# Skin of colour: Characteristics and disease

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## Abstract

Skin colour varies from pale white to very dark. Fitzpatrick's skin phototypes are based on the person's skin colour and its response to sun exposure in terms of burning and tanning of the skin. Fitzpatrick's type 1V-V1 is known as the skin of colour and type 1-111 is the fair or white skin. The colour of the skin and texture of the hair are the most apparent phenotype to differentiate the different races; this correlates closely with the geography and ultraviolet radiation of the sun.

There are notable differences in skin disease incidence, presentation, and treatment based on skin type. Differences in skin anatomy and physiology between the fair skin and the skin of colour may explain disparities in skin disorders and provide insight into appropriate differences in the management of cutaneous disease. Differences in culture and habits may produce skin lesions unknown to the local physicians. Temperature, humidity and rainfall are closely interwoven with the fauna and flora of the area. Hot and humid climate favours bacterial and fungal infections.

Today in this multicultural society due to globalization, a physician has to see patients from all over the globe. There is a need for the physicians to know the diseases of people from different racial and ethnic backgrounds for early diagnosis and treatment.


**Keywords:** Skin of colour, Characteristics, Disease, Changing demographics.

## Introduction

The colour of the skin depends mainly upon the density and distribution of melanin, the pigment which protects the skin from ultraviolet radiation (UVR) of the sun. The number of melanocytes in the skin is the same in all races; it is the amount and distribution of melanin that gives the skin its colour. The other chromophores of the skin are oxyhaemoglobin, reduced haemoglobin and carotene. The greater the melanin content of the skin, the darker is the skin colour. It is not only the melanin content which differentiates the people of colour, there are differences in the structure of the epidermis and dermis due to which they have certain specific skin diseases and the response to some dermatoses also differ.

Skin colour constitute a wide variety of racial and ethnic groups including Africans, African Americans, Afro-Caribbean's (Afro-Caribbeans), it also consists of certain races of a slightly lighter colour such as the Indians, Pakistanis, Arabs and Hispanics. There is not enough data on the structure, physiology and disease pattern in the skin of colour. Studies are done on small group of patients; and the studies that exist often have methodological flaws.[1]

The skin is the main organ of the body that is exposed to ultraviolet radiation of the sun. Melanin protects the skin from UVR and so the content of melanin is high in regions where the skin is exposed to increased intensity of UVR. As human beings migrated from out of Africa to areas where the UVR is low, their melanin content decreased and the skin took a lighter tone. Skin pigmentation has a definite correlation with latitudes. Near the equator where the UVR is highest, natural selection favours a darker skin, at high latitudes the fairer skin is prominent.[2]

## Fitzpatrick's Skin Phototypes

The Fitzpatrick skin type classification is based on a person's reaction to sun exposure. Types 1-111 are known as the white or fair skin, and types 1V -V1 are the dark skin or the skin of colour.[1,3]

## Variations in Structure and Function

(Throughout the article Fitzpatrick's Type V1 is used to describe the skin of colour, and Type 1 for the fairer skin).

**Fitzpatrick's Skin Phototypes.**

| Fitzpatrick's Skin phototypes | Reaction to sun exposure | Skin colour |
|---|---|---|
| 1 | Burns easily, does not tan | Pale white skin with reddish tinge |
| 11 | Burns easily, tans minimally | Pale white |
| 111 | Burns minimally, tans moderately | Pale white with beige tinge |
| 1V | Tans easily, burns minimally | Light brown |
| V | Tans profusely, rarely burns | Moderate brown |
| V1 | Tans profusely, does not burn | Dark brown/black |

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## Epidermis

The epidermis is the outermost and major protective layer of the skin. It is multilayered, that renews itself continuously by cell division in its deepest layer the stratum germinativum, or the basal layer. The epidermis is devoid of blood vessels and lymphatics, it derives its nutrition from the dermis, from which it is separated by the basement membrane. The stratum corneum is the outermost layer of the epidermis which is primarily responsible for the barrier properties of the skin. The main cells of the epidermis are the keratinocytes, these constitute about 95% of the epidermal cells, which consist of the stratum germinativum, stratum malpighii, stratum granulosum, stratum corneum and stratum lucidum in the palms and soles and . The other cells of the epidermis are the melanocytes, Langerhans cells, Merkel cells, the Granstein cells and the indeterminate cells.

The epidermal appendages constitute eccrine glands, apocrine glands, apoeccrine glands, sebaceous glands, the pilosebaceous unit, nails and hair. Embryologically they originate as downgrowths from the epidermis. Each appendage has its own special function; they can also serve as reserve media for regeneration when required.

**Stratum Corneum:** Stratum corneum is the outermost layer of the epidermis which is in direct contact with the external environment. The barrier properties of the skin reside in the outer 15 μm of the stratum corneum, which was previously regarded as rather inert or even dead, because the cells are anucleated and completely keratinized. It consists of protein rich corneocytes, embedded in a lipid matrix. The lipids are rich in cholesterol, ceramides and fatty acids. Alterations in the lipids are responsible for a number of skin diseases such as atopic dermatitis, ichthyosis, psoriasis and senile xerosis.

The thickness of the stratum corneum is the same in all races. The cells in the skin of colour are more compact due to greater intercellular cohesion and have more cell layers. In a fair skin 6-15 tape strips are needed to completely remove the stratum corneum, in the skin of colour 8-25 tape strips are required.[1] Due to greater cohesion in black skin, vesicles and bullae remain intact longer for similar lesions in the fairer skin.[3]

Transepidermal Water Loss (TEWL): TEWL represents the water barrier function of the skin. Skin maintains an effective barrier against loss of body fluids and absorption of externally applied substances. Water is lost from the skin through sweat, and by passive diffusion. TEWL through the stratum corneum and skin appendages under non-sweating conditions is the imperceptible water loss.[4] It is a true reflection of stratum corneum barrier function, when there is no sweat gland activity.

TEWL has been studied to predict the efficacy of therapeutic application of topical agents in skin disease. It is theoretically directly related to the ambient relative humidity and temperature. TEWL increases with the rise of skin temperature. Data regarding TEWL through the skin are conflicting, some studies indicate that TEWL depends upon the pigmentation of the skin, greater the pigmentation, greater the water loss.[5,6] Studies by Warrier et al show that TEWL is greater in the fairer skin.[7] No significant difference was found by Berardesca and Maibach.[8] It is clear that further studies have to be done to interpret the significance of measuring TEWL.

The lipid content of the black skin is somewhat higher than the white skin, this perhaps due to the greater intercellular cohesion, and difficulty in stripping off the black horny layer. This could also explain the slightly lower permeability to certain chemicals.[8]

Water Content: Water is known to play an important part in the stratum corneum barrier function. It keeps the stratum corneum supple, and prevents the cracking of the skin due to mechanical stress. It is also thought to regulate the normal desquamation of corneocytes from the skin. Proteases and protease inhibitors in the epidermis are important enzymes for desquamation.[9] Water content of the skin can be measured by many methods including skin capacitance, conduction, impedance and resistance.[4] Possible sources of error in measurements include sweat production, number of hair follicles, electrolyte content of the stratum corneum and artifacts from applied topical agents

No positive conclusion could be obtained about the water content between different races. There was low water content in the black skin in studies by Johnson et al.[10] There was no significant difference between the two phenotypes in studies by Berardesa et al.[8]

Melanin and Melanosomes: The hallmark biological feature in the people of colour is the amount and distribution of pigment melanin. Melanin besides functioning as a broadband UV absorbent also has antioxidant and radical scavenging properties.[11] The number of melanocytes is the same in all races. There is a direct correlation between the geographic distribution of UV radiation (UVR) and the distribution of indigenous skin pigmentation around the world. Areas that receive higher amounts of UVR, generally located closer to the equator, tend to have darker skin. Areas that are far from the tropics and closer to the poles have lower intensity of

UVR, is reflected in lighter skin colour. African skin transmits only 7.4% of the UVR as compared to 29.4 for Caucasoid epidermis.[2]

The number, size and aggregation of melanosomes also differ in different races. Dark skin have large, non-aggregating melanosomes, these are distributed throughout the epidermis. The fairer skin have smaller aggregated melanosomes, which are absent in the upper layers of the epidermis.[1] Large melanosomes have greater melanin content and can absorb more UVR.

## Epidermal Appendages

Studies regarding eccrine, apocrine, apoeccrine glands and sebaceous glands in the different races have given conflicting results.[1,12] Controversy is probably due to lack of well-controlled protocol, methodological flaws and small study population.[1]

Hair follicles. The hair of the Congoid skin is the most distinct phenotype characteristic of the skin of colour. Four types of hair are recognized, straight, wavy, helical and spiral. The spiral hair is characteristic of black skin. The cross section of the spiral hair has a flattened elliptical shape. Melanosomes are present in the outer root sheath and bulb of vellus hair in the dark skin. Black hair is more heavily pigmented. Elastic tissue anchorage to the hair follicle is reduced in the dark skin, resulting in greater damage with traction based alopecia.[1]

Formation of vitamin D. The epidermis synthesizes vitamin D in the presence of sunlight. It is produced in the skin from precursor 7-dehydrocholesterol. As the white skin absorbs more ultraviolet light, it has a greater capacity to form vitamin D than the black skin.[12]

Folate Deficiency: Folate deficiency has been extensively documented by analysis of human plasma, but the folate status within skin has not been widely investigated. Folate is sensitive to photolysis by exposure to UVR. Exposure to sunlight and UVR in particular is expected to lower folate levels in at least the superficial layers of the skin. Photolysis of folate stores in the skin have to be extensive to result in systemic depletion of folate. Indeed it has been reported that fair skinned patients undergoing photochemotherapy for dermatological conditions have low serum folate concentrations, suggesting that folate depletion may occur in vivo. More studies are required to determine folate levels in the skin both in normal condition and when exposed to ultraviolet light.[13]

## Dermis

The dermis is a thick layer located beneath the epidermis and above the subcutaneous layer. The dermis is composed of collagen fibers, elastic fibers, and the ground substance which comprises proteogylcans (PGs) and glycosaminogylcans (GAGs). The cellular components are the fibroblasts, macrophages, dendritic cells and the mast cells.

Collagen constitutes about 75% of the dry weight of the skin, it gives tensile strength, and protects the skin against external trauma. Collagen is the most important structural protein of the body. The elastic tissue forms a continuous network throughout the dermis and extends into the connective tissue; it is responsible for the elasticity of the skin. Elastic tissue constitutes only about 4% of the body weight. With increasing age the elastic fibers of the dermis degenerate and the skin tends to wrinkle. PGs and GAGs are molecules of the ground substance that embeds the fibrous components of dermis. These can absorb about 1000 times its own volume of water; they regulate the transmission of hormones and nutrients from the blood vessels to the cells.

Fibroblast are the key resident cells of the dermis, they are responsible for the synthesis and degradation of the fibrous and nonfibrous connective tissue proteins. Macrophages are phagocytic cells responsible for detecting, engulfing and destroying pathogens and apoptotic cells. The dermal dendrites are immunologically competent cells, they are highly phagocytic. Mast cells are special secretary cells responsible for immediate hypersensitivity reactions; they are also involved in subacute and chronic inflammatory disease. Mast cells and macrophages are intimately involved in regulating fibroblasts and thus participate in dermal changes under physiological and pathological conditions.

There is no difference in the thickness of the dermis between the skin of colour and the fair skin, differences are seen at cellular level.[1]

**Cells of the Dermis:** Fibroblasts are larger and greater in quantity in the black skin; these may be multinucleated or binucleated. Fibroblastic hyperactivity is thought to be due to the interaction between the various cells such as the mast cells and cytokines. This combined with the decreased activity of collagenase is responsible for the keloid formation in the black skin.[1]

**Mast Cells:** Most studies show that the number and size of mast cells are the same in the fair and coloured skin. Based on the finding that pruritus is more commonly observed in the black skin, a study was conducted by Sueki et al found that the granules of the mast cells were 1.5 times larger than in the white skin, they also found an increase in the distribution of protease.[14] The

**Table:** Differences between the skin of colour and fair skin.

| Characteristic | Skin of Colour | Fair Skin |
|---|---|---|
| Stratum corneum | Equal in thickness<br>Cells of the stratum corneum are compact with greater intercellular cohesion.<br>More cell layers | Equal in thickness<br>Cells less compact<br>Less cell layers |
| Melanocytes | Equal in number | Equal in number |
| Melanosomes | Large, not aggregated, present throughout the epidermis | Smaller, in aggregates, absent in the upper layers of the epidermis |
| Melanin content | Greater | Less |
| Hair | Spiral | Straight, wavy or helical |
| Follicular degeneration syndrome | ++ | - |
| Traction alopecia | ++ | - |
| Mast cells | Large granules | Smaller granules |
| Pruritus | ++ | + |
| Collagen fibers | Small and closely stacked | Larger, occasional fiber fragments present |
| Scars and keloid formation | +++ | + |
| Fibroblast | Large, more in number | Smaller, less in number |
| Vitamin D production | + | +++ |

participation of mast cells in aberrant fibrosis of skin disorders such as keloid and scars is due to the increased amounts of tryptase in the mast cells.[4]

Macrophages are larger and more numerous in the dark skin than in the fairer skin.[1]

## Connective Tissue of the Dermis

Collagen fibers: The collagen fiber bundles in black skin are closely stacked together, they are smaller and run more or less parallel to the epidermis; this is seen throughout the dermis, the fibers are more compact beneath the epidermis.[3] The collagen fiber bundles in the fair skin are larger with occasional fiber fragmentation 1. In addition fibrils and glycoprotein fragments are noted in the dermal interstices throughout the dermis in the black skin. In the white skin collagen fiber bundles are larger with occasional fragmentation.[1,15]

**Elastic Fibers:** Most studies show no significant difference in the elastic tissue in black and white skin. Warrier and Kligman demonstrated black skin had higher elastic recovery than white skin on the cheeks than on the legs; the difference was not statistically significant.[7] Elastic tissue anchorage to the hair follicle is reduced in the dark skin, resulting in greater damage with traction based alopecia.[1]

## Dermatoses that Show Racially Dependent Variations

Most diseases manifest similarly in all races; some disease differ due to the difference in the structure of the skin. Dermatoses that appear red or brown in Caucasoid skin appear black, gray or purple in the pigmented skin; pigmentation may mask an erythematous reaction. Inflammation in the pigmented skin is followed by hyperpigmentation or hypopigmentation, which is a problem while treating the dark skin with lasers and other physical modalities. Follicular, papular and annular lesions are more frequent in Afro-Caribbeans.[16]

Keloids occur in all races, more in Afro-Caribbeans. The exact incidence ratio for the Afro-Caribbeans over Caucasoids varies from 2 to 19 times according to a study by Kelly et al..[17] Keloids occur anywhere in the body but have a predilection for shoulders, ears, upper back and anterior chest. They usually follow trauma, but can occur spontaneously. In Afro-Caribbeans they may also develop in areas of scarification.[18]

Pomade acne in Afro-Caribbeans is due to the use of pomades, oils and creams on the hair; this can occur both in children and adults.[19,20]

**Contact Dermatitis:** The Afro-Caribbeans are less susceptible to irritants; this difference is not detectable if the stratum corneum is removed. It is due to increased cohesion found in the cells of the stratum corneum in the dark skin.[21]

Lichen Planus: Oral lesions are said to be uncommon in the black skin, but frequent in the Caucasoid people, while hypertrophic lichen planus, lichen planus pigmentosus, actinic variants and postinflammatory hyperpigmentation is a characteristic outcome of lichen planus in African Americans or dark skin individuals.[22,23]

**Palmoplantar Keratoderma:** Some palmoplantar keratodermas are more common in black Africans than other races. Keratosis palmaris et plantaris presents as small crateriform pits on the creases of the palms and soles, it is more frequent in Afro-Caribbean's Afro-Caribbeans.[24] Focal acral hyperkeratosis is characterized by oval or polygonal papules, with a central pigmented pit situated at the borders of the palms and soles is found exclusively in black Africans.[25]

**Dermatosis Papulosa Nigra:** These are characterized by hyperpigmented smooth-surfaced, round or filiform papules, 1-5 mm in diameter, usually on the face, sometimes seen on the neck and upper trunk affecting three fourths of the Afro-Caribbean's Afro-Caribbeans.[26,27]

**Pityriasis Rosea:** The Afro-Caribbeans show several unusual variations in the features of pityriasis rosea. An extreme inverse pattern with lesions on the face, neck, extremities and lower abdomen, rather than on the trunk is usual. In addition papular lesions can be seen on the palms and soles. The recurrence rate is also higher.[28,29]

**Sarcoidosis:** Black skin is more likely to develop cutaneous sarcoidosis. African Americans tend to develop sarcoidosis at a younger age; they are known to be at greater risk of developing severe pulmonary and cutaneous disease. Lupus pernio is associated with the worse prognosis both in systemic disease and the more chronic cutaneous lesions.[30]

**Kaposi Sarcoma:** The classical Kaposi's sarcoma is seen in Caucasoid of Jewish origin. African Kaposi sarcoma is seen as either an indolent neoplasm identical to the classic disease seen in Europe and North America or as an aggressive disease with fungating and exophytic tumours that may invade the subcutaneous and surrounding tissue including the underlying bone. Kaposi's sarcoma is also a common manifestation of AIDS. It is the most common AIDS-associated neoplasm. AIDS-related Kaposi sarcoma has an aggressive clinical course.[31] Approximately 15% of all AIDS patients develop Kaposi's sarcoma, sub-Saharan Africa harbours about three-quarters of the world's HIV-1-infected individuals.[32]

**Facial Afro-Caribbean Childhood Eruption (FACE):** This is a monomorphic flesh coloured or hypopigmented papular eruption seen on the face particularly around the mouth, eyelids and ear of Afro-Caribbean children. The eruption persists for several months and then resolves without scarring. The cause is unknown.[33,34]

**Infantile Acropustulosis:** Small intensely itchy papules appear between 2-10 months of age. The papules develop into pustules, mostly seen on the palms, soles, wrist and ankles. These clear within three months, but recur again. The disease resolves spontaneously by the age of 2-3 years. The cause is unknown. Early reports suggested a predominance of African Americans for infantile acropustulosis. Acropustulosis of infancy is now believed to affect all races equally.[35,36]

Papular Eruption in Black Males: The condition is characterized by pruritic papules with a predilection for the trunk, upper arm and postauricular areas. Condition is persistent and resistant to treatment.[37]

**Cutaneous Malignancy:** Afro-Caribbeans have the lowest incidence of melanoma, 1/70th that of Caucasoid, this is because the dark skin is well protected against UVR.[38] Squamous cell carcinoma is most common cancer as is basal cell carcinoma in Caucasoid.[39] Basal cell carcinoma often is pigmented, squamous cell carcinoma occurs in areas of chronic scarring and inflammation, and melanoma presents in non-sun-exposed areas, such as the soles and nail beds.

The Afro-Caribbeans also age less (extrinsic ageing) due to the protection they have from UVR by melanin pigment.

## Hair Disorders

Persons of African descent have the greatest degree of curl and increased fragility. The hair is spiral, tightly curled; dry and a few elastic fibers adhere to it which is responsible for a number of specific hair disorders.

Central centrifugal cicatricial alopecia (CCCA) is a term coined by the North American Hair Research Society (NAHRS) to describe a scarring hair loss, present on the vertex of the scalp that spreads peripherally. It is almost exclusively used to describe hair loss in African American women and replaces previously used terms such as hot comb alopecia and follicular degeneration syndrome.[40,41] Hot-combing was a method of straightening curled hair; it is now replaced by chemical methods. The hot-comb caused traction alopecia.[38] Follicular degeneration syndrome (FDS) was the term used for a distinct form of scarring alopecia due to premature desquamation of the inner root sheaths below the isthmus.[42]

Pseudofolliculitis barbae and acne keloidalis nuchae. This is due to inward growth of the spiral hair. When the hair is shaved the curved sharpened end of the hair re-enters the skin, causing a foreign body reaction and setting up an inflammatory process. The beard area is affected in pseudofolliculitis barbae and the occipital scalp in acne keloidalis nuchae. It can occur in any area that is shaved

such as the pubic area 38. The condition is prevalent in people of African ancestry.[43]

**Traction Alopecia:** This is often seen in Afro-Caribbeans due to the practice of plaiting or multiple braiding their hair. This is done by using tight rollers or picking the hair with a hard comb to create the African-hairstyle. Hair when pulled from its follicles gets inflamed, often followed by atrophy. The site of atrophy depends upon the pattern of braiding; it is generally seen on the temporal region of the scalp.[38] Traction alopecia is also seen in Sikh males due to constant wearing of the turban.[44]

**Dissecting Cellulitis of the Scalp:** This is a chronic suppurative scalp disorder mostly seen in Afro-Caribbean males. Painful boggy swellings are seen on the scalp, which are connected to one another by intercommunicating sinuses, as the disease progresses, scarring and alopecia result.[38,45]

## Variations of Normal Pigmentation

The normal physiological pigmentation differs in different races; which can be a diagnostic challenge to physicians if they are not aware of them. Pigmentary demarcation lines (PDLs) are areas of an abrupt transition from hyperpigmented to hypopigmented or normal skin colour. The aetiology of PDL is unknown, racial differences in PDLs may be related to genetic variations.[46]

**Futcher's or Voight's Lines:** These are physiological, abrupt transitions from deeper pigmented skin to lighter pigmented skin. Naturally occurring Voight's lines are described in the dark skin, these are on the lateral aspect of the upper arm extending over the pectoral area, on the posteromedial portion of the lower limb, a vertical hypopigmented line in the pre and para-sternal area, on the posteromedial aspect of the spine, and bilateral hypopigmented streaks, bands or lanceolate areas over the chest between the middle third of the clavicle and the periareola skin. The lines are symptomless. The pigmentation is proportional to the degree of pigmentation.[47,48] About 75% of Afro-Caribbeans have these lines.[49]

**Hyperpigmentation of the Palms and Soles:** These are discrete ill-defined or mottled macular pigmentation on the palms and soles of African people.[47,50]

**Midline Pigmentation:** These appear as a line or band of hypopigmentation or as oval macules on the anterior chest and midsternal area. The pigmentation can extend to the abdomen or the neck.[51] Linea nigra is more common in the women of colour.

**Nail Pigmentation:** There is no difference in the structure of the nails in different races, the frequency of melanonychia varies by the degree of skin pigmentation, There is a band of pigmentation on the nail plate in people of colour, extending from the nail matrix to the tip of the nail, it is absent in the fairer skin. It is due to increased formation of melanin by melanocytes in the nail matrix.[52] Among African-American patients, melanonychia affects up to 77% of young adults and almost 100% of those older than 50 years of age.[53] The condition should not be mistaken for a melanoma.

Oral Pigmentation: Oral macular physiological pigmentation is seen mostly on the gingiva; it may also involve the hard palate, buccal mucosa, and tongue.[54] Diffuse and bilateral physiologic pigmentation common in African, Asian and Mediterranean populations is due to greater melanocyte activity rather than a greater number of melanocytes. Physiologic pigmentation develops during the first 2 decades of life but may not come to the patient's attention until later.[55]

## Geographical Location and Climate

Macroclimate in the true geographical sense refers to the temperature, humidity and rainfall of a given area. These factors have a decided effect on the fauna and flora. The hot and humid climate predisposes to bacterial and fungal infections. In the flat lands of the savannah, deep mycoses, is of frequent occurrence. The heat combined with low humidity of the arid and semi-arid areas favour the development of coccidioidomycosis, desert sores and cutaneous diphtheria. Cooler temperatures and high altitudes favour skin disorders such as asteatotic eczema and frostbite.[56] Filariasis, onchocerciasis, loiasis, are found mainly in Africa. Human schistosomiasis causes severe morbidity second only to malaria, is found in sub-Saharan Africa. Mucocutaneous leishmaniasis is found in Central and South America. India has the highest incidence of leprosy.[57] Most cases of Lyme disease are seen in the United States. Due to globalization the demographics of the epidemiology of disease pattern is changing.

## Conclusion

The structure of the skin differs in the fair skin and skin of colour; it provides important biological markers for diagnosing skin disease. Demographics are changing all over the world, more so in industrialized nations. The demographics of the United States has changed dramatically, about 30% of the population of United States are mostly immigrants and people of colour. It is important that the general practitioner's should have knowledge of the diseases of different races, geographical background, culture and environment for early diagnosis

and treatment.

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## References


1. Taylor SC. Skin of colour: Biology, structure, function and implication for dermatologic disease. J Am Acad Dermatol 2002; 46: S41-62
2. Kaidley KH, Agin PP, Savre RM, Klihman AM. Photoprotection by melanin-a comparison between Black and Caucasian skin. J Am Acad Dermatol 1979; 1: 249-60
3. Czerkasij V. Skin of colour: A basic outline of unique differences. Nurse Pract 2013; 38: 34-40
4. Wesley NO, Maibach HJ. Racial differences in skin properties. Am J Clin Dermatol 2003; 4: 843-60
5. Wilson D, Berardesca E, Maibach HI. In vitro transepidermal water loss differences between black and white human skin. Br J Dermatol1988; 199: 647-52
6. Kompoare F, Marly JP, Dupont C. In vivo evaluation of the stratum corneum barrier functions in Blacks, Caucasians and Asians with two noninvasive methods. Skin Pharmacol 1993; 6: 200-7
7. Warrier AG, KligmanAM, Harper RA, Bowman J, Wickett RR. A comparison between black and white skin using non-invasive methods. J Soc Cosmetic Chem 1996; 47: 229-40
8. Berardesca E, Maibach HI. Racial differences in sodium laurate induced cutaneous irritation: black and white skin. Contact Dermatitis 1988; 18: 65-70
9. Hadgraft J, Lane ME. Transepidermal water loss and skin site. A hypothesis. Interternational J Pharmaceutics 2009; 373: 1-3
10. Johnson LC, Corah NL. Racial differences in skin resistance. Science 1962; 139: 766-7
11. Brenner M, Hearing VJ. The protective role of melanin against UV damage in human skin. Photochem Photobiol 2008; 84: 539-49
12. Andersen KE, Maibach HI. Black and white skin differences. J Am Acad Dermatol 1979; 1: 276-82
13. Williams JD, Jacobson EL, Kim H, Jacobson MK. Folate in skin cancer prevention. Subcell Biochem 2012; 56: 181-97
14. Sueki H, Whitaker-Menezes, Kligman AM. Structural diversity in mast cell granules in black and white skin. Br J Dermatol 2001; 144: 85-93
15. Montagna W, Carlisle K. The architecture of black and white facial skin. J Am Acad Dermatol 1991; 24: 929-37
16. McLaurin CI. Unusual patterns of common dermatoses in blacks. Cutis 1983; 32: 352-60
17. Kelly AP. Keloids. Dermatol Clin 1988; 6: 413-24
18. Shaffer JJ, Taylor SC, Cook-Bolden F. Keloidal scars: a review with critical look at therapeutic options. J Am Acad Dermatol. 2002; 46: S63-S97
19. Verhagen HR. Pomade acne in black skin. Arch Dermatol 1974; 110: 465
20. Lawson CN, Hollinger J, Sethi S, Rodney I, Sarkar R, Dloya N, et al. Updates in the understanding and treatments of skin and hair disorders in women of color. Int J Women's Dermatol 2015; 1: 59-75
21. Weigand DA, Haygood C, Gaylor JR. Cell layers and density of Negro and Caucasian stratum corneum. J Inves Dermatol 1974; 62: 563-8
22. Rosen T, Algra RJ. Papular eruption in black men. Arch Dermatol 1980; 16: 416-8
23. Gorouhi F, Davari P, Fazel N. Cutaneous and mucosal Lichen Planus: A comprehensive review of clinical subtypes, risk factors, diagnosis, and prognosis. Sci World J 2014: 2014
24. Penas PF, Rios-Buceta L, Sanchez-Perez J, Dorado-Bris JM, Aragues M. Keratosis punctata of the palmar creases: Case and prevalence study in Caucasians. Dermatology 1994; 188: 200-2
25. Lucker GPH, Van der Kerkhof PC, Steijlen PM. The hereditary palmoplantar keratoses; an updated review and classification. Br J Dermatol 1994; 131: 1-14
26. Grimes PE, Arora S, Minus HR, Kenny JA. Dermatosis papulosa nigra. Cutis 1983; 32: 385-6
27. Karadag SA, Ozkanli S, Mansuroglu C, Ozlu E, Zemheri E. Effectiveness of the pulse dye laser treatment in a Caucasian woman with dermatosis papulosa nigra. Indian J Dermatol 2015; 60: 321
28. McLaurin CI. Unusual patterns of common dermatoses in blacks. Cutis 1983; 32: 352-60
29. Vano-Galvan S, Ma DL, Lopez-Neyra A, Perez B, Munoz-Zato E, Jaen P. Atypical pityriasis rosea in a black child: a case report. Cases J 2009; 2: 6796
30. Guss LJ, Ghazarian SR, Daya N, Okoye GA. Cutaneous sarcoidosis in African Americans. Austin J Dermatol 2014; 1: 1028
31. Krown SE. Treatment strategies for Kaposi sarcoma in Sub-Saharan Africa: Challenges and Opportunities. Curr Opin Oncol 2011; 23: 463-8.
32. Shoa Y, Willaimson C. The HIV-1 Epidemic: Low- to Middle-Income Countries. Cold Spring Harb Perspect Med 2012 ; 2: a007187
33. Marten RH, Presbury DG, Adamson JE, Cardell BS. An unusual papular and acneiform eruption in the Negro child. Br J Dermatol 1976; 92 : 435-8
34. William HC, Ashworth C, Pembroke AC, Breathnach SM. FACE-Facial Afro-Caribbean Childhood Eruption. Clin Exp Dermatol 1990; 15: 163-6
35. Kahn G, Rywilin AM. Acropustulosis in infancy. Arch Dermatol 1979; 115: 831-3
36. Larralde M, Boldrini MP, Luna PC, Abad ME, Marín CC. Infantile acropustulosis. Dermatología Argentina 2013; 16: 268-71.
37. Rosen T, Algra RJ. Papular eruption in black men. Arch Dermatol 1980; 16: 416-8
38. Halder RM, Bang KM. Skin cancer in Blacks in the United States. Dermatol Clin 1988; 6: 397-405
39. Scotta DA. Disorders of the hair and scalp in blacks. Dermatol Clin 1988; 6: 387-95
40. Kyei A, Bergfeld WF, Piliang M, Summers P. Method and environmental risk factors for the development of central centrifugal cicatricial alopecia. A population study. Arch Dermatol 2011; 147: 909-11
41. Blattner C, Polley DC, Ferrito F, Elston DM. Central centrifugal cicatricial alopecia. Indian Dermatol Online J 2012; 3: 50-1
42. Sperling LC, Sau P. The follicular degeneration syndrome in black patients. Hair comb alopecia revisited and revised. Arch Dermatol 1992; 128: 68-74
43. Alexis A, Heath CR, Halder RM. Folliculitis keloidalis nuchae and pseudofolliculitis barbae. Are prevention and effective treatment within reach? Dermatol Clin 2014; 32: 183-91
44. James J, Saladi RN, Fox JL. Traction Alopecia in Sikh Male Patients. J Am Board Fam Med 2007; 20: 497-8
45. Ameja JS, Vashi CN, Gursel E, Lelli JL. Management of fulminant dissecting cellulitis of the scalp in the pediatric population: Case report and literature review. Can J Plast Surg 2007; 15: 211-4
46. Singh N, Thappa DM. Pigmentary demarcation lines. Pigment Int 2014; 1: 13-6
47. Nieuweboer-Krobotova L. Hyperpigmentation: types, diagnostics and targeted treatment options. J Eur Acad Dermatol Venereol 2013; 27 Suppl 1: 2-4
48. Somani VK and Razvi F. Pigmentary demarcation lines over the face. Indian J Dermatol Venereol Leprol 2004; 70: 336-41
49. Henderson AL. Skin variations in blacks. Cutis 1983; 32: 376-7

50. Chapel TA, Taylor RM, Pinkus H. Volar melanotic macules. Int J Dermatol 1979; 18: 222-5
51. Selmanowitz V, Krivo JM. Hypopigmented markings in Negroes. Int J Dermatol 1973; 12: 229-35
52. Leyden JJ, Spot DA, Goldschmidt H. Diffuse and banded melanin pigmentation in nails. Arch Dermatol 1972; 105: 548-50
53. Tosti A, Piraccini BM, de Farias DC. Dealing with melanonychia. Semin Cutan Med Surg 2009; 28: 49-54
54. Chandra S, Keluskar V, Sah K. Extensive physiologic melanin pigmentation on the tongue: An unusual clinical presentation. Contempt Clin Den 2010; 1: 204-6
55. Marisa P, Blanas N, Bradley G. Pigmented Lesions of the oral cavity: Review, differential diagnosis, and case presentations. J Can Dent Assoc 2004; 70: 682-3
56. Canizares O. Epidemiology and ecology of skin diseases in the tropics and subtropics. In: A Manual of Dermatology for Developing Countries. Oxford: Oxford University Press; 1982, 21-33
57. Jain A. Current status of leprosy as seen in a tertiary care hospital in North India. Indian J Med Res 2012; 135: 439-40.