Clinics in Dermatology (2019) **37**, 418–429

ELSEVIER

Clinics in Dermatology

Impact of skin color and ethnicity

Balaji Jothishankar, BS[a], Sarah L. Stein, MD[b,*]

[a]*University of Chicago Pritzker School of Medicine, Chicago, Illinois, USA*
[b]*Section of Dermatology, Department of Medicine and Pediatrics, University of Chicago Pritzker School of Medicine, Chicago, Illinois, USA*

As the field of medicine progresses toward an individualized approach to treating patients, race and ethnicity have become important topics of research and clinical conversations. Management decisions are being made on a more individualized basis, leading to better outcomes for patients and more appropriate clinical care. In the field of dermatology particularly, ethnicity and skin color are important considerations impacting the presentation and manifestation of many conditions and contributing to differences in outcome goals, skin and hair care product choices and routines among populations. We review how pigmentation affects skin and hair structure and function, highlight how cultural and ethnic differences impact skin and hair care, consider the dermatologic health care disparities among people with different skin colors, and address the importance of skin color differences in dermatology training.

Race versus ethnicity: what's the difference?

The terms race and ethnicity are frequently used almost interchangeably; however, there is a stark difference regarding the origin of these words, their definitions, and their use in clinical research and practice. The concept of race was used in the early 18th century as a tool by which to classify humans. The racial classifications were based primarily on skin color.[1] Although it is widely accepted that race is a social construct and is an arbitrary descriptor, it continues to be used in clinical research and practice as an explanatory variable, similar to educational status and socioeconomic status.[2] Ethnicity is a term that encompasses an amalgamation of social, religious, dietary, and other cultural components.[1] As such, the concept of ethnicity is a much more informative component to consider in the study of health, disease, and therapy.[3,4]

The use of race as a descriptor when practicing dermatology is likely to inaccurately categorize many of our patients. We risk disregarding the genetic makeup of patients, reflecting the interracial mixing that is increasingly common in our society.[5] Classifications by race are likely to overlook many differences in cultural practices, leading to stereotyped assumptions about certain populations. To avoid these pitfalls, efforts have been made to have patients self-identify their racial group[6]; however, studies have shown that self-reported race is a poor predictor of Fitzpatrick skin type, the most common method of assessing sunburn and skin cancer risk.[7] The Fitzpatrick scoring system was developed in 1975 based on an individual's skin color and response to sun exposure.[8] It is a common measure often used to assess skin cancer risk and sun sensitivity, and it has become an unofficial tool for characterizing an individual patient's skin color. The current Fitzpatrick skin type classification delineates six different skin types based on skin color:

- Very lightly pigmented, or white, skin is labeled skin type I.
- Very darkly pigmented, or black, skin is labeled skin type VI.

When initially introduced, the Fitzpatrick skin type was meant to be determined via a patient questionnaire self-reporting natural hair and skin color, tanning habits, and

* Corresponding author. Tel.: +1 773 834 2004.
E-mail address: sstein@medicine.bsd.uchicago.edu (S.L. Stein).

https://doi.org/10.1016/j.clindermatol.2019.07.009

reaction to sun exposure.[8] A study of a black population indicated that the Fitzpatrick questionnaire excluded many black patients and overestimated the number of black patients with a type IV Fitzpatrick skin rating.[9] This study suggested that more culturally sensitive language and skin categories are necessary to increase the utility of the instrument in patients with skin of color. In practice, many dermatologists will often estimate a patient's Fitzpatrick skin type based on observation or elicitation of some but not all of the criteria in an effort to find an objective measure by which to sensitively care for patients of varying skin colors (Figure 1).

As we progress through this chapter, we use the terms ethnic skin, pigmented skin, or skin of color instead of race.

Skin color as it influences skin structure

Skin color is dependent on several factors, including the type of melanin present, melanosome content, genetics, UV exposure, and additional chromophores in the skin.[10] The perception of skin color is, in part, impacted by the presence of varying combinations of four chromophores: hemoglobin, oxyhemoglobin, melanin, and carotenoids. The combination of these chromophores leads to differing absorption and reflection of light, causing the skin to appear as varying shades. In white skin, hemoglobin and oxyhemoglobin combine to produce a pink hue. In brown skin, melanin and carotenes combine to produce a yellow-orange hue.[11]

Melanocytes are the cells critical to the production of melanin pigment in skin. Melanosomes are the organelle within melanocytes in which melanin pigment is synthesized, deposited, and transferred. The number of melanocytes does not differ between ethnic groups or account for the differences in skin color; rather, differences in skin color are due to variations in size, amount, and distribution of melanosomes within melanocytes and keratinocytes.[12–14] In lightly pigmented skin, the melanosomes are smaller and clustered in groups within secondary lysosomes, whereas in darkly pigmented skin, the melanosomes are larger and more individually dispersed within lysosomes. The differences in melanosome density and size lead to the various shades of skin color that exist even within ethnic groups, with varying proportions of smaller aggregated melanosomes and larger singly dispersed melanosomes depending on the degree of pigmentation.[13,15]

Melanosome density is also influenced by sun exposure. Sun-exposed areas of the body tend to demonstrate more dispersed melanosomes than sun-protected regions. This finding was illustrated in a study of Asian skin in which it was shown that the pattern of dispersed melanosomes present in sun-exposed skin sites was similar to the pattern of melanosomes in a subset of white patients with darker skin tones, whereas the sun-protected sites had more aggregated melanosomes, similar to the pattern seen in very fair white skin.[13,15]

The location of melanosomes within the epidermis also varies by skin color. Melanosomes in darkly pigmented skin are found throughout the epidermis and are most concentrated and evenly dispersed along the basal layer, whereas in white skin, melanosomes are more quickly degraded, resulting in small aggregates of melanosomes in the basal cell and malpighian layers and a notable absence in the upper layers of the epidermis[16] (Figure 2). Within an ethnic group, more darkly shaded black skin demonstrates increased basal layer melanosomes compared with lighter shaded black skin. Similar findings are observed in darker shaded Asian skin and darker shaded white skin compared with their lighter counterparts.[13,17]

Melanin is the pigment present in skin. Eumelanin is brown/black in color and predominates in darker skin. Pheomelanin is yellow/red in color and predominates in whiter skin. The amino acid tyrosine is the starting material for both types of melanin, and the enzyme tyrosinase is the key regulatory enzyme in the synthetic pathway. When tyrosinase activity is decreased, pheomelanin production is the default pathway. Black skin is more melaninized as a result of increased enzyme activity and increased melanin production.[18–20] A variety of other factors involved in melanogenesis differ between skin colors contributing to variations in the production of melanin. The optimal pH for melanin synthesis is 6.8, which is closely approximated within melanosomes in black skin, whereas the pH within melanosomes in white skin is more acidic.[21–23] Another cellular factor that influences pigmentation is melanosome transfer to keratinocytes. This process involves protease-activated receptor 2. Darker pigmented skin has higher levels of protease-activated receptor 2 compared with lighter pigmented skin. Additionally, lighter skin demonstrates delayed upregulation of protease-activated receptor 2.[24] This increase in protease-activated receptor 2 activity could be a factor contributing to the development of hyperpigmentation seen more frequently in darker skin colors.

Beyond pigmentation, differences have been noted in the structure of the epidermis and dermis of skin of varying colors. Differences in epidermal structure have been associated with differing responses to aging observed across skin colors. Most

Fig. 1 Example of the use of the Fitzpatrick Scale to broadly classify skin by a visual appreciation of the degree of pigmentation.

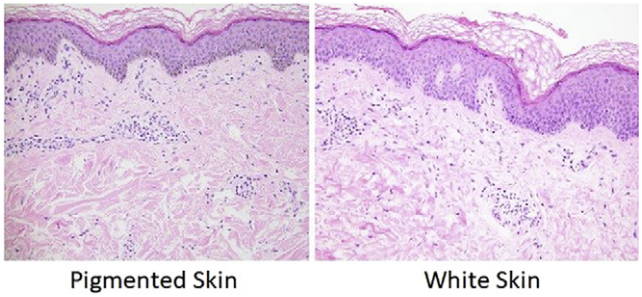

Fig. 2 Histology of epidermis and upper part of the dermis from pigmented skin and white skin, demonstrating concentration of pigment most notably along basal layer of the epidermis.

notably, the stratum lucidum is more compact in sun-exposed black skin, whereas it demonstrates hypercellularity and edema in sun-exposed white skin. A heavily pigmented epidermis is less vacuolated and dyskeratotic than less pigmented samples. Although a measurable difference in skin thickness between black and white skin has not been confirmed,[16] there are more corneocytes in the stratum corneum in black skin than in white skin, suggesting a more compacted and dense layer.[25] Likely as a result of this compaction, an increased mean electrical resistance is measured in black skin compared with white skin.[26] Further investigations have shown that corneocytes are the same size in black, white, and Asian skin,[27] and no differences in corneocyte maturation are detectable among the various skin colors.[28] The ceramide content of the epidermis appears to be highest in Asian and Hispanic skin and lowest in black skin.[29,30] No difference in skin surface pH has been detected between the ethnic groups.[31,32] Contributors to the skin flora, including *Cutibacterium acnes* (formerly *Propionibacterium acnes*), aerobic bacteria, and *Candida albicans,* are more abundant on black skin than white skin.[33,34]

Study of the dermis from skin of various colors has demonstrated equivalent thickness.[35] Although the number and size of dermal fibroblasts and macrophages is greater in black skin compared with white skin, collagen fibers are smaller and more closely stacked together in white skin.[16] A significant decrease in collagenase enzyme activity is detected in black skin.[14] This decrease in collagenase enzyme activity is one factor that is postulated to contribute to the higher incidence of keloid formation in the dark-skinned populations. Measurements of skin elasticity and viscoelasticity using a Twistometer have suggested differences in elastic recovery and skin extensibility between black, Hispanic, and white skin.[33,36]

Quantification of eccrine sweat gland numbers is not different between black and white skin[37,38]; however, conflicting data exist on the activity of eccrine sweat glands among ethnic groups. Some studies have demonstrated an increased quantity of sweat from white skin compared with black and Asian skin, with a resultant increase in retention of sodium detectable in black skin[39,40]; however, electrophysiology studies, which indirectly measure eccrine sweat gland activity by measuring skin resistance, have detected increased eccrine gland activity in black skin compared with white skin.[26,41,42] Considering the small sample sizes and the influence of environmental factors,[43] it is difficult to make definitive statements regarding eccrine sweat gland activity and sweat quantity.

The study of apocrine sweat glands suggests that black skin has an increased number of these sweat glands and larger structures compared with white and Chinese skin.[44] Data are not available on whether there are measurable differences in apocrine secretions. The number of sebaceous glands between skin colors does not seem to differ, although black skin demonstrates on average larger sebaceous glands.[45] Similar to functional eccrine sweat gland studies, the investigations of sebaceous gland function from varying skin colors have had conflicting results.[45–47]

Skin color as it influences skin function

Investigations into functional differences in the skin between patients of different skin colors have often been hampered by limitations such as small sample sizes, poor study designs, and conflicting results. Two studies examining the absorption of drugs through black and white skin showed that there was no difference in percutaneous absorption of topical medications,[48,49] whereas three other studies suggested decreased absorption of topical medications through black skin.[50–52] A study of white skin versus Hispanic skin showed no differences in absorption,[53] but a study of Asian skin versus white skin demonstrated decreased absorption through Asian skin compared with white.[54]

Assessments of the skin's susceptibility to irritants as a function of skin color has been similarly fraught by limitations and conflicting data. Investigator bias is a particularly relevant concern as many of these studies rely on erythema as an endpoint. Erythema is a subjective measure that is affected by background skin color and is notoriously underappreciated in pigmented skin (Figures 3 and 4). Studies using erythema as an endpoint tend to conclude that black skin is less susceptible to contact irritants than white skin and that lighter white skin is the most susceptible to skin irritants.[55–58] The difficulty in appreciating erythema in darker skin colors has been suggested as one reason why black residents of nursing homes are two to four times more likely to develop decubitus ulcers compared with white patients.[59] Transepidermal water loss as a function of stratum corneum integrity is another endpoint used to assess skin irritation. Even using this endpoint, the data have been conflicting, with some studies indicating Asian and black skin is more susceptible to irritants than white skin,[60,61] whereas others find no difference between groups.[62–64] A study comparing 15 light-skinned Chinese patients, 12 Malaysians with dark skin, and 11 Indians with very dark skin showed no significant differences in skin irritation between the groups using skin water vapor loss as the primary endpoint.[65] When patients were asked to self-assess skin sensitivity, there was no difference in frequency of sensitive skin by ethnic group.[66]

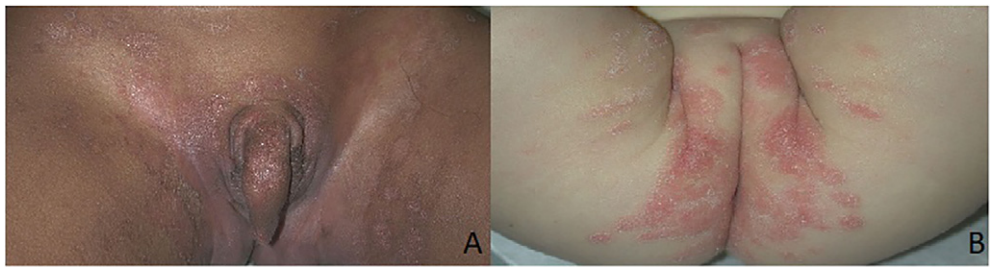

Fig. 3 Irritant diaper dermatitis presentations in skin of varying pigmentation, demonstrating the effect of pigmentation on erythema.

Investigators have looked at the effect of UV radiation on skin of different colors. The pigmented eumelanin of black skin is a fundamental photoprotective feature, serving to comprehensively block UV rays from penetrating through the epidermis.[67] Exposure to UV rays upregulates melanin production as demonstrated by an increase in production one week after exposure across all skin colors, but black skin shows a 12% increase compared with a 4% increase in Asian skin and a 1% increase in white skin.[68] UV radiation causes photo-induced damage to skin regardless of color but to varying degrees. The damage can be quantified by measuring the number of pyrimidine dimers and 6-4 photoproducts that are formed in the DNA of UV exposed cells and by measuring the activity of the p 53 gene in exposed cells.[68] This gene functions as a tumor suppressor, preventing cancer formation. Studies have shown that there is increased activity of the p 53 gene after UV exposure in black skin compared with white skin,[69] and a similar finding was observed in Asian skin compared with white skin,[70] suggesting that activity of this gene is one of the mechanisms protecting these skin types from the development of skin cancers.

Skin color and the impact on hair

Hair is a central component of self-identity and is a visually apparent characteristic that is frequently used by others to categorize and label. Hair color and texture have been correlated with differences in the cross-sectional shape of individual hair shafts. Cross sections of hair shafts obtained from African patients are noted to be elliptical, appearing flattened in cross section, and spontaneously curving into spirals.[71,72] Asian hair tends to be rounder in cross section and has the largest diameter of all hair types. Hair studied from a cohort of white-skinned subjects falls in between these ends of the spectrum, being somewhat smaller in diameter but more elliptical in cross-sectional shape than Asian hair.[71] African hair demonstrates a reduction in the numbers of elastic fibers anchoring the hair follicles to the dermis.[16] Whether this finding contributes to a higher risk of hair loss is unclear; furthermore, total hair density and total number of terminal hair follicles is lower when comparing African subjects with white subjects.[73] No differences have been found regarding the type of keratin or amino acid composition between the groups.[74,75]

Prevalence of skin disorders between ethnic groups

National databases have allowed for ranking of the top five dermatologic diagnoses made by US dermatologists by population: in black populations the top five diagnoses are acne, dermatitis, seborrheic dermatitis, atopic dermatitis, and dyschromia; in Asian populations the top five diagnoses are acne,

Fig. 4 Plaque-type psoriasis presentations in skin of varying pigmentation, demonstrating the effect of pigmentation on erythema.

Fig. 5 Hyperpigmented patches resulting from prior insults to the skin such as insect bites and resolving dermatitis.

Fig. 6 Ill-defined hypopigmented macules and patches on the face is typical of pityriasis alba.

dermatitis, benign neoplasms, psoriasis, and seborrheic keratoses; in Hispanic populations the top five diagnoses are dermatitis, acne, psoriasis, benign neoplasms, and warts; and in white populations the top five diagnoses are actinic keratoses, acne, benign neoplasms, dermatitis, and nonmelanoma skin cancer.[76]

Although there are similarities regarding diagnoses across ethnic groups, the most obvious difference is seen in the prevalence of the top three skin cancers: basal cell carcinoma, squamous cell carcinoma, and melanoma. The incidence of these skin malignancies among people with darker skin colors is relatively low compared with white patients, most likely due to the increased melanin content and broad melanosomal dispersion, which provides protection from the penetration of UV irradiation into the skin.[14,77] Persons with skin of color do still manifest these malignancies but at lower rates. Of concern is that these malignancies are often more advanced at the time of diagnosis.[78] This is likely a result of underrecognition within these populations, possibly less screening by health care providers, and the presence of these tumors more frequently in unexpected sites.

Melanoma is more likely to arise in nonsun-exposed areas such as the palms and soles in black, Asian, and Hispanic subjects, with acral lentiginous melanoma being the most common type of melanoma in these populations. Black and Hispanic transplant recipients who develop malignant neoplasms are more likely to manifest these tumors in sun-protected areas compared with the sun-exposed sites observed for white recipients.[79] Melanoma in particular is more likely to have been diagnosed after it has metastasized and to have a worse outcome when diagnosed in nonwhite populations.[78] The 5-year melanoma survival is 66.7% for black patients compared with 92.5% for white patients.[80] These differences are likely due to a combination of societal factors, including gaps in public education, gaps in education of health care providers, and inequality of access to health care.

The melanin content and melanosome dispersion pattern that protects against the development of skin cancer also protects against photoaging to a certain degree. Data suggests that the epidermis of darkly pigmented skin provides the equivalent of a sun protection factor (SPF) of 13.4 compared with a SPF of 3.4 inherent to white skin.[67] Clinical features of photoaging include wrinkles, dyspigmentation, loss of elasticity, and telangiectasias; histologically, chronically sun-exposed skin demonstrates epidermal atrophy with thinning of the spinous layer and degenerative dermal changes.[81] Studies have shown that these clinical and histopathologic findings occur on average at an older age for black patients than white patients.[82,83]

Pigmentation disorders disproportionately affect more darkly pigmented populations. The most common pigmentation disorder is postinflammatory hyperpigmentation[83] (Figure 5). This process is thought to be the default response to cutaneous injury. It has been proposed that melanocyte irritation triggers the release and subsequent oxidation of arachidonic acid, which upregulates the production and transfer of melanin from melanocytes to keratinocytes.[84] In some cases, the epidermal basal layer is additionally disrupted, allowing for melanin to be deposited in the dermis. Postinflammatory hyperpigmentation is often a sequela of acne in patients with darker skin colors and is often as concerning to patients as the primary inflammatory acne lesions.[83,84] Melasma is a pigmentation disorder that is more prevalent in black, Asian, and Hispanic skin. This condition is multifactorial with melanocyte lability playing a significant role.[85] Similarly, hypopigmentation conditions are more prominent in darker skin colors due to the contrast with the surrounding unaffected skin. Pityriasis alba is an example of a hypopigmentation disorder

Fig. 7 Acne presentations in skin of varying pigmentation, demonstrating the difference in type of lesions and the degree of redness and hyperpigmentation associated.

occurring commonly in atopic individuals but more frequently presenting for medical management when affecting those with darker pigmented skin (Figure 6).

Keloids are a scarring phenomenon that varies in prevalence by ethnic group, although whether this is a function of skin color or other factors is not currently understood. The incidence of keloids is higher in black patients than white patients[86–88] and higher in Chinese patients than Indian and Malaysian patients.[89] As alluded to previously, whether or not there is a true difference in prevalence of contact dermatitis between ethnic groups has been difficult to determine, possibly due to the use of erythema as the endpoint of most studies.[90–92]

The incidence of acne is similar across all ethnic groups,[93] and it is the most common skin disorder reported regardless of ethnicity. Interestingly, the type of acne lesions are described differently across ethnic groups, with white patients reported as manifesting inflammatory lesions and black patients more frequently described as manifesting comedones[10,11] (Figure 7). When looked at histologically, comedones in black skin often demonstrate an associated inflammatory component, whereas this is less commonly described in association with comedones in white skin. The presence of the associated inflammation observed in the pigmented skin samples could be a contributing factor to postinflammatory hyperpigmentation.[94] Treatment of acne in darker skin colors requires careful consideration of interventions to prevent or impact the expected hyperpigmentation. Such approaches may include earlier initiation of treatments, therapy initiation at low concentrations to avoid provoking irritation, use of retinoids or retinoid analogues to gradually lighten dark marks, and education on the importance of sun protection to optimize resolution of dyspigmentation.[94] Sensitivity to and recognition of cultural practices that may contribute to the development of acne, such as the use of hair pomades and comedogenic skin care products, are additionally important considerations in the management of acne in various populations.[95]

Atopic dermatitis is more prevalent in Asian and black populations compared with white populations, particularly in children. The odds ratio of developing atopic dermatitis is 3.1 for Asians, 2.2 for black patients, and 1.9 for Hispanics.[96–99] This predominance is present even after adjusting for sociodemographic and vitamin D levels.[100] The difference in prevalence has particular consequences regarding treatment, as historically much of the evidence for systemic therapy for atopic dermatitis in the United States has been based on randomized studies performed in Europe, where there is less ethnic diversity than is found in the United States, therefore making it difficult to generalize the results.[101]

Hair and scalp disorders differ in their presentations and etiologies among populations of varied skin colors and hair types. The biological differences in the structure of the hair shafts and follicles and their configuration, combined with cultural hair practices, may contribute to differences in prevalence of conditions such as pseudofolliculitis barbae, dissecting cellulitis, central centrifugal alopecia, acne keloidalis nuchae, traction alopecia, and tinea capitis.[102] Few studies have attempted to investigate the role of cultural practices and differences in hair physiology on the development of alopecia.

Effects of cultural practices on dermatologic disease

How people choose to care for their skin and hair is influenced by an ever expanding array of influences, fueled by the access to social media and other electronic inputs. Cultural roots provide the foundation shaping other choices and decisions. The structural and functional variations observed in skin of different colors likely influences the skin and hair care practices that have been historically adopted by ethnic groups. Some of these practices may be introduced as remedies for perceived imperfections or to concede to social pressures and can ultimately lead to complications that present to the dermatologist.

The use of skin lightening agents, such as hydroquinones, topical steroids, and mercurials, is popular in Africa, India, and other parts of Asia.[103–105] Many of the skin lightening products that are used by individuals in the United States are imported from other countries. These products are marketed in stores and online to target ethnic groups with darker pigmented skin colors. The use of these agents to lighten the skin has been observed to cause a variety of unintentional adverse effects. Hydroquinone-containing topical preparations can

lead to exogenous ochronosis[106–108] and contact dermatitis[109] and may have a carcinogenic potential.[110] Topical corticosteroids can lead to the development of acne, contact dermatitis, skin atrophy, rosacea, and telangiectasias.[111–113] The use of mercury-containing agents can lead to contact dermatitis, purpura, flushing, erythroderma, gingivostomatitis, and paradoxical hyperpigmentation.[114,115]

Hair care and styling accounts for some of the most varied practices among individuals. Styles such as braids, cornrows, and hair extensions can lead to chronic traction on the hair follicles and subsequent hair loss, which can become permanent. The use of chemicals and heat for straightening or curling hair can lead to hair breakage and contact dermatitis of the exposed skin. Hair pomades are often a combination of petrolatum, lanolin, and other oils. These products are used for hair styling, but prolonged contact with the skin of the forehead and temples can contribute to acne eruptions.[94]

Cultural practices, such as coining, cupping, and moxibustion, may be used as alternative approaches to managing health conditions but can have impacts on the skin that dermatologists will need to recognize. Coining involves using the edge of a coin to trace the skin around the edge of an inverted jar. This practice is felt to improve the circulation within soft tissue, but can lead to the development of ecchymotic circular streaks. Cupping involves inverting a jar on the skin while creating a vacuum within the jar, pulling the skin into the mouth of the jar. This practice results in round ecchymoses. Moxibustion is the application of incense to acupuncture sites, which can lead to second-degree burns.[116] Chili peppers are a common cooking ingredient for many cultures. The handling of chili peppers can lead to burning pain, erythema, and irritation of exposed skin and mucosa.[117]

Dermatologic health care disparities

Health care disparities exist for a multitude of reasons, ranging from lack of access to care to lack of trust in the health care system and providers to biased care delivery. The groups that tend to be most affected by these disparities are ethnic minorities and people of lower socioeconomic status.[118] Studies have shown that black patients are more likely than white and Hispanic patients to report cost as a barrier to dermatology follow-up care.[119] furthermore, only 8% of patients who are seen by dermatologists are nonwhite, whereas 16% of patients seen by nondermatologists are nonwhite. This 50% reduction in patient visits between nondermatologists and dermatologists suggests that populations of color do not have equal access to dermatologic care.[120]

Access-to-care limitations contribute to deficits in skin-directed health literacy among populations with skin of color, impacting such topics as the effects of chronic UV exposure, skin cancer risks, and the benefits of skin cancer screening.[121–123] The relative deficit of dermatologists in many parts of the country contributes to the dermatology access problem. A practice profile from 2009 showed that 38% of dermatologists reported that there was a shortage of dermatologists in their area, with an average wait time of 33.8 days.[124] Insurance status also impacts access to dermatologic care. Patients with private insurance are significantly more likely to visit a dermatologist than those with public insurance. Even when controlling for type of insurance, black patients and Hispanic patients are still significantly less likely to see a dermatologist; furthermore, black and Hispanic patients are much less likely to make nonreferred visits to a dermatologist compared with white patients.[125]

A systematic review examining diversity in dermatology clinical trials showed that only 60% of studies done within the United States reported on racial or ethnic demographics. African American patients were proportionally represented in these studies compared with the general population, but Hispanic representation was lower than that of the general population.[126] This lack of representation may be the result of failures in study recruitment but also mirrors the health care disparity issues in general. Distrust of the medical system is one of the issues of concern, potentially resulting in part from race discordance between patients and providers. Studies have demonstrated that patients with race discordant providers are more likely to feel unheard by their physicians and not be comfortable with treatment plans.[127] In a study of pediatric dermatology patients, black families indicated significant less trust for the medical research community and were significantly less likely to enroll their children in clinical studies. This study also found that black families had less exposure to research advertisements than white families.[128] The limited representation of ethnic minorities in research make the results difficult to generalize to minority populations and could lead to detrimental outcomes for patients.[129–131]

Diverse skin types and dermatology training

Dermatology training for US medical students is generally relatively limited, and any significant focus on skin disease by skin color is even more limited. Dermatology training makes up an average of 16 to 22 hours over the course of the medical school curriculum, or slightly less than 1% of the total undergraduate medical training hours. Only 40% of primary care residents report feeling prepared to manage common skin conditions in general.[132–135] A survey of dermatologists and dermatology residents found that 47% felt that they had received inadequate training on skin conditions in patients with skin of color due to lack of exposure or training material.[136] These sentiments were confirmed in a survey of dermatology chief residents and residency program directors who reported that only 52% and 66%, respectively, had didactics training specific to ethnic skin in their curriculum.[137]

By the year 2043, it is projected that no single ethnic group will make up a majority of America; however, black and Hispanic dermatologists make up only 3% and 4.2% of all dermatologists, respectively. This is in contrast to the 12.4% of black Americans and 16.3% of Hispanic Americans that make up the

American population currently. These differences are more exaggerated in the field of dermatology than among physicians overall. This trend is likely to continue to worsen as Hispanic and black students continue to be underrepresented in college and medical school.[138] Additionally, in part due to the competitive nature of dermatology, only 3% of Hispanic medical students and 7% of black medical students apply to dermatology for residency. In data compiled by the Electronic Residency Application Service in 2015, dermatology ranked 35th out of 47 specialties in terms of attracting a diverse applicant pool.[139]

This lack of a diverse dermatology workforce is troubling as studies have shown that race concordance between physicians and patients improves patient care and patient satisfaction.[140] Studies have confirmed that minority physicians are more likely to serve their own ethnic group, care for poor patients, and care for patients with Medicaid, thus improving the access to care that plagues many of these subpopulations.[141,142] Efforts to increase diversity in dermatology are underway, including making diversity an explicit goal in the dermatology selection process, working to improve exposure to dermatology in medical school, and encouraging implicit bias training for residency selection committees.[139,143–147]

Conclusions

Pigmentation of skin and hair impacts the structure, function, response to insult, and choices individuals make regarding their skin and hair health and care. More research is needed to better elucidate disease pathophysiology as it affects skin of color and to optimize management of skin and hair conditions in all populations. Dermatology training must ensure adequate exposure and education for all skin types. A diverse workforce of dermatologists is critical to appropriately caring for the diverse population that we live in.

References

1. Witzig R. The medicalization of race: scientific legitimization of a flawed social construct. *Ann Intern Med* 1996;125:675.
2. Rivara FP, Finberg L. Use of the terms race and ethnicity. *Arch Pediatr Adolesc Med* 2001;155:119.
3. Amberger J, Bocchini CA, Scott AF, et al. McKusick's Online mendelian inheritance in man (OMIM(R)). *Nucleic Acids Res* 2009;37:D793-D796.
4. King RA, Rotter JI, Motulsky AG. eds. *The genetic basis of common diseases.* 2nd ed. Oxford, England; New York. *NY: Oxford University Press* 2002;2:39-50.
5. Silver SE. Skin color is not the same thing as race. *Arch Dermatol* 2004;140.
6. Williams HC. Have you ever seen an Asian/Pacific islander? *Arch Dermatol* 2002;138.
7. He SY, McCulloch CE, Boscardin WJ, et al. Self-reported pigmentary phenotypes and race are significant but incomplete predictors of Fitzpatrick skin phototype in an ethnically diverse population. *J Am Acad Dermatol* 2014;71:731-737.
8. Fitzpatrick TB. The validity and practicality of sun-reactive skin types I through VI. *Arch Dermatol* 1988;124:869.
9. Pichon LC, Landrine H, Corral I, et al. Measuring skin cancer risk in African Americans: is the Fitzpatrick Skin Type Classification Scale culturally sensitive? *Ethn Dis* 2010;20:174-179.
10. Taylor SC. Skin of color: biology, structure, function, and implications for dermatologic disease. *J Am Acad Dermatol* 2002;46:S41-S62.
11. Rawlings AV. Ethnic skin types: are there differences in skin structure and function? *Int J Cosmet Sci* 2006;28:79-93.
12. Toda K, Pathak MA, Parrish JA, et al. Alteration of racial differences in melanosome distribution in human epidermis after exposure to ultraviolet light. *Nat New Biol* 1972;236:143-145.
13. Szabó G, Gerald AB, Pathak MA, et al. Racial differences in the fate of melanosomes in human epidermis. *Nature* 1969;222:1081-1082.
14. Johnson Jr BL, Moy RL, White GM, eds. *Ethnic Skin: Medical and Surgical.* St. Louis, Missouri: Mosby; 1998;3-5.
15. Olson RL, Gaylor J, Everett MA. Skin color, melanin, and erythema. *Arch Dermatol* 1973;108:541-544.
16. Montagna W, Carlisle K. The architecture of black and white facial skin. *J Am Acad Dermatol* 1991;24:929-937.
17. Goldschmidt H, Raymond JZ. Quantitative analysis of skin color from melanin content of superficial skin cells. *J Forensic Sci* 1972;17:124-131.
18. Alaluf S, Heath A, Carter N, et al. Variation in melanin content and composition in type V and VI photoexposed and photoprotected human skin: the dominant role of DHI. *Pigment Cell Res* 2001;14:337-347.
19. Alaluf S, Atkins D, Barrett K, et al. Ethnic variation in melanin content and composition in photoexposed and photoprotected human skin. *Pigment Cell Res* 2002;15:112-118.
20. Alaluf S, Barrett K, Blount M, et al. Ethnic variation in tyrosinase and TYRP1 expression in photoexposed and photoprotected human skin. *Pigment Cell Res* 2003;16:35-42.
21. Ancans J, Tobin DJ, Hoogduijn MJ, et al. Melanosomal pH controls rate of melanogenesis, eumelanin/phaeomelanin ratio and melanosome maturation in melanocytes and melanoma cells. *Exp Cell Res* 2001;268:26-35.
22. Watabe H, Valencia JC, Yasumoto K, et al. Regulation of tyrosinase processing and trafficking by organellar pH and by proteasome activity. *J Biol Chem* 2004;279:7971-7981.
23. Fuller BB, Spaulding DT, Smith DR. Regulation of the catalytic activity of preexisting tyrosinase in black and Caucasian human melanocyte cell cultures. *Exp Cell Res* 2001;262:197-208.
24. Scott G, Deng A, Rodriguez-Burford C, et al. Protease-activated receptor 2, a receptor involved in melanosome transfer, is upregulated in human skin by ultraviolet irradiation. *J Invest Dermatol* 2001;117:1412-1420.
25. Weigand DA, Haygood C, Gaylor JR. Cell layers and density of Negro and Caucasian stratum corneum. *J Invest Dermatol* 1974;62:563-568.
26. Johnson LC, Corah NL. Racial differences in skin resistance. *Science* 1963;139:766-767.
27. Corcuff P, Lotte C, Rougier A, et al. Racial differences in corneocytes. A comparison between black, white and oriental skin. *Acta Derm Venereol* 1991;71:146-148.
28. Roberts D, Marks R. The determination of regional and age variations in the rate of desquamation: a comparison of four techniques. *J Invest Dermatol* 1980;74:13-16.
29. Sugino K, Imokawa G, Maibach HI. Ethnic difference of varied stratum corneum function in relation to stratum corneum lipids. *J Dermatol Sci* 1993;6:108.
30. Jungersted JM, Høgh JK, Hellgren LI, et al. Ethnicity and stratum corneum ceramides. *Br J Dermatol* 2010;163:1169-1173.
31. Berardesca E, Pirot F, Singh M, et al. Differences in stratum corneum pH gradient when comparing white Caucasian and black African-American skin. *Br J Dermatol* 1998;139:855-857.
32. Berardesca E, Maibach HI. Racial differences in sodium lauryl sulphate induced cutaneous irritation: black and white. *Contact Dermatitis* 1988;18:65-70.

33. Warrier A, Kligman AM, Harper R, et al. Wickett R. A comparison of black and white skin using noninvasive methods. *J Soc Cosmet Chem* 1996;47:229-240.
34. Rebora A, Guarrera M. Racial differences in experimental skin infection with Candida albicans. *Acta Derm Venereol* 1988;68:165-168.
35. Whitmore SE, Sago NJ. Caliper-measured skin thickness is similar in white and black women. *J Am Acad Dermatol* 2000;42:76-79.
36. Berardesca E, de Rigal J, Leveque JL, et al. In vivo biophysical characterization of skin physiological differences in races. *Dermatologica* 1991;182:89-93.
37. Johnson LC, Landon MM. Eccrine sweat gland activity and racial differences in resting skin conductance. *Psychophysiology* 1965;1: 322-329.
38. Montagna W, Parakkal PF. *The Structure and Function of Skin.* New York. *NY: Academic Press* 1974;1:89-90.
39. McCance RA, Purohit G. Ethnic differences in the response of the sweat glands to pilocarpine. *Nature* 1969;221:378-379.
40. McCance RA, Rutishauser IH, Knight HC. Response of sweat glands to pilocarpine in the Bantu of Uganda. *Lancet* 1968;1:663-665.
41. Janes CL, Worland J, Stern JA. Skin potential and vasomotor responsiveness of black and white children. *Psychophysiology* 1976;13:523-527.
42. Juniper K, Dykman RA. Skin resistance, sweat-gland counts, salivary flow, and gastric secretion: age, race, and sex differences, and intercorrelations. *Psychophysiology* 1967;4:216-222.
43. Kawahata A, Sakamoto H. Some observations on sweating of the Aino. *Jpn J Physiol* 1951;2:166-169.
44. Thomson ML. A comparison between the number and distribution of functioning eccrine sweat glands in Europeans and Africans. *J Physiol* 1954;123:225-233.
45. Champion RH. ed. *An Introduction to the Biology of the Skin.* Oxford. *England: Blackwell Scientific* 1970;1:184-196.
46. Kligman AM, Shelley WB. An investigation of the biology of the human sebaceous gland. *J Invest Dermatol* 1958;30:99-125.
47. Pochi PE, Strauss JS. Sebaceous gland activity in black skin. *Dermatol Clin* 1988;6:349-351.
48. Wickrema Sinha AJ, Shaw SR, Weber DJ. Percutaneous absorption and excretion of tritium-labeled diflorasone diacetate, a new topical corticosteroid in the rat, monkey and man. *J Invest Dermatol* 1978;71:372-377.
49. Guy RH, Tur E, Bjerke S, et al. Are there age and racial differences to methyl nicotinate-induced vasodilatation in human skin? *J Am Acad Dermatol* 1985;12:1001-1006.
50. Wedig JH, Maibach HI. Percutaneous penetration of dipyrithione in man: effect of skin color (race). *J Am Acad Dermatol* 1981;5:433-438.
51. Stoughton RB. Bioassay of antimicrobials: a method for measuring penetration of agents into human skin. *Arch Dermatol* 1970;101:160.
52. Berardesca E, Maibach HI. Racial differences in pharmacodynamic response to nicotinates in vivo in human skin: black and white. *Acta Derm Venereol* 1990;70:63-66.
53. Berardesca E, Maibach H. Racial differences in skin pathophysiology. *J Am Acad Dermatol* 1996;34:667-672.
54. Gean CJ, Tur E, Maibach HI, et al. Cutaneous responses to topical methyl nicotinate in black, oriental, and Caucasian subjects. *Arch Dermatol Res* 1989;281:95-98.
55. Schwartz L, Tulipa L, Birmingham D. *Occupational diseases of the skin.* 3rd ed. PA: Lea & Febiger; 1939;2:83-95.
56. Frosch PJ, Kligman AM. The chamber-scarification test for irritancy. *Contact Dermatitis* 1976;2:314-324.
57. Marshall J, Heyl T. Skin diseases in Western Cape Province. *Afr Med J* 1963;37:1308.
58. Marshall J. New skin diseases in Africa. *Trans St Johns Hosp Dermatol Soc* 1970;56:3-10.
59. Clark M. Skin assessment in dark pigmented skin: a challenge in pressure ulcer prevention. *Nurs Times* 2010;106:16-17.
60. Kompaore F, Marty JP, Dupont C. In vivo evaluation of the stratum corneum barrier function in blacks, Caucasians and Asians with two noninvasive methods. *Skin Pharmacol* 1993;6:200-207.
61. Robinson MK. Population differences in acute skin irritation responses. Race, sex, age, sensitive skin and repeat subject comparisons. *Contact Dermatitis* 2002;46:86-93.
62. Pinnagoda J, Tupker RA, Agner T, et al. Guidelines for transepidermal water loss (TEWL) measurement. A report from the Standardization Group of the European Society of Contact Dermatitis. *Contact Dermatitis* 1990;22:164-178.
63. De Luca R, Balestrieri A, Dinle Y. [Measurement of cutaneous evaporation. 6. Cutaneous water loss in the people of Somalia]. Boll Della Soc Ital Biol Sper 1983;59:1499-1501 [in Italian].
64. Reed JT, Ghadially R, Elias PM. Skin type, but neither race nor gender, influence epidermal permeability barrier function. *Arch Dermatol* 1995;131:1134-1138.
65. 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;13:16-19.
66. Jourdain R, de Lacharrière O, Bastien P, et al. Ethnic variations in self-perceived sensitive skin: epidemiological survey. *Contact Dermatitis* 2002;46:162-169.
67. Kaidbey KH, Agin PP, Sayre RM, et al. Photoprotection by melanin—a comparison of black and Caucasian skin. *J Am Acad Dermatol* 1979;1: 249-260.
68. Tadokoro T, Kobayashi N, Zmudzka BZ, et al. UV-induced DNA damage and melanin content in human skin differing in racial/ethnic origin. *FASEB J* 2003;17:1177-1179.
69. de Winter S, Vink AA, Roza L, et al. Solar-simulated skin adaptation and its effect on subsequent UV-induced epidermal DNA damage. *J Invest Dermatol* 2001;117:678-682.
70. Bonnet Duquennoy M, Lachmann N, Noblesse E, et al. Influence of UV exposure on DNA damage in Chinese skin: abstracts. *Int J Cosmet Sci* 2008;27:69-73.
71. Vernall DG. A study of the size and shape of cross sections of hair from four races of men. *Am J Phys Anthropol* 1961;19:345-350.
72. Brauner GJ. Cutaneous disease in black children. *Am J Dis Child* 1983;137:488-496.
73. Sperling LC. Hair density in African Americans. *Arch Dermatol* 1999;135:656-658.
74. Hrdy D, Baden HP. Biochemical variation of hair keratins in man and non-human primates. *Am J Phys Anthropol* 1973;39:19-24.
75. Gold RJ, Scriver CG. The amino acid composition of hair from different racial origins. *Clin Chim Acta Int J Clin Chem* 1971;33:465-466.
76. Davis SA, Narahari S, Feldman SR, et al. Top dermatologic conditions in patients of color: an analysis of nationally representative data. *J Drugs Dermatol* 2012;11:466-473.
77. Halder RM, Bridgeman-Shah S. Skin cancer in African Americans. *Cancer* 1995;75:667-673.
78. 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: 246-252.
79. Chung CL, Nadhan KS, Shaver CM, et al. Comparison of posttransplant dermatologic diseases by race. *JAMA Dermatol* 2017;153:552.
80. Howlader N, noone AM, Krapcho M, et al, eds. SEER Cancer Statistics Review. Bethesda, MD: National Cancer Institute; 1975-2016..based on November 2018 SEER data submission, posted to SEER web site, April 2019https://seer.cancer.gov/csr/1975_2016/.
81. El-Domyati M, Attia S, Saleh F, et al. Intrinsic aging vs. photoaging: a comparative histopathological, immunohistochemical, and ultrastructural study of skin. *Exp Dermatol* 2002;11:398-405.
82. Halder RM. The role of retinoids in the management of cutaneous conditions in blacks. *J Am Acad Dermatol* 1998;39(2 Pt 3):S98-103.
83. Halder RM, Grimes PE, McLaurin CI, et al. Incidence of common dermatoses in a predominantly black dermatologic practice. *Cutis* 1983;32:388,390.
84. Tomita Y, Maeda K, Tagami H. Mechanisms for hyperpigmentation in postinflammatory pigmentation. *urticaria pigmentosa and sunburn Dermatologica* 1989;179(suppl 1):49-53.

85. Grimes PE, Stockton T. Pigmentary disorders in blacks. *Dermatol Clin* 1988;6:271-281.
86. Cosman B, Crikelair G, Ju DM, et al. The surgical treatment of keloids. *Plast Reconstr Surg* 1961;27:335-358.
87. Ketchum LD. Hypertrophic scars and keloids. *Clin Plast Surg* 1977;4: 301-310.
88. Oluwasanmi JO. Keloids in the African. *Clin Plast Surg* 1974;1:179-195.
89. Alhady SM, Sivanantharajah K. Keloids in various races. A review of 175 cases. *Plast Reconstr Surg* 1969;44:564-566.
90. Sherertz E, Schwartz S. Patch test interpretation on black skin. *Dermatitis* 1993;4:247-248.
91. Dickel H, Taylor JS, Evey P, et al. Comparison of patch test results with a standard series among white and black racial groups. *Am J Contact Dermat* 2001;12:77-82.
92. Deleo VA, Alexis A, Warshaw EM, et al. The association of race/ethnicity and patch test results: North American Contact Dermatitis Group, 1998-2006. *Dermatitis* 2016;27:288-292.
93. Henderson MD, Abboud J, Cogan CM, et al. Skin-of-color epidemiology: a report of the most common skin conditions by race: skin-of-color epidemiology. *Pediatr Dermatol* 2012;29:584-589.
94. Taylor SC, Cook-Bolden F, Rahman Z, et al. Acne vulgaris in skin of color. *J Am Acad Dermatol* 2002;46(2 suppl understanding):S98-S106.
95. Plewig G, Fulton JE, Kligman AM. Pomade acne. *Arch Dermatol* 1970;101:580-584.
96. Schachner L, Ling NS, Press S. A statistical analysis of a pediatric dermatology clinic. *Pediatr Dermatol* 1983;1:157-164.
97. Child FJ, Fuller LC, Higgins EM. Du Vivier AW. A study of the spectrum of skin disease occurring in a black population in south-east London. *Br J Dermatol* 1999;141:512-517.
98. Janumpally SR, Feldman SR, Gupta AK, et al. In the United States, blacks and Asian/Pacific Islanders are more likely than whites to seek medical care for atopic dermatitis. *Arch Dermatol* 2002;138: 634-637.
99. Horii KA, Simon SD, Liu DY, et al. Atopic dermatitis in children in the United States, 1997-2004: visit trends, patient and provider characteristics, and prescribing patterns. *Pediatrics* 2007;120:e527-e534.
100. Blomberg M, Rifas-Shiman S, Camargo C, et al. Differences in incidence and persistence of atopic dermatitis by race/ethnicity. *Dermatol Online J* 2016;22:9.
101. Bhattacharya T, Silverberg JI. Efficacy of systemic treatments for atopic dermatitis in racial and ethnic minorities in the United States. *JAMA Dermatol* 2014;150:1232.
102. Lindsey SF, Tosti A. Ethnic hair disorders. In: Ioannides D, Tosti A, eds. *Current Problems in Dermatology.* S. Karger AG; 2015. p. 139-149.
103. Wone I, Tal-Dia A, Diallo OF, et al. Prevalence of the use of skin bleaching cosmetics in two areas in Dakar (Sénégal). *Dakar Med* 2000;45:154-157. [in French].
104. Verma SB. Obsession with light skin—shedding some light on use of skin lightening products in India. *Int J Dermatol* 2010;49:464-465.
105. Ajose FOA. Consequences of skin bleaching in Nigerian men and women. *Int J Dermatol* 2005;44(suppl 1):41-43.
106. Findlay GH. Ochronosis following skin bleaching with hydroquinone. *J Am Acad Dermatol* 1982;6:1092-1093.
107. Hoshaw RA, Zimmerman KG, Menter A. Ochronosislike pigmentation from hydroquinone bleaching creams in American blacks. *Arch Dermatol* 1985;121:105-108.
108. Levitt J. The safety of hydroquinone: a dermatologist's response to the 2006 Federal Register. *J Am Acad Dermatol* 2007;57:854-872.
109. Camarasa JG, Serra-Baldrich E. Exogenous ochronosis with allergic contact dermatitis from hydroquinone. *Contact Dermatitis* 1994;31:57-58.
110. McGregor D. Hydroquinone: an evaluation of the human risks from its carcinogenic and mutagenic properties. *Crit Rev Toxicol* 2007;37:887-914.
111. Olumide YM, Akinkugbe AO, Altraide D, et al. Complications of chronic use of skin lightening cosmetics. *Int J Dermatol* 2008;47:344-353.
112. Otley CC, Sober A. Over-the-counter clobetasol propionate. *Arch Dermatol* 1994;130:121.
113. Nnoruka E, Okoye O. Topical steroid abuse: its use as a depigmenting agent. *J Natl Med Assoc* 2006;98:934-939.
114. Engler DE. Mercury 'bleaching' creams. *J Am Acad Dermatol* 2005;52: 1113-1114.
115. Goeckermann WH. A peculiar discoloration of the skin: probably resulting from mercurial compounds (calomel) in proprietary face creams. *J Am Med Assoc* 1922;79:605.
116. Look KM, Look RM. Skin scraping, cupping, and moxibustion that may mimic physical abuse. *J Forensic Sci* 1997;42:103-105.
117. Stoner JG. Miscellaneous dermatitis-inducing plants. *Clin Dermatol* 1986;4:94-101.
118. Carter-Pokras O, Baquet C. What is a 'health disparity. *Public Health Rep* 2002;117:426-434.
119. Friedman LC, Bruce S, Weinberg AD, et al. Early detection of skin cancer: racial/ethnic differences in behaviors and attitudes. *J Cancer Educ* 1994;9:105-110.
120. Stern RS. Dermatologists and office-based care of dermatologic disease in the 21st century. *J Investig Dermatol Symp Proc* 2004;9: 126-130.
121. Roetzheim RG, Pal N, Tennant C, et al. Effects of health insurance and race on early detection of cancer. *J Natl Cancer Inst* 1999;91:1409-1415.
122. Buster KJ, You Z, Fouad M, et al. Skin cancer risk perceptions: a comparison across ethnicity, age, education, gender, and income. *J Am Acad Dermatol* 2012;66:771-779.
123. Bilimoria KY, Balch CM, Wayne JD, et al. Health care system and socioeconomic factors associated with variance in use of sentinel lymph node biopsy for melanoma in the United States. *J Clin Oncol* 2009;27: 1857-1863.
124. Kostecki J. Dermatology Practice Profile Survey 2009 Report. *American Academy of Dermatology* 2009;3.
125. Harvey V, Paul J, Boulware E. Racial and ethnic disparities in dermatology office visits among insured patients, 2005-2010. *J Health Disparities Res Pract* 2016;9.
126. Charrow A, Xia FD, Joyce C, et al. Diversity in dermatology clinical trials: a systematic review. *JAMA Dermatol* 2017;153:193.
127. Harvey VM, Ozoemena U, Paul J, et al. Patient-provider communication, concordance, and ratings of care in dermatology: results of a cross-sectional study. *Dermatol Online J* 2016;22.
128. Shaw MG, Morrell DS, Corbie-Smith GM, et al. Perceptions of pediatric clinical research among African American and Caucasian parents. *J Natl Med Assoc* 2009;101:900-907.
129. Adams-Campbell LL, Ahaghotu C, Gaskins M, et al. Enrollment of African Americans onto clinical treatment trials: study design barriers. *J Clin Oncol* 2004;22:730-734.
130. Advani AS, Atkeson B, Brown CL, et al. Barriers to the participation of African-American patients with cancer in clinical trials: a pilot study. *Cancer* 2003;97:1499-1506.
131. Spears CR, Nolan BV, O'Neill JL, et al. Recruiting underserved populations to dermatologic research: a systematic review: recruiting underserved populations to dermatologic research. *Int J Dermatol* 2011;50: 385-395.
132. Hansra NK, O'Sullivan P, Chen CL, et al. Medical school dermatology curriculum: are we adequately preparing primary care physicians? *J Am Acad Dermatol* 2009;61:23-29.
133. McCleskey PE, Gilson RT, DeVillez RL. Medical Student Core Curriculum in Dermatology Survey. *J Am Acad Dermatol* 2009;61:30-35.
134. Knable A, Hood AF, Pearson TG. Undergraduate medical education in dermatology: report from the AAD Interdisciplinary Education Committee, Subcommittee on Undergraduate Medical Education. *J Am Acad Dermatol* 1997;36:467-470.
135. Ramsay DL, Mayer F. National survey of undergraduate dermatologic medical education. *Arch Dermatol* 1985;121:1529-1530.
136. Buster KJ, Stevens EI, Elmets CA. Dermatologic health disparities. *Dermatol Clin* 2012;30:53-59.

137. Nijhawan RI, Jacob SE, Woolery-Lloyd H. Skin of color education in dermatology residency programs: does residency training reflect the changing demographics of the United States? *J Am Acad Dermatol* 2008;59:615-618.
138. Pandya AG, Alexis AF, Berger TG, et al. Increasing racial and ethnic diversity in dermatology: a call to action. *J Am Acad Dermatol* 2016;74:584-587.
139. Van Voorhees AS, Enos CW. Diversity in dermatology residency programs. *J Investig Dermatol Symp Proc* 2017;18:S46-S49.
140. Cooper LA, Roter DL, Johnson RL, et al. Patient-centered communication, ratings of care, and concordance of patient and physician race. *Ann Intern Med* 2003;139:907-915.
141. Komaromy M, Grumbach K, Drake M, et al. The role of black and Hispanic physicians in providing health care for underserved populations. *N Engl J Med* 1996;334:1305-1310.
142. Marrast LM, Zallman L, Woolhandler S, et al. Minority physicians' role in the care of underserved patients: diversifying the physician workforce may be key in addressing health disparities. *JAMA Intern Med* 2014;174:289.
143. Lester J, Wintroub B, Linos E. Disparities in academic dermatology. *JAMA Dermatol* 2016;152:878-879.
144. Chen A, Shinkai K. Rethinking how we select dermatology applicants-turning the tide. *JAMA Dermatol* 2017;153:259-260.
145. Granstein RD, Cornelius L, Shinkai K. Diversity in dermatology-a call for action. *JAMA Dermatol* 2017;153:499-500.
146. McKesey J, Berger TG, Lim HW, et al. Cultural competence for the 21st century dermatologist practicing in the United States. *J Am Acad Dermatol* 2017;77:1159-1169.
147. Imadojemu S, James WD. Increasing African American representation in dermatology. *JAMA Dermatol* 2016;152:15-16.