

Selecting a Daily Skin Care Regimen for Today's Female Patients: Clinical and Cosmetic Considerations



TOPICS

Introduction: Skin Care and Its Relevance in Everyday Dermatology Practice

Integrated Functions of the Stratum Corneum: Implications for an Optimal Skin Care Regimen

The Role of Cleansing and Moisturizing Regimens in the Management of Patient Skin

Selecting Daily Regimens for Skin Care After Rejuvenation Procedures

Produced in affiliation with the
29th Annual Hawaii Dermatology Seminar

FACULTY

Howard I. Maibach, MD, *Chairman*
University of California, San Francisco,
School of Medicine
San Francisco, Calif.

Peter M. Elias, MD
University of California, San Francisco,
School of Medicine
UCSF and Dermatology Service, VAMC
San Francisco, Calif.

Dee Anna Glaser, MD
Saint Louis University,
School of Medicine
St. Louis, Mo.

Diane S. Berson, MD
Weill Medical College of Cornell University
New York-Presbyterian Hospital
New York, N.Y.

President, Elsevier/IMNG
Alan J. Imhoff

Vice President,
 Medical Education
 & Business Development
Sylvia H. Reitman, MBA

Program Manager,
 Medical Education
Sara M. Hagan

National Account Manager
Cheryl J. Gromann

Graphic Design
Lehner & Whyte, Inc.

Production Manager
Judi Sheffer

The articles in this supplement are based on presentations made during the Skin Disease Education Foundation's 29th Annual Hawaii Dermatology Seminar, a continuing medical education program, held March 18-24, 2005, in Maui, Hawaii. This educational supplement is supported by an educational grant from



It was produced by the medical education department of International Medical News Group. Neither the Editor of SKIN & ALLERGY NEWS, the Editorial Advisory Board, nor the reporting staff reviewed or contributed to its contents. The opinions expressed in this supplement are those of the faculty and do not necessarily reflect the views of the supporter or the Publisher.

Copyright © 2005 Elsevier Inc. All rights reserved. No part of this publication may be reproduced or transmitted in any form, by any means, without prior written permission of the Publisher. Elsevier Inc. will not assume responsibility for damages, loss, or claims of any kind arising from or related to the information contained in this publication, including any claims related to the products, drugs, or services mentioned herein.



INTERNATIONAL
 MEDICAL NEWS
 GROUP

Introduction: Skin Care and Its Relevance in Everyday Dermatology Practice	4
.....	
Integrated Functions of the Stratum Corneum: Implications for an Optimal Skin Care Regimen	5
.....	
The Role of Cleansing and Moisturizing Regimens in the Management of Patient Skin	9
.....	
Selecting Daily Regimens for Skin Care After Rejuvenation Procedures	13
.....	
CME Post-Test and Evaluation	16

Faculty

Howard I. Maibach, MD, *Chairman*

Professor of Dermatology
 Department of Dermatology
 University of California, San Francisco, School of Medicine
 San Francisco, Calif.

Diane S. Berson, MD

Assistant Professor, Department of Dermatology
 Weill Medical College of Cornell University
 Assistant Attending Dermatologist
 New York-Presbyterian Hospital
 New York, N.Y.

Peter M. Elias, MD

Professor and Vice Chairman
 Department of Dermatology
 University of California, San Francisco, School of Medicine
 UCSF and Dermatology Service, VAMC
 San Francisco, Calif.

Dee Anna Glaser, MD

Professor of Dermatology
 Director, Cosmetic Dermatology
 Vice Chair, Department of Dermatology
 Director, Dermatology Consultation Services
 Saint Louis University, School of Medicine
 St. Louis, Mo.

Female Patients: Clinical and Cosmetic Considerations

CME Recognition

The SKIN & ALLERGY NEWS supplement “Selecting a Daily Skin Care Regimen for Today’s Female Patients: Clinical and Cosmetic Considerations” is recognized by the American Academy of Dermatology for 1 hour of AAD Category 1 credit and may be used toward the American Academy of Dermatology’s Continuing Medical Education Award.

This program was developed in accordance with the Accreditation Council for Continuing Medical Education guidelines.

Term of approval: August 2005-July 31, 2006

Estimated time to complete this educational activity: 1 hour.

Target Audience

This activity has been developed for physicians and other clinicians who specialize in dermatology.

Educational Needs

Healthy skin starts with a healthy stratum corneum (SC), the outermost layer of skin that serves as a barrier against desiccation and environmental stressors. The primary objective of a daily skin care regimen is to protect the SC barrier and to allow natural repair and recovery processes to maintain its structural and functional integrity. Patients with a variety of skin disorders—ranging from atopic dermatitis and acne to rosacea and photoaged skin—often have compromised skin barriers, as do patients who have recently undergone such cosmetic rejuvenation procedures as glycolic acid peels and laser therapy. SC barrier damage is often associated with sensitive skin, a disorder marked by burning, stinging and itching, and a hyperresponsiveness to environmental signals. Notably, retinoids and other common dermatological treatments have been shown to exacerbate the signs and symptoms associated with a weakened barrier.

Skin care regimens are critical determinants of SC barrier integrity. Harsh surfactants found in soap-based cleansers, for example, often extract vital lipids and proteins, leading to profound disturbances in homeostatic mechanisms that main-

tain and repair the barrier. Selecting skin care strategies that minimally perturb the SC barrier—including, for instance, mild moisturizers and non-soap-based syndet cleansers—is thus an essential component of managing patient skin. Since female patients are particularly susceptible to skin barrier insult caused by hormonal changes and their frequent use of cosmetic products, they must take special care to identify a daily regimen best suited for cleansing and moisturizing their skin.

Learning Objectives

At the conclusion of this activity, participants should be able to:

- Outline the general principles of stratum corneum structure and function, and the relevant factors affecting barrier damage and repair.
- Review the clinical features of sensitive skin and the underlying dermatological conditions and/or cosmetic procedures that can increase skin sensitivity.
- Provide practical skin care recommendations for patients undergoing clinical and cosmetic treatments.
- Describe optimal skin care approaches for female patients.

Faculty and Unapproved Use Disclosures

Faculty/authors must disclose any significant financial interest or relationship with proprietary entities that may have a direct relationship to the subject matter. They must also disclose any discussion of investigational or unlabeled uses of products.

Dr Berson and **Dr Maibach** have nothing to disclose. **Dr Elias** has received funding for clinical research from the National Institutes of Health (NIH) and the US Department of Veterans Affairs. He is a consultant to and has a financial interest in Osmotics Corporation. **Dr Glaser** has received funding for clinical research from Allergan, Inc. and Novartis AG. She is a consultant to Allergan, Dermik, Novartis, and Unilever PLC.

Introduction: Skin Care and Its Relevance in Everyday Dermatology Practice

Howard I. Maibach, MD

Current understanding of skin structure and function in health and disease has demonstrated the importance of fundamental skin care in optimizing patient outcomes. This supplement focuses on the biology of the stratum corneum (SC), the clinical relevance of skin care disorders associated with sensitive skin, and principles of proper skin care during recovery from cosmetic procedures. When recommending skin care regimens, clinicians should give special consideration to female patients, whose presumably more extensive use of cosmetic products and facial rejuvenation procedures may render them particularly susceptible to skin irritation.

The SC is the selectively permeable outer layer of the epidermis consisting of layers of flat, overlapping, and interlocking protein-rich cells embedded in a lipid matrix.¹ The unique structure of the SC enables it to function as an effective partial barrier to water loss as well as a defense against external trauma, microbial infection, chemical insult, and ultraviolet radiation. The SC is our interface with the external environment and, as such, is vulnerable to environmental stressors that potentially interfere with its critical, interrelated functions. Furthermore, the SC maintains and repairs the barrier through a series of tightly regulated signaling cascades and metabolic pathways.² These important topics are reviewed by Dr Elias on pages 5-8.

Varying levels of barrier dysfunction can lead to common skin problems such as xerosis, psoriasis, dermatitis, eczema, acne, rosacea, and photodamage, all of which are associated with sensitive skin,³ a term used to describe a hyperresponsiveness to external stimuli that often presents with burning, stinging, and itching.⁴⁻⁶ Recommended strategies designed to avoid exacerbating sensitive skin conditions—ranging from the use of mild syndet cleansers to select moisturizers that promote SC hydration—are reviewed by Dr Glaser on

pages 9-12. The formulation of syndet bars includes a mild synthetic surfactant in addition to greater amounts of moisturizer and humectant than are found in soap-based products.

The number of cosmetic rejuvenation procedures has risen dramatically over the past several years. As a result, dermatologists have become increasingly

“Varying levels of barrier dysfunction can lead to common skin problems such as xerosis, psoriasis, dermatitis, eczema, acne, rosacea, and photodamage....”

aware of skin care strategies that facilitate recovery from such procedures as laser therapy and chemical peels. Although the techniques may vary, each procedure, in the final analysis, damages the SC, rendering the skin remarkably sensitive to environmental insults, particularly harsh cleansers and other skin care and cosmetic products. A

review of commonly used rejuvenation procedures/treatments, their impact on the SC barrier, and proper skin care during the recovery phase is provided by Dr Berson on pages 13-15.

Despite considerable progress, much needs to be done in elucidating the chemical and biological basis of optimal skin care regimens. Many of the recommendations and strategies contained in this supplement are based primarily on clinical experience and are not the results of controlled trials or published consensus guidelines.

References

1. Elias PM. Epidermal lipids, barrier function, and desquamation. *J Invest Dermatol*. 1983;80(suppl):44s-49s.
2. Elias PM, Feingold KR. Coordinate regulation of epidermal differentiation and barrier homeostasis. *Skin Pharmacol Appl Skin Physiol*. 2001;14(suppl 1):28-34.
3. Subramanyan K. Role of mild cleansing in the management of patient skin. *Dermatol Ther*. 2004;17(suppl 1):26-34.
4. Giacomoni PU, Muizzuddin N, Sparacio RM, et al. Sensitive skin and moisturization. In: Leyden JJ, Rawlings AV, eds. *Skin Moisturization*. New York, NY: Marcel Dekker; 2002:145-154.
5. Seidenari S, Francomano M, Mantovani L. Baseline biophysical parameters in subjects with sensitive skin. *Contact Dermatitis*. 1998;38:311-315.
6. Scheman AJ. Sensitive skin products. *Skinmed*. 2003;2:374-375.

Integrated Functions of the Stratum Corneum: Implications for an Optimal Skin Care Regimen

Peter M. Elias, MD

Stratum Corneum Structure and Interrelated Function

The stratum corneum (SC), the thin, outermost layer of the skin, is a selectively permeable, heterogeneous, composite outer layer of the epidermis that protects against desiccation and environmental challenge. Often described in terms of the “bricks and mortar” model, the SC is an enzymatically active tissue consisting of largely proteinaceous corneocytes (bricks) embedded in a continuous matrix of lipids (mortar) (Figure 1).^{1,2} SC lipids—primarily ceramides (about 50% by mass), free fatty acids (10% to 20%), and cholesterol (25%)^{3–5}—are an essential element of normal water barrier function. The corneocytes protect against chemical and physical insult and also contribute to maintaining the water balance within the SC.⁶

Specialized intercellular protein structures called corneodesmosomes⁷ hold neighboring corneocytes together in the SC layer and adjacent layers. Consistent with the “mortar” analogy, lipids also contribute to the intercellular cement,^{8,9} but the corneodesmosomal structures provide the principal cohesive force that, when degraded, facilitates desquamation.

The SC typically consists of 12 to 16 layers of flattened and terminally differentiated corneocytes, each with a mean thickness of around 1 μm and a mean surface area of approximately 1,000 μm^2 .^{2,10,11} Individual corneocytes contain a highly organized keratin macrofibrillar matrix that binds considerable amounts of water. The keratin matrix is stabilized through interkeratin and intrakeratin filament disulfide bonds and is encapsulated within an extensively cross-linked protein shell called the cornified cell envelope (CE). The most insoluble structure of the corneocyte, the CE consists of a 15-nm-thick layer of structural proteins and a 5-nm-thick layer of specialized lipids^{12–14} that help maintain water barrier function by associating with the intercellular lipids.

An essential mechanism that main-

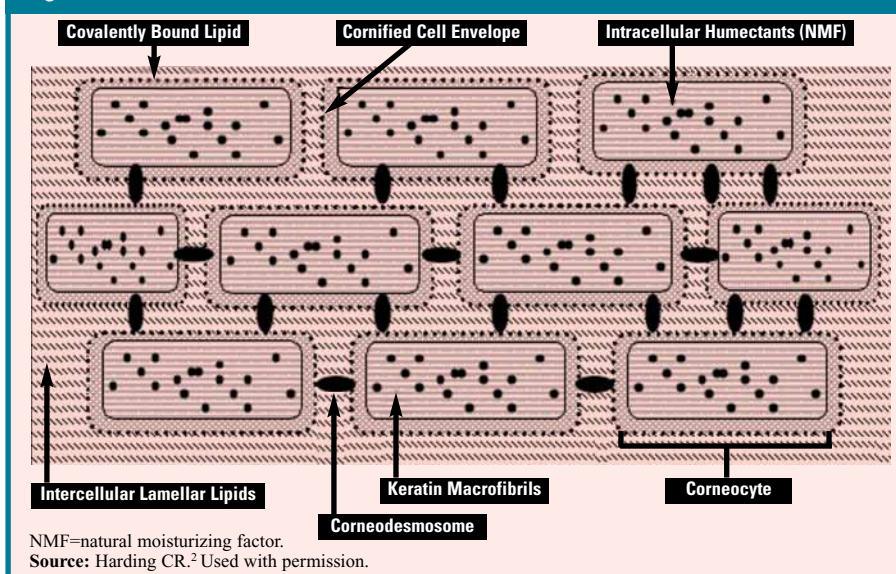
tains water balance within the SC is the formation of the natural moisturizing factor (NMF), a mixture of low-molecular-weight, water-soluble compounds consisting mainly of amino acids or their derivatives. NMF is generated and present in high concentrations exclusively within the corneocytes.⁶ The NMF compounds are primarily derived from the complete hydrolysis of filaggrin, a 37-kDa SC protein initially synthesized as profilaggrin in keratohyalin granules of the epidermis, which are the basophilic, irregularly shaped granules in the stratum granulosum that form the interfibrillary matrix. Profilaggrin is a large (>500 kDa), highly alkaline, heavily phosphorylated, histidine-rich protein.^{6,15–18}

Hygroscopic NMF components absorb atmospheric water and dissolve in their own water of hydration and, on that basis, are effective humectants.¹⁷ The humectant properties of the NMF maintain the hydration of the outermost layers of the SC despite the desiccating action of the environment. The “signal” that initiates the proteolysis of filaggrin to form NMF is the water gradient within the SC, which itself is the result of elaborate regulatory mechanisms and

differences in water-binding capacities between corneocyte cell layers.¹⁹ Environmental humidity determines, in part, the rate of evaporative water loss from the tissue and the steepness of the gradient and is therefore considered a critical determinant of the homeostatic signaling mechanism that generates NMF.

In addition to its role as a barrier to transepidermal water loss, the SC helps maintain basal barrier competence and facilitates homeostatic recovery and repair after external insults. The SC also defends against external trauma, microbial pathogens, chemical insult, and overexposure to ultraviolet radiation (UVR). These interrelated and protective roles are mediated by the unique structural components of the SC. For instance, the “mortar,” or extracellular lipids, form the basis of the permeability barrier and provide antimicrobial and antioxidant defense, whereas the corneocytes form a barrier to mechanical stress and UVR and help maintain optimal hydration levels. SC damage that initially results in suboptimal hydration may affect the enzymes that regulate corneocyte turnover, repair, and hydration, thus setting the stage for

Figure 1. Stratum Corneum Structure and Function: Bricks and Mortar Model



more pronounced damage over time.⁶ Alterations in pH can also adversely affect the permeability barrier and recovery, SC cohesion and integrity, and the antimicrobial barrier.^{6,20-22}

Factors That Perturb Barrier Homeostasis

A variety of factors may perturb barrier homeostasis (Figure 2). Some can be classified as modifiable, such as pH, UVR, and psychological stress, whereas others, such as gender and age, are nonmodifiable. As stated previously, the maintenance of SC hydration is under the profound influence of external humidity. Exposure to changes in environmental humidity induces changes in the SC moisture content and increases keratinocyte proliferation.²³ The external humidity has been shown to influence the number of lamellar bodies in stratum granulosum cells, the extent of lamellar body exocytosis, and the number of layers of SC.^{6,24} Humidity levels also influence the formation of the NMF and therefore the maintenance of SC hydration² and the process of desquamation.²⁵ Changes in environmental humidity can also contribute to seasonal exacerbations or amelioration of cutaneous disorders, such as atopic dermatitis and psoriasis, diseases that are characterized by a defective barrier, epidermal hyperplasia, and inflammation.²³

Alterations in SC pH can adversely affect barrier homeostasis and SC integrity and cohesion.^{20,26} Barrier repair after acute perturbations proceeds normally at an acidic skin pH.⁶ Higher pH can impede the phospholipid-to-free-fatty-acid processing for normal SC acidification, an important pathway not only for barrier homeostasis but also for SC integrity and cohesion.²¹ Furthermore, increased proteolytic activities have been observed under high pH conditions, leading to increased corneodesmolysis and aberrations in corneocyte cohesion.²²

Recent studies provide the first link between psychological status and cutaneous function in humans and suggest that stress-induced derangements in epidermal function precipitate inflammatory dermatoses.²⁷⁻²⁹ Psychological stress, for example, appears to stimulate production of glucocorticoids, which modulate the expression of a wide variety of target genes, and adversely affect permeability barrier homeostasis.²⁷

Continuous exposure to UVR damages cellular DNA and proteins and often leads to premature aging of the skin (photoaging).³⁰ Further, cumulative UVR harms keratinocytes and fibro-

“The structural, biochemical, and molecular anatomy of the SC equip it to function as a unique, sophisticated biosensor that signals the underlying epidermis to respond to external stresses.”

blasts, triggering signal transduction cascades that inhibit new collagen synthesis and promote the proteolytic breakdown of collagen in the extracellular matrix.³¹ UVR contributes to dry skin by disrupting differentiation in the granular layer, including the processing of profilaggrin, the precursor to the NMF.³² In addition, UVR leads to a depletion of Langerhan’s cells (believed to be a further factor in UVR-induced immunosuppression), epidermal hyperplasia, and thickening of the SC.³²

Chronologic aging of the skin is a universal and inevitable process associated with abnormalities in permeability barrier homeostasis, altered drug permeability, increased susceptibility to irritant contact dermatitis, and often

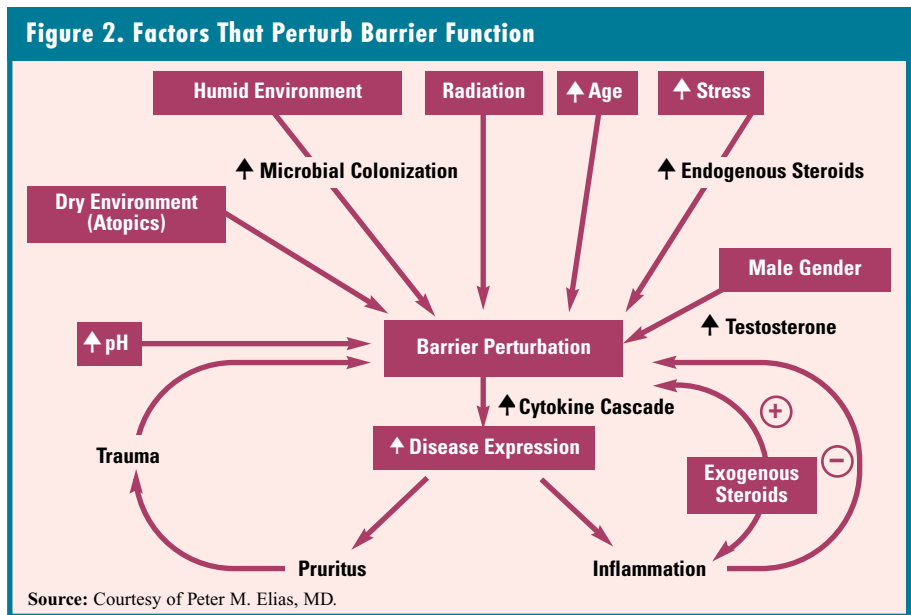
severe xerosis.³³ The biochemical basis of these and related changes appears to include, in part, impaired keratinocyte differentiation,³⁰ a profound reduction in SC lipid biosynthesis,³⁴ changes in the biophysical properties of collagen and elastin fibers,³⁵ and aberrant growth factor signaling pathways, particularly in the interleukin-1 family.³⁴ Notably, the diminished capacity for SC repair commonly found in aged skin may be due to cytokine dysregulation.³⁶

Gender may be a factor in the perturbation of barrier homeostasis as well. Fluctuations in testosterone, for example, are known to modulate barrier function, and testosterone repletion can have negative consequences for permeability barrier homeostasis.³⁷ In addition, sex-related differences have been observed at the level of the total ceramide concentration.³⁸

Various skin diseases are associated with perturbations in barrier homeostasis. Atopic dry skin and psoriasis are characterized by increased transepidermal water loss,³⁹⁻⁴¹ diminished water-binding properties,⁴²⁻⁴⁴ and abnormalities in lipid metabolism, including reduced SC ceramide levels.^{45,46} Dramatic changes in SC lipid structure contribute to some of the characteristic aberrations in SC function, including a lack of corneocyte cohesion and faulty desquamation.^{2,40,47,48}

Mechanisms That Regulate Barrier Homeostasis and Repair

Situated at the interface of the external environment, the SC maintains or



reestablishes barrier homeostasis in response to environmental stressors, such as reduced external humidity or barrier perturbations, through a variety of signaling cascades and metabolic processes.⁴⁹ Of particular importance are those pathways that drive DNA, lipid, and protein biosynthesis (Figure 3),^{2,49-51} each of which is influenced by ionic signaling, cytokines and growth factors, and the endogenous ligands of nuclear hormone receptors.

Transepidermal water loss is a regulatory signal for barrier homeostasis.⁴⁹ Perturbations in the barrier leading to altered water flux set in motion a series of processes within the epidermis that promote barrier recovery and repair.² The recovery of the barrier to a maintenance state begins within hours (rapid recovery phase) and takes approximately 3 days (prolonged recovery phase) (Figure 4).⁴⁹ In the elderly, the recovery takes approximately 1 week.

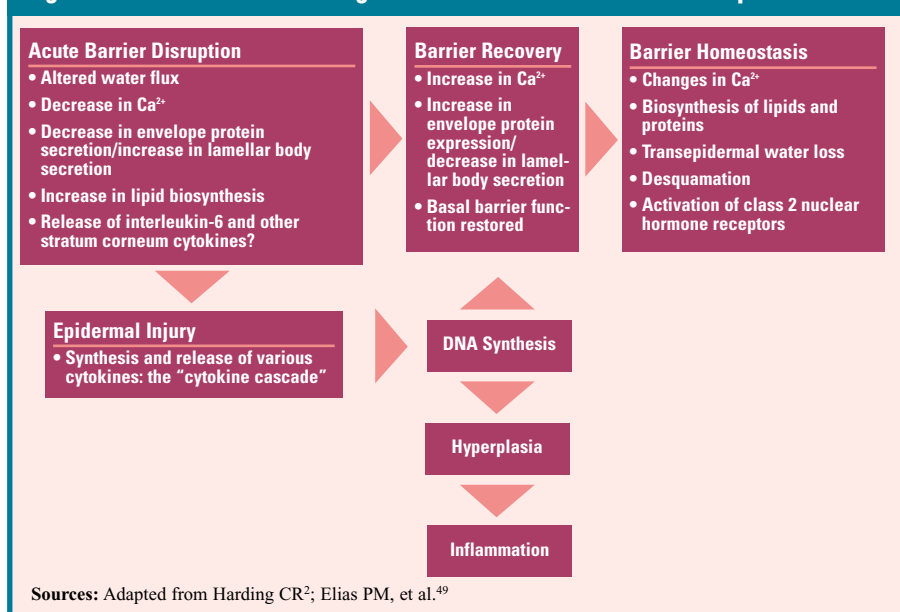
Although the precise mechanisms are not yet firmly established, the results of various studies have suggested that specific ions, particularly Ca^{2+} and K^{+} , are critical to the processes involved in barrier homeostasis and repair.^{2,49} With barrier disruption, the displacement of Ca^{2+} and K^{+} passively into the SC is followed by a burst of lamellar body secretion and lipid biosynthesis and a concurrent decline in the synthesis of at least some corneocyte proteins such as profilaggrin.⁴⁹

Following acute barrier disruption, a preformed pool of cytokines is released from the outer epidermis, and epidermal synthesis of many cytokines and growth factors increases.^{49,52} Changes in levels of epidermal cytokines and growth factors are potential candidates to mediate the metabolic responses to perturbation that lead to barrier recovery. Current data also suggest that the release of cytokines and growth factors is involved in cutaneous pathology.⁵¹ In addition, nuclear hormone receptors mediate epidermal growth, differentiation, development and barrier function, the antiinflammatory response, and sebaceous gland lipogenesis.⁴⁹

Summary

The structural, biochemical, and molecular anatomy of the SC equip it to function as a unique, sophisticated biosensor that signals the underlying epidermis to respond to external stressors. The proper maintenance of the SC

Figure 3. Mechanisms That Regulate Barrier Homeostasis and Repair

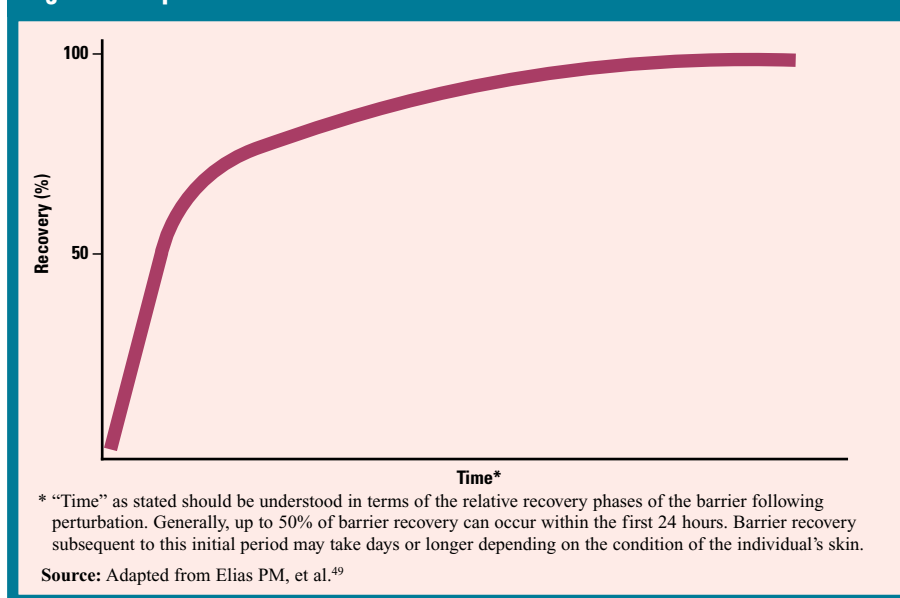


barrier and its recovery after perturbation are essential to the functions of the skin. The SC has several critical functions including but not limited to regulation of transepidermal water loss, mechanical defense against external trauma, and antimicrobial defense, as well as defense against chemical insult and overexposure to UVR. The functions of the SC are integrated and often dependent on one another in critical ways. Some of the factors that can perturb barrier homeostasis are modifiable—exposure to UVR, the pH of skin products, and psychological stress—whereas others, such as gender or increased age, cannot be modified.

Several coordinated signaling mechanisms that regulate lipid, DNA, and protein biosynthesis maintain barrier homeostasis or lead to its repair after perturbation. An understanding of the structure and function of the SC and the mechanisms that govern its maintenance, repair, and recovery will inevitably lead to improved skin care strategies that restore barrier functionality and prevent avoidable damage to the skin.

[Dr Elias' article provides a general update on stratum corneum structure and function that contains elements of his presentation, although it does not represent a summary of his talk.]

Figure 4. Rapid Restoration of Normal Function After Acute Barrier Insult



References

- Elias PM. Epidermal lipids, barrier function, and desquamation. *J Invest Dermatol.* 1983;80(suppl):44s-49s.
- Harding CR. The stratum corneum: Structure and function in health and disease. *Dermatol Ther.* 2004;17(suppl 1):6-15.
- Elias PM. Epidermal barrier function: Intercellular lamellar lipid structures, origin, composition and metabolism. *J Control Release.* 1991;15:199-208.
- Schaefer H, Redelmeier TE. *Skin Barrier: Principles of Percutaneous Absorption.* Basel: Karger; 1996:310.
- Wertz PW, van den Bergh B. The physical, chemical and functional properties of lipids in the skin and other biological barriers. *Chem Phys Lipids.* 1998;91:85-96.
- Rawlings AV, Harding CR. Moisturization and skin barrier function. *Dermatol Ther.* 2004;17(suppl 1):43-48.
- Chapman S, Walsh A. Desmosomes, corneosomes and desquamation: An ultrastructural study of adult pig epidermis. *Arch Dermatol Res.* 1990;282:304-310.
- Epstein EH Jr, Williams ML, Elias PM. Steroid sulfatase, X-linked ichthyosis, and stratum corneum cell cohesion. *Arch Dermatol.* 1981;117:761-763.
- Ranasinghe AW, Wertz PW, Downing DT, Mackenzie IC. Lipid composition of cohesive and desquamated corneocytes from mouse ear skin. *J Invest Dermatol.* 1986; 86:187-190.
- Marks R, Barton SP. The significance of the size and shape of corneocytes. In: Marks R, Plewig G, eds. *Stratum Corneum.* New York: Springer-Verlag; 1983:161-170.
- Ya-Xian Z, Suetake T, Tagami H. Number of cell layers of the stratum corneum in normal skin: Relationship to the anatomical location on the body, age, sex and physical parameters. *Arch Dermatol Res.* 1999; 291:555-559.
- Swartzendruber DC, Wertz PW, Madison KC, Downing DT. Evidence that the corneocyte has a chemically bound lipid envelope. *J Invest Dermatol.* 1987;88: 709-713.
- Marekov LN, Steinert PM. Ceramides are bound to structural proteins of the human foreskin epidermal cornified cell envelope. *J Biol Chem.* 1998; 273:17763-17770.
- Jarnik M, Simon MN, Steven AC. Cornified cell envelope assembly: A model based on electron microscopic determinations of thickness and projected density. *J Cell Sci.* 1998;111:1051-1060.
- Scott IR, Harding CR. Studies on the synthesis and degradation of a high molecular weight, histidine-rich phosphoprotein from mammalian epidermis. *Biochim Biophys Acta.* 1981;669:65-78.
- Scott IR, Harding CR, Barrett JG. Histidine-rich protein of the keratohyalin granules: Source of the free amino acids, urocanic acid and pyrrolidone carboxylic acid in the stratum corneum. *Biochim Biophys Acta.* 1982;719:110-117.
- Rawlings AV, Scott IR, Harding CR, Bowser PA. Stratum corneum moisturization at the molecular level. *J Invest Dermatol.* 1994;103:731-741.
- Harding CR, Scott IR. Stratum corneum moisturizing factors. In: Leyden J, Rawlings A, eds. *Skin Moisturization.* New York: Marcel Dekker; 2002:61-80.
- Scott IR, Harding CR. Filaggrin breakdown to water binding compounds during development of the rat stratum corneum is controlled by the water activity of the environment. *Dev Biol.* 1986;115:84-92.
- Hachem JP, Crumrine D, Fluhr J, Brown BE, Feingold KR, Elias PM. pH directly regulates epidermal permeability barrier homeostasis, and stratum corneum integrity/cohesion. *J Invest Dermatol.* 2003;121:345-353.
- Fluhr JW, Kao J, Jain M, Ahn SK, Feingold KR, Elias PM. Generation of free fatty acids from phospholipids regulates stratum corneum acidification and integrity. *J Invest Dermatol.* 2001;117:44-51.
- Fluhr JW, Elias PM. Stratum corneum pH: Formation and function of the acid mantle. *Exog Dermatol.* 2002;1:163-175.
- Denda M, Sato J, Tsuchiya T, Elias PM, Feingold KR. Low humidity stimulates epidermal DNA synthesis and amplifies the hyperproliferative response to barrier disruption: Implication for seasonal exacerbations of inflammatory dermatoses. *J Invest Dermatol.* 1998;111:873-878.
- Denda M, Sato J, Masuda Y, et al. Exposure to a dry environment enhances epidermal permeability barrier function. *J Invest Dermatol.* 1998;111:858-863.
- Rawlings A, Harding C, Watkinson A, Banks J, Ackerman C, Sabin R. The effect of glycerol and humidity on desmosome degradation in stratum corneum. *Arch Dermatol Res.* 1995;287:457-464.
- Fluhr JW, Mao-Qiang H, Brown BE, et al. Functional consequences of a neutral pH in neonatal rat stratum corneum. *J Invest Dermatol.* 2004;123:140-151.
- Denda M, Tsuchiya T, Elias PM, Feingold KR. Stress alters cutaneous permeability barrier homeostasis. *Am J Physiol Regul Integr Comp Physiol.* 2000;278:R367-R372.
- Garg A, Chren MM, Sands LP, et al. Psychological stress perturbs epidermal permeability barrier homeostasis: Implications for the pathogenesis of stress-associated skin disorders. *Arch Dermatol.* 2001;137:53-59.
- Altemus M, Rao B, Dhabhar FS, Ding W, Granstein RD. Stress-induced changes in skin barrier function in healthy women. *J Invest Dermatol.* 2001;117:309-317.
- Hashizume H. Skin aging and dry skin. *J Dermatol.* 2004;31:603-609.
- Fisher GJ. The pathophysiology of photoaging of the skin. *Cutis.* 2005;75(2 suppl):5-8; discussion 8-9.
- Nole G, Johnson AW. An analysis of cumulative lifetime solar ultraviolet radiation exposure and the benefits of daily sun protection. *Dermatol Ther.* 2004;17(suppl 1):57-62.
- Ghadially R. Aging and the epidermal permeability barrier: Implications for contact dermatitis. *Am J Contact Dermat.* 1998; 9:162-169.
- Elias PM, Ghadially R. The aged epidermal permeability barrier: Basis for functional abnormalities. *Clin Geriatr Med.* 2002;18:103-120.
- Wulf HC, Sandby-Møller J, Kobayasi T, Gniadecki R. Skin aging and natural photoprotection. *Micron.* 2004;35:185-191.
- Ye J, Garg A, Calhoun C, Feingold KR, Elias PM, Ghadially R. Alterations in cytokine regulation in aged epidermis: Implications for permeability barrier homeostasis and inflammation. I. IL-1 gene family. *Exp Dermatol.* 2002;11:209-216.
- Kao JS, Garg A, Mao-Qiang M, et al. Testosterone perturbs epidermal permeability barrier homeostasis. *J Invest Dermatol.* 2001;116:443-451.
- De Paepe K, Weerheim A, Houben E, Roseeuw D, Ponc M, Rogiers V. Analysis of epidermal lipids of the healthy human skin: Factors affecting the design of a control population. *Skin Pharmacol Physiol.* 2004;17:23-30.
- Werner Y, Lindberg M. Transepidermal water loss in dry and clinically normal skin in patients with atopic dermatitis. *Acta Derm Venereol.* 1985;65:102-105.
- Motta S, Monti M, Sesana S, Melleli L, Ghidoni R, Caputo R. Abnormality of water barrier function in psoriasis: Role of ceramide fractions. *Arch Dermatol.* 1994; 130:452-456.
- Tagami H, Yoshikuni K. Interrelationship between water-barrier and reservoir function of pathologic stratum corneum. *Arch Dermatol.* 1985;121:642-645.
- Thune P. Evaluation of the hydration and the water-holding capacity in atopic skin and so-called dry skin. *Acta Derm Venereol.* 1989;144(suppl):133-135.
- Imokawa G, Abe A, Jin K, Higaki Y, Kawashima M, Hidano A. Decreased level of ceramides in stratum corneum of atopic dermatitis: An etiologic factor in atopic dry skin? *J Invest Dermatol.* 1991; 96:523-526.
- Matsumoto M, Umemoto N, Sugiura H, Uehara M. Difference in ceramide composition between "dry" and "normal" skin in patients with atopic dermatitis. *Acta Derm Venereol.* 1999;79:246-247.
- Macheleidt O, Kaiser HW, Sandhoff K. Deficiency of epidermal protein-bound omega-hydroxyceramides in atopic dermatitis. *J Invest Dermatol.* 2002;119:166-173.
- Cui CY, Kusada S, Seguchi T, Takahashi M, Aisu K, Tezuka T. Decreased levels of prosaposin in atopic skin. *J Invest Dermatol.* 1997;109:319-323.
- Fartasch M. Epidermal barrier in disorders of the skin. *Microsc Res Tech.* 1997; 38:361-372.
- Ghadially R, Reed JT, Elias PM. Stratum corneum structure and function correlates with phenotype in psoriasis. *J Invest Dermatol.* 1996;107:558-564.
- Elias PM, Feingold KR. Coordinate regulation of epidermal differentiation and barrier homeostasis. *Skin Pharmacol Appl Skin Physiol.* 2001;14(suppl 1): 28-34.
- Elias PM, Ansel JC, Woods LD, Feingold KR. Signaling networks in barrier homeostasis. The mystery widens. *Arch Dermatol.* 1996;132:1505-1506.
- Elias PM, Wood LC, Feingold KR. Epidermal pathogenesis of inflammatory dermatoses. *Am J Contact Dermat.* 1999;10:119-126.
- Wang XP, Schunck M, Kallen KJ, et al. The interleukin-6 cytokine system regulates epidermal permeability barrier homeostasis. *J Invest Dermatol.* 2004;123:124-131.

The Role of Cleansing and Moisturizing Regimens in the Management of Patient Skin

Dee Anna Glaser, MD

Skin Disorders, Sensitive Skin, and Barrier Dysfunction

Common skin disorders such as xerosis, psoriasis, atopic dermatitis (AD), acne, rosacea, and photodamage are linked to barrier dysfunction, which leaves the skin vulnerable to various external insults and skin sensitivity.^{1,3} Sensitive skin is characterized by burning, stinging, and itching without any visible signs of inflammation.⁴ Visible signs of inflammation, when they do occur, may be related to one or more of the products patients use in their personal skin care regimens. Thus, selecting an optimal regimen is a critical component of sensitive skin care. Unfortunately, patients tend to receive inaccurate, inadequate, and even harmful advice regarding skin care from sources such as cosmetic counters at department stores, their local salon and spa, and their friends and family. A clearer understanding of sensitive skin will facilitate its treatment and the selection of proper cleansing and moisturization products, which are an important component of an integrated skin care regimen.

Approximately 40% to 50% of the population report some characteristics of sensitive skin. More women than men report sensitive skin, and there are no apparent differences in prevalence based on ethnicity.^{5,6} Patients with sensitive skin typically seen by dermatologists are adult females with acne, rosacea, mild atopic or perioral dermatitis, or seborrhea, all of whom are particularly susceptible to skin barrier insults. Patients without an underlying disease may present with sensitive skin as well. Female patients are particularly susceptible to skin barrier insult due to hormonal changes related to pregnancy or menopause⁷ and frequent use of cosmetic products⁸ that may irritate or perturb the skin barrier (eg, makeup, anti-aging treatments, and exfoliants).

Cleansing is an essential component of skin care that removes dirt, oil, environmental pollutants, and bacteria from the skin.¹ Paradoxically, the act of

cleansing typically leads to a weakening of the barrier as a result of the harsh surfactant ingredients in many cleansers that interact with the proteins and lipids of the stratum corneum (SC) detrimentally. For most skin disorders, cleansing with commonly used soap-based products exacerbates patients' skin disorders. The type of surfactant is a chief determinant of the irritancy potential of a personal wash product.⁹ The most aggressive surfactants are those with C10 to C14 chain lengths, which predominate in common soaps.

“...Changing to a mild syndet-based cleanser can lead to significant improvements in either normal or sensitive skin.”

The formulation of syndet bars includes a mild synthetic surfactant in addition to greater amounts of moisturizer and humectant than are found in soap-based products.⁹ The use of mild syndet-based cleansing formulations, especially in skin disorders characterized by barrier dysfunction and sensitive skin, is strongly advised.¹ A list of some commonly available cleansers is displayed on the basis of their formulation in the **Table** on page 11.⁹ High pH also increases the irritancy potential of a cleansing product. The pH of normal soaps is alkaline and in the range of 9.5 to 11.0.

Skin Improvement Following Modifications to the Skin Care Regimen

A series of trials evaluating the use of syndet bars in patients with AD, acne, rosacea, and normal skin was recently reviewed.¹ A 4-week, double-blind, parallel-group study was carried out in

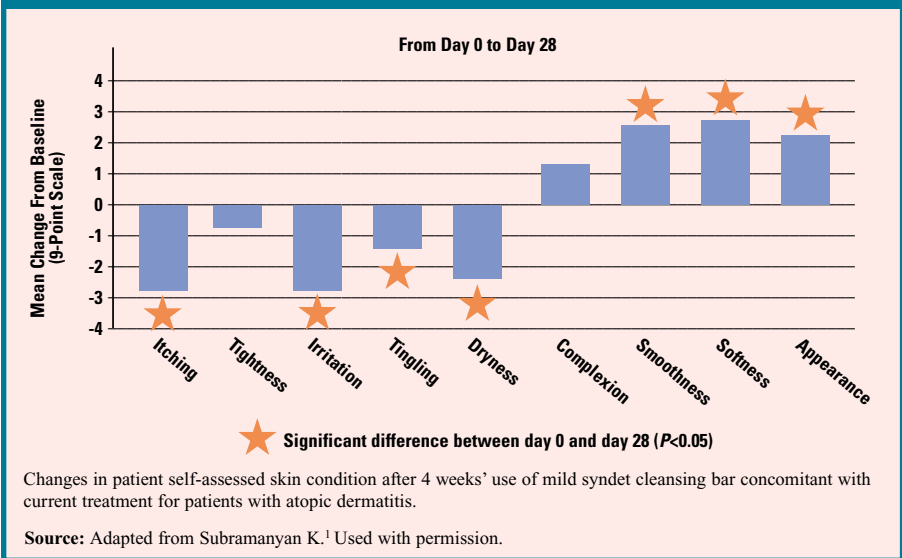
which 25 patients with mild AD (19 adults and 6 children) used a syndet cleanser instead of their normal cleanser (soap) for showering or bathing and continued their usual medication for AD.^{1,10} Eczema (whole body) and normal skin (forearm and calf) were evaluated at baseline and day 28 using the Eczema Area Severity Index (EASI). Improvements were noted in EASI scores after 4 weeks' use of a mild syndet cleansing bar concomitantly with current treatment for AD. Significant improvements were also noted in patient self-assessed skin condition, ie, itching, irritation, tingling, dryness, smoothness, softness, and appearance (**Figure 1**).¹

In a randomized double-blind study, 50 patients with moderate acne using topical erythromycin/benzoyl peroxide or erythromycin/benzoyl peroxide plus adapalene topical to treat their acne condition were recruited and instructed to use either a soap bar or a mild syndet bar to cleanse their face for a 4-week period.^{1,11} Patient skin was rated clinically for erythema, peeling, dryness, burning, stinging, itching, and tightness, each using a 4-point scale from 0 (none) to 3 (severe). An overall assessment of acne condition was made using a 6-point scale from 1 (very severe) to 6 (almost clear).

Dermatologist assessment of the patients revealed that for the patients using soap, the clinical measures of irritation, such as peeling, dryness, and irritation, worsened during the 4-week period, whereas no significant changes in irritation measures were seen for those patients using the syndet bar (**Figure 2**).¹ The results also clearly showed that the mild cleanser was more effective in significantly reducing scores for several negative characteristics such as itching, acne, and oiliness.

In another randomized, double-blind study, 25 patients with acne (15 using over-the-counter [OTC] acne medication and 10 using prescription [Rx] acne medication) were recruited and instructed to use a mild cleansing lotion

Figure 1. Use of Syndet Bar in Mild Atopic Dermatitis*



to cleanse their faces while continuing to use their medication (OTC or Rx) as usual.¹ Dermatologist assessment after 4 weeks indicated significant decreases in mean scores for key acne-related attributes such as the numbers of comedones and papules and pustules.

In a randomized double-blind study, 70 patients with moderate rosacea using topical metronidazole were instructed to use either a soap bar or mild syndet bar to cleanse their face for a 4-week period.¹¹ Patient self-assessment showed that soap use significantly worsened itching, irritation, tingling, and dryness, whereas the mild syndet cleanser alleviated many of the skin irritation measures (Figure 3).¹

Syndet use was also recently compared in a small group of female patients (N=28) who had either normal skin, patient-assessed sensitive skin problems, or dermatologist-assessed sensitive skin.¹² The 4-week study evaluated the effects of a daily regimen encompassing mild cleansing, moisturizing, and sun protection. In the morning, patients were instructed to use a mild self-foaming wash, a clarifying toner, and mild cream moisturizer with sun protection factor (SPF) 15. In the evening, patients were instructed to use a mild cleansing pillow, clarifying toner, and a mild cream moisturizer with no SPF. All groups showed improvement. A majority of patients (88%) perceived significant improvement over baseline in the state of moisturization and overall appearance of their skin. In 100% of the subjects, the consulting investigating dermatologist saw significant improvement in overall moisturization.

The results of these studies demonstrate that changing to a mild syndet-based cleanser can lead to significant improvements in either normal or sensitive skin. Equally important, reductions in skin irritation improve patient compliance especially in the population of individuals using Rx products for underlying dermatologic conditions.

Cosmetic Products That Contribute to Problematic Skin in Patients With Underlying Dermatologic Conditions

In patients with sensitive skin, it is important to avoid products that contain potentially irritating ingredients such as volatile solvents, penetrants, harsh surfactants, abrasives, and aromatic sun-

Figure 2. Dermatologist Assessment of Acne Patients*

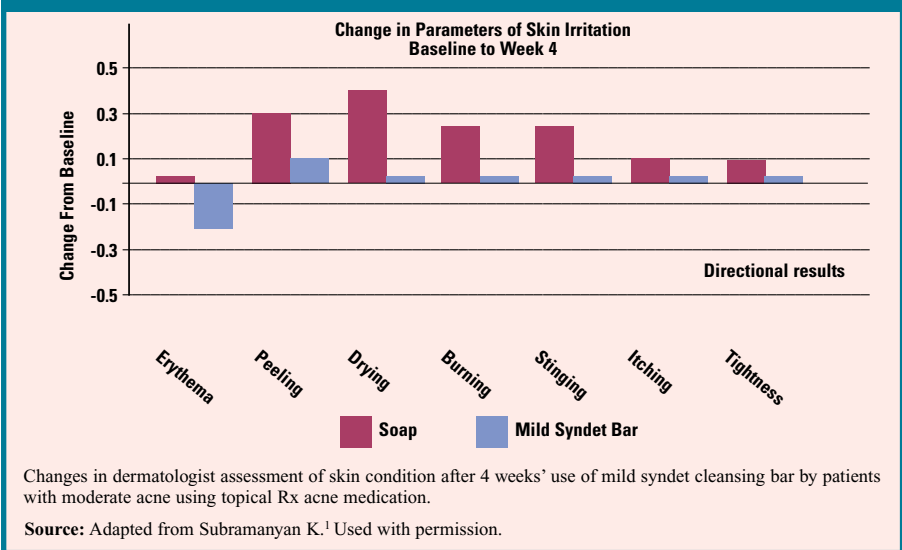
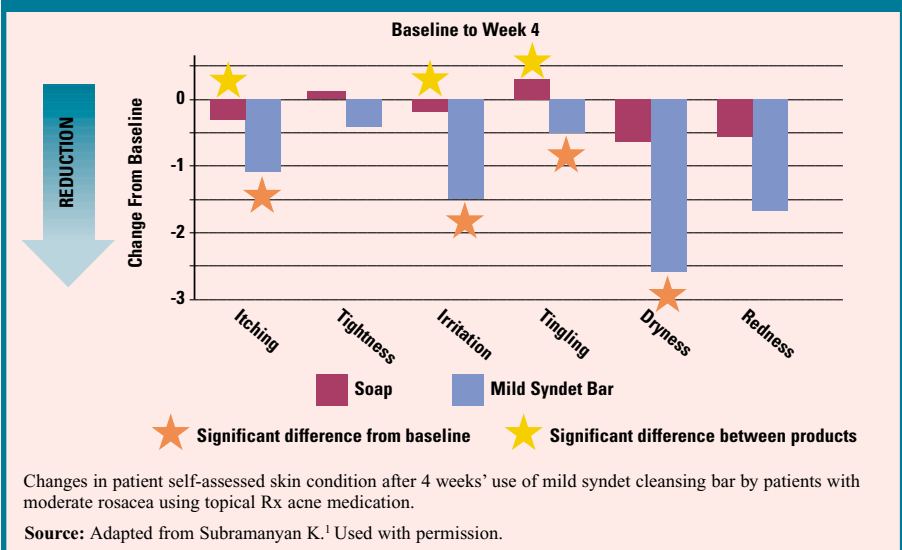


Figure 3. Patient Self-Assessment of Rosacea*



*In Figures 1, 2, and 3, the mild syndet bar used is Dove Sensitive Skin Bar.

screens.⁴ Specific examples of these classes of ingredients are listed in **Box 1**. Comedogenic products are those that induce open and/or closed comedones after 2 to 3 weeks, usually because of follicular plugging.^{13,14} Acnegenic products are those that can cause an acne papule or pustule that is usually related to follicular irritation.¹⁵ These types of products should also be avoided in patients with sensitive skin. “Irritating” products should also be avoided, but it should be noted that the irritancy potential of any product is related to the particular state of the individual’s SC, the concentration of the product, and the length of time the product is left on the skin.¹⁶

Recommended Strategies for Improving Skin Health

Cosmetic Use

It is important to assess all skin care or maintenance products, including cosmetics, for their capacity to exacerbate a patient’s existing condition. Indeed, not all ingredients are alike in their potential risk to induce damage to the SC barrier. The use of mild cleansers using synthetic detergents has been emphasized. There are also principles regarding cosmetic use that are important as well.¹⁵ Although many of these principles have not been formally tested in controlled trials, clinical experience and common sense suggest that they could be helpful to patients with problematic skin. Generally, powder cosmetics are better for sensitive skin than are creams or lotions. If a liquid facial foundation is used, it should be silicone based on cyclomethicone or dimethicone. Waterproof products are not recommended. Patients should replace their cosmetics frequently (every 3 to 6 months). Chemical-based sunscreens should be avoided in favor of microfine zinc oxide or micronized titanium dioxide or one of the physical blockers. Nail polishes, including colored and noncolored, should be avoided. For the use of eye cosmetics, generally black colors are better tolerated. The pencil forms of eyeliner and eyebrow cosmetics should be used. Light earth tone colors are favored for eye shadows rather than shiny colors or deep colors such as blue, purple, or green.

Strategy for Patients Experiencing Skin Flushing

For patients who experience skin

Box 1. Ingredients in Cosmetic Products That Should Be Avoided in Sensitive Skin

Ethanol
Menthol
Camphor
Benzyl alcohol
Propylene glycol
Sodium lauryl sulfate
Retinoids
Quaternary ammonium compounds
Mica
Silica
Bismuth oxychloride
Para-aminobenzoic acid
Benzophenones
Cinnamates

Source: Adapted from Scheman AJ.⁴

flushing, a skin care regimen that minimizes flushing is essential.¹⁵ Lukewarm water should be used during cleansing. A foaming liquid face wash containing mild synthetic detergents is preferred. The cleansing product should not be rubbed on the face and should be rinsed off thoroughly. Toners, astringents, chemical exfoliants, and all products designed to remain on the face should be avoided. As a replacement for chemical exfoliants, the use of a facial cleansing cloth two or three times weekly provides adequate mild mechanical exfoliation. The use of creams and powder skin care products is preferred to use of gels and thin lotions, especially those containing alcohols and solvents. Facial manipulation should be minimized, and a daily

cream-based sunscreen, preferably containing microfine zinc oxide, should be used.

Strategy for Patients With Worsening Skin Sensitivity

For patients whose sensitive skin condition is of apparent unknown origin and worsens on treatment, a comprehensive strategy that involves cessation of all skin care products followed by careful assessment of the effects of cessation and selective product reapplication may help⁸ (see **Box 2**). In the first phase of implementing such a strategy, all topical products, including prescribed products and those containing steroids, are discontinued over a period of 2 weeks. All the products patients have applied to their skin for the last 6 months should be brought into the office for assessment. Patients should be instructed to use a syndet cleanser as well as a bland moisturizer, the choice of which will depend on their individual skin integrity and moisture level. It is important to eliminate all sources of skin friction, including facials and apricot washes. Various tests can be administered 2 to 3 weeks after the implementation of this strategy to evaluate the patients for underlying dermatosis. Once the dermatosis, if present, or the skin sensitivity is under control, careful and selective reapplication of cosmetic products can be initiated. Generally, one product per week is added in order of importance to the patient. Each product can then be tested by provocative-use tests to determine whether or not it was the product(s) that caused the problem.

Summary

Dermatologists are the logical choice for good, accurate, and unbiased

Table. Commonly Available Personal Cleansing Products

Formulation:	Syndet	Soap
Product:	Aveeno	Dial
	Caress	Irish Spring
	Cetaphil	Ivory
	Dove	Neutrogena Transparent
	Neutrogena Extra Gentle	Tone
	Olay	Zest

The formulation of syndet bars includes a mild synthetic surfactant in addition to greater amounts of moisturizer and humectant than are found in soap-based products.

Source: Adapted from Abbas S, et al.⁹

Box 2. Strategy for Patients With Worsening Skin Sensitivity

1. Discontinue all topicals for 2 weeks
2. Discontinue all topical prescriptions or wean off steroids for 2 weeks
3. Switch to a mild syndet cleanser (see Table)
4. Use a bland moisturizer
5. Eliminate all sources of skin friction
6. Use pimecrolimus if needed
7. Evaluate the patient for underlying dermatoses in 2 to 3 weeks
8. Use a patch test, photopatch test if needed
9. Use a facial sting test with 10% lactic acid (nasolabial fold) or malar eminence
10. Add one facial cosmetic per week
 - Lipstick
 - Face powder
 - Powder blush
 - Let patients choose the most important to them
11. Provocative-use tests for products
 - Nightly x 5 nights
 - 2-cm area lateral to eye
 - Mascara, eyeliner, eyebrow pencil, eye shadow, foundations, and colored cosmetics
12. Use tests for products that remain on skin
 - Avoid cumbersome, time-consuming use tests

Source: Courtesy of Dee Anna Glaser, MD.

information, especially for patients with sensitive skin. The basic skin care regimen should involve mild cleansing, daily moisturizing, and sun protection. In patients with sensitive skin, it is important to avoid products that contain irritating ingredients. Strategies for skin care should be implemented on the basis of understanding skin structure and function (see the article by Dr Elias in this supplement), the particular individual aspects of a patient's skin

condition, the regimen of skin care products and cosmetics a patient uses, and any rejuvenation procedures the patient may have undergone (see the article by Dr Berson in this supplement).

References

1. Subramanyan K. Role of mild cleansing in the management of patient skin. *Dermatol Ther.* 2004;17(suppl 1):26-34.
2. Seidenari S, Francomano M, Mantovani L. Baseline biophysical parameters in subjects

- with sensitive skin. *Contact Dermatitis.* 1998;38:311-315.
3. Giacomoni PU, Muizzuddin N, Sparacio RM, et al. Sensitive skin and moisturization. In: Leyden JJ, Rawlings AV, eds. *Skin Moisturization.* New York, NY: Marcel Dekker; 2002:145-154.
4. Scheman AJ. Sensitive skin products. *Skinmed.* 2003;2:374-375.
5. Willis CM, Shaw S, De Lacharriere O, et al. Sensitive skin: An epidemiological study. *Br J Dermatol.* 2001;145:258-263.
6. Jourdain R, Lacharriere O, Bastien P, Maibach HI. Ethnic variations in self-perceived sensitive skin: Epidemiological survey. *Contact Dermatitis.* 2002;46:162-169.
7. Shah MG, Maibach HI. Estrogen and skin: An overview. *Am J Clin Dermatol.* 2001;2:143-150.
8. Draelos ZD. Sensitive skin: Perceptions, evaluation, and treatment. *Am J Contact Dermat.* 1997;8:67-78.
9. Abbas S, Goldberg JW, Massaro M. Personal cleanser technology and clinical performance. *Dermatol Ther.* 2004;17(suppl 1):35-42.
10. Solodkina G, Yan X, Johnson AW, Gottlieb AB. Mild synthetic detergent bars are helpful for patients with atopic dermatitis. Poster presented at: 60th Annual Meeting of the American Academy of Dermatology; February 22-27, 2002; New Orleans, La.
11. Subramanyan K, Johnson AW. Role of mild cleansing in the management of sensitive skin. Poster presented at: 61st Annual Meeting of the American Academy of Dermatology; March 21-26, 2003; San Francisco, Calif.
12. Hawkins SS, Subramanyan K, Liu D, Bryk M. Cleansing, moisturizing, and sun-protection regimens for normal skin, self-perceived sensitive skin, and dermatologist-assessed sensitive skin. *Dermatol Ther.* 2004;17(suppl 1):63-68.
13. Poli F. [Cosmetic treatments and acne]. *Rev Prat.* 2002;52:859-862. In French.
14. American Academy of Dermatology invitational symposium on comedogenicity. *J Am Acad Dermatol.* 1989;20(2 pt 1):272-277.
15. Knor T. The pathogenesis of acne. *Acta Dermatovenerol Croat.* 2005;13:44-49.
16. Draelos ZD. Cosmetics in acne and rosacea. *Semin Cutan Med Surg.* 2001;20:209-214.

Selecting Daily Regimens for Skin Care After Rejuvenation Procedures

Diane S. Berson, MD

Topical, Laser, and Chemical Skin Rejuvenation Treatments

The visible signs of aged skin include wrinkling, blotchiness, sagging, discoloration, scarring, acne, and uneven pigmentation. Restorative cosmetic skin procedures have become increasingly common in the field of dermatology, in part because of the effects of aging and chronic photodamage, in particular, which leads to premature aging of the skin. As the population ages and disposable income increases, the proportion of people seeking skin rejuvenation and enhancing procedures is likely to increase substantially.

The number of cosmetic procedures increased by 44% in 2004 from the previous year, according to the American Society for Aesthetic Plastic Surgery.¹ The most frequently used treatment was botulinum toxin type A injection, followed by laser procedures, chemical peels, microdermabrasion, and hyaluronic acid fillers (Table).¹ The vast majority of patients undergoing cosmetic procedures in 2004 were women (90%), but increasing percentages of men are seeking cosmetic treatments.

Topical Therapies

Topical therapies can be used as primary treatment of aged skin or as an adjunct to surgical or nonsurgical cosmetic procedures. Studies have shown that retinoids, which have been a mainstay in both the prevention and the treatment of the effects of photoaging, diminish the appearance of fine lines and wrinkles, reduce hyperpigmenta-

tion, and smooth the surface of the skin.² Other agents that are effective in improving the appearance of skin and preventing further damage include antioxidants (eg, vitamins C and E, green tea, co-enzyme Q10), alpha-hydroxy acids, bleaching agents (hydroquinone), moisturizers, emollients, and sunscreen.² Antioxidants generally protect the skin against the effects of free radicals formed by ultraviolet radiation (UVR). The use of alpha-hydroxy acids is thought to rejuvenate the skin, but its precise mechanism of action is currently unknown.² Bleaching agents, such as hydroquinone, are helpful for hyperpigmentation, and moisturizers and emollients help maintain hydration and restore the stratum corneum (SC) barrier. The use of a sunscreen helps protect against further photodamage.

Resurfacing, Chemical Peels, and Other Modalities

The optimal resurfacing procedure will vary depending on particular aspects of the patient's skin condition. Ablative laser skin resurfacing (in particular, carbon dioxide [CO₂] and erbium:YAG lasers) is considered the gold standard for treating deep rhytides, photodamage, and acne scars.³ Since laser skin resurfacing ablates the epidermis, intensive wound and skin care after the procedure is essential to achieving an optimal outcome (see Box).⁴ The use of occlusives, which retard water loss from the barrier, enhances the healing of the superficial thermal injury created by laser skin

resurfacing and reduces the risk of scarring. Effective postprocedure management can help minimize some of the expected effects of the procedure, such as crusting, discomfort, pruritus, erythema, and swelling. Ablative resurfacing procedures are associated with a 1- to 2-week recovery period.

Nonablative laser resurfacing procedures are commonly used for superficial wrinkling and dyschromia. The procedure bypasses the epidermis with a cooling mechanism and targets the dermis with heat, inducing a wound-healing mechanism and the generation of new collagen. Bypassing the epidermis avoids barrier disruption and explains the accelerated recovery time when compared to ablative procedures. Single-pass CO₂ laser skin resurfacing in conjunction with cold-air cooling minimizes intraoperative and postoperative adverse effects and has been shown to contribute to patient satisfaction.^{3,5}

Box. Laser Resurfacing: Postoperative Care

Soak (0.25% acetic acid, saline, or water) for 20 minutes, every 2 to 4 hours

Apply bland emollient ointments

Avoid products with antibacterials because they can irritate the skin

Gently cleanse with a mild syndet cleanser (see Table in Dr Glaser's article on page 11) after 24 to 48 hours

As healing progresses, switch from emollient ointment to a light moisturizer/sunscreen

Occlusive dressings may also be used for 2 or 3 days postprocedure

Source: Adapted from Batra RS.⁴

Table. Top Nonsurgical Cosmetic Procedures/Treatments, 2004

Nonsurgical	No. of procedures/treatments
Botulinum toxin type A	2,837,346
Laser hair removal	1,411,899
Chemical peel	1,110,401
Microdermabrasion	1,098,316
Hyaluronic acid	882,469

Source: American Society for Aesthetic Plastic Surgery.¹

The pulse dye laser can be helpful for wrinkling and also for vascular lesions.⁶ Infrared lasers are also well suited for mild acne scarring.⁷ Nonlaser treatments such as dermabrasion are useful in the treatment of severe acne scarring.⁸ Other nonlaser treatments—including intense pulsed light, which uses a broadband light source to treat hyperpigmentation and erythema—may also smooth the tone and texture of the skin.⁹⁻¹³ Radiofrequency tightens the skin and is used as a treatment for facial wrinkling and sagging.^{14,15}

Chemical peels are a popular cosmetic procedure for treatment of scars, pigmentation, and photodamage.¹⁶ Peeling reduces surface defects by destroying the outer layers of the epidermis, which stimulates the healing process and the generation of a new SC.¹⁷ Common chemical peel treatments include trichloroacetic acid, Jessner’s peel (salicylic acid, lactic acid, resorcinol), alpha-hydroxy acid (eg, glycolic acid, lactic acid) salicylic acid, and phenol.^{16,18,19} Because the SC is damaged following the chemical peel, mild cleansing products are necessary to avoid additional irritation.¹⁶

Factors Governing the Choice of Rejuvenation Procedures

The optimal rejuvenation procedure for each patient is dependent on several factors, including age, gender, skin type (dry, oily, or sensitive skin), propensity for scarring, and the patient’s underlying clinical conditions, if any, such as acne or hyperpigmentation. The patient’s lifestyle (eg, typical sun exposure or whether the patient smokes) should also be considered. These parameters may influence the outcome of treatment. For example, if a patient has acne scarring in addition to active acne, it would not make sense to use an ablative procedure to treat the scarring until the active acne is under control.

Posttreatment Skin Care

For patients who have undergone cosmetic procedures, the optimal post-treatment skin care and maintenance regimen is one that enhances the treatment response, improves the texture of their skin, and prevents new photodamage. Patients’ daily posttreatment skin care regimen should include mild cleansing; moisturization with a

light water-based moisturizer without added dyes, fragrances, or preservatives; and a sunscreen for UVR protection. Proper cleansing is necessary to remove dirt, oil, and bacteria from the skin. Indeed, infection can be a common complication of rejuvenation procedures.^{4,20} Soap-based cleansers contain harsh surfactants that can perturb the epidermal barrier by interacting with essential proteins and the lipid barrier. Mild, syndet cleansers are preferable to soap because syndet cleansers minimally interact with the lipids and proteins of the barrier (Figure 1)²¹ and are less irritating (Figures 2 and 3).²² Examples of syn-

det products are listed in the Table in Dr Glaser’s article in this supplement. Soap-based cleansers also have a high alkaline pH, which is known to disrupt skin barrier proteins and lipids.²³ Moisturizers containing humectants, occlusives, and emollients, which bind and trap water and aid in barrier repair, are optimal (Figure 4).²⁴ Finally, adequate protection from UVR helps prevent further photodamage.

Summary

Proper posttreatment skin care, including mild cleansing with minimal barrier perturbation, moisturization

Figure 1. Syndet Cleansers Minimally Perturb the Barrier

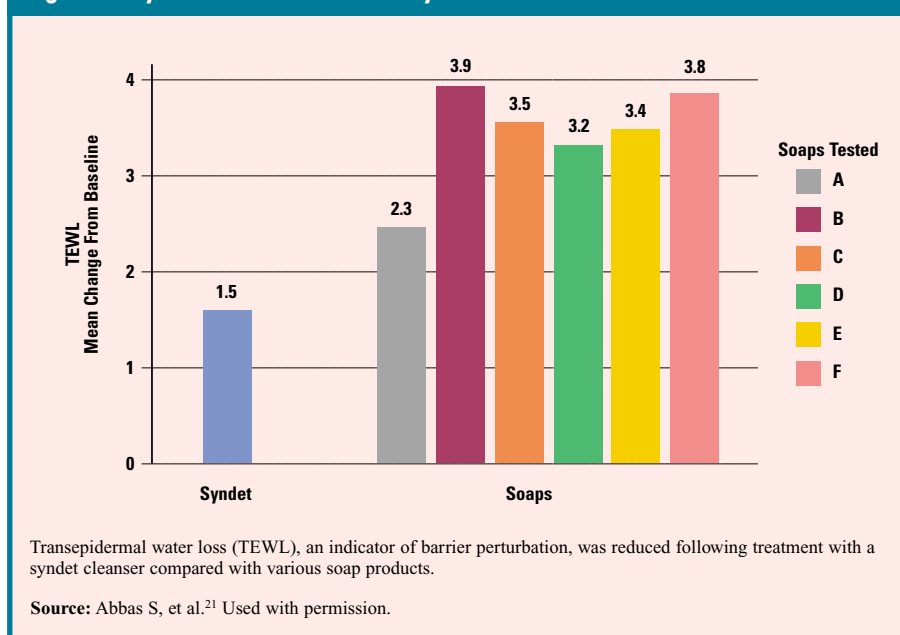
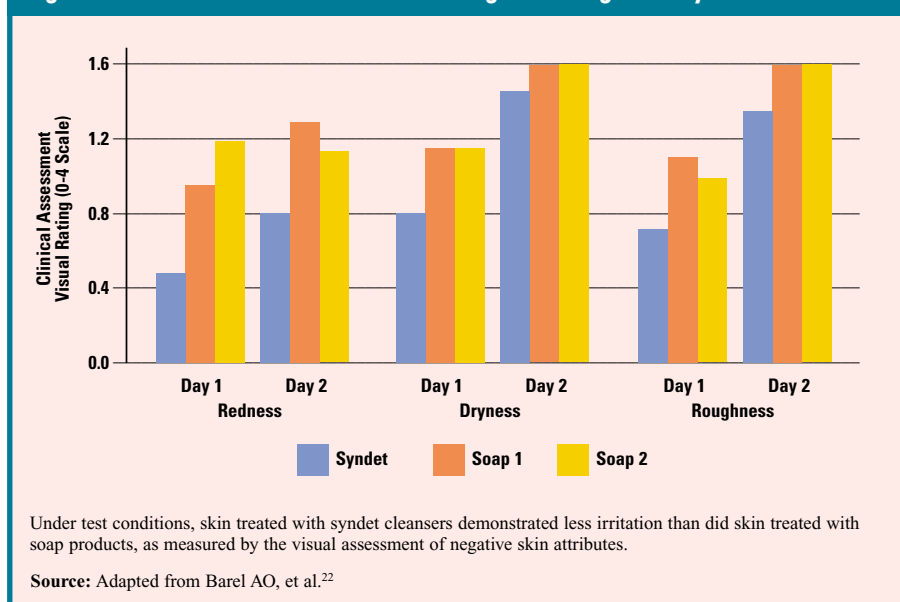


Figure 2. Reduced Skin Irritation Following Cleansing With Syndet Cleansers



(hydration and lipid replenishing), and UVR protection, can enhance the results of cosmetic rejuvenation procedures. Improvement in SC barrier integrity through mild cleansing and moisturization will reduce skin irritation associated with common clinical and cosmetic procedures and enhance the skin's overall appearance and skin health.

References

1. The American Society for Aesthetic Plastic Surgery 2004: *Cosmetic Surgery National Data Bank Statistics*. Available at: <http://www.surgery.org/download/2004-stats.pdf>. Accessed April 11, 2005.
2. Glaser DA, Rogers C. Topical and systemic

therapies for the aging face. *Facial Plast Surg Clin North Am*. 2001;9:189-196.

3. Raulin C, Grema H. Single-pass carbon dioxide laser skin resurfacing combined with cold-air cooling: Efficacy and patient satisfaction of a prospective side-by-side study. *Arch Dermatol*. 2004;140:1333-1336.
4. Batra RS. Ablative laser resurfacing: Post-operative care. *Skin Therapy Lett*. 2004;9:6-9.
5. Kopera D, Smolle J, Kaddu S, Kerl H. Nonablative laser treatment of wrinkles: Meeting the objective? Assessment by 25 dermatologists. *Br J Dermatol*. 2004;150:936-939.
6. Tanghetti EA, Sherr EA, Alvarado SL. Multipass treatment of photo-damage using the pulse dye laser. *Dermatol Surg*. 2003;29:686-690; discussion 690-691.

7. Goodman GJ. Management of post-acne scarring: What are the options for treatment? *Am J Clin Dermatol*. 2000;1:3-17.
8. Gold MH. Dermabrasion in dermatology. *Am J Clin Dermatol*. 2003;4:467-471.
9. Paquet P, Pierard GE. Intense pulsed light treatment of persistent facial hypermelanosis following drug-induced toxic epidermal necrolysis. *Dermatol Surg*. 2004;30(12 pt 2):1522-1525.
10. Alster TS, Tanzi EL, Welsh EC. Photo-rejuvenation of facial skin with topical 20% 5-aminolevulinic acid and intense pulsed light treatment: A split-face comparison study. *J Drugs Dermatol*. 2005;4:35-38.
11. Alam M, Dover JS. Treatment of photoaging with topical aminolevulinic acid and light. *Skin Therapy Lett*. 2004;9:7-9.
12. Chan HH, Kono T. The use of lasers and intense pulsed light sources for the treatment of pigmentary lesions. *Skin Therapy Lett*. 2004;9:5-7.
13. Kligman DE, Zhen Y. Intense pulsed light treatment of photoaged facial skin. *Dermatol Surg*. 2004;30:1085-1090.
14. Abraham MT, Chiang SK, Keller GS, Rawnsley JD, Blackwell KE, Elashoff DA. Clinical evaluation of non-ablative radiofrequency facial rejuvenation. *J Cosmet Laser Ther*. 2004;6:136-144.
15. Koch RJ. Radiofrequency nonablative tissue tightening. *Facial Plast Surg Clin North Am*. 2004;12:339-346.
16. Subramanyan K. Role of mild cleansing in the management of patient skin. *Dermatol Ther*. 2004;17(suppl 1):26-34.
17. Hevia O, Nemeth AJ, Taylor JR. Tretinoin accelerates healing after trichloroacetic acid chemical peel. *Arch Dermatol*. 1991;127:678-682.
18. El-Domyati MB, Attia SK, Saleh FY, Ahmad HM, Uitto JJ. Trichloroacetic acid peeling versus dermabrasion: A histometric, immunohistochemical, and ultrastructural comparison. *Dermatol Surg*. 2004;30(2 pt 1):179-188.
19. Fulton JE, Porumb S. Chemical peels: Their place within the range of resurfacing techniques. *Am J Clin Dermatol*. 2004;5:179-187.
20. Monheit GD. Chemical peels. *Skin Therapy Lett*. 2004;9:6-11.
21. Abbas S, Goldberg JW, Massaro M. Personal cleanser technology and clinical performance. *Dermatol Ther*. 2004;17(suppl 1):35-42.
22. Barel AO, Lambrecht R, Clarys P, Morrison BM Jr, Paye M. A comparative study of the effects on the skin of a classical bar soap and a syndet cleansing bar in normal use conditions and in the soap chamber test. *Skin Res Technol*. 2001;7:98-104.
23. Ananthapadmanabhan KP, Moore DJ, Subramanyan K, Misra M, Meyer F. Cleansing without compromise: The impact of cleansers on the skin barrier and the technology of mild cleansing. *Dermatol Ther*. 2004;17(suppl 1):16-25.
24. Rawlings AV, Harding CR. Moisturization and skin barrier function. *Dermatol Ther*. 2004;17(suppl 1):43-48.

Figure 3. Reduced Self-Perceived Skin Irritation Following Mild Cleansing

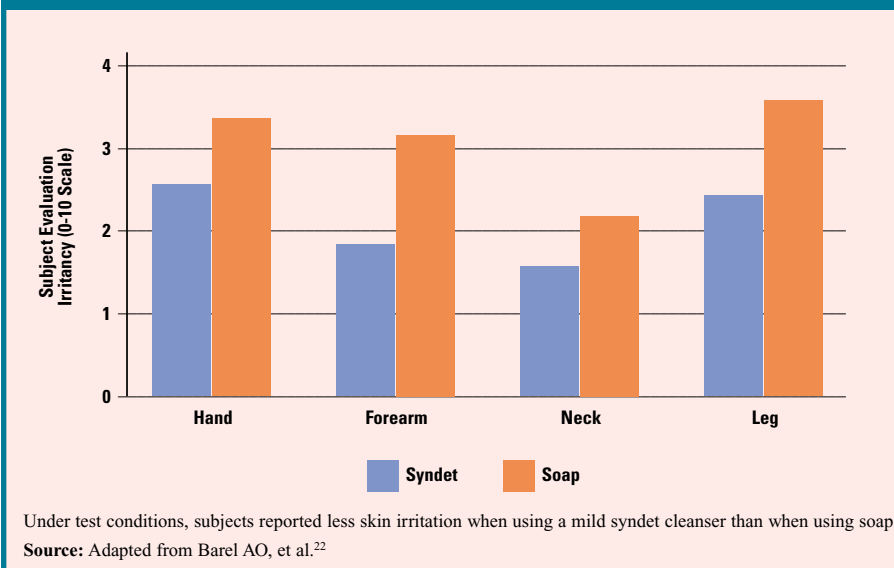


Figure 4. Moisturization and Barrier Function

