National Eczema Association

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ROADMAP TO A NEW ERA OF CARE: THE BURDEN OF ECZEMA

NATIONAL Eczema ASSOCIATION

IN THIS ISSUE

MISSION

The National Eczema Association (NEA) improves the health and quality of life for individuals with eczema through research, support, and education.

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nationaleczema.org

National Eczema Association is a national nonprofit patient-oriented organization dedicated to eczema education and research. The association was founded in 1988 in Portland, Oregon, by individuals with eczema, nurses, physicians, and others concerned with the enormous social, medical, and economic consequences of this disease. The association is supported by individual and corporate donations.

YOU + NEA = PARTNERS *in* CHANGE

Dear NEA Friends:

As we near the close of 2015, I've been reflecting on this past year at NEA, and am amazed and so very proud of all we've accomplished in such a short period of time. I'm especially excited to see our strategic plan, ***The Decade of Eczema: The Roadmap to Advocacy***, come to life and begin to expand NEA's research, advocacy, and education efforts to benefit more individuals and families who suffer from eczema.

Thanks to your support, NEA is poised as never before to act as a catalyst and leader in ushering in a new era of patient care — and 2015 is only the beginning. And, it's a great beginning. This year's accomplishments include:

- Presenting the Leaders in Eczema Educational Forums
- Hosting the Itching for Cure Walks this past May and October
- Testifying in front Federal Drug Administration (FDA)
- Funding research to advance effective and affordable treatments
- Producing an "Exposing Eczema" campaign for October Eczema Awareness Month, reaching over 7 million people
- Advancing opportunities to participate in clinical trials
- Bringing on two new staff to engage more of the eczema community

These are only the first steps on the path toward erasing the stigma of eczema, advancing impactful research, and ensuring access to effective and affordable eczema treatments.

Let's continue to lift up our voices together. **Join us by making a year-end financial contribution today in support of NEA's Roadmap**, ensuring a successful road to better care where patient needs — and patient voices — are front and center. Working together, we will continue to move this life-changing work forward.

You can make a contribution by December 31, 2015 by using the envelope included in your Advocate magazine, or by visiting: donate.nationaleczema.org

Thank you for all you do to support NEA.

With deep appreciation,

Julie Block
President & CEO

WELCOME GRETCHEN HORTON-DUNBAR

NEA's new Vice President, Expansion

The National Eczema Association (NEA) is pleased to introduce Gretchen Horton-Dunbar as NEA's Vice President for Expansion. Eczema care today is undergoing a dramatic transformation. With more than 40 new treatments in development, there is great potential for real, practical, and game changing impact for eczema patients. As the new Vice President for Expansion, Gretchen will take the lead on leveraging these opportunities by forging new relationships to build NEA's visibility, cultivating an engaged community of individual and philanthropic supporters, and increasing our positive impact. Her work will ensure that NEA will continue to expand our efforts to improve the health and quality of life for individuals with eczema.

Gretchen has been an active supporter of nonprofit organizations since elementary school in Pennsylvania, when she had a memorable experience raising funds to purchase a cow for a family through Heifer International. That positive experience made a lasting impression that she credits with shaping her career; it eventually led to Oregon in 1998, where Gretchen worked for her first nonprofit as a campaign manager for the Working Families Party.

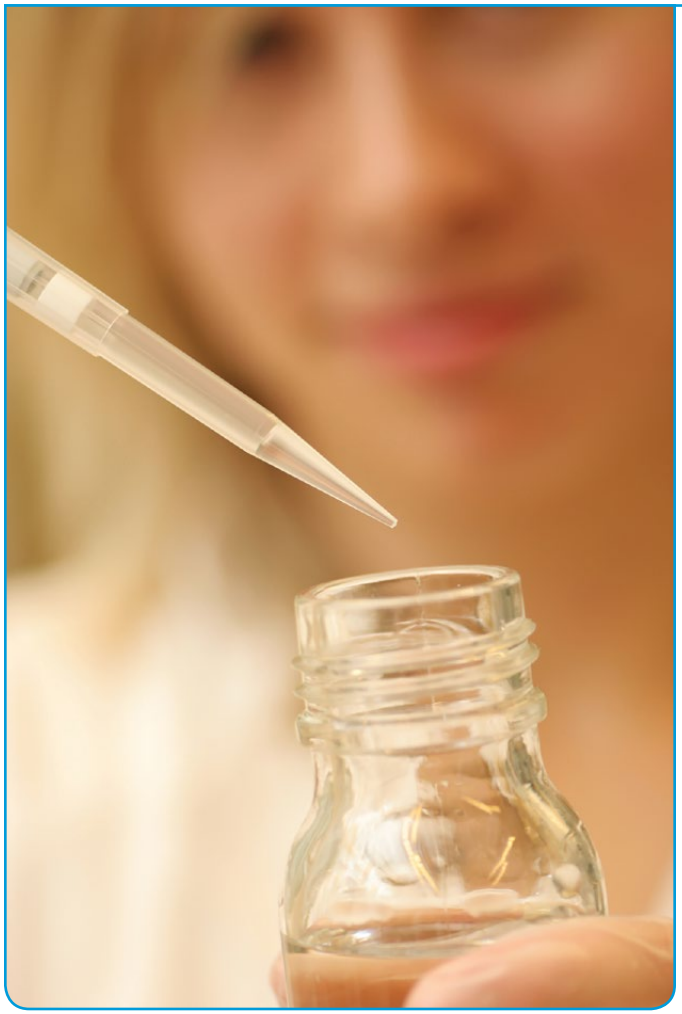
Over the last seventeen years since, Gretchen has continued to support mission-driven organizations by serving in leadership roles responsible for developing and advancing communication, community engagement, and development strategies. Most recently, from 2010 to 2014, Gretchen worked as the Director of Community Relations and Giving at Oregon College of Oriental Medicine. In fall 2014, she launched Sikhara Group, a consulting firm that specializes in helping health-oriented organizations increase their community impact and development success. As the principal consultant, Gretchen worked with both nonprofit and for-profit organizations, including the Oregon Public Health Institute and Oregon Reproductive Medicine, to advance their missions and develop sustainable funding for their futures.

Gretchen has a BA in English literature from Ohio University, and is currently working on a master's degree in public health. She's an avid hiker, yogi, and trail runner who loves to garden, cook, and read. She has two adult stepdaughters and a granddaughter, and is a frequent visitor to Georgia and southern Oregon, where the Horton-Dunbar families live.

Join us in welcoming Gretchen!

LEARNING MORE ABOUT ECZEMA — BIOMARKERS

by Adam Friedman, MD, FAAD



Eczema research continues to shed new light on several aspects of this chronic condition. NEA asked Dr. Friedman to help us understand a bit more about a particularly exciting aspect of emerging science.

What are biomarkers? Why are they important?

Biomarkers are reproducible and measurable indicators that can allow one to examine or follow biological activity or function over time. Biomarkers can be categorized in different types depending on their specific characteristics. They can be used to identify the risk of developing a disease, to diagnose a disease, predict disease progression, mark a particular response to an intervention (i.e. lifestyle change such as exercise, moisturizer use, diet), and for monitoring disease activity and clinical response to medication. This has HUGE implications in medicine, as identifying biomarkers can not only help us better understand how diseases develop, but also improve our ability to diagnosis a condition, follow its activity and severity, and monitor whether a medication is working or not working. In sum, biomarkers allow for a personalized approach, or what has become known in some respects as personalized medicine.

What will identifying biomarkers do?

Biomarkers can be used for many different purposes, including an objective (rather than observationally subjective) evaluation of disease severity, confirmation of clinical diagnosis, and to predict response to treatment. More importantly, understanding the importance of a biomarker in disease progression and severity could help identify NEW treatments based on said biomarker, a paradigm shift which has dominated the drug development world in oncology for over a decade.

What is exciting about new biomarker identification in relationship to eczema?

Eczema even today remains a clinical diagnosis without an objective approach for confirmation. Not all eczema is created equal, and while exceedingly common, for many years it was almost like an orphan disease given the limited advances made in treatment and management. This is all changing and biomarkers are central to this exponentially growing field of study. Identifying biomarkers will no doubt play an important role in research and personalized medical approach given the variation in disease severity from person to person. The use of biomarkers will enhance the success of treatment by creating therapies that target the patient's specific biological signature as well as help the physician predict and follow response to said medication. Many biomarkers are currently being studied, derived from different sources (blood, saliva, etc.), to develop the best and most accurate way to evaluate any and all sufferers of eczema.

What does this mean for eczema patients?

This means don't give up hope because we entering a new era! The same enthusiasm and drive to better understand and treat psoriasis over the past 20-30 years has overwhelmed the world of eczema. Multi-institutional teams are working together to identify panels of biomarkers

which can be used to better characterize unique differences in eczema individuals, age ranges, sexes, you name it. These same biomarkers being studied are being used to select the best and more effective treatments.

What can patients do to help this research along?

As of right now, the use of biomarkers to characterize and monitor eczema is not recommended as the science is still somewhat in its infancy. No question, advancing this science is of the utmost importance for EVERYONE and therefore this is a team effort. Patients can get involved with patient advocacy organizations, such as the National Eczema Association, to increase awareness, fundraise for research grants, participate in research studies, petition both local and federal government to make this work a priority. We are all in this together: patient, physician, scientist, etc.

Adam Friedman, MD, FAAD is an Associate Professor of Dermatology and serves as Residency Program Director and Director of Translational Research in the Department of Dermatology at The George Washington University School of Medicine & Health Sciences. Dr. Friedman is currently investigating novel nanotechnologies that allow for controlled and sustained delivery of a wide spectrum of physiologically and medicinally relevant molecules, with an emphasis on treating infectious diseases, accelerating wound healing, immune modulation, and correcting vascular dysfunction. He holds several patents derived from these investigations, and has published over 130 papers/chapters and 2 textbooks on both his research as well as a variety of clinical areas in dermatology. Dr. Friedman is also committed to resident and medical education. He currently serves on the AAD Sulzberger Committee on Education as well as the Poster Task Force, and is the Senior Editor of the Dermatology In-Review Online Workshop and Director of the Oakstone Institute Dermatology Board Review. He has received awards from multiple organizations such as the American Dermatologic Association, American Academy of Dermatology, and The American Society for Dermatologic Surgery.

THANK YOU!

Dear Isaiah's Fund Supporters:

Just like you, eczema keeps me from doing things that other people enjoy doing, like going to the beach, riding my bike, and just plain enjoying being outside.

But, I have new hope this won't always be true thanks to your generous gifts to "Isaiah's Fund" last spring.

NEA's Isaiah's Fund isn't just about me or my story — I was glad to share a glimpse into my life with eczema with the Food & Drug Administration (FDA) Advisory Panel in March, and with you. I now really do have hope that better medicines are on their way, and someday a cure!

Thank you for raising more than $5,000 to support NEA's work — you are helping make life better for all of us with eczema.

Sincerely/with gratitude,

Isaiah Dixon

Isaiah Dixon, Age 13
NEA member

*P.S. You can still support Isaiah's Fund — visit **nationaleczema.org/donate** to contribute today!*

Your Gifts in Action

Last spring, after Isaiah Dixon and Gracie Nye's compelling testimony at a Food & Drug Administration (FDA) hearing helped urge the FDA to move appropriate clinical drug trials for children forward, NEA asked you to support our mission by donating to "Isaiah's Fund." Thirty-one of you generously responded, donating a total of $5,274 in support of our efforts — thank you!

Thanks to your support:

- **NEA is taking the next steps toward ensuring clinical trials for promising new therapies continue to be possible, including break-through treatments for children suffering from eczema**. One example includes partnering with the Pediatric Dermatology Research Alliance, PeDRA, to draft a "Food & Drug Administration (FDA) Guidance Document" to direct drug manufacturers and government on the recommended approach and methods of these critically needed trials.

- **NEA has launched an exciting and ambitious strategic plan — The Roadmap for Advocacy — to empower, mobilize and engage the eczema community to improve the lives of patients and their families**. Read more about our Roadmap to Advocacy and the Decade of Eczema at nationaleczema.org

- **We've inspired others to donate to Isaiah's Fund and become active participants in the path toward better treatments and a cure**. Community support like yours allows NEA to fulfill our mission to improve the quality of life for individuals with eczema through research, support, and education. Thank you!

NEA BRINGS *the* VOICE *of* ECZEMA *to* CAPITOL HILL

to Protect and Enhance Patient Care

New treatments for eczema are on the horizon and the National Eczema Association (NEA) has been busy this Fall advocating to ensure these new therapies are accessible and affordable to those who need them when they become available. In September, NEA's CEO Julie Block and Vice President for Advocacy and Access Amy Fauver joined more than 130 dermatologists and members of the Coalition of Skin Diseases on Capitol Hill to advocate for policies that protect and enhance patient care.

Attendees from 35 states held 216 meetings with Members of Congress, Senators and key Congressional staff as part of the American Academy of Dermatology Association's (AADA) annual Legislative Conference. NEA's message on Capitol Hill focused on urging Congress to enhance access to care and treatment, and speed medical discovery through sustained federal investment in research. Specifically, NEA focused on the following legislative asks:

- **Co-sponsor the Patients' Access to Treatment Act**, introduced by Reps. David McKinley (R-W.Va.) and Lois Capps (D-Calif.), which limits out-of-pocket costs for new specialty drugs (typically Tier III) and diminishes cost barriers to effective medications.
- **Co-sponsor the Medicare Advantage Bill of Rights Act**, to be introduced by Rep. DeLauro (D-Conn.) and Sen. Sherrod Brown (D-Ohio), which improves transparency in the Medicare Advantage (MA) insurance market by prohibiting MA plans from removing patient's doctors mid-year and ensuring continuity of care requirements are satisfied when a provider is terminated from a network plan.
- **Support the upcoming Senate version of the 21st Century Cures Act** (passed by the House of Representatives last summer), which increases medical research funding to promote new cures and treatments.

In October, NEA's Vice President for Advocacy and Access Amy Fauver traveled back to Washington D.C. to attend the annual National Institute of Arthritis Musculoskeletal and Skin (NIAMS) Diseases Coalition meeting. NIAMS is part of the National Institute of Health, which is the primary federal agency conducting and supporting basic, clinical, and translational medical research, and is investigating the causes, treatments, and cures for both common and rare diseases.

NEA's Amy Fauver and other members of the Coalition of Skin Diseases' Oregon delegation meet with Senator Ron Wyden.

The NIAMS Coalition is an independent consortium of professional associations and patient advocacy organizations that raise awareness about NIAMS research into the basic understanding, causes, incidence, treatment, and prevention of diseases, including those of the skin. NEA's membership in the NIAMS Coalition allows us to serve as the voice of the eczema patients for whom NIAMS works.

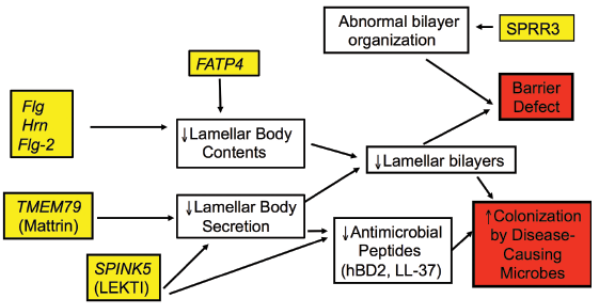
For more information about NEA's advocacy work or to get involved as an advocate in your community please contact Amy Fauver at **amy@nationaleczema.org**.

ATOPIC DERMATITIS UPDATE

AD is More Than One Disease

Peter M. Elias, M.D.
Department of Dermatology, UCSF, San Francisco, CA

Fig. 1: How Unrelated, Inherited Abnormalities Converge to Produce a Defective Permeability and Antimicrobial Barrier in Atopic Dermatitis (AD) (Yellow = Genes with mutations that predispose to AD)



Abbreviations: Fatp4, fatty acid transport protein 4; Flg, filaggrin; hBD2, human beta-defensin 2; Hrn, hornerin; KLK, kallikrein; LEKTI, lympho-epithelial Kazal-type trypsin inhibitor (modified from Elias & Wakefield, Ref 2 below)

It is widely recognized that the severity of atopic dermatitis (AD) can range from mild-to-moderate-to-severe, even though all patients demonstrate common features of dry, itchy and inflamed skin. Yet, while it is customary to consider it as a single disease of varying severity, AD is actually an umbrella term for a family of inherited disorders, often due to completely different mutations (Fig. 1). The type of mutation can also provide clues as to why disease severity varies from patient-to-patient. Best known and most closely associated with AD are mutations in the gene (*FLG*) that encodes the protein, filaggrin. Filaggrin (FLG) is a key contributor to the integrity of the permeability barrier. AD patients who carry two *FLG* mutations tend to have more severe disease than do patients who have inherited only one *FLG* mutation, and these so-called double-allele patients also often develop problematic dry skin and a variety of 'eczema' as adults. But it turns out that *FLG* mutations occur much more commonly in patients of northern European ancestry, than in AD patients of either Asian or African-American extraction. These populations usually have other associated mutations (Fig. 1).

Despite abundant evidence that darkly-pigmented skin displays superior barrier function in comparison to lightly-pigmented skin[1], African-Americans can exhibit very severe AD. One might ask: how could they have severe AD, if dark skin has a better barrier? In contrast to AD patients of northern European extraction[1], AD in African-Americans is often linked to another gene (*FLG-2*), which encodes a protein called filaggrin-2 (FLG-2) (Fig. 1). While FLG-2, like FLG, also impacts barrier function, it does so by unrelated mechanisms.[2] Hence, AD in African-Americans displays a different inherited basis, which could explain, at least in part, the more severe form of AD that can occur in these patients. Alternatively or in addition, many African-Americans live in crowded urban, rather than in dispersed, rural settings, which can favor the development of more severe AD[3]. AD patients that live in crowded urban settings are not exposed to environmental antigens that induce immune tolerance. Nor do they receive as much ultraviolet-B light exposure, which at moderate doses enhances barrier function and antimicrobial peptide production. In addition, AD can emerge under crowded conditions due to prolonged dust mite exposure. Finally, the prevalence of secondary bacterial infections, a common AD 'trigger,' also could be higher in many African-Americans due to more crowded living conditions. Thus, both genetic and cultural differences could account for the severe AD that occurs in some African-Americans, despite the fact that dark pigmentation in general confers a better barrier.

Then, there are AD patients with mutations in a group of enzymes (proteases or their inhibitors) that regulate stratum corneum shedding. For example, the rare inherited disorder, Netherton syndrome, is due to mutations in *SPINK5*, which provoke a form of AD that is usually much more severe than is AD associated with FLG mutations. In Netherton syndrome, the deficient gene product is a protease inhibitor (LEKTI), which when absent, unleashes a flood of proteases that provoke a particularly severe form of AD[4] (Fig. 1). Thus, AD patients with a wide variety of seemingly unrelated mutations can exhibit eczematous skin changes, including often severe itching, and an increased tendency to become colonized by pathogenic bacteria, particularly antibiotic-resistant strains of *Staphyloccus aureus*.

How can we explain that totally-unrelated mutations produce a similar (i.e., AD-like) clinical picture (or 'phenotype')? In a recently-published perspective in the

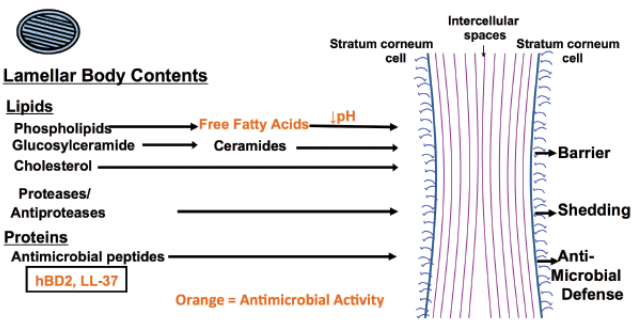


Fig. 2: Multifunctional Impact of Secreted Lamellar Body Contents

J. Allergy Clin Immunol (http://www.ncbi.nlm.nih.gov/pubmed/25131691)[2], we point out that these seemingly unrelated mutations all converge on (and compromise) the lamellar body secretory system (Fig. 1). Lamellar bodies are small, ovoid, subcellular particles or 'organelles' that deliver the lipids (fats) and proteins needed for a competent barrier to the outermost layers of skin, the stratum corneum. These lipids lie between the cells of the stratum corneum, where they form a repeated series of membrane sheets that provide the skin barrier (Figure 2). Once positioned there, these lipids and proteins regulate at least 3 key skin functions: 1) the permeability barrier; 2) antimicrobial defense; and 3) the ultimate shedding of stratum corneum cells from the skin surface[5]. In other words, patients with AD have inherited a variety of different mutations, but in all AD patients, the disease displays a related pathogenesis — either defective delivery or premature degradation of lamellar body contents necessary for the permeability barrier, antimicrobial defense and normal desquamation (shedding). Together, these observations support the deployment of barrier repair therapy, a logical step to overcoming the genetic predispositions that converge to produce the clinical features of AD.

References:

1. Gunathilake, R, Schurer, N Y, Shoo, B A, et al. pH-regulated mechanisms account for pigment-type differences in epidermal barrier function. J Invest Dermatol 2009;129:1719-29.

2. Elias, P M, and Wakefield, J S. Mechanisms of abnormal lamellar body secretion and the dysfunctional skin barrier in patients with atopic dermatitis. J Allergy Clin Immunol 2014;134:781-791 e1. 4186911.

3. Thyssen, J P, Zirwas, M J, and Elias, P M. Potential role of reduced environmental UV exposure as a 'driver' of the current epidemic of atopic dermatitis J Allergy Clin Immunol 2015;In press.

4. Hachem, J P, Wagberg, F, Schmuth, M, et al. Serine protease activity and residual LEKTI expression determine phenotype in Netherton syndrome. J Invest Dermatol 2006;126:1609-21.

5. Elias, P M. Structure and function of the stratum corneum extracellular matrix. J Invest Dermatol 2012;132:2131-3.

Dr. Elias is Professor (Emeritus) of Dermatology at UC San Francisco, and a Staff Physician at the San Francisco Veterans Administration Medical Center His laboratory is located at the new UCSF Medical Center at Mission Bay. Dr. Elias earned his undergraduate degree at Stanford University and then went to UCSF Medical School, followed by a dermatology residency in the Harvard-Massachusetts General Hospital program.

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** Eczema Area Severity Index*

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1. Data on file.

RESEARCH UPDATE

Your Dollars at Work to Understand the Burden of Eczema as a Disease

You know all about the struggles of living with eczema and the burdens of eczema as a disease: the constant itch; the difficulty of trying to manage work or school — and be your best — after another sleepless night; the high cost of needed medications and over-the-counter products that make a big dent in your budget and your lifestyle. The list could go on and on. Today, due to your financial support, NEA is one step closer to understanding the burden of eczema as a disease, through an exciting new NEA research grant award.

The challenges of eczema impact patients, families, the healthcare system, schools, places of work, and society as a whole. But, as you know, most people don't understand what it really means to have eczema and there are gaps in the data needed to document the burden of eczema as a disease. And that burden of disease data is critical to NEA's success in implementing the **Roadmap to Advocacy** to raise the profile of eczema as a serious disease, to deliver stronger, more united messages to insurers, medical providers and policy-makers on behalf of the eczema community, and to build the case to focus more funding on research for better eczema treatments and an eventual cure.

NEA is pleased to announce an exciting new research grant award to audit the existing burden of disease literature in both pediatric and adult eczema/atopic dermatitis. NEA has awarded the grant to investigator Aaron M. Drucker, MD, FRCPC, DABD, and co-investigators Abrar Quereshi, MD, MPH, DADB, Annie Wang, MD, and Wen-Qing Li, PhD, of Brown University for their "Burden of Eczema*" study.

The "Burden of Eczema" project will encompass a review of eczema co-morbidity, quality of life, family impact, healthcare impact, and society impact data. The study will also identify where the gaps in evidence are, helping to clarify where upcoming research should focus next. This will inform NEA's next targeted research strategies and will be of benefit to everyone in the eczema research field.

The need for more research, and to demonstrate the burden of eczema as a disease is clear. As outlined in NEA's **Roadmap to Advocacy**, NEA aims to be a steward of burden of disease research by serving as a clearinghouse for this type of research that can be readily shared with individuals and organizations interested in improving the lives of eczema patients.

Drive more progress! Grow and advance eczema research with NEA today — online at nationaleczema.org or with the remit envelope included in this magazine.

** Meet Dr. Drucker and learn more about the "Burden of Eczema" study on page 14.*

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NEA'S LATEST RESEARCH AWARD:

An Interview with Dr. Adam Drucker

Dr. Aaron Drucker is joining the Department of Dermatology at Brown University in Providence, RI as an Assistant Professor. He completed his dermatology residency at the University of Toronto after graduating from medical school at Queen's University in Kingston, Ontario. Dr. Drucker's clinical and research focus is atopic dermatitis, with his research specifically concentrating on the epidemiology and burden of disease of eczema.

How did you get interested in eczema? Why do you find this disease of particular interest?

Eczema is a skin disease that has a major impact on patients and their families. It is very satisfying to treat patients with eczema and see the difference that can be made with treatment. From a research perspective, it is incredibly interesting as it is a very common disease with many questions left to be answered about it. The field is changing rapidly and as a result we expect some exciting new treatments for eczema to be available in the near future.

What is "burden of disease data" and why is it important?

"Burden of disease" is a broad concept that encompasses the impact a disease has at an individual and population level. It includes things like quality of life, time off work or school due to the disease, the cost for patients and their families to manage the disease, and the cost to the health care system, among other things. Burden of disease data is incredibly important as it demonstrates to health care practitioners and policy makers how much of an impact a disease has on patients and society at large. If a disease like eczema is shown to have a large burden, that should lead to increased resources being put into more eczema research and better care for patients.

Tell us about your National Eczema Association "Burden of Eczema" project — what is your goal, what will you be auditing, and how will you proceed?

The goal of the project is to demonstrate the burden of eczema in the U.S.; to identify how much of an impact eczema is having on U.S. patients, their families, and the country as a whole. My research team and I will systematically audit existing medical literature, looking for studies on the impact eczema has on quality of life for patients and families and the economic burden of the disease, including medical costs and other costs such as time missed from work or school due to eczema. We will also employ social media in our study.

How can social media help this process?

Data published in the medical literature doesn't always do a great job of reflecting patients' and families' experience with a disease. We want to search social media (such as Facebook and Twitter) to learn about what people affected by eczema are saying about it directly. The question is, are there aspects of the burden of eczema from the patient perspective that have been missed by the medical and scientific communities? The answer is probably yes. We aim to explore this, ensuring that patient and caregiver voices are included in our research.

Can you explain for readers a little more about the following areas of eczema burden of disease and what questions will be considered in each of these areas in the audit?

COMORBIDITIES

Patients with eczema are more likely to have a variety of other medical conditions compared to people without eczema. These associated conditions are called comorbidities and they include asthma, hayfever, and food allergies as well as being overweight and having mental health conditions such as depression and anxiety. We will search the medical literature looking for studies that examine the rates of other conditions in patients with eczema compared to the general population.

QUALITY OF LIFE

Eczema can effect people's lives in many ways, from disturbed sleep to embarrassment about the way their skin looks to limitations in physical activities patients can comfortably participate in, and many more. We aim to search for data on the impact eczema has on quality of life, and to compare this impact to other diseases, including other skin diseases.

IMPACT ON THE FAMILY

Not only does eczema affect the quality of life of patients, but it can also adversely affect the family unit. Caregivers of children with eczema may need to devote considerable time and money caring for them. Caregivers and partners of children and adults with eczema, respectively, can also have their sleep disturbed. If patients limit their social or physical activities, that can limit the rest of the family as well. All of this can lead to significant cost and stress for a family unit. We will look for data that demonstrates these impacts.

IMPACT ON HEALTHCARE

Eczema is a condition that often requires the use of significant health care resources. Patients may visit their health care practitioners, both general practitioners and dermatologists, many times a year. Patients need to buy medication on an ongoing basis to keep their eczema under control and manage flares. Severe eczema flares and infected skin sometimes require visits to the emergency room or stays in hospital. We will search for data on how patients with eczema use the health care system and what the cost of this use is.

IMPACT ON SOCIETY/ECONOMIC BURDEN OF ECZEMA IN THE U.S.

The costs of eczema go beyond what patients, insurance companies and other payers spend directly on treating eczema. When patients miss work or school because they are having an eczema flare, that has an impact. When patients can't concentrate on their work because they didn't get a good night's sleep or their itch is distracting, that has an impact. We will search for data on these and other aspects of the indirect costs of eczema.

How can eczema burden of disease data help: patients, clinicians, researchers?

Burden of disease data can provide context for patients and their families. It can demonstrate to them that their experience is shared with others across the country and across the world, and that the large burden they feel having to cope with eczema is acknowledged by the medical and scientific community. More importantly, though, demonstrating that eczema has a significant burden should lead to increased resources to care for patients with eczema and to fund research to better treat eczema, ultimately decreasing its burden on patients and their families.

Burden of disease data helps clinicians better understand what their patients are experiencing. This allows clinicians to better communicate with their patients and to provide care better suited to their needs.

Burden of disease data is important for researchers to understand the scope of the disease. It also demonstrates the importance of eczema within medicine and society at large which can lead to increased attention to and improved funding for eczema research.

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GENETIC FRONTIERS *in our* UNDERSTANDING *of* ECZEMA

by Monica Enamandram, MD

Itching is one of the hallmark symptoms of a number of skin diseases. Chronic itch can cause substantial suffering and unfortunately affects millions of Americans. Although a variety of treatments are available for itch, nonetheless this symptom can be difficult to treat for both patients and providers. A significant clinical and research interest in dermatology is aimed at understanding the molecular basis of this process. Through genetic mouse models, mammalian studies, microneurography and other studies, recent advances have helped identify the mediators of itch. These have led to insights that are continuing to advance our understanding of this complex process.

This June, an exciting development in this field emerged as a result of a study performed by scientists in the Buck Institute for Research on Aging and the University of California, Berkeley, published in the early online version of the journal Neuron. Through collaboration between Diana Bautista PhD, Rachel Brem PhD, and colleagues, the study identified a serotonin receptor (HTR7) as a key mediator of itch. To begin this discussion, the first sections of this article will detail our understanding of the biology and mechanism of itching. After providing this framework, the second section of this article will shift to describe this groundbreaking work published in Neuron, and its clinical implications for atopic dermatitis.

The diversity of itching

The phenomenon of itching, known as pruritus, can be subdivided into one of four clinical categories. *Neurogenic itch* refers to a process that is generated in the central nervous system in response to pruritogens (substances that elicit an itch and the urge to scratch in the skin) but without underlying abnormalities of the nerves themselves. Conversely, *psychogenic itch* is derived from a psychological disorder. The third subtype, *neuropathic itch*, is complex and thought to be derived from neuronal damage or dysfunction that can result in itch in the absence of noxious stimuli. This type of itch may be accompanied by other abnormalities of sensation, such as numbness, tingling, or excessive physical sensitivity of the skin.

Finally, *pruritoceptive itch* is the fourth subtype of itch, which is one of the most common symptoms experienced by patients who present to the dermatology office. This process originates in the skin owing to either inflammation and/or skin damage. Inflammation can occur following a number of potential internal stimuli or external exposures (such as dust, clothing fabrics or bugs), which produces itch.

Regardless of the origin of an itch, a universal behavior results. As soon as we feel an itch, our first natural response is to scratch the affected area. The reason for this response is simple — we want to remove the irritant as soon as possible.

The biological underpinnings of itch

There are two theories as to how pruritoceptive itch is transmitted from the skin to the brain. The first, the "labeled line" theory suggests that there are itch-specific nerve fibers. These fibers are thought to extend through one or more connections in a circuit that begins in the skin and transmits to the spinal cord, and ultimately to the central nervous system.

The second "selectivity theory" posits that there are nerve fibers that send itching signals to the brain, which are also responsible for transmitting pain signals. Itching and pain, interestingly, share a number of commonalities. For example, the neurologic pathway that transmits both of these sensations in the spinal cord is shared, known as the spinothalamic tract. Brain areas activated by pruritus have also been implicated in the processing of pain in the central nervous system.

According to the selectivity theory, a specialized population of nerve fibers exists in the skin, the majority of which will only respond to pain. Yet a select subset exists that can respond to both itch and pain. The pain-related nerves are thought to inhibit the itch-sensitive nerve fibers. Therefore itch can occur only when itch-sensitive nerve fibers are selectively activated. In other words, if a stimulus triggers both itch- and pain-sensitive nerve fibers then the sensation of pain will predominate, masking the itch. This theory is supported by the observation that severe pain and itch are not simultaneously perceived. Moreover, the notion of pain as an inhibitor of itch can be translated to the fact that we can soothe an itch through scratching (theoretically triggering pain perception).

Various pruritogens interact with molecular detectors in the skin, which can be found on nerve fibers of the skin and cells in the superficial layer of the skin (epidermis) known as keratinocytes. These molecular detectors set off a cascade of events to transmit an itch signal. Their mechanisms are diverse and have attracted ongoing interest as potential drug targets to treat itch. For example, ion channels are a type of molecular detector that have been shown to transmit signals related to itch – among ion channels, the transient receptor potential (TRP) family has been extensively studied and shown to have an important role in certain pathways that transmit pruritoceptive itch.

New developments in an exciting field

Owing to a rapidly growing body of research, the field of itch has significantly expanded in recent years. Scientists have discovered a number of mediators that are thought to play a role in pruritoceptive itch. Histamine classically has been associated with this type of itching. Yet the fact that antihistamines are sometimes only a partially effective treatment is well recognized. This among other reasons led to the investigation of other mediators that could be potential drivers of these distressing symptoms.

Substances that are independent of histamine such as tryptase, cowhage, cathepsin S, kallikreins, cockroach and dust mite protease allergens have been shown to stimulate itch via a molecular detector known as protease-activated receptor-2 (PAR2). Other immune system-mediated factors such as interleukin-31, leukotriene B4 and Substance P have also been implicated in pruritoceptive itch. Ultimately, the diversity of these findings is a testament to the complexity and multifactorial biological origins of itch.

The role of serotonin

Serotonin is a neurotransmitter, which refers to a chemical produced by the body that enables brain cells and other nervous system cells to communicate with one another.

Serotonin is another potent inducer of itch, whose role is becoming increasingly elucidated. In humans, abnormal serotonin signaling has been linked to itch in atopic dermatitis (commonly known as eczema), as well as other disorders such as allergy, renal failure, hepatobiliary disorders, and psoriasis. In a study led by Zhou-Feng Chen PhD and colleagues at the Washington University School of Medicine Center for the Study of Itch, serotonin was first implicated in itch. As part of the study, Dr. Chen's research team genetically engineered a strain of mice that lacked the ability to make serotonin. When these mice were injected with a substance that normally produces itch in the skin, they demonstrated a reduced degree of scratching compared to their genetically normal littermates. After injection with serotonin, however, the genetically engineered mice scratched at a frequency that would be expected in response to the itchy stimuli.

Ultimately, the researchers demonstrated that a lack of serotonin expression corresponded to a reduced proclivity in mice for scratching. Although fascinating, the potential translation of this finding to clinical treatment would be unfortunately limited – serotonin is involved in various processes in the body, including aging, growth, bone metabolism, circadian rhythm, and mood regulation. Thus broad, non-specific serotonin blockade would have consequences on a number of physiological processes.

But further experiments were performed to investigate the communication between serotonin and spinal nerve cells that specifically transmit itch. Dr. Chen's research team successfully isolated the receptor used by serotonin to activate the itch-specific neurons, 5HT1A. Moreover, they blocked this specific receptor and found again that mice scratched with reduced frequency.

In this most recent study conducted by Drs. Bautisa and Brem's research teams, researchers examined the available scientific literature to identify genes whose expression correlated with itch behavior in experimental strains of mice. In doing so, the serotonin receptor HTR7 was isolated as a potential molecular candidate to study: mice that expressed the highest degree of HTR7 in nerve cells in the skin were also the ones most affected by itch.

A panel of follow-up experiments were performed that demonstrated how activation of HTR7 led to molecular signaling through TRPA1 (a member of the TRP family of ion channels that have been well-characterized in triggering itch). In a subsequent experiment examining a mouse model of atopic dermatitis, mice that lacked HTR7 or TRPA1 both displayed reduced scratching behaviors. In fact, their skin lesion severity was also reduced compared to equivalent mice with intact HTR7 and TRPA1 genes.

Future directions:

It is promising that altered serotonin signaling has been identified in a variety of dermatologic disorders such as allergy-induced itch as well as psoriasis. More broadly,

chronic itch is associated with systemic disorders as well — for example, kidney failure, cirrhosis and cancer — and can be equally as debilitating as it is for atopic dermatitis patients. It is of great interest that we expand our understanding of itch, as a common symptom for which effective treatments are limited, leading to frustration and suffering for affected patients. Thus the identification of HRT7 as a player in chronic itch, as led by the research teams of Dr. Bautista and Dr. Brem, is a major breakthrough in this field. Both HTR7 and the future directions now open for further research offer hope for the development of novel drug targets to treat chronic itch. For patients, this will ideally set the stage for greater symptom control and improved quality of life for patients who suffer from eczema and other conditions.

Sources:

Garibyan L, Rheingold CG, Lerner EA. Understanding the pathophysiology of itch. Dermatol Ther. 2013 Mar-Apr; 26(2):84-91.

Morita T, McClain SP, Batia LM, et al. HTR7 mediates serotonergic acute and chronic itch. Neuron. 2015 Jul 1; 87(1): 124-38. Doi:10.1016/j.neuron.2015.05.044. Epub 2015 Jun 11.

Potenzieri C, Undem BJ. Basic mechanisms of itch. Clin Exp Allergy. 2012 Jan; 42(1):8-19.

Zhao ZQ, Liu XY, Jeffry J, et al. Descending control of itch transmission by the serotonergic system via 5-HT1A-facilitated GRP-GRPR signaling. Neuron. 2014 Nov 19;84(4):821-34.

Monica Enamandram, MD is a resident physician in the Department of Dermatology at Stanford University Hospitals. She is a graduate of Harvard Medical School, and is interested in patient education, advocacy, and clinical research in dermatology. Monica is excited to develop successful preventive measures and improved treatment regimens for eczema.

Scratch Pad

Dear NEA Scratch Pad,
I have 2 children that are 3 years old and 10 months old. They suffered with eczema so bad that especially my youngest was cracked and bleeding all over her body. A friend introduced me to a nutritional science company. The nutrition was a powder that you mix in a liquid. The scientist developed Enfamil baby formula. I tried it and in 2 weeks my kids were symptom free of eczema. I am now a happy mom who was prescribed medications and was very frustrated. However, I was able to battle eczema another way.

Gaylynn M.
West Valley City, UT

Dear NEA Scratch Pad,
I have suffered from eczema mostly on my hands, arms, legs, back and neck. It was incredibly itchy and uncomfortable. After trying a number of products, I decided to create my own moisturizer, using coconut, aloe vera, and guava. I use it after showering and carry it with me everywhere I go. It has helped soothe and soften my skin and even my rash went away!

Margaret M.

Dear NEA Scratch Pad,
My daughter (17 years old) has had hand eczema since third grade and we have never been successful in getting it under control. After reading everything I could, I stumbled upon the suggestion for olive oil soap. I purchased the soap from earthsoap.com and bought some cotton gloves to wear. We washed everything (towels, blankets, sheets, and clothing) with non-fragrant laundry detergent and started 'fresh.'

All these changes have made a huge difference! For the first time my daughter is not suffering and scratching all the time. She hasn't needed to use steroid creams and the open sores on her hands have healed. Even the tough skin on her hands have softened! The olive oil soap is my favorite change we have made and she keeps it with her at all times.

Karen H.
Salt Lake City, UT

Scratch Pad

Dear NEA Scratch Pad,
I am 26 years old and I have found that applying a thin layer of toothpaste to my eczema spots every night before bed helps. It dries quickly and stops the itch. I even noticed that the next morning after showering my skin looks better. I also have severe food allergies, which makes dining out difficult. I take 10mg of hydroxyzine HCL, which has changed my life. I finally feel like I'm in control of my eczema. I hope what has worked for me will help someone else.

Maggie L.
San Bernadino

Dear NEA Scratch Pad,
I started taking bilberry pills, an herbal supplement, after I read that it would help with my cataracts. Shortly after I started taking the pills, I noticed my skin was changing. The spots on my skin that used to cause so much itching started going away. My hair started growing back and my arms started to look normal. Best of luck to those who try bilberry pills!

Rose M.
Ann Arbor, MI

Dear NEA Scratch Pad,
No more red ears, no more red neck, no more red forehead! No more seborrheic dermatitis!

Researching seborrheic dermatitis, I found that it might be caused by too much yeast in the body. If you want to rid yourself of the yeast, you need to quit feeding the yeast. Additionally, I got a new pillow, washed my sheets everyday for a month, washed my hair everyday for a month with a dandruff shampoo and I used only petroleum jelly on my skin. What a difference this has made in my life.

Jill V.
Michigan

The recommendations contained in the "Scratch Pad" are those of the contributors. NEA provides health information from a variety of sources; this information is not intended as medical advice. Persons with questions regarding specific symptoms or treatments should consult a professional health-care provider.

Email your "Scratch Pad" tip (along with a photo if you have it) to info@nationaleczema.org, so that we may publish it in an upcoming issue of *The Advocate* and help others!

NEA SUPPORT NETWORK

As a lifelong eczema sufferer, Ashley Blua donated her time and professional public relations skills to work on our Itching for a Cure Los Angeles outreach. Ashley worked with the media, bloggers, and other Public Relations outlets to spread the word about IFAC.

Thank you Ashley for all your hard work in promoting Itching for a Cure!

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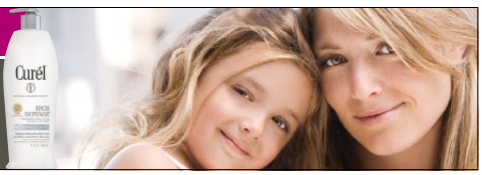

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