The 68th Montagna Symposium on the Biology of the Skin was held from 10 to 14 October 2019 at Salishan Lodge in Gleneden Beach, Oregon. The theme of the meeting was “Decoding Complex Skin Diseases: Integrating Genetics, Genomics, and Disease Biology.” The meeting emphasized the integration of multiple themes and disciplines to better understand some of the most common skin diseases, ranging from psoriasis to alopecia areata to vitiligo to lupus erythematosus to atopic dermatitis and food allergy. Promising therapeutic strategies are emerging for all of these diseases, providing clues for ways to connect the bench to the bedside.

A common thread was the success of GWASs, which have highlighted the importance of regulatory signals versus coding variation. These diseases also share an environmental component linked to immune system function. Hence, beyond GWASs, this meeting focused on gene regulatory mechanisms, the single-cell revolution, in vivo systems for dissection of disease pathogenesis, and the relationship between genetics and environment in the context of host defense. We concluded with a translational roundtable designed to explore how these interrelated fields can best be directed toward long-term disease control and, ultimately, a cure.

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The Montagna Symposium on the Biology of Skin, formerly the Annual Symposium on the Biology of Skin, was initiated at Brown University (Providence, RI) in 1950 by Dr William Montagna. The symposium grew from the need to communicate investigative work in cutaneous biology and to provide a link between basic scientists studying the skin in humans and animals and clinically trained scientists in investigative dermatology. Since then, over 5,000 scientists, physicians, and students from around the world have attended the symposium, which annually addresses a major topic in cutaneous biology. The topic of this year’s 68th Symposium was “Decoding Complex Skin Diseases: Integrating Genetics, Genomics, and Disease Biology.”

What have GWAS taught us about common skin diseases?

As of this writing, GWASs have generated over 4,346 publications involving at least 166,103 associations between genetic variants and many human traits and diseases (Buniello et al., 2019). The concept underlying this 68th Montagna Symposium was to nucleate the meeting with key skin-relevant GWASs, then to bring to bear the many other facets of what we have learned to elucidate the nature of complex genetic skin diseases in the post-GWAS era (Swindell et al., 2014), including genomics, immunology, environment, and tools for relating these findings to disease biology, including validation of pathogenic mechanisms and therapeutic translation (Figure 1).

After investing the better part of a decade in fine mapping of GWASs, one of the most consistent features of complex trait GWAS has been the observation that the majority of genetic signals underlying complex traits are regulatory in nature (Boyle et al., 2017). Keynote speaker Dr Paul Khavari (Stanford University, CA) provided a comprehensive look at the landscape of genetic regulation of gene expression in keratinocytes (KCs) as well as in immune cells, utilizing a combination of CRISPR perturbations and single-cell chromatin accessibility profiling using the assay for transposase-accessible chromatin and sequencing (ATAC-seq). The KC

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studies defined a trajectory of epidermal differentiation regulated by five key transcription factors (TFs) interacting either positively or negatively: CEBPA, EHF, ZNF750, KLF4, and JUNB (Rubin et al., 2019). Notably, ZNF750 and KLF4 have been implicated as psoriasis genetic loci in either GWAS (KLF4) (Tsoi et al., 2012) or pedigree studies (ZNF750) (Yang et al., 2008). Dr Khavari’s results also emphasized the role of chromatin looping in determining the nature of TF interactions (Rubin et al., 2019).

Led by Dr Anne Bowcock, the next session featured invited presentations by Drs Elder, Harley, Spritz, and Sara Brown (University of Dundee, Scotland) along with young investigator presentations selected from the abstracts by Viktoryia Laurynenka (University of Cincinnati, OH) and Bing-Jian Feng (University of Utah, Salt Lake City, UT). Noting that approximately 80–90% of GWAS signals are regulatory, Dr Elder pointed out how chromatin looping can complicate the mapping of GWAS hits to relevant target genes in the context of promoter-enhancer interactions. To address this, he presented ongoing studies of T-cell subsets from 150 individuals, analyzed for chromatin accessibility by ATAC-seq, coupled with transcriptome analysis by RNA sequencing (RNA-seq) and GWAS genotyping to generate chromatin quantitative trait loci and expression quantitative trait loci (eQTL). He reported that 10 of 11 evaluable GWAS hits displaying genotype-dependent chromatin accessibility in activated CD8+ T cells are also eQTLs in blood cells, as defined by the eQTLGen database (Vosa et al., unpublished data). Turning from regulatory to coding variants, he showed how the Act1 D10N variant in the TRAF3IP2 gene can enhance human T helper (Th) 17 cell polarization in vitro, in a manner that is enhanced by neutrophil extracellular traps (Lambert et al., 2019).

Dr Harley’s presentation summarized his longstanding interest in the genetics of systemic lupus erythematosus (SLE) (Harley et al., 2009) and its potential relationship with Epstein-Barr virus (EBV) (Arbuckle et al., 2003). He demonstrated a highly significant overlap of genetic signals for SLE and five other autoimmune diseases with binding sites for both virally encoded (EBNA-2) and host-encoded TFs involved in EBV latency (Harley et al., 2018). A recent expansion of the Harley laboratory chromatin immunoprecipitation (ChIP)-sequencing database to other EBV-encoded TFs identified EBNA3B, EBNA 3C, and EBNA1LP, all of which are involved in maintaining proliferation of EBV-transformed B-cells (Allday et al., 2015). These studies point toward a balancing act for the immune system between the need to control EBV (which is nearly ubiquitous in adults) and the development of autoimmunity.

Dr Spritz reviewed the current status of vitiligo GWAS, which has currently identified 52 vitiligo-susceptibility loci in individuals of European ancestry. He made the case for vitiligo being a relatively simple type of complex genetic disorder because vitiligo heritability is high (estimated at 46–72%), with about one-third of heritability represented by common variants and two-thirds by rare variants, and very little missing heritability. As is the case in psoriasis (Henseler and Christophers, 1985), vitiligo is a strongly HLA-associated disease with a bimodal distribution of age at onset (Jin et al., 2019). Dr Spritz and colleagues identified a major locus specific to the early onset case subgroup, which maps to an indel in a major histocompatibility complex class II enhancer. They identified a haplotype spanning this indel that confers high risk for vitiligo (OR of 8.1) and increases the expression of HLA-DQB1 mRNA and HLA-DQ protein on professional antigen-presenting cells, demonstrating a convergence of regulatory and protein-coding variants that is more important for conferring risk for early-onset vitiligo than traditional HLA class II alleles alone. Dr Spritz also noted that vitiligo age of onset has become more delayed in recent years, especially over 1973–2004, suggesting the importance of interactions between genetics and environment (Jin et al., 2020).

Dr Brown reviewed GWAS and meta-analyses that have revealed 31 loci associated with atopic dermatitis (AD) and noted that these loci jointly account for less than 20% of AD heritability, meaning that further work is needed to fully understand individual risk. Whereas some AD risk loci have confirmed pre-existing knowledge, including the role of skin barrier and type 2 inflammation in AD pathogenesis, others have yielded new insights, including evidence of autoimmunity and a role for Langerhans cells. Whereas most significant AD GWAS peak, mapping to the EDC on chromosome 1q21.3 contains the well-known FLG gene, loss-of-function mutations, and copy number variation within FLG, which do not fully explain this strong effect. Thus, the EDC is likely to contain additional risk variants that remain to be defined. As is the case in psoriasis, lupus, vitiligo, and other complex disorders, the majority of other AD risk loci are in intergenic regions, which will require detailed molecular studies carried out in cells and tissues of relevance to AD. She reported that one such region lies between the

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**Figure 1. Main themes of the meeting and integration with disease biology.**

**Decoding Complex Skin Diseases**

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<td>Single-cell transcriptomics</td>
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<td>Genetic effects on chromatin accessibility and gene expression</td>
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<td>Animal models</td>
<td>Ex vivo and in vitro models</td>
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<td>Effects of recent environmental changes</td>
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**JE Gudjonsson and JT Elder**

68th Montagna Symposium
EMSY and LRRC32 genes, both of which are strong candidate genes for AD risk.

Understanding GWAS signals—chromatin structure and gene regulation

Focusing further on the roles of chromatin structure and gene regulation as tools for understanding GWAS signals, a session directed by Dr Bogi Andersen included invited talks by Drs Evan Boyle (University of California, San Diego), Tõnu Esko (University of Tartu, Estonia), Roger Pique-Regi (Wayne State University, Detroit, MI), and Manuel Garber (University of Massachusetts Medical School, Worcester, MA) and included young investigator oral presentations selected from the abstracts by Sheng-Pei Wang, PhD (National Cheng Kung University, Taiwan) and Sarah T. Arron, MD, PhD (University of California, San Francisco). Dr Boyle’s talk expanded on a thought-provoking recent publication (Boyle et al., 2017), taking a global approach to understanding the links between genetic variation and disease. He proposed that gene regulatory networks interconnect in such a way that many different genes expressed in disease-relevant cells are liable to affect the functions of core disease-related genes and hence, the so-called missing heritability problem may be explained at least in part by effects of genetic variation on genes outside core pathways.

Dr Esko described his team’s efforts to determine how the genes and pathways through which disease-associated genetic variants identified by GWAS exert their effects on the phenotype. To this end, they combined genetic and transcriptomic data from over 30,000 blood samples within the eQTLGen Consortium (Vosa et al., unpublished data) to perform cis-eQTL mapping (i.e., the genetic variant influencing gene expression is near the target gene) and trans-eQTL mapping (i.e., genetic variant is far from the target gene) on >10,000 genetic risk factors identified by GWAS. They also calculated polygenic risk scores for over 1,300 complex traits and correlated those with gene expression levels. They found that 90% of all protein-coding genes show a significant cis-eQTL effect and that over 30% of all established genetic risk factors for disease manifested trans-eQTLs effects, impacting the expression of 4,700 unique genes, with examples of individual hub SNPs having downstream effects on over 200 genes. They found both cis and trans effects on gene expression for 2,600 risk SNPs, finding these interactions between their encoded proteins more often than would be expected by chance. In several cases, the trans-eQTL effect was mediated by the corresponding cis-eQTL gene. Overall, these findings emphasized the value of a highly powered sample to uncover the relationships between GWAS signals and genetic effects on gene expression.

Continuing the theme that genetic variants relevant for complex traits are more commonly found in noncoding regions, Dr Pique-Regi described his group’s efforts to understand the gene regulatory grammar encoded in the human genome. He emphasized that the heterogeneity of cell types found in most tissues and the additional complexity due to various environmental conditions both represent important knowledge gaps in decoding these grammar rules. He also summarized computational methods (such as CENTIPEDE [Pique-Regi et al., 2011]) for identifying gene regulatory elements from chromatin accessibility data as well as tools for predicting the effect of disease-associated genetic variants on gene regulation (Wen et al., 2017) and for studying the regulatory elements directing gene expression response to different environmental conditions using ATAC-seq and massively parallel reporter gene assays.

Dr Garber addressed the interesting question of why some enhancers are not functionally well-conserved across species (Villar et al., 2015) but others are. Continuing his laboratory’s interest in chromatin structure and gene regulation in dendritic cells (DCs), they focused on genes responsive to lipopolysaccharide in the DCs derived from mouse bone marrow and cytokine-induced human monocytes, followed by ATAC-seq, RNA-seq, and ChIP for H3K27Ac (Donnard et al., 2018). This work revealed that AP-1 family members tend to connect promoters and enhancers. They also found that gene expression programs are more divergent between man and mouse for mildly induced genes but are more highly conserved for strongly induced genes and early-response genes. Consistent with selective pressure for pathogen resistance, they found that the density of these conserved motifs strongly predicted the responsiveness of a gene to pathogen exposure, again emphasizing the interaction of genetics and environment.

Transcriptomics: The single-cell revolution

Moving on from the role of genetic regulation of gene expression and chromatin structure to understand GWAS signals, the next session focused on single-cell transcriptomic profiling to gain deeper insights into biological mechanisms in various disease states. Directed by Dr Gudjonsson, this session included invited talks by Drs Niroshana Anandasabapathy (Weill Cornell Medicine, New York City, NY), Muzifihani Hanifia (Newcastle University, United Kingdom), Michael D. Rosenblum (University of California, San Francisco), and Rui Yi (University of Colorado, Boulder, CO) as well as presentations selected from the abstracts by Fabian V. Filipp (Helmholtz Zentrum München, Bavaria, Germany) and Mrinal K. Sarkar (University of Michigan, Ann Arbor).

Dr Anandasabapathy addressed how DCs in peripheral tissues such as the skin influence T-cell behavior and memory formation through PD1L2 signaling. She addressed the consequences this activation has for immune memory, with a focus on response to melanoma. These findings were related to the behavior of immune populations in tumors and the consequences this has for melanoma-specific outcomes.

Dr Haniffa demonstrated the power and utility of single-cell RNA-seq to understand the functional organization of the developing human immune system. She identified, using single-cell transcriptomic profiling of approximately 140,000 liver cells and 74,000 skin, kidney, and yolk sac cells, the repertoire of human blood and immune cells during development (Popescu et al., 2019). Using this unique source of fetal tissue, her group was able to demonstrate physiological erythropoiesis in fetal skin and the presence of mast cells, NK, and innate lymphoid cell precursors in the yolk sac, thus
providing a blueprint for the study of pediatric blood and immune disorders and a reference for harnessing the therapeutic potential of hematopoietic stem cells (Popescu et al., 2019).

Dr Rosenblum showed novel findings in regard to the role of regulatory T cells (Tregs) in the skin and how they may actively suppress profibrotic immune responses in the skin. His work demonstrated that skin Tregs preferentially express high levels of GATA3, a master Th2 TF. Notably, Treg depletion resulted in a preferential increase in Th2 cytokine production in the skin, and this was accompanied by spontaneous fibroblast activation, profibrotic gene expression, and dermal fibrosis (Kalekar et al., 2019). This work suggests that Tregs play an important role in regulating fibroblast activation in the skin and play an active role in fibrotic skin diseases.

Dr Yi addressed single-cell transcriptomics and open chromatin analysis of developing skin. He discussed how few embryonic progenitors give rise to self-sustaining cell lineages that maintain tissue integrity throughout the lifespan of the organisms, how mammalian skin can be used as a model system, and how sophisticated genetic manipulation can be used to characterize this system at the single-cell level using both single-cell RNA-seq and single-cell ATAC-seq, providing unprecedented insights into understanding transcriptional mechanisms that govern epidermal development and maintenance of hair follicle stem cells (Fan et al., 2018).

The second keynote speaker of the symposium was Dr Richard Flavell (Yale University School of Medicine, New Haven, CT). As was noted during his introduction, during his highly decorated career, he has been a driving force in many of the key research developments upon which this meeting was based, including the discovery of gene regulation in trans through kissing chromosomes as well as the molecular basis of T-cell differentiation and the role of several receptor families in the innate immune response, including Toll-like receptors and intracellular nucleotide-binding oligomerization domain-like receptors. He was also among the first to demonstrate the role of inflammatory and microbial homeostasis in the pathogenesis of several chronic diseases, including inflammatory bowel disease and the metabolic syndrome. He presented new work focused on the role of neuronal innervation in the control of IL-18 expression and secretion in the gut. This neuroinflammatory connection was unexpected but fascinating and seems very much involved in keeping with the major role of the nervous system in regulating gut physiology.

**Immunopathogenesis of complex skin diseases**

The next session continued on the same theme but dove into how these technologies could be used to gain better insights into the immunopathogenesis of complex skin diseases. This session was directed by Dr Dennis Roop and included talks by Dr Nicole Ward (Case Western Reserve University, Cleveland, OH), Johann E. Gudjonsson (University of Michigan, Ann Arbor), Angela Christiano (Columbia University, New York City, NY), and J. Michelle Kahlenberg (University of Michigan, Ann Arbor). This session included young investigator oral presentations by Jessica Ludwig BS (Case Western Reserve University, Cleveland, OH) and Dr Matthew D. Veseley MD, PhD (Yale University, New Haven, CT).

In her talk, Dr Ward presented data showing how to use omics approaches to generate innovative mouse models of psoriasis and how these models can be used to gain new insights into disease pathogenesis that can be brought back to the patient to better understand their disease pathogenesis (Hawkes et al., 2018). She also presented data describing the utility of using mouse models to explore at the cellular and molecular levels epidemiological findings such as thrombosis, atherosclerosis, and psoriatic arthritis, which are associated with psoriasis (Wang et al., 2016, 2012), as well as addressing anecdotal reports from patients with psoriasis, such as the lack of improvement or worsening with anti–IL-6 agents in psoriasis (Fritz et al., 2017). This demonstrated the usefulness of using appropriate mouse models to study and understand psoriatic disease pathogenesis.

Switching over to a skin disease often characterized as allergic rather than autoimmune in origin, Dr Gudjonsson demonstrated the use of transcriptomic approaches to study the comparative pathogenesis of psoriasis and AD. This work shows that over 80% of genes dysregulated in AD skin overlaps with dysregulated genes in psoriasis. However, despite this overlap, AD was more heterogeneous in terms of transcriptomic variability and showed a dominance of IL-13 pathways, whereas, in contrast, psoriasis was dominated by IL-17 responses (Tsoi et al., 2019).

In her talk on genetics and immunology of alopecia areata, Dr Chris-tiano described findings from her laboratory on the identification of the genetic basis of alopecia areata as a complex genetic disease (Petukhova et al., 2010), including the role of effector CD8+ cells and the role of IL-15 in maintaining their effector function. She described how this led to the successful clinical trials using inhibitors of JAK signaling to inhibit IL-15 signaling (Xing et al., 2014). She also described exciting recent work using single-cell TCR sequencing to help identify the autoantigen(s) in alopecia areata and recent work focusing on the role of environmental triggers, especially gut microbiota. Her work demonstrates how GWASs can be used to uncover new disease mechanisms in complex autoimmune disorders and how this can inform the clinical investigation of repurposed drugs.

Dr Kahlenberg discussed her group's work outlining the pathogenesis of cutaneous lupus erythematosus (CLE). CLE is found in up to 70% of patients with SLE and can also occur as a skin-only condition (Stannard and Kahlenberg, 2016). In her talk, Dr Kahlenberg outlined recent developments in our understanding of CLE, including inflammatory mediators, differences in lesional subtypes, predisposition to inflammation in normal-appearing SLE skin, and mechanisms of flare by UV light (Sarkar et al., 2018; Wolf et al., 2018). These findings have been the driver behind new trials of medications for CLE treatment.

**Nature and nurture: Microbiome in complex skin diseases**

The symposium ended on a high note with a shift in the focus to nature and nurture—the role of microbiome in
complex skin disease. This session was led by Dr Elder and included talks by Drs Cathryn Nagler (University of Chicago, IL), Jose U. Scher (New York University), Lloyd S. Miller (John Hopkins University, Baltimore, MD), and Shruti Naik (New York University). Two young investigator presentations were given by Dr Tatsuya Ogawa (University of Tsukuba, Japan) and Dr Tamara Terzian (University of Colorado, Boulder, CO).

Noting that the prevalence of life-threatening food allergies is increasing rapidly in westernized societies, Dr Nagler suggested that emerging lifestyle practices, including antibiotic use, cesarean births, and formula feeding and other dietary changes are altering intestinal bacterial communities, especially early in life. Her team tested this idea by colonizing germ-free mice with feces from healthy or cow's milk allergic infants. They found that colonization with bacteria from healthy but not cow's milk allergic infants were protected against anaphylactic responses to a cow's milk allergen, accompanied by differences in gut bacterial composition. Using transcriptomic analysis, they identified a signature from one of the altered bacterial species (a Clostridium) that was also associated with distinctive human transcriptome signals in the ileal epithelium relating to its barrier function (Feehley et al., 2019). On the basis of this research, Dr Nagler et al. are working to construct a therapy to strengthen the gut barrier and prevent food allergens from passing out of the gut and into the bloodstream where they can trigger allergic responses.

Dr Scher addressed the role of microbiome in psoriasis and psoriatic arthritis. He described how there is increasing evidence for a role of the microbiome in contributing to and affecting the severity of psoriasis disease and how alterations in the composition of the microbiome can result in dysbiosis, which impacts the immune system leading to autoimmunity, persistent inflammation, and tissue damage. A part of his talk addressed pharmacomicrobiomics, which is a novel area of research that investigates the effect of variations within the human microbiome on drugs (Abdollahi-Roodsaz et al., 2016).

Dr Miller presented work on the interaction between FLG mutations such as those associated with AD, injury, and the skin microbiome. He showed, using a mouse model of skin injury in Flg-deficient mice, the development of a chronic AD-like skin inflammation associated with IL-1α intracellular release from KCs, which could be blocked by the inhibition of IL-1α but not IL-1β. Interestingly, topical antibiotics or cohousing of the Flg-deficient mice to alter or intermix the skin microbiota resolved the skin inflammation and restored IL-1α localization (Archer et al., 2019). This work has implications for AD pathogenesis and potential therapeutic targeting.

The final speaker of the symposium was Dr Shruti Naik who discussed choreographing of immune responses at the cutaneous interface. Dr Naik showed how commensal bacteria affect skin immunity and the critical cellular mediators involved. Furthermore, her work demonstrated that tissue-resident cells are poised to sense and respond to alterations in microbial communities, likely representing an evolutionary means by which the skin immune system uses fluctuating commensal signals to calibrate barrier immunity and protection against invading pathogens (Naik et al., 2015). Her work reveals the highly dynamic interactions that occur in the skin between the host and the microbiota and how these can be rapidly and specifically remodeled by encounters with defined commensals.

Translational roundtable
The final event of the meeting was the translational roundtable, where well-established members from academia, the National Institute of Health (NIH), and industry gathered together for a highly stimulating and interactive discussion with the audience about the future of skin research. This included a very lively discussion about the role of industry and academia in driving and prioritizing research areas and how these are shaping the future of cutaneous research and the role of the NIH to support and promote the field. It was generally agreed that the key to effective translation is to continue to advance our understanding of disease biology.

Summary and Perspectives:
Over 4 days, this symposium brought together 91 scientists, clinicians, and trainees (Figure 2) in a highly interactive and stimulating environment, with participants taking with them new connections and knowledge as they returned to their homes. The meeting emphasized how much research in common yet genetically complex skin diseases has moved toward the integration of genetics, genomics, and disease biology and outlined the opportunities and challenges ahead. As beautifully summarized by our keynote speakers, the meeting was highlighted by advances in the role of chromatin structure in gene regulation, the single-cell revolution, and the interaction of genetics and environment in maintaining the balance between response to pathogens and autoimmunity. These concepts provide important perspectives for the road ahead; whereas we should not expect that changing our genes will cure these diseases, we should expect that the insights provided by traditional GWAS will serve as a Rosetta Stone for leveraging the potential of epigenetics to regulate gene expression, for continued development of targeted immunotherapies, and for therapeutically exploiting the complex relationship between host defense and autoimmunity.
Data availability statement
No datasets were generated or analyzed in the process of preparing this meeting review.

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CONFLICT OF INTEREST
The authors state no conflict of interest.

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