

The Skin Sciences Institute

DIVISION PROFILE

Number of Faculty	14
Number of Fellows	
Research Fellows	3
Number of Graduate Students	13
Number of Other Students (full and part-time)	1
Number of Support Personnel	3
Number of Peer Reviewed Publications	32

FACULTY LISTING

Steven B. Hoath, MD, Professor of Pediatrics, Medical Director

Marty O. Visscher, PhD, Research, Director

Vivek Narendran, MD, Assistant Professor of Pediatrics, Director, Newborn Nursery, Christ Hospital;
Director Perinatal Outreach Program

Raymond E. Boissy, PhD, Professor of Dermatology and Cell Biology,

W. John Kitzmiller, MD, Associate Professor of Surgery, Head, Division of Plastic and Reconstructive
Surgery; Director of Wound Care Drake Hospital

Steven T. Boyce, PhD, Associate Professor of Surgery, Associate Professor of Biomedical Engineering;
Director, Department of Tissue Engineering, Shriners' Burns Institute

R. Randall Wickett, PhD, Professor of Pharmaceutics and Cosmetic Science, Director, Cosmetic
Science Graduate Program

Gerald B. Kasting, PhD, Associate Professor of Pharmaceutics and Cosmetic Science

Diya F. Mutasim, MD, Associate Professor of Dermatology, Director, Department of Dermatology

Charles L. Heaton, MD, Professor of Dermatology, Director, Sexually Transmitted Diseases Clinic and
Training Program, Department of Health

Zalfa Abdel-Malek, PhD, Associate Professor of Dermatology

James J. Nordlund, MD, Professor Emeritus of Dermatology, Director, Vitiligo Clinic

Glenn Talaska, PhD, Associate Professor of Environmental Health

Brian Adams, MD, Assistant Professor of Dermatology

OVERVIEW

The Skin Sciences Institute (SSI) is an interdisciplinary research group composed of investigators from TCHRF, the Shriners' Burns Institute, the Departments of Dermatology and Surgery, and the Colleges of Pharmacy, Engineering, and Environmental Health. The institute's goals are to generate new scientific information in basic and translational skin research, to provide the scientific information necessary for development of effective, evidence based skin care practices, while recognizing the skin as an important aspect of primary health care delivery, and to participate in effective research partnerships with the skin and health care industries.

Recent advances in the discerning the mechanism by which vernix caseosa may facilitate fetal epidermal barrier development in utero, the identification of pools of multiple anti-infective components in vernix and the associated functional activity, the effect of vernix caseosa on stratum corneum barrier restoration, the effect of stratum corneum water interactions on barrier repair and function, gene regulation and mechanisms of melanosome transfer, genetic modification to accelerate vascularization in cultured skin substitutes, regulation of keratinocyte activity in epidermal barrier repair, skin restoration with engineered cultured skin substitutes, and quantitative measurement of skin-water interactions, barrier damage and irritant dermatitis have been provided. Recognition of the investigators' expertise in epidermal barrier development, cell biology, pigment cell research and treatment of hyperpigmentary disorders, epidermal tissue engineering and wound healing, skin restoration, transdermal drug delivery and stratum corneum permeability, and quantitative biophysical characterization of skin characteristics motivated the formalization of the SSI.

The SSI's research on epidermal barrier development, vernix biology and infant skin adaptation is aimed at identifying the underlying biological mechanisms that a) produce a competent barrier in full term infants, b) facilitate the development of the barrier in premature infants, and c) influence the microbial interactions at the skin surface. Regulation of the water gradient at the skin surface is essential for the development of an effective stratum corneum barrier both in utero for the full term infant and under the dry environmental conditions of the NICU for the preterm infant.



Left to Right: S. Hoath, M. Visscher, V. Narendran

HIGHLIGHTS

Vernix Caseosa

Vernix caseosa is a complex proteolipid material synthesized in part by fetal sebaceous glands during the last trimester of pregnancy. The strategic location of vernix between the fetal skin surface and the amniotic fluid/environment suggests a potential role in multiple overlapping functions needed at the time of birth, ie, barrier to water loss, temperature regulation, moisturization, innate host defense, antioxidant, and wound healing. In vitro and in vivo investigations of native vernix have shown important and clinically relevant differences versus currently available treatments for use on premature infant skin, wounded or irritated skin, and compromised skin barrier. It protects the skin and allows for maturation using a fully natural material, as compared to conventional protectants (e.g., petrolatum) that are based on petrochemical derivatives. We have shown that vernix provides barrier repair properties in vivo, presumably by facilitating an optimal water gradient over the skin. Formulation efforts have resulted in high water containing, semipermeable prototypes based on the lipid classes found in native vernix, which can be evaluated in human trials in advance of translational research on preterm infants.

Chorioamnionitis and early onset infections are significant causes of morbidity and mortality in the newborn. Recent evidence supports a synergistic role for antimicrobial proteins in innate immune function and cutaneous host defense. The demonstration of host defense proteins (lysozyme, lactoferrin, human beta defensin) in vernix supports a role in innate immune function. Growth of group B streptococcus was inhibited by vernix. Endogenous antimicrobial peptides on or near the skin surface of the third trimester fetus may contribute to innate host defense mechanisms in early chorioamnionitis. Current research is aimed at determining the effectiveness of these and other antimicrobial proteins on the infant skin surface to establish their role in innate immunity.

Modulation of Skin Pigmentation

The SSI scientists investigated the mechanisms of action for deoxyarbutin (dA), a technology donated by The Procter & Gamble Company in October of 2001. Deoxyarbutin provides a therapeutic treatment for the modulation of post inflammatory skin hyperpigmentation that occurs as a result of tissue injury, e.g., acne scarring, chronic and acute wounds, burns, solar lentigines and other pigmentary disorders. The current treatment for hyperpigmentary disorders is hydroquinone, but the irritation associated with this modality is a significant limitation for patients. Our recent work has shown that deoxyarbutin is 4-fold less cytotoxic to melanocytes, keratinocytes, and fibroblasts than hydroquinone. At high concentrations and on intact cells, dA was significantly more inhibitory to tyrosinase than hydroquinone in light skin melanocytes. At the maximum viable dose, dA was more effective for tyrosinase/melanin inhibition than the vehicle control, while hydroquinone was ineffective at the maximum viable dose. Both dA and hydroquinone were equally effective in reducing post wounding hyperpigmentation on human skin (SCID mouse model) and dA resulted in a more uniform effect. In an in vivo human trial, dA significantly lightened tan induced human pigmentation while hydroquinone delayed the lightening process.

Hand Hygiene and Skin Barrier Integrity

A patient safety goal for 2004 is to reduce the risk of healthcare-acquired infections. Organizations have been directed to comply with the hand hygiene guideline issued in 2002 by the Centers for Disease Control (CDC) and developed to reduce the transmission of microorganisms to patients and health care workers (HCW). The guideline details the frequency and types of products (soaps, antibacterial soaps, alcohol rubs, surgical scrubs, and lotions) for various settings (ICU, operating room) to reduce microflora on the skin surface. Frequent, repetitive exposure to soap and water has significant negative effects on the structure and function of the outer layer of the skin, including inflammation, disruption of the lipid matrix, and increased permeability. In response, repair mechanisms are up-regulated, resulting in hyperproliferation, aberrant water binding properties, insufficient hydration and inadequate desquamation. Damaged skin has increased levels of bacteria. In collaboration with industrial partners, are investigating the effects of a hand hygiene regimen based on (a) minimally disruptive surfactants for soap and water washing or surgical scrub, (b) alcohol hand rubs with water-holding ingredients, and (c) skin lotion to determine the impact on hand skin barrier response and function relative to common hand care systems.

TRAINING

Valencia Walker, MD	PL-1	
Jon Cohen, MD	PL-3	
Richard Moraille, MD	PL-3	Ohio State University

PUBLICATIONS

1. Kadekaro AL, Kavanagh RJ, Wakamatsu K, Ito S, Pipitone MA, **Abdel-Malek ZA**. Cutaneous photobiology. The melanocyte vs. the sun: who will win the final round? *Pigment Cell Res* 2003;16(5):434-47.
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3. **Boissy RE**. Melanosome transfer to and translocation in the keratinocyte. *Exp Dermatol* 2003;12 Suppl 2:5-12.
4. **Boissy RE**, Manga P. On the etiology of contact/occupational vitiligo. *Pigment Cell Res* 2004;17(3):208-14.
5. Thong HY, Jee SH, Sun CC, **Boissy RE**. The patterns of melanosome distribution in keratinocytes of human skin as one determining factor of skin colour. *Br J Dermatol* 2003;149(3):498-505.
6. Holder IA, Durkee P, Supp AP, **Boyce ST**. Assessment of a silver-coated barrier dressing for potential use with skin grafts on excised burns. *Burns* 2003;29(5):445-8.

7. **Hoath SB**, Leahy DG. The organization of human epidermis: functional epidermal units and phi proportionality. *J Invest Dermatol* 2003;121(6):1440-6.
8. **Hoath SB**. Physiologic development of the skin. In: Polin RA, Fox WW, Abman SH, editors. *Fetal and Neonatal Physiology* 3rd ed. Philadelphia: Saunders; 2004. p. 597-611.
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12. Saiyasombati P, **Kasting GB**. In vivo evaporation rate of benzyl alcohol from human skin. *J Pharm Sci* 2004;93(2):515-20.
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15. Al-Ahmadie HA, **Mutasim DF**, Mutema GK. A case of intraepidermal Merkel cell carcinoma within squamous cell carcinoma in-situ: Merkel cell carcinoma in-situ? *Am J Dermatopathol* 2004;26(3):230-3.
16. Colvin JH, Lamerson CL, Cualing H, **Mutasim DF**. Cutaneous lymphoplasmacytoid lymphoma (immunocytoma) with Waldenstrom's macroglobulinemia mimicking rosacea. *J Am Acad Dermatol* 2003;49(6):1159-62.
17. Fein H, Sheth AP, **Mutasim DF**. Cutaneous arteritis presenting with hyperpigmented macules: macular arteritis. *J Am Acad Dermatol* 2003;49(3):519-22.
18. Lienesch DW, **Mutasim DF**, Singh RR. Eutrophilic eccrine hidradenitis mimicking cutaneous vasculitis in a lupus patient: a complication of cyclophosphamide. *Lupus* 2003;12(9):707-9.
19. Lienesch DW, **Mutasim DF**, Singh RR. Neutrophilic eccrine hidradenitis mimicking cutaneous vasculitis in a lupus patient: a complication of cyclophosphamide. *Lupus* 2003;12(9):707-9.
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21. **Mutasim DF**, Baumbach JL. Bullous autoimmune estrogen dermatitis. *J Am Acad Dermatol* 2003;49(1):130-2.
22. **Mutasim DF**. Lymphomatoid drug eruption mimicking digitate dermatosis: cross reactivity between two drugs that suppress angiotensin II function. *Am J Dermatopathol* 2003;25(4):331-4.
23. **Mutasim DF**. Confluent and reticulated papillomatosis without papillomatosis. *J Am Acad Dermatol* 2003;49(6):1182-4.
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31. Robinson MH, **Wickett RR**. Biochemical and bioengineering analysis of the skin's natural moisturizing factors. *J Cosmet Sci* 2004;55(2):211-2.
32. **Wickett RR**. Basics of skin structure. *J Cosmet Sci* 2004;55(1):132-3.