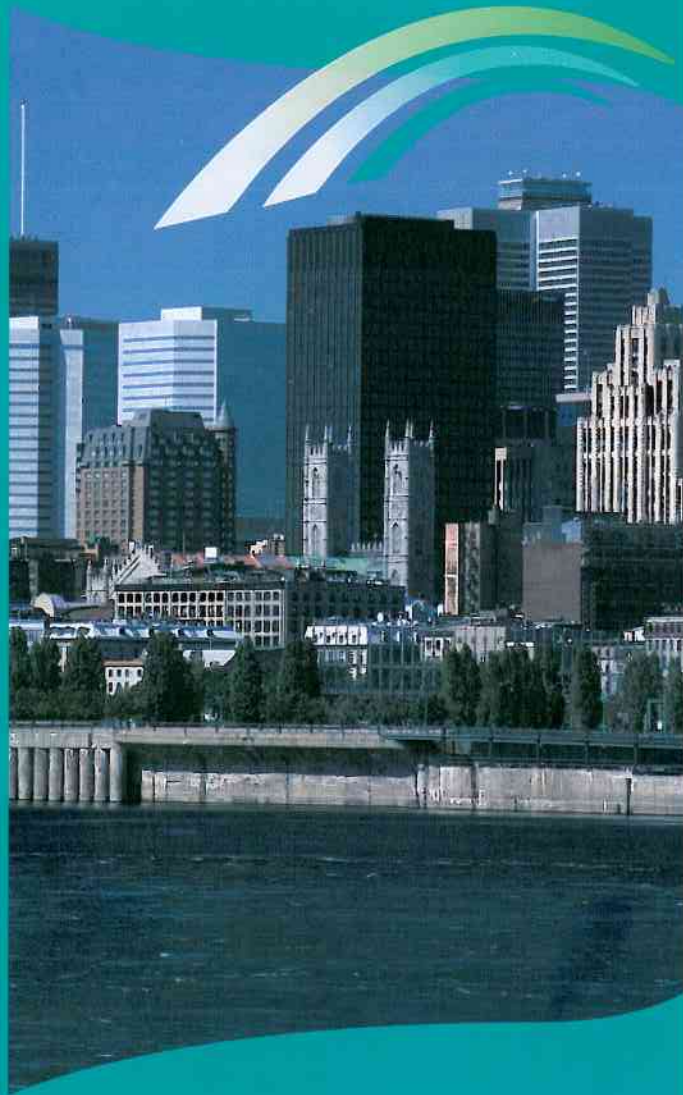


ADVANCES IN ROSACEA RESEARCH

A Satellite Symposium of the 69th Annual Meeting
of the Society for Investigative Dermatology - 2009



COMMITTEE ON ADVANCES IN ROSACEA RESEARCH

Richard Gallo, MD, PhD

University of California San Diego, San Diego, California

Yolanda Helfrich, MD

University of Michigan, Ann Arbor, Michigan

James Leyden, MD

Malvern, Pennsylvania

Diane Thiboutot, MD

Penn State University, Hershey, Pennsylvania

Jonathan Wilkin, MD

Columbus, Ohio

Satellite Symposium Sponsored by Galderma



Society for Investigative Dermatology

820 West Superior Ave., 7th Floor

Cleveland, OH 44113-1807

216-579-9300 fax: 216-579-9333

Thursday, May 7 2009

12:00 pm - 2:00 pm

Palais des Congrès, Room 517 A/B/C

Montreal, Canada

Satellite Symposium Sponsored by Galderma

ADVANCES IN ROSACEA RESEARCH

Thursday, May 7 2009, 12:00 pm - 2:00 pm
Palais des Congrès, Room 517 A/B/C

In partnership with Galderma, the SID instituted an annual Rosacea Research Award to advance Rosacea research along the road towards discovering new therapies and therapeutic targets in this common skin disease. The goal of the symposium is to present the latest progress in rosacea research and encourage young investigators to specialize in this area. This \$10,000 award is given to an investigator who published the best paper during the previous 18 months or submitted the best abstract to this year's SID meeting as determined by an ad hoc award committee. This award is not limited to members of the SID, but is open to scientists worldwide who will advance our progress along the road towards improvements in the understanding and treatment of rosacea.

CO-CHAIRS

Diane Thiboutot, MD

Penn State University College of Medicine, Hershey, Pennsylvania

Richard Gallo, MD, PhD

University of California, San Diego, California

SID & GALDERMA ROSACEA RESEARCH AWARD



2009 SID & Galderma Rosacea Research Award

Kenshi Yamasaki, MD, PhD

University of California, San Diego, California

Kenshi Yamasaki is currently a Project Scientist in the Division of Dermatology at the University of California San Diego. His primary research focus is on the role of cathelicidin peptides in the development of rosacea. Dr. Yamasaki received his M.D. and Ph.D. from Osaka University School of Medicine. He has authored over 40 publications and has received multiple funding awards, including the National Rosacea Society and the Japanese Society for the Promotion of Science.

12:00 pm - 12:10 pm

Overview and Presentation of Award

Diane Thiboutot, MD

*Penn State University College of Medicine,
Hershey, Pennsylvania*

12:10 pm - 12:30 pm

Aberrant Toll-Like Receptor Signaling Increases Kallikrein in Rosacea

Kenshi Yamasaki, MD, PhD

University of California, San Diego, California

12:30 pm - 12:45 pm

Adenosine Triphosphate Enhances Production of Inflammatory Factors by Human Dermal Microvascular Endothelial Cells: Possible Relevance to Rosacea

Richard Granstein, MD

*Weill Medical College of Cornell University,
New York, New York*

12:45 pm - 1:00 pm

Successful Treatment of the Erythema and Flushing of Rosacea Using a Topically Applied Selective α_1 -Adrenergic Receptor Agonist, Oxymetazoline

Stuart Shanler, MD

Cliffside Park, NJ

1:00 pm - 1:15 pm

A Pilot Quality of Life Instrument for Acne Rosacea

Kimberly Nicholson, MD

Northwestern University, Chicago, Illinois

1:15 pm - 1:30 pm

Rosacea: Physiology and Regulation Principles

Françoise Chanteloube

Bayer Santé Familiale, Lons, France

1:30 pm - 1:50 pm

Questions and Discussion

1:50 pm - 2:00 pm

Closing Comments

Richard Gallo, MD, PhD

University of California, San Diego, California



Richard Granstein MD

Richard D. Granstein, M.D. is the George W. Hambrick Jr. Professor in Dermatology and Chairman of the Department of Dermatology. Dr. Granstein obtained his undergraduate education at the Massachusetts Institute of Technology and his medical education at the UCLA School of Medicine. After completing his internship in 1979, he trained in dermatology at the Massachusetts General Hospital. As a Research Fellow, Dr. Granstein studied immunology and tumor biology at the National Cancer Institute-Fredrick Cancer Research Facility and at Harvard Medical School.

ADENOSINE TRIPHOSPHATE ENHANCES PRODUCTION OF INFLAMMATORY FACTORS BY HUMAN DERMAL MICROVASCULAR ENDOTHELIAL CELLS: POSSIBLE RELEVANCE TO ROSACEA

Rosacea is characterized by both vascular and inflammatory changes in response to trigger factors such as sunlight, heat, spicy foods, alcohol and stress. We hypothesized that adenosine triphosphate (ATP) (either secreted by neurons or released after cutaneous cell perturbation) may contribute to inflammation in rosacea by inducing release of pro-inflammatory factors from dermal microvascular endothelial cells through action on cell surface purinergic P2 receptors. It was found that ATP and the long-lived, hydrolysis-resistant ATP analogue ATP γ S induce production of interleukin-8 (IL-8, CXCL8), monocyte chemoattractant protein-1 (MCP-1, CCL2), and growth-regulated oncogene α (GRO α , CXCL1) in vitro by the transformed human microvascular endothelial cell line HMEC-1 and primary human dermal microvascular endothelial cells (HDMEC). IL-6 production and upregulation of intercellular adhesion molecule-1 (ICAM-1) were also induced by ATP γ S in HMEC-1 cells. These effects were suppressed by the presence of each of several non-specific P2 receptor inhibitors. Interestingly, ATP is a sympathetic nerve co-transmitter and sympathetic nerves innervate dermal blood vessels. Thus, hypothetically, under periods of stress, activation of the sympathetic nervous system may lead to release of ATP near dermal vessels that then binds to receptors on endothelial cells leading to release of chemokines and upregulation of ICAM-1. These chemokines then, in turn, chemoattract inflammatory cells while the induction of ICAM-1 facilitates transmigration of inflammatory cells from the vasculature into the interstitium with induction or augmentation of cutaneous inflammation. Such a mechanism may account, at least in part, for stress-induced exacerbation of rosacea. We have also found that the presence of tetracycline (TCN) inhibits the induction of IL-8 and GRO α production by HMEC-1 cells and primary HDMEC. Thus, inhibition of the production of these factors by endothelial cells may be one of the mechanisms by which TCN exerts its therapeutic effects in rosacea and other inflammatory skin disorders. A more complete understanding of the role of nerves and endothelial cell-derived pro-inflammatory factors in rosacea may lead to the development of novel therapeutic agents for this disease. Also, testing for the ability of agents to inhibit the induced release of pro-inflammatory factors by HDMEC may be useful for the identification of potential new therapeutics for rosacea.