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# Aging in skin of color

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**Abstract** Ethnic skin is quickly emerging as the norm in the United States, and as the population becomes more diverse, there is also a projected rise in the number of aging adults. Given the paucity of data regarding aging in ethnic skin, the authors have consolidated available information for this population. Literature examining structural and functional variation of aging in ethnic skin types was primarily found through PubMed and supplemental textbook chapters. Aging is comprised of two synergistic processes, intrinsic or chronologic aging, and extrinsic aging. Caucasian, African American, East Asian, and Hispanic skin each have distinguishing features of aging, but all populations share dyspigmentation, rhytides, and skin laxity. Increased melanin content predisposes skin of color to a greater degree of hyperpigmentation, but skin thickness may protect against the formation of rhytides. Tailored prevention and treatment are also paramount in attaining favorable outcomes for this growing cohort.
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## Introduction to aging

Aging is a complex, multifactorial process that is defined as the decreased function and capacity of all organs with an increased likelihood of illness and death resulting from the synergistic effects of intrinsic and extrinsic influences.[1] The skin is the most perceivable indicator of the aging process, with visible changes in the structure and function of the integument. Clinicians are faced with a growing elderly population as world demographics shift; by 2030, the elderly population is projected to increase to 71 million.[1] Individuals older than 65 years will account for almost 40% of the United States.[2] Skin is predicted to become more diverse over the same time period.[2] From the most recent census, Caucasians now comprise the minority of new births in the United States and are projected to comprise less than 50% of the population by 2060.[2] Ethnic skin is traditionally defined as skin of color, or Fitzpatrick skin types III to VI, but it may more inclusively define skin types darker than the Caucasian race, which includes an extensive range of phenotypes.[3]

These considerations have important consequences for dermatologists. A longer life expectancy confers a greater lifetime exposure to toxins, chemicals, radiation, and other environmental agents that may cumulatively affect the integument. Consumers are beginning to notice this trend as well; forecasts predict the anti-aging product market will grow to more than 300 billion dollars by 2021.[4] Not only is the population aging, but there is a growing interest in countering the biologic process. As life expectancy and diversity increase, dermatologists should prioritize appropriate prevention and care of the consequences of aging skin.

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## Methods

A total of 41 publications regarding aging in ethnic skin have been included. These were located via a PubMed literature search as well as a search of the e-book collection at the Boston University School of Medicine Alumni Medical Library. Keywords included: aging, ethnic skin, intrinsic aging, extrinsic aging, photoaging, skin of color, and skin ethnic differences. Contributions from 1970 to 2018 were searched. Select references of the reviewed publications were also considered. Included literature comprised basic science research, randomized controlled trials, observational studies, reviews, commentaries, and textbook chapters.

## Aging skin: clinical and molecular pathogenesis

### Intrinsic aging

Intrinsic, or chronologic aging, is a predetermined process that is affected by ethnicity, anatomic variation, and changes in hormones. Clinically, this is noted by skin thinning, laxity, wrinkles, and xerosis as a result of microscopic endogenous damage.[1] One of the primary changes is decreased skin thickness; progressive thinning of the skin occurs while the number of layers of skin remains constant.[5] Reduced skin thickness is attributable to various changes within each stratum. Initially, the stratum corneum has a decreased total lipid content with an unchanged composition of lipids.[5] Subsequently, the epidermis shows multiple changes, including flattening of the epidermal and dermal junction with decreased rete peg interdigitation.[5] This reduces communication between the dermis and epidermis, leading to a diminished transfer of oxygen and less resistance to shearing forces, making aged skin more vulnerable to insult.[6] Although the barrier function of skin at this level is grossly maintained with aging, the baseline transepidermal water loss decreases with age as water content is reduced overall in aged skin.[5]

Below the junction, the dermis exhibits decreased vascularity, cellularity, collagen synthesis, sweat glands, and degraded elastic fibers.[5] There is a progressive reduction in melanocytic and Langerhans cell number and function. Degraded elastic fibers and reduced collagen synthesis are responsible for reduced skin elasticity, or skin ptosis, and these alterations contribute to rhytides, or wrinkles. Wrinkling appears at the periorbital and perioral locations, particularly due to repetitive muscle action, and these features progress from dynamic to static rhytides. Excess skin with decreased elastic recoil in the jowl, submental, and nasolabial fold areas produces a sagging appearance.[7]

At the hypodermis level, there is fat atrophy and hypertrophy depending on location, causing changes in subcutaneous fat distributions and overall accentuating an unbalanced appearance.[8] The youthful face has a balanced topography of superficial and deep fat with primary arcs along the lateral cheek, the jawline, and forehead. With facial aging, there is loss of soft tissue on the cheeks, temples, and lateral chin, causing narrowing of the forehead and shortening and widening of the lower face.[9] Fat persists or hypertrophies in the submental region, lateral nasolabial folds, labiomental crease, and jowls, and these fat pockets become more distinguishable as separate entities.[9] The forward and downward descent of the malar fat pad is the cause of the prominent nasal fold of the aging face.[9]

Below the skin, there are notable changes in bone structure. Skeletal aging is the result of bone atrophy as well as dynamic changes in bone growth and loss. Bony resorption at the mandible and maxilla contribute to a reduction in facial height with an increase in facial width.[10] Maxillary resorption in conjunction with increased orbital size leads to displacement of the malar fat pad and an accentuated nasolabial fold.[7]

In women, estrogen has a notable effect on skin physiology and function. Estrogen may promote wound healing and contribute to variations in skin thickness during the menstrual cycle by influencing collagen synthesis.[1] Mitotic activity of epidermal keratinocytes respond to estrogen, and dermal fibroblasts also express an estrogen subtype beta receptor.[6] Postmenopausal skin exhibits increased dryness, decreased elasticity, and increased wrinkling, which may be secondary to changes in estrogen and estrogen receptor expression.[11] Other hormones, including melatonin, cortisol, thyroxine, growth hormone and insulin-like growth factor, decline in quantity, and there is an age-related decline in vitamin D and beta-adrenergic and dopaminergic receptors as well.[6]

### Extrinsic aging

Extrinsic aging is defined as any environmental factor that contributes to the aging process and may include exposures and lifestyle.[1] Photoaging, secondary to exposure of ultraviolet (UV) radiation, accounts for the majority of visible cutaneous manifestations of aging.[12] Photoaging involves all layers of the skin with primary effects in the dermis and paracrine effects into the other layers.[6] This phenomenon consists of a series of cellular processes, both acute and chronic, secondary to sun exposure leading to visible senescence, and is characterized by coarse wrinkles, loss of elasticity, pigmentation, xerosis, keratosis, and telangiectasias.[13] Photodamaged skin will initially have atrophy of the extracellular matrix with reduced mature collagen and denatured elastic fibers induced by UVA radiation.[14] UVB radiation induces fibroblast elastase and matrix metalloproteinases (MMP) 1, 3, and 9 in the epidermis, causing elastic fiber cleavage.[14] This exacerbates the intrinsic decline of the skin's tensile strength. Increased expression of certain transcription factors is induced by UV radiation as well, including activator protein 1, which decreases the levels of procollagens I and III.[14] Adverse effects of long-term UV exposure are thought to be driven by absorption of UVA waves through chromophores and oxidative damage as well as mitochondrial DNA damage.[14] Photoaged skin also has a reduction in cutaneous microvasculature, leading to potential

reduced nutritional supply, and this may contribute to skin thinning.[1]

Photoaging is responsible for much of the perceived notion of aging, as it significantly influences the cutaneous manifestations of skin aging. Hyperpigmentation may possibly contribute more to the perception of an aged appearance greater than wrinkles, while subsurface pigmentation reduces skin radiance.[15]

UV exposure is not the only source of extrinsic aging. Visible light and infrared radiation have also been shown to upregulate MMP-1 expression and reactive oxygen species.[16] Air pollutants have been implicated in skin changes, including wrinkle formation and pigmentation. Tobacco smoking is a common offender leading to characteristic skin changes, including prominent wrinkles, smoker's melanosis of the oral mucosa, facial pigmentation, and decreased skin radiance.[16] These changes may be attributable to increased MMP-1 expression and increased melanocytic activity.[6] Poor nutrition may contribute to cutaneous aging, as well as certain vitamin deficiencies that cause dermatitis, perleche, and dyspigmentation.[16]

## Photoaging and skin of color

The effects of photoaging are highly variable depending on the ethnicity of the patient. The most significant discrepancy between ethnic and Caucasian skin is the melanin content of the epidermis. There are several mediators of increased melanin and increased melanocytic activity, including melanocyte-stimulating hormone and its receptors in addition to tyrosinase-related proteins, which work to increase tyrosinase activity, melanocytic activity, and the size of melanosomes.[8] Skin of color typically contains larger, well-dispersed melanosomes with increased melanin content in contrast to individuals with lighter skin, which confers increased photoprotection and fewer signs of photoaging. A photoaging scale was developed to assess the severity of photodamage, but it is limited by its unique application to Caucasian skin.[13]

## Aging by ethnicity

### Caucasian

#### Structural aging

The term "Caucasian" specifically refers to individuals of European, North African, and southwest Asian ancestry.[3] Structurally, this skin type is characterized by a thinner stratum corneum and decreased water content. Features of skin aging in Caucasians include a narrower nasal base with larger tip projection and less volume in lips with intercanthal width consistent with that of African Americans.[8] On the upper aspect of the face, forehead and glabellar rhytides are visible.[7] In the midface, there is displacement of the orbital fat pad, descent of the melolabial fat pad, and laxity of the eyelids with redundancy of soft tissue.[7] Brow ptosis, crow's feet, and lower lid lag occur secondary to bony remodeling of the superomedial and inferolateral orbital rims.[8] The lower part of the face and the neck are characterized by skin laxity, which is visible as sagging and jowling.[17] Caucasian facial aging is also characterized by reduced lip volume with perioral rhytides.[17] Characteristic features of ethnic skin are listed in Table 1.

#### Photoaging

Caucasian skin contains small aggregations of melanosomes with reduced melanin content, predisposing these individuals to earlier photoaging.[18] Photoaging in Caucasian patients is best characterized by skin rhytides, increased laxity, and hyperpigmentation, such as solar lentigines, sallowness of skin, seborrheic keratosis, and dyschromia.[8] Signs of photoaging are evident in the fourth decade of life, appearing later in dark-skinned individuals.[19] Studies that analyzed the differences in aging appearance between Japanese and Caucasian women depicted that photoaging and wrinkles appeared more quickly and were more severe in light-skinned women.[19] The degree of sunlight exposure is also critical in these individuals; Australians with increased sun exposure revealed a higher incidence and severity of the signs of aging than their counterparts in Europe or the United States.[20]

### African American

#### Structural aging

The term "African American" is wide-reaching, encompassing several ethnicities with African, Caucasian, Native American, and Caribbean ancestry.[3] Anthropomorphic features unique to African American faces include longer forehead heights, shorter nasal length, shorter ear length, greater alar width, greater eye fissure width, and greater mouth width.[21] Structural characteristics of African American skin include a thicker stratum corneum with an increased number of layers in addition to amplified dermal fibroblast activity compared with Caucasian skin. This leads to overall thicker skin, which retains its youthful appearance with less pronounced wrinkling. The upper third of the face may show brow ptosis with forehead, glabellar, and perioral rhytides, but these changes may appear later in life in the fifth decade and beyond.[22]

The midface is a more prominent location for aging in African Americans, displaying laxity of the eyelids and descent of the malar fat pad, causing a double convexity of this area.[22] The upper eyelids tend to have the appearance of increased soft tissue fullness due to a shorter distance of the upper eyelid crease from the lid margin.[23] Additionally, a hypoplastic malar eminence and ocular proptosis create scleral show an infraorbital shadowing.[17] Hypertrophy of the malar

**Table 1** Ethnic differences in properties of the skin[18]

Adapted from Farage et. al

| Parameter | Comparison |
|---|---|
| Thickness | African American = Caucasian |
| Number of cell layers | African American > Caucasian > Asian |
| Lipid content | African American > Caucasian |
| Ceramide content | Asians > Hispanic > Caucasian > African American |
| Water content | Asian, Hispanic > African American > Caucasian |
| Melanosome size and distribution | African American: large, uniform, single units with membrane<br>Asian: small, uniform, clustered in groups of up to 10<br>Caucasian: variable shape and size, clustered in groups of up to 10 |
| Melanosome stage in keratinocytes | African American: stage IV<br>Asian: stages II, III, IV, with stage IV predominating<br>Caucasian: stages II and III |
| Melanosome induction | African American: UV induces all stages<br>Asian: UV induces stages II and III<br>Caucasian: UV induces stage IV |
| Sebum production | African American > Asian > Caucasian = Hispanic |

UV, ultraviolet light.

fat pad leads to early descent of the melolabial mounds, thereby causing accentuated nasolabial and nasal jugal folds.[17]

Increased skin weight may cause pronounced loss of the cervicomental angle in the lower portion of the face of African Americans.[22] This increased skin thickness is the main contributor to the jowling seen in lower facial aging.[22]

### Photoaging

A key characteristic of the African-American skin type include nonaggregated melanosomes that are widely dispersed through the epidermis layer.[18] The melanin content and dispersion prevent dermal UV penetration, reducing the impact of photoaging in African Americans. With matched UV exposure, African Americans may show fine wrinkles, mottled pigmentation, and dermatosis papulosa nigra in contrast to Caucasians with deep rhytides and dyschromias at an earlier age.[17] Dermatosis papulosa nigra is a variant of seborrheic keratosis and is particularly seen in African Americans, affecting up to one-third of the population.[17] It presents as dark brown papules and plaques clustered around the eyelids and upper malar region.

## Asian descent

### Structural aging

The term "Asian" is broad and refers to a wide group of individuals, but literature tends to focus on East Asians in particular, which includes those of Chinese, Japanese, and Korean descent.[3] Structurally, there have been several noted differences in facial shape. East Asians typically have wider bitemporal, bigonial, and bizygomatic width compared with Caucasians.[24] When viewed in profile, East Asian faces are notable for a flatter appearance due to retrusion of the midline skeletal structures, including the glabella, nasal bone, and mental protuberance.[24] There is literature to suggest that Asian faces structurally resemble that of an infant's, given a wider and rounder face, higher brow, fuller upper lip, lower nasal bridge, flatter malar prominence and midface, and a more receded chin.[3] Additionally, Asian faces have a narrower palpebral fissure and fuller upper eyelid, leading to a greater distance from the eyebrow to the upper lid margin.[17] Asians have also been found to have more midface malar fat with more prominent, fuller lips.[17] Some studies have shown that Asians have a weaker skeletal framework, leading to more soft-tissue descent of the midface, malar fat pad, and tear trough.[8]

There has been limited evidence showing distinct differences between the structure of East Asian and Caucasian skin. Some studies indicate that Asian skin has an increased lipid content in the stratum corneum when compared with other ethnicities[25]; however, the stratum corneum in Asian populations is thinner with higher eccrine gland density, which may contribute to higher sensitivity to exogenous chemicals.[25] Other studies have concluded that Asian skin has a greater functional loss in barrier function with aging with mechanical challenge, though the transepidermal water loss remains consistent.[25]

There is limited evidence evaluating the structure and function of Southeast Asian skin and facial structure. South Asian facial anatomy is typically more comparable with the Caucasian anatomic structure, as compared with the Mongoloid structure.[8]

### Photoaging

Asians tend to manifest photoaging as pigmentary changes more quickly than rhytides, which are less conspicuous and are seen after the fifth decade of life.[25] Asian skin tends to have a higher melanin content than Caucasians with smaller, more clustered melanosomes, which are protective against photodamage.[18] Pigmentary changes include hypochromia and

pigmentary growths with chronic UV exposure, such as ephelides, melasma, seborrheic keratoses, and lentigos.[25] A comparison of Chinese women with French-Caucasian women who have consistent sun exposure, wrinkle formation was delayed by 10 years, but pigmented spot intensity was much greater in Chinese women.[26] This is consistent with perceptions of aging, as seen in a study that evaluated the perception of aging between Chinese and Caucasian women, where Chinese women associated reduced dark spots with a younger age.[27]

### Cultural considerations

Both Caucasian and Asian populations ideally prefer the oval facial shape; however, the majority of Asian women requesting cosmetic procedures do not necessarily want Westernized changes, rather desiring an enhancement of their ethnic features.[24] Asian women prefer a facial shape similar to an upside-down egg, with more fullness in the upper half of the face tapering inward toward the chin.[24] Japanese women prefer to retain the greater epicanthal crease and seek procedures to create a full-lid contour and thinned outer lips.[28] Korean women prefer a more defined palpebral crease and fuller lips, and as Koreans tend to have a wider mandibular angle, they opt for procedures to create a more delicate angle and narrower lower part of the face. There are also significant cultural values regarding fair skin; old proverbs such as "white skin makes up for seven defects" in Japanese culture and similar adages in Chinese culture have persisted, and surveys show that as many as four out of 10 women in Hong Kong, Malaysia, the Philippines, and South Korea have used a skin-whitening cream.[3]

## Hispanic or Latino descent

### Structural aging

There is a broad range of ethnicities that fall under the umbrella term "Hispanic," which is defined by the United States Census Bureau to include all persons originating from a Spanish-speaking country. This ethnicity is characterized by a mixture of populations, with facial features that vary depending on the geographic location of origin. The diverse ancestry of this population may contribute to the relative paucity of literature on specific anthropomorphic and aging features. Hispanics from Central and South America may share more anthropomorphic features with Caucasians, whereas Hispanics from the Caribbean may exhibit features more similar to those of African Americans.[29] In general, the Hispanic face shape is round with prominent malar eminences and mildly retrusive chins.[29] Specific characteristics include increased bizygomatic distance, broad nose, abbreviated nasal length, and bimaxillary protrusion.[3] During aging, these features become more prominent, with increased eyelid hooding, drooping of the brows, conspicuous nasolabial folds, and jowling of the chin and neck.[29] The midface area also becomes prominent, like African Americans, due to fat pad accumulation and descent.[17]

### Photoaging

Latino skin may range between Fitzpatrick skin types II to IV, depending on ancestry.[30] This contributes to extensive differences on aging based on skin type with a relatively modest amount of published literature on photoaging in Hispanic skin. Latino skin contains more melanosomes than Caucasian skin, which confers some reduced risk of photoaging and typically presents as fine rhytides and pigmentary changes.[17] Wrinkles in Hispanic women were shown to be less prominent than in Caucasians, but more prominent than in East Asians.[31] Hispanics and East Asians were noted to have the least amount of hyperpigmentation with aging of all studied ethnicities.[31]

## Prevention and treatment

Prevention and treatment strategies for photoaging have been suggested in a three-tiered approach, including photoprotection, active formulations to mitigate the signs of aging, and invasive correction of photoaged skin.[32] Although increased melanin confers additional photoprotection, up to a sun protection factor of 13 in African Americans and Hispanics, it does not necessarily provide the optimal defense against sun exposure to prevent signs of photoaging.[33] A broad-spectrum sun protection factor of 30+ sunscreen daily, as well as avoidance of excess and unnecessary prolonged sun exposure, is recommended.[34] This recommendation applies to patients of all skin types, including ethnic skin, as it can slow the signs of photoaging.[35] Other general recommendations include avoiding smoking and artificial UV exposure and maintaining a balanced, healthy diet for optimal vitamin absorption.[16]

Topical therapies, such as retinoids, hydroquinone, alpha hydroxy acids, and antioxidants, prevent and reduce the signs of photoaging by impeding the melanin production pathway. Dyschromia is a common concern for aging patients with skin of color, and topical retinoids are a mainstay of treatment.[36] Retinoids, which comprise vitamin A and its derivatives, regulate the life cycle of keratinocytes and the spread of melanosomes in the epidermis.[32] Rapid turnover of keratinocytes and reduction of melanosome transfer ultimately enhance the loss of melanin.[37] At the level of the dermis, histologic findings include increased collagen, elastin, and glycosaminoglans.[32] In studies with a duration of at least 6 months, tretinoin was shown to improve the signs of photoaging, including the reduction of fine wrinkling and dyspigmentation.[38] Retinoids may cause a localized irritant reaction limiting compliance, but patients should be initiated on the lowest dose possible and encouraged to use the product consistently.

An efficacious and popular topical treatment, hydroquinone (HQ) diminishes hyperpigmentation by inhibiting the enzyme tyrosinase, thereby hindering the conversion of dihydroxyphenylalanine to melanin.[37] Hydroquinone also prevents the formation of melanosomes and stimulates melanocyte necrosis.[37] Its efficacy is proportional to product concentration, and more potent preparations have a quicker onset of action.[37] Though HQ has shown a significant reduction of

hyperpigmentation and melasma in skin of color, the use of HQ is limited by side effects such as irritant dermatitis, postinflammatory hyperpigmentation, hypopigmentation, and rarely, exogenous ochronosis.[37] Exogenous ochronosis may be averted by lower concentrations of HQ, careful consideration of formulation quality, and limited length of hydroquinone use.[37] A wide range of other topical lightening therapies are available as over-the-counter formulations. Alpha hydroxy acids, such as glycolic, lactic, tartaric, citric, malic, and pyruvic acids, work as calcium chelators to trigger desquamation, and antioxidants, including alpha-lipoic acid, niacinamide, L-ascorbic acid, and alpha-tocopherol, protect against photoaging by inhibiting damage induced by free radicals.[32] Bioavailability of these agents is hindered by formulation instability.

Fillers, neuromodulators, chemical peels, and lasers are also viable options for treatment of cosmetic concerns with aging. Dermal filler should be tailored to the individual, but overall, clinicians should consider the midface and nasolabial folds as common targets in ethnic skin, given the prevalence of midface fat atrophy and fat redistribution.[17] In patients of Asian descent, care should be taken to avoid excess volume enhancement of the lateral maxilla and zygoma, given larger inherent bizygomatic distances.[17] African Americans may especially benefit from stimulatory fillers such as calcium hydroxyapatite for volumizing the midface.[17] Postinjection erythema is common in ethnic skin types, and patients should be counseled appropriately.[34] Risk of keloids and postinflammatory hyperpigmentation in skin of color is also elevated, and this can be mitigated by meticulous technique.

Botulinum toxin type A treatment to smooth hyperdynamic wrinkles is a common cosmetic procedure requested by all ethnicities. Location of injection may vary, as ethnic patients tend to develop hyperdynamic wrinkles in the glabellar area compared with Caucasians, who develop brow ptosis and crow's feet at an early age.[17] Botulinum toxin type A is also becoming an increasingly popular treatment for benign masseter hypertrophy, especially in Korean populations.[17]

Superficial chemical peels have also been assessed as safe for pigmentary dyschromias in darker skin types, though there is a slightly elevated risk of side effects.[39] Chemical peels are agents that cause exfoliation of the epidermis and dermis, which subsequently regenerates. Compounds used for this purpose may include glycolic acid, salicylic acid, beta lipohydroxy acid, and trichloroacetic acid at high concentrations.[40] Superficial peels, which only exfoliate the stratum corneum to the papillary dermis, are generally well tolerated in skin of color, and these have been shown to improve dyspigmentation and rhytides.[40] There is a higher risk of postinflammatory hyperpigmentation with chemical peels in patients with darker skin, especially with trichloroacetic acid, which may be alleviated by titrating up from lower peel concentrations, postpeel depigmenting agents, and UV protection.[40]

Other treatments for hyperpigmentation and fine rhytides include ablative and nonablative lasers. Laser therapy is designed based on absorption of light by chromophores, and variance in wavelength, pulse duration, and fluence can dictate utility.[41] Signs of photoaging are treatable with the Q-switched neodymium-doped yttrium aluminum garnet laser in darker skin types. This is a popular choice for hyperpigmentation and melasma in skin of color given its longer wavelength, and preferentially targets the dermis[41] Nonablative fractional photothermolysis, which creates microscopic regions of thermal injury and subsequent rejuvenation, is also efficacious in improving the appearance of periorbital rhytides and pigmentation.[41] Ethnic skin types have higher rates of dyschromia with laser therapies, so careful consideration on an individual basis should be given after other topical treatments or chemical peels have been attempted.

## Conclusions

Aging is an inevitable process that manifests differently depending on a patient's skin type, exposures, and genetics. All skin types will at some point exhibit photodamage, bone remodeling, and soft tissue redistribution. Patients with skin of color may present with distinct concerns, including changes in pigmentation and variable changes in facial structure dependent on ancestry. Given increased melanin content, these individuals are overall less prone to signs of photoaging. African Americans typically exhibit increased skin thickness, contributing to reduced wrinkles but noticeable jowling. East Asian populations are predisposed to hyperpigmentation but delayed wrinkle formation. Hispanic skin is quite diverse and may age differently based on heritage but likewise demonstrates delayed rhytides.

In considering treatment, topical and invasive therapies are available to target common concerns. For patients with skin of color, dyschromia is a notable feature of aging, and topical agents, antioxidants, chemical peels, and lasers can be effective measures in mitigating pigmentary changes. Fillers and neuromodulators are also useful techniques to improve signs of volume loss and rhytides. All skin types would benefit from broad-spectrum UV protection. It is important for the clinician to take functional and structural changes of ethnic skin into consideration to tailor treatment for the specific needs and requests of the rapidly diversifying aging population.

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