. Her start was a one-year research grant in 2004 for work focused on Prenatal Risk Factors for Hemangiomas of Infancy. While documenting that hemangioma incidence is increasing each year and establishing low birth weight as the dominant risk factor, she learned not only “a different level of research skills, but a different level of leadership skills, which furthered my career.”

Dr. Drolet is now Medical Director of the multidisciplinary Birthmarks and Vascular Anomalies Center, Children’s Hospital of Wisconsin, and Vice-chair of Dermatology and Chief of Pediatric Dermatology at the Medical College.

HIG member Dr. Siegel, a current career development award recipient, will help clarify developmental vascular biology and hemangioma risk factors via her study Genetic Analysis of PHACES. PHACES is the developmental neurocutaneous condition that includes IH. “There is no question that DF funding enabled me to remain in academic medicine,” Dr. Siegel says. “Before DF funding, there was little money for research in pediatric dermatology—and no money for IH research,” Dr. Frieden says. “I am profoundly grateful to the Dermatology Foundation, and I feel very strongly about the value of the DF’s help.”

DF Partnerships Strengthen Specialty

The Foundation is thankful to the following national organizations for their generosity. Their contributions last year translated to 2010 research funding that will foster the development of tomorrow’s clinical scholars and scientific leaders in dermatology. Each has elected to partner with the DF to expand opportunities for research support. The Board of Trustees is grateful for the confidence they have placed in the DF’s ability to identify the most talented, promising new physician-scientists and investigators to help carry the specialty forward.

- American Academy of Dermatology—$55,000
- Women’s Dermatologic Society—$55,000
- Society for Pediatric Dermatology—$22,500
- American Society of Dermatopathology—$10,000
- Dystrophic Epidermolysis Bullosa Research Association of America—$10,000
It is not associated with HIV but presents an HIV-like picture, including reactive skin conditions and the classic early AIDS opportunistic pathogens.

Resurging/emerging infection. Our body responds to mycobacteria by TNF-induced IFN-γ release, which is potently suppressed by TNF agents—especially adalimumab and infliximab (much less with etanercept). We are seeing a resurgence of TB and the emergence of Legionella in these patients. Some negative pre-treatment PPDs are due to anergy. Starting INH with a positive PPD does not always prevent TB emergence. If signs and symptoms suggest TB, Elston advises a gamma-release assay and chest x-ray, and a look at the gut because bovine TB is also emerging. Syphilis is resurging—especially in men who have oral sex with men—and with an altered presentation. Lymphogranuloma venereum has also returned in this population.

Air travel. Recirculated cabin air has been implicated in the spread of measles and TB. “We will be seeing worldwide occurrence of diseases that in the past were geographically contained.”

Biologics: Emerging Drugs, Opportunities, and Emerging Side Effects

Erin E. Boh, MD, PhD

Background. When psoriasis was recognized as a dermal rather than an epidermal process, TNF was identified as a critical downstream factor. The first generation of anti-TNF drugs targeted its action, not its production, and monoclonal antibodies were humanized, not fully human. Before discussing the newest approved biologics targeting this pathway, Boh sketched out the pathway itself, especially the pivotal upstream cytokines IL-12 and IL-23 in orchestrating the psoriatic pathology. They each contain a common molecular subunit (called p40), and the scope of activity for each one includes inducing TNF-α synthesis.

Ustekinumab. Ustekinumab (Stelara™)—a monoclonal antibody to p40—inhibits IL-12 and IL-23. It was approved in June 2009 for adult psoriasis patients who are candidates for systemic therapy, and is in trials for psoriatic arthritis (PA). The first 2 doses are 4 weeks apart, with maintenance dosing every 12 weeks. The two critical trials compared 45-mg and 90-mg doses. Placebo subjects eventually crossed over to active drug. Follow-up was 40 weeks initially, then 52 weeks. Improvement in PASI scores was very rapid and nicely maintained. Patients >100 kg required the 90-mg dose for adequate benefit; patients <90–100 kg did well with the 45-mg dose. Reactions resemble those with anti-TNF agents—upper respiratory-like symptoms, nasopharyngitis, headaches, etc. A very few serious adverse reactions occurred. These safety data are not long term, making firm conclusions premature.

Golimumab. Golimumab (Simponi™) was approved in April 2009 for rheumatoid arthritis, PA, and ankylosing spondylitis, and is in psoriasis trials. It is a fully human anti-TNF monoclonal antibody to minimize antibody formation, and is intended for patients not responsive to existing anti-TNF agents. Dosing is monthly. A study comparing 50-mg and 100-mg doses found “nice improvement” in the ACR at 24 weeks. Close to 60% of the psoriasis subgroup showed a 75% improvement in PASI 75; ~35% showed respectable improvement in PASI 90. Efficacy at 52 weeks was comparable. And golimumab may actually reverse joint destruction in PA. The most common side effects were upper respiratory infections and pharyngitis, both very low compared to placebo. Several serious adverse reactions appeared in the longer study, including 1 death from small-cell lung cancer and 1 severe opportunistic infection, but duration is too short for gauging such events accurately.

Conclusion. The biologics require vigilant monitoring and post-marketing reporting of any adverse effects. Boh reviewed the updated FDA-issued box warning this past November for all anti-TNF agents.

Important Update to the Prescribing Information for Anti-TNFs:
Boxed Warning November 2009

- Malignancy
  - Lymphoma and other malignancies, some fatal, have been reported in children and adolescent patients treated with TNF blockers.
- Adverse reactions
  - Post-marketing Experience: Adalimumab
  - Skin reactions: ...new or worsening psoriasis (all subtypes including pustular and palmoplantar)
- Warnings: serious infections
  - Active tuberculosis (TB), including reactivation of latent TB
  - Invasive fungal infections
  - Bacterial, viral, and other infections due to opportunistic pathogens
  - The risks and benefits of treatment should be carefully considered prior to initiating therapy in patients with chronic or recurrent infection
  - Use of TNF blockers may increase the risk of reactivation of hepatitis B virus (HBV) in patients who are chronic carriers. Some cases have been fatal.
SOLODYN INNOVATION

SOLODYN is indicated to treat only inflammatory lesions of non-nodular moderate to severe acne vulgaris in patients 12 years of age and older. SOLODYN did not demonstrate any effect on non-inflammatory lesions. Safety of SOLODYN has not been established beyond 12 weeks of use. This formulation of minocycline has not been evaluated in the treatment of infections. To reduce the development of drug-resistant bacteria as well as to maintain the effectiveness of other antibacterial drugs, SOLODYN should be used only as indicated.

SOLODYN Tablets Important Safety Information

- The most commonly reported side effects were headache, fatigue, dizziness, and pruritus.
- Minocycline, like other tetracyclines, can cause fetal harm when administered to a pregnant woman.
- Tetracycline drugs should not be used during tooth development (last half of pregnancy and up to 8 years of age) as they may cause permanent discoloration of teeth.
- Pseudomembranous colitis has been reported with nearly all antibacterial agents and may range from mild to life-threatening; therefore, it is important to consider this diagnosis in patients who present with diarrhea subsequent to the administration of antibacterial agents.
- Central nervous system side effects, including light-headedness, dizziness, or vertigo, have been reported with minocycline therapy.
- In rare cases, photosensitivity has been reported.
- Should not be used during pregnancy nor by individuals of either gender who are attempting to conceive a child; concurrent use of tetracyclines with oral contraceptives may render oral contraceptives less effective.
- This drug is contraindicated in persons who have shown hypersensitivity to any of the tetracyclines.

See reverse side for brief summary of Full Prescribing Information.
Biologic Agents for the Management of Recalcitrant Atopic Dermatitis

Seth J. Orlow, MD, PhD

Introduction. Orlow discussed the off-label use of three biologics for patients with atopic dermatitis (AD) who cannot be controlled with the measures and medications normally used, or who cannot remain controlled on a reasonable maintenance regimen. Consider these biologics when contemplating the use of systemic immunomodulatory agents.

Interferon-γ. This recombinant protein—marketed as Actimmune®—is approved for treating chronic granulomatous disease and osteopetrosis. It inhibits the development of TH2 cells, which play a critical role in acute AD, in elevated IgE levels, etc. Multiple studies—open label and double-blind—in patients with severe AD show utility in reducing global scores, itch, and associated allergic symptoms. Some patients do well with 2–3 times/week; many need daily treatment. Flu-like symptoms are the primary side effect; allergic symptoms. Some patients do well with 2–3 times/week; many need daily treatment. Flu-like symptoms are the primary side effect, and diminish over time. Responsive patients are—counterintuitively—those with pretreatment IgE <1,500 and/or eosinophil levels <9%.

Omalizumab. This humanized monoclonal antibody against IgE (marketed as Xolair®) impairs the interactions of IgE with its receptors. It was approved a few years ago for resistant asthma in patients 12 or older with IgE levels 30–700. Although AD patients with much higher IgE levels can respond to omalizumab, improvement is most dramatic with low IgE regardless of severity. Dosing is every 2–4 weeks. In one study, by month 3 the majority of patients were classified as “mild,” with many able to stop other medications—including oral corticosteroids. Improvements can continue for the first 6–9 months of treatment. Side effects include asthmatic attack or dyspnea, anaphylaxis, and possibly a long-term increase in cardiovascular events.

Rituximab. This chimeric human–mouse monoclonal antibody against a surface antigen on B cells is approved for treating certain B-cell lymphomas and leukemias, and used off-label in lupus. In the initial study of 6 patients with very severe AD (EASI score of 30), the rapid and dramatic effects after 2 infusions 2 weeks apart were “tantalizing.” By week 8, EASI scores were 8.4, with markedly reduced B cells in skin and circulation—all maintained at 24 weeks. But rituximab also has “the most potential for severe side effects”: infusion reactions, possible severe mucocutaneous reactions, infections, and multifocal leukoencephalopathy.

Conclusion. Orlow concluded by stressing AD’s chronic nature and profound impact, and the fundamental needs for long-term planning, active patient/family education, and the ability to think outside the box for those with recalcitrant disease.
The Dermatology Foundation is grateful to the following corporations for supporting its mission to develop and retain tomorrow’s leaders in the specialty and advance patient care.

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fibers are lost through neurodegeneration and the remaining fibers become hypersensitive to compensate. He has identified this same phenomenon in psoriatic itch and nummular eczema. Yosipovitch suggested that the as-yet unidentified itch receptor in skin involves the keratinocyte. He discussed its neurosensory role, pointing out that it has muscarinic and nicotinic receptors for acetylcholine, receptors for nerve growth factors—which is very important in both sensitization and pain, receptors for opiates and for prostaglandins (E2 and histamine), and TRP (transient receptor potential) ion channel receptors (very important in itch and sensation).

Neurophysiology of itch. Antihistamines do not work for most of our patients with chronic itch, so it must be transmitted by other fibers. One search effort has led to several cysteine proteases that induce itch, and finding a cysteine protease receptor that is highly overexpressed in the epidermis, dermal-epidermal junction, and nerve fibers of patients with AD. This pathway represents a novel therapeutic target.

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Career Development Awards (CDAs)
Seven categories of CDAs are available for investigators in the early stages of their careers, and are intended to help recipients transition from fellowship to established investigator. These highly competitive awards provide an annual stipend of $55,000 for up to three years of support:

- Physician Scientist CDA
- Clinical CDA in Dermatologic Surgery
- Clinical CDA in Health Care Policy
- Medical Dermatology CDA
- Science of Human Appearance CDA
- Women’s Health CDA
- Research CDA

Fellowships
The DF offers two types of one-year fellowships for new dermatologists—the Dermatologist Investigator Research Fellowship and the Fellowship in Pediatric Dermatology. They provide salary stipends of $30,000 and $45,000, respectively.

Research Grants
These one-year grants offer $20,000 in seed money for research projects in a variety of concentrations: patient-directed investigation (medical and surgical studies that can directly benefit patients), basic dermatologic research, and dermatopathology.

Program Development Grant
This unique grant provides $10,000 to further the scientific infrastructure of an existing dermatology division or department.

Visit www.dermatologyfoundation.org/rap for detailed award information, application instructions, and downloadable forms. Funding begins July 1, 2011. Questions? Contact the DF staff at 847.328.2256 or dfrap@dermatologyfoundation.org.

C Nerve Fibers Extend to the Epidermis and are in Close Proximity to the Stratum Corneum
The Itchy Elderly Patient—A New Approach
Timothy G. Berger, MD

Introduction. Realizing that half of his patients in the referral practice at UCSF were elderly—past age 55–60—white men with chronically itchy skin spurred Berger to explore this large patient subset. He now views their varied morphologies as eruptions of senescence reflecting a common process, which has significantly improved his ability to treat them effectively.

Discovering the big picture. Berger began with a retrospective chart analysis of all patients over age 60 who had presented with an itchy rash between July 2005–June 2007. They represented 45.2%—almost half—of the 330 referrals. In this series, men outnumbered women (59.1% to 40.9%) and the average age was 73 (range 60–94). Although diagnosis and morphology produced a long list, a “Big Five” group represented 75% of the diagnoses: eczematous dermatitis, chronic itch/scratch lesions (PN/LSC), subacute prurigo, Grover’s disease, and neuropathic dermatoses. Most of the Big Five patients had multiple diagnoses over time, typically beginning with Grover’s disease. Asking “what happens with time and age that could cause all of these manifestations?” led him to: an aging immune system (immunosenescence), age-related barrier changes (barrier defect), and neural and spinal degeneration (neuropathic elements). The perceived itch in eruptions of senescence reflects “a delicate interaction of these physiologic age-related changes in the skin, immune system, and neural system.”

Understanding these changes. Immunosenescence—also underlying the difficulty in immunizing elderly patients—reflects the decreasing number of naïve T cells caused by thymic involution. This impairs recognition of new antigens and seriously diminishes both regulatory T cells and TH1 cells, allowing pro-inflammatory TH2 cells to proliferate. Skin barrier problems appear around age 55 as barrier repair begins to slow, worsening around age 70 when synthesis of the barrier’s lipid content ceases. Decreasing acidification impairs proper differentiation and permeability homeostasis. (Women’s more neutral pH means fewer eruptions of senescence; African-Americans are normally 1 point lower, explaining their rare appearance here.) Spinal degeneration due to arthritic changes often accounts for neuropathic cases. Some lesions reflect primarily one component—e.g., Grover’s disease and predominantly urticarial lesions are primarily immunosenescence disorders; eczematous eruptions and increased susceptibility to allergic contact dermatitis primarily reflect barrier defects. Others are multi-determined.

Patient management. Berger biopsies all existing morphologies, patch tests primarily eczematous patients, phototests photosensitive patients, and requests a spinal evaluation if he suspects brachioradial pruritus or other neuropathic alterations. He detailed the soak and smear therapeutic ladder for the primarily eczematous patient and a possible low-dose or very-low-dose immunosuppressive for patients with primarily Grover’s disease or urticarial papules.

You are Invited...
Everyone is welcome to become a part of dermatology’s progress, and the DF makes it easy. Visit dermatologyfoundation.org for more information regarding its ongoing mission to develop and retain tomorrow’s teachers and researchers. The need for research funding is great—and every contribution makes a difference. To put your membership in motion, call the DF office at 847.328.2256.
CLINICAL SYMPOSIA 2010 FACULTY Proceedings—Part II

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*We apologize for the inadvertent omission of LEO Pharma’s name in the Spring issue.

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John T. Seykora, MD, PhD: Aiming to Revolutionize SCC Prevention and Care

Noted dermatopathologist Dr. Seykora is a multidimensional physician at the University of Pennsylvania. He directly credits his 2001 Career Development Award (CDA) from the Dermatology Foundation for his contributions to the specialty.

Dr. Seykora is an Associate Professor with full teaching responsibilities and pursues research that takes him into areas with exceptionally promising clinical benefits: cell proliferation and differentiation, skin carcinogenesis, wound healing, and photoimmunology.

He is particularly excited about his research related to squamous cell carcinoma (SCC), which holds the potential for revolutionizing patient care. Dr. Seykora has discovered and characterized key regulators of keratinocyte growth, which represent potential new therapeutic targets. Recently, he has developed a groundbreaking mouse model for SCC that is the first to actually reproduce the spectrum of human skin cancer in a short time frame. Because this engineered mouse mimics human precursor lesions and tumors, it will enable breakthrough progress in understanding how skin cancer occurs and in the testing of candidate molecules effective for topical treatment.

Dr. Seykora’s first important steps in his research relating to SCC occurred during his fellowship at UPenn when he identified a novel gene that appeared to influence tumor development. However, the move to a junior faculty position meant he needed funding to support his research time and produce the data to become competitive for NIH funds. His only option was a Dermatology Foundation CDA, specifically designed to address the scarcity of transitional research support.

Dr. Seykora was awarded a CDA, and within two years began to receive steady federal funding.

“My DF CDA provided crucial support. It allowed me to continue my work, secure independent funding, and has ultimately led to a better understanding of skin cancer,” Dr. Seykora says. “Now, I am happy to give back to the DF—as a volunteer and a member.”