SUPPORTED BY AN EDUCATIONAL GRANT FROM GALDERMA LABORATORIES

Skin of color: Biology, structure, function, and implications for dermatologic disease

Susan C. Taylor, MD *New York, New York*

People with skin of color constitute a wide range of racial and ethnic groups—including Africans, African Americans, African Caribbeans, Chinese and Japanese, Native American Navajo Indians, and certain groups of fair-skinned persons (eg, Indians, Pakistanis, Arabs), and Hispanics. It has been predicted that people with skin of color will constitute a majority of the United States and international populations in the 21st century. There is not a wealth of data on racial and ethnic differences in skin and hair structure, physiology, and function. What studies do exist involve small patient populations and often have methodologic flaws. Consequently, few definitive conclusions can be made. The literature does support a racial differential in epidermal melanin content and melanosome dispersion in people of color compared with fair-skinned persons. Other studies have demonstrated differences in hair structure and fibroblast size and structure between black and fair-skinned persons. These differences could at least in part account for the lower incidence of skin cancer in certain people of color compared with fair-skinned persons; a lower incidence and different presentation of photo aging; pigmentation disorders in people with skin of color; and a higher incidence of certain types of alopecia in Africans and African Americans compared with those of other ancestry. However, biologic or genetic factors are not the only ones impacting on these differences in dermatologic disorders. Cultural practices also can have a significant impact. Further studies are needed to help dermatologists optimally treat people with skin of color. (J Am Acad Dermatol 2002;46:S41-62.)

Analysis of the population statistics of the United States reveals dramatically shifting demographics in the 21st century. These changes will significantly impact on the practice of dermatology. The United States is becoming a country in which the majority of its citizens will no longer have white skin, but instead pigmented skin, also referred to as skin of color. These people will be of diverse racial and ethnic backgrounds and primarily will include African Americans, Hispanics, and Asians, as well as individuals from these groups who have intermarried. The US 1990 census population data revealed that 76% of the population was composed of whites; 12% of the population was classified as black; 9% Hispanic; 2.8% as Asian/Pacific Islander; and 0.7% as American Indian, Eskimo, and Aleut. In sharp contrast, the US population projection for the year 2050 suggests a significant decrease in the white population to approximately 53% of the total population and concomitant increases in the other segments. The black population is projected to increase to 14% of the population; Hispanics to 25%, Asians to 8%, and American Indian, Eskimo, and Aleut to slightly less than 1%. Contributing to these dramatically changing demographics are changes in immigration patterns in major US cities. Only 50 years ago the majority of immigrants to the United States came from Europe. In contrast, between 1995 and 1996, most immigrants to New York City came from the

Table I. The modern races of *Homo sapiens**

Race	Representative peoples
Caucasoid	Europeans
	Arabs, Indians, Pakistanis
Mongoloid	Asians
Australoid	Australian Aborigines
Congoid or Negroid	Africans
	African Americans
	African Caribbeans
Capoid	Kung San tribe of Africa

*Data from Coon.[4]

From the Skin of Color Center, Department of Dermatology, St. Luke's-Roosevelt Hospital, New York.

This article is part of a supplement supported by Galderma Laboratories.

Disclosure: Dr Taylor is a clinical investigator for Allergan, Galderma, and Hill. She is also a member of the Advisory Board for Galderma, Medicis, Proctor and Gamble, and Roche, and is a speaker for Allergan, Galderma, Medicis, Novartis, and Roche.

Reprint requests: Susan C. Taylor, MD, Department of Dermatology, St. Luke's-Roosevelt Hospital Center, 1090 Amsterdam Ave, Suite 11D, New York, NY 10025. E-mail: staylor@slrhc.org

0190-9622/2002/$35.00 + 0 **16/0/120790**
doi:10.1067/mjd.2002.120790

Table II. Reactivity of human skin to solar radiation based on skin phototypes I to IV[6]*

Table available in print only

DT, Delayed tanning; *IPD*, immediate pigment darkening; *UV*, ultraviolet; *UVR*, ultraviolet radiation.
*Based on about 3 MED or 90-120 mJ/cm^2 of sun exposure of untanned skin without previous sun exposure.
†Based on about 5 to 6 MED exposure (mJ/cm^2) inducing redness at 24 hours.
Note: + + + +, very strong reaction, violaceous redness, edema, pain with or without blistering; + + +, strong reaction, brick-red color with edema, pain; + +, moderate reaction, redness without edema and pain; +, weak reaction, pink redness; ±, minimal redness.
From Pathak MA, Nghiem P, Fitzpatrick TB. In: Freedberg IM, Eisen AZ, Wolff K, et al, editors. Fitzpatrick's dermatology in general medicine. Vol. 1. New York: McGraw-Hill; 1999. p. 1606. Reproduced with permission of The McGraw-Hill Companies.

former Soviet Union, the Dominican Republic, China, Jamaica, Guyana, Bangladesh, India, Haiti, Ecuador, Trinidad, Tobago, and the Philippines.[1] Global population statistics also suggest a predominance of people with skin of color.[1]

These changing international, national, and regional demographics underscore the need for and importance of a thorough understanding of pigmented skin or skin of color. The dermatology community may be faced with cutaneous diseases that occur more often in people with pigmented skin, present differently, and/or are unique to this population. Additionally, all health care providers will need to understand the cultural habits and practices of people with skin of color that can impact on disease.

To meet these challenges, this article will attempt to (1) provide a definition of skin of color or pigmented skin; (2) review the existing literature on the structure, physiology, and function of skin of color; (3) discuss the disease implications of the preceding findings; and (4) review the cultural habits and practices of individuals with pigmented skin.

DEFINING SKIN OF COLOR

Racial and ethnic differences

Defining pigmented skin or skin of color obviously entails a discussion of the various races and ethnic groups of our species, *Homo sapiens*. Skin color and the skin's reaction to such environmental factors as sunlight, as well as other obvious phenotypic manifestations, were originally used to categorize the subspecies (ie, races) of *H sapiens*. More recently, molecular analysis has identified genetic differences between races and ethnic groups.[2] Most anthropologists believe that racial variation developed through natural selection processes; that is, different biologic traits in the races developed because these traits facilitated adaptation to a particular environment. For instance, it is believed that darkly pigmented skin evolved to protect those people living close to the equator from ultraviolet (UV) light. People who live north of the equator, on the other hand, probably have paler skin to ensure adequate absorption of UV rays to promote vitamin D formation in the basal layer of the epidermis.[3]

Despite these acknowledged differences, racial classifications are more or less arbitrary.[3] Furthermore, variation between individual members of a racial or ethnic group may at times assume greater importance than interracial variation in its impact on health and disease.

Our species has been divided into varying numbers of subspecies or races. These have included Caucasoid (eg, Europeans); Mongoloid (eg, Asians); Congoid or Negroid (eg, most African tribes); Capoid (eg, the Kung San African tribe); and

Australoid (eg, Australian aborigines)[4] (Table I). Obviously, these 5 races do not provide an exhaustive categorization of all people of the world. Based on this system of classification, most of these racial groups would consist of people with skin of color. Even certain Caucasoids (eg, Indians, Pakistanis, and Arabs) have pigmented skin.

In the United States, the racial and ethnic classification of those individuals with pigmented skin or skin of color would include African American black persons (including Caribbean American black persons), Asian and Pacific Islanders (including those of Filipino, Chinese, Japanese, Korean, Vietnamese, Thai, Malaysian, Laotian, or Hmog descent), Native Americans, Alaskans, and Aleuts, and those who report Latino or Hispanic ethnicity (including people of Mexican, Cuban, Puerto Rican, Central American, or Spanish descent). Also included are certain people traditionally categorized as Caucasoids, such as the majority of Indians, Pakistanis, and those of Middle Eastern origin. Though these people have been classified as Asians or Mongoloids, classic Mongoloids are those people who evolved in a cold climate and evidence certain phenotypic characteristics adapted to that environment, such as sparse body hair.

The Skin Phototype (SPT) system has been used classically by dermatologists to categorize all people, including those with pigmented skin. This system, developed by Fitzpatrick, is predicated on the reactions or vulnerability of various types of skin to sunlight and ultraviolet radiation (UVR)[5,6] (Table II). It correlates the color of skin with its dynamic ability to respond to UV light with burning or tanning. This classification system was developed to categorize white skin; all skin of color was initially classified as skin type V.[7] Obviously, skin of color encompasses greater color gradations. Subsequently, skin of color was divided into 3 groups: type IV, V, and VI. It is widely accepted in the dermatologic community that an individual with an olive skin tone, also characterized as beige or lightly tanned, is classified as having type IV skin; those with brown skin as type V; and black skin as type VI. For the purposes of this article, we will define pigmented skin or skin of color as that skin that meets the SPT IV to VI criteria. These skin types rarely or never burn on sun exposure and tan readily. These skin types include individuals of many racial and ethnic backgrounds. A majority of African Americans, Caribbean Americans, and Hispanic Americans would therefore be classified as having Fitzpatrick skin types IV through VI. Furthermore, there are many Asian Americans (eg, Vietnamese and Koreans) and even fair-skinned persons (eg, Arabs, Pakistanis, Indians) who also would be classified as having types IV and V skin. Of course, a segment of

Table III. Ultraviolet light-induced minimal erythema dose (MED) and minimum melanogenic dose (MMD) for skin phototypes

Table available in print only

From Pathak MA, Nghiem P, Fitzpatrick TB. In: Freedberg IM, Eisen AZ, Wolff K, et al, editors. Fitzpatrick's dermatology in general medicine. Vol. 1. New York: McGraw-Hill; 1999. p. 1606. Reproduced with permission of The McGraw-Hill Companies.

each of the previously described ethnic groups could be classified as having SPT III or even SPT II skin. This reflects the variability between the races as well as intermarriage between races. Indeed, one criterion for defining a subspecies or race is the inclination for members of these subspecies to interbreed.[3] Contemporary African Americans, for example, are a mosaic of indigenous Africans, European white persons, and Native Americans. US intermarriage patterns as of 1998 reveal 9% white/black intermarriage rates; 19% white/Asian; 12% white/Native American; 52% white/Hispanic; and the remaining 7% involving other admixtures. Although the racial or ethnic classification for the offspring of interbreeding may be more difficult to determine, a SPT still can be assigned to each person.

Despite being entrenched in the dermatologic community, the SPT classification system has limitations. For instance, the SPT has been used to predict the minimal erythema dose (ie, the MED or smallest amount of radiation needed to produce a perceptible erythema response) for various skin types or the minimum melanogenic dose (MMD), colloquially known as "tanning" (Table III). The SPT may be irrelevant to people with skin of color. It has been observed that in some individuals with skin of color there is often no relationship between constitutive skin color, skin phototype, and MEDs. Youn and colleagues[8] demonstrated this for Asians. In their study of a Korean population, skin types encompassed SPTs of II, III, IV, and V rather than only V. Furthermore, MED values ranged from 50 to 90 mJ/cm^2, whereas the SPTs would suggest MED values ranging from 25 to 90 mJ/cm^2. Leenutaphong[9] showed that individuals from Thailand encompassed phototypes II, III, IV, and V; constitutive skin color did not correspond well to the Fitzpatrick classification system in the older age group. Furthermore, there was also great variation in MED values as well as overlap in values between different skin types. Recognizing the limitations of a system designed to evaluate fair skin, which is then applied to skin from other races, Kawasa[10] adapted the SPT system to better categorize skin types of Japanese individuals. He created a 3-level classification system based on sensitivity to UV light.

Other skin classification systems, based on factors other than the effect of UV radiation, should be entertained. The Lancer Ethnicity Scale (LES) is a classification system designed to calculate healing efficacy and times in patients undergoing cosmetic laser or chemical peel procedures[11] (Table IV). The patient's skin color is an important factor to consider in assessing the risk involved with such procedures.

Individuals with skin of color might be better served by a skin classification system based on criteria other than sensitivity to UV radiation or healing efficacy. For instance, a classification system based on the propensity of the skin to become hyperpigmented caused by an inflammatory stimulus and to sustain that hyperpigmentation for prolonged periods may prove valuable. Dermatologic practitioners are aware of the reactivity of melanocytes and the profound tendency to hyperpigment as unique characteristics of pigmented skin.

BIOLOGY, STRUCTURE, AND FUNCTION OF SKIN OF COLOR

Biology of pigmentation

A hallmark biologic feature in people with skin of color is the amount and epidermal distribution of the cutaneous pigment, melanin. The biosynthesis of melanin occurs within the metabolic unit of the melanocyte, the melanosome.[12,13] The melanocyte, an exocrine cell, is present in the basal layer of the epidermis and in the matrix portion of the hair bulb. During the 18th week of embryonic development, neural crest melanoblasts migrate to the epidermis and differentiate into melanocytes.[14-16]

Each basal layer melanocyte is associated via the dendrites of the melanocytes with 36 keratinocytes located in the malpighian layer of the epidermis.[17,18] The association of each melanocyte with these keratinocytes is known as an epidermal melanin unit. It is within the machinery of the melanocyte that the 2 types of mammalian melanin, eumelanin and pheomelanin, are produced. The enzyme tyrosinase is critical to the formation of these 2 subtypes of melanin. This enzyme is formed within the Golgi apparatus of the melanocyte and is transferred to the melanosome in its first stage of development (stage I). Tyrosinase

Table IV. Lancer Ethnicity Scale (LES)

Table available in print only

From Lancer HA. Lancer Ethnicity Scale (LES) [correspondence]. Lasers Surg Med 1998;22:9. Reprinted by permission of Wiley-Liss, Inc., a subsidiary of John Wiley & Sons, Inc.

and additional proteins are assembled in stage II melanosomes. Tyrosinase first converts tyrosine to dopa, then converts dopa to dopaquinone. The subsequent cyclization and oxidation of dopaquinone results in the brown-black, alkali-insoluble eumelanin. These chemical processes occur in the stage III and IV melanosomes.[15,19] Predictably, the stage III and IV melanosomes have greater melanin content.[20] The yellow-red, alkali-soluble pheomelanin is formed by a shunt in the eumelanin pathway; dopaquinone combines with cysteine or glutathione to ultimately form pheomelanin in the stage III and IV melanosomes. The stage IV melanosomes move from the melanocyte dendrite into the keratinocyte either as single, discrete particles or as complex, aggregated particles.[21]

Biology of melanin and racial differences

It is well established that there are no racial differences in the number of melanocytes.[22,23] However, the actual number of melanocytes may differ from one individual to another and from one anatomic region of the body to another; for instance, the head and forearm have the highest number.[24]

Racial and ethnic differences in skin color are due to variations in the number, size, and aggregation of the melanosomes within the melanocyte and keratinocyte.[22] Racial or ethnic differences in the size and aggregation of melanosomes within keratinocytes have been clearly demonstrated.[20-26] In 1969, Szabo and colleagues[25] examined melanosome distribution in Caucasoids, Mongoloids (Japanese and Chinese), and Negroids. The melanosomes of the 5 Caucasoid subjects studied were grouped or aggregated together within a surrounding membrane. The melanosomes of the 3 Mongoloid subjects were likewise grouped in aggregates but there was a more compact configuration compared with those of the Caucasoid subjects. This finding was believed to be secondary to less ground substance. In contrast, the melanosomes of the 7 Negroid subjects were not aggregated but individually dispersed. Additionally, in the Negroid group of subjects, there were occasional closely packed doublets of melanosomes and only a few of the melanosomes were in groups or aggregates.[25] Toda et al[24] and Olson et al,[27] expanding on this initial research, demonstrated that different groupings of melanosomes correlated with the lightness or darkness of the individual subject's skin color. For instance, dark-skinned black subjects had nonaggregated, large melanosomes, whereas light-skinned black subjects had both large nonaggregated and smaller aggregated melanosomes.

Melanosome groupings were also affected by sun exposure. Asian skin exposed to sunlight, such as forearm skin, had a predominance of nonaggregated melanosomes, whereas unexposed skin, such as on the lower abdomen, had predominately aggregated melanosomes. Dark-skinned white subjects with sunlight-exposed skin had nonaggregated melanosomes, whereas light-skinned white subjects with no sun exposure had aggregated melanosomes.[24,27]

A comparison by Mitchell[28] of Australoid and Caucasoid subjects from Australia revealed that the melanosomes of the Australoid subjects were nonaggregated, very dense, and much larger than those of the Australian Caucasoid subjects. The melanosomes of the Caucasoid subjects were also aggregated.

Toda et al[24] attached size restrictions to the ability of a melanosome to aggregate in a single package. They concluded that melanosomes smaller than 0.8 × 0.3 μm are physically able to be grouped together in the form of a membrane-bound unit, known as a phagosome, whereas melanosomes larger than 0.8 × 0.3 μm cannot physically or structurally be grouped together in this unit.

Olson et al[27] made similar observations. Melanosomes in Negroid subjects with dark complexions averaged 0.69 × 0.28 μm compared with 0.40 × 0.17 μm in fair-skinned subjects. They concluded that melanosomes larger than 0.35 μm are usually not found in complexes in either Caucasoid or Negroid subjects.

These data reveal that in subjects with skin of color, especially those of African-American descent, there is a trend for melanosomes to be large and nonaggregated. However, all black persons do not have nonaggregated melanosomes. This is consistent with the variation in skin hues and the range of SPTs in this racial group. Darker-skinned black subjects (those of SPT VI) have larger, nonaggregated melanosomes, whereas those black subjects with lighter skin tones have smaller, aggregated melanosomes. Additionally, it has been determined that melanosomes are larger, more oval, and denser in dark-skinned individuals compared with lighter-skinned individuals.[21] Similarly, all white and Asian subjects do not have small melanosomes, nor are the melanosomes always aggregated in the skin of people from these racial groups. These facts again underscore the significance of individual variation in each racial group.

Total melanin content is greater in individuals with SPT VI compared with those with SPT I and II, as determined through melanocyte cultures.[29]

In addition to differences in the grouping of the melanosomes, Toda et al[24] demonstrated an increased number of basal layer melanosomes (340 per basal cell) in dark-skinned black subjects compared with the light-skinned black subjects (120 melanosomes per basal cell). This same trend toward fewer basal layer melanosomes in more lightly pigmented Asian and fair-skinned subjects compared with darkly pigmented subjects was demonstrated by Goldschmidt and Raymond.[30]

Racial differences in the epidermal distribution of melanosome were highlighted by Montagna and Carlisle.[26] Melanosomes in black skin were distributed throughout the entire epidermis, including the stratum basale, granulosum, lucidum, and corneum. This finding was confirmed by Herzberg and Dinehart.[31] In contrast, in fair white skin, only a few melanosomes were observed in the stratum basale and malphighian layer; melanosomes were reported to be absent in the upper layers of the epidermis in fair, white, unexposed skin.

However, the distribution of melanosomes in dark, white skin was noted to resemble that of black skin.[26,27] Thus, there appears to be a correlation between melanosome distribution and skin tone or color. Kotrajaras and Kligman[32] demonstrated in a group of Thai subjects that melanosomes were distributed throughout the entire epidermis with dense clusters in the basal layer and heavy pigmentation in the stratum corneum.

Function of melanosomes and melanin

Both the epidermal content of melanin and packaging and distribution of melanosomes can impact on photoprotection.[30] Many studies have demonstrated that melanin confers protection from UV light.[27,28,33] Melanin appears to absorb and deflect the rays of UV light.[33] In addition, a greater number of individually dispersed[33] and stage IV melanosomes[34] also appears to confer photoprotection.[25] The larger, individually dispersed, stage IV melanosomes have a higher melanin content and are able to absorb more UV light energy than the aggregated, smaller melanosomes with less melanin content found in fair-skinned white subjects.[33]

In 1968, Mitchell[28] observed that the Australoid subjects with nonaggregated, large melanosomes were protected from UV-light-induced skin malignancies. Australians of European descent, on the other hand, had a high incidence of skin cancer. In the 1950s Thompson[35] evaluated stratum corneum thickness in Nigerian Africans, including one albino, and white Europeans. They concluded that skin color rather than stratum corneum thickness was mainly responsible for racial differences in skin reflectance measurements. They noted that the transmission of light through the skin of the albino African was similar to that of the white Europeans. Olson et al[27] demonstrated a racial differential in the MED. Individuals with darkly pigmented, black skin had an average MED 15 to 33 times greater than that of individuals with white skin. Kaidbey et al[33] measured the transmission of UV radiation through white and black cadaveric skin to assess quantitatively the photoprotective effect of melanin. By using skin samples as filters, they determined that (1) the main site of UV filtration in fair-skinned subjects is

the stratum corneum, whereas in black subjects, it is the malphighian layer; (2) because no differences in thickness or composition of epidermal layers in the 2 racial groups exist, the differences in transmission were mainly due to the pigment melanin; (3) melanin acts as a neutral density filter reducing all wavelengths of light equally; (4) although wide interindividual variations among black subjects apparently was due to differences in pigmentation, the protection afforded by a black epidermis against sunburn was on average equivalent to a sun-protective factor (SPF) of 13.4.[33] Abe et al[36] compared the skin color and MED of 101 Japanese women. They demonstrated that the greater the epidermal melanin content, as evidenced by darker complexion, the less severe the reaction to the sun.

A recent study by Lee and Kim[37] determined that darker skin color conferred photoprotection, at least in younger subjects. The patient population was divided into 2 groups: 40 subjects aged 20 to 39 years, and older subjects aged 43 to 63 years. Both groups encompassed SPTs II through V. Both the UVB-induced MED and the UVA-induced minimal immediate pigment darkening dose (MIPDD) were determined. An inverse relationship between SPT and UV-light sensitivity was found in the younger patient population; however, no relationship was found between SPT and the older patient population. The authors speculate that aging of the skin may have altered constitutive skin color; the obvious skin color recorded in these patients may not have reflected the actual skin color. Alternatively, the authors suggested that the SPT system may not be as predictive of UV-light sensitivity in darkly pigmented races. (The most common skin phototype in this population was skin type V.) Thus, as previously discussed, other skin classification systems may be warranted.

Although melanin confers a protection from UV radiation, Kotrajaras and Kligman[32] reported that pigmented skin can also experience significant photodamage, manifested by epidermal atypia and atrophy, dermal collagen and elastin damage, and marked hyperpigmentation. They studied the use of topical tretinoin on photodamaged facial skin in Asian women from Thailand (average SPT IV). They were surprised at the extent of actinic damage in subjects over the age of 60 years, and noted that melanin is not an efficient absorber of UV light of longer wavelength (eg, UVA spectrum > 320 nm). In addition to speculating that the damage was UVA induced, they also suggested that infrared radiation may also overwhelm the protective effects of melanin. Furthermore, it has been suggested that melanin is both a photoprotector and a photosensitizer. This pigment can be photoreactive, resulting in the production of damaging free-oxygen radicals.[38]

Racial differences in stratum corneum structure

There are conflicting data regarding racial differentials in stratum corneum structure. Many of the studies cited in the literature have small patient populations and less-than-optimal study designs. Consequently, definitive conclusions cannot be made, and further research is warranted.

An early study by Weigand et al[39] investigated the cell layers and density of the stratum corneum in black and fair-skinned subjects, as measured by the number of tape strips necessary to completely remove the stratum corneum layer, microscopic observation, and measurement of air density in the stratum corneum. In fair-skinned subjects, 6 to 15 tape strips (mean 10.3) were required to completely remove the stratum corneum compared with 8 to 25 strips (mean 16.6 strips) in black subjects. Although there was overlap between these 2 ranges, the mean difference between the races was significant ($P < .01$). There was also greater variability in the amount of tape strips used in black subjects compared with fair-skinned subjects, but this variability was not correlated with pigmentation. Microscopic visualization also demonstrated racial differences in stratum corneum. Black subjects demonstrated greater air density in the stratum corneum; however, buoyant densities were similar between the races. Because the average stratum corneum thickness was found to be similar between the 2 racial groups, it was concluded that black stratum corneum was more compact than the white stratum corneum, perhaps reflecting greater intercellular cohesion.[39]

Reed et al[40] compared the skin structure of subjects with skin types V and VI (4 African American, 2 Filipino, and 1 Hispanic) to those with lightly pigmented skin types II and III (6 Asian and 8 fair-skinned subjects.) The darkly pigmented subjects required more tape strippings to disrupt the epidermal barrier. The authors drew 2 conclusions from these data. First, darkly pigmented skin probably had more cornified cell layers, and therefore was more compact than lightly pigmented skin. Secondly, dark skin was thought to display superior epidermal barrier function. These data are particularly interesting because the differences in cell layers and barrier protection were demonstrated to be related to the skin hue and SPT, not race.

La Ruche and Cesarini,[41] in a review of other authors' research, concluded that stratum corneum compactness and lipid content were greater in black skin compared with white skin. Again, their conclu-

sions were based on studies involving small patient populations.

Corcuff et al[42] examined corneocyte surface area, mean surface area, and spontaneous desquamation in African Americans, white Americans, and Asians of Chinese extraction (18 to 25 subjects per group). There was no difference in corneocyte surface area between all 3 racial groups, and no difference in spontaneous corneocyte desquamation between the Chinese and white group. However, spontaneous desquamation was increased in the black group compared with the white and Asian groups. These data appear contradictory to Weigand's findings. If black skin requires more tape strippings to remove the stratum corneum, then we would expect that the spontaneous desquamation to be less not greater in black skin. Research by Warrier et al[43] does not vindicate these findings regarding desquamation. They studied 30 black and 30 white age-matched subjects. The desquamation index was found to be greater on the cheeks and forehead of white subjects compared with black subjects.[43]

Johnson[20] has reported that black skin shows a trend toward having a thicker stratum corneum compared with white skin. However, other studies do not confirm his findings. Thomson[35] measured the stratum corneum thickness of 17 white Europeans and 20 Nigerian Africans and found no significant difference between the 2 groups. Further studies[20,26,28] that used various methodologies also failed to demonstrate differences in stratum corneum thickness between black skin (of Africans, Australoids, and African Americans) and white skin (of Europeans and Americans).

Finally, other studies have suggested that there is a greater lipid content in black stratum corneum compared with white stratum corneum.[41,44] This could account for the greater density of black stratum corneum, a conclusion supported by Weigand et al.[39] They demonstrated that when lipids were removed from stratum corneum samples extracted from subjects of both races, the specimens were of equal weight.

The structure of the stratum corneum ultimately impacts on barrier function and hence the occurrence of irritant reactions. The literature on racial and ethnic differentials in cutaneous irritants will be reviewed later in this article.

Racial differences in epidermal structure

Racial differences in epidermal structure between various races, especially Negroid and Caucasoid, have also been documented by various studies. These differences become especially pronounced during extrinsic aging (eg, sun-induced) and intrinsic aging.

Montagna and Carlisle,[26] using punch biopsy specimens, investigated differences in epidermal structure between 19 black and 19 white American female subjects. Their findings included the following: (1) There were interindividual variations in the thickness of the epidermis within both groups; the epidermis was described as thin in some subjects and thick in others; (2) there was a stratum lucidum of 1 to 2 layers of non–sun-exposed skin in both ethnic groups; (3) there was a compact and unaltered stratum lucidum in sun-exposed black skin but a swollen, cellular stratum lucidum in sun-exposed white skin; and (4) there was a stratum granulosum consisting of up to 3 layers in people from both racial groups. Marked differences in atrophy and cell cytology were noted between the 2 racial groups. Minor epidermal atrophy in one of 19 black subjects was noted. Although some black subjects had no histologic flaws at all, others evidenced vacuoles and dyskeratosis in their malpighian layer. On the other hand, subjects with white skin evidenced numerous focal areas of atrophy and necrosis with readily apparent vacuoles and dyskeratosis. Herzberg and Dinehart[31] did observe certain predictable skin changes due to chronologic aging in black subjects of varying ages. Older black adults clearly evidenced epidermal thinning compared with young adults and children. Additionally, rete ridges were less pronounced as black skin matured.

Whitmore and Sago[45] measured epidermal and dermal skin thickness on non–sun-exposed forearm skin with Harpenden calipers in 86 white and 40 black women. Their motivation for hypothesizing that there would be a difference between the races is based on findings of greater bone density in black versus white premenopausal women.[46] (Both the bone matrix and skin dermis contain the same type of collagen.) They controlled for potential confounding variables, such as age, oral contraceptive use, postmenopausal hormone replacement therapy, and cigarette smoking. No statistically significant difference in skin thickness between these 2 racial groups was observed.

Kotrajaras and Kligman[32] demonstrated epidermal atrophy, cell atypia, and poor polarity as well as disorderly differentiation in the skin of Thai subjects over age 50 with heavy sun exposure.

Racial and ethnic differences in cutaneous appendages

Eccrine sweat glands. Eccrine sweat glands are a key part of the body's thermoregulatory system. Because of the established premise that the races evolved as a result of environmental selection, it is plausible to believe that a racial differential in eccrine

sweat glands between a race adapted to a hot-and-humid climate (eg, the Negroid race) compared with races adapted to colder climates could exist. Ethnic and racial differences in the quantity, structure, and function of the eccrine sweat glands are controversial. Indeed, the bulk of the evidence suggests no significant differences between the races.

Although interindividual differences in the number of eccrine sweat glands across races have been established by Szabo,[47] a similar differential does not appear to exist between black and white persons.[48,49] However, a racial differential in functional activity of eccrine sweat glands has been noted.

In 1941 Robinson et al[50] reported higher sweating rates by white Americans compared with black Americans during physical labor. McCance et al[51,52] measured sweat production on the forearm of subjects from 3 different racial groups in response to physical labor and cholinergic stimulation by pilocarpine. (The eccrine glands are innervated by the cholinergic system.) They reported that white Europeans had a higher sweating rate than either black Africans or Asian Indians. However, there were technical difficulties associated with the measurements. Additionally, in this study, the measurement of pilocarpine-stimulated sodium concentration in sweat revealed that the black Africans had a significantly lower sodium concentration in their sweat compared with white Europeans or Asian Indians. This may reflect the more efficient electrolyte conservation observed with those of African ancestry.[53] Herrmann et al[54] measured the onset of sweating and quantity of sweat produced from 4 black male Americans compared with 16 white male Americans. They found no significant difference between the 2 groups. Rebel and Kirk[55] also found no differences between sweating in black and white subjects.

A number of electrophysiologic studies indirectly compared the sweating of white and black subjects through skin resistance measurements.[56-59] (Skin resistance is correlated with greater eccrine gland activity.) All studies found a higher skin resistance in black subjects compared with white subjects. One of the trials also assessed this activity in Hispanic subjects of Mexican origin and Spanish subjects.[56] Both Hispanic and Spanish subjects had a mean resistance between that of the white and black subjects. Because Spanish subjects are also considered to be Caucasoid, whereas many Mexicans represent racial admixtures, pigmentation may also have played a role in these findings. Therefore, dark-skinned individuals are reported to have higher skin resistance than white or fair-skinned individuals.

It is not clear whether these observed differences are based on genetics or environmental adaptations. A study by Kawahata and Saramoto[60] of the Ainu, a Japanese ethnic group that appears to represent a mixture of Australoid and Caucasoid races, supports the influence of the latter. They demonstrated that Ainu born in Japan who migrated to the tropics had the same number of sweat glands as Ainu born in Japan who continued to live in Japan. However, Ainu born in the tropics had a larger number of sweat glands than the other groups.

Apocrine sweat gland. The apocrine glands, located in the axillae, perineum, and external auditory canal, develop from the pilosebaceous unit.[60,61] These glands become active just before puberty. Though their physiologic function remains unknown, their odor may serve as a sexual signal.

A review of the literature pertaining to apocrine gland structure and function among the races reveals only 3 studies of less-than-optimal design. The small number of subjects in these studies and the absence of investigator-blinded assessment preclude definitive conclusions.

The first was published in 1922 by Schiefferdecker,[62] who evaluated the apocrine glands of 3 black subjects, 1 Asian subject of Chinese origin, and 12 white subjects of German ancestry. He concluded that the black subjects had larger apocrine glands and in greater numbers than either of the other subjects. In 1926 Homm[63] did a histologic analysis of 538 skin sections from white subjects and 631 sections from black subjects. The skin samples were taken from 4 apocrine-bearing areas of the body. He observed that apocrine glands occurred 3 times as often in black subjects compared with white subjects. However, none of the specimens were obtained from the axillae—the area of the body containing the greatest number of apocrine glands. The final study performed in 1960 by Hurley and Shelley[64] also investigated apocrine gland structure and function in 30 black and white subjects. They reported larger apocrine glands in black subjects compared with white subjects. They also evaluated apocrine secretions as induced by emotional stimulation or epinephrine and found a greater amount of secretions in the black subjects. The researchers also engaged in a subjective appraisal of the secretions and determined that the secretions from black subjects were more turbid and produced a "unique axillary odor."

Apoeccrine gland. The apoeccrine gland develops at puberty from an eccrine gland or an eccrine-like precursor gland. It is present in the axilla, perianal, and nasal skin.[65] This gland, sometimes referred to as the "mixed sweat gland," has characteristics of both eccrine and apocrine glands. The secretory rate of the gland is estimated at 10 times that of the eccrine gland. It is felt to be an eccrine gland that underwent apocrinization.[65]

There is great interindividual variation in the number of apoeccrine sweat glands.[61] At least one study has documented interracial variation as well. Montagna and Carlisle[26] found that the apoecccrine gland occurred more frequently in black than white female facial skin. The significance of this finding is unclear.

Sebaceous gland. The sebaceous gland, which forms during the 14th week of gestation, is attached to the hair follicle by a duct. The sebaceous gland produces sebum. Sebum is composed of various lipids, including squalene, cholesterol, cholesterol esters, wax esters, and triglycerides that transcend the follicular canal to the skin surface.[66]

The few studies of racial differences in sebaceous gland size and activity are often contradictory. Again, such controversy is probably due to lack of well-controlled protocols, methodologic flaws, and small study populations. Racial differences in sebaceous gland size and activity have been suggested. Nicolaides and Rothman[67] assessed sebum production in black and white subjects by measuring the lipid concentration in the hair. They found that black subjects had 60% to 70% more lipid content in their hair compared with white subjects. Based on this finding, they concluded that blacks evidenced greater sebum production. Racial differences in hair density, length, and diameter were not controlled for in the study and could have confounded the results. Furthermore, hair lipid levels are probably not an accurate measure of either sebum production or sebum levels; these levels may merely reflect the follicular reservoir of sebum.

Kligman and Shelley[68] measured sebum production as well as sebaceous gland size in 5 black and 5 white subjects. Taking measurements during a 4-hour period, they found that black subjects had higher sebum levels compared with white subjects. By using biopsy specimens from 2 black subjects who produced high or moderate levels of sebum and 2 white subjects who produced low sebum levels, they also found that the black subjects had sebaceous glands that were much larger than those of the white subjects.

Champion et al[69] determined that the sebaceous glands of black subjects were larger than those of white subjects. However, both racial groups evidenced a comparable quantity of glands.

Pochi and Strauss[70] measured sebum production on the foreheads of 30 black male subjects compared with 373 white male subjects. Mean sebum production was slightly but not statistically increased in the black male subjects compared with the white male subjects. The opposite results were obtained when they evaluated 37 black and 209 white females. Sebum levels were significantly lower in these black woman compared with age-matched white women. Because there was an unequal number of subjects in the racial groups, the data were analyzed differently to try to control for this variable. By using the total male and female white population as a control group, the researchers evaluated whether the sebum production values for each of the 67 black subjects were within the 95% confidence limits for the control group (ie, statistically equivalent). Only 4 of the 67 black subjects had sebum production values that exceeded the range of 2 standard deviations from the mean of the white patients.

In a more recent study, Abedeen et al[71] measured the rate of sebum production, using both sebutape and a sebumeter, in 3 different racial groups: 20 white, 20 black, and 20 Asian subjects. They concluded that there was no statistical difference in sebum excretion rate among these racial groups.

Abe et al[36] assessed the relation between sebum production and skin pigmentation in 101 Japanese women. They demonstrated a positive correlation between the amount of skin surface lipids and darker pigmentation.

Hair follicles

The hair of individuals from the Negroid race has been deemed a more distinctive phenotypic characteristic than skin color.[71] Four types of hair are recognized: straight, wavy, helical, and spiral—the last being the type of hair present in the vast majority of blacks.[72] The coils constituting the spiral form show reduced diameter from the scalp outward. A cross-sectional evaluation of hair from 4 different racial groups reveals that black subjects have the longest major axis, ultimately giving hair a flattened elliptical shape.[73] The follicles of the scalp and the hair itself are curved.[74]

The hair of Asian subjects as demonstrated in the Chinese is the most nearly round or circular, and with the largest cross-sectional area. In contrast, the hair of Western European subjects was found to have the smallest cross-sectional area.[73] Similar findings were demonstrated by Steggerda and Seibert.[75]

In another study comparing the hair of different racial and ethnic groups, white subjects of Dutch ancestry had the smallest hair diameter. Black subjects had the most elliptical hairs and Mayan Indians had the most circular or round hairs. Native Americans (Hopi, Navajo, and Zuni) had large round hairs. There was no discernible difference in the thickness of the cuticle, shape and size of scale, and cortical cells between the hair of white and black subjects.[76]

Other racial differences in hair follicles have been observed. Montagna and Carlisle[26] did an ultrastruc-

tural study of hair in black subjects compared with white subjects and found fewer elastic fibers anchoring the hair follicles to the dermis in the black subjects. This may have implications for the cause of certain types of alopecia, such as traction alopecia, and follicular degeneration syndrome. Also of interest, melanosomes were noted in both the outer root sheath and in the bulb of vellus hairs in black subjects but not white subjects.[26] Black hair was found to be more heavily pigmented compared with white hair.[76] Swift,[77] using electron microscopy, measured the size of isolated melanin granules and found that those from the hair of black subjects were larger than those from the hair of fair-skinned and Asian (Chinese) subjects.[77]

Sperling[78] recently published a retrospective analysis of patients who had undergone a biopsy of the scalp to assess hair density in 22 African Americans compared with 12 white subjects. He found that the total hair density (ie, number of follicles found in a 4-mm punch biopsy specimen) and the total number of terminal hair follicles were significantly lower in African Americans compared with white subjects ($P < .001$).

Khumalo et al[79] examined the ultrastructure of hair from white, African, and Asian subjects. He determined that African hair had a tendency to form knots and longitudinal fissures and splits along the hair shaft compared with the hair of white or Asian subjects. The majority of the tips of African hair had fractured ends representing breakage, whereas the majority of hair from the white and Asian subjects were shed.

Despite these differences, the hair of members from all races share common structural elements. For instance, there is no difference in the type of keratin between black and white individuals.[80] However, Menkart et al[76] found differences in the amino acid composition between the hair of white subjects compared with black subjects. There was a deficiency of serine and threonine and an excess of tyrosine, phenylalanine, and ammonia in the hair of black subjects.[76] Gold and Schriver,[81] on the other hand, found no differences in the amino acid composition of hair from different races.

Racial differences in the dermis

As noted earlier, a study of caliper-assessed skin thickness—a measurement encompassing both the epidermis and dermis—found no difference between black and white females.[45] Despite this gross similarity, there appear to be differences on the cellular level between the dermis of black and white individuals.

An analysis of the dermis of black and white skin by Montagna and Carlisle[26] showed differences in certain cell types. Fibroblasts in black female facial skin were larger and occurred in greater quantity compared with those in white female facial skin. The fibroblasts in black females were either binucleated or multinucleated. The actual collagen fiber bundles in black individuals were smaller, more closely stacked, and ran more parallel to the epidermis. In addition, many collagen fibrils and glycoprotein fragments were noted in the dermal interstices and throughout the dermis. White individuals showed greater interindividual variability in fibroblast numbers. The collagen fiber bundles in the white females were larger; occasional fiber fragments were noted.

Fibroblast hyperreactivity is thought to be due to an interaction between various cells (eg, mast cells), cytokines, and fibroblasts. This, combined with a decrease in the activity of the collagenase enzyme, probably results in keloid formation.[20] There were no significant differences in the number or size of mast cells between the 2 racial groups as demonstrated by Montagna and Carlisle.[26] However, in the black individuals macrophages in the papillary dermis were larger and more numerous.[20]

As will be noted in a later section, a number of these biologic properties of fibroblasts and their interactions with other cells and growth factors could account for the increased incidence of keloid formation seen in black individuals.

FUNCTION OF PIGMENTED SKIN

Research on racial and ethnic differentials in various aspects of skin function is also controversial. Small patient populations and different test methodologies make it difficult to arrive at definitive conclusions.

Percutaneous absorption

The barrier properties of the skin are predicated on the integrity of the stratum corneum.[82] The fact that the stratum corneum is metabolically inactive suggests passive diffusion as the mechanism of percutaneous absorption. Substances then penetrate the remainder of the epidermis, dermis, and enter the circulation via the capillary system. Transappendage penetration through the hair follicle wall and sebaceous gland may also play a small initial role.[83,84] The variation in skin permeability depends on a number of factors, including the thickness of the intact stratum corneum and to a lesser extent the density of the cutaneous appendages.[83-85]

Studies on the interracial and interethnic variation in percutaneous absorption have produced conflicting results. Wickrema-Sinha et al[86] studied the skin penetration of the topical steroid, diflorasone diacetate, in 3 white and 3 black subjects by measuring the excretion of a radiolabeled drug. No differ-

ences were found between the 2 racial groups. Guy et al[87] investigated racial differences in percutaneous absorption of methyl-nicotinate by measuring the vasodilative response to this chemical in 6 young white subjects and 6 age-matched black subjects. Methyl-nicotinate was applied to the arm of each subject. There was no difference in absorption, as measured by peak response and area under the response-time curve. There was also no observed difference in cutaneous blood flow, as measured by laser Doppler velocimetry (LDV). However, when the method of photoplethysmography was used to assess cutaneous blood flow, a decreased maximum response was seen in the black subjects. Wedig and Maibach[88] evaluated percutaneous absorption of a radiolabeled preservative (dipyrithione) in 4 white subjects compared with 4 black subjects through measuring urinary excretion of this chemical over a 7-day period. Lower urinary excretion in black subjects suggested 34% lower absorption of the chemical. Hence, the researchers concluded that white skin was more permeable to certain chemicals than black skin. Possible experimental errors in the study were incomplete urine collections and differences in excretion, distribution, or metabolism of absorbed material between the groups.

Stoughton[89] measured the percutaneous absorption of the steroid, fluocinolone acetonide, through normal-appearing white and black cadaveric skin. (The skin was amputated because of gangrene.) He found greater absorption in the white skin compared with the black skin. Berardesca and Maibach[90] studied the racial differences in vasodilative response to nicotinate in 10 black subjects and 9 white subjects. To facilitate absorption through the stratum corneum, they altered this layer by either stripping cell layers or by removing the lipids. They then measured the subjects' pharmacodynamic response to the nicotinate agents with LDV. Lower measurements were recorded in black subjects compared with white subjects. The researchers concluded that black subjects evidence either reduced percutaneous absorption or reduced vasodilative reaction to nicotinate.

The same researchers used the same methodology to assess ethnic differences between white Americans and Hispanic Americans.[91] No difference in percutaneous absorption or vasodilation was observed between the groups.

Gean et al[92] compared percutaneous absorption of methylnicotinate in 5 black subjects, 5 Asian subjects of Chinese ancestry, and 5 white subjects. Methods included the use of LDV and researcher observation of erythema. The diameter of the visually perceptible erythema, the laser Doppler output as a function of time, and the maximum LDV response were similar among the 3 groups. The laser Doppler output as a function of time was greater in black and Asian skin compared with white skin.

Skin irritants

Assessment of the racial differential in skin irritability is another controversial subject. There are multiple scientific studies with conflicting data concerning susceptibility of skin of color to skin irritants. Earlier studies relied on the investigators' observation of erythema as the primary endpoint in determining irritability. This subjective evaluation could vary, depending on the observers' clinical experience with skin of color and their ability to discriminate erythema within the context of pigmented skin. Furthermore, interindividual variability in susceptibility to irritants is an omnipresent confounding variable in all studies evaluating racial differentials in skin irritant reactions. Human subjects appear to express great heterogeneity in skin reactivity, regardless of race or ethnic group.[93]

Studies on the racial and ethnic differences in skin irritant reactions began as early as 1919 when Marshall et al[94] examined racial variations in skin irritation induced by mustard gas (dichloroethylsulphide). Observation of erythema was the method used to assess irritability. Fewer black men evidenced erythematous reactions to various concentrations of dichloroethylsulfide compared with the white men in the study.[94] Two subsequent studies by Schwartz et al[95] and Shelly[96] also produced findings suggesting that black subjects were less likely to experience cutaneous irritation as induced by either chemicals or UV light. In both studies, skin irritability was evaluated by the observation of erythema. Weigand and Mershon[97] and Weigand et al[39] assessed the racial differential in skin irritation to o-chlorobenzylidenemalononitrite and dinitrochlorobenzene, respectively. Again, perceptible erythema was the endpoint for evaluation of skin irritation. This reaction was less evident in black subjects compared with white subjects. Frosch and Kligman[98] used the chamber-scarification test for assessing the irritablenes of topically applied substances. Through subjective assessment of erythema, they concluded that black subjects were less susceptible to irritants than white subjects. Furthermore, those white subjects with the lightest complexions were most susceptible. Other researchers also concluded that black subjects experience less skin irritation compared with white subjects.[99,100]

Hence, many researchers concluded, based on perceptible morphologic skin changes, that black subjects were less susceptible to developing chemical irritation than white subjects. The accuracy of this subjective evaluation, especially within the context

of pigmented skin, is questionable. More recent studies have used more objective measurements for assessing skin irritation, such as measurements of transepidermal water loss (TEWL) and other objective measurements of irritant-induced breeches in the stratum corneum. The advent of these more objective methodologies, coupled with a better understanding of the difficulty of perceiving erythema in highly pigmented skin, have prompted a number of investigators to question the results of these earlier studies. Though these studies represent progress in the assessment of skin of color function, a number of the studies are not without methodologic flaws.

In the 1980s and early 1990s, several groups of researchers attempted to characterize racial and ethnic differences in irritant reactions to topically applied chemicals by using several biologic parameters.[101-108] Most groups looked at irritation provoked by the topical application and occlusion of sodium lauryl sulfate (SLS). Two concentrations (0.5% and 2.0%) of this irritant chemical were applied to the skin of black, white, and Hispanic subjects for a 24-hour period via Finn chambers. Three different skin models were used: untreated skin; skin pretreated with ethyl acetate to remove the stratum corneum lipids; and skin preoccluded with plastic for 30 minutes to increase the stratum corneum water content and enhance penetration of the irritant. Thus, two thirds of these studies were designed to measure irritation in skin that has been altered in some respect. One can only question the clinical relevance of these data, which are frequently quoted in the literature. The irritant effect of SLS was assessed through its effect on stratum corneum integrity. Stratum corneum integrity in turn was measured through a number of objective methods: TEWL with evaporimetry; water content of the stratum corneum through a measurement of capacitance; and microcirculation via LDV. Berardesca and colleagues assessed racial and ethnic differential in stratum corneum integrity through a number of studies.[101,102,104,105] Based on their findings, they arrived at a number of conclusions frequently quoted in the literature: (1) black subjects' skin displays a stronger skin irritant reaction than white subjects'; (2) the skin of black subjects is more sensitive to irritants than the skin of white subjects; (3) black subjects display less erythema, less blood vessel reactivity, and cutaneous blood flow to irritants than white or Hispanic subjects; (4) Hispanic subjects show a stronger irritant reaction compared with white subjects; their irritant reaction is similar to that of black subjects; (5) Hispanic subjects have stronger irritant reactions when injured with concentrated chemical; and (6) Hispanic and white subjects have similar erythematous reactions.

A critical appraisal of these studies undermines these conclusions. For the untreated skin model, there was no significant difference among black, white, and Hispanic subjects for any measurement of stratum corneum integrity. Baseline values for water content, TEWL, and microcirculation were similar among all groups. Predictably, after irritation with the 2% SLS, higher TEWL, water content, and LDV values were recorded in the same range for all groups. Significantly higher TEWL values were seen only in black subjects with preoccluded skin exposed to 0.5% SLS. Thus, the assertion that Hispanic and black subjects have a stronger irritant reaction to chemicals is based on nonstatistical data. The conclusion that black subjects have a more sensitive response to irritants compared with white subjects is supported only by the data in the preoccluded skin model mentioned above.

The assertion of stronger irritant reactions in Hispanic subjects compared with white subjects is based on increased TEWL values seen with the delipidized skin model, as assessed by linear regression analysis. The claims of increased sensitivity would be better substantiated if a significant increase in TEWL values were seen in the untreated skin model exposed to the lower SLS concentration.

The higher concentration of SLS caused a significant increase in cutaneous blood flow in all patient groups. In the untreated skin model, there were no significant differences in cutaneous blood flow between white, Hispanic, and black subjects. In the untreated skin model exposed to the lower SLS concentration, cutaneous blood flow was lower in black subjects compared with white subjects.

Goh and Chia[106] evaluated skin irritation to 2% SLS also through transepidermal water loss (termed skin water vapor loss or SVL in this study) in 15 fair-skinned Chinese, 12 Malaysians with darker skin, and 11 Indian subjects with very dark skin. No significant differences in the mean baseline SVL values among the 3 different groups were found. After exposure to SLS, no significant difference in mean SVL values was found between any of the pair-group comparisons: Chinese and Malaysians, Chinese and Indians, or Malaysians and Indians. The mean irritation indices associated with the 3 different levels of pigmentation also were not significantly different.

Several other groups looked at susceptibility to contact irritants in ethnic and racial groups by assessing stratum corneum barrier function. They hypothesized that a compromised barrier results in increased susceptibility to irritants.

Kompaore et al[107] compared the barrier function of the stratum corneum in 3 racial groups: 7 African

Table V. Therapeutic implications of key biologic differences between races[20-28,30,33,37,71,77,109-116]

Biologic factor	Therapeutic implications
Epidermis	
Increased melanin content, melanosomal dispersion in people with skin of color	Lower rates of skin cancer in people of color Less pronounced photoaging Pigmentation disorders due to both biologic predispositions and cultural practices (eg, use of lightening agents)
Dermis	
Multinucleated and larger fibroblasts in black persons compared with white persons	Greater incidence of keloid formation in black persons compared with white persons
Hair	
Curved hair follicle/spiral hair type in black persons compared with white persons	Pseudofolliculitis in black persons who shave
Fewer elastic fibers anchoring hair follicles to dermis in black persons compared with white persons	Use of hair products that may lead to hair and scalp disorders in black persons Increased incidence of alopecias in black women

black subjects, 8 white European subjects, and 6 Asian subjects, using TEWL and LDV measurements. In this trial, subjects were not exposed to irritants or other chemicals that would disrupt the stratum corneum. Baseline TEWL measurements were significantly higher in Asian subjects and black subjects compared with white subjects. No baseline differences were seen between black and Asian subjects. On the basis of these results, the authors concluded that black and Asian subjects have a more compromised barrier function compared with white subjects and therefore would be more susceptible to irritants.

Wilson et al[104] in an in vitro study used biopsy specimens from cadaveric skin to assess TEWL. Biopsy specimens were obtained from the thighs of 12 white and 10 black subjects of African Caribbean origin. When the skin samples were exposed to increased temperatures, TEWL increased exponentially in the samples from both racial groups; however, higher absolute values were observed in samples from the black subjects. DeLuca et al[108] on the other hand studied baseline TEWL in 29 African black subjects compared with white European subjects and found no difference in this parameter. Finally, Pinnagoda et al[109] found no apparent difference in baseline TEWL among racial groups.

Reed et al[40] also found no significant differences in baseline TEWL values among subjects with skin type V/VI (African American, Filipino, and Hispanic subjects) and those with type II/III skin (Asian and white subjects). However, after subjects were exposed to a cutaneous irritant, those subjects with skin types V and VI demonstrated superior barrier integrity and permeability barrier recovery.

CUTANEOUS DISEASE IMPLICATIONS

There are many reviews of common dermatoses that either present differently or occur more often in people with skin of color compared with people with lighter skin. We will not attempt an exhaustive review of such issues in this article. Rather, we will address some salient disease implications on the basis of the few distinctive differences in dermatologic physiology seen in people with skin of color that is supported by the literature (Table V). In addition, we will also review cultural practices in these racial and ethnic groups that impact on dermatologic disorders. Needless to say, many of these cultural practices are prompted by certain hallmark features of the skin and hair of people with skin of color.

Racial differences in skin cancer incidence

Though skin cancer is the most common malignancy in the United States, the incidence among people with skin of color is relatively low.[110,111] In white subjects, sun exposure—either chronic exposure or episodic high-intensity exposure—is thought to be a major etiologic factor in the development of basal call carcinoma (the most common cutaneous malignancy in fair-skinned subjects), squamous cell carcinoma (the most common cutaneous malignancy in black subjects), and melanoma.[110,111] The melanin content and melanosomal dispersion pattern in people with phototypes V and VI is thought to be responsible for providing protection from the carcinogenic effects of UV radiation and hence, a lower incidence of skin cancer.[110,111]

The development of melanoma is inversely correlated with the degree of skin pigmentation on skin that is exposed to the sun.[112] In white subjects, it has been found that there is an increased susceptibility to melanoma compared with Hispanic, Asian, and black subjects. Furthermore, melanoma among Hispanic, Asian, and black subjects differs in incidence, site distribution, stage at diagnosis, and histologic type from melanoma in white subjects.[113] The melanoma data

from the California Cancer Registry for a 6-year period revealed average, annual, age-adjusted incidence rates per 100,000 population for white men of 17.2 and for white women 11.3; for Hispanic men 2.8 and for Hispanic women 3.0; for Asian men 0.9 and for Asian women 0.8 and for black men 1.0 and for black women 0.7. Hence, cutaneous melanoma incidence in Hispanic subjects has been reported to be among white, black, and Asian subjects.[113]

In Hispanic, Asian, and black subjects, melanoma arises most often on non–sun-exposed sites with less pigment such as the palms, soles, and subungual areas compared with white subjects. In white subjects, melanoma occurs primarily on sun-exposed or intermittently sun-exposed skin. When individuals with skin of color have a melanoma develop, they are more likely to have acral lentiginosus melanoma develop compared with white subjects. However, Clark et al[114] suggested that the rate of plantar melanoma per 100,000 cases per year is identical in white and dark-skinned individuals, although plantar melanoma accounts for only 5% of all melanomas in white subjects but 50% to 73% of melanomas in dark-skinned individuals.

The outcome of melanoma is not as favorable in individuals with skin of color compared with white skin. The California Cancer Registry, for example, reported that melanoma was diagnosed after it had metastasized to a remote site for 15% Hispanic men, 13% Asian men, and 12% black men compared with 6% white men. Clinicians should not be complacent about the risk of skin cancer in people with skin of color. First, early detection and surgical intervention offers the best chance of long-term survival. Second, it has been found that a single exposure to low-dose UV radiation in black subjects caused derangements in immune function.[115] Because immune surveillance is the body's main protection against the development of malignancies, it has been suggested that people with dark skin may still need to take the same precautions against sun exposure as recommended for individuals with lighter skin.[116]

Racial differential in the development of photoaging

Dermatologists who have experience treating an African American patient population can attest to the albeit anecdotal observation that people with dark skin appear to "age better" than those with lighter skin. Individuals with black skin are thought to evidence firmer and smoother skin than individuals with lighter skin of the same age.[117] Again, the melanin content and melanosomal dispersion pattern is thought to confer protection from the accelerated aging induced by exposure to UV radiation.[33] Kaidbey et al[33] demonstrated that black epidermis on average provided an SPF of 13.4, which would provide the scientific basis for the "better aging" observation. Photoaging among black subjects does occur, but it is more common in individuals with relatively fair skin.[117] Photoaging also tends to occur at a later age in black subjects than in white subjects.[118] Predictably, inconsistent pigmentation (eg, hypopigmentation or hyperpigmentation) is a sign of photoaging in people with skin of color.[117] Many black individuals report overall darker facial skin than non–sun-exposed skin.

These findings are consistent with the study mentioned earlier on Asian women with an average SPT of IV. Signs of photoaging (eg, epidermal atrophy, cell atypia and poor polarity, and disorderly differentiation) have been observed in Asian subjects' skin.[32] In short, deeply or darkly pigmented skin can still experience photodamage, as evidenced by pigmentation disorders and other signs of epidermal and dermal damage (eg, collagen damage).[32,117]

Pigmentary disorders

In an analysis of 2000 black patients who had seen dermatologists, Halder et al[118] found that the third most common diagnosis involved pigmentation disorders other than vitiligo; most patients with pigmentation disorders presented with postinflammatory hyperpigmentation. Postinflammatory hyperpigmentation can be considered the default pathophysiologic response to cutaneous injury in people with skin of color. This response is thought to be predicated on the labile response of melanocytes to irritation or inflammation.[119] For instance, postinflammatory hyperpigmentation is a common sequelae of acne vulgaris and the acne hyperpigmented mocule (AHM) is often the chief complaint in acne patients with skin of color as opposed to the acne per se.[113] These changes should not be construed as trivial cosmetic changes; they can have significant psychological impact in subjects who experience them. (A thorough review of acne vulgaris and the acne hyperpigmented macule is found in another article in this supplement.)

Melasma is another pigmentary disorder that occurs frequently in people with skin of color. This disorder is reported to be more prevalent in black, Hispanic, and Asian subjects.[119] Hormonal factors, UV radiation, and the lability of melanocytes are all thought to be contributing etiologic factors.[116,119]

As will be discussed shortly, the prevalence of pigmentation disorders in black subjects has resulted in their use of many topical products that are not always prescribed or monitored by physicians. These cultural practices can result in further dermatologic disorders.

Hair and scalp disorders in African Americans

The intrinsic properties of hair in black individuals impact on hair and scalp disorders. Many of these disorders appear to be the result of an interaction between some unique biologic features and certain cultural practices, a topic that will be discussed in more depth shortly. For instance, the curvature of the hair follicle and the configuration of the actual hair of many blacks are the biologic attributes that appear to impact on the development of pseudofolliculitis barbae in black men and women. (A fuller discussion of this disorder can be found in an article in this supplement.) Histologic analysis demonstrates that the curved hair forms an arc in the dermis and is almost parallel to the skin surface. After these facial hairs are cut, a few millimeters of growth will cause them to puncture the skin, resulting in extrafollicular penetration. A neutrophil-mediated inflammatory response then occurs.[120] Because of these detrimental consequences of shaving, many black men grow beards. This is a cultural practice that can redress a potential hair follicle disorder. Unfortunately, this is not an attractive option for black women with this condition.

Alopecia is a disease that commonly occurs in African-American women and some Hispanic women. Traction alopecia, occurring primarily at the frontal and temporal hairline, is well recognized and is related to tension on the hair. As previously noted, fewer elastic fibers anchor hair follicles to the dermis in black persons compared with white persons.[26] Again, this biologic feature, coupled with cultural practices of tight braids, ponytails, and the addition of weighty artificial hair attached to hair weaves and braid extensions, may impact on the development of traction alopecia. Furthermore, the demonstration of knots in the hair of black persons, along with the marked breakage of hair due to normal grooming practices (eg, combing and braiding), underscores the importance of trauma in the development of traction alopecia.

Another common and dramatic form of alopecia occurs in the vertex and midscalp of African-American women. This presents clinically with a decrease or absence of follicular openings and a shiny appearance of the scalp. This form of alopecia has been identified under several labels, including hot-comb alopecia,[121] follicular degeneration syndrome,[122] centrifugal scarring alopecia,[123] and hot-comb alopecia/follicular degeneration syndrome in African-American women is traction alopecia.[124] Despite provocative explanations for this form of alopecia by leaders in pathology and dermatology, the cause of this alopecia remains an enigma and a quandary unresolved. Clinically, traction alopecia and what has been called "follicular degeneration syndrome" differ in the location on the scalp as well as pattern and appearance.

In attempting to more thoroughly define and identify this entity, it may be useful to review both the known biologic differences between the hair of white and black persons as well as the known hair care practices. These would include (1) curvature of the hair follicles; (2) flattened, elliptically shaped hair prone to knotting and fracturing in black persons; (3) fewer elastic anchoring fibers in black hair; (4) melanosomes in the outer root sheath of the hair; and (5) large melanin granules found in the hair.[72,73,78,120] Documented cultural practices that can impact on the development of this disorder include hot-comb and pomade use coupled with braiding during childhood and adolescence, the use of chemical relaxers 6 to 12 times per year coupled with blow drying, the use of hot iron curling, hair rollers, and hair dyes during adulthood, and/or the use of various hair styles (eg, ponytails, braids with extensions, and microbraids.) Questions to direct future areas of investigation elucidating on what has been called "follicular degeneration syndrome" could include the following: (1) Does the genetically determined curvature of the hair and hair follicles in some way predispose some African-American women to this type of alopecia or does the curvature predispose this group to use hair products and processes that may be responsible for the alopecia? (2) Are elastic fibers further compromised by these hair products and processes? (3) Does melanin in the outer root sheath of the hair produce damaging free radicals which then destroy the stem cells located in the bulge region of the outer root sheath? (4) Do thermal/steam damage from hot combing during childhood or irritant contact dermatitis from alkali-based chemical relaxers or microscopic burns during adulthood destroy the bulge region of the outer root sheaths over time? What is undoubtedly required to solve this quandary is serial scalp biopsies from large numbers of African-American women over an extended period. Concurrent documentation and analysis of hair grooming practices (hot-comb use, chemical relaxer use, use of hair braids, extensions, weaves, hair rollers) and an extensive family history to uncover familial predisposition must be obtained as well.

Keloidal scar formation

Although keloidal scarring occurs in all races, it is thought to occur much more frequently in black persons,[125-127] ranging from 3 to 18 times more often in black persons compared with white persons. Although the relative frequency of incidence in other

racial and ethnic group is unclear, a study of keloidal scarring in Malaysians, Indians, and Chinese suggests keloidal scars were more common in the Chinese population. Keloidal scarring develops through a complex and poorly understood interaction between fibroblasts, various cutaneous cells, and cytokines that facilitates the production of excessive collagen and inhibits the degradation of the extracellular matrix components.[128]

As noted previously, research suggests that fibroblasts are larger and binucleated or multinucleated in black persons.[26] It is of interest, when thinking about keloidal formation in black skin, that the fibroblasts in these individuals are large and numerous with nuclear machinery possibly awaiting stimulus to begin the overproduction of collagen. That stimulus, thought by many to be transforming growth factor beta 1 (TGF-β1) also acts to retard degradation of collagen and other extracellular matrix proteins.[128] A thorough review of keloidal scars with an emphasis on therapeutics is found in another article in this supplement.

Contact dermatitis

The literature is equivocal on the incidence of contact dermatitis in people with black skin. Research suggests both an increased and decreased susceptibility of black skin to contact allergic dermatitis. As noted previously, some of the difficulty is related to the perception of erythema, an endpoint in determining contact dermatitis through patch testing. Sherertz and Schwartz[129] have emphasized the fact that patch test interpretation of black skin responsivity is more difficult given the challenge of detecting erythema. A review of the patch-testing data by various groups reveals the following: Rosenberg and Kanof[130] determined that black skin was less readily sensitized to the allergens dinitrochlorobenzene and paranitrosodimethylaniline. Epstein and Kligman[131] confirmed these results. However, in another study Kligman and Epstein[132] determined similar sensitization to p-phenylenediamine, monobenzyl ether of hydroquinone, and nickel sulfate between fair-skinned white persons and dark-skinned black persons. In this study, the sensitization rates for the allergens that were termed weak antigens (penicillin A and neomycin sulfate) were statistically higher in white subjects compared with black subjects. Kligman and colleagues concluded that black skin is less responsive to exogenous insults. This lower responsivity is not due to a compromised immune reaction but the inability of black skin to demonstrate ostensible inflammation-based changes by which allergic states are recognized. Kligman and Epstein[132] reiterated that fair-skinned persons have skin that is more reactive to allergens. Menne and Maibach[133] also presented experimental evidence that black persons are less susceptible to allergens and irritants than white persons.

Other data do not vindicate the above observed racial differentials. Leyden and Kligman[134] studying allergic contact reactions to multiple allergens found no significant difference in reaction rates between black and white persons. However, a gender differential was reported among black persons, with females experiencing slightly less sensitization than males. Fisher[135] reported a similar incidence of allergic contact dermatitis in black and white persons.

The North American Contact Dermatitis Group reported information on contact dermatitis in approximately 10,000 patients (10.5% of whom identified themselves as black) who received patch tests between 1992 and 1998. (See article by DeLeo et al in this supplement.) The percentage of patients with positive patch tests was similar between black and white patients. The sites of the dermatitis were likewise similar between the races; the hands and face were the most commonly affected areas. The most common allergen was nickel, and the response rates to this allergen was comparable between white and black persons. Of interest are findings that there was a trend among white persons for higher rates of sensitization to formaldehyde and formaldehyde-related preservatives compared with black persons. This may be explained by certain racial differences in cultural practices. White persons appear to use cream-based products more often than black persons and therefore have more exposure to cream-based products containing these preservatives. Black persons use more ointment-based products that do not contain these preservatives.

Racial or ethnic cultural practices may provide an explanation rather than genetic or biologic factors in explaining another observed racial differential in chemical sensitivity. In the article on contact dermatitis in this supplement, the black population was shown to have a higher incidence of contact dermatitis to paraphenylenediamine, a hair dye frequently used by this racial group. Dickel[136] found similar results but in addition reported a gender difference in sensitivity, with black men experiencing this sensitivity more often than black women. While black persons probably do not use more hair dye than white persons, black persons may use darker shades of hair dyes and these darker products contain higher levels of paraphenylenediamine. This could lead to higher sensitization rates among users. Finally, it may also be possible that higher sensitivity to paraphenylenediamine in black persons represents cross-sensitization to other chemically related substances such as thiazide diuretics and oral hypoglycemic agents, which are

more frequently used in black persons compared with white persons. However, as pointed out by DeLeo et al in this supplement, one would expect to find higher levels of sensitization to a similarly related allergen, benzocaine, which was not seen in that study.

In summary, a synthesis of the available data related to contact dermatitis indicates equal ability of white and black persons to mount an immunologic response to antigens. Racial differences in the rates of positive patch tests to specific antigens are most likely related to differences in baseline exposures.

Atopic dermatitis

A recent prospective, 12-month, observational study of 61 fair-skinned white, 61 Chinese, and 59 Vietnamese infants, all living in Australia, found that atopic dermatitis developed in 21% of the fair-skinned infants, 44% of the Chinese infants, and 17% of the Vietnamese infants, underscoring the importance of the role of systemic allergens.[137] Because the fair-skinned infants and Chinese infants were of similar socioeconomic background, the authors concluded that genetics probably played a predominant role in the difference in incidence. Because the Vietnamese infants were of a lower socioeconomic background but of the same racial group as the Chinese infants, the authors suggested that environmental factors contributed more than genetic factors in the difference in incidence between these 2 Asian groups. A major methodologic flaw in this study is lack of control or knowledge about the infants' diet other than access to breast feeding.[137] Because dietary antigens are an important cause of allergic disease in infants, definitive conclusions from this study are probably not warranted. However, the study does underscore the importance of both genetic and environmental contributions to dermatologic diseases, although unfortunately, the role of allergens was not adequately investigated. Environmental influences in the form of cultural practices will be the final area of discussion of this review.

IMPACT OF CULTURAL PRACTICES ON DERMATOLOGIC DISEASE AND CONVERSELY, THE IMPACT OF DERMATOLOGIC DISEASE ON CULTURAL PRACTICES

The use of facial lightening agents

As noted, cultural practices, including the use of commercially available, nonprescribed pharmacologic and other interventions by individuals from various ethnic and racial groups, have an impact on the development of skin and hair disorders.

Beauty supply stores in major metropolitan areas that service African, Afro-Caribbean, African American, and Asian communities have reported a marked increase in the sales of skin-lightening agents. Many of these products contain potent pharmaceuticals that should be prescribed and monitored by dermatologists. For instance, products known as facial fading creams, imported from Saudi Arabia, Europe, and Africa, contain potent topical corticosteroids such as clobetasol propionate. These are over-the-counter products sold under the names of Dermovate, Betnovate, Topsone, and Movate. As expected, the chronic use of steroids has the potential to produce skin atrophy, hypopigmentation, erythema, and telangiectasia. The lay press has taken a role in educating the public about the dangers of using these products; however, dermatologists must also be aware of such product use and counsel their patients about the dangers.

Products containing hydroquinone in concentrations of 2% to 4% are the most frequently used bleaching agents. Concentrations greater than 4% may be obtained from foreign countries as well as from distributors in the United States. Especially with the use of hydroquinone in higher concentrations, there is the risk of exogenous ochronosis. Although exogenous ochronosis is an uncommon problem in the United States, with the influx of immigrants, we will probably see more of this disorder. Finally, contact dermatitis to hydroquinone has been well described.

African-American hairstyles and hair preparations

As noted previously, hairstyles popular in the African-American community include braids, braids with extensions, microbraids, twists, cornrows, and tightly gathered ponytails. These hairstyles, in combination with the unique hair follicle characteristics mentioned previously, can lead to temporary or permanent traction alopecia. At the Skin of Color Center, we frequently observe receding hairlines in African and African-American women due to hairstyle-induced traction. Prolonged and repeated use of these hairstyles can lead to permanent alopecia.

Hair pomades and conditioners are also popular with members of the African, Afro-Caribbean, and African-American communities. The hair pomades, containing various mixtures of petrolatum, lanolin, and vegetable, mineral, or animal oils,[138] serve to coat and improve the texture and manageability of the hair, making the hair shafts less brittle. These pomades also are applied to the scalp to treat the itching and scaling associated with seborrheic dermatitis. However, when these agents are spread to the forehead and temples, papular and comedonal acne develops.

The use of other hair and skin products

Henna is a natural dye derived from the dry leaves of a shrub, *Lawsonia alba*, indigenous to Africa and the Middle East.[139] The soaked leaves yield colors ranging from orange to reddish brown to black. Decoration with henna usually centers around rituals such as circumcisions and celebrations or holidays such as weddings or Ramadan. Henna has several applications for the skin and hair. It is used to provide reddish highlights in the hair of women but also of men from many cultures. In some cultures, it is used for decorative purposes on palms, soles, tips of the fingers and toes, and on the nails of women and teenagers. Of interest, *L alba* is an oxidizing agent that can produce hemolysis of red blood cells. The hemolysis can occur in glucose-6-phosphate dehydrogenase normal cells and especially in G6PD-deficient cells.[140] There are cases of hemolysis and hyperbilirubinemia in newborns in whom henna was used to decorate their entire bodies. The color of henna is enhanced when combined with paraphenylenediamine (PPD). There have been reports from the Sudan of adults and children experiencing angioneurotic edema, shock, and renal failure from this mixture.[141]

Contact dermatitis due to henna has been reported at the sites of application of the henna.[142,143] Patch testing to *Lawsonia* have revealed both positive and negative results, suggesting that other allergens in addition to *Lawsonia* in henna may be responsible.[144]

Other cultural practices: Coining, cupping, and moxibustion

Many Asians engage in traditional healing practices such as coining, cupping, and moxibustion. Coining is used to treat a variety of illnesses, including febrile illnesses in children, headaches, myalgia, and malaise in adults. A warm oil or Tiger Balm (a topical analgesic agent) is rubbed on the skin and a coin or other instrument is firmly rubbed along the back or chest. This produces linear petechiae and ecchymosis. Though the process can induce pain in children, it should not be confused with child abuse. These lesions resolve after several days. Cupping, involving the application of a bell-like suction device to the skin, is another healing practice common in Asian cultures. The cupping device produces circular ecchymoses on the neck, chest, back, and arms. This dermatologic disorder also resolves quickly. The practice is designed to "release wind" from the body and therefore restore balance and health. A traditional healing practice in Asian and African societies is the production of circular burns sometimes termed moxibustion. The burns are produced through contact with a heated stick or embers from burning incense or herbs. Common areas of involvement include the umbilicus, chest, wrist, ankles, and scalp. The presence of burn scars may indicate that the child has had a serious febrile illness. Reports from the Northwestern United States reveal that this treatment has been used for chronic eczema and ear infections. Again, these practices do not reflect child abuse. In many African societies, scarification is widely practiced. They may be used for decorative purposes on the faces or for medicinal purposes.

CONCLUSION

People with skin of color constitute a wide range of racial and ethnic groups, including African, African American, Asian, Native American, Hispanic, and certain groups of fair-skinned persons (eg, Arabs, Indians, and Pakistanis). These people have been categorized by the Fitzgerald SPT system as having skin types IV through VI.

There is a paucity of well-controlled studies on people with skin of color. Most studies evaluate differences between fair-skinned persons of European ancestry and African/African-American persons. Few definitive conclusions about racial and ethnic differences in skin structure, physiology, and dermatologic disorders can be made. The literature does support a racial/ethnic differential in epidermal melanin content and melanosome dispersion, and in black persons compared with fair-skinned persons, and differences in hair structure and fibroblast structure.

Any observed racial or ethnic differences in dermatologic disorders may not be solely predicated on genetics, but also the unique cultural practices of the groups in question. Obviously further research is needed on people with skin of color who will shortly constitute the majority of people in this world.

REFERENCES

1. As city immigration thrives, diversity bounds. The New York Times; November 8, 1999.
2. Distribution of haplotypes from a chromosome 21 region distinguishes multiple prehistoric human migrations. Proc Natl Acad Sci USA 1999;96:3796-800.
3. Diamond J. The third chimpanzee. New York (NY): Harper Perennial; 1992.
4. Coon CS. The origin of races. New York (NY): Alfred A. Knopf; 1962.
5. Fitzpatrick TB. The validity and practicality of sun reactive skin type I through VI. Arch Dermatol 1988;124:869-71.
6. Pathak MA, Nghiem P, Fitzpatrick TB. Acute and chronic effects of the sun. In: Freedberg IM, Eisen AZ, Wolff K, Austen KF, Goldsmith LA, Katz SI, et al, editors. Fitzpatrick's dermatology in general medicine, vol 1. New York (NY): McGraw-Hill; 1999. p. 1598-608.
7. Pathak MA, Fitapatrick TB. Preventive treatment of sunburn, dermatoheliosis and skin cancer with sun protective agents. In: Freedberg IM, Eisen AZ, Wolff K, Austen KF, Goldsmith LA, et

al, editors. Fitzpatrick's dermatology in general medicine, vol 1. New York (NY): McGraw-Hill; 1999. p. 2742-63.
8. Youn JI, Oh JK, Kim BK, Suh DH, Chung JH, Oh SJ, et al. Relationship between skin phototype and MED in Korean, brown skin. Photodermatol Photoimmunol 1997;13:208-11.
9. Leenutaphong V. Relationship between skin color and cutaneous response to ultraviolet radiation in Thai. Photodermatol Photoimmunol 1995;11:198-203.
10. Kawasa A. UVB-induced erythema delayed tanning, and UVA-induced immediate tanning in Japanese skin. Photodermatol 1986;3:327-33.
11. Lancer HA. Lancer Ethnicity Scale (LES) [correspondence]. Lasers Surg Med 1998;22:9.
12. Baker RV, Joseph M. Melanin granules and mitochondria. Nature 1960;187:392.
13. Seiji M. Chemical composition and terminology of specialized organelles (melanosomes and melanin granules) in mammalian melanocytes. Nature 1963;197:1082.
14. Rawles ME. Origin of melanophores and their role in development of color patterns in vertebrates. Physiol Res 1948;28:383.
15. Jimbow K, Quevedo W, Fitzpatrick T, Szabo G. Biology of melanocytes. In: Freedberg IM, Eisen AZ, Wolff K, Austen KF, Goldsmith LA, et al, editors. Fitzpatrick's dermatology in general medicine, vol 1. New York (NY): McGraw-Hill; 1999. p. 192-219.
16. Zimmerman AA, Cornbleet T. The development of epidermal pigmentation in the Negro tevus. J Invest Dermatol 1948;11: 383.
17. Quevedo WC Jr, et al. Human skin color: original variation and significance. J Hum Evol 1985;14:43.
18. Fitzpatrick TB, Breathnach AS. Das epidermale melanin—einheit system. Dtsh Med Wochenschr 1963;147:481.
19. Jimbow K, Bereiter-Hahn J. Formation, chemical composition and functions of melanin pigments in mammals. In: Matoltsy AG, editor. Biology of the Integument. New York (NY): Springer Verlag; 1986.
20. Johnson BL Jr. Differences in skin type. In: Johnson BL Jr, Moy RL, White GM, editors. Ethnic skin: medical and surgical. St. Louis (MO): Mosby; 1998. p. 3-5.
21. Masson P. Pigment cells in man. In: Miner RW, Gordon M, editors. The biology of melanosomes. vol IV. New York (NY): New York Academy of Sciences; 1948. p. 10-7.
22. Szabo G. Pigment cell biology. In: Gordon M, editor. Mitochondria and other cytoplasmic inclusions. New York (NY): Academic Press; 1959.
23. Starkco RS, Pinkush. Quantitative and qualitative data on the pigment cell of adult human epidermus. J Invest Dermatol 1957;28:33.
24. Toda K, Patnak MA, Parrish A, Fitzpatrick TB. Alteration of racial differences in melanosome distribution in human epidermis after exposure to ultraviolet light. Nat New Biol 1972;236: 143-4.
25. Szabo G, Gerald AB, Patnak MA, Fitzpatrick TB. Racial differences in the fate of melanosomes in humane epidermis. Nature 222 1969;1081-2.
26. Montagna W, Carlisle K. The architecture of black and white facial skin. J Am Acad Dermatol 1991;24:929-37.
27. Olson RL, Gaylor J, Everett MA. Skin color, melanin, and erythema. Arch Dermatol 1973;108:541-4.
28. Mitchell R. The skin of the Australian Aborigines: a light and electron microscopical study. Australas J Dermatol 1968;9:314.
29. Smit NPM, Kolb RM, Lentjes EGWM, Noz KC, van der Meulen H, Koerten HK, et al. Variations in lelanin formation by cultured melanocytes from different skin types. Arch Dermatol Res 1998;290:342-9.
30. Goldschmidt H, Raymond JZ. Quantitative analysis of skin colour from melanin content of superficial skin cells. J Forensic Sci 1972;17:124.
31. Herzberg AJ, Dinehart SM. Chronologic aging in black skin. Am J Dermatopathol 1989;11:319-28.
32. Kotrajaras R, Kligman AM. The effect of topical tretinoin on photodamaged facial skin: the Thai experience. Br J Dermatol 1993;129:302-9.
33. Kaidbey KH, Agin PP, Sayre RM, Kligman A. Photoprotection by melanin—a comparison of black and caucasian skin. J Am Acad Dermatol 1979;1:249-60.
34. Strum RA, Box NF, Ramsay M. Human pigmentation genetics: the difference is only skin deep. Bioessays 1998;20:712-21.
35. Thomson ML. Relative efficiency of pigment and horny layer thickness in protecting the skin of Europeans and Africans against solar ultraviolet radiation. J Physiol 1955;127:236-8.
36. Abe T, Arai S, Mimura K, Hayakawa R. Studies of physiological factors affecting skin susceptibility to ultraviolet light irradiation and irritants. J Dermatol 1983;10:531-7.
37. Lee JH, Kim TY. Relationship between constitutive skin color and ultraviolet light sensitivity in Koreans. Photodermatol Photoimmunol Photomed 1999;15:231-5.
38. Hill HZ, Li W, Xin P, Mitchell DL. Melanin: a two-edged sword? Pigment Cell Res 1997;10:158-61.
39. Weigand DA, Haygood C, Gaylor JR. Cell layers and density of negro and caucasian stratum corneum. J Invest Dermatol 1974;62:563-8.
40. Reed JT, Ghadially R, Elias PM. Effect of race gender, and skin type of epidermal permeability barrier function [abstract]. J Invest Dermatol 1994;102:537.
41. La Ruche G, Cesarini JP. Histology and physiology of black skin. Ann Dermatovenereologica 1992;119:567-74.
42. Corcuff P, Lotte C, Rougier A, Maibach HI. Racial differences in corneocytes. Acta Derm Venereol 1991;71:146-8.
43. Warrier AG, Kligman AM, Harper RA, Bowman J, Wickett RR. A comparison of black and white skin using noninvasive methods. J Soc Cosmet Chem 1996;47:229-40.
44. Rienertson RP, Wheatley VR. Studies on the chemical composition of human epidermal lipids. J Invest Dermatol 1959;32:49-51.
45. Whitmore SE, Sago NJ. Caliper-measured skin thickness is similar in white and black women. J Am Acad Dermatol 2000;42: 76-9.
46. Leil R, Edwards J, Shary J, Spicer KM, Gordon B, Bell NH. The effects of race and body habitus on bone mineral density of the radius, hip, and spine in premenopausal women. J Clin Endocrinol Metab 1988;66:1247-50.
47. Szabo G. The number of eccrine sweat glands in human skin. In: Montagna W, Ellis R, Silver A, editors. Advances in biology of skin, vol 3. New York (NY): Pergamon Press; 1962.
48. Johnson LG, Landon MM. Eccrine sweat gland activity and racial differences in resting skin conductance. Psychophysiology 1965;1:322-9.
49. Montagna W, Parakkal PF. The structure and function of skin. 3rd ed. New York: Academic Press; 1974.
50. Robinson S, Dill DB, Wilson JW, Nielsen M. Adaptation of white men and Negroes to prolonged work in humid heat. Am J Trop Med 1941;21:261-87.
51. McCance RA, Purohit G. Ethnic differences in response to the sweat glands to pilocarpine. Nature 1969;221:378-9.
52. McCance RA, Rutishauser IHE, Knight HC. Response to sweat glands to pilocarpine in the Bantu of Uganda. Lancet 1968; 1:663-5.
53. Calhoun DA, Oparil S. Racial differences in the pathogenesis of hypertension. Am J Med Sci 1995;310(Suppl 1):S86-90.
54. Herrmann F, Prose PH, Sulzberger WB. Studies on sweating v. studies of quantity and distribution of thermogenic sweat delivery to the skin. J Invest Dermatol 1952;18:71.

55. Rebel G, Kirk D. Patterns of eccrine sweating in the human axilla. In: Montagna W, Ellis R, Silver A, editors. Advances in biology of skin, vol 3. New York (NY): Pergamon Press; 1962. p. 108-26.
56. Homma H. On apocrine sweat glands in White and Negro men and women. Bull Johns Hopkins Hosp 1956;38:365.
57. Johnson LC, Corah NL. Racial differences in skin resistance. Science 1960;139:766-7.
58. James CL, Worlana J, Stern JA. Skin potential and varometer responsiveness of black and white children. Psychophysiology 1976;13:523-7.
59. Juniper K Jr, Dykman RA. Skin resistance, sweat-gland counts, salivary flow, and gastric secretion: age, race, and sex differences, and intercorrelations. Psychophysiology 1967;4:216-22.
60. Kawahata A, Saramoto A. Some observations on sweating of the Aino. Jpn J Physiol 1951;2:166.
61. Goldsmith LA. Biology of eccrine and apocrine sweat glands. In: Freedberg IM, Eisen AZ, Wolff K, Austen KF, Goldsmith LA, et al, editors. Fitzpatrick's dermatology in general medicine, vol 1. New York (NY): McGraw-Hill; 1999.
62. Schiefferdecker P. Dsaael be (vollkomin. Mitt.). Zoologica 1922;27:1-154.
63. Homma H. On apocrine sweat glands in white and negro men and women. Bull Johns Hopkins Hosp 1926;38:365.[dupl]
64. Hurley HJ, Shelley WB. The physiology and pharmacology of the apocrine sweat gland. In: The human apocrine sweat gland in health and disease. Springfield (IL): Charles Thompson; 1960.
65. Ito T. Morphological connections of human apocrine and eccrine sweat glands: occurrence of the so called "mixed sweat glands"—a review. Okajimas Folia Anat Jpn 1988;65:315-6.
66. Downing DT, Stewart ME, Strauss JS. Lipids of the epidermis and the sebaceous glands. In: Freedberg IM, Eisen AZ, Wolff K, Austen KF, Goldsmith LA, et al, editors. Fitzpatrick's dermatology in general medicine, vol 1. New York (NY): McGraw-Hill; 1999.
67. Nicolaides N, Rothman S. Studies on the chemical composition of human hair fat: II, the overall composition with regard to age, sex and race. J Invest Dermatol 1952;21:90.
68. Kligman AM, Shelley WB. An investigation of the biology of the sebaceous gland. J Invest Dermatol 1958;30:99-125.
69. Champion RH, Gillman T, Rood AS, Burton L. An introduction to the biology of the skin. Philadelphia (PA): FA Davis; 1970. p. 418.
70. Pochi P, Strauss JS. Sebaceous gland activity in black skin. Dermatol Clin 1988;6:349.
71. Abedeen SK, Gonzalez M, Judodihardjo H, Gaskell S, Dykes P. Racial variation in sebum excretion rate. Program and abstracts of the 58th Annual Meeting of the American Academy of Dermatology; March 10-15, 2000; San Francisco, CA. Abstract #559.
72. Halder RM. Hair and scalp disorders in blacks. Cutis 1983; 32:378-80.
73. Vernall DG. Study of the size and shape of hair from four races of men. Am J Phys Anthropol 1961;19:345.
74. Brauner GJ. Cutaneous disease in the black races. In: Moschella SL, Pillsbury DM, Hurley JH, editors. Dermatology. Philadelphia (PA): WB Saunders; 1975.
75. Steggerda M, Seibert H. Size and shape of head hair from six racial groups. J Hered 1941;20:315-8.
76. Menkart J, Wolfram L, Mao I. Caucasian hair, Negro hair and wool: similarities and differences. J Soc Cosmetic Chemists 1966;17:769-87.
77. Swift JA. Fundamentals of human hair science [thesis]. Leeds, UK: Leeds University; 1963.
78. Sperling LC. Hair density in African Americans. Arch Dermatol 1999;135:656-8.
79. Khumalo NP, Doe PT, Dawber PR, Ferguson DJP. What is normal black African hair? A light and scanning electron-microscopic study. J Am Acad Dermatol 2000;43:814-20.
80. Hardy D, Baden HP. Biochemical variation of hair keratins in man and non-human primates. Am J Phys Anthropol 1973;39: 19-24.
81. Gold RJM, Schriver CH. The amino acid composition of hair from different racial origins. Clin Chem Acta 1971;33:465-6.
82. Bereson PS, Burch GE. Studies of diffusion through dead human skin. Am J Trop Med Hyg 1971;31:842.
83. Malkinson FD, Gehlman L. Factors affecting percutaneous absorption. In: Drill VA, Lazar P, editors. Cutaneous toxicology. New York (NY): Academic Press; 1977.
84. Scheuplein RJ, Blank IH. Permeability of the skin. Physiol Rev 1971;51:702.
85. Marzulli FN. Barriers to skin penetration. J Invest Dermatol 1962;39:387.
86. Wickrema-Sinha WJ, Shaw SR, Weber OJ. Percutaneous absorption and excretion of tritium-labelled diflorasone diacetate: a new topical corticosteroid in the rat, monkey and man. J Invest Dermatol 1978;7:372-7.
87. Guy RH, Tur E, Bjerke S, Maibach H. Are there age and racial differences to methyl nicotinate-induced vasodilatation in human skin? J Am Acad Dermatol 1985;12:1001-6.
88. Wedig JH, Maibach HI. Percutaneous penetration of dipyrithione in men: effect of skin color (race). J Am Acad Dermatol 1981;5:433-8.
89. Stoughton RB. Bioassay methods for measuring percutaneous absorption. In: Montagna W, Stoughton RB, van Scott EJ, editors. Pharmacology of the skin. New York (NY): Appleton-Century-Crofts; 1969. p. 542-4.
90. Berardesca E, Maibach HI. Racial differences in pharmacodynamic responses to nicotinates in vivo in human skin: black and white. Arch Derm Venereol 1990;70:63-6.
91. Berardesca E, Maibach HI. Effect of race on percutaneous pepetration of nicotinates in human skin: a comparison of white and hispanic-Americans. Bioeng Skin 1988;4:31-8.
92. Gean CJ, Tur E, Maibach HI, Guy RH. Cutaneous responses to topical methyl bicofinate in black, oriental and caucasian subjects. Arch Dermatol Res 1989;281:95-8.
93. Judge MR, Griffiths HA, Basketter DA, White IR, Rycroft RJG, McFadden JP. Variation in response of human skin to irritant challenge. Contact Dermatitis 1996;34:115-7.
94. Marshall EK, Lynch V, Smith HV. Variation in susceptibility of the skin to dichloroethylsulfide. J Pharmacol Exp Ther 1919;12: 291-301.
95. Schwartz L, Tulipa L, Birmingham DJ. Occupational Diseases of the Skin. Philadelphia, PA: Lea & Febiger; 1939.
96. Shelley WB. Newer understanding of ecology in dermatology. In: Rees RB, editor. Dermatosis due to environmental and physical factors. Springfield (IL): Charles C. Publisher; 1962. p. 12.
97. Weigand DA, Mershon MM. The cutaneous irritant reaction to agent O-chlorobenzylidene malonitrile. (cs) (1): Quantitation and racial influence in human subjects. Edgewood Arsenal Technical Report 4332, February, 1970.
98. Frosch RJ, Kligman AM. The chamber scarification test for assessing irritancy of topically applied substances. In: Drill VA, Lazar P, editors. Cutaneous toxicology. New York (NY): Academic Press; p. 150.
99. Marshall J, Heyl T. Skin diseases in Western Cape Province. S Afr Med J 1963;37:1308.
100. Marshall J. New skin diseases in Africa. Trans St Johns Hosp Dermatol Soc 1970;56:3-10.
101. Berardesca E, Maibach HI. Racial differences in sodium lauryl sulfate induced cutaneous irritation: black and white. Contact Dermatitis 1988;18:65-70.

102. Berardesca E, Maibach HI. Sodium-lauryl-sulphate-induced cutaneous irritation comparison of white and hispanic subjects. Contact Dermatitis 1988;19:136-40.
103. Berardesca E. Racial differences in skin function. Acta Derm Venereol 1994;185(suppl):44-6.
104. Wilson D, Berdardesca E, Maiback HI. In vivo transepidermal water loss: differences between black and white skin. Br J Dermatol 1988;119:647-52.
105. Berardesca E, Maibach HI. Sensitive and ethnic skin: a need for special skin care agent? Dermatol Clin 1991;9:89-92.
106. Goh CL, Chia SE. Skin irritability to sodium-lauryl sulphate-as measured by skin water vapour loss—by sex and race. Clin Exp Dermatol 1988;13:16-9.
107. Kompaore F, Marty JP, Dupont C. In vivo evaluation of the stratum corneum barrier function in blacks, Caucasions and Asians with two non-invasive methods. Skin Pharmacol 1993; 6:200-7.
108. DeLuca R, Balestrier A, Dinle Y. Measurement of cutaneous evaporation. 6. Cutaneous water loss in the people of Somalia. Boll Soc Ital Biol Sper 1983;59:1499-501.
109. Pinnagoda J, Tupker RA, Agner T, Serup J. Guidelines for transepidermal water loss (TEWL) measurement. Contact Dermatitis 1990;22:164-78.
110. Taberi DP, Narurkar V, Moy RL. Skin cancer. In: Johnson BL Jr, Moy RL, White GM, editors. Ethnic skin: medical and surgical. St. Louis (MO): Mosby; 1998.
111. Halder RM, Bridgeman-Shan S. Skin cancer in African Americans. Cancer 1995;75:667-73.
112. Crombie IK. Racial differences in melanoma incidence. Br J Cancer 1979;40:185.
113. Cress RD, Holly EA. Incidence of cutaneous melanoma among non-Hispanic whites, Hispanics, Asians, and blacks: an analysis of California Cancer Registry data, 1988-93. Cancer Causes Control 1997;8:246-52.
114. Clark WH, Elder DE, Van Horn M. The biologic forms of malignant melanoma. Hum Pathol 1986;76:403-14.
115. Vermeer M, Schmieder GJ, Yoshikawa T, van den Berg J-W, Metzman MS, Taylor JR, et al. Effects of ultraviolet B light on cutaneous immune responses of humans with deeply pigmented skin. J Invest Derm 1991;97:729-34.
116. Stephens TJ, Oresajo C. Ethnic sensitive skin: a review. Cosmetics Toiletries 1994;109:75-80.
117. Halder RM. The role of retinoids in the management of cutaneous conditions in blacks. J Am Acad Dermatol 1998;39:S98-S103.
118. Halder RM, Grimes PE, McLaurin CI, Kress MA, Kenney JA Jr. Incidence of common dermatoses in a predominantly black dermatology practice. Cutis 1983;32:388.
119. Grimes PE, Stockton T. Pigmentary disorders in blacks. Dermatol Clin 1988;6:271-81.
120. White GM. Pseudofilliculitis barbae. In: Johnson BL Jr, Moy RL, White GM, editors. Ethnic skin: medical and surgical. St. Louis (MO): Mosby; 1998.
121. LoPresti P, Papa CH, Kligman A. Hot comb alopecia. Arch Dermatol 1968;98:234-9.
122. Sperling LC, Sau P. The follicular degeneration syndrome in black patients. Arch Dermatol 1992;128:68-74.
123. Sperling LC, Solomon AR, Whiting DA. A new look at scarring alopecia. Arch Dermatol 2000;136:235-420.
124. Ackerman AB, Walton NW, Jones RE, Charissi C. "Hot comb alopecia"/"follicular degeneration syndrome" in African-American women is traction alopecia. Dermatopathol Practical Conceptual 2000;6:320-36.
125. Cosman B, Crikelair GF, Ju MC, Gaulin JC, Lattes R. The surgical treatment of keloidal scars. Plast Reconstr Surg 1961;27:355-8.
126. Ketchum LD, Cohen IK, Masters FW. Hypertrophic scars and keloidal scars. Plast Reconstr Surg 1974;53:140-54.
127. Oluwasanmi JO. Keloidal scars in the African. Clin Plast Surg 1974;1:179-95.
128. Johnson BL Jr. Keloids. In: Johnson BL Jr, Moy RL, White GM, editors. Ethnic skin: medical and surgical. St. Louis (MO): Mosby; 1998. p. 167-70.
129. Sherertz E, Schwartz S. Patch test interpretation of black skin. Am J Contact Dermatitis 1993;4:247-8.
130. Rosenberg A, Kanof NM. Studies in eczematous sensitization. J Invest Derm 1941;4:505.
131. Epstein W, Kligman AM. The interference phenomenon in allergic contact dermatitis. J Invest Derm 1958;31:175.
132. Kligman AM, Epstein W. Updating the maximization test for identifying contact allergens. Contact Dermatitis 1975;1: 231-9.
133. Menne T, Maibach HI. Exogenous dermatoses: environmental dermatitis. Boca Raton (FL): CRC; 1991. p. 66-9.
134. Leyden JJ, Kligman AM. Allergic contact dermatitis: sex difference. Contact Dermatitis 1977;3:333-6.
135. Fisher AA. Contact dermatitis in black patients. Cutis 1977; 20:905-22.
136. Dickel H, Taylor JS, Evey P, Merk HF. Comparison of patch test results with a standard series among white and black racial groups. Am J Contact Dermat 2001;12:77-82.
137. Mar A, Tam M, Jolley D, Marks R. The cumulative incidence of atopic dermatitis in the first 12 months among Chinese, Vietnamese, and caucasian infants born in Melbourne, Australia. J Am Acad Dermatol 1999;40:597-602.
138. McMichael AJ. Scalp and hair disease in the black patient. In: Johnson BL Jr, Moy RL, White GM, editors. Ethnic skin: medical and surgical. St. Louis (MO): Mosby; 1998. p. 214-30.
139. Kupicha FK. Lythraceae. In: Heywood V, editor. Flowering plants of the world. New York (NY): Oxford University Press; 1993. p. 156-7.
140. Zinkham WH, Oski FA. Henna: a potential cause of oxidative hemolysis and neonatal hyperbilirubinemia. Pediatrics 1996; 97:707-9.
141. Sir Hazsnim M. Poisoning from henna dye and paraphenylenediamine mixture in children in Khartoum. Ann Trop Paediatr 1992;12:3-6.
142. Lynn MJ, Shaw JC. Allergic contact dermatitis reaction to henna. Arch Dermatol 2000;136:124-5.
143. Sahoo B, Handa S, Penchallaiah K, Kumar B. Contact anaphylaxis due to hair dye. Contact Dermatitis 2000;43:244.
144. Al-Mejed SA, Harakati MS. The effect of henna paste on oxygen saturation reading obtained by pulse oximetry. Trop Georg Med 1994;46:38-9.