



AMERICAN ACNE & ROSACEA SOCIETY

2nd Annual Meeting Program



American Acne & Rosacea Society

2nd Annual Meeting

Friday, February 2, 2007

7:00—10:00 PM

The Occidental Restaurant
1475 Pennsylvania Avenue, NW
Washington, DC 20004



AMERICAN ACNE & ROSACEA SOCIETY

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PROGRAM

- 7:00 PM Registration and Cocktail Reception
- 8:00 PM **Introduction of Kligman Speaker**
Guy Webster, MD, PhD
- 8:03 PM **Perspectives on Acne and Rosacea**
Peter Pochi, MD
- 8:35 PM **Introduction of Scientific Program**
Diane Thiboutot, MD
- 8:40 PM **Rosacea and Innate Immune Defense**
Richard Gallo, MD, PhD
- 9:00 PM **Innate Immunity and Acne**
Jenny Kim, MD, PhD
- 9:15 PM **Gene expression in inflammatory acne**
Diane Thiboutot, MD
- 9:45 ***AARS Business Meeting***
- President's statement/ State of the AARS**
Guy Webster, MD, PhD
- Incoming President's Statement**
Hilary Baldwin, MD
- Announcement of Grants program**
Diane Thiboutot, MD and Sewon Kang, MD
- Election announcement**
Lee Zane, MD
- Bylaws Announcement**
Lee Zane, MD
- Election Results**
Sewon Kang, MD
- Adjournment.**



GENERAL INFORMATION

Verification of Attendance/Evaluation

You will receive evaluation forms for the meeting in your registration packet. Please complete the form and deposit them in the collection box at the registration desk following the meeting.

Appropriate credit for attendance should be ascertained and reported by individual physicians to the particular state or medical society to which he or she belongs. A certificate of attendance will be mailed to all registrants.

CME Credits

The American Acne and Rosacea Society Annual Meeting is recognized by the American Academy of Dermatology for 1.75 hours of AAD category 1 CME credit and may be used toward the American Academy of Dermatology Continuing Medical Education Award.

Program: 499100

AARS Membership

Membership in AARS is open to dermatologists, physicians, researchers and medical professionals with an interest in acne and Rosacea research and practice.

Disclosure Statement

The AARS requires balance, independence, objectivity and scientific rigor in all of its educational activities. The Board of Directors requires that all presenters and audience members comply with all applicable laws and regulations governing disclosure.

AARS Address

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ABSTRACTS

Kligman Lecture

Perspectives on Acne and Rosacea



Peter Pochi, MD
Professor Emeritus, Boston University School of Medicine

Acne and rosacea share a number of similar clinical characteristics, but with distinctive differences as well. Therapies directed toward both disorders are reviewed, with a particular look back to the 1950s, at which time treatments for them began to emerge from a state of comparative ineffectiveness into the antibiotic era. Tetracycline and successor antibacterial/anti-inflammatory drugs afforded for the first time a fairly predictable assurance of efficacy. Of interest in this regard has been a striking unanimity in the panoply of therapeutic agents for both acne and rosacea, which suggests a correlative relationship between the two, albeit one not likely of the first order.

Cathelicidins: A link between innate immunity and Rosacea

Richard L. Gallo, M.D., Ph.D.
Division of Dermatology, University of California, San Diego

The cause of rosacea is unknown, although its symptoms are exacerbated by vasoactive agents and improved by some antibiotics. Antimicrobial peptides (AMPs) are innate antibiotics produced in the skin and triggered by external stimuli similar to those that exacerbate rosacea. AMPs of the cathelicidin family are vasoactive, leading us to ask if they may be abnormal in rosacea. Punch biopsies and tape strip samples of rosacea patients demonstrated a large increase in cathelicidin. Furthermore, rosacea patients showed abnormal post-translational processing of cathelicidin by



SCTE, a protease that activates cathelicidins. Combined, this resulted in forms of cathelicidin in rosacea that are not detectable in normal human skin. Injection of mice with these AMPs at physiological concentrations mimicked aspects of rosacea but injection of peptides found in normal patients did not. Furthermore, targeted deletion of the cathelicidin gene from mice decreased inflammation following contact irritation, suggesting that cathelicidins not only act as antibiotics, but can mediate inflammation. Thus, we hypothesized that the etiology of rosacea involves the abnormal expression and processing of cathelicidin. Support for this hypothesis could be demonstrated in humans, as minocycline suppresses elevated protease activity while leading to decreased inflammation, and topical vitamin D induces cathelicidin and leads to increased inflammation. Combined, these findings provide a novel explanation for the pathogenesis of rosacea, suggest a link between innate immunity and this common skin disease, and provide alternative therapeutic strategies by limiting the processing or production of AMP.

Innate Immunity and Acne

Jenny Kim, MD, PhD
Chief, Dermatology
GLAHS VA

The role of the innate immune system is to rapidly recognize microbial pathogen and lead to destruction yet at the same time, the same innate immune response to microbes can also contribute to clinical inflammatory response that leads to disease state. We have used acne as a model to study the innate host response. Previously our study demonstrated that *P. acnes* induces inflammatory cytokine production through activation of TLR2. We now show that specific metalloproteinase, important in inducing inflammation and tissue remodeling, is also induced by *P. acnes* through TLR2 and that all-trans retinoic acid can inhibit this immune response. We also studied the host defense mechanisms against the bacterium and found that two key cells of the innate immune system, CD209⁺ macrophages and CD1⁺ dendritic cells could be rapidly induced by stimulation of peripheral blood monocytes with *P. acnes*. This correlated with the localization of CD209⁺ and CD1⁺ cells in acne lesions which suggests that the *in vitro* generation of these cell subsets is relevant to disease pathogenesis. Although both subsets of cells were activated by *P. acnes*, the CD209⁺ cells were found to phagocytize *P. acnes* and

mount a superior antimicrobial response. Our data suggest that the recognition of *P. acnes* by the host immune system leads to antimicrobial activity but also at the same time leads to inflammatory response involved in pathogenesis of acne. The ability of all-trans retinoic acid to modulate TLR-mediated innate immune response is one way in which we can control *P. acnes* growth and treat acne as well as other cutaneous infection.

Gene Expression in Inflammatory Acne

Diane Thiboutot, MD, Professor of Dermatology, The Pennsylvania State University College of Medicine, Hershey, PA 17033

The pathogenesis of acne has been linked to multiple factors such as increased sebum production, inflammation, follicular hyperkeratinization, and the action of *Propionibacterium acnes* within the follicle. In an attempt to understand the specific genes involved in inflammatory acne, we performed gene expression profiling in acne patients. Skin biopsies were obtained from an inflammatory papule and from normal skin in six patients with acne. Biopsies were also taken from normal skin of six subjects without acne. Gene array expression profiling was conducted using Affymetrix HG-U133A 2.0 arrays comparing lesional to nonlesional skin in acne patients and comparing nonlesional skin from acne patients to skin from normal subjects. Within the acne patients, 211 genes are upregulated in lesional skin compared to nonlesional skin. A significant proportion of these genes are involved in pathways that regulate inflammation and extracellular matrix remodeling, and they include matrix metalloproteinases 1 and 3, IL-8, human b-defensin 4, and granzyme B. These data indicate a prominent role of matrix metalloproteinases, inflammatory cytokines, and antimicrobial peptides in acne lesions. These studies are the first describing the comprehensive changes in gene expression in inflammatory acne lesions, they compare well with other reports of the expression of inflammatory mediators in acne lesions and they are valuable in identifying potential therapeutic targets in inflammatory acne.



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