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4 The Baumann Skin-Type Indicator: A Novel Approach to Understanding Skin Type

Leslie Baumann
University of Miami, Cosmetic Medicine and Research Institute, Miami, Florida, U.S.A.

INTRODUCTION

Over the latter part of the last century, the dry, oily, combination, or sensitive skin-type classifications, which were identified in the early 1900s by cosmetics magnate Helena Rubinstein, have held sway in terms of characterizing the skin. While there have been significant innovations and even more substantial growth in the skin care product market during this time span, few notable advances have been made to further our understanding or ability to characterize skin types. Consequently, practitioners have had insufficient information to use in divining the most appropriate skin care product selections for their patients. The Baumann skin-type indicator (BSTI) is a novel approach to categorizing skin types, which greatly expands on the skin-type designations of Rubinstein and, in the process, provides assistance to practitioners and patients/consumers alike in making sense of the numerous available skin care formulations, many of which are now touted for particular skin types, as well as in selecting the most suitable products. The BSTI is based on the identification of skin type using four dichotomous parameters characterizing the skin: dry or oily, sensitive or resistant, pigmented or nonpigmented, and wrinkled or unwrinkled (tight). A four-letter skin-type designation is derived from the answers to a 64-item questionnaire and considers all the four skin parameters at once. Sixteen possible skin types, each delineated using the four-letter code denoting one end of each parameter, characterize the BSTI (Fig. 1). Ideally, patients will self-administer the BSTI to ascertain baseline skin type and reuse the questionnaire after significant life changes (e.g., moving to a different climate, pregnancy, menopause, andropause, chronic stress), which can induce modifications to skin type (1). This chapter focuses on the basic science underlying the four fundamental skin-type parameters and, in the process, characterizes in varying levels of depth the 16 skin types. In addition, some attention is paid to treatments, mainly topical and noninvasive, on the basis of the BSTI system.

SKIN HYDRATION

Oily (O) Vs. Dry (D)

"Dry skin," also known as xerosis, results from a complex, multifactorial etiology and is characterized by dull color (usually gray-white), rough texture, and an elevated number of ridges (2). The primary factors that regulate the level of skin hydration and that contribute to dry skin are the levels of stratum corneum (SC) lipids, natural moisturizing factor (NMF), sebum, hyaluronic acid (HA), and aquaporin. The role of the SC and its capacity to maintain skin hydration is the most important of these factors in terms of dry skin. The SC is composed primarily of ceramides, fatty acids, and cholesterol. These constituents help protect the skin and keep it watertight when they are present in the SC in the proper balance. SC equilibrium is also thought to be maintained via stimulation of keratinocyte lipid production and keratinocyte proliferation by primary cytokines (3).

When the primary components of the SC are not in proper balance, the skin's capacity to maintain water is decreased, and the skin becomes more susceptible to environmental factors. With the skin barrier thus impaired, transepidermal water loss (TEWL) increases and the skin is left dry and sensitive. This occurs because the enzymes essential for desmosome metabolism are inhibited by inadequate hydration, leading to the abnormal desquamation of corneocytes (4). At the same time, superficial SC desmoglein I levels remain high. The resultant compromised

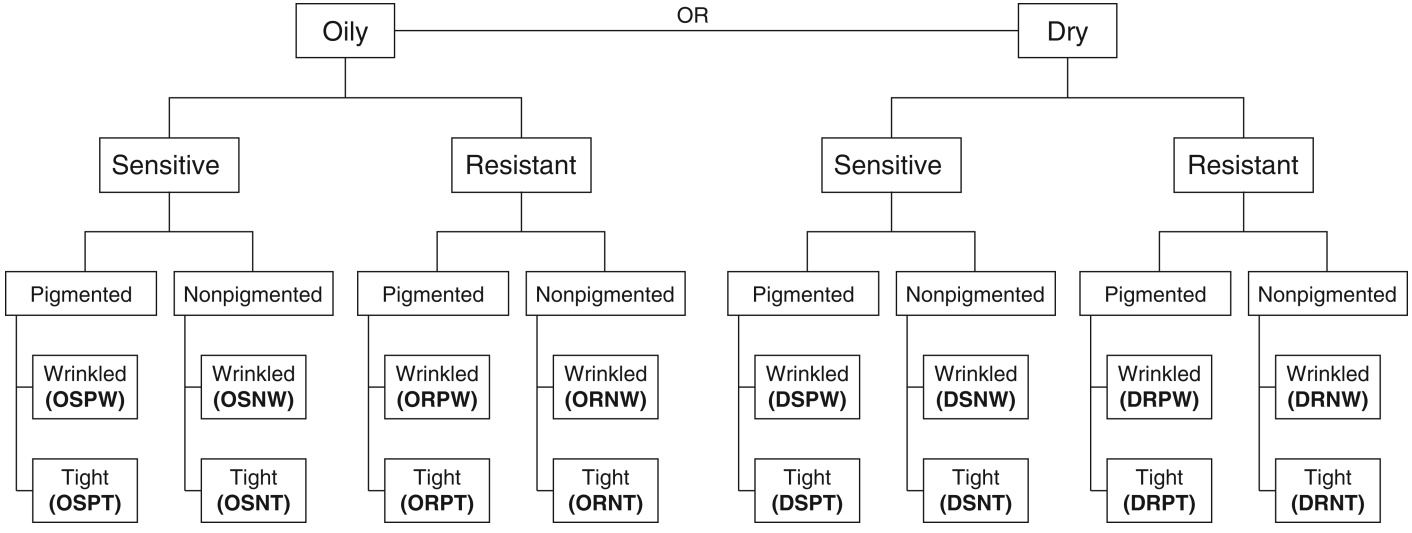

Figure 1 The BSTI skin types. The BSTI questionnaire can be located by registering online at http://www.SkinIQ.com. The Web site is frequently updated with the latest data as new questions are developed. The nonidentifying data collected on this Web site will be used to expand knowledge of skin-type prevalence around the world.

desquamation leads to a visible accrual of keratinocytes, leaving a rough and dry appearance to the skin (5). Dry skin has also been associated with a perturbation in the lipid bilayer of the SC as a result of elevated fatty acid levels and reduced ceramide levels (6). Exogenous factors, such as UV irradiation, acetone, chlorine, detergents, and protracted exposure to or immersion in water, can also affect and inhibit the lipid bilayer. In addition, recent studies have suggested that local pH fluctuations may account for the initial cohesion and ultimate desquamation of corneocytes from the SC surface. These alterations are thought to selectively activate numerous extracellular proteases in a pH-dependent manner (7).

NMF, derived from the breakdown of the protein filaggrin, is an intracellular, hygroscopic compound present only in the SC that is released by lamellar bodies and plays an integral role in maintaining water within skin cells. Filaggrin, which is composed of lactic acid, urea, citrate, and sugars, imparts structural support and strength to the lower layers of the SC. A cytosolic protease breaks it down into free amino acids, such as arginine, glutamine (glutamic acid), and histidine, in the stratum compactum, an outer SC layer (8). These water-soluble substances remain inside the keratinocytes and avidly cling to water molecules. Aspartate protease (cathepsin) initiates this chain of events and is believed to regulate the pace of filaggrin decomposition into NMF as well as the level of NMF (9). It is important to note that external humidity levels can affect cathepsin, resulting in changes in NMF production. After an individual enters a low-humidity environment, the pace of NMF production typically increases over the course of several days of getting acclimated (10). Notably, xerosis and icthyosis vulgaris are associated with low NMF levels. In addition, UV irradiation and surfactants can inhibit NMF production. However, NMF production cannot yet be artificially regulated through the use of any products or procedures.

HA can bind 1000 times its weight in water, and its presence in the dermis assists the skin in retaining water. HA is also found in the epidermal intercellular spaces, particularly the middle spinous layer, but is not present in the SC or stratum granulosum (11). Produced primarily by fibroblasts and keratinocytes, HA has an estimated turnover rate of 2 to 4.5 days in mammalians (12). Although the role of HA in skin hydration has not been fully elucidated, aged skin, which is less plump than youthful skin, is characterized by decreased levels of HA. Significantly, topically applied HA does not penetrate the skin (13). Nevertheless, several manufacturers include HA in topical skin care products and claim that they are effective.

Aquaporin-3 (AQP3) is a member of a family of homologous integral membrane proteins and a subclass of aquaporins called aquaglyceroporins that facilitate water transport and small neutral solutes, including glycerol and urea, across biological membranes (14). Present in the urinary, respiratory, and digestive tracts as well as the kidney collecting ducts and, notably, epidermis, AQP3 was shown recently to be expressed copiously in the plasma membrane of epidermal keratinocytes in human skin (15). The water conduction function in the skin is thought to occur along an osmotic gradient below the SC, where high AQP3-mediated water permeability is manifested. In this context, AQP3 water clamps viable epidermal layers to promote the hydration of cutaneous layers beneath the SC. A high concentration of solutes (Na^+, K^+, and Cl^-) and a low concentration of water (13–35%) have been shown to exist in the superficial SC that produce in the steady-state gradients of solutes and water from the skin surface to the viable epidermal keratinocytes (16–19). Nevertheless, the relationship between keratinocyte fluid transport and SC hydration as well as the molecular mechanisms of fluid transport across epidermal keratinocyte layers remains poorly understood. It is thought though that AQP3 enhances transepidermal water permeability to protect the SC from water evaporating from the skin surface and/or to spread water gradients throughout the layer of epidermal keratinocytes (15). In a study evaluating the functional expression of AQP3 in human skin, researchers observed that the water permeability of human epidermal keratinocytes was inhibited by mercurials and low pH, which was consistent with AQP3 involvement (15). Some of the same investigators considered skin phenotype in transgenic mice lacking AQP3 and discovered substantially decreased water and glycerol permeability in AQP3 null mice, supporting earlier evidence that AQP3 functions as a plasma membrane water/glycerol transporter in the epidermis (20). In most areas of the skin, conductance measurements revealed significantly diminished SC water content in the AQP3 null mice. Epidermal cell water permeability is not an important determinant of SC hydration, however, because water movement across AQP3 is slower in skin than in other tissues (21). Currently, only extracts of

the herb Ajuga turkestanica have been demonstrated to exert an influence in regulating AQP3 (22). Ajuga turkestanica is included as an ingredient in a high-end line of skin care products. Eventually, pharmacological manipulation of AQP3 may lead to its use in treating skin conditions caused by excess or reduced hydration.

Sebum, the oily secretion of the sebaceous glands containing wax esters, sterol esters, cholesterol, di- and triglycerides, and squalene, imparts an oily quality to the skin and is well known to play an important role in acne development (23). A significant source of vitamin E, sebum is also believed to confer cutaneous protection from exogenous elements and, perhaps, when production is decreased, contribute to dry skin (24). The xerosis aspect of this theory has not received much support though, as low sebaceous activity has not been found to foster dry skin. In fact, a more complex role for sebum production in the causal pathway of xerosis has been expounded. It has been previously assumed that sebum does not alter epidermal permeability barrier function because skin with few sebaceous glands, such as that in prepubertal children, manifests normal basal barrier function (25). Indeed, prepubertal children (aged 2–9 years) often present with eczematous patches (pityriasis alba) on the face and trunk, which are not associated with sebaceous gland activity. In addition, the pharmacological involution of sebaceous glands with supraphysiological doses of isotretinoin has no impact on barrier function or SC lamellar membranes (26–28).

Although sebum levels do not alter barrier function, sebum may still play a role in the etiology of xerosis in people with dry, resistant skin (DR in the BSTI system). Lipids from meibomian glands, which are modified sebaceous glands found in the eyes, act against dryness by preventing tear evaporation (29,30). TEWL is prevented in a similar fashion, as sebum-derived fats form a lipid film over the skin surface. This theory received support from a recent study that assessed permeability barrier homeostasis and SC hydration in asebia J1 mice that demonstrated sebaceous gland hypoplasia (31). Investigators observed normal barrier function in these sebum-deficient mice, which they ascribed to unaltered levels of the three primary barrier lipids—ceramides, free sterols, and free fatty acids—and the persistence of normal SC extracellular membranes. The mice did exhibit reduced SC hydration, however, suggesting that an intact intercellular membrane bilayer system, although sufficient for permeability barrier homeostasis, does not necessarily imply normal SC hydration. It is worth noting that normal SC hydration levels were restored with the topical application of glycerol. Sebaceous gland-derived triglycerides are hydrolyzed to glycerol before they are transported to the skin surface in normal skin. In individuals with low sebum production, replacing this glycerol may be an effective way to ease their xerosis. Using glycerol has also been demonstrated to be successful in accelerating SC recovery (32).

Patients rarely, if ever, complain about reduced sebum production, but elevated sebum production, yielding oily skin that can be a precursor to acne, is a common complaint. Several factors are known to influence sebum production. Age, in particular, has a significant and well-known impact, as sebum levels are usually low in childhood, rise in the middle-to-late teen years, and remain stable into the seventh and eighth decades until endogenous androgen synthesis dwindles (33). Sebum production is also affected by one's genetic background, diet, stress, and hormone levels. In a study of 20 pairs each of identical and nonidentical like-sex twins, nearly equivalent sebum excretion rates with significantly differing acne severity were observed in the identical twins, but a significant divergence was seen in both parameters among the nonidentical twins, suggesting that acne development is influenced by genetic and exogenous factors (34). Using oral retinoids to reduce sebaceous glands is a well-established approach, but this capacity has not been demonstrated in topical retinoids. No topical products have been shown to lower sebum production.

Skin Care for the O–D Parameter

An intact SC and barrier, normal NMF and HA levels, normal AQP3 expression, and balanced sebum secretion are qualities of the skin that fall in the middle of the oily–dry spectrum. Increased sebum secretion, regardless of whether it contributes to acne development, is typically the reason that the skin may be described as falling on the oily side of this continuum. Oily skin that is also prone to acne would be characterized as oily, sensitive (OS within the BSTI framework), as acne-infiltrated skin is distinguished by heightened sensitivity (see section "Acne Type"). Treatment for individuals with OS skin should concentrate on lowering sebum levels using retinoids, reducing or eliminating cutaneous bacteria with antibiotics,

benzoyl peroxide, or other antimicrobials, and complementing with anti-inflammatory agents. Individuals with oily skin but no acne (the OR type within the BSTI) should be treated only to decrease sebum production, unless other skin-type parameters dictate otherwise (e.g., hyperpigmentation or wrinkling). Sebum secretion has been shown to be effectively reduced using oral ketoconazole as well as oral retinoids, but no topical products have yet shown such success (35,36). Further, unwanted sebum in OR skin can be camouflaged using sebum-absorbing polymers and talc.

Treatment of dry skin starts with the identification of factors contributing to dryness. The other BSTI skin parameters can provide clues. The skin barrier is likely impaired in a patient whose skin is dry and sensitive (DS in the BSTI system). To treat such skin, products that repair the skin barrier (i.e., formulations that include fatty acids, cholesterol, ceramides, or glycerol) should be used. In a patient with dry photodamaged skin (with a high score on the W vs. T parameter), lower HA levels likely account, at least in part, for the dryness. Skin care products that include HA are useless in this context as topically applied HA is not absorbed into the skin. Recent studies have suggested that HA levels may be boosted through the use of glucosamine supplements (37). The role of glucosamine has not been established though, as one small single-blind study demonstrated wrinkle enhancement but no improvement in skin hydration (38). Dry skin that is habitually exposed to the sun likely exhibits an impaired skin barrier and diminished NMF. Treatment for such skin should concentrate on repairing the barrier and reducing or avoiding sun exposure. If sun exposure cannot be avoided, adequate sun protection is necessary, of course.

Harsh foaming detergents, which remove hydrating lipids and NMF from the skin, should be avoided by all patients with dry skin. Such detergents are found in body and facial cleansers as well as in laundry and dish cleansers. All patients with dry skin should also abstain from bathing for prolonged periods, especially in hot or chlorinated water. Humidifiers are recommended for people with very dry skin who live in low-humidity environments, as application of moisturizers is recommended two to three times daily and after bathing. Several over-the-counter (OTC) moisturizers (e.g., occlusives, humectants, and emollients) are effective in hydrating the skin and serve as worthy adjuncts to the aforementioned pharmacological and behavioral approaches to treating dry skin. Indeed, moisturizers are the third most often recommended type of OTC topical skin product (39). Moisturizers are typically formulated as water-in-oil emulsions (e.g., hand creams) and oil-in-water emulsions (e.g., creams and lotions).

SKIN SENSITIVITY

Sensitive (S) Vs. Resistant (R)

A potent SC that provides especially reliable protection to the skin, rendering harmless allergens and numerous irritating exogenous substances, characterizes resistant skin. Individuals with such skin are unlikely to experience erythema (unless overexposed to the sun) or acne (though stress or hormonal fluctuations could lead to a breakout). Such skin also confers an interesting set of advantages and disadvantages. On the positive side, resistant skin allows for the use of most skin care formulations with an extremely low probability of incurring adverse reactions (e.g., acne, rashes, or a stinging sensation). However, resistant skin also renders many skin care products ineffective, with individuals with such skin experiencing difficulty in detecting differences among cosmetic formulations and exhibiting an exceedingly high threshold for product penetration and efficacy.

Sensitive skin is more complex than resistant skin in terms of characterization, presentation, diagnosis, and treatment. Nevertheless, the diagnosis of sensitive skin is increasingly common (40). The majority of people that complain to a dermatologist about sensitive skin are healthy women of childbearing age. On an individual basis, sensitive skin incidence diminishes with age, fortunately. The prevalence of sensitive skin continues to increase, though. While numerous skin care products are increasingly touted as suitable for sensitive skin, such skin remains challenging to treat. Variations in the qualities of sensitive skin and poor self-diagnosis account for this difficulty. Indeed, four discrete subtypes of sensitive skin have been identified: acne type, rosacea type, stinging type, and allergic type. Consequently, the products marketed for sensitive skin are not necessarily suitable for all sensitive skin subtypes, which is

a phenomenon that presents some unusual treatment challenges. All four sensitive skin subtypes do share a significant feature, though: inflammation. The treatment approach to any kind of sensitive skin understandably begins with a focus on alleviating and eliminating inflammation. Treatment for patients with more than one sensitive skin subtype, which is not uncommon, is, of course, more complicated.

Acne Type

This is the most common subtype of sensitive skin because of the prevalence of acne, which is by far the most common skin disease. Individuals with such sensitivity are prone to developing acne, black heads, or white heads. Acne typically affects adolescent and young adults, equally by sex, between 11 to 25 years old. Most of the remainder of the millions of those suffering from acne are adult women, who display a hormonal aspect to their acne. The complex interplay of four primary factors is at the heart of acne pathogenesis: an increase in sebum production, clogging of pores (which results from dead keratinocytes inside the hair follicles clinging more strongly than in people without acne and can also result from elevated sebum production), presence of the bacteria *Propionibacterium acnes*, and inflammation. Significantly, acne can occur as a result of various causal pathways or in idiopathic presentations, but the sine qua non of the condition is the amassing and adherence of dead keratinocytes in the hair follicles due to elevated sebum production, leading to clogged follicles and appearance of a papule or pustule. This is followed by the migration of *P. acnes* into the hair follicle, where the combination of the bacteria, sebum, and dead keratinocytes stimulates the release of cytokines and other inflammatory factors. In turn, an inflammatory response is provoked that manifests in the formation of redness and pus. Indeed, in chronic inflammatory conditions such as acne, high levels of primary cytokines, chemokines, and other inflammatory markers are typically present (3). To treat acne, the therapeutic intention is to target the four main etiological factors. This translates to decreasing sebum production (using retinoids, oral contraceptives, and/or stress reduction), unclogging pores (using retinoids, α-hydroxy acids, or β-hydroxy acid), eliminating bacteria (using benzoyl peroxide, sulfur, antibiotics, or azelaic acid), and reducing inflammation (using any of a wide array of anti-inflammatory products).

Rosacea Type

The acneiform condition rosacea affects 14 million people in the United States, typically adults aged between 25 and 60 years, according to the National Rosacea Society (41). Those with the rosacea subtype of sensitive skin exhibit a tendency toward recurrent flushing, facial redness, and experiencing hot sensations. The etiology of rosacea remains elusive, but this condition shares the aforementioned symptoms with acne, along with papules, but is distinguished by the formation of salient telangiectases. Avoiding the triggers that exacerbate symptoms is, of course, recommended for rosacea treatment, as is using anti-inflammatory ingredients to reduce the dilation of the blood vessels. Eosinophils, which are versatile leukocytes, contribute to the initiation and promotion of various inflammatory responses (42,43). The aim of rosacea therapy is to inhibit eosinophilic activity, decrease vascular reactivity, neutralize free radicals, and hinder immune function, the arachidonic acid pathway, and degranulation of mast cells (which frequently migrate to areas of eosinophil-mediated disease). Several anti-inflammatory medications are available for the treatment of rosacea, including antibiotics, immune modulators, and steroids. The most effective anti-inflammatory ingredients (many of which are botanically derived) in the copious supply of topical rosacea therapeutic agents include aloe vera, arnica, chamomile, colloidal oatmeal, cucumber extract, feverfew, licochalcone, niacinamide, quadrinone, salicylic acid, sulfacetamide, sulfur, witch hazel, and zinc (44).

Stinging Type

People with this particular subset of sensitive skin exhibit a predilection to experiencing stinging or burning sensations in response to various factors and triggers. This tendency is best characterized as a nonallergic neural sensitivity. "Stingers" or the stinging tendency can be identified through the use of numerous tests. The lactic acid stinging test is the best-regarded, standard way to assess patients who complain of invisible and subjective cutaneous irritation (45). This test has, in fact, been used to show that individuals with "sensitive skin" experienced a much stronger stinging sensation than those in a healthy

control group (46). It is worth noting that erythema does not necessarily accompany the stinging sensation, as many patients report stinging without experiencing redness or irritation (47). Nevertheless, exposure to lactic acid is more likely to elicit stinging in patients with rosacea distinguished by facial flushing (48). Topical products that contain α-hydroxy acids (particularly glycolic acid), benzoic acid, bronopol, cinnamic acid compounds, Dowicel 200, formaldehyde, lactic acid, propylene glycol, quaternary ammonium compounds, sodium lauryl sulfate, sorbic acid, urea, or vitamin C should be avoided by patients that are confirmed to have the stinging subtype of sensitive skin.

Allergic Type

Over the course of a year, the use of personal care products, including deodorants, perfumes, nail cosmetics, as well as skin and hair care products, elicit adverse reactions in 23% of women and 13.8% of men, according to a recent epidemiological survey in the United Kingdom (49). Individuals with the allergic subtype of sensitive skin are more prone to exhibit erythema, pruritus, and skin flaking. Patients tested for allergies to cosmetic ingredients are typically patch tested for 20 to 100 ingredients, with erythema or edema in the tested area indicating an allergy to the particular ingredient. Several studies have demonstrated that approximately 10% of dermatological patients who were patch tested were found to have an allergy to at least one ingredient common in cosmetic products (50). Fragrances and preservatives are the most common allergens, and most reactions, approximately 80%, arise in women aged 20 to 60 years (50). Overexposure to common allergens, by using several skin care products, raises the risk of inducing allergic reactions. In particular, individuals with the D skin type (within the BSTI system) who have an impaired SC manifested by xerosis are more likely to exhibit an increased incidence of allergic reactions to topically applied allergens (51).

On the basis of the guidelines of the BSTI, oil control is necessary for those with OS skin. An acne or rosacea regimen would also likely be necessary for the OS type. Treatment to repair the SC is indicated for people with DS skin. Therapy to ameliorate wrinkles and to prevent the development of new ones is recommended for individuals with sensitive, wrinkled (SW) skin. Frequently, people with sensitive, pigmented (SP) skin request procedures or topical applications to reduce or remove hyperpigmentation and therapy to lessen the likelihood of developing new dyschromias.

SKIN PIGMENTATION: PIGMENTED (P) VS. NONPIGMENTED (N)

This skin-type parameter refers to the proclivity to develop unwanted hyperpigmentations on the face or chest. Within the BSTI framework, the focus is on the pigmentary changes or conditions that can be ameliorated with topical skin care products or minor dermatological procedures. In this context, melasma, solar lentigos, ephelides, and postinflammatory hyperpigmentation are representative conditions for the pigmented skin type. Considerable anxiety is often associated with the presentation of these skin lesions, and patients often pay substantial sums in the attempt to treat these conditions. To best treat these pigmentary problems, it is incumbent upon the physician to understand the source of pigmentation. In addition, the practitioner can be well served in terms of making suitable product selections for patients to place such knowledge within the context of other aspects of an individual patient's full (BSTI) skin type.

The enzymatic breakdown of tyrosine into dihydrophenylalanine (DOPA) and then dopaquinone leads to the synthesis of two types of skin pigment (melanin), eumelanin and pheomelanin (52). These skin pigments (of which eumelanin is the more abundant and which regularly correlates with the visual phenotype) are produced by melanocytes, which use melanosomes to transport the pigments to keratinocytes (53). One melanocyte is typically attached to approximately 30 keratinocytes. Melanosomes are surrounded by keratinocytes, which absorb the melanin after activation of the protease-activated receptor (PAR)-2 (54). Expressed in keratinocytes but not melanocytes, PAR-2 is a seven transmembrane G-protein-coupled trypsin/tryptase receptor activated by a serine protease cleavage. PAR-2 is believed to regulate pigmentation via exchanges between keratinocytes and melanocytes (55). Notably, melanogenesis can also be initiated by UV irradiation. Under these conditions, melanogenesis is a defensive manifestation to protect the skin and is characterized by accelerated melanin

synthesis and transfer to keratinocytes, leading to darkening of the skin in the exposed areas (56). Melanocytes synthesize more melanin in darker-skinned people, and their larger melanosomes accommodate this comparatively greater abundance of melanin and consequently break down more slowly than in lighter-skinned people (55).

Inhibiting tyrosinase, thus preventing melanin formation, and blocking the transfer of melanin into keratinocytes represent the two main pathways through which the development of skin pigmentation can be hindered. Hydroquinone, vitamin C, kojic acid, arbutin, mulberry extract, and licorice extract are the most effective tyrosinase inhibitors. Skin pigmentation is also thought to be inhibited by two small proteins contained in soy—soybean trypsin inhibitor (STI) and Bowman–Birk inhibitor (BBI). Both STI and BBI have been shown in vitro and in vivo to exhibit depigmenting activity and to prevent UV-induced pigmentation by inhibiting the cleavage of PAR-2 (57). Consequently, STI and BBI are thought to influence melanosome transfer into keratinocytes, thereby exerting an effect on pigmentation. Niacinamide, a vitamin B_3 derivative, has also been demonstrated to hinder the melanosome transfer from melanocytes to keratinocytes (58). Soy and niacinamide, the most effective PAR-2 blockers, are the main agents for preventing this transfer.

There are three classes of topical agents used within the two pathways of inhibiting melanin formation. In addition to the inhibitors of tyrosinase and PAR-2, exfoliating products (e.g., α-hydroxy acids, β-hydroxy acid, retinoids) have the capacity to increase cell turnover to outpace the rate of melanin production. Such exfoliation can also be achieved through microdermabrasion and the use of facial scrubs. Broad-spectrum sunscreens should also be employed in any skin care program intended to reduce or eliminate undesired pigmentation. The most effective way of preventing pigmentary alterations remains the avoidance of chronic sun exposure. Within the BSTI framework, a person with a penchant for developing unwanted dyspigmentations has "P" type skin, or, otherwise, "N" type skin.

SKIN AGING: WRINKLED (W) VS. TIGHT (T)

Cutaneous aging is a complex multifactorial phenomenon described in terms of endogenous and exogenous influences that ultimately manifest in alterations to the outward appearance of the skin. Endogenous aging—known as natural, chronological, or intrinsic aging in this case—is a function of heredity or cellular programming. The aging-related manifestations of such forces that occur over time are, therefore, considered inevitable and beyond human volition. Exogenous aging—known typically as extrinsic aging—is driven by chronic exposure to the sun and other deleterious environmental elements (e.g., cigarette smoke, poor nutrition) and, therefore, can be avoided, though not always easily. While these etiological strains appear, and have been typically evaluated, as discrete processes, recent findings suggest that UV irradiation—the leading cause of extrinsic aging—may also alter the normal course of chronological aging. Therefore, it is possible that there is a significant overlap in the processes of intrinsic and extrinsic aging. For the purposes of this discussion, however, intrinsic and extrinsic aging will be considered separately.

Cellular or intrinsic aging is currently best understood with reference to telomeres, specialized structures that shield the ends of chromosomes. Telomere length shortens with age, and this erosion is considered an internal aging clock as well as the source for one of the currently espoused theories on chronological aging (59). The enzyme telomerase, which lengthens telomeres and imparts stability, is expressed in approximately 90% of all tumors and in the epidermis, but is absent in several somatic tissues (59,60). This suggests that most cancer cells, as opposed to normal healthy cells, are not programed for apoptosis or cell death. For this reason, cancer and aging are thought to represent opposite sides of the same coin. Current knowledge regarding telomeres and telomerase has not yet been harnessed for any viable antiaging therapies, primarily because little is known regarding the safety of artificially increasing telomere length.

As implied in the definition, extrinsic aging is a premature aging of the skin that is the result of the interplay of external factors and human behaviors resulting in the chronic exposure to such factors, and thus falls within the realm of human control. By far, exposure to UV irradiation is the leading cause of extrinsic aging; indeed, such premature aging is often referred to as photoaging. Of course, other factors such as smoking, other pollution, poor

nutrition, excessive alcohol consumption, and protracted stress among additional exogenous influences can contribute to accelerating cutaneous aging. Significantly, photodamage precedes photoaging, and this evolves through several mechanisms, including the formation of sunburn cells, thymine and pyrimidine dimers, production of collagenase, and induction of an inflammatory response. In addition, photodamage and aging have been associated with signaling through the p53 pathway subsequent to UV-induced (especially by UVB) telomere disturbance (61,62). The best-known deleterious effects of UV (UVA, 320–400 nm, in particular) include photoaging, photoimmunosuppression, and photocarcinogenesis, but much has yet to be discovered regarding the mechanisms through which UV irradiation engenders such extensive harm (63). Nevertheless, as the aforementioned theory implies, intrinsic aging can be thought to be impacted by the primary source of extrinsic aging, as chronic UV exposure can damage DNA and accelerate the diminution of telomeres, which is known to play a role in chronological aging.

Cutaneous aging is evidenced, first and foremost, by the formation of rhytides, which develop in the dermis. Because few topical skin care products can actually penetrate to this layer of the skin to affect wrinkles, the dermatological approach to antiaging skin care concentrates on preventing the formation of wrinkles (64). This translates to a focus on replenishing or maintaining the three primary structural constituents of the skin, collagen, elastin, and HA, which are known to degrade with age. Despite the inadequacy of most topical formulations to deliver active ingredients that alter these components, some products have been shown to exert such an impact on collagen and HA. Specifically, collagen synthesis has been shown to be spurred by topical retinoids, vitamin C, and copper peptide as well as oral vitamin C (65–67). The synthesis of HA and elastin has been demonstrated in animal models to be stimulated by retinoids (68,69). In addition, HA levels are thought to be enhanced through glucosamine supplementation (37). However, no products have yet been demonstrated or approved for inducing the production of elastin.

Collagen, elastin, and HA can also be broken down by inflammation; therefore, targeting ways to reduce inflammation represents another significant approach to preventing or mitigating cutaneous aging. Skin inflammation can result from reactive oxygen species (ROS) or free radicals acting directly on growth factor and cytokine receptors in keratinocytes and dermal cells. Although their effects on cutaneous aging are not fully understood, growth factors and cytokines are known to act synergistically in a complex process involving several types of growth factors and cytokines (70). Antioxidants protect the skin from ROS via various mechanisms not yet fully explained. However, the events through which ROS directly impact the aging process are known. UV exposure is thought to induce a chain of events, acting on growth factors and cytokine receptors in keratinocytes and dermal cells. This yields downstream signal transduction from the activation of mitogen-activated protein (MAP) kinase pathways, which accrue in the cell nuclei, developing into cFos/cJun complexes of transcription factor activator protein 1, in turn leading to the breakdown of cutaneous collagen as a result of the induction of matrix metalloproteinases, including collagenase, stromelysin, and 92-kDa gelatinase (71,72). The use of antioxidants is thought to delay or act against photoaging in this context by preventing these pathways from synthesizing collagenase. Kang et al. demonstrated that production of the UV-induced cJun-driven enzyme collagenase was inhibited by the pretreatment of human skin with the antioxidants genistein and *N*-acetyl cysteine.

Numerous antioxidants, such as vitamins C and E, and coenzyme Q10, as well as botanically derived ingredients (e.g., caffeine, coffeeberry, ferulic acid, feverfew, grape seed extract, green tea, idebenone, mushrooms, polypodium leucotomos, pomegranate, pycnogenol, resveratrol, rosemary, silymarin) are found in skin care products. Despite compelling evidence in the literature substantiating the potency of these antioxidant ingredients, there is a paucity of data demonstrating their efficacy in topical formulations. Research is ongoing to harness their potential in such products, however. Research and development might also yield technological advances in tissue engineering and gene therapy that result in innovative therapeutic applications of growth factors, cytokines, and, perhaps, telomerase (73). Currently, the best approaches to combat cutaneous aging remain behavioral—avoiding sun exposure (particularly between 10 a.m. and 4 p.m.); using broad-spectrum sunscreen daily; avoiding cigarette smoke, pollution, and excessive consumption of alcohol; reducing stress; eating a diet high in fruits and vegetables; taking oral antioxidant supplements or topical antioxidant formulations; and regularly using prescription retinoids.

CONCLUSION

The four traditional expressions used to describe skin type have remained prominent and largely unchallenged over the last century. However, the terms "dry," "oily," "combination," and "sensitive" as characterizations of the skin have been found to be inadequate guides or gauges for finding the most suitable formulations among the ever-burgeoning supply of skin care products. The BSTI proposes that four fundamental skin parameters, covering the spectra from dry to oily, sensitive to resistant, pigmented to nonpigmented, and wrinkled to tight, can be used to better understand and more accurately depict the nature of human skin and identify an individual's skin type among the 16 possible permutations. Because the skin qualities described in the BSTI are not mutually exclusive, all four parameters must be considered when identifying skin type. A four-letter BSTI code is derived from answers to a 64-item self-administered questionnaire, with each letter corresponding to the end of the spectrum of each parameter that an individual favors. With this code, consumers and physicians can more readily select the most suitable OTC skin products, and practitioners may be assisted in treating various skin conditions with the topical formulations most appropriate for a patient's skin type.

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