The Baumann Skin Typing System 117

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Abstract

The biology of various skin phenotypes, such as oily, dry, acne-prone, rosacea-prone, dyschromia, and photoaged, affects the interaction and efficacy of cosmeceutical ingredients. This chapter will review how cosmeceuticals influence basic skin biology and how they interact with each other in various patient phenotypes, as characterized by the Baumann Skin Typing System (BSTS). Developed in 2004, the BSTS, derived from a scientifically validated questionnaire, offers specific guidance for physicians and patients/consumers in identifying the most suitable ingredients and skin products as it takes into account multiple concurrent cutaneous characteristics and gathers historical data. The reader will be provided with knowledge of the basic science of different skin issues and will obtain a scientific perspective on how to design skin care regimens, combine cosmeceuticals with prescription medications, educate staff and patients on their proper use, and ethically prescribe skin care products using this standardized methodology.

Introduction

As of 2011, the cosmeceutical industry was considered to be a $6.5 billion business [1], and global sales of topical cosmeceuticals surpassed $33 billion in 2012, with projected sales exceeding $42 billion by 2017 [2]. The promise of

financial success has resulted in the discovery of many new cosmeceutical technologies and significant advances in understanding skin science and the effects on skin by topically applied substances. The emergence of many new ingredients and topical formulations has manifested in an abundance of products on the market, exaggerated marketing claims, and confusion on the part of consumers, aestheticians, and physicians. The major current challenge is to sift through the copious technologies and formulations and match cosmeceutical technologies to the appropriate type of skin. As new genetic data lead to advances in skin care technologies, the importance of the genotype as well as the phenotype cannot be overstated. An ingredient is only efficacious when it is placed on the correct skin phenotype. Combining ingredients affects their chemical structure, efficacy, and penetration; therefore, the order in which products are placed on the skin is important. The phenotype of the skin will also affect how ingredients penetrate into the skin and react with each other. In order to maximize outcomes with cosmeceuticals, there are many steps that should be taken to properly match a skin care regimen with the proper skin phenotype (Table 1).

The Baumann Skin Typing System (BSTS) was developed in 2004 and classifies skin according to four dichotomous parameters: dry or oily, sensitive or resistant, pigmented or nonpigmented, and wrinkled or unwrinkled. Simultaneously assessing the skin based on these four non-mutually exclusive parameters yields 16 potential and distinct skin phenotypes (Table 2). The Baumann Skin Type® designations for each phenotype are derived from a scientifically validated questionnaire known as the Baumann Skin Type Indicator (BSTI). The Baumann Skin Type can change after significant alterations in lifestyle habits, hormones, medical condition, and environment and therefore should be retaken annually or when the current skin care regimen seems insufficient [3, 4].

Table 1 How to maximize outcomes from cosmeceuticals

1. Know ingredient science
2. Understand cosmeceutical formulation
3. Understand manufacturing and packaging
4. Understand ingredient interactions
5. Choose the best products from each brand
6. Combine products from various brands to form set regimens
7. Diagnose the patient's Baumann Skin Type® using the validated BSTI (5)
8. Match the regimen to the correct skin type
9. Test the regimens on each individual skin type
10. Take baseline photographs and standardized measurements
11. Educate your patients (clients) to increase compliance
12. Schedule regular follow-up visits to adjust the regimen
13. Ensure proper use of the correct products

The BSTS, by nature, offers specific guidance for physicians and patients/consumers in identifying the most suitable ingredients and skin products as it takes into account multiple concurrent cutaneous characteristics and gathers historical data. The questionnaire probes how the skin reacts in various situations, allowing the collection of historical data and the current skin condition.

Each of the distinct Baumann Skin Types display characteristics that determine which ingredients are beneficial and which are deleterious. For example, a person with dry, sensitive, pigmented, wrinkle-prone skin (DSPW) would require significantly different skin care products than a person with oily, resistant, nonpigmented, "tight" (not wrinkle-prone) skin (ORNT). Oily-resistant skin types have a strong skin barrier with an extra layer of sebum to prevent penetration of ingredients; therefore, individuals with this skin type require an increased concentration of active ingredients or penetration enhancers to demonstrate efficacy. Dry, sensitive skin types, on the other hand, have an impaired barrier that allows increased penetration of ingredients; these individuals are more prone to inflammation. In the DSPW type, the skin-lightening ingredients and antiaging ingredients that are required should not be ones that will incite inflammation. Lower concentrations can be used because of the defect in the skin barrier.More than 300,000 people worldwide have taken the BSTI, and it is used by multiple dermatologists around the world to diagnose patient skin phenotype. It has been shown to be valid for all ethnicities, ages, and genders. The most recent version

Table 2 **The BSTS skin type paradigm.** Each of the Baumann Skin Types® is assigned a color and a number

of the BSTI questionnaire is only accessible online to physicians who have met various criteria and completed a training program to ensure the proper use of ingredients [5]. The BSTI is instrumental in diagnosing skin type and helping the doctor or designee prescribe the proper cosmeceuticals, over-the-counter and prescription medications, and procedures that are most appropriate for the patient's Baumann Skin Type®. The non-identifying data culled from the BSTI has the potential to broaden our knowledge of skin types, their prevalence around the world, and how to improve patient outcomes. It also allows standardization in study subject recruitment so that only certain skin phenotypes are included in research studies. The BSTI and its use are beyond the scope of this chapter; however, information on how to apply to be chosen to use this system can be found at STSFranchise.com. The program is designed to be used under the guidance of a physician and incorporates prescription and nonprescription products and in-office therapies.

The four parameters on which the BSTI is based will guide the discussion in this chapter. Emphasis will be placed on defining the characteristics of these dichotomies and focusing on pertinent basic science. Various aspects of the 16 skin phenotype variations will be described in the process. Cutaneous aging is explained in the context of the wrinkled (W) to tight (T) continuum. Approaches to skin care or treatment options that follow from the BSTS will also be cited, with noninvasive, mostly topical therapies addressed.

Skin Hydration: The Oily (O) To Dry (D) Continuum

Oily skin is caused by the excess production of sebum; dry skin, also known as xerosis, is associated with a complex, multifactorial etiology including an impaired barrier. Individuals with oily skin often are unable to find a sunscreen that they can tolerate due to greasiness, while people with dry skin types are more easily irritated by retinoids and acne medications. The most important factors that regulate the degree of dryness/oiliness are stratum corneum lipids, sebum, natural moisturizing factor, and aquaporin, a discussion of which follows [6].

Stratum Corneum (SC)

The role of the stratum corneum (SC), particularly its ability to maintain skin hydration, is the most significant factor in preventing or setting the stage for xerosis. The SC is composed of keratinocytes surrounded by a coating of ceramides, cholesterol, and fatty acids, among other constituents. These primary components of the SC, when present in the appropriate amount and balance, assist in protecting the skin and keeping it watertight. The stimulation of lipid synthesis and keratinocyte proliferation by multiple factors, including diet, medications, hormones, and immunomodulators such as cytokines, contributes to maintaining SC equilibrium [7].

When ceramides, cholesterol, and fatty acids are imbalanced, the SC endures a cascade of interrelated events, resulting in a reduced capacity to maintain water and increased susceptibility to exogenous elements, thereby elevating susceptibility to skin surface sensitivity and xerosis. Such an impairment in the SC is measurable as an increase in transepidermal water loss (TEWL). When the skin becomes dehydrated, the desquamation of corneocytes is adversely affected because the enzymes necessary for desmosome metabolism require water [8]. The abnormal or compromised desquamation leads to a visible collection of keratinocytes manifesting in skin surface irregularities that result in skin that feels rough and loses radiance because it poorly reflects light [9]. This is a common complaint of patients that say that their skin "looks old" or "is no longer glowing." Many skin care companies take advantage of this imbalance by offering demonstrations of their products that exfoliate, resulting in an instant glow to the skin that often prompts the customer to purchase the product. However, these exfoliating products are treating the symptom of roughness but not the underlying cause, which is dehydration.

Not all moisturizers that contain ceramides, cholesterol, and fatty acids are effective at repairing the skin barrier because the ceramides and fatty acids must mimic the naturally occurring three-dimensional structure of the multilamellar bilayer of lipids known as the skin barrier. For example, a moisturizer that contains increased fatty acid levels and decreased ceramide levels causes a perturbation in the lipid bilayer of the SC, which is also linked to xerosis [10]. For this reason, moisturizers designed to repair the skin barrier must contain the proper ratio of fatty acids, ceramides, and cholesterol. In addition, the type of fatty acid is important. Oleic acid, because of the structure of its fatty acid chains, causes tiny holes in the skin barrier, while other fatty acids such as stearic acid are able to pack in closer together, thereby strengthening the skin barrier [11]. There are synthetic "multilamellar emulsions" such as MLE technology (myristoyl/palmitoyl oxostearamide/arachamide MEA) that utilize fatty acids and ceramides that mimic the same three-dimensional structure as the native bilayer membrane [12].

The skin's protective lipid bilayer needs constant replenishment in those whose underlying mechanisms are insufficient to produce adequate amounts of ceramides, fatty acids, and cholesterol because this lipid bilayer is susceptible to deleterious effects induced by exogenous factors such as ultraviolet (UV) radiation, detergents, acetone, chlorine, and prolonged water exposure. Changes in temperature, humidity, and pH affect cohesion and desquamation of corneocytes from the SC by activating numerous extracellular proteases [13],

which in turn can impact the skin's protective layer by thinning or thickening the SC.

Improving skin hydration by strengthening the skin barrier in dry skin types begins with the selection of a nonfoaming cleanser. Foaming cleansers should be avoided because they often have detergents (surfactants) that surround and remove the lipids from the SC. A barrier repair moisturizer should be used twice a day and should contain a 1:1:1 ratio of ceramides, fatty acids, and cholesterol. The optimal fatty acids are linoleic, palmitic, and stearic acids, which have been shown to repair the skin barrier within 2 h when applied exogenously [14]. These fatty acids are found in large amounts in some natural ingredients such as almond oil, argan oil, shea butter, sunflower oil, safflower oil, grape seed extract, and macadamia oil, as well as in synthetic ingredients such as glyceryl stearate and myristoyl/palmitoyl oxostearamide/arachamide MEA.

Natural Moisturizing Factor (NMF)

Natural moisturizing factor (NMF) is an intracellular, hygroscopic substance present only in the SC. Low NMF levels are correlated with xerosis and ichthyosis vulgaris. NMF is produced by lamellar bodies via the breakdown of the protein filaggrin (or filament-aggregating protein). Filaggrin is composed of lactic acid, urea, citrate, and sugars and is broken down into free amino acids, such as arginine, glutamine (glutamic acid), and histidine by a cytosolic protease in the outer layer of the SC [15]. These water-soluble free amino acids remain inside the keratinocytes as NMF and play an integral intracellular humectant role by maintaining water within skin cells. The process of NMF production is elegantly controlled by ambient humidity levels. Aspartate protease (cathepsin), the enzyme responsible for the rate of filaggrin decomposition, has been shown to be susceptible to changes in ambient humidity, allowing fluctuations in NMF production [16]. After an individual enters a low-humidity environment, NMF synthesis usually increases over the course of several days [17]. UV radiation as well as surfactants (detergents) can suppress the development of NMF.

As of yet, no products or procedures have been developed that have the capacity to artificially influence or regulate NMF synthesis because its intracellular location makes it difficult to replace topically. The best course of action is to avoid foaming cleansers (detergents) and UV exposure to preserve native NMF.

Aquaporin-3

Aquaporin (AQP)-3 is member of a subclass of aquaporins labeled aquaglyceroporins, which selectively transport water, glycerol, urea, and other small solutes through a water channel protein. AQP-3 exerts an influential role in skin hydration by regulating this water channel [18]. AQP-3 is found in the kidney, lungs, and GI tract and in human epidermal keratinocytes [19]. The water conduction function in the skin occurs along an osmotic gradient beneath the SC, thereby facilitating the hydration of skin layers below the SC.

In the superficial SC, a high concentration of solutes (Na^+, K^+, and Cl^-) and a low concentration of water (13–35 %) [20] generate in the steady-state gradients of solutes and water from the skin surface to the viable epidermal keratinocytes [21–23]. The molecular mechanisms of fluid transport across epidermal keratinocyte layers and the relationship between keratinocyte fluid transport and SC hydration have not been elucidated. It is hypothesized that AQP-3 enhances transepidermal water movement to protect the SC from evaporation from the skin surface and/or to spread water gradients throughout the epidermal keratinocyte layer [19]. In one study, researchers noted that the water permeability of human epidermal keratinocytes was inhibited by mercurials and low pH, which was consistent with AQP-3 involvement [19]. Another study found significantly lower water and glycerol permeability, and conductance measurements revealed much lower SC water content in AQP-3 null mice,

supporting previous evidence that AQP-3 functions as a plasma membrane water/glycerol transporter in the epidermis [24]. Water transport across AQP-3 was found to be slower in skin than in other tissues [25].

Despite the fact that some cosmeceutical manufacturers boast of aquaporin on their ingredient lists, it is not possible to exogenously insert AQP-3 into the keratinocyte membrane. At this time, the only way to affect aquaporin function with a topical formulation is with extracts that stimulate AQP-3 function. A solitary example is an extract of the herb *Ajuga turkestanica*, which has been shown to enhance the activity of AQP-3 [26]. At the time of publication of this chapter, no other ingredients have been able to mimic this activity.

Sebum

Sebum is the oily secretion of the sebaceous glands that contains wax esters, sterol esters, cholesterol, di- and triglycerides, and squalene. Sebum imparts an oily protective film on the surface of the skin but also plays a causal role in the formation of comedones and acne [27]. The exact composition of sebum is determined by genetics, diet, medications, and other poorly understood factors. Sebum, which is an important source of the antioxidant vitamin E, confers cutaneous protection from exogenous factors such as UV light. It also functions as an occlusive moisturizer on the skin's surface, impeding TEWL. Subjects with an impairment in the lipid bilayer surrounding the keratinocytes in the SC skin may suffer less from dehydration and irritant (or allergen)-mediated inflammation when a higher level of protective sebum is present. When sebum production is below the normal range, it is thought by some to play a role in the development of dry skin, especially when it coincides with an impaired skin barrier [28]. It is important to note that low sebaceous gland activity has not been shown to cause xerosis in the absence of an impaired skin barrier. In fact, the protective role that sebum plays in preventing skin xerosis seems to be independent of skin barrier function because skin with few sebaceous glands, as in prepubertal children, can manifest normal skin barrier function [29]. In addition, barrier function or SC lamellar membranes are not impacted by the pharmacologic involution of sebaceous glands with supraphysiologic isotretinoin doses resulting in dry skin [30–32].

Sebum likely exerts its skin-hydrating effect through occlusive activity rather than an influence on barrier function. The protective effect of glandular lipids is demonstrated by the meibomian glands, which are modified sebaceous glands located in the eyes that have the capacity to stave off dryness by preventing tear evaporation [33, 34]. The separate role of sebum in skin hydration was demonstrated in a study that assessed permeability barrier homeostasis and SC hydration in asebia J1 mice with sebaceous gland hypoplasia [35]. The asebia mice had consistent levels of the three primary barrier lipids (ceramides, free sterols, and free fatty acids) and normal barrier function but were sebum deficient. The researchers demonstrated that the asebia J1 mice manifested reduced SC hydration, implying that while an intact intercellular membrane bilayer system suffices for permeability barrier homeostasis, the presence of sebum is necessary for normal SC hydration.

The investigators in the asebia J1 study noted that the topical application of glycerol restored normal SC hydration to the sebum-deficient mice. Glycerol is a humectant that helps bind water to the skin's surface. Sebaceous gland-derived triglycerides (TG) are hydrolyzed to glycerol before transport to the skin surface in normal skin. In fact, the use of glycerol has also been demonstrated to be effective in accelerating SC recovery [36]. For this reason, sebum has both an occlusive and a humectant effect on the skin's surface and, through hydrolyzation of its triglycerides to glycerol, has the ability to hasten barrier repair.

Many factors contribute to sebum production rates. The age-related trajectory of sebum production levels is well understood. During childhood, sebum levels are usually low, then rise in the middle-to-late teens, and remain relatively stable for decades until declining in the 7th and 8th decades as endogenous androgen production falls

[37]. Sebum production is also affected by diet and stress, but the influence of these is not well understood. Androgenic hormones are well known to increase sebaceous gland activity. Genetic predisposition to sebum production was demonstrated in a fascinating study of 20 pairs each of identical and nonidentical like-sex twins. Almost equivalent sebum excretion rates were observed in the identical twins, but there were significant variations in sebum production among the nonidentical twins, demonstrating the strong genetic influence on sebum secretion rates [38].

Differences in Skin Care for Oily and Dry Skin

An intact SC and skin barrier, normal levels of NMF and hyaluronic acid (HA), normal AQP-3 activity, and balanced sebum secretion together characterize an ideal cutaneous state. The goal of a properly designed skin care regimen is to achieve this idealized state by removing excess sebum in oily skin types and repairing the skin barrier in dry skin types.

Oily Skin Care

Treatment of individuals with oily skin should be aimed at decreasing surface sebum levels with surfactant-containing foaming cleansers because there are no topical ingredients that have convincingly demonstrated the ability to decrease sebum production, despite manufacturer claims to the contrary. Oral retinoids, oral spironolactone, and oral contraceptives have been well established as effective in reducing sebaceous gland activity, but topical retinoids and topical antiandrogens have not yet been demonstrated to exhibit this capacity. Oily skin types should avoid lipid-laden moisturizers such as heavy creams and oils. Gels, serums, and light lotions are a better choice for oily skin types. Oily types often omit sunscreen because of the greasiness associated with chemical sunscreen ingredients and dimethicone, which is often found in SPF preparations. Choosing an SPF that does not have oily components will increase sunscreen compliance in oily types. Omitting a moisturizer in the morning and using a sunscreen instead is one way to increase sunscreen compliance in oily skin types. Oily skin types must cleanse the face completely at night to remove make up, sunscreen, dimethicone, dirt, and other debris that can contribute to comedone formation.

Dry Skin Care

An impaired skin barrier and diminished NMF characterize xerotic skin. Skin care should aim to preserve and replace skin lipids. Harsh foaming detergents (present in hand, body, and facial cleansers) strip lipids and NMF from the skin and should be avoided by all patients with dry skin. Individuals with dry skin should also be advised to abstain from protracted bathing, especially in hot or chlorinated water. People with extremely dry skin use humidifiers in low-humidity environments and apply moisturizers two to three times daily and after bathing. Moisturizers should include barrier repair ingredients in a 1:1:1 ratio of ceramides, fatty acids, and cholesterol. In addition, occlusive and humectant ingredients can be added to boost skin hydration. Dry skin is very prevalent, especially in the winter as evidenced by the fact that of all the OTC topical skin care product types, moisturizers are the third most frequently recommended [39]. It is important to remember that sebaceous glands are only found on the face, back, and chest. Therefore, some patients who demonstrate oily facial skin that is masking an impaired skin barrier will exhibit dry skin on the limbs and body.

Moisturizers are typically packaged as water-in-oil emulsions or oil-in-water emulsions or as an oil. A brief discussion follows of the differences among moisturizer types, which is important to a practitioner's knowledge base in terms of offering appropriate product selection recommendations to patients.

Occlusives

Occlusive agents are lipid-filled compounds that mimic the effects of sebum and are incorporated

into skin care formulations in order to coat the SC and prevent TEWL. In addition to inhibiting TEWL, occlusives exhibit emollient properties and are therefore appropriate products for smoothing the roughness associated with dry skin.

Petrolatum and mineral oil were once thought to be the most effective occlusive ingredients. Used as a skin care product since 1872, petrolatum was considered one of the best moisturizers and is still the gold standard by which other occlusive agents are measured [40]. For example, petrolatum displays a resistance to water vapor loss that is 170 times that of olive oil [41]. Many consumers deem petrolatum to be cosmetically unacceptable because of its greasy texture, and prefer a more "environmentally friendly" option, but efficacy of moisturizers is still often compared to that of petrolatum. Other frequently used occlusive ingredients include paraffin, squalene, silicone derivatives (dimethicone, cyclomethicone), almond oil, argan oil, soybean oil, grape seed oil, macadamia nut oil, propylene glycol, lanolin, lecithin, stearyl stearate, and beeswax [42, 43].

Lanolin, which is derived from the sebaceous secretions of sheep, warrants special mention. It contains the important SC lipid cholesterol and can coexist with SC lipids as solids and liquids at physiologic temperatures. However, lanolin has been identified as an allergen [44] and is derived from animals, which has greatly lessened its popularity. Functioning as both a humectant and an occlusive agent, propylene glycol (PG) is an odorless liquid that also exhibits antimicrobial and keratolytic activity. In addition, PG has been demonstrated to contribute to the cellular penetration of some drugs, such as minoxidil and steroids. Although believed to be a weak sensitizer, PG may provoke or factor into contact dermatitis by facilitating allergen penetration into the epidermis [45].

Occlusive ingredients are a temporary solution for smoothing skin and helping prevent TEWL, but they are not a replacement for barrier repair ingredients, and they do not confer long-lasting benefits. Once an occlusive product is removed from the skin, TEWL returns to its previous level. The reduction of TEWL by more than 40 %, which can result from overuse of occlusive agents, poses a risk of maceration with increased bacteria levels; therefore, occlusive agents are typically used in combination with humectant ingredients to decrease the amount of occlusion needed to restore hydration [46].

Humectants

Humectants are hygroscopic, water-soluble substances that strongly bind water. In conditions with at least 80 % humidity, humectants applied to the skin exhibit the capacity to attract water from the external environment to the skin surface. In low-humidity conditions, however, humectants applied to the skin can absorb water from the deeper epidermis and dermis, thus contributing to TEWL and exacerbating xerosis [47]. Combining humectants with occlusive products decreases TEWL and skin dehydration in a low-humidity environment. Cosmetic moisturizers are often formulated with humectants in order to prevent product evaporation and thickening, thus extending the product's shelf-life. By drawing water into the skin, humectants engender a minor swelling of the SC, leaving a perception of smoother skin with fewer wrinkles, but these effects are temporary. Some humectants impart other benefits such as emollient and bacteriostatic properties [48]. Glycerin and glycerol are considered the most effective humectant ingredients found in skin care products. Alpha hydroxy acids, carboxylic acid, gelatin, honey and other sugars, panthenol, propylene glycol, sodium hyaluronate, sodium and ammonium lactate, sodium pyrrolidine carboxylic acid, sorbitol, and urea are among other substances that function as active humectant ingredients [43]. Moisturizers typically incorporate occlusive as well as humectant ingredients, but the benefits of these are short lived. Some barrier repair moisturizers incorporate occlusives and humectants so that the preparation will have both long-term and short-term benefits.

Glycerin

Glycerin is a potent humectant [49]. Using ultrastructural analyses of skin treated with high-

glycerin formulations, investigators have demonstrated that this humectant expands the SC by enhancing corneocyte thickness and creating greater distance between corneocyte layers [50]. In addition, after a 5-year study that compared two high-glycerin moisturizers with 16 other popular moisturizers, including petrolatum preparations, used by 394 patients with severe xerosis, researchers reported that the high-glycerin products were the most effective, rapidly restoring dry skin to normal hydration with longer-lasting results than the other products [9]. Glycerin has also been shown to stabilize and hydrate cell membranes along with the enzymes essential for desmosome degradation [9]. Glycerin is another name for glycerol. Glycerol forms the backbone of fatty acids and is therefore released when fatty acids are digested [51].

Urea

Also known as carbamide, urea is an end product of mammalian protein metabolism as well as an NMF constituent. This versatile compound exhibits humectant and mild antipruritic activity [52]. Urea has been included as an ingredient in several hand cream formulations since the 1940s [53]. In addition, it has been successfully used in combination with hydrocortisone, retinoic acid, and other ingredients to facilitate the cutaneous penetration of these agents [54, 55]. However, despite such findings in the mid-to-late 1980s, skepticism lingered regarding the ability of urea to promote such action. In 2005, the Cosmetic Ingredient Review (CIR) Expert Panel declared that urea does indeed have the capacity to enhance the percutaneous absorption of other chemicals and, further, that urea is safe for use in cosmetic products [56]. Regarding its humectant activity, a 3-week double-blind study comparing 3–10 % urea cream revealed the study formulations to be more effective in ameliorating clinical signs of dry skin than the vehicle control. Both creams successfully reduced scaling and enhanced hydration. The 3 % cream caused the skin to appear gold or yellow and had no impact on TEWL, whereas the 10 % cream reduced TEWL, although subjects reported the creams to be equally effective [57].

Hydroxy Acids

Alpha hydroxy acids (AHAs) are naturally occurring organic acids that have been discovered to display humectant and exfoliant activity. Glycolic and lactic acids, respectively derived from sugar cane and sour milk, are the AHAs most often used in moisturizing products and were the first ones to become commercially available. Citric, malic, and tartaric acids are among the other AHAs. Topical preparations that contain AHAs were demonstrated more than 40 years ago to confer significant effects on epidermal keratinization [58]. Nearly 20 years ago, glycolic acid was shown to act as a photoprotective agent [59]. Salicylic acid, the only beta hydroxy acid (BHA), is derived from willow bark, wintergreen leaves, and sweet birch. BHA functions as a chemical exfoliant and is found in synthetic form in several topical formulations [60]. At the lowest levels of the SC, corneocyte cohesiveness is attacked and eroded by AHAs and BHA, influencing pH in the process, as these ingredients break down desmosomes, thus contributing to desquamation [61, 62].

Lactic acid is an AHA as well as a component of NMF. Lactic acid was first used as part of the dermatologic armamentarium in 1943 for the treatment of ichthyosis [63]. Since then, in vitro and in vivo experiments have demonstrated that lactic acid can augment ceramide synthesis by keratinocytes [53, 64]. This moisturizing AHA ingredient has also been shown to combat signs of photoaging. Specifically, 8 % L-lactic acid was found to be superior to the vehicle in a double-blind vehicle-controlled study, with statistically significant improvements measured in sallowness, skin coarseness, and blotchiness [65].

Emollients

Emollients are substances that fill in the gaps between desquamating corneocytes, yielding a smooth skin surface [42]. In addition, emollient formulations improve cohesion, flattening out the curled edges of individual corneocytes [43]. A smoother skin surface, in turn, lessens friction while enhancing light refraction. Emollients are composed primarily of lipids and oils and may also fall into the categories of occlusives or humectants. Emollients are included in cosmetics

to hydrate, soften, and smooth the skin. Emollient ingredients are divided into classes of compounds, including those that exhibit astringent, desiccating, fatting, protective, and protein-rejuvenating activity [49].

Reports of adverse effects linked to moisturizing agents are very rare; reactions are more likely to be due to other ingredients added to the formula. There have been reports of allergic contact dermatitis associated with products that contain preservatives, perfumes, solubilizers, sunscreens, and some other classes of compounds. Specifically, cases of contact dermatitis associated with lanolin, propylene glycol, vitamin E, and Kathon CG have been reported [66, 67].

Collagen and Polypeptide Ingredients

It is important for physicians and patients to know that the preponderance of collagen "extracts" contained in the host of expensive moisturizers touted for the capacity to restore collagen lost due to aging has a molecular weight of 15,000–50,000 Da, but only compounds with a molecular weight of 5000 Da or less can actually penetrate the SC [46]. In other words, these products cannot deliver on their advertised claims of replacing collagen. However, the collagen and other hydrolyzed proteins and polypeptides yield a temporary film on the epidermis that, upon drying, fills in surface depressions and other irregularities. Essentially, the film generated by these products provides a subtle stretching out of fine skin wrinkles. Using a humectant product can further enhance the fuller or somewhat plumper appearance created by collagen and polypeptide ingredients. Formulations that contain collagen and polypeptide ingredients have little or no effect on TEWL but are usually labeled as moisturizers and firming creams.

Skin Sensitivity: The Sensitive (S) to Resistant (R) Continuum

Sensitive skin is defined as skin that is susceptible to inflammation, while resistant skin is not. Individuals with resistant skin rarely experience acne, rosacea, stinging skin, or contact/irritant dermatitis. Although resistant skin is as likely to sunburn as other skin types, it has less of a chance of inflammation due to other causes (such as allergens, irritants, and friction) as compared to sensitive skin types. In terms of skin care product usage, resistant skin might be considered a double-edged sword, because individuals with resistant skin can use most skin care products without experiencing inflammation or irritation, but products may demonstrate less efficacy because of less ingredient penetration through resistant skin. Individuals with resistant skin will achieve better efficacy when hydroxy acids or penetration-enhancing ingredients are added to the skin care regimen.

Sensitive skin is an increasingly common complaint, which may be due in part to the popularity of botanical ingredients and fragrances that can incite an allergic response [68]. Sensitive skin can be accurately categorized into four discrete subtypes: Type 1 (acne type) exhibits the proclivity to develop acne; Type 2 (rosacea type) has the propensity to experience facial redness; Type 3 (stinging type) suffers from episodes of stinging or burning sensations; and Type 4 (allergic type) is prone to allergic and irritant reactions resulting in erythema, pruritus, and skin flaking. Such variations in sensitive skin characteristics present treatment challenges to the consumer as products marketed for "sensitive skin" are not specific about which subtype they are formulated for and therefore not suitable for all sensitive skin subtypes. The four subtypes of sensitive skin share one salient quality: inflammation. Consequently, any sensitive skin treatment program must focus on reducing and eradicating inflammation and should be undertaken under the care of a physician who is knowledgeable about the underlying etiology of the sensitive skin subtypes. Patients may suffer simultaneously from more than one sensitive subtype, thus requiring more complex treatment regimens.

Acne Type

Acne is estimated to affect 40–50 million people in the USA annually [69] and is easily the most

common skin disease [70]. Teenagers and adult women are particularly susceptible. The confluence of four primary factors has been implicated in the pathogenesis of acne including elevated sebum production, a buildup of incompletely desquamated keratinocytes inside the hair follicles, the presence of *Propionibacterium acnes* bacteria, and inflammation. The characteristic cycle of acne is the adherence of dead keratinocytes in the hair follicles due to augmented sebum production, leading to comedones and increased levels of *P. acnes*, followed by initiation of inflammatory cascades by toll-like receptors and other cell signaling mechanisms. Initiation of the inflammatory response results in the development of papules and pustules. The acne cycle from buildup of keratinocytes in the hair follicle to the resulting pustule typically takes 8 weeks. For this reason, acne treatment regimens must include continuous preventative measures in order to break the acne cycle.

Acne therapy targets the four primary etiologic factors: decreasing sebum production (with retinoids or oral contraceptives), unclogging pores (with retinoids or hydroxy acids), stabilizing keratinization (with retinoids and hydroxy acids), eliminating bacteria (with benzoyl peroxide, antibiotics, antimicrobials, blue light, or silver), and reducing inflammation.

Rosacea Type

Approximately 16 million Americans, usually adults between 25 and 60 years of age, are affected by rosacea [71]. Rosacea overlaps with acne because many rosacea sufferers also exhibit papules and pustules in addition to characteristic facial redness, flushing, and the formation of prominent telangiectasias. Although the pathophysiology of rosacea remains to be elucidated, there are many unproven hypotheses about the cause(s). Recent topical vasoconstrictive medications have been used to successfully treat the facial flushing of rosacea through alpha-agonist activity; however, these treat the symptoms rather than the underlying cause of rosacea. In addition to vasoconstrictive topical medications, rosacea therapy should focus on addition of anti-inflammatory ingredients to the diet and skin care regimen. Triggers such as spicy and hot food and/or alcohol should also be avoided. Moisturizers, serums, and oils used on the face in these patients should include anti-inflammatory ingredients such as aloe vera, argan oil, arnica, chamomile, colloidal oatmeal, cucumber extract, feverfew, grape seed extract, licochalcone, licorice extract, niacinamide, salicylic acid, sulfacetamide, sulfur, and zinc [72]. These should be combined with oral and topical prescription medications and vascular laser therapies to slow progression of this bothersome disorder.

Stinging Type

In reaction to various triggers, some people experience a stinging sensation, which is a nonallergic neural sensitivity. The stinging propensity, or patients characterized as "stingers," can be identified through various available tests. In particular, the lactic acid stinging test is well regarded and established as a method for assessing patients who report invisible and subjective cutaneous irritation. The problem is that not all patients sting in response to the same substance. For example, one patient might sting to lactic acid, while another stings when in contact with benzoic acid. For this reason, historical data are more accurate at identifying a "stinger" than any physical test. It is worth noting that the stinging response seen in these patients is not necessarily associated with erythema; many patients experience stinging without exhibiting redness or other visible skin changes [73]. However, rosacea patients often associate facial flushing with a sensation of stinging or burning, especially when exposed to lactic acid [74]. Patients that are confirmed to have the stinging subtype of sensitive skin should be advised to avoid topical products containing the following ingredients: AHAs (particularly glycolic acid), ascorbic acid, benzoic acid, bronopol, cinnamic acid compounds, Dowicil 200, formaldehyde, lactic acid, propylene glycol, quaternary ammonium compounds, sodium lauryl sulfate, sorbic acid, or urea. When patients desire a form of vitamin C, they can tolerate ascorbyl

phosphate much better than ascorbic acid because ascorbic acid is formulated at a low pH, which leads to stinging in susceptible patients. It is worth noting that any patient who is beginning a retinoid may exhibit stinging to almost all ingredients including water during the first few weeks of retinoid therapy initiation, but this is transient and will resolve once the skin has acclimated to the retinoid.

Allergic Type

An epidemiologic survey in the UK published in 2004 reported that 23 % of women and 13.8 % of men displayed adverse reactions to a personal care product (e.g., deodorants and perfumes, skin care products, hair care products, and nail cosmetics) over the course of 1 year [75]. More recently, in a 1999–2006 Brazilian study of 176 patients (154 women and 22 men) who were seen in a private office and complained of dermatoses resulting from cosmetics, 45 % had dermatoses linked to cosmetics, and 14 % had skin lesions that were found to be caused by inappropriate use of cosmetics [76]. In addition, several studies have demonstrated that about 10 % of dermatologic patients who are patch tested for 20–100 ingredients exhibit allergic sensitivity to at least one ingredient common in cosmetic products [75]. Fragrances and preservatives are the most common allergens, and women aged 20–60 years old represent the demographic group that experiences the majority of these reactions [77]. Individuals that are overexposed to skin care products and patients with an impaired SC, as manifested by dry skin, reportedly have increased susceptibility to allergic reactions [78]. These findings underscore both the significance of the allergic subtype as well as the need for matching skin type and skin care products, which the BSTI facilitates.

Treatment Approaches for Sensitive Skin

The treatment of sensitive skin depends on the underlying subtype. It is crucial that the type of sensitive skin be identified and that the patient be referred to a physician who can offer a prescription medication combined with the proper skin care regimen to treat the particular type of sensitive skin. Those patients that suffer from acne will require antimicrobial ingredients and retinoids, while those that suffer from redness will need to avoid causative agents and add prescription rosacea medications, anti-inflammatory foods, supplements, and skin care products to their daily regimen. Patients undergoing procedures such as surgery or laser treatments need to discuss pre- and postoperative skin care measures with their physicians to avoid complications from their sensitive skin. In some cases, silver-containing pillowcases and other textiles in combination with skin care products can be used to reduce symptoms.

Skin Pigmentation: The Pigmented (P) to Nonpigmented (N) Continuum

This skin type parameter refers not to skin color but to the tendency to develop dyschromia (hyperpigmentation), mainly on the face or chest. Within the BSTS, pigmentary conditions that can be ameliorated using topical formulations or minor dermatologic procedures include ephelides, melasma, post-inflammatory hyperpigmentation, and solar lentigos. Congenital nevi, seborrheic keratoses, and other skin lesions that require excision or treatment beyond topical skin care are outside the scope of the BSTS system. The mechanisms of pigmentation should be clearly understood in order to prepare physicians to prevent and treat these anxiety-producing pigmentary conditions.

The enzymatic breakdown of tyrosine by tyrosinase into dihydroxyphenylalanine (DOPA) and then dopaquinone ultimately results in the production of the skin pigment melanin, specifically the two melanin types eumelanin and pheomelanin [79]. Melanin is produced by melanocytes utilizing the enzyme tyrosinase and then transferred via melanosomes to keratinocytes. Melanogenesis can be induced by UV exposure and infrared heat. Melanin production represents

the cutaneous defense against the insult of UV and infrared irradiation because when melanocytes accelerate melanin synthesis and transfer it to keratinocytes [80], the melanin surrounds and protects cellular DNA from damage. This is visually noted as skin darkening or tanning [81].

The melanocytes, each of which are typically attached to about 30 keratinocytes, load melanin into melanosomes and then transfer the melanosomes into keratinocytes through the PAR-2 receptor [82]. PAR-2 is believed to regulate melanosome transfer and thus pigmentation, through interactions between keratinocytes and melanocytes [83].

Cutaneous pigmentation can be hindered via three primary pathways: inhibition of tyrosinase, blocking of the PAR-2 receptor, or exfoliation of the melanin-containing keratinocytes. Hydroquinone, vitamin C, kojic acid, arbutin, mulberry extract, and licorice extract are examples of ingredients that inhibit tyrosinase. Soybean trypsin inhibitor (STI) and Bowman-Birk inhibitor (BBI), which are proteins contained in natural soy, have been found to suppress skin pigmentation development. STI and BBI have also been shown, in vitro and in vivo, to prevent UV-induced pigmentation [84]. Melanosome transfer into keratinocytes is influenced by STI and BBI by dint of their inhibition of the cleavage of PAR-2. The introduction of niacinamide, a vitamin B_3 derivative, has also been demonstrated to impede the transfer of melanosomes to keratinocytes [85]. Soy and niacinamide, which are considered the most effective PAR-2 blockers, are the most commonly used topical agents for inhibiting melanin transfer to keratinocytes. Not all soy is able to block PAR-2, but a complete discussion of soy is beyond the scope of this chapter [86].

In addition to the tyrosinase inhibitors and PAR-2 blockers, exfoliating agents such as AHAs, BHA, and retinoids can sufficiently accelerate cell turnover to outpace melanin synthesis. A broad-spectrum sunscreen and sun-protective clothing should also be included in any skin care regimen intended to diminish or eliminate the development of undesired dyschromias. The most effective way to prevent pigmentary and other harmful changes to the skin is to practice sun and heat avoidance, within reason. A "P" skin type designation in the BSTS correlates with the presence of dyschromia and the need for skin-lightening ingredients, while the "N" designation implies that the skin is even toned.

Skin Aging: The Wrinkled (W) to Tight (T) Continuum

Exogenous and endogenous factors play considerable roles in the complex, multifactorial process of cutaneous aging. Extrinsic aging, which results from chronic exposure to various environmental insults, particularly UV radiation, is affected by lifestyle choices such as tanning or smoking. Natural intrinsic aging is genetically driven, or cellularly programmed, and is thus inevitable until the genes responsible are identified and understood. Both pathways ultimately manifest in visible skin alterations, particularly wrinkles, lost elasticity, and skin fragility and thinning.

The BSTI questionnaire identifies "W" or wrinkle-prone individuals based on their habits and chronological age. Individuals with the following habits are more likely to develop wrinkles because of the cellular damage that these habits cause: excessive sugar intake, smoking, exposure to pollution, poor nutrition, tanning bed use, increased stress, lack of sleep, and – the biggest culprit – solar exposure. The diverse mechanisms through which UV radiation results in damage to the skin include the development of sunburn cells, as well as pyrimidine and thymine dimers, collagenase synthesis, loss of elastin, and the promotion of an inflammatory response. Aging and photodamage, in particular, have been associated with signaling through the p53 pathway after UV (especially UVB)-induced telomere disruption [87, 88]. Although much remains to be learned regarding the mechanisms by which UV irradiation initiates and promotes deleterious effects, UV (particularly UVA) irradiation is well known as the cause of photoaging, photocarcinogenesis, and photo-immunosuppression [89]. Insofar as UV irradiation impairs DNA and accelerates telomere shortening, this chief cause of extrinsic

aging can be thought of as exerting an impact on the natural course of intrinsic aging.

The primary evidence of cutaneous aging is the development of rhytides (wrinkles), the formation of which is initiated in the lower dermal layers. It is important to note that few skin care formulations can actually penetrate far enough into the dermis or the deeper epidermis to reverse deep wrinkles, despite the multitude of products that tout such a capacity. Preventing loss of collagen, HA, and skin elasticity and at the same time coercing fibroblasts into increasing collagen and HA production is the primary focus of antiaging skin care [90]. Accordingly, topical products are formulated to prevent the degradation or promote the synthesis of the three primary skin constituents – collagen, elastin, and HA. Specifically, collagen production has been demonstrated to be promoted by topical preparations of retinoids, vitamin C, and copper peptides, as well as oral vitamin C [91–93]. The synthesis of HA has been shown in animal models to be stimulated by retinoids [94, 95], and HA levels are also thought to be enhanced through glucosamine supplementation [96]. Currently, no products have been shown to be effective, or approved, for spurring elastin synthesis.

Inflammation reduction is also a significant target for wrinkle prevention, because inflammation is known to influence the degradation of collagen, elastin, and HA. Antioxidants, which protect the skin through several mechanisms, are used in this approach to mitigate ROS activity. This is important because ROS act directly on growth factor and cytokine receptors in keratinocytes, and cutaneous inflammation can be initiated in these epidermal cells [97]. ROS can also contribute to glycation, a process by which a sugar is bound to a protein causing damage to the protein.

UV irradiation is the biggest culprit in aging, and it causes its harmful effects by initiating a cascade of events that result in downstream signal transduction by activating mitogen-activated protein (MAP) kinase pathways (extracellular signal-regulated kinase, c-jun N-terminal protein kinase, and p38). These then amass in cell nuclei, forming c-Fos/c-Jun complexes of transcription factor activator protein 1, and provoking the matrix metalloproteinases collagenase, 92 kDa gelatinase, and stromelysin to degrade collagen and other cutaneous connective tissue [98, 99].

The direct effects of ROS on cutaneous aging and the overall aging process are less clearly understood. In 2003, Kang et al. demonstrated that ROS activation of MAP kinase pathways induces collagenase production, thus contributing to collagen degradation [99]. The use of antioxidants is believed to block these pathways, thus inhibiting the process of photoaging by preventing collagenase synthesis and its ensuing deleterious impact on collagen. Specifically, Kang et al. found that the pretreatment of human skin with the antioxidants genistein and N-acetyl cysteine hindered UV induction of the cJun-driven enzyme collagenase.

The vast array of topical skin care products include antioxidants such as vitamins C and E, coenzyme Q_{10}, argan oil, caffeine, coffeeberry, ferulic acid, feverfew, ginger, grape seed extract, green tea, idebenone, mushrooms, phloretin, polypodium leucotomos, pomegranate, pycnogenol, resveratrol, rosemary, and silymarin [100]. The antioxidant capacity of these compounds is well established in the literature; however, their efficacy in topical formulations designed to reverse or diminish the cutaneous signs of aging is unclear because long-term aging prevention is difficult to prove. Antioxidant use should be combined with several practical measures including avoiding/limiting solar exposure (especially from 10 am to 4 pm), using broad-spectrum sunscreen on a daily basis, avoiding cigarette smoke and pollution, eating a diet high in fruits and vegetables, taking oral antioxidant supplements and topical antioxidant formulations, regular use of topical retinoids, reduction of sugar in the diet, and averaging 7 h of sleep per night in addition to stress reduction activities. In the near future, technological innovations in tissue engineering and gene therapy may lead to breakthroughs in the therapeutic uses of growth factors, cytokines, and telomerase [101], including dermatologic applications. At this time, stem

cells and peptides are not associated with sufficient scientific data to support their use in topical skin care geared to treat wrinkle-prone skin.

Skin Care Regimens Based on Baumann Skin Type

Assessing the four skin type dichotomies together, as discussed above, provides insight into the simultaneous state or tendencies of an individual's skin along four different spectra, yielding 16 different possible skin type phenotypes (Table 2). Each of the individual Baumann Skin Types requires a regimen designed specifically for its needs. For example, formulations containing ingredients with the capacity to repair the skin barrier and provide anti-inflammatory activity would be appropriate selections for a person with dry, sensitive, nonpigmented, tight skin (DSNT). In addition to barrier repair and anti-inflammatory ingredients, DSNW (dry, sensitive, nonpigmented, wrinkle-prone) skin would require antiaging ingredients such as ascorbic acid and retinol. However, ascorbic acid and retinol can cause stinging and retinoid dermatitis in the susceptible DSNW skin types; therefore, the order of delivery of the agents is essential. In this example, retinoids should be applied after a moisturizer that contains both occlusive and barrier repair ingredients, which will decrease retinol penetration and minimize side effects. The moisturizer should also contain anti-inflammatory ingredients. In each skin type, the regimen steps are adjusted to maximize efficacy and decrease adverse events. Particular care is taken in choosing the order in which products are applied so that the ingredient interactions are maximized. The actions of ingredients can be greatly altered by pH, exposure to oxidizing agents, interaction with other ingredients, and exposure to penetration inhibitors or enhancers. All of these effects must be taken into account when choosing the steps of the skin care regimen.

Environmental conditions, diet, exercise, sleep, and stress can impact skin type by affecting the barrier, cortisol levels, and inflammatory cascades. For this reason, it is recommended that individuals take a baseline BSTI questionnaire and retake the test when stress, significant life changes, or cutaneous symptoms are present. Specifically, stress, pregnancy, menopause, exposure to variable climates or moving to a different climate, and various other significant exogenous or endogenous alterations can manifest in skin type changes. Essentially, if the skin care regimen stops working, it is time to retake the BSTI questionnaire. With baseline and updated BSTI scores, a physician is better equipped to arrive at a more holistic, integrated, or informed skin type assessment and treatment approach.

Summary

The use of the Baumann Skin Typing System (BSTS), based on the results of the Baumann Skin Type Indicator (BSTI), a self-administered questionnaire, allows for the evaluation of skin according to four dichotomous spectra – dry or oily, sensitive or resistant, pigmented or nonpigmented, and wrinkled or tight (unwrinkled). The BSTI is only available through physicians and is used to develop preset regimens for patients. By developing the regimens ahead of time and using a consistent methodology to diagnose skin type and prescribe skin care, outcomes are improved through staff and patient education. The discussion that arises from the use of the BSTI can also improve the physician–patient relationship, with such communication serving also to educate the patient about the importance of proper skin care. The BSTS is also used in research trials to select what types of patients are most likely to benefit from a cosmeceutical ingredient. The Fitzpatrick skin typing system is another skin type classification system that categorizes skin according to the skin's response to ultraviolet light. The Glogau photoaging scale classifies skin according to the level of skin aging. Patients self-classify their skin as dry, oily, combination, or sensitive, but there is much disparity about the meaning of each of these. Studies show that patients self-classify incorrectly, especially along

the realms of oily versus dry [102]. Researchers and cosmetic scientists should take the time to properly categorize the skin type of the subjects used in cosmeceutical research studies so that the efficacy of ingredients can be properly evaluated. Scientific advances are happening rapidly, and more advanced ingredients are expected to enter the market in the next 5 years. These may include ingredients designed to address genetic deficiencies, the immune system, or organelles such as lysosomes or mitochondria.

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