As another year winds down, it’s time to think about our accomplishments and plan for the future. Penn Dermatology has had a great year as you will see in the pages of this newsletter. Our dermatologists saw a record number of patients with every conceivable diagnosis. Our dermatopathologists read over 84,000 cases and made some of the most difficult diagnoses in our region (over 21,000 pigmented lesions!). Our research investigators published high impact research. Our trainees worked tirelessly, presented their work at national meetings, and are well on their way to becoming outstanding dermatologists, dermatopathologists, Mohs surgeons, educators and investigators. Overall, our department continues to have a major impact on patient care, education and research.

We are definitely in a growth mode, especially on the clinical side. We started new practices at Valley Forge, PA and Woodbury, NJ. We currently have 36 full-time faculty, 74 affiliated faculty and 185 staff members. We hired three new faculty members this past July and are actively recruiting as our space is scheduled to increase at HUP/PCAM in January of 2015.

We moved into the new research space on the tenth floor of the BRBII/III building this past July. Having the basic science research faculty adjacent to each other increased interactions and invigorated our research. The lithographs are in storage but the portraits are all hanging. Please pay us a visit if you are in the neighborhood.

The size of our residency class has remained stable. Though in theory we could train a much larger group of residents based on our patient volumes and size of our faculty, we are limited by the number of slots supported by funding for Graduate Medical Education (GME). Though the number of medical schools in the US has increased, the number of residency slots has not, and even existing GME funding remains precarious.

In the near future, we face many challenges. Congressional dysfunction has led to sequestration, resulting in unprecedented cuts to the NIH budget. Only 1 out of every 10 to 20 grant applications submitted to the NIH receives funding now. Existing NIH grants have been cut 10% per year. These short-sighted cuts jeopardize the future of scientific innovation and discovery in the US at a time when other countries are increasing their biomedical research budgets. For instance, the Japanese government recently approved an additional $3.2 billion to foster research in regenerative medicine. We need to do the same. Otherwise, cutting edge research that will lead to better treatments for disease will never reach fruition.

For these reasons, especially at this time, support from our alumnae and through philanthropy becomes exceedingly important. Support comes in many different forms. Keep in mind that as funds for research and education falter, we depend even more on our clinical activities to support our tripartite mission. Your utilization of our dermatopathology and other clinical services makes an enormous difference in our ability to support research and education. Of course, contributing to research and education through donations is the most direct means for supporting these missions.

Please contact me to discuss ways of giving to the department.

Enjoy the Holidays and Have a Happy New Year!

Sincerely,

George Cotsarelis, M.D.
It might be difficult to believe that time goes by so fast, but our Penn SDRC is approaching the end of its five years of funding support. It was established in July 2009 and will terminate in June 2014, unless refunded, through a competitive renewal, by the National Institute of Arthritis, Musculoskeletal and Skin Diseases. We have submitted a massive (467 pages to be exact) grant that details our success over the last five years and our vision for a new SDRC.

The success of our current SDRC can be measured in many ways. One, of which we are most proud, is the development of new physician-scientists in our field. Few programs have been successful at developing physician-scientists in dermatology. Our Department is proud of our focus on developing new clinician-scientists in our field and we designed our current SDRC to help this effort through formal mentoring, a special resident-scientist selection and recruitment program, and research grants and travel awards to support our trainees. This program has been very successful with six of the seven mentored trainees over the last four years pursuing careers as physician-scientists and one pursuing an academic-clinical career. Another aspect of our current SDRC has been the awarding of Pilot and Feasibility (P&F) Grants. We have awarded a total of 17 grants to junior scientists in dermatology, trainees in dermatology, and scientists outside dermatology who wanted to pursue skin-related projects. These grants had excellent success in seeding additional funding through NIH and foundations, as well as in publications. Finally, the SDRC established scientific cores that facilitated research in our field. These were widely utilized by investigators inside and outside dermatology. One phenomenal example is that the Skin Histology and Characterization Core, directed by Dr. John Seykora, processed over 56,000 histologic slides for skin investigators over 3.5 years! Finally, the SDRC has sponsored superb lecturers and research conferences. These successes have helped give dermatology a highly visible presence and great reputation throughout Penn Medicine.

What about the new version of the SDRC for which we have applied for funding? First, we have kept the parts of the original SDRC which were most successful. These include three scientific research cores: the Skin Histology and Characterization Core (Director, John T. Seykora, MD, PhD), the Tissue Keratinocyte and Procurement Core (Director, Aimee Payne, MD, PhD), and the Stem Cell and Xenograft Core (Director, George Cotsarelis, MD) which has been expanded (see below). We have also kept the highly successful mentoring and career development program for physician-scientist trainees.

However, we have also made some fundamental changes in our newly proposed SDRC. We have expanded Dr. Cotsarelis’ core, newly named the Keratinocyte Stem Cell, Xenograft and Tissue Engineering Core, to include tissue engineering that includes three dimensional cultures of skin with various genetic manipulations. Todd Ridky, MD, PhD, who is an expert in this field, will be a co-Director of this core. Based on the feedback and requests from our researchers, we have added a new scientific core called the High Throughput Sequence Analysis and Interpretation Core (Director: Elizabeth Grice, PhD) which will provide guidance in planning and interpreting experiments that involve deep sequencing technology, which is becoming increasingly important in many aspects of molecular and cell biology. Such technology generates massive amounts of data that require special expertise to handle and analyze, which this core will provide.

In the new SDRC, we proposed focusing the P&F Grant program, under the Directorship of Dr. Sarah Millar, PhD, to only include “Physician-Scientists in Training Mini-Grants”, the most successful part of our previous program. These type of grants will help support our trainee scientists at the beginning of their research careers, give them an opportunity to learn how to prepare effective grant applications, and help them obtain experience and data for additional grant support.

Finally, in the new SDRC we proposed a program to encourage diversity in dermatologic research. This program will encourage women and under-represented minorities in their careers and bring focus to unconscious gender and other biases in recruitment and promotion.

We are excited about the potential of our newly proposed SDRC and hope to report in the next newsletter that we have been funded to establish it. For the moment, if you are reading this, please keep your fingers crossed.
Why does a melanocyte look and function so differently from a fibroblast, or a hepatocyte for that matter, when they all have essentially the same DNA sequence? Similarly, how do we humans form into such a complex and diverse array of tissues and cell types from a genetically identical small cluster of cells in the womb? The study of epigenetics provides answers to these questions. Very broadly, epigenetics can be defined as the study of the regulation of genes, their expression, and how that translates into particular phenotypes, independent of any change to the underlying DNA sequence. Human disease is increasingly being linked to the turning on or turning off genes due to epigenetic changes that control the regulation of our genomes. For example, altered epigenetics is an important driver of cancer, therefore age-associated epigenetic changes in apparently normal tissues might help to explain why aging is the single biggest risk factor for cancer and why the organization and packing of DNA into chromatin is the most influential factor on regional mutation rate in cancers. Along these lines, several cardinal features of cancer and aging, including elevated inflammation and dysregulated gene transcription, have been linked to epigenetic mechanisms.

Similarly, senescence, the process by which cells cease to proliferate but remain alive and metabolically active, is thought to contribute to both aging and cancer. For instance, the number of senescent cells increases with age in numerous human tissues, including the skin. And remarkably, the clearance of senescent cells dramatically prevents age-related tissue dysfunction in mouse models. However, beyond a deleterious role, senescence can be protective. In the most well-known in vivo example of human senescence, melanocytic nevi are benign lesions harboring activated oncogenes such as BRAFV600E. These oncogenes first act mitogenically to stimulate proliferation and nevus formation, but the cell then undergoes oncogene-induced senescence, a state of proliferative arrest from which it may ultimately escape and transform into melanoma. There is evidence that epigenetic reprogramming may be playing a role in both the maintenance and loss of this senescent state. This Mini-grant aims to define the major epigenomic chromatin-based mechanisms of both the onset of and the escape from senescence in the skin.

Specifically, we hypothesize that dramatic epigenetic reorganization occurs during senescence involving the general opening up of the normally compact genome within cells due to functional disruptions in chromatin organization across the genome. Our previous data has demonstrated unequivocally that dramatic changes in the chromatin landscape occur in both model organisms with aging and in human cells with senescence (Figure 1). However, these changes have not been explored in the human skin. Better understanding of these phenomena are likely to yield insights into neoplastic changes that occur in the skin with aging and oncogenesis. Importantly, as epigenetic changes are generally considered to be relatively reversible, unlike genetic mutations in DNA, an understanding of these processes will potentially provide opportunities for intervention and modulation.

To test our hypothesis, we are employing several powerful techniques and approaches, including chromatin-immunoprecipitation (ChIP) followed by genome-wide parallel sequencing (ChIP-seq) as well as genome-wide transcriptome sequencing (RNA-seq) to correlate these global epigenetic alterations with gene expression changes. This Mini-grant supports these studies and closely aligns with the overall goal of my work, which is to understand the role of epigenetic mechanisms in skin function and disease. We anticipate that this work will ultimately have major implications for understanding both degenerative and neoplastic changes that occur in the skin during aging and carcinogenesis and provide evidence for ways in which these changes may be prevented or modified. Furthermore, given its incredible accessibility, the skin is considered a model system for the study of human cancer and aging in general, and thus our findings here may in fact have implications far beyond the skin only.

![Figure 1](image-url)
Fibroblast growth factors bind to Fibroblast Growth Factor Receptor 3 (FGFR3) on the surface of cells. Signaling through this receptor influences mitogenesis and differentiation. Mutations of FGFR3 are implicated as primary oncogenes in subsets of bladder, cervix, and head and neck carcinoma as well as multiple myeloma. The exact role of FGFR3 is still being studied in these carcinomas and myelomas, but in response to initial findings on the role of the protein in cancer development, a number of pharmaceutical companies have begun to develop FGFR3 inhibitors as potential anti-cancer drugs.

Curiously, in the epidermis, mutations of FGFR3 have only been associated with benign tumors, such as seborrheic keratoses (SK) and congenital epidermal nevi. Despite the strong association between FGFR3 mutations and these benign skin lesions, no functional data establishes them as sufficient on their own to cause these tumors, nor is it clear why the same mutation would lead to cancer in some epithelial tissues, while causing only benign hyperproliferation in skin. Establishing the physiologic roles of FGFR3 in vivo is made more complex by additional research indicating FGFR3 activation may actually serve as a tumor suppressor by normally blocking the activity of the Ras oncogene that commonly drives squamous cell carcinoma (SCC).

Elizabeth Kennedy, a UPenn Cancer Biology graduate student in our laboratory, has spearheaded recent efforts to define the role of FGFR3 in the epidermis. To undertake this endeavor, our lab leveraged the human xenograft approach, modeling epidermal diseases in engineered human skin tissue grafts. This type of modeling creates a three-dimensional environment, recapitulating hallmark features of the living human organism and associated tumor we hope to study. Skin is the only solid human organ that can be rebuilt in the lab from its multiple component cells and subsequently used in organ transplants where the tissue functions properly in its normal location in a living animal. The ability to isolate pure populations of living keratinocytes, fibroblasts, melanocytes, and endothelial cells from a single piece of human skin allows us the opportunity to genetically engineer the cells to express or silence one or more genes of interest. This expression is controlled by gene promoters that allow the gene to be turned “on and off”. The genetic manipulations are structured with the flexibility to express genes in the experimental tissues for a little as one day or up to 2 years (typical life-span of a laboratory mouse used as a host for the human skin grafts). Examples of normal human unpigmented and pigmented engineered skin transplants, along with model SCC, and melanoma tumors expressing oncogenes commonly seen in spontaneous human tumors are shown in Fig. 1.

Using this three-dimensional modeling approach, our lab set out to determine the role of FGFR3 in the epidermis. Specifically we sought to understand: 1) the ability of FGFR3 mutations to serve as the sole drivers of SK or SCC, 2) the ability of FGFR3 mutations to work in combination with other mutations to drive SK or SCC, 3) whether FGFR3 activation actually blocks the effects of Ras in human skin, and 4) whether FGFR3 is necessary for either normal skin function or Ras-driven epidermal SCC. To extend these findings to patients, we 5) examined a human tumor in which SCC developed in continuity with a SK, testing the hypothesis that one of the tumors evolved from the other and exploring the role of FGFR3 activation in each.

FGFR3 mutations in human skin are not sufficient to drive an SK or SCC

Our first experiments set out to explore if mutations in FGFR3 would lead to SK or SCC in associated tissue. Cysteine substitutions at FGFR3 positions 248 and 249 lead to increased FGFR3 signaling. In one experiment, we expressed these mutant FGFR3 proteins in otherwise normal human keratinocytes. Both of the mutant FGFR 248C and 249C proteins led to activation of downstream MAPK kinase activity – a hallmark of tumor tissue from skin, as well as tumors originating in most other organs. However, while the level of MAPK activation was more than that seen in control cells, it was less than that seen in cells expressing an active Ras mutation commonly seen in SCC or epidermal nevi.

Skin grafts expressing these active FGFR3 mutants were subsequently transplanted onto host mice, and histologically analyzed at several time points. Again, epidermis expressing FGFR3 248C or 249C was mildly hyperproliferative, but lacked many of the hallmark features of typical seborrheic keratosis. This suggests that other factors must cooperate with FGFR3 mutations in people to form the SKs seen clinically.
This finding is supported by prior mouse models in which active FGFR3 was targeted to epidermis using the K5 promoter. In this model, mice are born expressing active FGFR3 in all of their skin. However, their skin is normal at birth, and it is only over time that these animals eventually develop small hyperproliferative epidermal papules in a few specific areas subject to mechanical trauma including nose and eyelids, again suggesting FGFR3 activation alone is not sufficient to support SK formation.

**FGFR3 mutations may work in combination with other mutations to drive SK**

While the FGFR3 mutants are clearly not sufficient to cause SK or epidermal SCC on their own, we thought that maybe they could cooperate with other common SCC-associated mutations to drive skin tumor formation. Active FGFR3 was expressed in epidermis along with active CDK4, and dominant negative p53. These two later elements are common in human cancers and inhibit anti-proliferative safeguards that normally prevent tumor growth. While there was mild hyperproliferation in the grafts, the FGFR3 mutants did not develop SCC, even in the permissive tissue environment created by CDK4 and dnp53. Therefore, FGFR3 is likely not a primary oncogene in skin, but may cooperate with additional activating mutations in other (potentially weak) oncogenes to support tumor formation.

**FGFR3 is not a tumor suppressor in human skin**

We remained intrigued by the possibility suggested in prior studies that FGFR3 would inhibit Ras-driven tumors. We therefore tested this directly by establishing human skin transplants expressing active Ras and CDK4 with and without active FGFR3. In both grafts, FGFR3 did not affect tumor speed or size of tumor growth, degree of invasiveness, or differentiation status. Thus, through two complementary lines of evidence, FGFR3 is not a tumor suppressor — at least in epidermis.

**FGFR3 is not necessary for either normal skin function or Ras-driven epidermal SCC**

While FGFR3 activity was not sufficient to drive or inhibit epidermal tumorigenesis in our system, we conducted a series of experiments to determine whether FGFR3 was necessary for either normal skin or SCC. FGFR3 expression was suppressed in keratinocytes using RNAi approaches. These cells were then used to establish skin with and without the CDK4/ Ras oncogene pair. Tissues with FGFR3 suppression were not noticeably different than controls. This is consistent with the FGFR3 knockout mouse which has bone abnormalities, but grossly normal skin. This result further indicates that despite their potential utility in myeloma, FGFR3 small molecule inhibitors, and blocking antibodies, are not likely to be useful therapeutics for Ras-driven epidermal cancers.

**FGFR3 activation is insufficient to drive SCC**

With the help of Dr. John Seykora (UPenn Dermatopathology), we were able to study a histologically classic SCC that developed in continuity with a pre-existing SK. This is an occasionally-seen clinical scenario that provided us with a great opportunity to help clarify the effects of FGFR3 activation in epidermis. Sections were cut from the standard block of formalin-fixed, paraffin-embedded tissue. Using the laser microscope, the regions containing the histologically normal tips, classic SK, and SCC were all separated from each other. The RNA was isolated from each region and sequencing was performed on the FGFR3 gene. As expected, a FGFR3 248C mutation was seen in the SK. Interestingly, the same mutation was not found in the adjacent epidermis or SCC. This indicates that, despite the histological appearance, the SCC arose independently from the SK. We also identified the common dominant-negative TP53 R248W mutation present in the SCC, but not in the adjacent epidermis or SK, further supporting our finding that the tumors arose independently. This isolated clinical case suggests that perhaps those occasionally-encountered cases in which an SK appears to progress into an SCC are in fact collision lesions of two very common genetically distinct tumors of sun damaged, aged skin. To date, FGFR3 mutations have not been observed in SCC. This is perhaps a bit unexpected given that SK lesions are hyperproliferative with elevated MAPK activity, which would seem to make them more prone to malignant transformation. While we have demonstrated that the FGFR3 mutation itself does not prevent progression to SCC, it is possible that other mechanisms work to minimize the development of additional mutations. It is likely that that the increased tissue thickness, hyperpigmentation and hyperkeratosis associated with SK lesions prevent additional UV-related mutations in the proliferative basilar keratinocytes. Alternatively, FGFR3-mediated MAPK activation may alter the KC response to UV radiation, promoting either apoptosis or DNA damage repair pathways making progression to SCC an unlikely event.

**Lessons from FGFR3 modeling and next steps**

The medical relevance of basic science cancer studies is maximized by conducting experiments in three-dimensional environments that recapitulate hallmark features of the living human organ and the associated tumor. Our work on FGFR3 provided evidence that FGFR3 inhibitors are unlikely to be useful to stop skin cancers. Though the mutations are, in part, involved in the development of SK, they are not sufficient alone to cause SK. Moreover, we found that FGFR3 is not necessary for either normal skin function or Ras-driven epidermal SCC. Analogous models are not available to test the impact of FGFR3 mutations outside of the epidermis. New models would be needed to better characterize the events that lead the gene to become an oncogene in other tissue.

Skin is the only solid human organ that can be rebuilt in the lab. As demonstrated through our work on FGFR3, this three-dimensional modeling approach helps pave the way for differentiated, unique models that can be activated in countless ways. As leaders in the dermatology field, our department is in a unique position to take advantage of these models in innovative and groundbreaking ways to find answers surrounding the most common to the most complex epidermal issues facing the industry today. Similar three-dimensional experimental platforms are being utilized in multiple ongoing efforts in the lab, with the general theme of defining specific roles for factors governing malignant transformation of keratinocytes to squamous cell carcinoma, and melanocytes to melanoma. Specific to FGFR3 and SK, findings from our work will be leveraged to further refine models of the potential stromal stressors of SK that are related to its development. We are also further exploring why the FGFR3 mutation is only involved with benign tumors in the epidermis as opposed to the carcinomas and multiple myeloma seen in other parts of the human body. These answers would impact not only practitioners in the dermatological field, but across the whole medical field.
A vast diversity of microbes colonize our skin. Research in the Grice laboratory focuses on these microbial populations (the “microbiome”), how they influence skin health and disease, and factors that contribute to their variability. Through a series of unique collaborations with the Penn School of Veterinary Medicine, the Grice laboratory is looking to furry members of our families to examine environment influences on skin microbiota and as models of complex skin disease.

Ana Misic, PhD, a post-doctoral fellow in the laboratory, was recently awarded a Morris Animal Foundation fellowship to examine the shared microbiomes of humans and their furry companions in households with members with active methicillin resistant Staphylococcus aureus (MRSA) infections. The project is a collaborative effort with researchers from Penn Medicine (Ebbing Lautenbach MD MPH MSCE, Irving Nachamkin DrPH MPH), Penn Veterinary Medicine (Dan Morris DVM MPH), and Johns Hopkins University (Meghan Davis DVM MPH PhD).

MRSA, a skin pathogen in pets and people, produces high rates of re-infection even after antibiotic treatment. In companion animals, species such as S. aureus, S. pseudintermedius, and S. schleferi are major pathogens that produce a wide array of virulence factors and result in many disease syndromes. Humans and pets live in the same environments, share skin-to-fur contact, and share microbiota; both pathogens and commensals. The objective of this project is to determine if companion animals share microbiota with humans and if the microbiome has a role in modulating MRSA colonization and infection in households with and without pets. In the Grice laboratory, microbiomes are examined using a culture-independent approach of deep sequencing of the bacterial 16S ribosomal RNA gene. These gene sequences are used to identify and examine bacterial community dynamics and, in this case, microbiota that is shared between pets and their humans.

Preliminary results show that there are fundamental differences between pet and human microbiomes, with animal species being one of the greatest determinants of microbiome composition. For example, cats and dogs harbor much higher relative abundances of Proteobacteria than humans. Proteobacteria are mainly Gram-negative, environmental bacteria. Humans that self-identify as being licked by their pets share a greater degree of microbiota than those that aren’t licked. Members of the same households share more features of their microbiota with each other than with those outside of the household. Index patients with MRSA infection have distinct microbiota compared to other members of the household.

In addition to this unique collaboration, the Grice laboratory recently received funding from Penn Vet’s new Center for Host Microbe Interactions (CHMI) as part of a collaborative effort to characterize the skin microbiota, antibiotic resistance, and the skin barrier function in dogs with spontaneous atopic dermatitis. Charles Bradley VMD, an Instructor at Penn Vet, will spend a year in the Grice lab working on this project. Other investigators involved in the project from Penn Vet include Shelley Rankin PhD, Dan Morris DVM MPH, Christine Cain DVM, and Elizabeth Mauldin DVM.

Figure: Principle coordinate analysis to illustrate differences in the human, cat, and dog microbiomes. Red dots represent cat samples (from nose and mouth), green dots represent dog samples (from nose and mouth), and blue dots represent human samples (from nose, inguinal crease and axilla). Grey spheres with labels indicate the bacterial order-level taxa that are contributing to observed clustering of samples. For example, cats and dogs have more abundant Gram-negative Proteobacteria, including Pseudomonadales, Pasteuralles, and Neisseriales, than humans.
Autoimmune Skin Disease Study Unit
- Victoria Werth, MD

Clinical and Translational studies

The autoimmune skin disease study unit at Penn has completed a number of validation studies for disease severity tools that are now being used in international trials for lupus (CLASI) and dermatomyositis (CDASI). These studies required demonstrating that multiple investigators can obtain similar results when looking at the same patients (inter-rater reliability) and that the same investigator can get a similar result when re-rating a given patient (intra-rater reliability). The disease severity tools were also shown to respond to change in the disease activity by using another “gold standard” that demonstrated improvement in the disease and showing that the specific disease tool also improves. Our recent studies have shown a correlation between specific inflammatory blood biomarkers and disease activity. These validated tools have stimulated interest in development of medications for previously unstudied diseases, with several ongoing international trials in lupus and now dermatomyositis. In addition, Dr. Werth has co-led an international effort to develop disease severity tools for autoimmune blistering diseases. The potential for developing new and effective medications specifically for more rare diseases that have previously required high doses of systemic immunosuppressives is particularly critical in the setting of increasing denial by insurance companies of payment for off-label medications used for autoimmune skin diseases. The disease severity tools are also helpful for performing translational research related to disease mechanism.

Ongoing clinical research, utilizing longitudinal databases for both lupus and dermatomyositis, is demonstrating the impact of these diseases on skin-specific quality of life. When disease activity improves, even in the setting of persistent skin dyspigmentation and scarring, the quality of life improves. The data is also demonstrating that patients frequently require treatment for their skin disease with systemic immunosuppressives. In particular, data we presented at the American College of Rheumatology in October 2013 shows a large number of dermatomyositis patients have ongoing active skin disease despite years of treatment with potent immunosuppressives.

The lupus database has shown that patients who smoke have more severe cutaneous lupus. Previous studies have demonstrated an increased risk of lupus in smokers. One large international study of 500 SLE patients examined the CLASI and found smokers had higher mean CLASI scores, with greater percentages of patients with moderate or severe skin disease relative to nonsmokers.

Ongoing studies are modeling in vitro the mechanism for pro-inflammatory cytokine production by peripheral blood mononuclear cells in response to therapy, to better understand the mechanism and heterogeneity of responses seen clinically. In addition, our recent lab studies have demonstrated the effect of tobacco smoke exposure on the induction of proinflammatory microvesicles, that, when isolated, stimulate pro-inflammatory cytokine production by immune cells. New ongoing studies will further clarify the mechanism for these effects.

Active Clinical Trials

Bullous Pemphigoid Study (Novartis): A randomized, placebo-controlled clinical trial to study the safety and efficacy of an IgE receptor blocker, QGE031, in bullous pemphigoid. QGE031 is a human monoclonal antibody that binds to IgE antibodies. The goal is to reduce mortality in BP and limit the use of high dose systemic steroids for long periods of time.

Basic Criteria for screening include:
- Diagnosis of bullous pemphigoid (BP)
- Active disease is defined as 3% body surface area with any combination of urticarial plaques, erosions or blisters
- Lab results within study limits
- Stable prednisone dose of 10 mg/day
- Age 20-80 years old

Pemphigus Vulgaris Study (Novartis): A randomized, partial-blind, placebo-controlled trial evaluating the efficacy, safety, pharmacokinetics and pharmacodynamics of VAY736 in the treatment of patients with pemphigus vulgaris.

Basic Criteria for screening include:
- Diagnosis of pemphigus vulgaris (PV)
- Mild to moderate active disease
- Lab results within study limits
- Stable dose of corticosteroid +/- azathioprine or myophenolate mofetil.
- Age 20-70 years old
- No use of any B-cell depleting therapy (rituximab) within preceding 12 months

Annual Skin Cancer Screening Event
May 18, 2013

The Annual Free Skin Cancer Screening Event took place this year on Saturday, May 18th in the Department of Dermatology in the Perelman Center for Advanced Medicine. The event allowed for patients to come into the practice and get their skin checked by providers without an appointment or any costs. The event was a huge success, and we were able to provide skin checks for 237 patients in the tri-state area.

The event was followed by a celebratory softball match. Congrats to the Clinic team, captained by Dr. Bill James, who defeated Dermatology Pathology for the Philly Phanatic Trophy. Rematch date TBD.

A big thank you goes out to all volunteers both staff and faculty that donated their time that Saturday morning!
Take your Child to Work Day
Thursday, April 25, 2013

During the spring, the Department of Dermatology hosted 14 of the Dermatology faculty and staff’s children for a fun-filled and educational day. The kids were excited to start the day with a visit to the “PennSTAR” helicopter. They were able to see and learn about some diseased body parts and visit our art gallery in memory of Dr. Johnson before getting lunch with their parents. They ended the day with a visit to the School of Medicine to see and even go inside a hyperbaric chamber. Special thanks to Brandi Eldridge, James Close and Kristina DiStefano for organizing this event and making it a special day for the kids.

Move to Biomedical Research Building II/III

Over the summer, the Department of Dermatology finalized the move to the tenth floor of the Biomedical Research Building II/III. All together, eight faculty members, 30 laboratory staff, ten administrative staff and over 450 pieces of equipment made the move.

The move centralized our research laboratories into one location, creating efficiencies and enhancing collaborations among our basic science research teams. The common space is much better organized, allowing us to eliminate redundancies and share equipment for laser capture microdissection and live cell imaging.

There is state-of-the-art cell culture, tissue processing, microscopy and molecular biology cores. The new space not only encourages more interaction between lab members, we are in closer proximity to powerhouse labs in other departments, which we know will help to further cut across departmental boundaries and drive more innovative research.

As the department grows through its research missions, as well as its clinical and teaching missions, this new space will help facilitate the vastly expanding presence of Penn Dermatology.

IT Updates
Matt Zarkos

Dermpoint is even better!

Last year the department “DermPoint” site was introduced. DermPoint was developed exclusively for staff and faculty to make it easier to share knowledge and to drive a deeper sense of community within the department.

While DermPoint was met with enthusiasm, finding it was difficult. To make the site more accessible, we now have easy-to-remember addresses. Internally on the UP SHA network, the address is “dermpoint” or http://dermpoint. From outside the UP SHA network (even from home), the address is www.pennmedicine.org/dermpoint. When prompted, enter your UP SHA network ID and password.

Over the past year, several groups have taken advantage of DermPoint’s ability to be used as a central repository. Clinical staff are using it to make training information (including videos and how-to guides) easily accessible. New hires view content as part of their orientation. Residents use a special section, specifically created just for them, to help manage schedules and share documents. Frequently used links, contact information, current calendar, and sharing of “how tos” are accessible 24x7. Department phone directories are also online.

On the lighter side, upcoming activities (such as the Staff Appreciation lunches) are advertised as well as photos from past events. HUP and Radnor sections list local restaurants and public transportation information. Easy access to Penn resources, such as online paycheck information, are available in the “Quick Links” section.

We’re always looking for suggestions for improvement, new features, and feedback. Please send your ideas or comments to dermit@uphs.upenn.edu.

Dermatology Public Website
(www.pennmedicine.org/dermatology)

The long awaited update to the Dermatology website is now live and available on the Internet. The new site brings a fresh look to the patient and physician services offered by the department, as well as updated faculty and event information.

The site was developed in conjunction with the Marketing and Web teams leveraging the UP SHA public facing model. It provides an integrated and consistent interface for users. While the standard design is being used for other departments, we are able to highlight Dermatology specialties, faculty, and events to target audiences. Integration with Google Analytics provides the ability to understand what pages are capturing the attention of viewers and allows us to follow their path as they click through the site.

Revisions will be made frequently throughout the year. Faculty and staff are encouraged to offer suggestions and updates to the site by sending an email to dermit@uphs.upenn.edu.
Under the direction of Dr. Joel Gelfand, we are conducting the Vascular Inflammation in Psoriasis (VIP) trial and the VIP Extension trial at PENN Dermatology.

The primary objectives of our research are to determine the efficacy of adalimumab (Humira) and phototherapy compared to placebo on ameliorating vascular inflammation (measured by FDG-PET/CT imaging) and cardiovascular risks (measured by novel cardiometabolic biomarkers) in patients with psoriasis. All study treatments will be provided at no cost and study participants will be compensated for their time and travel up to $1130.

Eligible patients will be randomized to adalimumab, phototherapy or placebo for a treatment period of 12 weeks. After completion of the 12 week VIP Trial, subjects will have the opportunity to continue onto the Vascular Inflammation in Psoriasis Extension (VIP-E) trial in which all patients receive adalimumab (open label) for a period of up to 52 weeks to determine if systemic treatment is associated with durable improvements in cardiometabolic disease.

Below is a schematic representation of the study and sample FDG PET/CT images. For additional study details, please visit: http://clinicaltrials.gov/ct2/show/NCT01553058 and http://clinicaltrials.gov/ct2/show/NCT01866592 or have interested patients call us at 215-662-SKIN (7546).

Penn in Guatemala
Rudolf Roth, MD & Carrie Kovarik, MD

Rudolf Roth, MD, Associate Professor of Clinical Dermatology, is spearheading the dermatology partnership in Guatemala with INDERMA, the leading dermatologic care and training center for Central America. Carrie Kovarik, MD, Assistant Professor of Dermatology, has continued to work with INDERMA in conjunction with Dr. Roth. Garbine Riley, MD, INDERMA faculty, visited Penn in October 2012 to give a Grand Rounds lecture on leprosy and Michelle Scott, MD, a Penn dermatology resident, visited Guatemala in November 2012 to provide technical and programmatic support for INDERMA’s newly established teledermatology services. Dr. Scott also attended the Guatemalan National Congress for Dermatology’s annual conference. Dr. Roth and Dr. Kovarik’s goals for the Penn-INDERMA relationship are to: further develop this partnership, including resident and staff exchanges; develop an electronic network among INDERMA and Penn dermatologists that would be useful in sharing difficult cases and consider a global expansion of this network, which would not be limited to Guatemala or Central America; assist in the expansion of dermatology teaching in Guatemala beyond INDERMA; assist in the development of a dermatology service for Hospitalito Atitlán (currently not available in Santiago Atitlán); continue to explore the further development of teledermatology in Guatemala, including for educational purposes; explore the feasibility of using “DoctoDoc” as a platform for case-based dermatology teaching at USAC/UFM; help foster dermatology research in Guatemala and encourage collaborative research.

Dr. Roth accompanied Drs. Branas, Strom, and Bream on the March Guatemala visit in 2013. Dr. Roth visited INDERMA to discuss collaborative possibilities and plans (see above). Two INDERMA residents visited Penn in August 2013 for two weeks and participated in our clinical rotations. Dr. Roth will return to INDERMA in September 2013, with another faculty member Dr. Jules Lipoff, to give lectures and work collaboratively with INDERMA residents on cases/surgeries. The department also hopes to help develop the research of Monica Paz, MD who recently graduated from the Master of Science in Clinical Epidemiology program at Penn and has returned to Guatemala City. One planned cooperative program will be to go with her to Hospitalito Atitlán for a week in February 2014 to help determine the incidence of atopic dermatitis in the Mayan population.

Vascular Inflammation in Psoriasis (VIP) Trial
Joel Gelfand, MD

Under the direction of Dr. Joel Gelfand, we are conducting the Vascular Inflammation in Psoriasis (VIP) trial and the VIP Extension trial at PENN Dermatology.

The primary objectives of our research are to determine the efficacy of adalimumab (Humira) and phototherapy compared to placebo on ameliorating vascular inflammation (measured by FDG-PET/CT imaging) and cardiovascular risks (measured by novel cardiometabolic biomarkers) in patients with psoriasis. All study treatments will be provided at no cost and study participants will be compensated for their time and travel up to $1130.

Eligible patients will be randomized to adalimumab, phototherapy or placebo for a treatment period of 12 weeks. After completion of the 12 week VIP Trial, subjects will have the opportunity to continue onto the Vascular Inflammation in Psoriasis Extension (VIP-E) trial in which all patients receive adalimumab (open label) for a period of up to 52 weeks to determine if systemic treatment is associated with durable improvements in cardiometabolic disease.

Below is a schematic representation of the study and sample FDG PET/CT images. For additional study details, please visit: http://clinicaltrials.gov/ct2/show/NCT01553058 and http://clinicaltrials.gov/ct2/show/NCT01866592 or have interested patients call us at 215-662-SKIN (7546).

Penn in Guatemala
Rudolf Roth, MD & Carrie Kovarik, MD
CPUP Service Excellence Awards

Penn Medicine Service Excellence Award: Kathy Malatesta, Medical Assistance

Kathy Malatesta received the Penn Medicine Service Excellence Award for the first quarter of 2013. Kathy is a medical assistant that has been with Penn since 2004 and with the Dermatology Department for the past four years. “Kathy is a ray of sunshine in our department. While I have many great things to say concerning the care, compassion and empathy that Kathy shows to our patients every day, the comments and notes concerning the experience that our patients have received say it all.” “The care Kathy extends doesn’t stop at our patients. She takes care of all of her co-workers and physicians. I am very fortunate to have Kathy as one of our team members. While I’m thankful for all of our dedicated staff, Kathy’s warmth and compassion embraces all.”

Team of the Quarter: Radnor Dermatology Clinical Staff

CPUP Service Excellence Rewards and Recognition Program Team of the Quarter Award for 1st Quarter FY 2013 was awarded to Radnor Dermatology Clinical Staff, Vicky Byers MA, Tacara Cain MA, Catherine Casino MA, Emily Nowell MA, Nicole Ewing LPN, Ashley Hall MA, Jane Johnsen RN, Ellen Judy RN, Anita Ledridge MA, Jessica Maitland MA, Kathy Malatesta MA, Jeannine Masalaitis RN, Laurie Molino MA, Sonia Sun (Histotech), Isabel Sy (Histotech) and Marie Terrell LPN, for the dedication, compassion and team effort that they put forth through a very trying past year and in particular the first quarter of FY 2013.

Last year the Radnor Dermatology Clinic started an immense expansion and renovation project that took over seven months to complete. This project encompassed three divisions of the department but affected all four divisions. Throughout the renovations and painting of the department, planned and unplanned medical leaves, vacations and more, the clinical staff worked together to make sure that our physicians had the support that they needed and our patients continued to receive the care that they expect from our facility. The team went above and beyond by working directly with other team members to get training in clinic areas where they had no previous experience. “We have an amazing department and although it was a trying time, it has strengthened our team to be even more outstanding.”

Twenty-Ninth Pillsbury Lectureship in Dermatology

The Twenty-Ninth Pillsbury Lectureship in Dermatology was held on Thursday, May 2, 2013 in the Smilow Center for Translational Research Auditorium at the University of Pennsylvania Perelman School of Medicine. Lynn Anne Cornelius, MD, presented the lecture entitled, “Melanoma and UV: Topics throughout the Stages”.

Dr. Cornelius is a Professor of Medicine and Chief of Dermatology at Washington University, St. Louis, MO.

Dr. Cornelius received her MD from the University of Missouri School of Medicine in Columbia, MO. She trained at the University of North Carolina School of Medicine and completed her residency in dermatology at Washington University School of Medicine. She performed her post-doctoral fellowship at Emory University School of Medicine in Atlanta, GA.

Associated Faculty Anniversaries Announced:
The Pillsbury Dinner was held on Wednesday, May 1, 2013 at The Downtown Club in Philadelphia, Pennsylvania. Listed below are individuals who received special recognition.

5 year Anniversary
Ari Gutman
Stephen Hess
Barbara Mathes
Mordechai Tariow
James Treat

10 year Anniversary
Joseph Kist

15 year Anniversary
Eric Bernstein
Bruce Brod
John Laskas
Rebecca Suchin
Toby Frank Zachian

20 year Anniversary
Christine Egan
Michael Saruk
Jonathan Wolfe

30 year Anniversary
William Horn

35 year Anniversary
Waine Johnson

40 year Anniversary
Alexander Ehrlich

Dr. Lynn Anne Cornelius, Pillsbury Lecturer
Dr. Lynn Anne Cornelius and Dr. George Cotsarelis
**Hail and Farewell!**

**Hail to our 2013 Incoming Residents and Fellows**

**First Year Residents**

- **Aileen Chang, MD**
  University of Pennsylvania
  Perelman School of Medicine

- **Sotonye Imadojemu, MD, MBE**
  University of Pennsylvania
  Perelman School of Medicine

- **Badri Modi, MD**
  Yale University
  School of Medicine

- **Cory Simpson, MD, PhD**
  Northwestern University
  Feinberg School of Medicine,
  Medical Scientist Training Program, Chicago, IL

- **Joy Wan, MD**
  University of Pennsylvania
  Perelman School of Medicine

**Dermatopathology Fellows**

- **Filiberto Cedeno-Laurent, MD, PhD (CTCL Fellow)**
  Escuela de Medicina Ignacio A. Santos, Monterrey, Mexico

- **Sasha Stephen, MD (Former CTCL Fellow)**
  University of Pennsylvania
  Perelman School of Medicine

- **Roberto Novoa, MD**
  Columbia University Medical Center,
  New York Presbyterian Hospital

- **Campbell Stewart, MD**
  University of Vermont
  College of Medicine

**Clinical Educator Fellow**

- **Nada Elbuluk, MD**
  University of Michigan
  Medical School

**Procedural Dermatology Fellow**

- **Thuzar Shin, MD**
  Keck School of Medicine of the
  University of Southern California

**CHOP Pediatric Dermatology Fellows**

- **Lisa Arkin, MD**
  University of Pennsylvania
  School of Medicine

- **Lara Wine Lee, MD, PhD**
  University of Pennsylvania
  School of Medicine

**Farewells to Graduating Residents:**

- **Inbal Braunstein** – has accepted an Assistant Professor position at Johns Hopkins University Department of Dermatology
- **Brian Capell** – has accepted a Postdoctoral fellowship at the University of Pennsylvania, Department of Dermatology
- **Robert Micheletti** – has accepted an Assistant Professor position at the University of Pennsylvania, Department of Dermatology
- **Kelly Morrissey** – has accepted a Faculty position at the University of Colorado followed by Dermatopathology Fellowship at Boston University
- **Campbell Stewart** – has accepted a Dermatopathology fellow position at the University of Pennsylvania, Department of Dermatology
- **Lara Wine Lee** – has accepted a Pediatric Dermatology Fellow position at Children’s Hospital of Philadelphia (CHOP)
Welcome New Dermatology Faculty

Robert G. Micheletti, MD
Assistant Professor of Dermatology

Robert G. Micheletti, MD, joins the department of dermatology as an assistant professor at the Perelman School of Medicine at the University of Pennsylvania. Dr. Micheletti received his medical degree from Duke University School of Medicine. He completed a combined internal medicine and dermatology residency at the University of Pennsylvania.

Dr. Micheletti is board certified in dermatology and internal medicine. He is active in research and is a member of a number of medical organizations. He has a clinical focus in complex medical dermatology, including inpatient dermatology, graft versus host disease, vasculitis, and infectious diseases.

Dr. Micheletti sees patients at the Hospital of the University of Pennsylvania and at the Perelman Center for Advanced Medicine.

Temitayo A. Ogunleye, MD
Assistant Professor of Clinical Dermatology

Temitayo A. Ogunleye, MD joins the Department of Dermatology as an assistant professor of clinical dermatology at the Perelman School of Medicine at the University of Pennsylvania. Dr. Ogunleye received her medical degree from the University of Pennsylvania School of Medicine. She completed an internship with the Department of Medicine at the Albert Einstein Medical Center, Philadelphia and a dermatology residency at the University of Michigan Hospital System, Ann Arbor.

Dr. Ogunleye is board certified in dermatology and is a member of several medical organizations, including the American Academy of Dermatology, the American Medical Association, the Association of Professors of Dermatology, and the National Medical Association. She has a clinical focus in general dermatology, with special interests in skin of color and hair disorders.

Dr. Ogunleye sees patients at the Perelman Center for Advanced Medicine, Presbyterian Medical Center, and at the HUP/Dermatology site in Woodbury, NJ.

Lisa K. Pappas-Taffer, MD
Assistant Professor of Clinical Dermatology

Lisa K. Pappas-Taffer, MD, joins the Department of Dermatology as an assistant professor of clinical dermatology at the Perelman School of Medicine at the University of Pennsylvania. Dr. Pappas-Taffer received her medical degree from Wayne State University School of Medicine. She completed an internship with the Department of Medicine at New York University Medical Center and a dermatology residency at Brown University, Rhode Island Hospital. Dr. Pappas-Taffer also completed a clinician-educator fellowship at Penn focusing on complex medical dermatology.

Dr. Pappas-Taffer is board certified in dermatology and is a member of several medical organizations, including the American Academy of Dermatology, the Rheumatologic Dermatology Society, the Medical Dermatology Society, and the Association of Professors of Dermatology. She has a clinical focus in general dermatology, autoimmune conditions (including connective tissue diseases and blistering disorders), side effects of chemotherapy agents, and urticaria.

Dr. Pappas-Taffer sees patients at the Perelman Center for Advanced Medicine, the HUP/Bucks County practice, and the Philadelphia VA Medical Center.

Dr. Pappas-Taffer sees patients at the Perelman Center for Advanced Medicine, the HUP/Bucks County practice, and the Philadelphia VA Medical Center.
**Awards & Recognitions:**

**July 2013 to Present**

- **James Treat, MD - Assistant Professor**
  Awarded the 2013 CHOP Master Clinician Award, which is given in recognition of compassionate care, clinical excellence and collegiality.

- **Sarah Millar, PhD – Professor**
  Selected as an ELAM (Executive Leadership in Academic Medicine) Fellow for 2013-14.

- **Aimee Payne, MD, PhD – Assistant Professor**
  Received the Endowed Chair designation as the Albert M. Kligman Assistant Professor of Dermatology effective May 1, 2013-June 30, 2021

- **Abby VanVoorhees, MD – Associate Professor**
  Appointed Chairperson-Elect of the National Psoriasis Foundation Medical Board.

- **Ellen Kim, MD - Assistant Professor**
  Awarded the 2013 Leonard Berwick Memorial Teaching Award, in recognition of outstanding teaching, particularly among the younger medical faculty.

- **Rudolf Roth, MD – Associate Professor**
  Awarded Honorary Professor at INDERMA, Instituto de Dermatologia in Guatemala City, Guatemala (the leading dermatologic care and training center for Central America) at their Graduation Ceremony.

- **Remembering Colleagues**
  **Donald J. Adler (1949-2013)**

  Donald J. Adler, 64, died peacefully on October 5, 2013 surrounded by his loving family. He fought a courageous battle over the past two years with unbelievable strength, determination, and dignity until the very end.

  Dr. Adler was born in Reading, PA, but grew up in Cleveland, OH, and graduated from Ohio State University. He went to medical school at Michigan State University, College of Osteopathic Medicine, and did his dermatology residency at Wright State in Dayton, Ohio. He loved his work as a dermatologist and opened his own office in Doylestown in 1980 where he was much loved by his patients and office staff during his 32 years of practice. He often took patients with challenging cases to Grand Rounds at the University of Pennsylvania, where he had a reputation for bringing the most interesting cases to share with his colleagues. The residents he trained looked forward to being in his office and considered him a wonderful teacher.

  Dr. Adler was named Clinical Teacher of the Year by Lehigh Valley Hospital for his role in training dermatology residents. This past June, he was honored as Dermatologist of the Year by the Philadelphia Dermatological Society. Dr. Adler was voted as First Place Dermatologist by the Intelligencer ‘Best of Bucks-Mont’ numerous times.

  Dr. Adler loved classical music. He felt that music filled his soul. He was also an incredible chef, a talented photographer, and an avid traveler. He will be greatly missed by all who knew him.

  He is survived by his loving wife, Barbara, and their children, Lauren and Brandin Dear, Josh Adler and Catherine Haas-Adler, and his grandson Max. He also is survived by Richard and Mary A. Adler, Kenneth and Ellen Adler, Lesley Weissman-Cook and Jamie Cook; and his many nephews and nieces.
Partnering With Penn Dermatology

From our scientists and physicians to our nurses and everyone in between, Penn has consistently moved dermatology care forward with personalized care and therapeutic advances. The Department of Dermatology works continuously to develop new techniques and therapies through research and to educate the next generation of outstanding physicians and researchers.

To maximize our expertise and potential, improvements to our research infrastructure are required. Basic, translational, and clinical research activities are the hallmark of our clinical care and patient outcomes. With significant investment, this department will not only move forward on pressing medical challenges in dermatologic care, but also will be instrumental with diagnosis, new surgical techniques, and quality of life. Lastly, offering the best multidisciplinary care for our patients remains top priority.

Department of Dermatology Funding Priorities

Endowed Professorships – Rewarding Innovation
Supporting the work of Penn’s physician scientists is of utmost priority. Endowed professorships in investigative dermatology provide Penn Dermatology with the ability to retain and attract exceptional faculty. For decades, Penn’s preeminent dermatologists and researchers consistently receive recognition for excellence in patient care, research discoveries and education. Endowed professorships are instrumental in permanently recognizing the dedication of the department’s faculty and their important work.

Pilot Research Projects – Honoring Leaders
As the oldest dermatology department in the country, Penn Dermatology has been shaped by many great leaders whose legacies live on through their scientific breakthroughs. Established in 1874 by Dr. Louis Duhring, Penn Dermatology follows the traditions of many great 19th and 20th century physician researchers who worked collaboratively and across disciplines, such as with the engineering school. As a contributor to pilot research projects in cutaneous regeneration, Penn investigators gain the ability to impact patients worldwide with novel approaches to skin diseases, innovative treatments and the potential for cures.

Laboratories and Research Facilities – Promoting Scientific Advancement
Research space is of great necessity. New laboratories and instruments provide the path to great discoveries. With the right resources, Penn Dermatology will develop a cutaneous regeneration and tissue engineering effort focused on developing new treatments for skin disorders.

Fellowship Training Programs – Supporting New Investigators
Penn Dermatology’s training programs attract the most outstanding candidates, developing leaders in dermatologic research, academic and clinical dermatology. Funds directed toward fellowship training programs guarantee Penn Dermatology’s long tradition of educating exceptional scientists and clinicians.

Private philanthropy meets funding needs not covered by government grants or insurance reimbursements. Your donation enables us to break new ground and to improve upon existing therapies.

Philanthropic gifts of all sizes to support our research, educational and clinical endeavors are greatly appreciated. Naming opportunities within the department begin at the $25,000-level. Additionally, any gift can be given outright, through a planned giving vehicle, or can be structured to be paid over a 5-year period.

For more information about partnering with Penn Dermatology, please contact Caitlin Crowe at Penn Medicine Development & Alumni Relations at (215) 746-2167 or ccrowe@upenn.edu.
ASSOCIATED FACULTY 2013-2014

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SENA LEE, MD
Pigmented Lesion Group
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STEVEN S. FAKHARZADEH, MD
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ALBERT C. VAN, MD
Acne, Atopic Dermatitis, Childhood Blistering Disease, Genetic Skin Disease, Hemangiomata and Vascular Lesions
215.590.2169
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