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## Abstract

Black children are children from Africa, the Caribbean, and Latin America whose ancestry is partially or fully Black African. Children who are black have large and active melanosomes producing eumelanin and providing an intrinsic sun protection to the skin, yielding a Fitzpatrick Skin Type of IV–VI. The skin tone is accompanied by curled hair of lesser density and reduced oil distribution along the follicles as well as hyperactive and plumper fibroblasts. This chapter highlights the biological basis of skin tone in children who are of Black descent, with a focus toward clinical correlation with disease states and susceptibility in the Black population.

## Keywords

Black skin • Hair • Nails • Africa • Caribbean • Latin America • Fitzpatrick skin type • Melanosomes • Eumelanin

## Introduction

- **Black children are children of African ancestry, with matrilineages dating back to 200,000 years ago.**
- **The development of skin pigmentation in black children is felt to have derived from a genetic selection process favoring ultraviolet radiation protection among other valuable features of darker skin.**

Approximately 200,000 years ago, modern day humans first appeared in Eastern Africa. Since then, genetic analyses identified new matrilineages approximately 40,000–80,000 years ago as the *Homo sapiens* dispersed out of Africa to Eurasia and then 15,000–30,000 years ago to the Americas [1].

Questions about what our ancestors looked like invariably lead to questions about the wide diversity of skin pig-

mentation. For example, “Black” skin spans a wide range of color and comprises not only Africans and people of African descent, but also African Americans, Caribbean Americans, and Latin Americans. There have been numerous explanations for skin of color. Consistent in these theories is that differences in skin color developed with a strong influence from natural selection and genetic mutations as the first *Homo sapiens* migrated out of Africa from a climate with consistently high UVR and daytime temperatures into climates with more seasonal variations and lower UVR and daytime temperatures. The ability to adapt to different conditions in this way genetically and phenotypically over centuries has been very important to human survival. While skin pigmentation has a definitive correlation with latitude [2–4], the UV minimal erythral dose (UVMED) is the environmental factor most strongly correlated with skin pigmentation when measured by skin reflectance [5]. Nearer the equator where UVR is the highest, natural selection favored evolution of darker skin. Several major hypotheses have arisen to explain this evolutionary phenomenon: (1) Protection from sunburn and skin cancer; (2) Greater camouflage in forest environments [6]; (3) Improved permeability barrier function [7], (4) Melanin’s antimicrobial

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characteristics [8]; (5) Folate deficiency from UVR-mediated cell division, DNA repair, and melanogenesis [9]; and (6) Thermoregulation.

A discussion of the origins of dark skin should include that of its opposite: the development of fairer skin. The main theory that complements the positive correlation of skin pigmentation with the amount of UVR present is the vitamin D theory, which postulates that lighter skin color evolved in humans migrating from the equator to higher latitudes in order to allow for adequate production of vitamin D. The amount of UVB light needed to generate vitamin D in dark skin is six times as much as in fair skin [10]. It is well known that vitamin D deficiency has many effects on bone health including rickets and osteomalacia and it may also contribute to cardiovascular disease [11], cancer risk, infection, autoimmune disease, and fertility [12]. Yuen and Jablonski argue that effects such as these would affect viability of the young, survival throughout life, fecundity, selection, and longevity [13]; one exception to this would be the Arctic Inuits, whose diet is rich in vitamin D-rich fish oils. Other notable theories of skin lightening include sexual selection [14] and genetic drift [15].

## Melanocyte Biology

- **Melanin is the pigment formed in the melanocyte, but is not the only pigment in skin.**
- **The development of melanin in Black children involves enzymatic favoring of eumelanin production as well as larger melanosomes.**

Skin, hair, and eye color is determined by the amount and type of melanin present. Synthesis of this organic polymer takes place in melanocytes located in the basal layer of the epidermis, hair bulb, and iris. The enzyme tyrosinase is the key enzyme overseeing tyrosine's hydroxylation to dihydroxyphenylalanine (dopa), followed by oxidation to dopaquinone. All of this takes place in lysosome-like organelles, melanosomes. Dopaquinone can then proceed down biochemical pathways to either the dark brown/black insoluble DHI (5, 6-dihydroxyindole)-eumelanin (in the absence of cysteine), light brown/alkali-soluble, DHICA (5,6-dihydroxyindole-2-carboxylic acid)-eumelanin (in the absence of cysteine), or red/yellow pheomelanin (in the presence of cysteine) [16]. Following this, melanosomes are secreted into keratinocytes and melanosomes are transported to the epidermal surface [17].

Melanocyte density can differ between body parts, with the highest densities in the forehead, cheeks, and genital areas; however, melanocyte size, shape, and population density are similar between races with the ratio of keratinocytes to melanocytes in the epidermis staying relatively stable

**Table 2.1** Ultrastructural differences in melanocyte distribution and melanosome packaging by race and ethnicity

Race	Pigmentary differences
Black (in the USA: African American or Afro Caribbean)	Large (Stage IV) melanosomes Eumelanin constitutes majority of pigment production Closely packed doublet or singlet melanosomes, rare aggregates <sup>a</sup> Larger melanophages (may account in part for greater incidence of melasma and erythema dyschromicum perstans) UV filtration is in the malpighian layer
Caucasian	Small, aggregated melanosomes Pheomelanin Few small melanophages UV filtration in the stratum corneum

<sup>a</sup>Taylor SC. Skin of color: biology, structure, function, and implications for dermatologic disease. *J Am Acad Dermatol*. 2002; 46(2 Suppl):S41–62

at 36:1 (Table 2.1) [18]. The key factors affecting skin pigmentation then are the amount and type of melanin as well as the size and distribution of melanosomes. Dark skin has more DHI-eumelanin and lighter skin has more light-brown DHICA-eumelanin and yellow/red pheomelanins [19]. Furthermore, melanosomes in dark skin are larger and found in single bodies whereas light skin has smaller melanosomes that are clustered together (Table 2.1) [20]. The most widely used scale of skin phototype, the Fitzpatrick scale, was developed in 1975 and initially included skin types I–IV (moving from light, always burns to dark, never burns), but was modified in 1988 to include darker skin types V and VI. This further delineates the cutaneous photoresponse of the darkest patients with type VI skin often being at the greatest risk for dyspigmentation (e.g., hypopigmentation from hair removal laser).

## Genetics of Pigmentation

- **Pigmentation is polygenic with contribution from many types of genes ranging from melanin production, distribution, and dispersion genes to melanoblast migration genes.**
- **Alteration in pigmentation genes produces pigmentary alterations ranging from mild skin tone alterations to complete absence of melanin production.**

Pigmentation is polygenic with many different types of genes contributing to the formation of skin tone. Over the last century, many of the studies that discovered genes controlling skin color were investigating pigmentation disorders in humans and animal models. For example, in mice there are greater than 100 genes known to contribute to over 800 phenotypic alleles [21]. With the sequencing of the

**Table 2.2** Genes that contribute to pigmentation

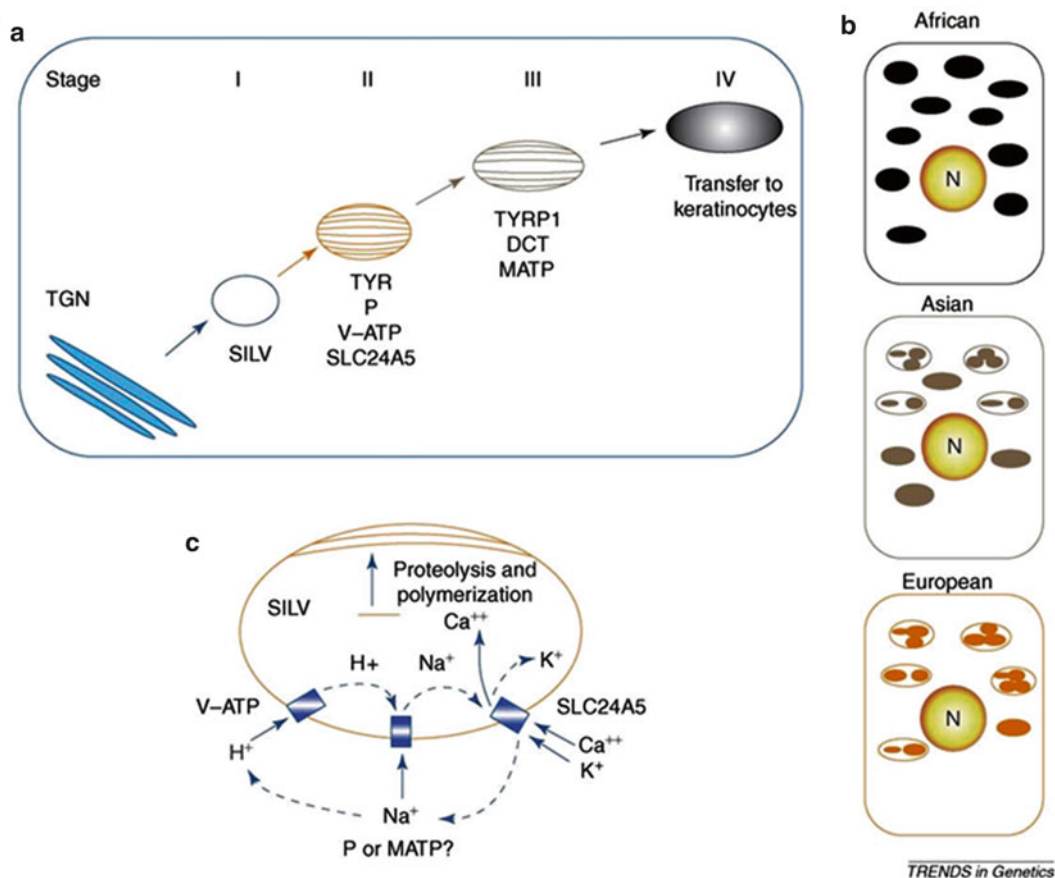
Type	Gene	Associated conditions
Tyrosinase enzyme complex	TYR	Oculocutaneous albinism type I/amelanotic melanoma/vitiligo
	TRP1	Oculocutaneous albinism type 3/vitiligo
Melanosomal proteins	MATP	Oculocutaneous albinism type 4
	P gene	Oculocutaneous albinism type 2
	PMEL/SILV	Juvenile xanthogranuloma/melanoma
	SLC24A5	OCA6/Loeys Dietz/patent ductus arteriosus
Regulators of melanin synthesis	MC1R/Alpha-MSH	Melanogenesis/eumelanin production/Skin Cancers
	ASIP	Hair pigmentation; skin cancers
	ATRN	Radioulnar synostosis
Transcription factors of melanin production	PAX3	Waardenburg syndrome, alveolar rhabdomyosarcoma
	MITF	Waardenburg syndrome, types 2 and 2a
	SOX10	Waardenburg syndrome, type 4/Nodular melanoma
Melanosomal transport proteins	MYO5A	Griscelli syndrome, types I and III; Elejalde syndrome
	MYO7A	Usher syndrome Ib
	RAB27A	Griscelli syndrome, type II
Melanosomal construction/protein routing	CHS1	Chediak–Higashi syndrome
	HPS1-6	Hermansky–Pudlak syndrome
Developmental ligands controlling melanoblast migration and differentiation	EDN3	Hirschsprung syndrome/Waardenburg's syndrome
	KITLG	Familial progressive hyperpigmentation
Developmental receptors controlling melanoblast migration and differentiation	KIT	Piebaldism and urticaria pigmentosum/mastocytosis

human genome just over a decade ago, there has been an explosion of new knowledge in this area from studies including comparative genomic and specific allele association studies. Single nucleotide polymorphisms (SNPs) have also been identified in genome-wide association studies that have allowed illumination of genetic variants associated with human pigmentation of the skin, eye, and hair [22]. The most commonly studied genes, TRY, TRP1, P, MATP, MC1R, ASIP, SLC24A5, and MATP, will be described briefly.

The TYR gene encodes for tyrosinase, a copper-dependent enzyme responsible for catalyzing melanin. This gene is mutated in oculocutaneous albinism type 1, with complete or partial loss of gene function (types Ia and Ib, respectively). At present, there are over 100 mutations associated with albinism or skin color dilution [23]. Also contributing to the tyrosinase enzyme complex is TRP1, mutations in which result in oculocutaneous albinism type 3. In individuals of sub-Saharan African heritage with oculocutaneous albinism type 2, mutations in the P gene cause a defective melanocytic transporter protein resulting in light blond or yellow hair, vision problems, and white skin. MATP is a membrane-associated transporter protein associated with OCA type 4 that shows strong selection in European populations.

Another widely studied pigmentation gene is the melanocortin 1 receptor (MC1R, also called alpha melanocyte stimulating hormone) gene, which codes for a G protein-coupled receptor important in melanocytic switching between production of eumelanin and pheomelanin. Loss-of-function mutations of MC1R have been associated with people with red hair and fair skin (autologous to an autosomal recessive trait), but are also seen in up to 30 % of the population and may play a role in lighter skin color [24]. European populations show a higher sequencing diversity of MC1R, which reflects neutral expectations of selection under relaxation of functional constraints especially when compared to sub-Saharan African and other dark-skinned populations, which are thought to be under stronger functional constraints and show a lack of sequencing diversity [25, 26].

ASIP, the agouti signaling protein, acts antagonistically at the MC1R receptor to inhibit the production of both eumelanin and pheomelanin; and the 8818G allele is strongly associated with dark hair, brown eyes, and dark skin [27, 28]. Lastly, a gene contributing to the “lightening” of skin is the “golden” gene (SLC24A5) coding for a melanosomal cation exchanger and responsible for up to 25–38 % of the difference between the European versus African melanin index of the skin [29]. Other genes implicated in pigmentation are seen in the Table 2.2 (Fig. 2.1).



**Fig. 2.1** Melanosome formation and the role of ion transport in their maturation. (a) The four-stage model of melanosome formation is shown together with key proteins that are necessary for each step of maturation before melanosomes are passed to keratinocytes. (b) Keratinocyte distribution of melanosomes in ethnic populations, note that the melanosomes often form a cap surrounding the nucleus that might have a role in photoprotection. (c) A model for ion transport

that is essential to melanosome function. The coupling of H<sup>+</sup>, Na<sup>+</sup> exchange by the V-ATP complex, with possible involvement of the P or MATP proteins, enables SLC24A5 (also known as NCKX5) to transport K<sup>+</sup>, Ca<sup>2+</sup> ions into the melanosome. Ca<sup>2+</sup> might have an essential role in activating the proteolytic cleavage of SILV, which polymerizes to form the melanosomal matrix copied with permission from Sturm R. 2006 [30]

## Hair

- **Hair type can be categorized by shape of hair in cross section, curvature or lack thereof of the follicle, density of the hairs, and content of sulfur in the hairs.**
- **Hair type may have racial or ethnic association for the general public.**
- **Hair in Black patients can be more susceptible to illness due to reduced density, less elastic anchorage, and cultural styling practices.**

In the literature, research often focuses on grouping hair textures into African, Caucasian, or Asian. These distinctions, though heavily studied and helpful, do not take into account the multitude of the world's population that may not fall into one of these categories due to inter- and inner-group variation of hair types. A study of 1,442 people from 18 countries revealed 8 different hair types [31]. Hardy classified hair

types without incorporating race, but the classifications have not been widely used [32]. Khumalo suggests that race has been used as a proxy for describing hair forms, despite obvious inter-racial variation [33, 34].

Khumalo expressed the need for an easy to use classification of hair forms that is inclusive of multiple hair types. Despite this desire, the most commonly used terminology to describe hair types still utilizes racial classifications.

When cross sections of hair are viewed, Asian hair is round. Caucasian hair is thinner and more elliptical than Asian hair. African hair is often textured, is coiled, and is the most elliptical [35]. On cross section, Black hairs will be flattened. Textured hair and dryness of the scalp and hairs are common in Black hair, due to reduced sebum production/distribution along the hair shaft and reduced water content in the hairs. The hair follicle is expected to be helical or curved, with limited elastic fiber anchorage. Lower hair density is noted in Black

patients (0.6 follicular units per square mm vs. 1 follicular unit per square meter in Asians and Caucasians) [36–38].

It is well known that African textured hair may be straightened permanently with chemical relaxers and temporarily with heat, with side effects ranging from frizziness to follicular destruction [39]. Close observation has revealed that the African texture is also noted to change during certain types of illnesses and states of health such as AIDS, rheumatoid arthritis, systemic lupus erythematosus, pulmonary tuberculosis with cachexia, and Behçet's disease, especially those with anemia of chronic illness, high erythrocyte sedimentation rate, and mild hypocalcemia [40].

In infants, the hair whorl may be hard to note in Black children due to the curl of the hair compounded by the popularity of shaven hairstyles. Microscopy of hair in Black children demonstrates discrete hair packets and curled hairs.

Follicular prominence can be noted in Black adolescents resulting in a light halo near each sebaceous hair follicle of the face. Follicular inflammation is more common in Black children with consequently greater amounts of follicular eczema and folliculocentric allergic contact dermatitis.

Some studies have suggested that sebaceous glands are larger in Blacks than in Caucasians, and therefore, sebum, the oil produced by sebaceous glands, has greater lipid content [41, 42]. This may possibly allow for greater bacterial and yeast overgrowth.

## Dermal

- **Fibroblasts are larger in the dermis of Black children contributing to the increased incidence of keloidal lesions in this racial group.**
- **Elastic tissue anchorage of the hair follicle is reduced resulting in greater damage with traction-based hairstyles.**

Skin thickness is the same in Blacks and Whites [43], despite the compact nature of the stratum corneum in Blacks [44]. Fibroblasts in Black skin are larger than those in White skin [45]. Elastic tissue anchorage of the hair follicle is reduced in Black patients resulting in greater risk of traction-induced damage.

Keloids result from unbalanced extracellular matrix production and degradation [46]. Hyperactive fibroblasts contribute to keloid formation and are influenced by transforming growth factor beta, epidermal growth factor, mast cells, and decreased collagenase activity [47–49].

Keloid development is influenced by many factors including genetic susceptibility including racial prevalence amongst Blacks, Asians, and Hispanics, family linkage, and HLA associations and corroborated by twin studies. Environmental contributory factors include hormones, wound tension, infection, and foreign body granulomas. Another factor that authors note in practice is the comorbidity

of nickel contact allergy, often induced by piercing, as a trigger of keloids secondary to piercing.

## Other Considerations

- **Genetic differences in metabolism and skin structure can affect response to medications.**
- **Awareness of G6PD deficiency, an X-linked recessive enzymatic defect, is needed for practitioners who prescribe dapsone and antimalarial medications, due to the risk of severe hemolysis.**
- **The role of environment on development of skin diseases is especially contributory in the development of atopic dermatitis in developed countries.**

Some dermatologic diseases affecting patients with skin of color have been linked to genetic propensity. For example, sarcoid, is associated with specific HLA types in Black patients [50, 51].

Vitiligo is more prominent in children of color, but despite this, no specific linkage genes to race have been identified in Black children. Vitiligo genetics is actually polygenic and multifactorial [52]. On the other hand, OCA2, an autosomal recessive albinism, has a specific gene defect and is the most prevalent autosomal recessive disease among South African Blacks, P protein is defective in OCA2 leading to abnormal tyrosinase enzyme function and defective melanin production [52].

Keloids have long been observed to occur more frequently in skin of color populations, especially in those of African descent. Studies now suggest that certain environmental triggers may spur keloid formation in those who are genetically susceptible [53].

Other pertinent genetically common illnesses in patients of Black or African descent include G6PD deficiency, an X-linked recessive enzymatic defect that affects metabolism of medications such as dapsone and hydroxychloroquine and can result in severe hemolysis with drug administration of these agents. Male patients should be suspected most, but all black patients should be screened prior to usage of these agents as female patients may be homozygous or have low expression based on lyonization.

Sickle cell anemia can confer susceptibility to bacterial infection (e.g., *Streptococcus*) [54] and is associated with severe hemolysis requiring hospitalization for transfusion in children with G6PD deficiency. Sickle cell carriers may be less prone to malaria, generating the hypothesis as to why sickle cell carriage and disease are more common in patients of African descent [55].

Type II diabetes mellitus is associated with acanthosis nigricans, skin tags, candidal infections, and poor wound healing. In the USA, Black, Native American/Inuit, and Mexican American children are at increased risk. Signs of insulin resistance, especially acanthosis nigricans, are noted



in pre-teen years with disease becoming full blown in some cases by the mid-teen years [56, 57].

Black children also have specific reduction in the formation of infantile hemangiomas [58] and lifetime risk of skin cancers [59] (lifetime risk is lower). Collagen vascular diseases are more common from birth, i.e., neonatal lupus through childhood/adolescence when Black children may develop the first features of lupus erythematosus, with specifically increased risk of nephritis [60]. More than 60 % of patients under the age of 20 years with systemic lupus erythematosus will be Black [61].

The effect of the environment/country/place of birth on the disease incidence cannot be ignored, e.g., atopic dermatitis being more common in Afro-caribbeans in London, but relatively less common in Africans on the continent, etc. In addition, differences in the pattern of skin disease exist between races. Henderson et al. found that more than 60 % of all pediatric patients seen at their dermatology clinic had diagnoses of acne (28.6 %), dermatitis (19.4 %), and warts (16.2 %) [62]. But when the patients were further stratified, they found that African-American pediatric patients in their study were most commonly seen for dermatitis (29.0 %), acne (27.5 %), and dermatophytosis (10.2 %), whereas Caucasian children were most commonly seen for acne (29.9 %), warts (22.6 %), and dermatitis (13.1 %).

Another study of both adults and children at our Skin of Color Clinic at St. Luke's Roosevelt Hospital in New York City found similarities between common diagnoses in Black and Caucasian skin. However, dyschromia and alopecia were two conditions commonly seen in black patients that were not even in the top ten diagnoses of Caucasian patients [63]. The most common diagnoses in African-American patients were acne, dyschromia, contact dermatitis/other eczema of unspecified cause, alopecia, and seborrheic dermatitis. In Caucasian patients, the most common diagnoses were acne, lesion of unspecified behavior, benign neoplasm of skin of trunk, contact dermatitis/other eczema of unspecified cause, and psoriasis.

Those studies, along with others from around the world, show similar patterns comparing disease incidence in skin of color to Caucasian skin, but modern travel has enabled entire groups of people to be mobile and migrate globally. While there are numerous genetically determined biological factors discussed earlier in the chapter that are responsible for the characteristics of skin of color and resultant epidemiological differences of diseases between races, environment also plays a role in disease incidence.

One representation of environment playing a role in disease incidence is examining atopic dermatitis and eczema. London-born Black Caribbean children were thought to have an increased risk of atopic dermatitis [64] and were also thought to be more likely to develop atopic eczema when compared to their counterparts in Kingston, Jamaica [65].

Another London study of a Black population showed the most frequent dermatoses in their pediatric population were atopic eczema (36.5 %) and tinea capitis (26.5 %), whereas adults were most commonly diagnosed with acneiform eruptions (27.4 %) and eczema (9.6 %) [66]. Compare this to a Jamaican study, which did not separate their data between adults and children, that identified the most common skin diseases as acne vulgaris (29.21 %), seborrheic eczema (22.02 %), pigmentary disorders (16.56 %), and atopic eczema (6.1 %) [67]. The frequency of dermatitis and atopic eczema in Black patients found in the Western countries was greater than those found in less developed countries, with theories including increased hygiene in countries that are developed (e.g., varicella vaccination) [68], indoor heating, and cultural factors such as washing [69].

Furthermore, the different rates of atopic eczema between countries can be explained at least partially by their natural environment/climates and not only by immunogenic theory. For example, eczema is known to be triggered by skin dryness, so comparing the rates of atopic dermatitis (13.8 %) and contact dermatitis (5.8 %) seen in the more tropical Nigeria [70] to those rates of eczema seen in the drier and semi-arid weather of South Africa (32.7 %) [71] would support this. That environmental factors are important in disease expression was also suggested by the authors of a multi-country cross-sectional study, which showed symptoms of atopic eczema with widely varying rates of prevalence both within and between countries with similar ethnic groups [72].

## Conclusion

Black children today, and their skin, reflect the culmination of 200,000 years of environmental exposure and genetic selection. Understanding of these contributory factors is crucial in the appreciation of Black skin.

## References

1. Campbell MC, Tishkoff SA. The evolution of human genetic and phenotypic variation in Africa. *Curr Biol*. 2010;20(4): R166–73.
2. Tasa GL, Murray CJ, Boughton JM. Reflectometer reports on human pigmentation. *Curr Anthropol*. 1985;26:511–2.
3. Roberts DF. Human pigmentation: its geographical and racial distribution and biological significance. *J Cosmet Sci*. 1977;28: 329–42.
4. Roberts DF, Kahlon DPS. Environmental correlations of skin colour. *Ann Hum Biol*. 1976;3(1):11–22.
5. Chaplin G. Geographic distribution of environmental factors influencing human skin colouration. *Am J Phys Anthropol*. 2004;125(3):292–302.
6. Cowles RB, Hamilton WJ, Heppner F. Black pigmentation: adaptation for concealment or heat conservation? *Science*. 1967; 158(3806):1340–1.

7. Elias PM, Menon G, Wetzel BK, Williams JW. Barrier requirements as the evolutionary “driver” of epidermal pigmentation in humans. *Am J Hum Biol.* 2010;22(4):526–37.
8. Mackintosh JA. The antimicrobial properties of melanocytes, melanosomes and melanin and the evolution of black skin. *J Theor Biol.* 2001;211:101–13.
9. Jablonski NG. Evolution of human skin colouration and its relevance to health in the modern world. *J R Coll Physicians Edinb.* 2012;42(1):58–63.
10. Clemens TL, Henderson SL, Adams JS, Holick MF. Increased skin pigment reduces the capacity of skin to synthesize Vitamin D3. *Lancet.* 1982;1(8263):74–6.
11. Perna L, Schöttker B, Holleczeck B, Brenner H. Serum 25-hydroxyvitamin D and incidence of fatal and nonfatal cardiovascular events: a prospective study with repeated measurements. *J Clin Endocrinol Metab.* 2013;98(12):4908–15.
12. Lerchbaum E, Obermayer-Pietsch B. Vitamin D and fertility: a systematic review. *Eur J Endocrinol.* 2012;166:765–78.
13. Yuen AW, Jablonski NG. Vitamin D: in the evolution of human skin colour. *Med Hypotheses.* 2010;74(1):39–44.
14. Aoki K. Sexual selection as a cause of human skin colour variation: Darwin’s hypothesis revisited. *Ann Hum Biol.* 2002;29(6):589–608.
15. Juzeniene A, Setlow R, Porojnicu A, Steindal AH, Moan J. Development of different human skin colors: a review highlighting photobiological and photobiophysical aspects. *J Photochem Photobiol B.* 2009;96(2):93–100.
16. Parra EJ. Human pigmentation variation: evolution, genetic basis, and implications for public health. *Am J Phys Anthropol.* 2007;45:85–105.
17. Jimbow K, Quevedo WC, Fitzpatrick TB, Szabo G. Some aspects of melanin biology: 1950–1975. *J Invest Dermatol.* 1976;67(1):72–89.
18. Quevedo WC, Fitzpatrick TB, Jimbow K. Human skin color: origin, variation, and significance. *J Hum Evol.* 1985;14(1):43–56.
19. Alaluf S, Atkins D, Barrett K, Blount M, Carter N, Heath A. Ethnic variation in melanin content and composition in photoexposed and photoprotected human skin. *Pigment Cell Res.* 2002;15:112–8.
20. Szabo G, Gerald AB, Pathak MA, Fitzpatrick TB. Racial differences in the fate of melanosomes in human epidermis. *Nature.* 1969;222:1081–2.
21. Bennett DC, Lamoreux ML. The color loci of mice – a genetic century. *Pigment Cell Res.* 2003;16(4):333–44.
22. Han J, Kraft P, Nan H, Guo Q, Chen C, Qureshi A, et al. A genome-wide association study identifies novel alleles associated with hair color and skin pigmentation. *PLoS Genet.* 2008;4(5):e100074.
23. Oetting WS, Fryer JP, Shriram S, King RA. Oculocutaneous albinism type 1: the last 100 years. *Pigment Cell Res.* 2003;16(3):307–11.
24. Rees JL. The melanocortin 1 receptor (MC1R): more than just red hair. *Pigment Cell Res.* 2000;13(3):135–40.
25. Rana BK, Hewett-Emmett D, Jin L, Chang BH, Sambuughin N, Lin M, et al. High polymorphism at the human melanocortin 1 receptor locus. *Genetics.* 1999;151(4):1547–57.
26. Harding RM, Healy E, Ray AJ, Ellis NS, Flanagan N, Todd C, et al. Evidence for variable selective pressures at MC1R. *Am J Hum Genet.* 2000;66(4):1351–61.
27. Graham A, Wakamatsu K, Hunt G, Ito S, Thody AJ. Agouti protein inhibits the production of eumelanin and pheomelanin in the presence and absence of alpha-melanocyte stimulating hormone. *Pigment Cell Res.* 1997;10(5):298–303.
28. Bonilla C, Boxill LA, Donald SA, Williams T, Sylvester N, Parra EJ, et al. The 8818G allele of the agouti signaling protein (ASIP) gene is ancestral and is associated with darker skin color in African Americans. *Hum Genet.* 2005;116(5):402–6.
29. Lamason RL, Mohideen MA, Mest JR, Wong AC, Norton HL, Aros MC, et al. SLC24A5, a putative cation exchanger, affects pigmentation in zebrafish and humans. *Science.* 2005;310(5755):1782–6.
30. Sturm R. A golden age of human pigmentation genetics. *Trends Genet.* 2006;22(9):464–8.
31. De la Mettrie R, Saint-Leger D, Loussouarn G, Garcel A, Porter C, Langaney A. Shape variability and classification of human hair: a worldwide approach. *Hum Biol.* 2007;79(3):265–81.
32. Hrdy D. Quantitative hair form variation in seven populations. *Am J Phys Anthropol.* 1973;39(1):7–17.
33. Khumalo NP. Yes, let’s abandon race – it does not accurately correlate with hair form. *J Am Acad Dermatol.* 2007;56(4):709–10.
34. Franbourg A, Hallegot P, Baltenneck F, Toutain C, Leroy F. Current research on ethnic hair. *J Am Acad Dermatol.* 2003;48(6 Suppl):S115–9.
35. Ramos-e-Silva M. Ethnic hair and skin: what is the state of the science? *Clin Dermatol.* 2002;20(3):321–4.
36. Heath CR, McMichael AJ. Chapter 17: Biology of hair follicle. In: Kelly AP, Taylor SC, editors. *Dermatology for skin of color.* New York: McGraw Hill; 2009. p. 105–9.
37. Silverberg NB. Chapter 1: Atlas of pediatric cutaneous biodiversity. Springer; 2013. p. 6–7.
38. Khumalo NP. African hair morphology: macrostructure to ultrastructure. *Int J Dermatol.* 2005;44 Suppl 1:10–2.
39. Shetty VH, Shetty NJ, Nair DG. Chemical hair relaxers have adverse effects a myth or reality. *Int J Trichol.* 2013;5(1):26–8.
40. Ajose FO. Diseases that turn African hair silky. *Int J Dermatol.* 2012;51 Suppl 1:12–6, 14–9.
41. Kligman AM, Shelley WB. An investigation of the biology of the human sebaceous gland. *J Invest Dermatol.* 1958;30(3):99–125.
42. Taylor SC. Skin of color: biology, structure, function, and implications for dermatologic disease. *J Am Acad Dermatol.* 2002;46(2 Suppl):S41–62.
43. Whitmore SE, Sago NJ. Caliper-measured skin thickness is similar in white and black women. *J Am Acad Dermatol.* 2000;42:76–9.
44. La Ruche G, Cesarini JP. Histology and physiology of black skin. *Ann Dermatovenereologica.* 1992;119:567–74.
45. Montagna W, Carlisle K. The architecture of black and white facial skin. *J Am Acad Dermatol.* 1991;24:929–37.
46. Hahn JM, Glaser K, McFarland KL, Aronow BJ, Boyce ST, Supp DM. Keloid-derived keratinocytes exhibit an abnormal gene expression profile consistent with a distinct causal role in keloid pathology. *Wound Repair Regen.* 2013;21(4):530–44.
47. Shih B, Garside E, McGrouther DA, Bayat A. Molecular dissection of abnormal wound healing processes resulting in keloid disease. *Wound Repair Regen.* 2010;18(2):139–53.
48. Satish L, Babu M, Tran KT, Hebda PA, Wells A. Keloid fibroblast responsiveness to epidermal growth factor and activation of downstream intracellular signaling pathways. *Wound Repair Regen.* 2004;12(2):183–92.
49. Johnson Jr BL. Differences in skin type. In: Johnson Jr BL, Moy RL, White GM, editors. *Ethnic skin: medical and surgical.* St. Louis, MO: Mosby; 1998. p. 3–5.
50. Iannuzzi MC. Advances in the genetics of sarcoidosis. *Proc Am Thorac Soc.* 2007;4(5):457–60.
51. Adrianto I, Lin CP, Hale JJ, Levin AM, Datta I, Parker R, et al. Genome-wide association study of African and European Americans implicates multiple shared and ethnic specific loci in sarcoidosis susceptibility. *PLoS One.* 2012;7(8):e43907.
52. Manga P, Kerr R, Ramsay M, Kromberg JG. Biology and genetics of oculocutaneous albinism and vitiligo – common pigmentation disorders in southern Africa. *S Afr Med J.* 2013;103(12 Suppl 1):984–8.
53. Halim AS, Emami A, Salahshourifar I, Kannan TP. Keloid scarring: understanding the genetic basis, advances, and prospects. *Arch Plast Surg.* 2012;39(3):184–9.

54. <http://emedicine.medscape.com/article/200390-overview>. Accessed 16 Nov 2014..
55. Gong L, Parikh S, Rosenthal PJ, Greenhouse B. Biochemical and immunological mechanisms by which sickle cell trait protects against malaria. *Malar J*. 2013;12:317.
56. Gahagan S, Silverstein J, American Academy of Pediatrics Committee on Native American Child Health; American Academy of Pediatrics Section on Endocrinology. Prevention and treatment of type 2 diabetes mellitus in children, with special emphasis on American Indian and Alaska Native children. American Academy of Pediatrics Committee on Native American Child Health. *Pediatrics*. 2003;112(4):e328.
57. Sinha S, Schwartz RA. Juvenile acanthosis nigricans. *J Am Acad Dermatol*. 2007;57(3):502–8.
58. Amrock SM, Weitzman M. Diverging racial trends in neonatal infantile hemangioma diagnoses, 1979–2006. *Pediatr Dermatol*. 2013;30(4):493–4.
59. Agbai ON, Buster K, Sanchez M, Hernandez C, Kundu RV, Chiu M, et al. Skin cancer and photoprotection in people of color: a review and recommendations for physicians and the public. *J Am Acad Dermatol*. 2014;70(4):748–62.
60. Tejani A, Nicastrì AD, Chen CK, Fikrig S, Gurumurthy K. Lupus nephritis in black and Hispanic children. *Am J Dis Child*. 1983;137(5):481–3.
61. Klein-Gitelman MS, Jung LK. Pediatric systemic lupus erythematosus. *Medscape (US)* 2014. Available from: <http://emedicine.medscape.com/article/1008066-overview#a0156> [updated 6 May 2013, cited 7 May 2014]
62. Henderson MD, Abboud J, Cogan CM, Poisson LM, Eide MJ, Shwayder TA, et al. Skin-of-color epidemiology: a report of the most common skin conditions by race. *Pediatr Dermatol*. 2012;29(5):584–9.
63. Alexis AF, Sergay AB, Taylor SC. Common dermatologic disorders in skin of color: a comparative practice survey. *Cutis*. 2007;80(5):387–94.
64. Williams HC, Pembroke AC, Forsdyke H, Boodoo G, Hay RJ, Burney PG. London-born black Caribbean children are at increased risk of atopic dermatitis. *J Am Acad Dermatol*. 1995;32(2 Pt 1):212–7.
65. Burrell-Morris CE, LaGrenade L, Williams HC, Hay R. The prevalence of atopic dermatitis in black Caribbean children in London and Kingston, Jamaica. *Br J Dermatol*. 1997;137 Suppl 50:22. (Abstr.).
66. 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(3):512–7.
67. Dunwell P, Rose A. Study of the skin disease spectrum occurring in an Afro-Caribbean population. *Int J Dermatol*. 2003;42(4):287–9.
68. Silverberg JI, Norowitz KB, Kleiman E, Silverberg NB, Durkin HG, Joks R, et al. Association between varicella zoster virus infection and atopic dermatitis in early and late childhood: a case-control study. *J Allergy Clin Immunol*. 2010;126(2):300–5.
69. Silverberg JI, Hanifin J, Simpson EL. Climatic factors are associated with childhood eczema prevalence in the United States. *J Invest Dermatol*. 2013;133(7):1752–9.
70. Yahya H. Change in pattern of skin disease in Kaduna, north-central Nigeria. *Int J Dermatol*. 2007;46(9):936–43.
71. Hartshorne ST. Dermatological disorders in Johannesburg. *S Afr Clin Exp Dermatol*. 2003;28(6):661–5.
72. Williams H, Robertson C, Stewart A, Ait-Khaled N, Anabwani G, Anderson R, et al. Worldwide variations in the prevalence of symptoms of atopic eczema in the International Study of Asthma and Allergies in Childhood. *J Allergy Clin Immunol*. 1999;103(1 Pt 1):125–38.



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