



# Analysis and Assessment of Facial Aging

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## Abstract

**Purpose of Review** The goal of this review is to highlight the important clinical considerations that impact the facial aging process and discuss current assessment tools that are available to help guide the clinician on appropriate facial rejuvenation modalities.

**Recent Findings** Extrinsic and intrinsic factors and their effects on facial aging continue to be studied. Ethnic and racial backgrounds play a critical role in the facial aging process. Gender's impact on facial aging is explored. Several assessment tools have been developed to examine various subunits of the face, and other assessments have been created that account for the patient's ethnic background.

**Summary** Facial aging is a dynamic and complex process. Understanding facial aging requires understanding changes in all layers of facial tissues as well as extrinsic and intrinsic factors that can affect the process, along with ethnic differences. Using appropriate assessment tools can help guide the clinician toward optimal treatment modalities.

**Keywords** Facial aging · Facial analysis · Facial aging assessment · Aging face

## Introduction

Facial aging is a strong area of international interest as the global demand for facial rejuvenation procedures has grown. Multiple factors contribute to the changes associated with facial aging including physiologic, cellular, environmental, and genetic considerations. Appropriate knowledge of these factors as well as the options for treatment is critical in order to tailor aesthetic treatments and optimize the outcomes for patients.

## Morphologic Considerations of Facial Aging

Structural changes to the aging face are multifactorial; they involve multiple facial structures including skeletal, soft tissue, and skin [1]. In a young face, superficial and deep fat is evenly distributed. As the face ages, there is simultaneous volumetric fat loss as well as some localized adipose hypertrophy. These lead to topographic irregularities to the face, including the temples, cheek, and lateral chin (Fig. 1) [1].

The medical literature has described how structurally, skin, fat, muscle, and bone all play a role in facial aging. While each facial structure is affected by the aging process individually, the structures act in dynamic harmony to determine the phenotypic presentation of the face as it ages. Though the extent to which each of these structures contributes to the aging process remains a subject of debate, it seems clear that some facial aging is the result of gravitational effects on the facial skin and soft tissues [1].

In addition to gravity, other forces that contribute to facial aging include skeletal resorption, superficial and deep volumetric fat redistribution, photoaging, hormonal changes, smoking, and chronic solar exposure [1]. Additional environmental factors that are purported to affect

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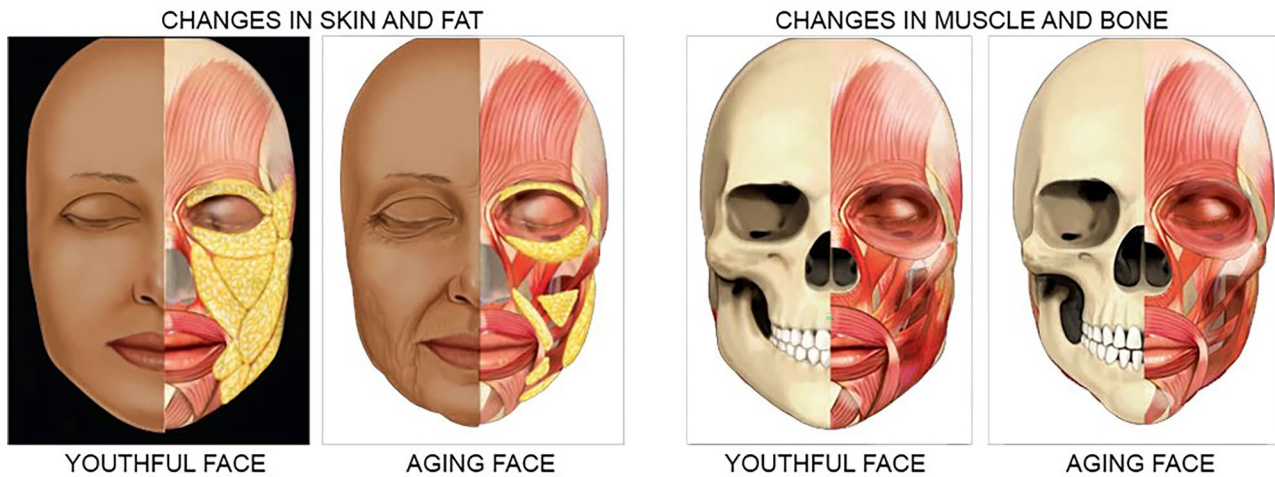
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**Fig. 1** Diagram illustrating the facial changes that occur with age. In the youthful face, subcutaneous fat deposits are appropriately positioned. With age comes progressive loss of facial volume and the

resulting involution and eventual descent of soft tissues. This causes underlying fat, soft tissue, and skeletal structures to become more evident and creates contour deficiencies

facial appearance include psychologic stress, diet, drug abuse, and comorbidities [1].

### Skeletal Changes

Age-related changes of the craniofacial skeleton are the result of both bone atrophy and change in the dynamics of bone expansion and bone resorption<sup>2</sup>. Due to changes in the maxilla and mandible, there is a noticeable reduction in facial height and a marginal increase in facial depth and width. Over time, there is an increase in orbital size, along with a decrease in maxillary projection, compounding the displacement of the malar fat pads inferiorly and accentuating the nasolabial fold [2]. The increase in resorption of the maxilla may also lead to a loss of structural support of the upper lip, thus contributing to perioral wrinkling. In the mandible, chin shape and projection change with age, and loss of teeth leads to marked bony resorption of the alveolar ridge [2]. The bony protuberances become more discernable at the insertion points of the masticatory muscles (e.g., inferior edge of the zygomatic eminence and the gonial angle) [2].

Shaw and Kahn described the volumetric changes of the aging face using computed tomography and three-dimensional volume rendering [3]. Using three separate patient groups (young, middle-aged, and old), they noted morphologic craniofacial changes including increase in orbital area and orbital aperture width, increase in piriform aperture area, and significant reduction in glabellar and maxillary angles [3].

### Subcutaneous Fat and Soft Tissue Distribution

The gradual loss of underlying soft tissue support and fullness is responsible for the soft tissue descent and relative excess of facial skin associated with aging [4].

The youthful face demonstrates a well-balanced distribution of superficial and deep fat, the topography of which is delineated by a series of arcs and convexities, including the jawline, temples, and lips in the frontal view, and the forehead, lateral cheek projection (the “ogee” curve), and jawline in the profile view [4]. With aging, there is a loss of soft tissue fullness in the forehead, glabellar, temporal, periorbital, malar, perioral, mandibular, and mental sites and hypertrophy in the infraorbital fat pads, malar fat pads, lateral nasolabial folds, jowls, and labiomental creases [4]. As such, there is a disruption in the arcs and convexities that define a youthful face. In the frontal view, the jawline scallops, the suborbital, temporal, and buccal areas are hollow, and the lips become thinner and straighter. In the profile view, the lateral arc of the cheek is disrupted, the mandibular arc has more pronounced jowling, and there is loss of projection of the forehead and brow [4, 5].

A convenient method for assessing the morphological effects of aging is to divide the face into thirds.

### Upper Third (Forehead and Brows)

There is a loss of temporal support to the lateral brow and an overall loss of fullness to the upper eyelid, which gives the appearance of brow ptosis. The outline of the skull and supraorbital rims becomes more apparent, along with the

brow muscles (particularly the procerus and corrugators), and the temporal blood vessel appearance becomes more tortuous [4]. As the orbital septum weakens, infraorbital fat may begin to protrude. In the youthful face, the subcutaneous volume of the forehead conceals the muscles of facial expression in this area (e.g., glabella, procerus, frontalis, corrugator). As this fullness diminishes with age, the effect of intrinsic tone of these muscles is accentuated, giving rise to fixed rhytides [4].

### Middle Third (Midface)

With age, there is loss of subcutaneous fullness over the malar prominence and progressive hollowing of the buccal space [4]. There is loss of fullness of the infraorbital subcutaneous tissues, accentuates the intrinsic tone of the orbicularis oculi muscle, giving rise to crow's feet rhytides [4]. This loss of subcutaneous fullness along the inferior border of the orbicularis oculi muscle also contributes to the development of the nasojugal fold medially and the malar crescent over the zygomatic eminence laterally. Infraorbital volume loss can accentuate the appearance infraorbital fat pads and tear-trough depressions [4]. Loss of subcutaneous fullness between the orbicularis oculi muscle and the overlying skin brings these lower eyelid tissues into closer proximity and makes the skin appear darker, and the eyes more tired [4]. However, darker coloration of the lower eyelid skin may also be due to dermal melanin deposition [4]. Flattening of the medial forehead bones causes blunting of the nasofrontal angle, which confers an appearance of a longer nose [4]. The upper and lower lateral nasal cartilaginous attachments weaken, causing loss of tip support and progressive tip ptosis [4]. Resorption at the pyriform and maxilla affects the alar base and causes a narrowing of the nasolabial angle, further highlighting the tip ptosis [4].

### Lower Third (Chin, Jawline, and Neck)

There is progressive loss of definition along the jawline as skin laxity increases, malar and perioral fat deposits become depleted, and alveolar bone is resorbed [4]. Jowls occur due to loss of masseteric ligamentous support allowing for descent of fat to the mandibular border [1]. There is loss of lateral projection of mandible, lowering the appearance of the angle of the mandible from the buccal region lower toward the neck. In the chin, there is volume loss laterally and inferiorly, causing the central part of the chin to appear wider and more prominent on frontal view [4]. As support of the skin of the lower face lessens with age, the submental fat pad may either protrude from between the free borders of the platysma or bulge from behind the submental portion of the platysma, and coupled with the downward vector of the platysma, this causes a characteristic “turkey neck” deformity

[4]. Plastysmal bands become more obvious as the muscle continues to support the floor of mouth structures and deep neck, and laxity of the overlying skin may create horizontal rhytids [4]. With age, the larynx and hyoid descend, causing blunting of the cervicomental angle [4].

## Intrinsic and Extrinsic Factors in Facial Aging

The two main processes that induce age-related changes of the face occur as a result of intrinsic and extrinsic pathways. Extrinsic (environmental) factors include sun exposure, smoking, alcohol abuse, air pollution, and poor nutrition. Intrinsic (genetic) factors depend upon various factors of each individual patient [6].

### Intrinsic Factors

Reactive oxygen species cause damage to the cellular membranes, enzymes, and DNA [7]. Telomeres, terminal portions of the eukaryotic chromosome, play a critical role in aging [7]. It has been widely shown that telomeres shorten with each cell division, impacting longevity. Growth factor modifications and decline in sex hormone levels also play a role in the aging process [7].

### Extrinsic Factors

Severe physical and psychological stress, diet, ionizing radiation, alcohol intake, environmental pollution, and exposure to UV radiation all play a role in extrinsic skin aging. Among this list, UV radiation is the most important as it contributes up to 80% of extrinsic aging [7]. Both UVB and UVA rays contribute to extrinsic skin aging [8•]. UVA (320–400 nm) penetrates deeper into the dermis, inducing both epidermal and dermal damage [8•]. There is roughly 10–100 times more UVA compared to UVB in ambient light [7]. The longer wavelength part of UVA might be of particular importance, but more research is needed in this area [8•]. UVB, on the other hand, induces primarily epidermal damage, affecting keratinocytes and melanocytes. UVB is also the cause of thymidine dimer covalent bonds, the accumulation of which leads to mutations causing actinic keratosis, lentigines, and carcinomas [7].

## Ethnic Differences in Facial Aging

The aging process is a particular area of interest in cosmetic surgery as beauty standards are impacted by cultural, geographical, and anatomical variations. A detailed knowledge of the morphological and histological variations of the facial aging process across ethnic groups is critical to providing

appropriate guidance on the ideal facial rejuvenation treatments [9••]. Understanding each patient's internal and external motivations, culture, mentality, language, and what procedures are best suited for these complaints is essential to optimal outcomes.

### Individuals of African Descent

The term “individuals of African descent” includes multiple ethnicities and persons of African, Caucasian, Afro-Caribbean, and Native American descent, or some mixture of these. Global generalizations can be difficult for this group as all these lineages will possess a unique array facial structures and characteristics [11].

The soft tissue envelope (STE) in individuals of African descent often has a more compact stratum corneum and thicker dermal collagen bundles, along with more apocrine and sebaceous glands, which causes increased transdermal water loss [12]. The most obvious difference between the skin of individuals of African descent and of Caucasians is the melanin content of the epidermis. Although the number of melanocytes is the same, the number and size of melanosomes varies, with individuals of African descent's epidermal melanosomes being more dispersed and larger [12]. The melanin content is higher in the skin of individuals of African descent, which confers protection against UV damage, and therefore, manifestations of photodamage are less severe and typically occur 10 to 20 years later than those of Caucasian counterparts of the same age [11]. Although photoprotection can be beneficial, the differences in melanin and melanosome characteristics can also cause more skin hyperpigmentation issues in individuals of African descent. Causes of hyperpigmentation include postinflammatory hyperpigmentation, melasma, and dyschromia of photoaging [12]. Indeed, in a survey of 100 women of color, 86% had complaints about dark spots, and nearly 50% had complaints about sensitive or very sensitive skin [13]. In addition to hyperpigmentation, keloids are 3–18 times more common in individuals of African descent compared to Caucasians, due to differences in the composition of fibroblasts [12]. In the skin of individuals of African descent, fibroblasts are both larger and multinucleated, and it is theorized that these fibroblasts' interaction with cytokines results in overproduction of collagen and under degradation of components of the extracellular matrix [12]. Individuals of African descent tend to have more stimulation of collagen from fillers with less thinning of collagen bundles after treatment, and thus may require fewer treatments [14].

There are structural differences between the faces of individuals of African descent and Caucasian faces. As discussed, individuals of African descent have skin that maintains its youthful appearance and structural integrity for longer than lighter skin types. On the other hand, skin of

individuals of African descent can exhibit marked descent of the malar fat pads, jowl formation, and increased soft tissue laxity [15]. The unique skeletal morphology of individuals of African descent leads to signs of facial aging more in the periorbital and midface regions compared to the upper face and brow regions.

### Latino

The skin type among Latino patients varies often according to the patient's racial mix. Those of European heritage may have a lighter complexion, while those with more African Indigenous heritage tend to have a darker complexion. The most common skin type among Latin Americans is the Mestizo skin type, which is thicker than Caucasian skin and confers a higher melanin content [10]. Because of these features, this skin tends to be more resistant to the effects of photoaging [10]. Though consultations for rhytides may be less common among this population, this patient group will present more frequently for hyperpigmentation complaints and hyperinflammatory reactions such as Pseudofolliculitis barbae [10].

Structurally, those from Central and South America often have similar anthropometric measurements to Caucasian, while those from the Caribbean are often more morphologically similar to those of African descent<sup>11</sup>. Overall, Latinos have increased bimaxillary protrusion, greater bizygomatic distance, broader nose, shorter nasal length, more rounded face, recessed chin, heavier eyelids, and prominent midface region [11]. Similar to African Americans, the midface fat pad descent is more prominent, highlighting the nasolabial folds, along with eyelid and eyebrow drooping and lower lid fat herniation [11].

### Individuals of Asian Descent

The term “individuals of Asian descent” encompasses an enormously diverse population, and the literature tends to focus on particular ethnicities or subsets of this large group of individuals, such as East Asian and South Asian. Of course, the phenotypic variations of individuals of Asian descent should be considered when addressing and analyzing the aging face.

Galzote et al. evaluated the facial skin of various Asian subsets (e.g., China, Japan, South Korea, India, and the Philippines) [16]. They reported that Japanese patients had more skin surface moisture across age groups, while Chinese patients had the greatest amount of transepidermal water loss, suggesting less robust stratum corneum function [16]. Across all studied populations, skin surface moisture, transepidermal water loss, and sebum content tended to decrease with age [16]. It was also reported that patients from Calicut,

India, had the darkest skin tone, and those from Sendai, Japan, had the lightest [16].

Structurally, there are notable differences between the face of Caucasians and of individuals of Asian descent. East Asians have narrower mouths with fuller lips, wider nasal bases, more receded chins, and elongated intercanthal widths [11]. South Asians often have higher cheekbones with more buccal fat, giving the lower cheek a more rounded contour [9••]. The Asian supratarsal crease is smaller and more camouflaged. The adipose fullness of the upper eyelid, along with epicanthal folds and narrow palpebral fissures is the hallmark of the Asian eyelid [9••]. The eyebrow to upper eyelid distance is greater in Caucasians than individuals of Asian descent [9••]. In individuals of Asian descent, a relatively weaker facial skeletal framework is seen, resulting in greater gravitational descent of the midface soft tissues and malar fat pads, along with ptosis and tear trough formation [11]. Shome et al. evaluated facial aging patterns of North, East, South, and West Indians and noted that Indians overall have more prominence of the nasolabial folds compared to Caucasians or other individuals of Asian descent [9••]. West Indians tend to have more prominent forehead wrinkles and the earliest signs of dermatochalasis, whereas South and East Indian ethnicities had more brow ptosis than West and North Indian populations over 70 years old [9••].

### Middle Eastern

Patients of Middle Eastern descent display a broad range of skin types. Similar to other ethnic skin types, postinflammatory and posttraumatic dyschromia is often a concern in this group, though the risk of keloid formation is not increased compared to Caucasians [17].

### Gender-Related Changes in Facial Aging

Gender is a significantly influential factor of facial aging. Throughout the aging process, there is a reduction in the production of sex hormones as functional reserves become depleted [18]. Postmenopausal women will experience a marked decline in serum estrogen levels, whereas most aging men will experience a slow and steady decrease in circulating testosterone levels, by about 1% annually beginning at age 30, though this can vary considerably [18]. Estrogen upregulates nuclear gene expression of antioxidant enzymes in women, conferring greater protection against reactive oxygen species [19]. Men have thicker skin at all ages, and mirroring the changes in sex hormones, postmenopausal women experience a dramatic decrease in skin and soft tissue thickness, while men have a gradual decrease over time [18]. The difference in thickness is thought to be due to differences in the amount of dermal collagen [1].

Men are more likely to partake in high-risk health behaviors, such as smoking and alcohol use, and tend to underutilize preventative dermatological evaluations compared to women [18]. Thus, rhytides develop earlier and are more severe in men, particularly in the forehead [18]. Women do develop deeper rhytides in the perioral area, however, likely due to the smaller pilosebaceous units in this region [18]. The pattern of wrinkle development differs in men, due to differences in facial musculature [18]. For example, men have more “U”-shaped glabellar lines due to increased contribution of the procerus [18]. The lateral orbicularis oculi muscle has different contraction patterns in men, with more than half demonstrating a downward fan pattern in the crow’s feet area, while women have equal likelihood of a downward, central, or full fan pattern [18].

Androgenic alopecia is a common problem, with 30–50% of Caucasian men having some degree of hair loss by age 50, and around 80% by age 70 [19]. In women, there is prevalence of mid-frontal hair loss in 57% of women over the age of 80 [19, 20].

### Assessment Scales for Facial Aging

Facial aging assessment tools are useful in assisting with the characterization and treatment of the aging face. The ideal facial aging assessment tool would be easy for clinicians to use and include all relevant signs of aging. Several facial aging assessment scales have been described, though none is considered standard, partly due to the complex features that contribute to skin aging including genetics, age, sex, and ethnic backgrounds, which create a broad spectrum of skin types that defy simple categorization [21].

#### Fitzpatrick Scale

The Fitzpatrick classification was developed in 1975 to classify skin colors and their response to UV radiation [22]. There are six types on this scale: (I) white skin, always burns, never tans, (II) white skin, always burns, minimal tan; (III) white skin, burns minimally, tans moderately and gradually; (IV) light brown skin, burns minimally, tans well; (V) brown skin, rarely burns, tans deeply; and (VI) dark brown/black skin. Though this scale is widely used in the clinical setting to describe skin color and predict the skin’s response to minimally invasive treatments such as lasers and chemical peels, it is not without limitations [21]. Primarily, the system has limited use in communication information to patients and in helping clinicians treat various ethnic skin types appropriately [21]. Patients with pigmented skin would benefit more from a classification system that characterized the unique features of ethnic skin, such as propensity of the skin to become hyperpigmented and/or to scar [21].

## Glogau Scale

The Glogau scale was developed in 1996 to characterize the clinical signs of photoaging with the purpose of organizing discussions about appropriate therapies and clinical results [23]. There are four types in this scale, type I “no wrinkles,” type II “wrinkles in motion,” type III “wrinkles at rest,” and type IV “only wrinkles.” The Glogau scale also does not account for the ethnic background of the patient.

## Roberts Scale

The Roberts scale was introduced to aid the clinician in predicting the skin’s response to inflammation and/or injury by including a four-part patient profile, including obtaining clinical and ancestral history, physical examination, visual examination, and test site reactions. [24] Patient’s skin is given an alphanumeric descriptor that describes propensity for pigmentation ( $H_{0-4}$ ) and scar morphology ( $S_{0-5}$ ) [24].

## Other Scales

Several multidimensional assessment scales have been developed with the intention of being more comprehensive for all skin types [25–29]. While these scales are considered methodologically robust, they are not currently widely used, possibly because they are seen as overly complex and contain unclear terminology, such as “pigmented spot” and “benign tumor,” which may have different interpretations depending on the provider [30]. Additionally, some of these scales require permission prior to use, which limits widespread clinical use.

In recent years, Carruthers et al. have developed a set of validated grading scales for brow positioning, forehead lines, melomental folds (marionette lines), and crow’s feet [31–34]. The scales were designed for use in clinical trials as well as in assessing outcomes after facial fillers and neurotoxins, though the test–retest reliability on some of these assessments was only moderate [35]. While these newer assessment scales do well to address some of the limitations of older tools, more studies are needed to test validity and reliability.

## Conclusions

Facial aging is a dynamic and complex process that involves knowledge of skin physiology, extrinsic and intrinsic factors of aging, facial anatomy, ethnic differences in facial aging, and assessment tools that can help guide the clinician on the most appropriate treatment modalities of each individual patient.

## Declarations

**Research Involving Human and Animal Participants** This article does not contain any studies with human or animal subjects performed by any of the authors.

**Conflict of Interest** The authors declare no competing interests.

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● Of importance

●● Of major importance

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