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Montagna Symposium 2017—Precision Dermatology: Next Generation Prevention, Diagnosis, and Treatment

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Society for Investigative Dermatology Eugene M. Farber Travel Awards for Young Investigators: Mohammed Dany (Medical University of South Carolina), Ayman Grada (Boston University), Eunjeong Kwon (Harvard Medical School), Catherine Lee (University of Minnesota), Landon Kyle Oetjen (Washington University School of Medicine), Xiaoming Ouyang (Oregon Health & Science University), Christine Prodinger (Paracelsus Medical University), Julia Riedl (University of Minnesota), Saranya Wyles (Mayo Clinic); **Epidermolysis Bullosa Medical Research Foundation Travel Awards for Young Investigators**: Kristen Hook (University of Minnesota), Michael Pickett-Leonard (University of Minnesota); **National Psoriasis Foundation Travel Grants:** Min-Kyung Choo (Harvard Medical School), Yan Zhou (University of California, Davis); **Japanese Society for Investigative Dermatology Travel Award for Young Investigators:** Yoshihiro Ishida (Kyoto University), Yoskuke Ishitsuka (University of Tsukuba)

CONFLICT OF INTEREST

The authors state no conflict of interest.

The Annual Montagna Symposium on the Biology of Skin is a unique event in the scientific world, inspired by the passion for knowledge and collegiality of the late William Montagna, Ph.D. He was a great scientist in an age of giants, with interests as broad as ornithology, primatology, histology, entomology, taxidermy, and the French horn. It was this breadth of knowledge and willingness to share it that marked the early meetings (once known as the Brown Meeting, after Brown University, and then the Green Meeting, after the verdure of the Pacific Northwest). Every meeting is imbued with the sense of responsibility to live up to the extraordinary life and ideals of Dr. Montagna, and the results resonate far beyond the meeting itself.

The goal of the 66th annual meeting was to combine molecular understanding of disease and health with clinical applications of big data by exploring a progression of topics—from genes to cells to tissues to devices and ending with people—focused on the future of personalized dermatology. “Precision Dermatology: Next Generation Prevention, Diagnosis, and Treatment” was held October 12–16, 2017, in Stevenson, Washington, USA. Jakub Tolar (University of Minnesota) served as Program Chair, with John McGrath (King’s College London), Daniel Kaplan (University of Pittsburgh), Johann Bauer (Paracelsus Medical University), Sancy Leachman (Oregon Health & Science University), and Amy Paller (Northwestern University) serving as Session Chairs.

Genes: Digital Code

The advent of next generation sequencing has had an immense impact on gene discovery and the identification of disease mutations, germline and somatic. In the era of precision medicine, attention is turning toward the predictive value of data in both clinical care and understanding disease behavior. John McGrath chaired and introduced the “Genetically defined diagnosis and therapy” session with a presentation on “gene hunting” for Mendelian genodermatoses. Examples are emerging of useful reductionist dissection of genetic traits, often into more than one disease entity in the same individual, such as the occurrence of inherited hair and nail abnormalities clinically categorized as a form of ectodermal dysplasia but genetically reduced to two separate autosomal recessive hair and nail gene disorders (Hsu et al., 2017). Regarding clinical care, “pre-emptive” management is now alive in the clinic, improving physician astuteness and precision, for example in anticipating the future clinical course in infants with seemingly non-specific forms of ichthyosis (Saito et al., 2017) or in more complex genetic disorders like inherited poikilodermatous syndromes with potential for life-threatening complications (Takeichi et al., 2017). Such advances have an immediate benefit for patients and families, extending the goal of making an accurate genetic diagnosis into improving clinical management.

In tumors, molecular dissection of mutational events is also reworking models of cancers such as melanoma. Boris Bastian (University of California, San Francisco) discussed the revision of classic models of oncogene activation based on the timing of events such as activation of telomerase expression (Chiba et al., 2017), underscoring new dynamic models of pre-neoplastic lesions that reflect an equilibrium state of slow proliferation and attrition. Understanding this state—and dissecting key underlying mechanisms—is set to herald the

development of new biomarkers for diagnosis and prognostication (Shain and Bastian, 2017).

Precision at a gene level is also fundamental to dissecting human epidermal reprogramming, with relevance to aging, cancer, and inflammation. Raymond Cho (University of California, San Francisco) presented new work on advances in single cell RNA-seq technologies that have allowed for transcriptomic profiling of thousands of keratinocytes from different anatomic sites. These studies have identified more detailed and complex coordinate transcriptional changes during keratinocyte differentiation, identifying discrete regulatory programs on a genome-level scale. Understanding keratinocyte behavior in such a detailed manner provides a compendium for precision dissection of hundreds of disease states, with grand openings for future preventative or novel therapeutic interventions.

Johann Bauer and Mark Osborn (University of Minnesota) presented other approaches to precise, personal therapeutics, already in pre-clinical phases. These include the application of spliceosome-mediated RNA trans-splicing and gene editing technologies, particularly use of the clustered regularly interspaced short palindromic repeats (CRISPR)-Cas9 nuclease system, both of which are in development for testing in recessive dystrophic epidermolysis bullosa (RDEB) associated with mutations in the type VII collagen gene, *COL7A1* (Peking et al., 2017, Perdoni et al., 2016). At a gene level, precision medicine is making its clinical mark.

Cells: Biobricks

At a cellular level, also, the increased understanding of how cells and their mechanisms interact with their environments is creating improved treatment options. One of the critical aspects for the success of precision medicine is the accurate identification of specific pathways driving pathogenesis in disease states and the development of selective approaches to interfere with these processes. The path to translation relies on a solid understanding of disease mechanisms. Past success stories include psoriasis and atopic dermatitis where IL-23/IL-17 and IL-4, respectively, were found to be central components of each disease, and new biologic therapies targeting these cytokines have revolutionized the therapy of both diseases. Building on these successes, the goal of the “Cells as living medications” session was to explore 1) novel aspects of skin biology that may ultimately provide novel therapeutic targets and 2) new translational approaches that capitalize on recent discoveries.

Kenji Kabashima (Kyoto University) described, in the setting of the elicitation phase of contact hypersensitivity, a murine model of contact dermatitis, a structure of sequential leukocyte clusters forming along postcapillary venules through a coordinated chemokine driven process. This structure does not exist in the steady state but is “induced” in response to local inflammatory condition. He terms it “inducible SALT (iSALT),” reminiscent of the skin associated lymphoid tissue (SALT) initially proposed by Streilein in 1978. This tissue’s lymphoid structure is likely key to maintaining chronic inflammation (Kogame et al., 2017).

Paola Di Meglio (King’s College London) presented data on the aryl hydrocarbon receptor (AhR), an environmental sensor and transcription factor activated by the environmental

pollutant dioxin but also by physiological ligands of dietary and microbial origin. AhR is a critical regulator of homeostasis at barrier organs (gut, skin, lung). In the skin, physiological AhR ligands in keratinocytes decrease their responsiveness to inflammatory stimuli. Thus, AhR acts as an anti-inflammatory brake to constrain chronic inflammation and maintain skin homeostasis.

Daniel Kaplan presented his group's ongoing work on the importance of pain-sensing nerves in the skin for the development of innate resistant to epicutaneous infection with *C. albicans*. He showed that nerves are required to elicit IL-23 from dermal dendritic cells that then promote a Type 17 immune response required for host defense. He also showed how *C. albicans* directly activated two subsets of nerves that can be distinguished based on expression of IB4 through distinct ligand/receptor interactions. These three observations provide potential novel targets for therapeutic future interventions.

Anthony Oro (Stanford University) contributed elegant data on chromatin dynamics in "stemness" and cell fate commitment of skin cells, and Markus Frank (Harvard Medical School) described a novel and highly relevant type of ABCB5 positive dermal stem cell. Capitalizing on past results showing the potential promise of allogeneic bone marrow/cord blood transplantation (BMCBT), Jakub Tolar presented his recent results using BMCBT for the treatment of RDEB. He found encouraging results with evidence of type VII collagen expression in 20/39 patients. Junctional EB (JEB) was also treated but was found to help only patients with α3 laminin 332-deficient subtype.

Aimee Payne (University of Pennsylvania) discussed two strategies for antigen-specific immunotherapy currently in preparation for first-in-human clinical trials, including autoantigen peptide-coupled cells, which employ ex vivo alteration and reinfusion of autologous cells to induce antigen-specific immune tolerance, and chimeric autoantibody receptor (CAAR) T cells, which induce antigen-specific B cell depletion (Ellebrecht et al., 2016). Opportunities and challenges in translating these novel immunotherapies to trials were discussed, focusing on the potential for long-lasting and targeted disease remissions with CAAR T cells, akin to those observed with the recently FDA-approved chimeric antigen receptor (CAR) T cell therapies for B cell cancers, versus the current high cost of supplies and processes for such complex cellular therapy approaches, which is expected to drop dramatically as additional cellular immunotherapies come to market.

Tissues: Bioplastics

A spectacular change is also occurring at the tissue level, as regenerative medicine and bioengineering expand the possibilities of replacing and restoring skin to health. The "Engineering new skin" session, chaired by Johann Bauer, began with M. Peter Marinkovich's (Stanford University) presentation of his data on *COL7A1* gene grafting in patients suffering from RDEB (Siprashvili et al., 2016). Currently, seven patients have been treated without experiencing major side effects. Clinical efficiency varied, with a gradual decline over a period of 12 months in some treated individuals.

Michele De Luca reported on combined ex vivo cell and gene therapy by transgenic epidermal stem cells in one JEB patient. After three operations and a follow-up period of 2.5 years, no blisters were seen, and all junctional proteins were expressed. Importantly, no clonality could be detected. Progenitors were diluted from 91% to 13% after eight months, thus proving that holoclones constitute the regenerative compartment of the epidermis (Hirsch et al., 2017). Continuing with regeneration, Michael McAlpine (University of Minnesota) showed that 3D printing is moving fast to regenerate parts of the human body such as the ear and prostate. Currently, microelectronics are used to reproduce nervous content to fabricate 3D tactile sensors.

In a series of "**2020 Vision Soap Box Talks**," speakers presented short talks on forward-looking, precision-oriented topics in cancer biology. Molly Kulesz-Martin (Oregon Health & Science University) elaborated on how precision oncology is used to identify drugs that interact with patient-specific biological targets of interest. She introduced the concept of a unified Cancer Targetome that aggregates drug-target interactions in an evidence-based framework (Blucher et al., 2017). Genomic data mining and evaluation of new drug functionality is complemented by available small-molecule drugs. One example of this approach is the designed combination of an ALK inhibitor with an EGFR inhibitor that was tested on eight patients' head and neck cancer-derived cells.

Naoki Oshimori (Oregon Health & Science University) presented data on gene signatures of putative cancer stem cells in squamous cell carcinomas. TGF-β-activated squamous cell carcinoma stem cells were found to be moving on the edge of the tumor. Gene expression profiling showed that *ADAP1* high and low expression was associated with risk of progression. Mohammed Dany (Medical University of South Carolina) looked into signalling pathways regulated by ceramides as a means of overcoming drug resistance in cancer. Ceramides can act as tumor suppressors by inducing lipid mitophagy. He showed that ceramides are translocated into mitochondria after kinase inhibitor therapy. Upon development of resistance, ceramide analogues can induce lethal mitophagy.

In addition to addressing biological mechanisms of the skin, the meeting also examined individual and societal factors that impact the field of dermatology. An affecting example was presented by Kelly Griffith-Bauer (University of California, San Francisco) in a "Perspective Talk" on the Melanoma SCAR Project, a collection of artistic photographic images displayed with quotes from 25 participants diagnosed with stage 0 to stage 4 melanoma. Dr. Griffith-Bauer's project aims to raise melanoma awareness and personalize skin malignancy.

Banquet speaker Douglas R. Lowy (NIH/NCI) addressed the critical importance of prevention and early detection, taking up a subtheme in the meeting, that "imprecise precision medicine is with us." Adding that we need to increasingly define common molecular subsets for patients who do and do not respond to treatment—and why—Dr. Lowy shared his work on prevention of human papilloma virus (HPV) driven cancers. The high titer vaccine to capsid proteins of oncogenic HPV types enabled by his work can provide close to 100% protection against persistent infection and premalignant disease when given to people before they become sexually active (Lowy and Schiller, 2017). In telling this

success story, Dr. Lowy underscored team science, testing of early detection and prevention strategies against current standards of care, cost effective “one dose” vaccination, incorporating new technologies in developing nations without trained screening specialists, and, by 2030, the possibility of eradicating cervical cancer as a worldwide epidemic.

Devices: Living software

One of the more robust debates held during the meeting was in the “Big data, bigger picture” session, chaired by Sancy Leachman and designed to highlight new and powerful technologies, such as novel skin imaging devices (in vivo confocal, optical coherence tomography, photoacoustic imaging) and the use of mobile phone application technologies, that could be applied to personalized approaches in the prevention of melanoma and in Serial Measurement of Molecular and Architectural Responses to Therapy (SMMART) trials, detailed by Joe Gray (Oregon Health & Science University), which tailor sequential treatments to individual patients based upon 3D and genomics monitoring of serial samples over time. A formal Debate session, chaired by Amy Paller and Dr. Leachman, centered around whether technologies are currently able to improve medical care or are still in the “blind men and an elephant” phase in which technology reveals part of the answer but isn’t able to capture the entire picture.

Although Drs. Paller and Leachman agreed that creating and effectively utilizing new technology will benefit medicine as a whole, the audience raised questions about how technologies should be validated, whether they are ready for clinical application, and how to overcome barriers to dissemination. For example, Alexander Witkowski (University of Modena and Reggio Emilia) reported on rapid adoption of in vivo reflectance confocal microscopy (RCM) in European clinics. The use of RCM in the United States has not been as widespread, and the audience discussed whether or not 1) this lack of dissemination was due to a failure of the technology to be completely validated relative to traditional dermatopathology, and 2) it was reasonable to validate in vivo RCM by comparison to histology.

This led to further discussion about whether the poor correlation coefficient between dermatopathologists (when reading difficult melanocytic lesions) creates a barrier to improvement—it is difficult to prove that a technology is better than the “gold standard” (traditional histopathology) when the gold standard is error prone. There was also discussion about how reimbursement for technologies impacts the uptake rate in the United States and whether a conflict of interest exists when a specialty that may lose revenue when new technology is implemented (e.g., dermatopathology) is responsible for expert review to validate the same technology.

Whether a human provider will be necessary in the era of artificial intelligence (AI)-driven practice formed a lively debate topic in this session, following Andre Esteva’s (Stanford University) presentation on the potential for deep neural networks to enhance diagnostic capabilities of mobile technologies (Esteva et al., 2017). Although there was some concern about the loss of the human side of medicine, many patients have been shown to prefer an

on-screen avatar to a live physician due to a belief that they will receive more objective information in a non-judgmental way and that their privacy may be better protected.

The ability to link AI with mobile applications, such as MoleMapper™, a research-directed mole-tracking app (Webster et al., 2017), was also discussed. The audience was passionately in favor of increasing access to care for all members of society through the use of these devices but only after validity has been confirmed.

People: Patients

Although advances in technology for gene sequencing and mRNA and protein expression patterns have improved our understanding of skin disease, options for clinical use of gene therapy as a tool in personalized dermatologic care remain limited both in access and impact. However, new approaches are emerging from evaluation of the functional impact of genetic alterations (e.g., suppressing pathway activation or replenishing insufficient end products). The use of biologic therapy is an example of the value of this approach for two polygenic inflammatory skin disorders, psoriasis (inhibition of TNF, IL-23, or IL-17 pathway signaling) and atopic dermatitis (inhibition of IL-4/IL-13 signaling). The “Adaptive clinical trials” session, chaired by Amy Paller, explored these topics.

Dr. Paller’s presentation emphasized the need to seek therapies, including repurposed therapies, for groups of disorders with similar phenotypic changes. Twenty-one patients with various orphan forms of ichthyosis (Netherton syndrome, lamellar ichthyosis, congenital ichthyosiform erythroderma forms of autosomal recessive congenital ichthyosis, and epidermolytic ichthyosis) had strong cutaneous Th17 cytokine skewing that correlated with disease severity that closely resembles psoriasis (Paller et al., 2017). Blood flow cytometry of 50 ichthyosis patients showed Th17+ and Th22+ CLA+ CD4+, suggesting systemic Th17/IL-22 immune activation in ichthyosis. These studies led to an ongoing study of secukinumab, an IL-17 inhibitor for adults with ichthyosis.

Eddy Wang (Columbia University) introduced the role of increased Janus kinase/signal transducers and activators of transcription (JAK/STAT) signaling in a variety of autoimmune disorders, including alopecia areata. Discovery of the upregulation of several common γ-chain cytokines and of interferon gamma (IFN-γ) pathway expression in alopecic scalp led to the demonstration that these signatures are significantly upregulated in the lesional skin from both the C3H/HeJ mouse model and humans. Inhibition of the downstream JAK/STAT pathway (ruxolitinib or tofacitinib) has led to biologic and clinical responses in two open-label clinical trials (Kennedy Crispin et al., 2016, Mackay-Wiggan et al., 2016).

Joyce Teng (Stanford University) discussed the somatic *PIK3CA* mutations that underlie many lymphatic and venous malformations and overgrowth syndromes. Somatic *PIK3CA* mutations alone, however, do not account for phenotypic variation and differences in clinical response, highlighting the need to understand the overall gene expression profiles in vascular malformations and to explore the role of additional mutations and the particular cell types involved in pathogenesis. Preliminary studies revealed additional mutations in other PI3K subunits and molecules involved in Ras/Erk–MAP kinase signaling. Gene expression

profiles from the peripheral blood of patients with lymphatic malformations showed activation of cell cycle, DNA replication, and protein phosphorylation pathways in addition to AKT/mTOR/PI3K, highlighting disease heterogeneity and the need to develop new targets, either used alone or as part of combinatorial therapies for vascular anomalies.

The most rapid advances in understanding pathway inhibition have been leveraged to treat melanoma. Roger Lo (University of California, Los Angeles) discussed the development of melanoma BRAFi and BRAF-+MEKi resistance at the genomic, epigenomic, and immune microenvironmental levels. Newer combinatorial approaches incorporate inhibitors of RAF dimerization, ERK/MAPK, and the RAF-MEK hetero-complex. Melanomas with additional *PI3K-AKT* mutations may respond to BRAFi+MEKi+AKTi. Acute withdrawal of BRAFi +MEKi in MAPK inhibitor-resistant melanomas induces “drug addiction,” with MAPK/ERK pathway rebound and melanoma cell toxicity that can be increased by adding a PARP inhibitor to prevent DNA damage repair (Hong et al., 2018). Melanomas also escape from MAPK inhibitors by gaining immune suppressive properties (innate anti-PD-1 resistance (IPRES)) (Hugo et al., 2016, Song et al., 2017), suggesting blockade of both the PD-1 axis and IPRES processes during MAPK-targeted therapy to improve tumor control.

This meeting, as the 65 that have preceded it, provided a broad range of information and brought clinicians and researchers together to expand understanding of future directions and current patient need in the hopes of stimulating new connections, collaborations, and knowledge in the area of “Precision Dermatology” as the participants (Figure 1) dispersed back to their research and practice homes.

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The Montagna Symposium on the Biology of Skin, directed by Molly F. Kulesz-Martin, Ph.D., is an annual non-profit scientific meeting, inaugurated in 1950 by William Montagna, Ph.D., that gathers leading cutaneous biologists and dermatologists to discuss new findings, techniques, and goals in skin biology. Podcast and video interviews of speakers are available via the JID Multimedia collection at

http://www.jidonline.org/.

Joint Montagna Symposium/PASPCR Annual Meeting 2018

October 17 – 22, 2018, Salishan Resort, Gleneden Beach, Oregon, USA

“Melanoma to Vitiligo: The Melanocyte in Biology & Medicine”

Program Chairs

Sancy A. Leachman, M.D., Ph.D.

Thomas J. Hornyak, M.D., Ph.D.

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Figure 1.
Group photo (courtesy of Jeremy Bauer).