

## SKIN AGING

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**SUMMARY** – Skin aging is a multisystem degenerative process that involves the skin and skin support system. Young faces tend to be convex with full lips, sweeping jaw line with full temples and cheeks. Aged face tends to be concave with flat lips, sunken temples and cheeks, scalloped mandible and more shadows. Aging caused by the genes we inherit and depending on the passage of time *per se* is called chronological or intrinsic aging. Intrinsic skin aging is characterized by atrophy of the skin with loss of elasticity and slowed metabolic activity. The signs of intrinsic aging are fine wrinkles, thin and transparent skin, loss of underlying fat, facial bone loss, dry skin, inability to sweat sufficiently to cool the skin, hair loss and unwanted hair. The other type of aging is known as extrinsic aging and is caused by environmental factors. Among harmful environmental factors that contribute to extrinsic aging, long-term effects of repeated exposure to ultraviolet light are most significant and are referred to as photoaging. It is a cumulative process and depends primarily on the degree of sun exposure and skin pigment. UV irradiation invokes a complex sequence of specific molecular responses that cause damage to the skin connective tissue. Photoaging affects the sun-exposed areas and is characterized clinically by fine and coarse wrinkling, roughness, dryness, laxity, telangiectasias, loss of tensile strength and pigmentary changes. There is also an increase in development of benign and malignant neoplasms on photoaged skin.

Key words: *skin aging, photoaging, UV radiation*

By the third decade of life, the skin begins to change. Skin aging is a progressive process in which environmental damage superimposed on aging skin determines the ultimate skin appearance. Aging is a multisystem degenerative process that involves the skin and the skin support systems including the bone, cartilage, and subcutaneous compartments<sup>1</sup>.

There are two distinct types of aging. Aging caused by the genes we inherit and depending on the passage of time *per se* is called chronological or intrinsic aging. The other type of aging is known as extrinsic aging and is caused by environmental factors such as sun exposure. Age is not the determining factor in the condition of mature skin. Environmental factors that influence aging of the skin play a central role. Tone, elasticity and

epidermal regeneration capacity do not decline until advanced age in areas not exposed to light, whereas they do so prematurely in areas exposed to light<sup>2</sup>.

### Intrinsic Aging

Intrinsic aging, also known as the natural aging process, is a continuous process that normally begins in the mid-twenties. Within the skin, collagen production slows down and elastin has a bit less spring. Dead skin cells do not shed as quickly and turnover of new skin cells may decrease slightly. While these changes usually begin in the twenties, the signs of intrinsic aging are typically not visible for decades. The signs of intrinsic aging are fine wrinkles, thin and transparent skin, loss of underlying fat, bones shrink away from the skin due to bone loss, which causes sagging skin, dry skin, inability to sweat sufficiently to cool the skin, graying hair, hair loss and unwanted hair<sup>3</sup>. Young faces tend to be convex with full lips,

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sweeping jaw line and full temples and cheeks. Aged face tends to be concave with flat lips, sunken temples and cheeks, scalloped mandible and more shadows<sup>4</sup>.

The skin support system includes the bone, cartilage, and subcutaneous compartments, which provide the architectural support for the dermis, epidermis and stratum corneum. A multipronged approach to aging involves reversing the undesirable changes in each of these structures. One of the most important areas for consideration is the bony architecture over which the skin lies. Without a strong framework, the skin hangs formless over the face. Bone demineralization begins at around 25 and leads to dulling of the facial features. Bone replacement therapy, such as bisphosphonates, is usually not initiated until overt evidence of osteoporosis is present. The architecture of the cartilage of the face with bones defines the shape of the face. The most important facial structure dependent on cartilage is the nose. The cartilage does not disappear with advancing age, but does change in shape. The change occurs during pregnancy due to relaxins that are secreted at high levels during the final trimester<sup>1</sup>.

The subcutaneous compartment undergoes much of the change that contributes to the aged appearance of the face. In intrinsic aging, there is a decreased fat cell size, diminished fat cell function, impaired fat cell differentiation and redistribution of fat cells. Subcutaneous fat from all over the body is removed and lost, including facial fat, and redeposited intra-abdominally, probably to lower growth hormone levels<sup>5</sup>. The face shows excess concavity with prominent muscles and bony landmarks as the result of lipoatrophy<sup>4</sup>. Fat redistribution in the body occurs with menopause<sup>6,7</sup>. In postmenopausal women, fat is typically redistributed to the breast, arms, waist, thighs and buttock with loss of facial fat. Volume loss begins to occur under the eyes in the tear troughs, as well as in the cheek area. Volume loss becomes evident around the nasolabial folds due to the loss of volume in the cheek and perioral area. Prominence of the nasolabial fold is a major change associated with midface aging. This fold has a dynamic stage in earlier life and a static appearance with aging.

Viable epidermis and dermis are the essence of the skin. Structural destruction and loss of dermal collagen fiber bundles lead to wrinkling and increased appearance of muscular attachments. Irregular melanization

leads to lentigines, poikiloderma, and melasma and prominent telangiectasias lead to erythema<sup>8</sup>.

With aging, the loss of extracellular matrix and its major component hyaluronate, which stabilizes the intracellular structures by forming viscoelastic network in which collagen and elastin fibers are embedded, induces loss of the skin mechanical functions. Hyaluronate provides a cushion effect to the skin structures including the epidermis. Solidity of the skin is provided by the extracellular matrix and the loss of hyaluronate and consequently of the viscoelastic buffering system would contribute to easy tearing resulting in skin lacerations<sup>9</sup>. The treatable stratum corneum problem that leads to fine wrinkling is dehydration. The corneocytes and intercellular lipids can be restored to their normal brick-and-mortar lamellar organization<sup>1</sup>.

### Extrinsic Aging

A number of extrinsic or external factors often act together with the normal aging process to prematurely age our skin. External factors that prematurely age the skin are repetitive facial expressions, sun, gravity, sleeping positions, and smoking.

Repetitive facial movements actually lead to fine lines and wrinkles. When we use a facial muscle, a groove forms beneath the surface of the skin, which is why we see lines to form with each facial expression. As skin ages and loses its elasticity, the skin stops springing back to its line-free state, and these grooves become permanently etched on the face as fine lines and wrinkles. Changes related to gravity become more pronounced with aging. When the skin elasticity declines in middle age, the effects of gravity become evident. Gravity causes the tip of the nose to droop, the ears to elongate, the eyelids to fall, the jowls to form, and the upper lip to disappear while the lower lip becomes more pronounced. Resting your face in the same way every night for years eventually also leads to wrinkles. Sleep wrinkles become etched on the surface of the skin and no longer disappear.

Cigarette smoking causes biochemical changes in the body that accelerate aging. People who smoke for a number of years tend to develop an unhealthy yellowish hue and deeply wrinkled, leathery skin not seen in non-smokers. These signs can be greatly diminished by stopping smoking<sup>2,3</sup>.

Most premature aging is caused by sun exposure. Unlike chronological aging, which depends on the passage of time *per se*, photoaging depends primarily on the degree of sun exposure and skin pigment. Individuals who have outdoor lifestyles, live in sunny climates and are lightly pigmented will experience the greatest degree of photoaging<sup>10</sup>.

Among harmful environmental factors that contribute to extrinsic aging, long-term effects of repeated exposure to UV radiation are most significant and are referred to as photoaging. Photoaging is directly correlated to the quantity of UV rays received during the course of lifetime. The effects of photodamage are often evident many years before intrinsic aging is apparent. Young people who are exposed to a great amount of UV rays appear prematurely aged.

### Mechanisms of Photoaging

Photoaging is a multisystem degenerative process that involves the skin and the skin support system. In skin with long-term sun exposure, the ratio of melanocyte density is approximately twice that of non-exposed skin<sup>11</sup>. Prominent telangiectasias lead to erythema and loss of hydration in stratum corneum leads to fine wrinkling. Photoaging affects the sun exposed areas and is characterized clinically by fine and coarse wrinkling, roughness, dryness, laxity, telangiectasias, loss of tensile strength and pigmentary changes. There is also an increased development of benign and malignant neoplasms on photoaged skin. It is a cumulative process and depends primarily on the degree of sun exposure and skin pigment. The epidermis and dermis are both affected by UVB, but the dermis is also affected to a significant extent by UVA. It has long been thought that the majority of human photolesions are due to UVB rays, while now it is believed that UVA play a substantial role in photoaging. Because UVB is essentially completely absorbed in the epidermis, it is important to understand that photoaging changes can be produced by UVA alone. Indeed, these changes are produced in photoprotected skin by a small number of low-dose exposures of UVA radiation<sup>12</sup>.

With chronic skin exposure to UV rays, the epidermis responds with hypertrophy. The stratum corneum thickens, the epidermis becomes acanthotic, and there is progressive dysplasia with cellular atypia, and anaplasia. Keratinocytes are irregular with the loss of polarity. Melanocytes are irregular with pockets of in-

creased and decreased numbers. The Langerhans cell population in the epidermis is reduced, which contributes to an impaired immune response to antigen and skin cancer cells<sup>10,11</sup>. The roughness of photoaged skin is the result of a combination of changes in stratum corneum and changes in the glycosaminoglycan content of the dermis. With age, there is a decrease in glycosaminoglycans in the dermis. In photoaged skin, there is a paradoxical increase in glycosaminoglycans when compared with intrinsically aged skin. But, they are deposited on the abnormal elastotic material rather than in the papillary dermis and this location may make them unavailable as a source of hydration<sup>12</sup>. Photoaged skin displays thickened basement membrane. Dermal changes in photoaged skin are reduction in collagen and precursors of types I and III collagen, degeneration of elastic fibers, which are replaced with time by an amorphous mass, and chronic inflammation with an increase in degranulated mast cells, macrophages and lymphocytes. Blood vessels are dilated and tortuous<sup>13</sup>. In addition, because of the diminution of the collagen framework, the blood vessels are poorly supported; they can easily rupture, resulting in solar purpura.

### Conclusion

Skin aging is the end result of both intrinsic aging, which is the result of the passage of time, and photoaging, which refers to alterations in skin structure and function that result from chronic sun exposure, in addition to the passage of time. It is important to distinguish between chronological skin aging and photoaging.

Chronological skin aging can be summarized as atrophy with structural and functional decline of the skin. Sun-protected aged skin is finely wrinkled with exaggeration of facial expression lines, laxity, and pallor.

Extrinsic skin aging involves hypertrophy as an inflammatory, protective response to the damaging effects of UV rays. Photoaging is manifested by xerosis, leathery skin, irregular pigmentation (freckles, lentigines), and more pronounced wrinkling. Telangiectasias, purpura, comedones, and a variety of benign and premalignant skin tumors including seborrheic keratoses, acrochordons, sebaceous gland hyperplasia, and actinic keratoses are frequent findings. Ultraviolet

irradiation from the sun damages human skin, causing it to age prematurely. Sun protection can prevent many of the changes associated with photoaging.

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## Sažetak

### STARENJE KOŽE LICA

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Starenje kože je degenerativni proces koji uz kožu zahvaća i potporne strukture kože. Mlado lice je konveksno s punim usnama i obrazima, a linija mandibule je uzdignuta prema obrazima. Lice koje pokazuje znakove starenja obilježavaju tanke usne, nejednolikosti pigmentacije, opušteni obrazi i čelo, opuštena mandibula i cijelo lice se doima konkavnim. Starenje uzrokovano genskim nasljeđem i koje ovisi o prolasku vremena zovemo kronološkim starenjem ili intrinzičnim starenjem. Ono je obilježeno atrofijom kože, gubitkom elastičnosti i usporenjem metaboličnih aktivnosti. Znakovi kronološkog starenja su sitne bore, tanka i transparentna koža, gubitak podložnog masnog tkiva i koštane strukture lica, suhoća kože, nemogućnost žlijezda znojnice da dostatno hlade kožu, gubitak kose i pojava neželjene dlakavosti. Drugi tip starenja naziva se ekstrinzično starenje i uzrokovano je utjecajem vanjskih čimbenika. Između brojnih štetnih utjecaja okoline koji sudjeluju u starenju dugotrajno i opetovano izlaganje ultraviolettom zračenju je najznačajnije te uzrokuje foto-starenje kože. To je kumulativni proces koji prvenstveno ovisi o stupnju izloženosti sunčevim zrakama i pigmentu kože. UV zračenje uzrokuje složene procese na specifičnim molekulama odgovor kojih uzrokuje oštećenja vezivnog tkiva kože. Foto-starenje se očituje pojavom naboranosti, crvenilom kože, suhoćom, gubitkom elastičnosti, pojavom telangiektazija te pigmentnim promjenama. Izloženost UV zračenju povećava pojavu dobroćudnih i zloćudnih novotvorina kože na fotoekspoziranim dijelovima.

Ključne riječi: *starenje kože, fotostarenje, ultraviolettom zračenje*