THE EVOLUTION OF HUMAN SKIN AND SKIN COLOR

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■ **Abstract**   Humans skin is the most visible aspect of the human phenotype. It is
distinguished mainly by its naked appearance, greatly enhanced abilities to dissipate
body heat through sweating, and the great range of genetically determined skin colors
present within a single species. Many aspects of the evolution of human skin and skin
color can be reconstructed using comparative anatomy, physiology, and genomics.
Enhancement of thermal sweating was a key innovation in human evolution that allowed
maintenance of homeostasis (including constant brain temperature) during sustained
physical activity in hot environments. Dark skin evolved *pari passu* with the loss of body
hair and was the original state for the genus *Homo*. Melanin pigmentation is adaptive
and has been maintained by natural selection. Because of its evolutionary lability, skin
color phenotype is useless as a unique marker of genetic identity. In recent prehistory,
humans became adept at protecting themselves from the environment through clothing
and shelter, thus reducing the scope for the action of natural selection on human skin.

INTRODUCTION

When humans visualize a body, they see mostly skin. The skin is the body's
direct interface with the physical environment, conveying a state of health and
personal identity. The skin comprises a sheet-like investiture that protects the
body from attack by physical, chemical, and microbial agents. It is the organ that
regulates body temperature through control of surface blood flow and sweating and
detects critical information about the ambient environment and objects touched.
The largest and most massive of the organs of the body, the skin of the average
adult human exceeds 2 m^2 yet is generally no thicker than 2 mm (Odland 1991).
The skin also provides a forum for advertising. It provides information about a
person's age, health, and some aspects of ancestry, and furnishes a placard upon
which further information is placed through temporary and permanent decoration.

Research on the evolution of human skin and skin color has not been com-
mensurate with the importance of skin in human evolution. Skin is generally not
preserved in the fossil record and so details of its evolution can be gained only from

comparative anatomical and physiological evidence. Skin has also been overlooked as a topic of research interest in anthropology and human biology in recent decades because of the social sensitivity surrounding discussions of skin color and because of the use and misuse of skin color in biological and social concepts of race.

The goal of this review is to provide a comprehensive yet economical survey of the biology, evolution, and culture of human skin and skin color, with an emphasis on new research—especially on the evolution of skin color. The review begins with an overview of the basic biology of skin itself, followed by discussions of the evolution of skin and skin color, and of skin color and race.

THE STRUCTURE AND FUNCTIONS OF HUMAN SKIN

The skin serves as an effective physical barrier because its laminar structure renders it relatively resistant to abrasion, puncture, and percutaneous absorption, and because its immune cells mount a first line of defense against pathogens coming in contact with the body. Lacking adequate protection from hair, human skin has undergone numerous adaptive structural changes that give it strength, resilience, and sensitivity (Montagna 1981). The skin of humans, like that of all tetrapods, acts as a sun shield to protect the body from most solar UV radiation (UVR) and is the locus for the initiation of the important, UVR-driven process of vitamin D production in the body.

Epidermis

The laminar structure of human skin comprises two major tissue layers, a thinner outer layer, the epidermis, and a thicker and more internally complex inner layer, the dermis (Figure 1). The epidermis is a stratified keratinizing epithelium with a smooth, abrasion-resistant surface that is interrupted only by hair follicles and the pores of sweat glands. The barrier properties of the skin are predicated on the integrity of the stratum corneum (Elias et al. 2003, Taylor 2002). Keratinocytes are the principal cell type found in epidermis and are composed largely of filamentous proteins known as keratins, which are imbedded in an amorphous matrix. The skin's elasticity and resistance to physical and chemical attack can be attributed to the high elastic modulus and unique amino acid composition of the keratinized layer of the epidermis (Marks 1991, Odland 1991). The epidermis also contains populations of three types of immigrant dendritic cells: melanocytes, Langerhans cells, and Merkel cells. Melanocytes produce the skin's primary pigment, melanin, and are discussed in greater detail below. Langerhans cells are specialized cells of the immune system that present and respond to antigens coming in contact with the skin, and Merkel cells are associated with nerve terminals that together function as slow-adapting mechanoreceptors for touch; they are most common on the glabrous skin of the fingertips (Chu et al. 2003, Kripke & Applegate 1991, Lynn 1991, Odland 1991). The epidermis is subdivided into four layers from deep to superficial: the stratum basale (the germinative layer of keratinocytes), the
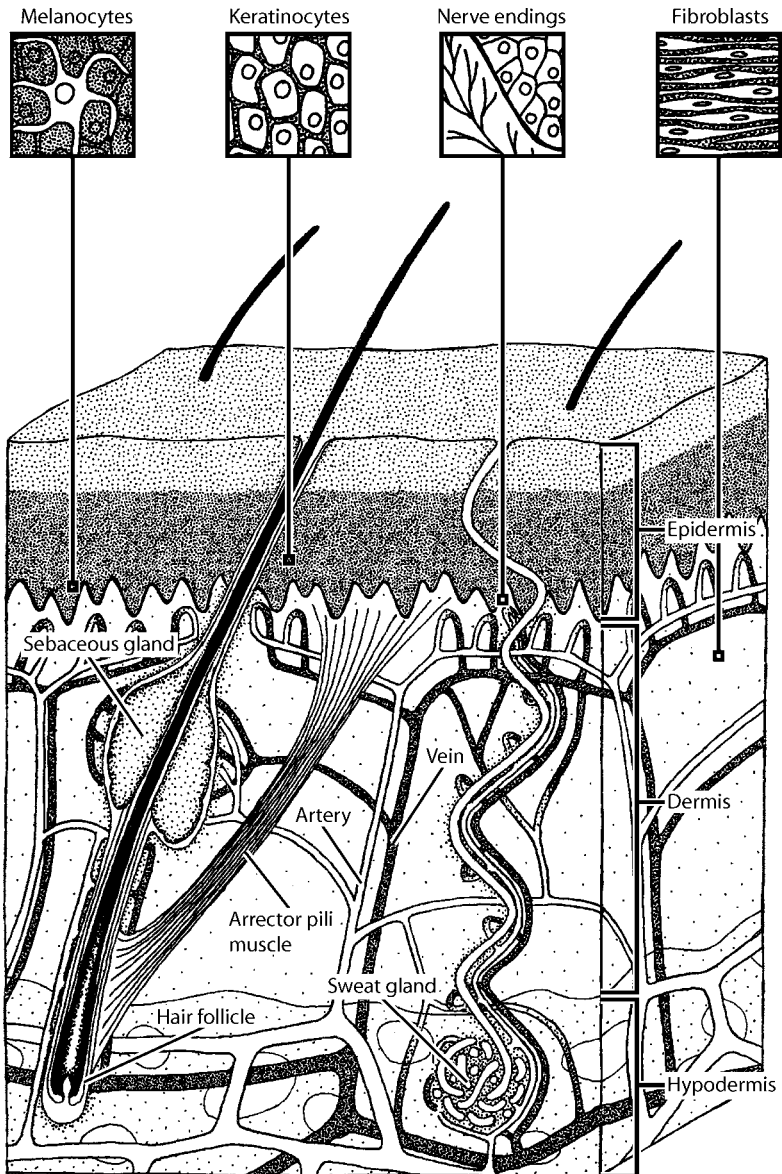
Figure 1 Schematic rendering of a cross-section of human skin, showing its laminar structure, main cell types, and appendages.

stratum spinosum, the stratum granulosum, and the stratum corneum. The stratum corneum consists of flattened, nonviable keratinocytes. In darkly pigmented or heavily tanned individuals, these keratinocytes contain specks of melanin "dust" (Kollias 1995a). The stratum corneum acts as a barrier to the unrestrained passage of water and solutes through the skin, defends against invasion by microorganisms and the penetration of toxic substances, and protects against most mechanical injury caused by friction, abrasion, pricks, or arthropod bites (Marks 1991). These functions are successfully served despite the epidermis being in a constant state of turnover, as the outermost cornified cells of the stratum corneum are shed as they are replaced from below.

Differences between human groups in epidermal structure and thickness have been reported, but most studies of this topic have been based on small samples with poorly controlled experimental designs, as reviewed elsewhere (Taylor 2002). Considerable variation in epidermal thickness exists within human populations and is likely related to age and history of sun exposure. The stratum corneum of darkly pigmented or heavily tanned people is more compact and consists of more cornified cell layers than that of lightly pigmented people; these characteristics enhance the barrier protection functions of the skin (Taylor 2002).

In all primates, the epidermis of the volar surfaces of the hands and feet exhibit well-developed epidermal ridges or dermatoglyphics, which impart greater resistance against friction and help to insure secure purchase on locomotor substrates and on objects being gripped or manipulated. Dermatoglyphics are also found on the ventral surfaces of the tails of prehensile-tailed New World monkeys and on the knuckle pads of chimpanzees and gorillas (Ellis & Montagna 1962, Montagna 1971).

The melanocytes of the epidermis warrant close attention because of their role in the production of the skin's primary pigment or chromophore, melanin. Melanocytes are specialized dendritic cells that reside in the stratum basale of the epidermis and in the matrix portion of the hair bulb. They originate in the neural crest as melanoblasts proliferate and migrate to the epidermis during the eighteenth week of embryonic development (Rawles 1948). Melanocytes produce melanins in specialized cytoplasmic organelles called melanosomes, which vary in size and degree of aggregation depending on skin type and pigmentation (Figure 2) (Szabo et al. 1969). The density of melanocytes varies over the surface of the body, and the number of active (melanin-producing) melanocytes varies with age and can be increased by exposure to UVR (Halaban et al. 2003, Jimbow et al. 1991, Quevedo et al. 1975). The total number of melanocytes is relatively invariant from one person to another, however, and is not related to variation between human groups in skin pigmentation (Fitzpatrick et al. 1961, Jimbow et al. 1991, Robins 1991, Young & Sheehan 2001). MacKintosh, following Wasserman, has recently advanced the hypothesis that melanocytes, melanosomes, and melanin together function as part of the immune system against invading microorganisms and that the more darkly pigmented skins of the indigenous peoples of the tropics have evolved primarily to serve this function (MacKintosh 2001; Wassermann 1965b, 1974).
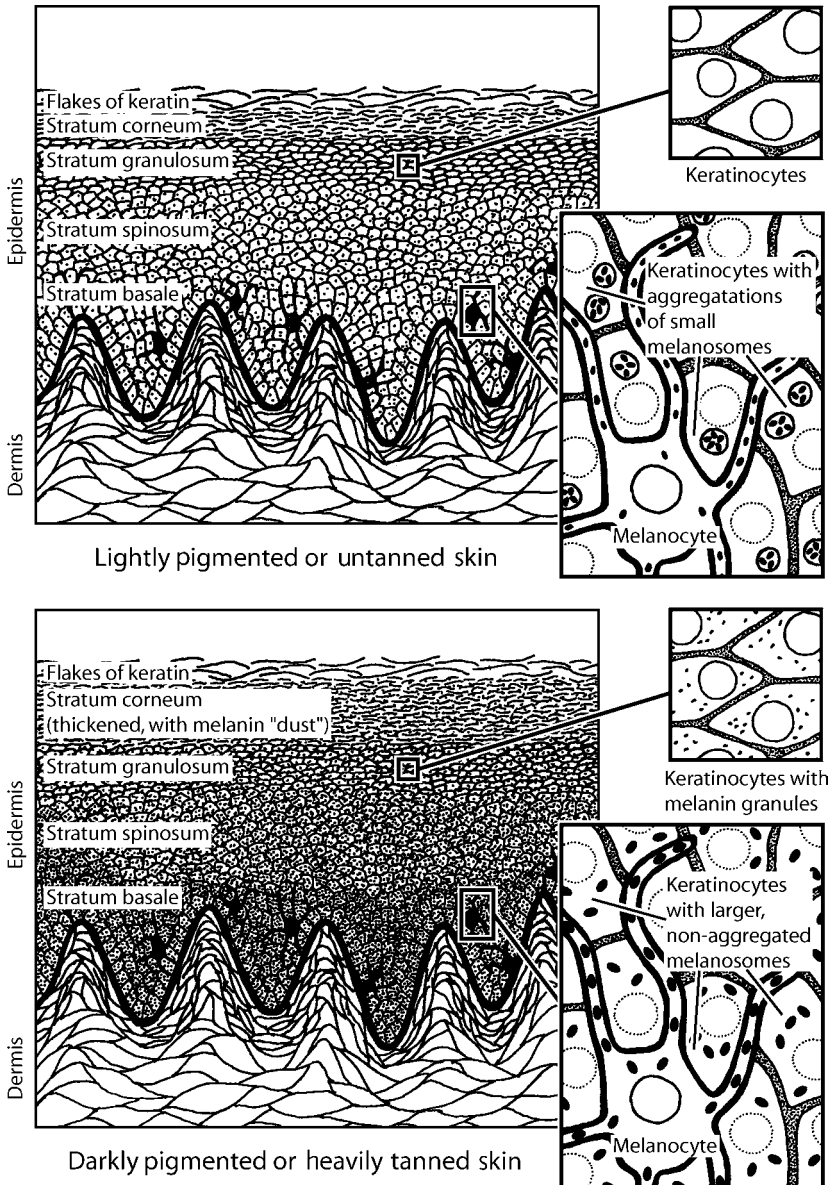
Figure 2   Schematic rendering of cross-sections of lightly and darkly pigmented human skin, showing differences in stratum corneum structure and in the size and aggregation of melanin-containing melanosomes.

Melanocytes project their dendrites into keratinocytes where they then transfer mature melanosomes (Figure 2). Melanosomes are ellipsoidal, membrane-bound organelles containing melanin. After melanosomes have been transferred to keratinocytes, they become aggregated and surrounded by a membrane in a melanosome complex (Jimbow et al. 1991, Szabo et al. 1969). In darkly pigmented skin, melanosomes are large and are not clumped in aggregations, whereas in lightly pigmented skin these organelles are smaller and aggregated (Szabo et al. 1969). Intensity of skin coloration is determined by many factors: (*a*) The total number of melanosomes in the keratinocytes and melanocytes, and their degree of dispersion; (*b*) the rate of melanin production (melanogenesis); (*c*) the degree of melanization of melanosomes; (*e*) the rate of transport and type of incorporation of melanosomes into keratinocytes; (*f*) the degradation of melanosomes within the keratinocytes; and (*g*) a person's chronological age because the number of metabolically active melanocytes decreases over time (Halaban et al. 2003, Jimbow et al. 1976, Ortonne 1990, Parker 1981). Larger melanosomes break down more slowly in keratinocytes and contribute to higher levels of pigmentation (Sulaimon & Kitchell 2003).

MELANIN PIGMENTATION AND ITS MEASUREMENT   Human skin derives most of its pigmentation from melanin, an extremely dense, virtually insoluble, high molecular weight polymer that is attached to a structural protein (Jimbow et al. 1991, Ortonne 2002, Parker 1981, Sulaimon & Kitchell 2003). Human skin contains the two types of melanin found in all mammals, the brownish-black eumelanin and the reddish-yellow pheomelanin (Thody et al. 1991). Higher concentrations of eumelanin characterize darker skin phenotypes including tanned skin. Concentrations of pheomelanin in the skin vary considerably from individual to individual within any given human group, but pheomelanin-rich skin phenotypes are more common among red-haired northern Europeans, as well as East Asians and Native Americans (Rana et al. 1999, Thody et al. 1991). Melanin is synthesized by oxidation of tyrosine via the enzyme tyrosinase (Fitzpatrick et al. 1950, Jimbow et al. 1976, Ortonne 2002). Eumelanins and pheomelanins arise from a common metabolic pathway in which dopaquinone is the key intermediate (Ortonne 2002). As is discussed in greater detail below, production of melanins is regulated by pigmentation genes, hormones, and UVR (Fitzpatrick & Ortonne 2003, Sulaimon & Kitchell 2003, Thody & Smith 1977). A balance of many regulatory factors is essential for normal pigment production in the melanocyte, and derangements of these factors can lead to anomalies of cutaneous pigmentation such as albinism piebald spotting, and various types of hyperpigmentation (Robins 1991, Sulaimon & Kitchell 2003, Thody & Smith 1977).

The optical and chemical properties of melanins have been studied in detail (Ito 2003, Kollias et al. 1991, Ortonne 2002, Prota 1992c), but detailed chemical characterization of the compounds has been difficult to obtain because melanin polymers are composed of many different units connected through strong carbon-carbon bonds (Ito 2003). The optical properties of natural melanin in vivo are

related to its abilities to absorb, scatter, and reflect light of different wavelengths (Kollias et al. 1991, Ortonne 2002). The melanins in human skin are a heterogeneous mixture of melanin polymers, precursors, and metabolites, characterized by a continuous absorption capacity in the UV range and exponentially declining absorption capacity from the UV to the visible range (Kollias 1995b, Sarna & Swartz 1998). Natural protection against sunburning (photoprotection) is due to the absorption and scattering of UVR by melanin (Kaidbey et al. 1979; Kollias 1995a,b). Both processes are influenced by the density and distribution of melanosomes within keratinocytes (Figure 2), with the larger, singly dispersed and heavily melanized melanosomes of darkly pigmented skin absorbing more energy than the smaller, less dense, and lightly melanized melanosomes of lightly pigmented skin (Kaidbey et al. 1979).

Melanin was long considered to act as a passive screening filter against UVR, but it is by no means inert (Fitzpatrick et al. 1961). Photodegradation (photolysis) and/or oxidative polymerization of melanin may occur when it absorbs photons (Ortonne 2002). Recent evidence indicates that the photoprotective role of melanin in darkly pigmented skin may be augmented by its ability to scavenge oxygen-derived radicals (reactive oxygen species), such as superoxide anion and hydrogen peroxide, which are cytotoxic compounds generated by the interaction of UV photons with membrane lipids and other cellular components (Ortonne 2002, Prota 1992c, Sulaimon & Kitchell 2003, Young & Sheehan 2001). At the physiological level, the protective role of melanin pigmentation against UVR exposure derives from its ability to prevent direct and indirect (oxidative) damage to DNA at wavelengths where it is most vulnerable (Cleaver & Crowley 2002, Kielbassa et al. 1997, Shea & Parrish 1991).

Melanin pigmentation in human skin is considered as either constitutive skin color or facultative skin color (Quevedo et al. 1975). Constitutive skin color is the amount of genetically determined cutaneous melanin pigmentation that is generated without any influence of solar radiation (Jimbow et al. 1976, Quevedo et al. 1975). Facultative skin color or "tan" constitutes the short-lived, immediate, and delayed tanning reactions elicited by exposure to UVR (Jimbow et al. 1991, 1976; Quevedo et al. 1975). Lighter constitutive pigmentation is associated with a higher sunburn response, a lower tanning response, and a greater susceptibility to skin cancers (Kollias et al. 1991, Sturm 2002, Wagner et al. 2002).

Objective and reproducible assessment of melanin pigmentation has long been a goal of anthropology and dermatology. In anthropology, verbal descriptions of skin colors ("white," "yellow," "black," "brown," and "red") were replaced by color-matching methods during the early twentieth century (Olivier 1960, von Luschan 1897). The most popular of these methods was the von Luschan scale, based on the use of colored tablets or tiles of different colors and hues with which the colors of unexposed skin were matched. These and similar matching methods could not be consistently reproduced, however, and were swiftly abandoned when reflectance spectrophotometry was introduced in the early 1950s (Lasker

1954, Wassermann 1974). Reflectance spectrophotometry remains the method of choice for the objective study of skin pigmentation, color definition, and the spectral reflectance curves of skin because the incident light used and the distance between the light source and the subject are invariable and because subjective factors inherent in the visual matching methods are excluded (Wassermann 1974). All instrumental approaches to skin color evaluation depend on the illumination of the skin site by a standard light source at a fixed relative angle that minimizes the reflected light from the stratum corneum. The detector collects light re-emitted by the skin site from a particular angle and with a chosen color filter (Kollias 1995a). Because of the importance of assessing constitutive skin color on a part of the body that is not routinely exposed to sun, the inner (medial) surface of the upper arm has long been the standard reference site for studies of skin color. Portable reflectance spectrophotometers came into use with Weiner's (1951) study, with two types of instruments being commonly employed in anthropology during the latter part of the twentieth century. The instrument manufactured by the Evans Electroselenium Company (EEL) has been the most widely used, especially in studies of the skin colors of Old World peoples (Wassermann 1974), whereas that made by the Photovolt Corporation was more widely used in studies of New World peoples. Unfortunately, the skin reflectance measurements obtained by these two instruments are not directly comparable, requiring conversion formulae to make them so (Lees & Byard 1978). Research is now underway that may make possible the conversion of skin color assessments made by von Luschan color tablets to values comparable with those derived from reflectance spectrophotometry (M. Henneberg, personal communication).

In clinical medicine, constitutive skin color and skin sensitivity has been classified commonly according to skin phototypes or sun-reactive skin types, from Type I (very sensitive, easily burned, with little or no potential for tanning) to Type VI (insensitive, never burns, and deeply pigmented) (Fitzpatrick 1988, Fitzpatrick & Ortonne 2003, Jimbow et al. 1991). Skin type does not correspond well to constitutive skin color, however, and has limited applicability with respect to the responses of moderately or deeply pigmented skin (Kollias et al. 1991, Prota 1992c, Taylor 2002, Wagner et al. 2002, Westerhof et al. 1990). Despite these limitations, skin phototyping has been widely embraced by many clinicians because assessments can be made without instrumentation. In recent years, highly sensitive diffuse reflectance spectrophotometers such as the DermaSpectrometer and the Datacolor International Microflash as well as chromaticity meters have been used increasingly to measure skin pigmentation and skin response to UVR (Kollias 1995a, Wagner et al. 2002).

The photoprotective benefits of melanin have been assessed using several different measures including minimal erythemal dose (MED), DNA damage, and incidence of skin cancer (Kollias et al. 1991). The MED represents the minimum amount of UVR necessary to bring about a slight visible reddening of lightly pigmented skin. It is the easiest and most common method of assessing skin reactions to UVR but is difficult to determine for deeply pigmented individuals in whom

visual redness is difficult to assess (Kaidbey et al. 1979, Ortonne 2002, Shono et al. 1985).

Exposure of human skin to UVR results in a profound alteration of the metabolism, structure, and function of epidermal cells. These activities include increased activation of melanocytes, augmentation of melanosome production, an increase in the size of melanosome complexes incorporated within keratinocytes, and initiation of vitamin D synthesis (Parker 1981, Prota 1992a, Urbach 2001). The erythema response or sunburn reaction is related to constitutive skin color: Dark-skinned individuals can tolerate longer sun exposure than light-skinned individuals can. The skin of individuals with dark constitutive pigmentation exhibits a sun protection factor (SPF) of 10–15, whereas that of moderately pigmented people (e.g., from the circum-Mediterranean) achieves an SPF of only 2.5 (Kaidbey et al. 1979, Kollias et al. 1991, Ortonne 2002). In vitro studies of the reactions of human melanocytes to UVR have shown that heavily pigmented melanocytes have a greater capacity to resume cell division after irradiation with short wavelength UVR (UVB) than do their lightly pigmented counterparts, which suggests that they suffered less damage to their DNA (Barker et al. 1995). In contrast, UVB damages the immune system of the skin regardless of constitutive pigmentation by depleting both heavily and lightly pigmented skin of Langerhans cells (Cleaver & Crowley 2002, Kripke & Applegate 1991). The protective role of melanin in connection with skin cancer thus derives from its role in preventing damage to DNA in the first place, not in protecting against damage to the cutaneous immune system (Vermeer et al. 1991). Tanning or facultative pigmentation induced by UVR is photoprotective to some degree against the deleterious effects of further UVR exposure, but it does not significantly increase the SPF of individuals with light constitutive pigmentation or protect the DNA of their skin from UVR-induced damage (Kaidbey et al. 1979, Ortonne 2002). Although repeated exposure of tanned skin to UVR increases the number of metabolically active melanocytes and the intensity of melanogenesis (Lock-Anderson et al. 1998), the increased concentration of melanin in the tanned skin of inherently lightly pigmented people does not approach the photoprotection conferred by natural melanin in intrinsically darker-skinned people (Kaidbey et al. 1979). Individuals with lightly to moderately pigmented skin, who are repeatedly exposed to UVR, experience premature aging (photoaging) of the skin, which is characterized by wrinkling and anomalies of pigmentation (Chung 2001, Fisher et al. 2002, Kollias et al. 1991). This process is initiated by the photochemical generation of reactive oxygen species causing degradation of structural proteins in the dermis that confer strength and resiliency to the skin (Fisher et al. 2002).

Dermis

The dermis is a thick, dense fibroelastic connective tissue composed of collagen fibers, elastic fibers, and an interfibrillar gel composed of glycosaminoglycans, salts, and water. The primary cells of the dermis are collagen-rich fibroblasts. Collagen, which constitutes 77% of the fat-free dry weight of skin, largely accounts

for the tensile strength of the skin's fabric and for some of the ability of the dermis to scatter visible light (Kollias 1995a, Shea & Parrish 1991). Interwoven with the collagen is a network of abundant elastic fibers that restore the skin to its normal configuration after stretching. The dermis is equally thick in people with dark or light constitutive pigmentation (Taylor 2002).

The dermis encloses a widely ramifying network of blood vessels, an extensive nerve network, sweat glands, and a pilosebaceous complex of hair follicles and sebaceous glands (Figure 1). Of these, only the sweat glands are addressed in detail in this review because of their importance in thermoregulation.

The rich vascular supply of the skin is responsible for supplying the needs of the sweat glands, hair follicles, and rapidly multiplying epidermal cells in the stratum basale. The density of cutaneous blood vessels varies throughout the body's surface and is related to temperature and blood pressure regulation and the relative amounts of intermittent physical pressure different parts of the body must withstand, with the highest concentrations found in the skin covering the head, nipples, palms, soles, and ischial tuberosities (Edwards & Duntley 1939). The perineal skin of female macaques, baboons, and chimpanzees is richly suffused with blood vessels (Montagna 1967, Montagna 1971) that create large sexual swellings advertising the female's state of reproductive receptivity and lifetime reproductive potential (Domb & Pagel 2001). The oxygenated and deoxygenated forms of hemoglobin carried in the skin's blood vessels are some of the skin's main pigments, with a person's skin color determined mainly by the skin's melanin and hemoglobin content (Edwards & Duntley 1939). The erythema or strongly red appearance of the skin caused by exposure to UVR is the result of increases in the number and diameter of vascular capillaries through which blood is flowing and an increase in the blood flow through each capillary (Kollias 1995a). Sunburned skin feels hot to the touch because of the increased vascularization of the skin and the inflammatory response mounted by the skin as it works to repair UVR-induced damage (Ryan 1991, Shea & Parrish 1991).

The nerve supply of the skin is highly complex because the skin is a major sensory surface that contains varied types of receptors sending signals to the central nervous system about the external environment and the internal state of the skin (Chu et al. 2003, Lynn 1991). These receptors include two types of temperature sensors, diverse mechanoreceptors associated with both hairy and glabrous skin, and an important group of cutaneous sensory cells (nociceptive afferents) specialized for the detection of tissue-threatening stimuli or the presence of injury or inflammation (Lynn 1991). The glabrous skin of the hands and feet of primates is densely packed with sensory nerve endings that permit highly sensitive tactile discrimination and exquisite differentiation of temperature and texture (Chu et al. 2003, Lynn 1991, Martin 1990). These attributes greatly enhance the manipulative functions of these appendages, especially the hand (Martin 1990).

Numerous hairs, which grow from hair follicles located in the dermis, are associated with mechanoreceptors and sebaceous glands. Hair performs a range of

functions from insulation, to protection against the sun, enhancement of cutaneous sensation to communication of emotion (through piloerection), and ornamentation (Lavker et al. 2003; Montagna 1967, 1971; Wheeler 1984, 1985). Humans are unique among primates in possessing effectively naked skin, except on the scalp, the male chin, the axilla, and the groin. Although human skin bears millions of hairs, most of them are so small as to be nearly invisible (Montagna 1981).

SWEAT GLANDS   Human dermis contains two main types of sweat glands, eccrine and apocrine. The former are widely distributed throughout the surface of the body, whereas the latter are concentrated in the axilla, perineum, and external auditory canal. Eccrine glands are tubular in form (Figure 1) and lie in the outer portion of the dermis. They produce copious amounts of dilute, watery fluid expressed to the surface of the skin through an individual pore. Humans have two to four million eccrine glands on the surface of their bodies, with an average distribution ranging from ~150–340/cm^2 (Folk & Semken 1991, Goldsmith 2003). Both apocrine and eccrine sweat glands are stimulated by the sympathetic division of the autonomic nervous system and produce sweat in response to thermal stimulation (thermal sweating). In contrast, the eccrine glands of the palms and soles respond only to emotional stimuli, whereas those of the face and axilla respond to both (Folk & Semken 1991, Zihlman & Cohn 1988).

Considerable attention has been placed on comparisons of the quantity, structure, and function of sweat glands between human groups. The number of strictly controlled comparisons between members of different populations after equivalent periods of deliberate acclimatization is quite small (Weiner 1977). The results of most rigorous comparative study of sweat gland densities in humans (Knip 1977) indicate that only small differences in the total number and average density of sweat glands exist between disparate human populations. As yet it has proven virtually impossible to design studies that can determine conclusively whether differences in sweating performance between human groups are due to genetic influences or environmental adaptations.

The Skin in Thermoregulation

Dissipation of heat is the function that most conspicuously distinguishes human skin from that of all other animals (Montagna 1981). The reasons for the evolution of this unique capacity are discussed in the following section. Humans encounter heat stress more or less year round in equatorial areas and for varying lengths of time in the rest of the world except for circumpolar and alpine environments. Heat stress is exacerbated by prolonged or rigorous exercise. Maintenance of homeostasis requires that the body's core temperature remain close to a neutral point, which varies from about 36.8 to 37.2°C, in order to permit uninterrupted functioning of the temperature-sensitive cells of the human central nervous system. If the rates of production or loss of heat are excessively out of balance, core

temperature can quickly increase or decrease to dangerous levels (Kraning 1991, Wenger 2003).

Temperature regulation in humans includes involuntary (physiologic) and voluntary (behavioral) activity (Wenger 2003). Voluntary temperature regulation involves the conscious actions taken by people to maintain thermal comfort, including the seeking of shade and shelter, and the wearing or shedding of clothing.

Involuntary temperature regulation in the skin has been studied in great detail in the past 50 years by both physiologists and anthropologists, and only a superficial summary of this corpus of work is presented here. Regulation of temperature by the skin is accomplished through its roles in (*a*) perceiving and transmitting its own temperature to the central nervous system; (*b*) regulating heat transfer between the body's core and the skin through the cutaneous circulation; (*c*) serving as a superficial casing through which body heat is conducted from the vascular layers to the surface; (*d*) acting as an interface for the loss or gain of heat to or from the environment by radiation, convection, or conduction; and (*e*) acting as a surface for the spreading of sweat necessary for evaporative cooling (Frisancho 1981, Kraning 1991). The relative role of the four avenues of heat loss (radiation, convection, conduction, and evaporation) depends on the interaction of the ambient temperature and humidity (Chaplin et al. 1994; Frisancho 1981; Wenger 2003; Wheeler 1984, 1991b). The ability of sweat glands to respond to heat stress is adversely affected by sunburn (Pandolf et al. 1992). Protection of the integrity of sweat glands against damage caused by UVR, therefore, has been of great importance during the long course of human habitation of the tropics.

Experimental studies and simulations undertaken to determine how thermal homeostasis is maintained under the stressful environmental conditions of the tropics have shown that heat loss is maximized in people with a high ratio of skin surface area to body weight, such as Nilotic tribespeople, the Kung San, and Australian Aborigines (Frisancho 1981; Wheeler 1991a,b, 1992). This relationship supports Allen's Rule in mammals, which states that mammals living in cold regions will minimize the size and surface area of their extremities, whereas those inhabiting hot areas will increase the relative size of appendages.

The Role of the Skin in Vitamin D Biosynthesis

Synthesis of vitamin D in the skin of vertebrates is the only unanimously agreed positive effect of UVR exposure. Vitamin D_3 is the form of vitamin D that is synthesized by vertebrates, whereas vitamin D_2 is the primary form found in plants (Holick 2003). Vitamin D_3 is more accurately characterized as a prosteroid hormone than as a vitamin because, in mammals, it is derived from a cholesterol-like precursor (7-dehydrocholesterol) found in the skin (Holick 2003). Vitamin D is a unique natural product thought to have first occurred on Earth as a photosynthetic product in marine phytoplankton more than 750 mya (Holick 1995). Although the physiological role of vitamin D in plants and invertebrates is not clear, vitamin D was essential for the evolution of terrestrial vertebrates (Holick 1991, 1995).

Holick has reasoned that early tetrapods depended on vitamin D for the efficient use of scarce dietary calcium to preserve their rigid calcified skeletons (Holick 1995). Vitamin D can be synthesized only by a photochemical process, so early tetrapods could only satisfy their body's vitamin D requirements by exposing themselves to sunlight to photosynthesize vitamin D in their own skin or by ingesting foods containing vitamin D (Holick 1995).

Vitamin D_3 synthesized in the skin requires successive hydroxylations in the liver and kidney to be converted to its biologically active form, 1α, 25-dihydroxy-vitamin D_3 (Holick 1991, Jones et al. 1998). This functionally active form is important for the regulation of calcium and phosphorus metabolism, skeletal development and mineralization, the regulation of normal cell growth, and the inhibition of cancer cell growth (Holick 1991, 2001). The production of vitamin D_3 is optimally stimulated by UVR wavelengths of 295–300 nm, in the UVB range (MacLaughlin et al. 1982). High-energy UVB photons penetrate the skin and are absorbed by the 7-dehydrocholesterol in the keratinocytes of the epidermis (especially of the strata basale and spinosum) and fibroblasts of the dermis, catalyzing the formation of previtamin D_3 (Holick 2001, Webb et al. 1988). Once formed in the skin, previtamin D_3 can undergo isomerization to vitamin D_3 at body temperature and then undergo further chemical conversions to 1α, 25-dihydroxyvitamin D_3. The conversion of previtamin D_3 or vitamin D_3 to the functionally active form is rate-limited, however. In the presence of biologically sufficient amounts of 1α, 25-dihydroxyvitamin D_3 in the circulation, previtamin D_3 and vitamin D_3 are transformed by UVA or UVB into a variety of inert byproducts, thus averting overproduction of the biologically active form and subsequent "vitamin D intoxication" (Holick 2001, Holick et al. 1981). This finding disproves the hypothesis that dark constitutive skin pigmentation evolved in the tropics as an adaptation to protect against the overproduction of 1α, 25-dihydroxyvitamin D_3 (Loomis 1967).

Melanin pigments are highly effective at absorbing and scattering the UVB wavelengths that catalyze vitamin D_3 synthesis. Thus, high concentrations of melanin in the skin result in a decrease in the efficiency of conversion of 7-dehydrocholesterol to previtamin D_3; pigmentation slows but does not prevent cutaneous production of the vitamin (Holick et al. 1981, Webb et al. 1988). Individuals with very deep constitutive pigmentation often require 10 to 20 times longer exposure to sunlight than those of lighter pigmentation in order to promote an adequate synthesis of vitamin D_3 (Holick et al. 1981). This finding explains why dark-skinned individuals living at high latitudes with low levels of environmental UVB are at greater risk of vitamin D_3–deficiency diseases than are light-skinned people (Clemens et al. 1982, Holick 2001, Mitra & Bell 1997). The evolutionary significance of this observation is discussed further below. The photoconversion of 7-dehydrocholesterol to previtamin D_3 in the skin is also adversely affected by increasing age (Holick 1995), the wearing of clothing (Matsuoka et al. 1992), and by the use of topical sunscreens, which block the UVB wavelengths responsible for both sunburn and vitamin D_3 production (Holick 1997, Webb et al. 1988).

THE EVOLUTION OF MODERN HUMAN SKIN

Reconstruction of the evolution of human skin relies on evidence provided by comparative anatomy and physiology, as well as study of the evolution of the genes and gene complexes that determine the function and pigmentation of skin. Using basic principles of historical morphology, one can reconstruct the major steps in the evolution of human skin by utilizing a well-established phylogeny to examine historical transformations of structure and function (Jablonski & Chaplin 2000). This method leads to the reconstruction of the probable appearance of the skin in the last common ancestor of the human and chimpanzee lineages as being lightly pigmented and covered with dark hair, like most catarrhine primates today (Jablonski & Chaplin 2000).

The skin of modern humans is distinguished from that of other primates mainly by its naked appearance, its greatly enhanced abilities to dissipate body heat through sweating, and by the great range of genetically determined skin colors present within a single species. Most investigators have considered these attributes to be adaptations forged by natural selection.

The Evolution of the Thermoregulatory Properties of Human Skin

Human skin is not hairless, but—as discussed above—the hairs over most of the body's surface are so fine and present at a sufficiently low density that the skin appears essentially naked. Explanations for the evolution of human hairlessness have been many, varied, and often highly creative. The most cogent explanations are based on the importance of a functionally naked skin in maintaining body temperature in hot environments.

Many animals, including primates, which live in hot environments, have heavy coats of insulating fur or feathers. In the heat caused by strong sunlight, such insulation reduces environmental heat gain (Folk & Semken 1991, Walsberg 1988). This is the case even for black coats, which absorb short-wave radiation near or at the surface of the fur and reradiate large amounts of long-wave radiation before it reaches the skin (Dmi'el et al. 1980). The effectiveness of fur insulation in reducing environmental heat gain is lessened by sweating. The most efficient evaporative cooling occurs at the skin's surface; in heavily furred animals, water vapor is transferred through the fur to the atmosphere (Folk & Semken 1991). If the fur is wet from sweating, however, maximum evaporation occurs at the surface of the fur, and heat from the blood vessels cannot be transferred as efficiently to the site of evaporation (Folk & Semken 1991). Under these circumstances, much more water must be used for evaporative cooling. Thermal sweating as a method of cooling becomes more important as environmental temperatures rise or as activity levels increase because the lower gradient between core and environmental temperatures restricts the amount of heat loss that can be achieved by radiation, convection, and conduction (Frisancho 1981, Wheeler 1991b). Removal of excess heat is,

therefore, greatly facilitated by the loss of body hair because it increases thermal conductance and permits additional heat loss through sweating (Wheeler 1985, Zihlman & Cohn 1988).

A strong case can be made for the evolutionary loss of apocrine sweat glands in humans because these sweat glands are most common in heavily furred animals (Folk & Semken 1991). The African apes exhibit a ratio of approximately 40% apocrine sweat glands to 60% eccrine; the great preponderance of eccrine sweat glands in modern humans probably evolved under the strong influence of natural selection, following the loss of the apocrine component to sweating (Folk & Semken 1991, Montagna 1981, Zihlman & Cohn 1988). This process was probably propelled by increases in body size and activity levels associated with modern limb proportions and striding bipedalism, which occurred in the transition from the primitive hominins of the late Miocene to the genus *Homo* of the Plio-Pleistocene (Chaplin et al. 1994; Folk & Semken 1991; Jablonski & Chaplin 2000; Montagna 1981; Schwartz & Rosenblum 1981; Wheeler 1984, 1996).

The importance of body cooling through the skin in modern humans has been emphasized repeatedly by both physiologists and anthropologists because of the primacy of preventing hyperthermia and attendant damage to the central nervous system (Cabanac & Caputa 1979, Falk 1990, Wheeler 1984, Zihlman & Cohn 1988). The temperature of the brain closely follows arterial temperature, requiring that the temperature of the circulating blood be carefully regulated (Nelson & Nunneley 1998). This process became increasingly important as activity levels and brain size increased in the genus *Homo* through the Pleistocene. Simulations and experimental studies have confirmed that maintenance of stable core temperature under conditions of increased environmental heat load or exercise is best accomplished via recruitment of a whole-body cooling system, involving cooling of peripheral blood vessels through sweating (Desruelle & Candas 2000, Nelson & Nunneley 1998). A recently mooted hypothesis that human hairlessness evolved late in human evolution as a result of the adoption of clothing and the need to reduce the load of external parasites (Pagel & Bodmer 2003) finds no support in light of the overwhelming evidence of the importance of hairlessness in thermal sweating and whole-body cooling in maintaining stable core temperature and homeostasis.

The Evolution of Human Skin Pigmentation

RECONSTRUCTION OF SKIN COLOR IN EARLY *HOMO*   The early members of the genus *Homo* from the late Pliocene and Early Pleistocene of Africa exhibited larger bodies, relatively larger brains, and relative longer lower limbs than did their australopithecine predecessors (McHenry & Berger 1998; Ruff et al. 1993, 1997). The higher activity levels and larger day ranges reconstructed for them (Wheeler 1991a, 1992) would have required that their skin be functionally naked and endowed with a high density of eccrine sweat glands in order to facilitate heat loss (Jablonski & Chaplin 2000, Wheeler 1984). This situation created a new physiological challenge for human skin: protection of a naked integument against

UVR. Dense hairy coats protect the skin of mammals from UVR-induced damage to the skin because the hairs themselves absorb or reflect most short-wavelength solar radiation. In mammals with sparse coats of hair, however, 3%–5% of incident UVR is transmitted to the skin (Walsberg 1988). Nonhuman mammals that are active in hot, sunny environments exhibit sparse coats because they facilitate passive heat loss; they also display highly melanized skin on their exposed (dorsal) surfaces to effectively block the UVR transmitted to the skin (Walsberg 1988). This evidence clearly indicates that hair loss in the human lineage was coupled with increased melanization of the skin as activity levels in hot environments increased. The early members of the genus *Homo*, the ancestral stock from which all later humans evolved, were, thus, darkly pigmented (Jablonski & Chaplin 2000). This interpretation has recently been supported by genetic evidence demonstrating that strong levels of natural selection acted about 1.2 mya to produce darkly pigmented skin in early members of the genus *Homo* (Rogers et al. 2004).

Heavily pigmented skin does not, in fact, perceptibly increase the body's heat load under conditions of intense solar radiation (Baker 1958, Walsberg 1988). This is because for half of the solar radiation reaching the Earth's surface—in the infrared—there is essentially no difference in absorption between dark and light skin (Baker 1958, Daniels 1964). This evidence negates the claim by Blum (1961) and others (Morison 1985) that heavily melanized pigmentation in humans could not be adaptive in the hot tropics because of the increased heat load caused by greater amounts of absorbed solar radiation.

SKIN PIGMENTATION IN MODERN HUMAN POPULATIONS   Many of the accounts of travelers and explorers from the fifteenth century onward include reports of the skin color of the peoples they encountered. As natural historians and human geographers—mostly from Europe—ventured into Asia, Africa, Australia, and the Americas and began to study the indigenous human populations in detail, maps depicting the worldwide distribution of human skin color were slowly assembled. The best known of these maps is that composed by the Italian geographer Renato Biasutti, which was based on the von Luschan skin color scale. This map has gained broad circulation in several widely distributed publications (Barsh 2003, Lewontin 1995, Roberts 1977, Walter 1971), despite the fact that, for areas with no data, Biasutti simply filled in the map by extrapolation from findings obtained in other areas (Robins 1991). A more accurate and exhaustive compilation of the skin colors of indigenous peoples based only on published skin reflectance measurements is now available (Jablonski & Chaplin 2000). Both maps show similar trends, with darkly pigmented peoples found near the Equator and incrementally lighter ones found closer to the Poles. A larger percentage of people with dark skin is found in the Southern Hemisphere as compared with the Northern Hemisphere (Relethford 1997) because of a latitudinal bias in the distribution of land masses (Chaplin & Jablonski 1998).

The data compiled by Jablonski and Chaplin also provide conclusive evidence of sexual dimorphism previously observed in human skin pigmentation (Frost

1988, van den Berghe & Frost 1986), with females being consistently lighter than males in all populations studied (Jablonski & Chaplin 2000).

One of the major problems encountered in assembling data on the distribution of human skin color in indigenous populations is determining exactly what an indigenous population represents. For most anthropologists and human geographers, an arbitrary cutoff date of 1500 has been adopted to distinguish native or indigenous peoples from immigrant populations. This date is reasonable with respect to the inauguration of the modern era of European colonization but fails to recognize the several major movements of human groups within continents (such as the so-called Bantu expansion within Africa) that occurred before 1500. These movements, along with European colonization and the increasingly rapid and distant migrations of human populations through time, have fundamentally altered the human landscape established in prehistoric times. This has made the interpretation of geographically and biologically significant trends in human populations much more difficult.

ENVIRONMENTAL CORRELATES OF HUMAN SKIN COLOR   The skin pigmentation of indigenous human populations shows remarkable regularity in its geographic distribution. Darker skins occur in more tropical regions and lighter skins in temperate, although the gradient is less intense in the New World as compared to the Old World. Even within Africa, the continent with the largest equatorial land mass, there is considerable heterogeneity of skin color, with the deepest colors occurring not in the lowest latitudes but in the open grasslands (Chaplin 2001, Roberts 1977). The strong latitudinal signal in skin color led most early workers to conclude that skin pigmentation represented an adaptation to sunlight or other solar-driven phenomena such as temperature. Walter (1958, 1971) was the first researcher to suggest that the pigmentation gradient observed was linked to the intensity of UVR, and he established this relationship by calculation of correlation coefficients between skin color (as measured on the von Luschan scale) and estimated UVR. The relationship between skin color and environment was further explored by studies in which the relationship of skin color, as measured by reflectance spectrometry, to latitude, temperature, and humidity was studied by correlation and regression analyses (Roberts 1977, Roberts & Kahlon 1976). These analyses showed the dominant association of skin reflectance with latitude, which was then deduced to be an effect related to the intensity of UVR (Roberts 1977, Roberts & Kahlon 1976).

In recent years, studies of the relationships between morphological and physiological variation and attributes of the physical environment have been advanced by the availability of remotely sensed data on levels of UVR, total solar radiation, temperature, humidity, precipitation, and other environmental variables at the Earth's surface. These data, which were not widely available to workers before 1990, have permitted correlation, regression, and other analyses of skin reflectance to be conducted against actual measurements, rather than estimates, of environmental variables (Chaplin 2001, 2004; Jablonski & Chaplin 2000).

Using data on the minimal erythemal dose of UVR (UVMED) at the Earth's surface collected by the NASA TOMS 7 satellite, Jablonski & Chaplin were able to establish a conclusive correlation between latitude and annual average UVMED, and thence between annual average UVMED and skin reflectance (Jablonski & Chaplin 2000). This publication was followed by a study in which the influence of minimum, maximum, and seasonal levels of UVR, as well as other directly measured environmental variables, relative to skin reflectance were studied (Chaplin 2001, Chaplin 2004). This study showed that skin reflectance was correlated with autumn levels of UVMED, and that skin reflectance could be almost fully modeled as a linear effect of this variable alone (Chaplin 2001, 2004). This study also showed that the relationship between summer levels of UVMED and skin reflectance appeared to reach a threshold past which higher levels of UVR were not correlated with incrementally lower skin reflectance (darker pigmentation) (Chaplin 2001, 2004).

Low reflectance values for human skin (dark pigmentation) are primarily a function of UVMED (Jablonski & Chaplin 2000), with regression analysis demonstrating that autumn UVMED levels have the strongest effect. This indicates that skin color is more strongly correlated with UVA, which is consistently higher throughout the year at all latitudes, than with UVB (Chaplin 2001, 2004). Maximum UVMED had the next most significant effect (Chaplin 2001, 2004). Winter levels of precipitation have the opposite effect, being positively correlated with high reflectance values (light pigmentation) (Chaplin 2001, 2004). Multiple regression formulae relating skin reflectance to these environmental parameters can then be used to derive a map of predicted human skin colors, with the colors shown being realistic approximations of the true color of skin (Chaplin 2001, 2004) (Figure 3). This map depicts an idealized situation in which humans worldwide are assumed to have inhabited their respective regions for the same lengths of time, and have followed similar cultural practices that could affect skin color (e.g., diet, activity schedules, use of clothing and shelter).

NATURAL SELECTION AND THE EVOLUTION OF HUMAN SKIN PIGMENTATION   The geographical distribution of human skin colors has invited many explanations, most of which have claimed melanin pigmentation to be an adaptation to some attribute of the physical environment that varies primarily by latitude. Ever since the harmful effects of UVR began to be appreciated by scientists, explanations for the evolution of deeply melanized skin have centered on the importance of resistance to sunburn, solar degeneration, and skin cancer (Daniels et al. 1972). Equally popular has been the vitamin D hypothesis, which stated that lightly pigmented skins were necessary outside of the tropics in order to permit vitamin D biosynthesis in the skin by low levels of UVR, whereas darkly pigmented skin afforded protection against production of toxic doses of vitamin D in equatorial regions (Loomis 1967). Lightly pigmented skin has also been explained as an adaptation to resist cold injury, on the basis of experimental and epidemiological data that have documented more severe injuries incurred by pigmented skin exposed to

freezing conditions (Post et al. 1975, Steegmann 1967). Other explanations have imputed highly melanized skin as providing effective concealment in habitats such as tropical forests with differing light intensities and environmental illumination (Cowles 1959, Morison 1985), and still others have reasoned that tropical diseases and parasites rather than tropical climate were the major selective forces leading to the evolution of differential pigmentation in humans (MacKintosh 2001; Wassermann 1981, 1965a).

Although adaptive explanations for human pigmentation have dominated the literature, others have downplayed or discounted the role of adaptation by natural selection. Some workers have emphasized the role of sexual selection, especially by way of explaining the lighter constitutive pigmentation of females relative to males (Aoki 2002, Frost 1988). Deol claimed that differences in skin color between human populations were the pleiotropic byproducts of natural selection on other functions of pigmentation genes (Deol 1975). Others have simply discouraged the "amusing pastime" of adaptive reconstruction in the absence of data on the differential survival and reproduction of varying skin pigmentation phenotypes (Blum 1961, Lewontin 1995). Adaptive explanations "for" any given phenotypic trait require demonstration that the trait increases the real or probable reproductive success of the organism. Although such evidence is often difficult to muster in the case of traits borne by long-lived mammals, it is incumbent that adaptive reconstructions be tethered by this responsibility.

In the past, adaptive explanations for different levels of melanin pigmentation in human skin have suffered from an inability to demonstrate probable or real differences in survivorship and reproduction of different skin color phenotypes under the same environmental conditions. Blum introduced this mode of critical appraisal of competing hypotheses when he drew attention to the fact that dark skin pigmentation could not have evolved primarily as adaptive protection against skin cancer because such cancers rarely cause death during peak reproductive years (Blum 1961, Jablonski & Chaplin 2000). Other adaptive explanations for light or dark skin pigmentation (e.g., protection against cold injury; camouflage) have similarly failed to demonstrate real or probable increases in reproductive success as a result of possession of these phenotypes.

MELANIN AS A REGULATOR OF THE PENETRATION OF UVR INTO THE SKIN Recently, Jablonski & Chaplin (2000) published a new adaptive hypothesis for the evolution of human skin pigmentation stating that melanin pigmentation evolved to regulate the penetration of UVR into the skin in order to prevent the photolysis of photo-labile compounds while permitting the photosynthesis of others. This hypothesis was based on two equally important observations: (*a*) that the B vitamin folate is destroyed by long wavelength UVR (UVA), and that folate deficiencies can markedly reduce individual reproductive success by adversely affecting cell division; and (*b*) that vitamin D_3 is synthesized in the skin by short wavelength UVR (UVB) and that severe vitamin D deficiencies adversely affect reproductive success by interfering with normal calcium metabolism (Jablonski & Chaplin

2000). Natural selection has produced two opposing clines of skin pigmentation. The first is a cline of photoprotection that grades from darkly pigmented skin at the Equator to lightly pigmented skin near the Poles. The second is a cline of vitamin D_3 photosynthesis that grades from lightly pigmented near the Poles to darkly pigmented at the Equator. In the middle of the two clines we find peoples with enhanced abilities to develop facultative pigmentation according to seasonal UVR levels.

THE FOLATE HYPOTHESIS   The potential importance of dark skin pigmentation in protecting folate from UVR-induced photolysis was first recognized upon discovery that folate undergoes photolysis in vitro when subjected to UVA (360 nm) and that serum folate levels of human subjects dramatically declined when humans underwent long-term exposure (minimum of 3 months) to the same wavelength, for 30–60 min once or twice a week (Branda & Eaton 1978). The potential significance of the finding to the evolution of human skin pigmentation was echoed later, but a causal mechanism was not mooted (Zihlman & Cohn 1988).

Few nutrients compare with folate (folic acid) for its impact on health. Adequate folate status is vital for the synthesis, repair, and expression of DNA, and therefore for all processes involved in cell division and homeostasis (Kesavan et al. 2003, Lucock et al. 2003, Suh et al. 2001). The subtle influence of folate on the cell's genomic machinery has led to the realization that even marginal folate deficiencies may have significance in developmental disorders and degenerative diseases associated with high morbidity and mortality (Lucock et al. 2003). Now that folate deficiency is widely acknowledged as a risk factor for neural tube defects, recurrent early pregnancy loss, and other complications of pregnancy, the maintenance of adequate folate status in women of reproductive age has become a primary public health concern (Bower & Stanley 1989, Fleming & Copp 1998, Minns 1996, Suh et al. 2001). Folate's importance in spermatogenesis also highlights its important role in maintaining male reproductive competence (Cosentino et al. 1990, Mathur et al. 1977).

The recognition of folate's pivotal roles in DNA synthesis and repair—and thus most processes associated with reproductive success in both sexes—has underlined the importance of protecting the body's folate stores from physical or chemical degradation. Because folate is susceptible to oxidative damage as a result of exposure to UVR and ionizing radiation (Branda & Eaton 1978, Hirakawa et al. 2002, Kesavan et al. 2003), the primary evolutionary function of melanin in regions receiving high annual UVR is to protect folate from photodegradation (Jablonski & Chaplin 2000). Photolysis of folate has been experimentally demonstrated at 340 nm and 312 nm, in the UVA and near-UVA wavelengths (Hirakawa et al. 2002, Lucock et al. 2003). With skin reflectance being most closely correlated with autumn levels of UVMED dominated by UVA, one can conclude that the longer wavelengths of UVR, which are capable of penetrating deep into the dermis of the skin, have been the most important agents of natural selection in connection with the evolution of skin pigmentation (Chaplin 2001) (Figure 4). The results of
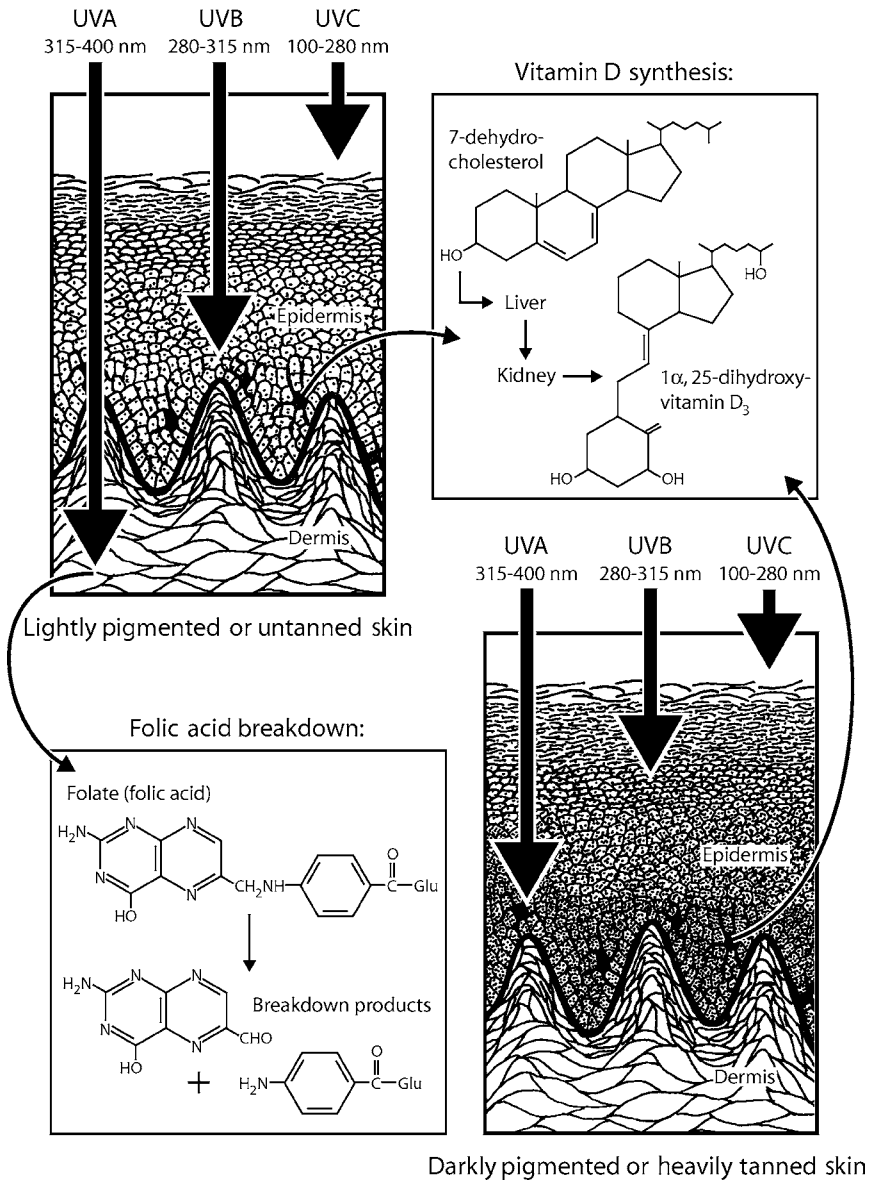
Figure 4   The effects of UV radiation on the skin. Different wavelengths of UVR penetrate to different thicknesses in the skin, with UVA penetrating more deeply than UVB. UVC generally does not penetrate the Earth's atmosphere.

a recent study (Gambichler et al. 2001) did not confirm the photolytic effect of UVA on serum folate levels in a small number of human volunteers. This finding runs counter to the results of previous in vivo and in vitro studies demonstrating profound photodegradation of folate upon exposure to UVR (Hirakawa et al. 2002, Lucock et al. 2003) and to X- and γ-irradiation (Kesavan et al. 2003). A true and statistically robust test of the folate hypothesis would require a case-control study involving a large number of human volunteers experiencing long-term (once or twice a week for a minimum of three months) exposure to UVR, with measurement of more labile folate species such as specific red cell folate coenzymes (Lucock et al. 2003).

SKIN PIGMENTATION AND VITAMIN D BIOSYNTHESIS   In the millennia prior to about 1.6 mya, the earliest members of the genus *Homo* appear to have been restricted in their distribution to the high-UVR regimes of equatorial Africa. Under these environmental conditions, possession of highly melanized skin was critical for survival. As populations of early *Homo* moved both northward and southward, they began to experience different schedules and intensities of UVR exposure.

UVR levels at the Earth's surface are affected by latitude, altitude, season, moisture content, cloud cover, the depth of the ozone column, orbital parameters, and other factors (Hitchcock 2001, Madronich et al. 1998). Short wavelength UVR (UVB, 280–315 nm) is more effectively absorbed by atmospheric ozone than are longer wavelengths (UVA, 315–400). Thus, as one moves away from the Equator and the angle of solar elevation decreases, the thickness of the atmosphere (including the ozone layer), through which sunlight must pass, increases. This results in a greater attenuation of UVR, especially of UVB, by scattering and absorption by ozone, and consequently very low levels of UVB in high-latitude ecosystems (Caldwell et al. 1998). Very small increments or decrements of UVB lead to substantial biological effects (Madronich et al. 1998); thus, it is highly biologically significant that regions north and south of 50° latitude receive only tiny doses of UVB, and only then at the peak of summer (Caldwell et al. 1998, Chaplin 2001, Johnson et al. 1976, Neer 1985).

As discussed earlier, deeply melanized skin confers excellent protection against the deleterious effects of UVR, but it also greatly slows the process of vitamin D_3 synthesis in the skin. As hominins moved out of the tropics, their exposure to UVR—especially to vitamin D–inducing UVB—was dramatically reduced. Levels of UVR at the Earth's surface are not thought to have been appreciably different in the Pleistocene as compared to today because similar conditions of solar emissivity and orbital parameters existed at the time, and similar levels of UVR have been reconstructed from biological proxies (Rozema et al. 2002). Even before remotely sensed data on UVB levels outside of the tropics were available, theorists surmised that early humans living in high latitudes with deeply pigmented skin would have not been able to produce sufficient vitamin D_3 in their skin to meet their physiological demands and that strong selective pressure for depigmentation

of the skin had been exerted in order to facilitate photosynthesis of vitamin D_3 (Loomis 1967, Murray 1934, Neer 1975).

Using known values of UVMED at the Earth's surface (Herman & Celarier 1996) and the precise dosage of UVB necessary to catalyze vitamin D synthesis in human skin at a specific latitude (Webb et al. 1988), researchers can calculate the worldwide potential for vitamin D_3 synthesis for lightly pigmented skin (Jablonski & Chaplin 2000) (Figure 5). Zone 1 (shown without hachure in Figure 5) corresponds closely to the tropics and represents an area in which there is adequate UVR throughout the year to catalyze vitamin D_3 synthesis in the skin (Jablonski & Chaplin 2000). Zone 2 (area covered by vertical hachure in Figure 5) represents the region in which there is insufficient UVR during at least one month of the year to produce vitamin D_3, and Zone 3 (cross-hatched area of Figure 5) represents that in which there is insufficient UVR, as averaged over the entire year, to photosynthesize vitamin D_3 in the skin (Jablonski & Chaplin 2000). The configuration of vitamin D synthesis zones for darkly pigmented skin differs markedly from this depiction, with Zone 1 being greatly reduced in area, and Zones 2 and 3 significantly expanded because of the attenuation of UVB absorption by dark melanin pigmentation and concomitant prolongation of the length of UVB exposure required for vitamin D_3 biosynthesis (Jablonski & Chaplin 2000). This analysis clearly demonstrates the profound impact of constitutive pigmentation on the potential for vitamin D_3 synthesis in the skin. An empirical demonstration of this was recently provided by a school population of darkly pigmented and albino children in South Africa, in which the former group of children required a significantly higher dietary intake of vitamin D_3 to attain the same levels of vitamin D_3 and plasma calcium than did the albinos (Cornish et al. 2000). The importance of the synthesis and physiological activity of vitamin D have been further born out by studies of the worldwide polymorphism in the vitamin D–binding protein (or group-specific component, *Gc*) that show a clear relationship between the frequency of specific *Gc* alleles and levels of sunlight (OMIM 2003).

Vitamin D_3 insufficiency and deficiency can exert sinister effects on the body throughout life and have the demonstrated potential to reduce fitness when they afflict children and adolescents. The most serious and notorious of the vitamin D_3–deficiency diseases is rickets, caused by a failure of mineralization in the cartilaginous matrix of developing bones as a result of calcium and phosphate malabsorption (Shaw 2003, Wharton & Bishop 2003). Comprehensive clinical descriptions of rickets (Bereket 2003, Holick 1995, Shaw 2003, Wharton & Bishop 2003) catalog the devastating osseous and nonosseous effects of the disease on children and adolescents, including the delayed closure of fontanelles, bowing of the lower limb bones, and narrowing of the pelvic outlet in females, which can cause obstructed labor and a high incidence of infant and maternal morbidity and mortality. Vitamin D_3 deficiency in adults produce osteomalacia, a softening of the bone matrix, but inadequate vitamin D_3 status in pregnant women contributes to hypocalcemia and rickets in their babies (Wharton & Bishop 2003). The deleterious effects of vitamin D_3 deficiency encompass a suite of problems affecting

evolutionary fitness, including those involving the formation and maintenance of the skeleton, control of normal cell growth, inhibition of cancerous cell growth, and maintenance of normal immune system function (Grant 2002; Holick 1991, 2001; Wharton & Bishop 2003). An important, but little reported consequence of vitamin D deficiency in laboratory mice and rats is a marked reduction in female fertility and female reproductive failure apparently due to failure of vitamin D to interact normally with its receptor on the ovary (Jones et al. 1998).

An abundance of clinical and epidemiological evidence now supports the argument that depigmentation of the skin evolved in humans living outside of the tropics because of the importance of maintaining adequate vitamin D_3 production in the skin for as long as possible throughout the year. Alterations in the function of the vitamin D endocrine system in darkly pigmented people as a consequence of diminished exposure to sunlight result in vitamin D_3 insufficiency and deficiency, as recently reviewed elsewhere (Mitra & Bell 1997, Wharton & Bishop 2003). These problems potentially afflict dark-skinned people who have migrated to or who inhabit UVB-poor regions (e.g., northern Europe, the northern United States, or Canada) or darkly pigmented people living in sunny regions who habitually stay indoors or consistently wear concealing clothing when outdoors (Atiq et al. 1998, Bereket 2003, Brunvand & Haug 1993, Fogelman et al. 1995, Fonseca et al. 1984, Gessner et al. 1997, Hodgkin et al. 1973, Holick 1995, Wharton & Bishop 2003). In these populations, vitamin D_3 deficiency is exacerbated by breast feeding because of the low concentration of vitamin D_3 in human breast milk (Gessner et al. 1997, Shaw 2003, Wharton & Bishop 2003).

Vitamin D_3 insufficiency and deficiency also afflict lightly pigmented people who are not exposed to sufficient sunlight because of occupation, advanced age, or hospitalization, or people who consistently wear protective clothing or sunscreen when outdoors (Holick 1995, 1997, 2001; Thomas et al. 1998). Rickets (known to many as the English disease) was, in fact, first recognized as a disease of light-skinned children living in dark, multistoried structures devoid of sunlight (Holick 1991, 1995).

Brace (1963) argues that depigmentation of human skin occurred not as the result of active selection for lighter pigmentation, but because of the relief of selective pressure on pigmentary systems as humans populated increasingly high latitudes where dark pigmentation was no longer required as a shield against UVR. This structural reduction hypothesis is based on the "probable mutation effect" whereby mutations in the genes controlling melanin pigmentation would accumulate, leading to reduction of or failure to produce melanin (Brace 1963). A recent variation on this argument states that where natural selection for dark skin is sufficiently weak, a sexual preference for lighter skin could have driven the evolution of light skin (Aoki 2002, Ihara & Aoki 1999) (see below also). These arguments find limited support today with respect to the evolution of human skin coloration in light of the impressive body of recent clinical evidence cited above that attests to the many and highly significant functions of vitamin D_3 in humans, which directly impact human health and reproductive competence.

Strong natural selection for vitamin D_3 production in human skin was likely a powerful factor influencing the evolution of skin pigmentation in human populations at high latitudes. Preliminary study of the distribution of paleontological and archaeological sites for the genus *Homo* in relationship to the vitamin D_3 synthesis zones described above indicates that year-round hominin habitation of Zone 3, i.e., latitudes generally higher than $50°$, occurred only after human populations had developed the technological competence to harvest fish, marine mammals, or other sources of food [such as reindeer lichen, or reindeer meat, organs, or milk (Bjorn & Wang 2000)] rich in vitamin D_3 (N. Jablonski, G. Chaplin & D. Tyler, manuscript in preparation). This capability is associated almost primarily with Upper Paleolithic peoples, living approximately 15,000–10,000 years ago, who are known to have made extensive use of fish hooks, fish traps, nets, harpoons, and other implements for the harvesting of marine animals.

SEXUAL DIMORPHISM IN HUMAN SKIN COLOR   The observation that females exhibit lighter skin pigmentation than do males in all populations examined (Jablonski & Chaplin 2000, van den Berghe & Frost 1986) has invited speculation that the phenomenon may be due to infantile mimicry, sexual selection, or a combination of both factors (Aoki 2002, Frost 1988, Ihara & Aoki 1999, van den Berghe & Frost 1986). These hypotheses are based on the observations that the attraction of human infants and human females is partly due to their lighter pigmentation, and that lighter-colored adult females are perceived as more feminine than are darker females, and therefore are preferred as partners (Frost 1988). Jablonski & Chaplin (2000) have advanced the idea that sexual dimorphism in skin pigmentation is primarily due to natural selection, on the basis of the need of females to maximize cutaneous vitamin D_3 production in order to meet their absolutely higher calcium requirements of pregnancy and lactation. Also, darker pigmentation may have been the object of natural selection in males because of the importance of maintaining optimal levels of folate in order to safeguard sperm production, a process dependent on folate for DNA synthesis (G. Chaplin, personal communication). Sexual selection is thus considered to have played a role in increasing the disparity in skin color between the sexes in some societies through preference for more lightly pigmented females, but this was not its ultimate cause (Jablonski & Chaplin 2000).

TANNING AND BLEACHING   The temporary development of increased melanin pigmentation through exposure to UVR is called facultative pigmentation or tanning. Individuals with very light constitutive pigmentation (skin phototypes I and II) never tan or tan minimally, whereas those with moderate to dark constitutive pigmentation (phototypes V and VI) tan profusely (Taylor 2002). Considerable variation in tanning potential exists even between people with ostensibly very similar levels of constitutive pigmentation (Lee & Lasker 1959). Tanning develops in two stages (immediate and delayed) over the course of several hours or days, depending on the wavelength and duration of UVR exposure (Ortonne 1990). Exposure to UVA causes tanning to develop quickly (Ortonne 1990), possibly as an adaptation

to protect against photodegradation of essential biomolecules. Facultative pigmentation is probably most important in areas such as the circum-Mediterranean that receive low levels of UVB but receive moderate levels of UVA that cause photodegradation of folate, DNA, and vitamin D_3.

The practice of recreational tanning has been eschewed by health care workers in the past 20 years because of the explosion in skin cancer rates due to increased UVR exposure. A tanned skin is still viewed by many as fashionable or as a sign of well-being, however, and this positive image has spurred the development of a simulated tanning industry in Europe, the Americas, and Australia (Brown 2001, Randle 1997).

In many countries, however, tanned or dark skin does not connote membership in a fashionable class, and the possession of light skin—especially among women—was and still is viewed as highly desirable and indicative of higher social standing. In many Asian countries, most women practice sun avoidance diligently. In other countries where constitutive pigmentation is darker, skin-bleaching agents (including potent topical corticosteroids and hydroquinone formulations) have become popular (Taylor 2002).

THE MULTIFACTORIAL DETERMINATION OF SKIN PIGMENTATION IN MODERN HUMANS   The evolution of skin pigmentation in humans has been determined by many factors (Figure 6). By far the most important of these is the UVR regime of the environment because intensity of UVR has been the main selective factor influencing the evolution of melanin pigmentation in the skin. Through time, the number of factors influencing the evolution of human skin pigmentation has increased, and culture clearly has reduced the scope for the action of natural selection on human skin. Cultural behaviors such as the wearing of clothes and the utilization of shelter have become more common through time and have affected the evolution of skin pigmentation in some populations because of their effects of reducing an individual's UVR exposure. Related to this phenomenon is the length of time that a population has inhabited an area with a particular UVR regime and the latitudinal distance traversed from the ancestral to the new homeland. There is certainly a considerable lag time between the time of settlement of an area and time that a population reaches its "optimum" skin color for the UVR conditions of the area. The length of that lag period for any population is not known but would depend on the intensity of natural selection exerted on the population by environmental influences. In early prehistory, humans possessed a simpler material culture, spent considerable time accumulating food, and had fewer cultural trappings to buffer themselves against the environment. Under these conditions, natural selection would have promoted mainly biological adaptations to the environment—including changes in skin coloration, body proportions, and regulation of thermal cooling. With increasing cultural competence over time, cultural solutions to the environmental challenges of sun, heat, and cold became preeminent. The oft-cited example of the skin colors of the native populations of equatorial South America is worth revisiting in this connection. These populations have long been recognized
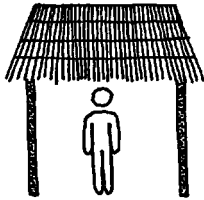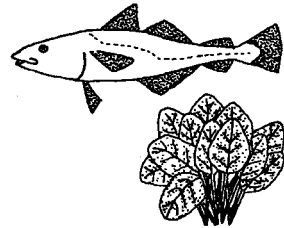
In evolutionary time:
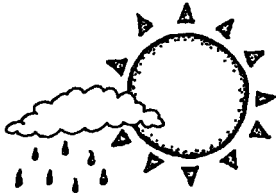
Environment:
- UV radiation levels
- Precipitation
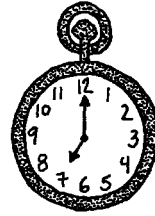
Migration:
- Length of migration
- Recency of migration

Other cultural adaptations to climate:
- Clothes wearing
- Shade seeking
- Shelter
- Timing of daily activities

Diet:
- Vitamin D content
- Folate content

During a human lifetime:

Environmental influences:

- UV radiation exposure
- Application of tanning or bleaching agents
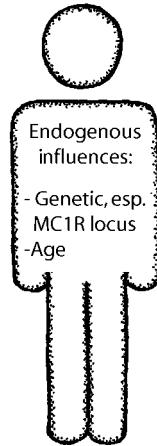
Endogenous influences:

- Genetic, esp. MC1R locus
- Age

Figure 6 The factors influencing human skin pigmentation, through evolutionary time and during the course of a human lifetime.

as being more lightly pigmented than are their counterparts at similar latitudes and altitudes in the Old World (Frisancho 1981, Jablonski & Chaplin 2000). This fact is almost certainly due to the recency of populations' migration into South America from Asia (within the past 10,000–15,000 years) and the fact that the immigrant populations into South America possessed many cultural behaviors and accoutrements that protected them from high UVR exposure (Jablonski & Chaplin 2000).

Diet has also played a part in the evolution of human skin pigmentation in very recent human history, as is well illustrated by the Eskimo-Aleut peoples of the northeast Asian and North American Arctic. Eskimo-Aleuts exhibit skin pigmentation darker than would be predicted on the basis of the UVMED in their habitats (Jablonski & Chaplin 2000). Several factors have likely contributed to this phenomenon, including the relative recency of their migration to the far north from a lower-latitude Asian homeland and its implication that their skin color has not caught up with their current location. This is almost certainly not the entire story, however. The UVR regime of the latitudes in which Eskimo-Aleuts reside comprises almost exclusively UVA throughout the year, with virtually no vitamin D–inducing UVB except for extremely small doses in the summer months (Chaplin 2001, Johnson et al. 1976). Habitation of this latitude (Figure 5, Zone 3) by humans would be impossible without reliance on a highly vitamin D–rich diet. The major components of the aboriginal Eskimo-Aleut diet—marine mammals, fish, and caribou—provide vitamin D_3 in abundance. Much of the dietary vitamin D_3 is stored in body fat (Mawer et al. 1972), denoting a possible evolutionary connection between the development of generous subcutaneous fat stores and vitamin D_3 storage in these populations. With selection pressure on depigmentation apparently relaxed because of diet, Eskimo-Aleuts have evolved darker skin to protect themselves from high levels of UVA as a result of direct solar irradiation and reflection from snow and ice. This scenario is supported by epidemiological studies showing that departure from traditional diets in Eskimo-Aleut populations has resulted in a high prevalence of vitamin D_3–deficiency diseases, especially rickets (Gessner et al. 1997, Haworth & Dilling 1986, Moffatt 1995).

THE GENETICS OF HUMAN SKIN COLORATION The study of genetics of human skin pigmentation has lagged considerably behind the study of the diversity and causation of diverse human skin color phenotypes. This situation is now changing rapidly as comparative genomics, especially detailed studies of the genes regulating coat color pigmentation in mice (Barsh 1996, Sturm et al. 2001), begin to permit identification of the genes responsible for the pigmentation of human hair, skin, and eyes. Sixty of the 127 currently recognized pigmentation genes in the mouse appear to have human orthologs (Bennett 2003).

Human skin pigmentation has long been considered a polygenic trait that follows a quasi-Mendelian pattern of inheritance (Brues 1975, Byard 1981, Byard & Lees 1981), with a few major genes of dramatic effect and additional modifier genes (Sturm et al. 2001). Because pigmentation is a trait determined by the

synchronized interaction of various genes with the environment (John et al. 2003), determination of the relative roles of variant genes and varying environments has proven extremely challenging (Sturm et al. 1998). Classical genetic studies of inheritance of human skin coloration have shed little light on the molecular basis of skin color variation, beyond showing that interbreeding between light and dark skin color phenotypes produces offspring of intermediate pigmentation (Robins 1991; Sturm et al. 1998, 2001). As a result of recent advances in the understanding of the chemistry and enzymology of the biosynthesis of melanins, the genetic regulation of the many steps in melanin production is now beginning to be understood. Among the numerous mutations affecting melanocyte function in human populations are the *P*-gene and members of the *TYRP* and *SILV* gene families, which direct the assembly and maturation of melansomes within melanocytes (Sturm et al. 2001). Investigation of the influence of these genes on skin pigmentation phenotypes is just beginning (Akey et al. 2001), however, and it remains to be demonstrated whether polymorphism in these gene systems correlates with pigmentary differences between populations.

To date, the greatest scholarly attention has been focused on the melanocortin-1 receptor (*MC1R*) gene, which is the human homologue of the *Agouti* locus that in mice regulates the production of the eumelanin and pheomelanin pigments of the coat (Barsh 1996, Rana et al. 1999). In humans, the synthesis of eumelanin is stimulated by the binding of α-melanotropin (α-melanocyte-stimulating hormone) to the functional *MC1R* expressed on melanocytes (Scott et al. 2002). The *MC1R* appears to be one of the major genes involved in the determination of human hair and skin pigmentation, with *MC1R* polymorphisms in northern European populations associated with red hair and fair skin, reduced tanning ability, and high risk of melanoma and nonmelanoma skin cancer (Healy et al. 2001, Scott et al. 2002, Smith et al. 1998). The *MC1R* locus is characterized by high levels of polymorphism in light-skinned individuals outside of Africa and lower levels of variation in dark-skinned individuals within Africa (John et al. 2003, Rana et al. 1999). This is opposite the pattern observed in most other loci, where Africans are most polymorphic (Shriver et al. 1997). The observed pattern of variation in the *MC1R* suggests that different selective pressures among individuals with dark and light skin have shaped the genetic variation at this locus, with functional constraints operating to limit variation in African populations (John et al. 2003). The numerous *MC1R* polymorphisms in light-skinned individuals were originally thought to denote relaxation of selection for production of eumelanin outside of tropical latitudes (Harding et al. 2000). A reinterpretation of these data indicates, however, that adaptive evolution for sun-resistant *MC1R* alleles began when humans first became hairless in tropical Africa, and that human movement into the less sunny climes of Eurasia favored any mutant *MC1R* allele that did not produce dark skin (Rogers et al. 2004). Recent study of the *MC1R* promoter function casts doubt on the relaxation hypothesis and suggests instead the possible action of purifying or diversifying selection on some *MC1R* variants in Asian and Europeans (Makova et al. 2001). A study comparing populations in southern Africa of Bantu-language

speakers and San people showed some variation in *MC1R* sequences, but investigators concluded that although some MC1R mutants are tolerated in Africa, this gene has been the object of purifying selection and has played an important role in the maintenance of dark pigmentation in Africans (John et al. 2003). The presence of higher levels of *MC1R* variation in dark-skinned populations subjected to lower levels of UVR in southern Africa (as compared to equatorial Africa) supports the notion that number and kinds of *MC1R* variants are strongly influenced by purifying selection (John et al. 2003). Further genetic studies of more African populations are needed to determine if the great diversity of skin color observed in populations in sub-Saharan Africa (Relethford 2000) can be related to specific patterns of *MC1R* or other polymorphisms that evolved in response to the region's considerable heterogeneity of UVR and precipitation regimes (Chaplin 2001).

The study of the genetics of human skin pigmentation is still in infancy, and much remains to be learned about the levels, effects, and interactions of polymorphisms in the loci influencing skin color phenotype. The production of eumelanin is under strong functional constraint as a result of natural selection in regions of the world with high levels of UVR (Sturm et al. 2001), and there is increasing evidence that at least *MC1R* variation is an adaptive response to selection for different alleles in different environments (Makova et al. 2001, Sturm et al. 2001). From what is known of the timing and nature of movements of groups of early *Homo* species and of *Homo sapiens* in prehistory, it appears that populations of humans have moved in and out of regions with different UVR regimes over the course of thousands of years. This finding would suggest that natural selection would have favored the evolution of dark and light skin pigmentation in disparate places at different times, resulting in the independent evolution of dark and light skin phenotypes and possibly involving recurrent episodes of repigmentation and depigmentation (Jablonski & Chaplin 2000). This phenomenon would have been pronounced in the early history of the genus *Homo* (including the early history of *Homo sapiens*) when cultural buffers against the environment were less effective and sophisticated.

SKIN COLOR AND RACE   Skin color is the most obvious visible attribute of the human body. It has been the primary characteristic used to classify people into purportedly genetically distinct geographic groups or "races." The biological basis of skin pigmentation in humans, however, strongly argues against its use as a diagnostic classificatory trait. Critical examination of the distribution of skin color phenotypes in humans leads to the conclusion that skin pigmentation is adaptive, and its evolution in specific populations has been strongly influenced by the environmental conditions (the UVR regimes, in particular) of specific places. Highly adaptive phenotypic characteristics of organisms are of little use in classification because they are subject to homoplasy (parallelism or convergent evolution) and are extremely labile. Emerging genetic evidence indicates that the evolution of pigmentation genes has been driven by purifying and diversifying selection working to produce adaptive responses in different environments (Makova et al. 2001,

Rogers et al. 2004, Sturm et al. 2001). This evidence indicates that similar skin colors have evolved independently in human populations inhabiting similar environments. Darkly or lightly pigmented skin, therefore, provides evidence only about the nature of the past environments in which people have lived, rendering skin pigmentation useless as a marker for membership in a unique group or "race."

The continued social importance of skin color in human affairs reflects a high degree of sensitivity to skin color, brought about by historic and complex cultural attitudes toward skin colors (Ehrlich & Feldman 1969, Lewontin 1995, Parra et al. 2003). The apparent existence of a difference between so-called human races and subgroups is predicated on an exaggerated perception and heightened sensitivity to a visually obvious attribute of human appearance. The enormity of this bias is revealed when the small amounts of actual genetic variation within purported racial groups are revealed (Lewontin 1995, Marks 2002). Overall, human populations are remarkably similar to one another, with the greatest fraction of human variation being accounted for by differences between individuals (Lewontin 1972, 1995; Marks 2002). This collective evidence militates that the concept of biological race be abandoned and publicly disavowed (Lewontin 1995, Marks 2002, Muir 1993). Race thus emerges as a cultural construct devoid of explanatory power and destructive of human and social relations (Lewontin 1972, 1995; Muir 1993).

PROSPECTUS

The past decade has witnessed a tremendous advance in the understanding of the evolution of human skin and especially skin color, largely as a result of two phenomena. First is availability of remotely sensed environmental data that permit hypotheses about the adaptive value of properties of skin to be thoroughly tested. Second is the proliferation of studies of the molecular genetics of the skin color that are permitting new insight into the origins of skin color phenotypes and the mechanisms by which they have evolved. Growth is anticipated in both of these areas, and great potential exists for their interaction, in particular for the testing of hypotheses of adaptation through the simultaneous and detailed study of patterns of phenotypic, genotypic, and environmental variation, such as has been done in the study of butterfly pigmentation (Watt et al. 2003). The study of the evolution of human skin and skin color will also be advanced by the documentation of differential survival of well-defined phenotypes and genotypes in different environmental regimes through the use of epidemiological data, as has been undertaken recently in the study of geographic patterns of melanoma (Garland et al. 2003).

Continued study of the evolution of human skin and skin color is important not only to our realization of a more complete picture of human evolution, but also it is important because the skin is involved in so many aspects of human well-being. Many humans now live in regions far distant from their ancestral homelands, but they retain a covering of skin adapted to remote Pleistocene conditions. As is evidenced by modern rates of skin cancer and vitamin D deficiencies, human

behavior and culture are not perfect buffers against the effects of these major translocations. An appreciation of the many roles of skin will improve human health and attitudes toward diversity and will promote the fundamental understanding of why it is that people look the way they do.

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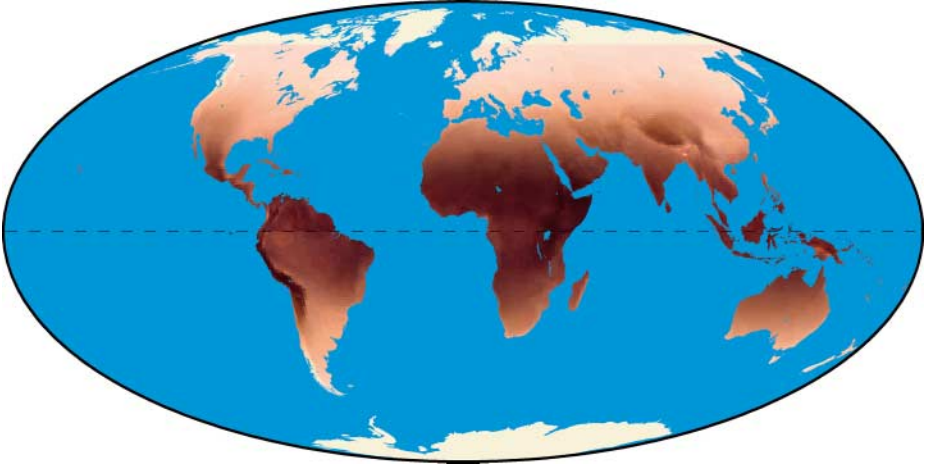
Figure 3   Human skin coloration as predicted from multiple regression formulae. See text for discussion.
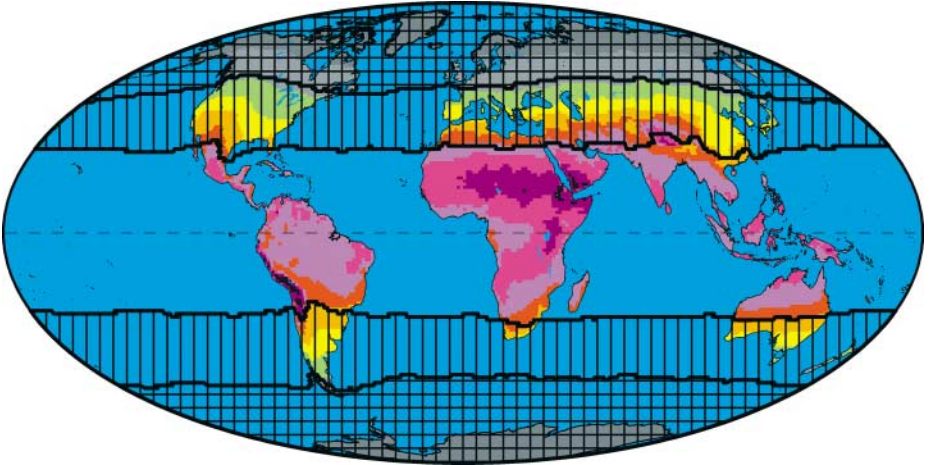
Figure 5   The potential for synthesizing vitamin D_3 in the skin relative to levels of annual average UVMED. The highest UVMED levels are indicated in deep violet, with incrementally lower levels indicated in shades of red, orange, yellow, green, and gray. Zone 1 (*area without hachure enclosing the tropics*) represents the region with adequate UVR throughout the year to catalyze vitamin D_3 synthesis. Zone 2 (*vertical hachure*) represents the area in which there is insufficient UVR during at least one month of the year to produce vitamin D_3. Zone 3 (*cross-hatched area*) represents the region in which there is insufficient UVR averaged over the entire year to photosynthesize vitamin D_3. See text for further description.

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