Cosmeceuticals: The Science of Designing a Skincare Regimen

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Dermatologist, Author, Researcher
Financial Disclosure

- CEO of Skin Type Solutions
- Advisory Board and Researcher for many skincare brands
- Author multiple books
Diagnose the skin type before designing a regimen
Baumann Skin Type

- Oily vs Dry
- Sensitive vs Resistant
- Pigmented vs Nonpigmented
- Wrinkled vs Tight
Fitzpatrick Skin Type is Limited to Skin Pigmentation Response

- The concept of sun-reactive "skin typing" was created in 1975
- Classified persons with white skin
- Used to select the correct initial doses of ultraviolet A and psoralen (PUVA) for the treatment of psoriasis
- Fitzpatrick realized that the estimation of the white-skinned person’s tolerance level to oral PUVA could not be based solely on hair and eye color
- Systems asks about tanning response to sun exposure.

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Validation of a Questionnaire to Diagnose the Baumann Skin Type in All Ethnicities and in Various Geographic Locations

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Abstract

The Baumann Skin Typing System diagnoses patients as having one of 16 skin types based on their answers to a validated questionnaire [i] known as the Baumann Skin Type Indicator [ii]. The BSTI questionnaire has been tested over the last decade on over 200,000 people of various ages and ethnicities in different geographic locations around the world. In this study, data were collected from 52,862 patients to compare skin type prevalence between those who presented to doctor’s offices and those who took the quiz without supervision online. The most common skin types varied only slightly between patients that took the quiz online and those that completed the questionnaire in their doctor’s office. This indicates that the prevalence of skin types seen in the doctor’s office is similar to that in the general population and that supervision is not necessary to get an accurate result on the BSTI. [iii] In addition, comparison of data gathered in China, Korea, and the US did not show a significant difference in skin type prevalence between Asian and Caucasian skin types. [iv] This study demonstrates that the English version of the BSTI is valid for English speaking patients online, and in doctors’ offices in the US, China and Korea.
Skincare Should Be Designed With The Baumann Skin Type in Mind
Consider The Product Order and Time of Use

AM

PM

DIAGNOSIS: 

<table>
<thead>
<tr>
<th>Time</th>
<th>Product</th>
<th>Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>MORNING</td>
<td>CLEANSER</td>
<td></td>
</tr>
<tr>
<td>STEP 1</td>
<td>EYE PRODUCT</td>
<td></td>
</tr>
<tr>
<td>STEP 2</td>
<td>TREATMENT PRODUCT</td>
<td></td>
</tr>
<tr>
<td>STEP 3</td>
<td>MOISTURIZER</td>
<td></td>
</tr>
<tr>
<td>STEP 4</td>
<td>SUNSCREEN</td>
<td></td>
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<tr>
<td>STEP 5</td>
<td>EVENING</td>
<td></td>
</tr>
<tr>
<td>STEP 1</td>
<td>CLEANSER</td>
<td></td>
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<td>STEP 2</td>
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<tr>
<td>STEP 4</td>
<td>MOISTURIZER</td>
<td></td>
</tr>
<tr>
<td>STEP 5</td>
<td>RETINOID</td>
<td></td>
</tr>
</tbody>
</table>

NOTES: ________________________________
Ingredients Are Chosen By Skin Type

- Which To Use
- Which To Avoid
PRODUCT LAYERING
Ingredients Combine To Form New Compounds With Different Characteristics

Effects:
- Activity
- Penetration
- Solubility
- Stability
Some Ingredients Are Very Reactive

Ascorbic acid
Benzoyl peroxide
Hydroquinone
Peptides
Retinoids
Each product should improve the efficacy of the other products.
Basic Regimen Structure
Methodology of Regimen Structure

AM
1. Cleanser
2. Eye Product
3. Treatment Product
4. Moisturizer
5. Sunscreen

PM
1. Cleanser
2. Eye Product
3. Treatment Product
4. Moisturizer
5. Retinoid
First We Will Focus On:

**AM**
1. Cleanser
2. __________
3. __________
4. Moisturizer
5. __________

**PM**
1. Cleanser
2. __________
3. __________
4. Moisturizer
5. __________
Cleansers

- Prepare Skin
- Remove Lipid
- Deposit Lipids
- Effect pH
- Loosen Cell Attachments
- Promote Desquamation
Cleansers Affect The Skin Barrier
What is the Skin Barrier?
Stratum Corneum
Stratum Corneum

Made of keratinocytes
Keratinocytes are Surrounded by Lipids

Resembles a Brick Wall

Bricks = Keratinocytes

Mortar = Lipids
The Skin Barrier

Lipids form bilayer membranes
Multilamellar structure

- Multiple bilayers
- Protects the keratinocytes
Skin Barrier

Bilayer membrane composed of 3 lipids

- Cholesterol
- Ceramides
- Fatty Acids
A Ratio of 1:1:1 is Optimal

Cholesterol                  Ceramides                   Fatty Acids

Optimization of Physiological Lipid Mixtures for Barrier Repair

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Three stratum corneum lipids, ceramides, cholesterol (CHOL), and free fatty acids (FA), are required for permeability barrier homeostasis. Recent studies have shown that application of one or two of these lipids to perturbed skin delays barrier recovery; only equimolar mixtures allow normal recovery. We asked here whether any physiological lipid mixtures improve barrier repair, as assessed by transepidermal water loss. Whereas an equimolar ratio of ceramides, CHOL, and FA (either the essential fatty acid, linoleic acid, or the nonessential FAs, palmitic or stearic acids) allows normal repair, further acceleration of barrier repair occurs as the ratio of any of these ingredients is increased up to 3-fold. Similar preliminary results were obtained in damaged human skin. Likewise, while acylceramides alone delay barrier recovery, acylceramides:CHOL mixtures within a specific range of molar ratios dramatically improve barrier repair. Furthermore, glycosyl ceramides, sphingomyelin, and triglycerides substitute effectively for ceramides and FA, respectively, but neither phospholipids nor cholesterol esters substitute for FA and CHOL, respectively. These studies show the specific requirements of selected stratum corneum lipid mixtures for optimized barrier repair in murine skin, with further validation in human skin. Utilization of physiologic lipids according to these parameters could lead to new forms of topical therapy for dermatoses (e.g., psoriasis, atopic dermatitis, and irritant dermatitis) triggered by abnormal barrier function. Key words: stratum corneum/bARRIER function/transepidelial water loss/epidermal lipid/epidermal ultrastructure. J Invest Dermatol 106:1096–1106, 1996

Manuscript received October 24, 1995; revised January 9, 1996; accepted for publication January 26, 1996.
Reprint requests to Dr. Peter M. Elias, Dermatology Service (103), Department of Veterans Affairs Medical Center, 4150 Clement Street, San Francisco, CA 94121.
Abbreviations: SC, stratum corneum; CEL, ceramides; CHOL, cholesterol; FAs, fatty acids; L.A., linoleic acid; TEWL, transepidermal water loss; LB, lamellar bodies; FAE, fatty acid esters.
Healthy Skin Barrier
Skin Barrier Prevents TEWL

- **Hydrophilic heads** - *love* water
- **Hydrophobic tails** - *hate* water
Skin Barrier Prevents TEWL
Skin Barrier

An intact multilamellar membrane prevents water movement across the membrane.
Cleansers and the Skin Barrier

<table>
<thead>
<tr>
<th>FOAMING CLEANSER</th>
<th>NON FOAMING CLEANSER</th>
</tr>
</thead>
<tbody>
<tr>
<td>Injure the barrier</td>
<td>Preserve the barrier</td>
</tr>
</tbody>
</table>
Detergents = Surfactants

- Cleansers
- Shampoo
- Soap
- Bubble bath
- Laundry detergent
Foaming Cleansers Damage the Barrier

Detergents (surfactants) pry themselves between lipids.
Disrupted Skin Barrier

TEWL (Transepidermal water loss)

Dehydration
Disrupted Skin Barrier

Allows entry of:
Allergens
Irritants
Bacteria
Sebum Protects the Skin Barrier
Oily Skin

If excessive sebum is present, the detergents will surround sebum instead of barrier lipids.
Sebum is Occlusive
Cleanser Choice Should Consider Presence of Sebum

Oily Skin

Dry Skin
Cleansers Effect The Skin’s pH

- pH 2: Stomach Acid
- pH 2-2.5: Vitamin C serum
- pH 4-6.0: Skin
- pH 6-7.0: P.Acnes Bacteria
- pH 7-8.0: Water
- pH 10: Foaming Cleanser
- pH 12: Household Bleach

Acidic, Neutral, Alkaline
Soap Cleansers

Raise the pH to 9 or 10
Acidic Cleansers

Decrease the pH to 2 - 4
Both exposure of stratum corneum to neutral pH buffers and blockade of acidification mechanisms disturb cutaneous permeability barrier homeostasis and stratum corneum integrity/cohesion, but these approaches all introduce potentially confounding variables. To study the consequences of stratum corneum neutralization, independent of hydration, we applied two chemically unrelated superbases, 1,1,3,3-tetramethylguanidine or 1,8-diazabicyclo [5,4,0] undec-7-ene, in propylene glycol:ethanol (7:3) to hairless mouse skin and assessed whether discrete pH changes alone regulate cutaneous permeability barrier function and stratum corneum integrity/cohesion, as well as the responsible mechanisms. Both 1,1,3,3-tetramethylguanidine and 1,8-diazabicyclo [5,4,0] undec-7-ene applications increased skin surface pH in parallel with abnormalities in both barrier homeostasis and stratum corneum integrity/cohesion. The latter was attributable to rapid activation (<20 min) of serine proteases, assessed by in situ zymography, followed by serine-protease-mediated degradation of corneodesmosomes. Western blotting revealed degradation of desmoglein 1, a key corneodesmosome structural protein, in parallel with loss of corneodesmosomes. Coapplication of serine protease inhibitors with the superbase normalized stratum corneum integrity/cohesion. The superbases also delayed permeability barrier recovery, attributable to decreased β-glucocerebrosidase activity, assessed zymographically, resulting in a lipid-processing defect on electron microscopy. These studies demonstrate unequivocally that stratum corneum neutralization alone provokes stratum corneum functional abnormalities, including aberrant permeability barrier homeostasis and decreased stratum corneum integrity/cohesion, as well as the mechanisms responsible for these abnormalities. **Key words:** corneodesmosome/permeability barrier function/serine protease/serine protease inhibitor/stratum corneum/superbase/transepidermal water loss. *J Invest Dermatol* 121:345–353, 2003
pH Affects Penetration

Topical L-Ascorbic Acid: Percutaneous Absorption Studies

Sheldon R. Pinnell, MD, * Huanshu Yang, MD, ‡ Mostafa Omar, PhD, † Nancy Monteiro Riviere, PhD, ‡ Holly V. DeBuys, MD, * Linda C. Walker, * Yaqhui Wang, MD, § and Mark Levine, MD §

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BACKGROUND. Reactive oxygen species generated by ultraviolet light result in photocarcinogenic and photoaging changes in the skin. Antioxidants protect skin from these insults.

OBJECTIVE. This study defines formulation characteristics for delivering L-ascorbic acid into the skin to supplement the skin’s natural antioxidant reservoir.

METHODS. L-ascorbic acid or its derivatives were applied to pig skin. Skin levels of L-ascorbic acid were measured to determine percutaneous delivery.

RESULTS. L-ascorbic acid must be formulated at pH levels less than 3.5 to enter the skin. Maximal concentration for optimal percutaneous absorption was 20%. Tissue levels were saturated after three daily applications; the half-life of tissue disappearance was about 4 days. Derivatives of ascorbic acid including magnesium ascorbyl phosphate, ascorbyl-6-palmitate, and dehydroascorbic acid did not increase skin levels of L-ascorbic acid.

CONCLUSIONS. Delivery of topical L-ascorbic acid into the skin is critically dependent on formulation characteristics.
Moisturizers

AM
1
2
3
4
5

PM
1
2
3
4
5

MOISTURIZER

MOISTURIZER
Moisturizers

- Can Repair Barrier
- Deposits Lipids
- Affects Penetration
- Affects Treatment Product
  - Efficacy
  - Side effects
Skin Barrier

Bilayer membrane composed of 3 lipids

- Cholesterol
- Ceramides
- Fatty Acids
Fatty Acid Choice Is Critical

Fatty acid type affects membrane permeability

Oleic Acid

Larger spaces between membrane lipids
Ingredients Can Influence Penetration
Oleic Acid Increases Penetration

Mechanism of oleic acid-induced skin penetration enhancement in vivo in humans

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Received 13 September 1994; accepted 13 June 1995

Abstract

The outermost layer of mammalian skin, the stratum corneum (SC), by virtue of its unique architecture, presents a significant barrier to the transdermal delivery of drugs. Penetration enhancers such as oleic acid (OA), which increase skin permeability, appear to act selectively on the extracellular lipids representing the principal regulatory channel for the penetration of small molecules. In vitro studies investigating the mode of action of OA have posited two mechanistic scenarios, which may account for the action of this enhancer: (a) lipid fluidization, and (b) lipid phase separation. In the studies presented here, attenuated total reflectance infrared spectroscopy was used to determine the mode of action of OA in vivo, in vivo. The use of perdeuterated OA ([1H]OA) enabled the behaviour of endogenous lipids to be observed independently of that of the exogenously applied enhancer as a result of their spectrally distinct methylene group vibrations. Human forearm was treated topically with 1 ml of either (a) a solution of 5% (v/v) [1H]OA in ethanol, or (b) ethanol alone, for a period of 16 h. After removal of the delivery systems, the SC at the application site was progressively removed by adhesive tape-stripping, while sequential IR spectra were obtained at each newly exposed surface. In this way, we were able to monitor (a) the distribution profile of [1H]OA across the SC, (b) the conformational order of the SC lipids as a function of depth, and (c) the phase behaviour of the enhancer in the SC. Our results indicate that [1H]OA induces lipid disordering only in the superficial layers of the SC, albeit of a smaller magnitude than that associated with a gel to liquid crystalline conformational change. Additionally, [1H]OA was found to exist in a liquid phase at all levels of the SC spectrascopically examined. These results suggest, therefore, that OA-induced skin penetration enhancement results from a mechanism involving both SC lipid fluidization and phase separation, with the latter probably predominating.
Penetration Enhancers in Moisturizers

- Isopropyl myristate
- Propylene glycol
- Glycerol

<table>
<thead>
<tr>
<th>Chemical class</th>
<th>Compounds</th>
</tr>
</thead>
<tbody>
<tr>
<td>Water</td>
<td>Water</td>
</tr>
<tr>
<td>Hydrocarbons</td>
<td>Alkanes, alkenes, squalene, mineral oil, halogens</td>
</tr>
<tr>
<td>Alcohols</td>
<td>Glycerols, glycols, polyglycols, ethanol, caprylic alcohol</td>
</tr>
<tr>
<td>Acids</td>
<td>Oleic acid, Undecanoic acid and other fatty acids</td>
</tr>
<tr>
<td>Amines</td>
<td>Primary, secondary and tertiary, cyclic and acyclic amines</td>
</tr>
<tr>
<td>Amides</td>
<td>Pyrrolidone(N-methyl-2-pyrrolidone, 2-pyrrolidone)azonies (Azone®) (1-dodecylazacycloheptan-2-one)urea</td>
</tr>
<tr>
<td>Esters</td>
<td>Isopropyl myristate</td>
</tr>
<tr>
<td>Surfactants (anionic, cationic, non-ionic, Zwitterionic)</td>
<td>Sodium lauryl sulfate, cetyltrimethyl ammonium bromide, sorbitan monolaurate, polysorbate 80, dodecyl dimethyl ammoniopropionate sulfate</td>
</tr>
<tr>
<td>Terpenes, terpenoids and essential oils</td>
<td>Menthol, limonene</td>
</tr>
<tr>
<td>Sulfoxides</td>
<td>Dimethyl sulfoxide, dodecyl methyl sulfoxide</td>
</tr>
<tr>
<td>Lipids</td>
<td>Phospholipids</td>
</tr>
</tbody>
</table>

Hyaluronic acid: a unique topical vehicle for the localized delivery of drugs to the skin

Hydrodynamic potential of HA-related drug delivery systems

ABSTRACT

Hyaluronic acid (HA) is a naturally occurring polysaccharide that consists of N-acetyl-D-glucosamine and D-glucuronic acid. It is present in the intercellular matrix of most vertebrate connective tissues, especially skin where it has a protective, structure stabilizing and shock-absorbing role. The unique viscoelastic nature of HA along with its biocompatibility and non-immunogenicity has led to its use in a number of clinical applications, which include the supplementation of joint fluid in arthritis as a surgical aid in eye surgery, and to facilitate the healing and regeneration of surgical wounds. More recently, HA has been investigated as a drug delivery agent for various routes of administration, including ophthalmic, nasal, pulmonary, parenteral and topical. In fact, regulatory approval in the USA, Canada and Europe was granted recently for 5% diclofenac in 2.5% HA gel, Solaraze®, for the topical treatment of actinic keratoses, which is the third most common skin complaint in the USA. The gel is well tolerated, safe and efficacious and provides an attractive, cost-effective alternative to cryosurgery, curettage or dermabrasion, or treatment with 5-fluorouracil. The purpose of this review is to describe briefly the physical, chemical and biological properties of HA together with some details of its medical and pharmaceutical uses with emphasis on this more recent topical application.

Key words: actinic keratoses, dermatology, diclofenac, hyaluronic acid, Solaraze, topical drug delivery
Sebum Can Affect Transdermal Delivery

- Hydration of the SC
- Occlusion
We covered:

**AM**
1. CLEANSER
2. EYE PRODUCT
3. TREATMENT PRODUCT
4. MOISTURIZER
5. SUNSCREEN

**PM**
1. CLEANSER
2. EYE PRODUCT
3. TREATMENT PRODUCT
4. MOISTURIZER
5. RETINOID
Treatment Product Focus

**AM**
1. Cleanser
2. Eye Product
3. Treatment Product
4. Moisturizer
5. Sunscreen

**PM**
1. Cleanser
2. Eye Product
3. Treatment Product
4. Moisturizer
5. Retinoid
Skin Lightening Treatment Products
Skin Pigmentation

Epidermis

Dermis

Melanocytes
Melanosomes

Site of melanin production by tyrosinase.
Epidermal Melanin Unit

PAR-2 Receptor
Targets For Skin Lightening

Cell Structure

Tyrosinase
PAR-2
Melanosomes

Action

Block tyrosinase
Block receptor
Desquamation
Tyrosinase Inhibitors Are Notoriously Unstable and Reactive

Care must be taken when combining with other products
Hydroquinone

- Unstable due to rapid oxidation
- Turns brown when oxidized
- Poor skin penetration because of hydrophilic structure
Arbutin

- Derivative of hydroquinone

- Decomposition is 4x higher at pH of 9 than at pH of 5
pH affects efficacy of products
Ascorbic acid

- Photoinstability
- Poor absorption

Topical L-Ascorbic Acid: Percutaneous Absorption Studies

Sheldon R. Pinnell, MD, * Huanshu Yang, MD, ‡ Mostafa Omar, PhD, † Nancy Monteiro Riviere, PhD, ‡ Holly V. DeBuys, MD, * Linda C. Walker, * Yaohui Wang, MD, § and Mark Levine, MD §

* Duke University Medical Center, Durham, North Carolina, † PhytoCeuticals, Elmwood Park, New Jersey, ‡ College of Veterinary Medicine, North Carolina State University, Raleigh, North Carolina, and § National Institute of Diabetes & Digestive & Kidney Diseases, NIH, Bethesda, Maryland
Enhancing Tyrosinase Inhibitor Efficacy

- Light and air avoidance
- Penetration enhancers to increase absorption
- Combine with antioxidants
- Avoidance of oxidizing agents such as:
  - benzoyl peroxide
  - hydrogen peroxide
Hydroquinone Should Be Used with an Antioxidant Product Because it Causes:

- Depletion of glutathione
- Generation of reactive oxygen species
- Oxidative damage of membrane lipids and proteins
Cleansers and Moisturizers Effect Efficacy

- Solubility
- Stability
- Hydration of the SC
- Desquamation
- The pH
- Occlusion
Proteasomes are multicatalytic protease complexes within cells that selectively degrade ubiquitinated proteins. We have recently demonstrated that fatty acids, major components of cell membranes, are able to regulate the proteasomal degradation of tyrosinase, a critical enzyme required for melatonin biosynthesis, in contrasting manners by relative increases or decreases in the ubiquitinated tyrosinase. In the present study, we show that altering the intracellular composition of fatty acids affects the post-Golgi degradation of tyrosinase. Incubation with linoleic acid (C18:2) dramatically changed the fatty acid composition of the endoplasmic reticulum (ER) and the degradation of tyrosinase was increased after its maturation in the Golgi. Retention of tyrosinase in the ER was observed when cells were treated with linoleic acid in the presence of proteasome inhibitors, explaining why melanin synthesis was decreased in cells treated with linoleic acid and a proteasome inhibitor despite the abrogation of tyrosinase degradation. These results suggest that the intracellular composition of fatty acids affects the processing and function of tyrosinase in connection with the ubiquitin–proteasome pathway and suggest that this might be a common physiological approach to regulate protein degradation.

**Abstract** This study was conducted to evaluate the effects of mono-/polyunsaturated fatty acids on ultraviolet-induced hyperpigmentation of the skin. An efficient lightening effect was observed following topical application of linoleic acid or α-linolenic acid to UV-stimulated hyperpigmented dorsal skin of brownish guinea pigs. The number of melanocytes in the treated skin was similar to the number in the skin of the pigmented control, indicating that the pigment-lightening effect was not due to depletion of melanocytes. In vitro experiments using cultured murine melanoma cells showed that melanin production was inhibited most effectively by γ-linolenic acid, followed by linoleic acid and then by oleic acid. Furthermore, the turnover of the stratum corneum, which plays an important role in the removal of melanin pigment from the epidermis, was accelerated by linoleic acid and by γ-linolenic acid. Taken together, the results suggest that the pigment-lightening effects of linoleic acid and α-linolenic acid are, at least in part, due to suppression of melanin production by active melanocytes, and to enhanced desquamation of melanin pigment from the epiderm. is not possible in mammals [9, 35, 36]. The upstream fatty acids of these strains are oleic acid (OA, 18:1n-9), linoleic acid (LA, 18:2n-6), and α-linolenic acid (α-LNA, 18:3n-3), respectively (Fig. 1). UFAs and their metabolites have been used as therapeutic agents in patients with various skin diseases [5, 6, 34]. The physiological functions of fatty acids in the skin have been investigated by analyzing syndromes of essential fatty acid deficiency [28, 29], and early studies have shown that such deficiencies induce loss of epidermal barrier function [4, 9]. The administration of LA to essential fatty acid-deficient ani- mals not only restores the barrier function of their epider- mals, but also reduces the redness and scaling of the dis- eased skin [10].

Melanin synthesis is enzymatically regulated by the rate-limiting enzyme tyrosinase. Tyrosinase (EC 1.14.18.1) is a bifunctional copper-containing enzyme which plays a pivotal role in the modulation of melanin production [23, 26]. Initially by catalyzing the hydroxylation of ty- rosine to dopa, and, in a second enzymic step, by cata- lyzing the oxidation of dopa to dopaquinone. Melanin pigment is a heterogeneous biopolymer formed from various intermediate products derived from dopaquinone; it is synthesized within melanocytes in the epidermis. To study mammalian melanogenesis in vivo, and particularly to evaluate the effects of chemical and physical agents on skin pigmentation, guinea pigs [1, 11, 12, 15-17, 19, 31]
Fatty Acids Effect
Skin Lightening Product Efficacy

Unsaturated fatty acids:
- α-linolenic acid, linoleic acid, oleic acid
- Decrease tyrosinase function
- Linoleic acid and by α-linolenic acid
- Increase cell turnover of the stratum corneum

Saturated fatty acids
- Palmitic acid
- Increases tyrosinase function

Tyrosinase function

α-Linoleic acid  Linoleic acid  Oleic acid  Palmitic acid

baseline
Designing An Efficacious Regimen Takes Time.
Use a Methodology To Prepare Regimens and Patient Instructions Ahead of Time

I use software to generate regimens!
Email me at
DrB@SkinTypeSolutions.com
or text Manny at
786-512-1674

Thanks for your attention!
Leslie Baumann MD, FAAD