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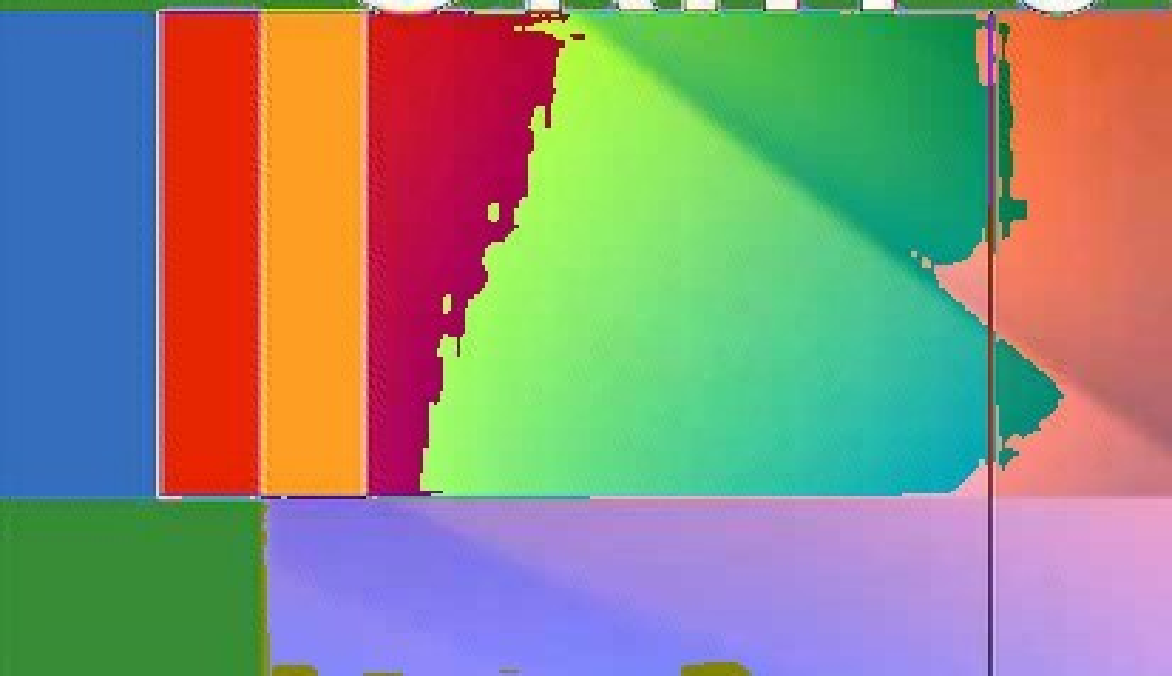

Skin of Color

Andrew F. Alexis • Victoria H. Barbosa
Editors

Skin of Color

A Practical Guide to Dermatologic Diagnosis and Treatment

Foreword by A. Paul Kelly

Springer

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Foreword

As a history buff with a long-standing interest in the history of dermatology, I have a particular interest in the development of specialized treatment for skin of color. The introduction of slavery into the United States in the early seventeenth century resulted in a variety of skin hues as the races mixed. White doctors of the nineteenth century, while recognizing that darker skin often presented different pathology from lighter skin, either applied standard treatments or, worse, experimental ones based on prejudice, myths, and folktales.

It was African-American medical practitioners of the early twentieth century who pioneered new approaches to treating skin of color. Prominent among many other innovators were Drs. T.K. Lawless, John A. Kenney, Jr., Harold Pierce, and Vernal Cave.

Legally sanctioned segregation and the Civil Rights Movement in the United States led to the establishment of racially and ethnically centered hospital complexes, such as the Martin Luther King, Jr. Multi-Service Ambulatory Care Center, formerly known as Martin Luther King Jr./Drew Medical Center, in Los Angeles, CA; medical colleges at Meharry and Howard Universities; Cook County Hospital, Chicago, IL; Homer G. Phillips Hospital, St. Louis, MO; and many others. By serving large populations of darker-skinned patients, dermatologists were able to hone in on their treatments for skin of color.

In Oman, as a 2010–2011 Fulbright Regional Research Scholar at the College of Medicine & Health Sciences, Sultan Qaboos University, I saw firsthand the racial and ethnic mixtures in the population, the result of close Omani ties with East Africa, along with centuries of seafaring ventures. No wonder keloids, the focus of my research project, are as prevalent in Oman as in the United States.

As the world's population swells, the need for dermatologists skilled in treating a variety of racial and ethnic groups becomes ever more urgent. Advances in DNA research undoubtedly will lead to the redefinition of racial and ethnic mixtures. Still, the fact remains that more darkly pigmented skin reacts or displays differently to many dermatological conditions. *Skin of Color: A Practical Guide to Dermatologic Diagnosis and Treatment* provides the sound medical information dermatologists all over the world are seeking.

The editors, Andrew F. Alexis and Victoria H. Barbosa, are present-day pioneers in improving our knowledge and treatments for skin of color, both in clinical and cosmetic dermatology. The authors of the 21 chapters also break new ground in sharing their experience and treatment options. All leading authorities on dermatology address topics ranging from the basics to skin diseases in children, to cultural considerations in treating different racial and ethnic groups, and to issues related to cosmetic dermatology for skin of color. Written in a clear and concise manner, using a vocabulary familiar to a variety of medical professionals who treat skin of color patients, this book will be an indispensable resource in your daily dermatology practice.

Muscat, Oman A. Paul Kelly, M.D.

Preface

Demographic projections in the United States and worldwide indicate that a growing proportion of the global population will consist of individuals belonging to the various nonwhite racial and ethnic groups that are characterized as having skin of color. As such, understanding racial and ethnic variations in the epidemiology, clinical presentation, and treatment of skin and hair conditions is of growing importance to providers of dermatologic care. *Skin of Color: A Practical Guide to Dermatologic Diagnosis and Treatment* is intended to serve as a practical, clinically oriented reference for the management of dermatologic disorders that are more prevalent in the rapidly growing, richly pigmented patient population.

This book is designed to be a useful guide for dermatologists, dermatology residents, physician extenders, medical students, and other health-care providers who are involved in the treatment of skin and hair conditions in patients with skin of color. To this end, the chapters provide practical, "how-to" descriptions by world-renowned dermatologists. The content of the book includes a comprehensive range of medical and aesthetic dermatologic conditions that are relevant to the darker-skinned patient. Chapters covering alopecias, pigmentary disorders, keloids, pseudofolliculitis barbae, and inflammatory disorders with unique manifestations in skin of color provide the reader with practical approaches to the common "bread and butter" dermatologic conditions characteristic of skin of color, while chapters on lasers, chemical peels and microdermabrasion, botulinum toxin, soft tissue fillers, and hair transplantation address the nuances of performing cosmetic procedures in nonwhite patients. In addition, chapters on cultural considerations in African-American, Asian, and Latino populations are intended to serve as a useful guide to ensuring cultural competence when treating various racial and ethnic groups with skin of color.

It has been an honor and a pleasure to invite leading authorities in the field of dermatology to share their practical insights into treating darker-skinned patient populations for the completion of this important project. It is our hope that this book will serve as a practical tool that will help clinicians provide optimum dermatologic care for their patients with skin of color.

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Contents

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Chapter 1
Structural, Physiological, Functional, and Cultural Differences in Skin of Color

Adedamola Adegbenro and Susan Taylor

Skin of color is defined as non-Caucasian skin types, which by Fitzpatricks' classification comprise skin types III-VI. There is great variability in skin pigmentation among distinct racial and ethnic groups, making it difficult to define skin types simply by ethnicity, race, or culture. Individuals with darker skin comprise a wide range of racial and ethnic groups including Africans, African-Americans, African-Caribbean, Japanese, Chinese, Asians, Latinos, Indians, and Pakistanis, to name just a few (Table 1.1) [1].

The US census indicates that by the year 2056, greater than 50% of the US population will be non-Caucasian [2]. Presently, individuals with pigmented skin comprise 80% of the world population [3]. As expected, demand for health care services by individuals with skin of color will grow substantially as this population grows. This demand will also expand as newer technologies and treatment for skin diseases develop. Therefore, the importance of studying the differences that exist in the structure, physiology, function, and culture of ethnic skin cannot be overemphasized.

Most of the early literature on pigmented skin has produced conflicting results that are difficult to interpret or generalize. This is as a result of small sample sizes, non-standardized approaches and methodologies, varying anatomic sites, and the use of subjective parameters. This chapter will summarize the current data regarding ethnic skin differences. An understanding of structural, physiological, and functional differences in pigmented skin will lead to a better grasp of the pathophysiologic mechanisms of skin diseases particularly those that disproportionately affect the ethnic population.

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Table 1.1 Groups that comprise persons defined as having ethnic skin[a]

"Black"	African, people of African descent, including African American, Caribbean-American, and Latin-American persons
Latino or Hispanic	Persons of Spanish and indigenous Central/South American descent, including Central Americans, South Americans, and Caribbean-American; persons of Spanish descent, including Cuban, Puerto Rican, and Dominican
East Asian	Chinese, Japanese, Korean
Southeast Asian and Pacific islander	Filipino, Vietnamese, Cambodian, Thai, Malaysian, Laotian, Burmese, Hmong descent, Polynesian, Micronesian
Australoid	Australian aborigine, Melanesian descent (the Republic of Guinea, Papua, Solomon Islands)
Native Americans	More than 560 recognized tribes, including Inuit (Native Alaskans)
South Asia	Indian, Pakistani, Bangladesh, Sri Lanka
Middle Eastern	Iranian, Iraqi, persons from Saudi Arabia and the Arabian Peninsula (including Kuwait, Bahrain, Oman, Qatar, the United Arab Emirates, Yemen), Lebanese, Afghani, Jordanian, Syrian, *Israeli*, Turkish, North African (Egypt, Morocco, Algeria, Libya)

From Talakoub and Wesley [1], with permission of Elsevier

[a]Traditionally, there are nine geographic races, each with particular genetic similarities. These geographic races include Europeans (which include Middle Eastern and Mediterranean persons), Eastern Indians, Asians, American Indians, Africans, Melanesians, Micronesians, Polynesians, and Australian aborigines. We modify these schema into categories in which ethnic persons share similar anatomic characteristics

Basic Structure and Function of Skin

Stratum Corneum

The stratum corneum deserves special mention given that, as the outermost layer of the skin, it is the skin's primary protective barrier from the environment. It is also the layer of the skin that has received the most attention in studies on differences between skin types. The stratum corneum is a two-compartment system comprised of disk-like corneocytes embedded in a matrix of lipid-enriched membranes. Individual corneocytes contain keratin filaments and osmotically active molecules such as filaggrin encased in a resistant, flexible cell envelope. Within the intercellular spaces of the corneocytes are lipids which provide the permeability barrier of the skin. The lipids include ceramides, cholesterol, long-chain saturated fatty acids, and other less active constituents. These lipids are elaborately organized into multilamellar structures. The stratum corneum functions primarily to prevent water loss from the interior cell layers of the skin. It serves as the interface barrier between the body and the external environment. Consequently, it protects against mechanical insult, irritant/foreign chemicals, ultraviolet light, and microorganisms.

Epidermis

The other layers of the epidermis are highly cellular and composed mainly of keratinocytes. These layers are divided into the basal layer, stratum spinosum, and stratum granulosum based on the morphology of the cells comprising each layer. The basal layer, which is the innermost layer of the epidermis, contains an actively dividing population of keratinocytes. An important constituent of the basal layer of the epidermis is melanocytes, which are derived from neural crest cells and are responsible for the production of melanin pigment. Melanocytes produce melanin which is packaged into organelles termed melanosomes. The melanosomes are subsequently transferred to adjacent keratinocytes via phagocytosis. It is thought that melanin absorbs radiant energy from the sun and protects the skin from the harmful effects of ultraviolet radiation. The ratios of melanocytes to keratinocytes in the epidermis vary by anatomic sites as a function of the degree of sun exposure.

Dermal-Epidermal Junction

The dermal-epidermal junction (DEJ) is an undulating basement membrane zone where the epidermis and dermis adhere to each other. It is composed of two layers known as the lamina lucida and lamina densa. The lamina lucida is an electron-lucent region beneath the basal cell layer. Anchoring filaments from the hemidesmosomes of the basal cells above cross into the lamina lucida below. In turn, anchoring fibrils extend down from the lamina densa into the papillary dermis. Beneath the lamina densa are fingerlike projections of the papillary dermis termed rete ridges. The DEJ is thus a highly irregular junction that greatly increases the surface area over which oxygen, nutrients, and waste products are exchanged between the dermis and the avascular epidermis [4].

Dermis

The primary function of the dermis is to sustain and support the epidermis. The dermis is a more complex structure and is composed of two layers, the more superficial papillary dermis and the deeper reticular dermis. It is made up of collagen, elastic fibers, fibroblasts, blood vessels, lymphatics, and various glands all surrounded by a gel-like ground substance. The fibroblast is the major cell type of the dermis producing mainly collagen and sparse elastic fibers. Although elastic fibers constitute less than 1% of the weight of the dermis, they play an enormously important functional role by resisting deformational forces and returning the skin to its resting shape [4]. Sebaceous glands, eccrine sweat glands, and apocrine glands are epidermal appendages embedded deep within the dermis.

Structural and Physiological Differences

Biophysical Properties: Transepidermal Water Loss, Water Content, and pH

Currently, literature defines transepidermal water loss (TEWL) as the total amount of water vapor lost through the skin and appendages, under non-sweating conditions. TEWL remains the most studied biophysical property in defining differences between various skin types. The data obtained from all previous studies does not lead to consistent conclusions. An in vitro study by Wilson et al. [5] demonstrated that TEWL was 1.1 times greater in Black compared to White skin. In other studies, in vivo evaluation of baseline TEWL showed higher values in Blacks and Asians compared with Whites, but this was after treatment with methyl nicotinate [6, 7]. This led to the conclusion that Black and Asian subjects have a more compromised epidermal barrier function (discussed later in this chapter) that would most likely be more susceptible to irritants.

In contrast, several in vivo studies conducted by Berardesca et al. demonstrated no significant differences in TEWL and water content (WC) between Black, Hispanic, and White skin at baseline [8–10]. However, in these studies, Blacks had higher TEWL than Whites after application of sodium lauryl sulfate, a water soluble irritant, to preoccluded skin. Yet another study using a larger sample size found TEWL in Blacks to be significantly lower on the cheeks and legs compared with that of Whites [11]. This study also found a lower TEWL in the volar forearms of Black compared to White skin, but this difference was not significant. Importantly, these studies have varied in the anatomic sites used with studies using the back and inner thighs demonstrating a greater TEWL in Blacks compared with Whites, while studies of the cheeks and legs found TEWL to be lower in Blacks. One might conclude that inconsistencies in study results may be due to variation in anatomic sites for a particular skin type or to differences in measurement parameters. TEWL is known to be inversely proportional to water content [12].

Water content (WC), a measure of hydration of the skin, has also been studied extensively. WC is measured as a factor of skin resistance, capacitance, conductance, and impedance with the use of skin electrodes. A review of early data on WC demonstrated conflicting results and the use of varying methodologies in assessing ethnic skin differences. One recent study on water content measured by skin electric capacitance found no significant differences between Black, African/Carribean mixed-race, and Caucasian women [13]. Anatomic site variation, skin microrelief, sweat production, and artifacts from topical agents may modify the quality of electrode contact and serve as confounding factors in these studies [14]. This idea is further substantiated by a recent study of a large sample of Chinese individuals that demonstrated SC hydration changes with age, gender, and anatomic site [15]. More recently, a large-scale study using new skin capacitance imaging technique to

compare skin WC among African-American, Chinese, Caucasian, and Mexican subjects in Chicago found that with age, African-American and Caucasian women exhibit lower WC than Chinese and Mexican women [16]. These findings are yet to be validated using the new imaging technique.

Ethnic differences in stratum corneum pH have been explored and deserve some mention. Recent studies have shown that pH regulates homeostasis in epidermal permeability [17]. The most recent study examining pH of the cheek, forehead, and arm did not find any differences among African-Americans, Asian Indians, Caucasians, East Asians, and Latinos [18]. A prior study found a significantly lower pH in Black women compared to Caucasian women only after three tape strippings [19]. Moreover, there were no significant differences in pH with further tape strippings suggesting pH is similar in both races within deeper layers of the skin. Another study by Warrier et al. demonstrated a lower baseline pH on the cheeks in Blacks but not on the legs [11]. The overall results of these studies trend toward the hypothesis that Black skin has a lower pH compared to Whites with some variations according to body sites and depth of skin layers.

Corneocyte Variability and Cell Layers

Differences in the thickness, density, and compactness of the stratum corneum have been studied. Various studies have found that there is no significant difference in thickness of the stratum corneum (SC) between Black and White skin. A comparative study found that in Black subjects, a greater number of tape strippings were required for complete removal of the SC when compared to Whites [20]. Microscopically, Black skin demonstrated higher average numbers of stratum corneum layers than White skin. This led to the conclusion that the stratum corneum is more compact and cohesive in Blacks accounting for the higher number of layers.

Differences in corneocyte surface area have also been examined. A comparative study among African-Americans, White Americans, and Asians of Chinese descent demonstrated no difference in corneocyte surface area, but there was a significant increase in spontaneous corneocyte desquamation by a factor of 2.5 in the Black group [21]. This was attributed to a difference in the composition of the lipids of the stratum corneum. However, another study showed a higher desquamation index of corneocytes on the cheeks and forearms of White subjects [11]. Corcuff et al., in the former study, examined the upper outer arm whereas Warrier et al., in the later study, examined the cheeks, forearms, and lower legs. A recent study using the forehead and volar forearm found no differences in desquamation index among Black, African/Carribean mixed-race, and Caucasian women [13]. Given that corneocyte surface area is believed to vary by anatomic sites in Caucasians, it is possible that corneocyte desquamation also varies by site and not necessarily by race.

Stratum Corneum Lipid Composition

Studies have shown that the intercellular matrix composition of the stratum corneum may vary among ethnic groups. The earliest in vitro studies on epidermal lipid content demonstrated that Black skin has higher lipid content than White skin [22]. However, this study had a small sample size (two Black and four White subjects) and compared skin from both living and cadaveric subjects at different body sites. One experiment demonstrated the lowest levels of ceramide in Blacks followed by Whites, Hispanics, and Asians [12]. This is partially confirmed by another study which reported lower ceramide levels in African-Americans but similar levels for Asians and Caucasians [18]. Lower ceramide levels correlate with higher TEWL and lower water content, suggesting that alterations in the composition and organization of the lipids may have an effect on transepidermal water loss (TEWL) [12].

Of note, a recent study evaluating racial differences in dandruff found no differences in scalp SC lipid content between UK and Thai subjects during the wet season regardless of the presence or absence of dandruff [23]. In the dry season, the Thai subjects had higher levels of free SC lipids. However, this study failed to characterize the specific ethnic groups to which these subjects belonged. Overall, there seems to be some ethnic differences in lipid composition and probably in lipid content as well but current data is inconclusive.

Melanin/Melanosomes

It has been confirmed in various studies that melanin protects the skin from damage by UV light, and it is the source of skin pigmentation [24–26]. There is strong evidence from various studies demonstrating no significant difference in the total number of melanocytes across racial groups [27, 28]. However, studies have shown that melanosomes, the melanin-containing organelles assembled in melanocytes, are more numerous in Africans, African-Americans, and Australian aborigines [25]. Heredity appears to play a role in skin pigmentation, distribution of melanocytes, melanin content, and melanin activity [29]. Variations in melanosome quantity, size, type, and arrangement within melanocytes are deemed responsible for the differences in skin color [28].

Darker skin types have larger, non-aggregated melanosomes that are degraded more slowly than lighter skin types who have smaller, aggregated melanosomes [30]. More recent studies by Alaluf et al. showed an increasing size of melanosomes across ethnicities with Africans having the largest melanosomes followed by Indians, Mexicans, Chinese, and Europeans [31, 32]. Another recent study using electron microscopy confirms that African-Americans have predominantly individual melanosomes (88.9%), Caucasians have predominantly clustered melanosomes (84.5%), while Asians have a combination of individual (62.6%) and clustered (37.4%) melanosomes [33]. This study also found that dark-skinned Africans have larger non-aggregated melanosomes while light-skinned Africans have smaller, aggregated melanosomes. This is consistent with the premise that variations in aggregation and size of melanosomes exist within individual ethnic groups.

The degree of sun exposure correlates with aggregation of melanosomes as demonstrated by a study of Asian skin showing that anatomic sites with less sun exposure have more aggregated melanosomes than more sun-exposed sites [34]. Epidermal distribution of melanosomes varies across races. A contributing factor may be the differences that exist in the rate of degradation of melanosomes. Melanosomes in Black skin are distributed throughout the epidermis with increased numbers in the basal layer, whereas in White skin, they are confined to the basal layer (in fewer numbers) and absent in the upper layers [35, 36]. After transfer of mature melanosomes into the keratinocytes in the basal layer, the keratinocytes become terminally differentiated and migrate to the SC, where the melanosomes are generally degraded [37]. As demonstrated in a recent study, the pattern of degradation of melanosomes in the stratum corneum may vary among ethnic groups given that Black skin had higher numbers of melanosomes in the SC followed by Asian skin and White skin in whom melanosomes are almost absent [33].

Research has also shown a greater rate of melanogenesis and higher melanin content in darker skin types. Lighter skin types (European, Chinese, and Mexican) have approximately half as much epidermal melanin as the most darkly pigmented skin types (African and Indian) as demonstrated in various studies [30, 31]. In vitro cultures of melanocytes from Black skin produce more melanin than those from White skin [38]. The rate of melanogenesis may be determined by intracellular pH of melanocytes. In Black skin, the pH of melanocytes was found to be closer to a neutral pH and optimal for melanin synthesis. A recent study showed that decreased intracellular pH in Whites compared with Blacks correlated with reduced melanogenesis [37]. In contrast to these findings, a recent study analyzing the effects of stratum corneum pH on barrier function demonstrated that vesicular organelles that correspond to melanosomes in darkly pigmented melanocytes (SPT 1V-V) are significantly more acidic than those in their counterparts (SPT I-II) [39]. These findings are yet to be validated by other studies.

In another recent study, rates of melanogenesis following UV insult were found to vary among racial groups [40]. Seven days after a single UV dose, this study found that only darker and more UV-resistant skin types demonstrated significant increase in melanin content. A study by De Winter et al. [41] demonstrated increased and higher p53 immunoreactivity in the epidermis after UV exposure in darker-skinned individuals. p53 is a tumor suppressor gene that upregulates DNA repair enzymes following DNA damage. This would suggest that no skin type is immune to photodamage, but it also suggests more efficient repair system against UV damage of the skin in Black subjects compared with Whites. Further details will be discussed later in this chapter.

Dermal Structure

There appears to be no difference in thickness of the dermis across ethnic groups. A recent study by Bernard et al. using modern imaging studies of the skin of female subjects shows no significant difference in papillary dermis thickness across four ethnic groups (African-Americans, Mexicans, Caucasians, and Chinese) [42].

However, this study demonstrated an increased thickness of the dermal-epidermal junction in African-Americans compared to Caucasians. Also, the subepidermal nonechogenic band (SENEB), a marker of skin aging, was lower in African-Americans than Caucasians with intermediate thickness in Mexicans and Chinese [42]. A more recent study also found no difference in superficial dermis thickness between African and Caucasian skin [43]. This study found that the DEJ in African skin was increased about threefold and more convoluted than in Caucasian skin. Another study on facial skin found more dermal papillae in African-Americans and Hispanics than Caucasians and Asians [44].

Pertaining to the cellular composition of the dermis, studies have shown that the dermis in Black subjects is more compact with smaller, more closely stacked collagen fiber bundles; more numerous fiber fragments; larger and greater number of fibroblasts; and more macrophages compared to White subjects [35, 45]. One of these studies also found no differences in the size and number of mast cells. However, there were greater numbers of and larger melanophages in Black skin as compared to White skin. A more recent study demonstrated that mast cells in African-American skin contained larger granules, increased parallel linear striations (PLS) and increased tryptase levels localized to the PLS in Black compared to White skin [46]. Black skin is known to have a higher predisposition to keloidal scarring, and other early studies suggested the participation of mast cells in aberrant fibrosis seen in these disorders [47–49]. Given the higher levels of tryptase found in Blacks, the investigators in this study suggested involvement of tryptase in the development of keloidal and hypertrophic scars. These differences may also account for more pruritus experienced in Black skin compared to White skin [46].

Functional Differences

Percutaneous Absorption and Barrier Function

Variations in permeability and barrier function of the skin are dependent on epidermal thickness, stratum corneum structure, density of cutaneous appendages, and several other factors [50–52]. Studies of interracial differences in percutaneous absorption of the skin have been contradictory. Most of these studies have used TEWL as a measure of the skin's permeability to water. Although inconclusive, dark skin is thought by some to display superior epidermal barrier function because darkly pigmented subjects typically require more tape strippings to disrupt the epidermal barrier. A study in Black, White, and Asian subjects evaluating TEWL and LDV after irritant exposure showed an increase in TEWL in Asians and Blacks compared to Whites [6]. Another study by Reed et al. showed that darker skin types (SPT V-VI) have a faster recovery back to baseline TEWL than light skin (SPT II-III) after tape stripping [53]. However, a more recent study found no differences in TEWL when comparing Black, African/Caribbean mixed-race, and Caucasians [13].

Studies have suggested that pigment type (not ethnicity, race, or gender) seems to determine differences in barrier function. Reed et al. demonstrated that neither the number of tape strippings required to perturb the barrier nor the rates of barrier recovery were significantly different in White versus Asian subjects or in female versus male subjects [53]. However, patients with skin types II/III required only 29.6 +/− 2.4 tape strippings to perturb the barrier, while the skin type V/VI group required 66.7 +/− 6.9 tape strippings. This was confirmed by another more recent study showing that subjects with darkly pigmented skin (SPT IV-V) have an enhanced epidermal barrier than lightly pigmented skin types (SPT II-III) [39]. The investigators also demonstrated that these differences are secondary to differences in epidermal lipid content and pH-regulated enzymatic mechanisms. Skin surface temperature differences may also contribute to differences in TEWL, which may be a confounding factor in attempts to assess TEWL as a measure of epidermal barrier function.

Racial differences in the skin's rate of absorption of different chemicals have been studied extensively, albeit with inconclusive evidence. This is probably due to the use of several different methodologies and varying techniques for assessing barrier function in these studies. Though it is difficult to make comparisons across studies, there seems to be a trend toward greater permeability to chemicals in White skin compared to Black skin. Studies by Stoughton et al. and Berardesca et al. show less absorption of fluocinolone and nicotinate respectively in Black skin compared to White skin [54, 55]. The study by Berardesca et al. showed no difference in absorption between White and Hispanic skin although the researchers applied nicotinate after skin structure had been altered by stripping or lipid removal. Earlier studies by Wickrema et al. and Guy et al. found no differences in radiolabeled excretion of diflorasone and absorption of methyl nicotinate respectively in Black and White skin [56, 57].

Blood Vessel and Skin Reactivity

Differences in skin reactivity to irritants, sometimes measured by cutaneous blood flow among different ethnic groups, have been studied with inconclusive evidence of differences [58]. Interobserver variability in evaluation of subclinical degrees of irritancy and subjective assessment of erythema as a function of degree of irritation in pigmented skin poses a major challenge in attempts to make comparisons across these studies. There are some recent studies using new objective techniques like laser Doppler velocimetry (LDV) and photoplethysmography (PPG) to assess blood vessel reactivity as a measure of skin irritancy, but they have also produced conflicting results. Consequently, there is no consensus about the relationship between propensity to develop irritant dermatitis and ethnicity.

Earlier studies using erythema as a measure of skin reactivity demonstrated that Black men showed less erythema in reaction to various chemicals [29]. Furthermore, another study showed that White subjects with the lightest complexions were most susceptible to erythema as a measure of skin irritation [59]. However, given the

difficulties in assessing erythema in darker skin, these studies are likely unreliable. Other subsequent studies assessed skin reactivity through its effects on SC integrity and used objective measures such as TEWL, WC, and microcirculation via LDV.

Several studies by Berardesca et al. using these measures demonstrated that Black and Hispanic skin displays a stronger irritant reaction than Whites, whereas Hispanic and White subjects have similar erythematous reactions, albeit more erythema than Black subjects [8–10]. One of these studies by Berardesca show higher TEWL in Blacks and Hispanics after irritant exposure compared to Caucasians, but this was only significant after 0.5% SLS was applied to preoccluded skin [9]. There were no differences in TEWL, WC, and microcirculation of untreated skin after application of sodium lauryl sulfate (SLS) in Blacks, Whites, and Hispanics. However, in another study by Berardesca et al. on racial differences in irritation induced by topical corticosteroid, Blacks showed a decrease in baseline blood vessel reactivity as measured by LDV compared with Whites [60].

Yet another study evaluating skin irritation to 2% SLS as measured by TEWL showed no differences in fair-skinned Chinese, darker-skinned Malaysians, and very dark Indians [61]. Studies in Asian subjects have also been contradictory. One study found increased reactivity to octanoic acid, SLS, acetic acid, and decanol in Asian subjects compared to Caucasians [62]. A study by Foy et al. reported that irritation responses were greater in Japanese women when compared to Caucasian women [63]. However, another study by Aramaki et al. reported no differences in reactivity after an SLS challenge in Japanese and German (Caucasian) subjects [64].

Photoprotection

It is well documented that melanin, by absorbing and deflecting rays of UV light, helps protect the skin from the damaging effects of UV light [24]. A study found that five times more UV light reaches the upper dermis of White skin compared to Black skin [26]. The melanin content and distribution of melanosomes can impact the degree of photoprotection across racial groups. Studies by Szabo et al. [65] and Kaidbey et al. [26] have shown that larger, individually dispersed stage-IV melanosomes (predominantly in Black skin) have a higher melanin content and absorb more UV light than aggregated, smaller melanosomes with less melanin content (predominantly in White skin).

In 1968, Mitchell observed that the Australoid subjects with non-aggregated, large melanosomes were protected from UV-light-induced skin cancers [25]. Australians of European descent, on the other hand, had a high incidence of skin malignancies. Studies have found that rates of basal/squamous carcinomas and melanomas in the USA are 50 and 13 times higher respectively in White skin than in Black skin [66, 67]. An earlier study attempting to explain these differences demonstrated that the main site of UV filtration in fair-skinned subjects is the stratum corneum, whereas in Black subjects, it is the malpighian layer of the epidermis [26]. This study also found that the protection afforded against sunburn in Black epidermis

was on average equivalent to a sun-protective factor (SPF) of 13.4. Also, a study in Japanese women comparing skin color and minimal erythema dose (MED) shows that greater melanin content as evidenced by darker complexion correlated with less severe reaction to the sun [68].

A more recent study demonstrated a higher rate of melanin increase in darker and more UV-resistant skin types 7 days after a single UV dose [40]. However, this study found that neither melanin content nor ethnic groups correlated with the efficiency of DNA damage removal. In contrast, another study by Yamaguchi showed that melanin content correlated inversely with cyclobutane pyrimidine dimers (CPD markers of DNA damage) [69]. This study demonstrated that the correlation coefficient was significantly higher in the lower epidermis compared to the upper epidermis in dark skin. In contrast, fair skin demonstrated similar CPD levels in both the upper and lower epidermis [69]. One prior study demonstrated that changes in the distribution of melanin from the lower layers upward to the middle layer of the skin were more pronounced in Black skin 1 week after UV exposure than in White skin [70]. Rijken et al. recently compared the responses of Black and White skin to solar-simulating radiation and found that subjects with skin of color did not show an increase in neutrophils, active proteolytic enzymes, or diffuse keratinocyte activation unlike Caucasians [71]. The authors suggested that this may explain why Black skin is less predisposed to sunburn and photoaging.

De Winter et al. demonstrated increased p53 (a tumor suppressor gene) immunoreactivity in the epidermis after a single UV exposure, and the levels were higher in darker-skinned individuals [41]. Conversely, another recent study found more p53 protein in nuclei of White skin than Black skin after UV exposure. However, in this study, phosphorylated p53, which actively mediates apoptosis of damaged DNA, was absent in White skin compared to Black skin which also contained a greater number of apoptotic cells [72]. The investigators concluded that UV-induced apoptosis is less frequent in White skin after low dose of UV although it is significant in Black skin, suggesting that Black skin removes UV-damaged cells by apoptosis more effectively. Taken together, these studies suggest that Black skin possesses more efficient mechanisms including facilitating apoptosis of damaged DNA, thus conferring better protection against UV-induced photodamage than White skin. These mechanisms may or may not be independent of melanin content or melanosome distribution. Notably, there are very few studies comparing Asian skin to Black or Caucasian skin.

Vitamin D Production

In the study of human evolution, a number of authors have suggested that lighter skin developed to optimize the production of vitamin D [73, 74]. However, a review of these studies demonstrates equivocal evidence for the theory that populations with lighter skin types maintained selective survival in higher latitudes as a function of vitamin D production [75]. Vitamin D is initiated in the skin at the optimal UVB

wavelengths of 297 nm by conversion of 7-dehydrocholesterol to cholecalciferol, also termed vitamin D3 [76]. It has been documented that UVA does not participate in the initiation of vitamin D production but rather breaks it down in the skin [77, 78]. Vitamin D deficiency was originally known to cause rickets, osteomalacia, and osteoporosis. However, many recent papers have suggested that vitamin D deficiency may correlate with higher rates of cancer, infections, hypertension, cardiovascular disease, and autoimmune diseases [79].

Vitamin D insufficiency is far more common in Blacks than Whites as demonstrated in current literature. Data from the Third National Health and Nutrition Examination Survey showed that in the winter months, 53–76% of non-Hispanic Blacks compared with 8–33% of non-Hispanic Whites had vitamin D levels below 50 nmol/l [80]. The lower cholecalciferol levels in individuals with darker skin may be due to greater pigmentation reducing UV radiation and, hence, vitamin D photosynthesis in the skin. Given sufficient UV exposure, darker skin can produce adequate vitamin D levels as demonstrated in one study [81]. This study further demonstrated that Black and White skin has similar capacity to produce cholecalciferol, but, at usual levels of sun exposure, vitamin D synthesis is less efficient among Blacks.

However, under normal conditions at most latitudes in North America, even young, black, healthy African-Americans do not attain optimal levels of vitamin D at any time of the year [82]. One study shows that children with darker complexions are at risk for suboptimal vitamin D levels [83]. Low dietary intake may contribute to this deficiency of vitamin D in Blacks as demonstrated in several studies [84]. Blacks have a higher prevalence of the medical conditions that have been seen to correlate with vitamin D deficiency. In light of these findings, clinicians should be encouraged to promote improved vitamin D status among Blacks.

Cutaneous Appendages

The eccrine, apocrine, and apoeccrine sweat glands and sebaceous glands are four separately recognized cutaneous appendages contained in the dermis. Race is believed to have evolved as a function of adaptation to variable environmental conditions in a continuum from cold climates to hot and humid climates. This leads to the speculation that differences should exist in sweat gland quantity, structure, and activity among various ethnic groups and race. There are a few inconclusive studies analyzing the differences among sweat glands from various ethnic groups.

A majority of evidence demonstrates no significant racial differences between the quantity and structure of eccrine sweat glands [85, 86] but does suggest differences in the function of these glands. One author found that in response to physical labor and cholinergic stimulation, White Europeans had a higher sweating rate than either Black Africans or Asian Indians [87, 88]. These studies also found that after cholinergic stimulation by pilocarpine, Black Africans had significantly lower concentrations of sodium chloride in their sweat compared to White Europeans or Asian Indians, thus suggesting that Black skin has a more efficient electrolyte

conservation system [87–89]. Other electrophysiology studies that correlate skin resistance with sweat activity consistently demonstrate a higher skin resistance in Black subjects compared to White subjects [58]. One of the studies found that Hispanic and Spanish subjects had an intermediate skin resistance falling between Black and White subjects [90]. However, a couple of studies found no significant differences in onset of sweating and quantity of sweat in Black and White skin [58].

There are a few very early observational studies that suggest that Black subjects have larger apocrine glands and in greater numbers than Caucasians and Chinese [91–93]. However, the small-scale nature and lack of investigator-blinded assessment preclude definitive conclusions. One of these studies noted that Black subjects secreted more turbid fluid with a different odor from their apocrine glands compared to White subjects [93]. The apoeccrine gland which develops at puberty from eccrine glands is localized in the axilla, perianal regions, and nasal skin. It is larger and produces secretions at a rate 10× the rate of eccrine gland secretions. One study showed no differences in the average number of apoeccrine glands among racial groups but did demonstrate high interindividual variability [35]. In contrast, another study found that apoeccrine glands occur in greater numbers in Black versus White female facial skin [94].

Literature analyzing differences in sebaceous glands has produced conflicting results. One study suggests the presence of larger glands with higher sebum production in Black compared to White subjects [95]. However, another study found a higher sebum production on the forehead of Black men compared to White men but an opposite finding in Black compared to White women [96]. In yet another study, no significant difference was seen in sebum excretion of Whites, Blacks, and Asians [97]. A recent study also found no significant difference in sebum quantity among Black, African/Carribean mixed-race, and Caucasian women [13]. The most recent study on facial skin shows greater facial pore area and pore size in African-Americans compared to Asians [43]. This study also found that Hispanics and Caucasians have similar pore area and size as African-American skin. Of note, facial pores correspond to enlarged openings of pilosebaceous units which consist of hair shaft, hair follicle, arrector pili, and sebaceous gland.

The literature contains very few studies on racial differences in sebum composition. One study found that the sebum of both Japanese and Caucasian subjects contains more straight chain fatty acids than branched chain fatty acids [98]. However, Japanese skin had a greater number of C16 isobranched chain fatty acids and greater sebaceous gland activity than Caucasian skin [98]. This study also demonstrated that sebum levels consistently decline with age.

Cultural Differences

Cultural practices and cosmetic needs vary among racial and ethnic groups. These factors may play a role in presentation and predisposition to dermatologic diseases and other conditions in certain ethnic groups. Practices unique to some ethnic groups

include but are not limited to the use of skin-lightening agents, unique hair grooming practices and styles, henna use by Africans and Middle Easterners, and dietary differences such as chili peppers in Hispanic diet. It also includes alternative medicine practices such as cupping, moxibustion, and Cao gio as practiced by Southeast Asians. Dermatologists should be knowledgeable of these practices so as to provide culturally sensitive care to their ethnic patients.

The misuse and excessive use of skin-lightening agents in ethnic populations, particularly of African descent, has been widely reported in literature [99–101]. Several of the products used in the United States are often purchased from the internet or beauty supply stores that cater to the cosmetic needs of African, Caribbean, and Asian communities. Some of these over-the-counter products contain hydroquinone or potent topical steroids such as clobetasol [58]. Potential adverse side effects of these practices have also been studied, and they include exogenous ochronosis, irritant and allergic contact dermatitis, extensive striae, skin atrophy, and adrenal insufficiency [99, 100].

In one study, 33 out of 35 subjects reported that they make mixtures of a superpotent topical steroid diluted with skin-lightening creams, some of which contained hydroquinone concentrations as high as 4–16% [100]. This study also found a distinctive late stage of exogenous ochronosis termed *pigmented colloid milium* in 11% of Africans living in Paris, who reported current or past use of skin-bleaching creams. Another study on the use of skin-lightening products in pregnant women in Senegal found a higher rate of low birth weight and small placentas in those who reported using highly potent steroid creams [102]. These reports underscore the need for dermatologists to increase public awareness and assess for the use of these OTC agents by their ethnic patients.

Due to the unique properties of ethnic hair, African, African-American, African-Caribbean, and Hispanic women use hairstyles and techniques that may lead to temporary or permanent traction alopecia [58]. These hairstyles and techniques include braids with extensions, cornrows, microbraids, twists, hot combing, and curl ironing. Also, hair pomades, which contain mixtures of comedogenic agents like petrolatum and lanolin, are more frequently used in Black and Hispanic populations. The waxy nature of these pomades helps alleviate dry scalp and itching associated with seborrheic dermatitis and also aids in coating and protecting the hair shafts [58, 102]. Adverse events include pomade acne, comedogenic acne involving the forehead and temples.

Henna is a natural dye made from a shrub indigenous to Africa and the Middle East, known as *Lawsonia alba* [102]. When the dry leaves of this shrub are soaked, it produces various colors. This dye is then applied to the skin, hair, and nails for decorative purposes during certain ceremonies and rituals such as circumcisions. However, there have been reported cases of hemolysis and neonatal hyperbilirubinemia secondary to the oxidizing effect of *L. alba* on red blood cells, especially those that are G6PD-deficient [103]. Capsaicin dermatitis is a contact dermatitis caused by chile peppers which is more commonly used by Hispanics as a condiment in their diet [2]. These chile peppers can produce irritation, burning pain, and erythema when handled, and patients should be advised to wear gloves prior to coming into contact with these peppers.

Traditional healing practices common to the Asian population include Cao gio (also termed coining), cupping, and moxibustion. These practices are used on various parts of the body such as the back, chest, shoulders, forehead, and arms for treatment of flu-like symptoms, febrile illnesses, and headaches [104]. They are believed to bring bad blood to the surface and cause bad or excessive wind to escape from the body, thereby restoring balance [105]. Cao gio involves application of an analgesic ointment such as "Tiger balm" or "Monkey balm" to the area to be abraded, after which the skin is firmly rubbed or pinched with the smooth edge of a metal object such as a spoon or coin. This procedure results in erythema, streaks of petechiae, and ecchymosis that eventually disappear in a few days [104–106]. Care must be taken to avoid interpreting these lesions as physical or child abuse.

Cupping is another healing practice that makes use of cups or jars with bell-like suction applied to the skin which pulls the involved skin into the cup or jar for brief moments. This produces circular and ecchymotic lesions that also resolve after a few days. Moxibustion on the other hand results in circular burns from skin contact with heated stick and embers from burning incense [58]. Again, dermatologists should be aware of these presentations in order not to associate them with physical abuse.

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