

Lesson 16 The aging process and assessment tools

Intrinsic Aging (Chronological Aging):

Intrinsic aging is the natural aging process that occurs due to internal factors and the passage of time. It primarily involves changes in the skin's structure and function at the cellular and molecular levels.

Causes:

Genetics: Genetic factors play a significant role in determining how our skin ages. The rate of intrinsic aging is partly determined by our genetic makeup.

Cellular Changes: Over time, skin cells, particularly fibroblasts, which produce collagen and elastin, become less active. This leads to a decrease in the production of these essential proteins, resulting in reduced skin elasticity and firmness.

Decline in Hyaluronic Acid: Hyaluronic acid, a molecule responsible for skin hydration, decreases with age. This contributes to a loss of skin plumpness and moisture.

Effects:

Wrinkles and Fine Lines: Reduced collagen and elastin production results in the formation of wrinkles and fine lines, particularly around the eyes and mouth.

Thinning of the Epidermis: The outermost layer of the skin, the epidermis, becomes thinner, making the skin more susceptible to injury and damage.

Dryness: A decrease in oil production by sebaceous glands can lead to dry skin.

Slower Wound Healing: Skin's ability to repair itself diminishes, leading to slower wound healing and a higher risk of infection.

Physiology of Chronological Skin Aging at Cellular Level

Aging takes place at cellular and molecular levels. These changes significantly impact how cells function and contribute to the aging of tissues and organs, including the skin.

Cellular Senescence: One hallmark of cellular aging is cellular senescence. Senescence is a state in which cells lose their ability to divide and replicate. This occurs for several reasons:

Telomere Shortening: Telomeres are protective caps at the ends of chromosomes that shorten with each cell division. When telomeres become critically short, cells enter a state of senescence to prevent genomic instability.

DNA Damage: Accumulation of DNA damage over time, caused by factors like UV radiation and oxidative stress, can trigger cellular senescence.

Activation of Tumour Suppressor Genes: Certain genes, like p53, become activated in response to cellular stress and can induce senescence as a protective mechanism against cancer development.

Decreased Cellular Function: Aging cells often exhibit a decline in various cellular functions, including:

Reduced Energy Production: Mitochondria, the cell's energy factories, become less efficient with age, resulting in decreased energy production, and increased oxidative stress.

Impaired Protein Synthesis: Protein synthesis becomes less accurate, leading to the production of misfolded or damaged proteins.

Altered Signalling Pathways: Signalling pathways that regulate cellular processes, such as growth and repair, can become dysregulated in aging cells.

Decreased Cellular Turnover: The rate of cellular turnover and renewal decreases with age, resulting in a slower replacement of damaged cells.

Accumulation of Cellular Damage: Aging cells accumulate various types of damage, including:

Oxidative Damage: Reactive oxygen species (ROS) generated during normal cellular processes can damage lipids, proteins, and DNA, contributing to cellular aging.

Protein Aggregates: Misfolded and damaged proteins can aggregate within cells, impairing cellular function.

Lipofuscin Accumulation: Lipofuscin, a yellow-brown pigment, accumulates in cells over time and is associated with age-related cellular dysfunction.

Inflammation and Senescence-Associated Secretory Phenotype (SASP): Senescent cells often exhibit a pro-inflammatory state known as the SASP. These cells release pro-inflammatory cytokines, chemokines, and growth factors. SASP can contribute to chronic low-grade inflammation, a hallmark of aging known as "inflammaging." This inflammation can further damage neighbouring cells and tissues.

Autophagy Decline: Autophagy is a cellular process responsible for the removal of damaged organelles and proteins. With aging, autophagic activity tends to decline, leading to the accumulation of cellular waste and damaged components.

Reduced Repair and Regeneration: Aging cells have a reduced capacity to repair DNA damage, recover from stress, and regenerate damaged tissues. This contributes to the overall decline in tissue function and repair seen in aging individuals.

Telomere Shortening and Aging Clock: Telomere shortening is often considered a cellular aging clock. It's associated with aging-related diseases and mortality. Shortened telomeres can trigger cellular senescence and contribute to the aging process.

Understanding these cellular changes in aging is crucial for developing strategies to mitigate their effects and promote healthy aging. Researchers are exploring various interventions, such as caloric restriction, exercise, and pharmacological agents, to target these cellular processes and potentially slow down the aging process or reduce the risk of age-related diseases.

Extrinsic Aging (Environmental Aging):

Extrinsic aging is primarily influenced by external factors, such as exposure to the sun, pollution, smoking, and lifestyle choices. These factors accelerate the aging process and result in visible skin changes.

Causes:

Ultraviolet (UV) Radiation: UV radiation from the sun is one of the most significant extrinsic factors. It damages collagen and elastin fibres, causing premature wrinkles and skin sagging.

Tobacco Smoke: Smoking releases harmful chemicals that constrict blood vessels, reducing blood flow to the skin. This diminishes the skin's oxygen and nutrient supply, leading to premature aging and wrinkles.

Pollution: Air pollution, particularly fine particulate matter (PM2.5), can contribute to oxidative stress and inflammation in the skin, accelerating aging.

Diet and Lifestyle: Poor nutrition, excessive alcohol consumption, and stress can contribute to skin aging by increasing oxidative stress and inflammation.

Effects:

Photoaging: UV exposure leads to photoaging, characterized by deep wrinkles, brown spots (solar lentigines), and a leathery texture.

Uneven Skin Tone: Prolonged sun exposure can result in uneven skin tone and the development of age spots or sunspots.

Loss of Skin Elasticity: Smoking and exposure to pollutants can cause a breakdown of collagen and elastin, resulting in sagging skin.

Reduced Skin Barrier Function: Environmental factors can weaken the skin's natural barrier, leading to increased sensitivity and a higher risk of irritation.

Mitigation and Prevention:

Sun Protection: Regular use of broad-spectrum sunscreen with a high SPF, protective clothing, and seeking shade can help prevent UV-induced damage.

Smoking Cessation: Quitting smoking can improve skin health and slow down the aging process.

Healthy Lifestyle: A balanced diet rich in antioxidants, proper hydration, and stress management can support skin health.

Topical Skincare: The use of topical products containing antioxidants, retinoids, and moisturizers can help mitigate some signs of aging.

In summary, skin aging is a complex process influenced by intrinsic and extrinsic factors. Understanding the causes and effects of aging on the skin is crucial for developing effective strategies to maintain skin health and minimize the visible signs of aging.

Changes to the skeletal structure with aging

Bone Resorption:

With age, the process of bone resorption becomes more pronounced in the facial skeleton.

The bones lose density and volume due to a decrease in osteoblastic activity and an increase in osteoclastic activity.

Maxillary Atrophy: The maxilla undergoes resorption, leading to a decrease in height and projection.

This contributes to changes in dental occlusion and can lead to sagging of the soft tissues in the midface.

Mandibular Changes: The mandible experiences bone loss, leading to a reduction in its dimensions.

The angle of the mandible can become more obtuse, impacting the overall jawline definition.

Orbital Rim Changes: The orbital rims may thin and recede, resulting in changes to the eye socket's shape and depth.

These changes can contribute to the appearance of sunken eyes and the formation of tear troughs.

Changes to the Nasal Skeleton: The nasal bones and nasal septum can experience resorption and deformity. These changes contribute to alterations in nasal shape, including drooping of the nasal tip.

Zygomatic Arch Changes: The zygomatic arch may undergo thinning and resorption. This can result in a loss of facial width and contribute to changes in midface proportions.

Changes to the Chin and Mandible: The chin and mandible may lose projection due to bone resorption. This can lead to a weaker jawline and reduced chin prominence.

Changes to Facial Symmetry: Asymmetry becomes more pronounced with age due to uneven bone loss and remodelling. Changes in bone volume and density contribute to variations in facial symmetry.

Changes to Cranial Bone Structure: The bones of the cranial vault may experience subtle changes, leading to alterations in head shape and size.