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The Impact of Environmental Factors on Skin and Tissue Ageing: Mechanisms, Effects, and Preventive Strategies

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Abstract:

Introduction: Ageing is a natural biological process that affects every living being. With the passing of years, due to both internal and external factors, the skin's functions gradually weaken, and its regenerative processes slow down. This manifests as a decrease in elasticity, discoloration, and other visible signs of ageing. Factors such as sunlight, air pollution, smoking, alcohol consumption, diet, and physical activity play a crucial role in determining the rate of the ageing process.

The aim of the study: The aim of this article is to review the ageing process of the skin and other tissues, along with the impact of environmental factors. The paper focuses on mechanisms like oxidative stress, inflammation, and the decline in collagen and elastin production.

Material and Methods of Research: The literature was collected through searches in the PubMed, Google Scholar databases, and references from the initially retrieved articles with keywords including “skin aging” “environmental factors” and “oxidative stress”.

Conclusion: The ageing process encompasses structural and functional changes in the skin and other tissues, as well as alterations at the cellular and molecular levels. With advancing age, collagen and elastin production decreases, resulting in reduced elasticity, firmness, and resilience. Additionally, diminished bone and fat volume weakens structural support, leading to sagging skin and the development of fine wrinkles. The facial contour changes, and the wound-healing process becomes slower. Both genetic and environmental factors influence the ageing process. Environmental contributors such as UV radiation, air pollution, smoking, alcohol consumption, and an unhealthy diet exacerbate oxidative stress, thereby accelerating skin ageing. Conversely, adopting a healthy lifestyle—including regular physical activity, a diet rich in antioxidants, and avoiding excessive sun exposure—can help mitigate these effects and slow the ageing process.

Keywords: skin aging, oxidative stress; inflammation; antioxidants; environmental factors

Introduction

Ageing is a natural biological process that affects every living being. The skin, as the largest organ in the human body, performs a range of essential functions, including protection, thermoregulation, and sensory perception. Over time, both intrinsic and extrinsic factors lead to a gradual decline in these functions and a slowdown in the skin's regenerative processes.[1] This decline manifests as reduced elasticity, discoloration, and other visible signs of ageing.[2] However, it is not only the skin that influences appearance and perceived attractiveness. Changes in other tissues, such as subcutaneous fat, muscle, bone, and connective tissue, also play a significant role in the ageing process.[3] Environmental factors, such as sun exposure, air pollution, smoking, alcohol consumption, diet, and physical activity, are critical determinants of the rate at which ageing occurs. In particular, UVA and UVB radiation, along with infrared and visible light, contribute significantly to photoageing by generating reactive oxygen species (ROS). [4] Furthermore, environmental pollution and unhealthy lifestyle choices exacerbate oxidative stress, accelerating the ageing of both the skin and underlying tissues. A comprehensive understanding of the mechanisms underlying ageing, as well as the influence of modifiable lifestyle factors, is essential for developing effective preventive strategies. By addressing these factors, it is possible not only to improve external appearance but also to enhance overall health and quality of life

The Processes of Ageing in the Skin, Adipose, Bone, and Muscle Tissues

Skin ageing is a multifactorial biological process characterized by progressive changes at the cellular, molecular, and tissue levels. This process can be classified into two distinct types: intrinsic ageing, also known as chronological ageing, which is primarily governed by genetic and endogenous factors, and extrinsic ageing, commonly referred to as photoageing, which is predominantly induced by environmental stressors, including ultraviolet radiation, air pollution, and lifestyle-related behaviors.[5] Both types of ageing contribute to the structural and functional decline of the skin, leading to visible signs such as wrinkles, loss of elasticity, and impaired barrier function. Keratinocyte renewal slows with age, leading to an accumulation of dead cells on the skin's surface and a rough texture. The flattening of the dermal-epidermal junction impairs the transport of nutrients between the dermis and epidermis. A reduction in fibroblast activity and collagen synthesis results in stiff, disorganized collagen fibers.[6] Furthermore, changes occur in the extracellular matrix, particularly in glycosaminoglycans and proteoglycans, such as hyaluronic acid, chondroitin sulfate, and dermatan sulfate. Proteoglycans like versican show reduced expression at both the mRNA and protein levels. [7] Skin ageing leads to the weakening of the epidermal barrier, with a reduction in sebum and lipid production. This results in increased transepidermal water loss, skin dryness, and heightened vulnerability to micro-injuries, irritation, as well as bacterial, fungal, and viral infections. Pruritus associated with dehydration diminishes patient comfort.[8]A reduced number of blood vessels results in compromised blood circulation in the skin, weakening its regenerative capacity and impairing the delivery of essential nutrients. Consequently, this vascular dysfunction leads to prolonged wound healing and an increased propensity for scarring.[9] Changes in melanocytes contribute to uneven skin pigmentation, often presenting as solar lentigines or age spots, particularly in sun-exposed areas.[10]The reduction in the number and function of dermal blood vessels compromises circulation, which further impairs the skin's regenerative abilities. Telangiectasias may develop, appearing as small, visible blood vessels. Furthermore, erythrocyte extravasation into the dermis leads to purpura, which eventually resolves as brownish-yellow discoloration due to hemosiderin deposition. These changes are more visible as a result of

thinning of the epidermis. In older patients, such lesions may appear spontaneously, but cognitive impairments, such as dementia, may prevent them from recalling prior injuries.[1,8]

Adipose tissue

With age, subcutaneous white adipose tissue (sWAT), primarily composed of adipocytes, undergoes significant changes that directly affect the mechanical properties of the skin. These changes include a reduction in sWAT thickness [11] and alterations in collagen structure, such as decreased organization and quantity of collagen fibers.[12] Notably, a marked reduction in sWAT is observed in key facial regions, including the suborbital area, medial cheek, temporal region, and nasolabial cushions, contributing to decreased skin elasticity and changes in overall appearance.[13]

Studies have shown that facial adipocytes exhibit distinct characteristics compared to adipocytes from other regions of the body, such as the abdomen. Notably, they possess lower lipolytic activity and reduced expression of beta-adrenergic receptors, resulting in decreased fat mobilization. Additionally, differences in the expression of genes, including homeobox genes—key regulators of adipogenesis—have been identified. These distinctions may have significant implications for understanding regional variations in fat deposition and metabolism.[12] On the face, adipose tissue, in addition to contributing to the subcutaneous tissue layer, serves as a support structure for the skin. A reduction in adipose tissue volume leads to diminished support, resulting in skin laxity and the formation of superficial wrinkles. These changes significantly affect the overall facial appearance. The loss of fat in specific compartments, such as the malar fat pad and suborbicularis oculi fat, results in sunken cheeks and the development of "shadows" beneath the eyes. The gravitational displacement of fat across the face induces alterations in facial contours, including the formation of nasolabial folds and the development of "bags" under the eyes. The redistribution of fat from the lower face may also contribute to the formation of a sagging chin and loose skin on the neck.[14]

Muscle tissue

Facial muscles, also known as the muscles of expression, play a crucial role in the manifestation of emotions and non-verbal communication. The ageing of these muscles is a significant process that influences both the appearance and the expressiveness of the face. A distinctive feature of facial muscles is their direct attachment to the skin. Major groups of these muscles include the frontalis (forehead), orbicularis oculi (around the eyes), zygomaticus major (cheek), depressor anguli oris (muscle of the mouth corners), orbicularis oris (around the mouth), and buccinator (cheek). Repetitive contractions of these muscles lead to the formation of dynamic wrinkles, which, over time, may become static wrinkles. These wrinkles are most commonly observed in the periorbital region (crow's feet), on the forehead (fine lines), and around the mouth (marionette lines and nasolabial folds). [15] Ageing is associated with sarcopenia, a process characterized by the progressive loss of muscle strength and mass in all skeletal muscles, including the facial muscles. The loss of muscle mass results in impaired function and a reduction in volume, which subsequently alters the shape and reduces the expressiveness of the face. Additionally, diminished blood supply and nutrient delivery further compromise muscle function. The less efficient transport of oxygen and nutrients contributes to muscle weakness and atrophy. Ageing also results in a progressive decline in the elasticity and tone of the facial muscles, leading to the formation of wrinkles and skin folds. This is particularly pronounced in regions subjected to frequent muscular activity, such as the forehead, periorbital area, and around the lips, and may contribute to the sagging of the eyelids, cheeks, and eyebrows [16]

Bone tissue

Facial bones play essential structural and protective roles by providing support for facial muscles, skin, and other soft tissues, which shape the overall appearance of the face. The primary facial bones include the maxillary, mandibular, zygomatic, nasal, lacrimal, and palatal bones. Like bones in other regions of the body, facial bones undergo a loss of mass and volume with age, resulting from an imbalance between bone resorption and formation. [17] This loss of bone mass is particularly evident in the maxillary and mandibular bones, leading to a reduction in facial projection and a collapse of the bony framework. Additionally, the orbital and nasal regions become more hollow and more defined.[18] Similar to adipose tissue, the reduction in bone mass and the associated changes in facial bone structure diminish the support for the overlying skin and muscles. Consequently, this leads to sagging skin and the formation of wrinkles and folds, including marionette lines and nasolabial furrows. Moreover, these alterations contribute to the displacement of facial muscles, changes in facial expressions, and the shifting and atrophy of fatty tissue.[17]

Biological Mechanisms of Skin Ageing

In addition to the structural changes observed in the skin and other tissues, ageing is characterized by a range of mechanisms at the cellular and molecular levels. These mechanisms encompass oxidative stress, the processes of protein glycation, the activity of matrix metalloproteinases, and alterations in hormonal metabolism.[19-27]

Oxidative stress

Free radicals are highly reactive molecules that are produced during normal metabolic processes and as a consequence of exposure to UV radiation, environmental pollution, and tobacco smoke. These reactive species have the potential to damage cellular components, including DNA, proteins, and lipids, within skin cells. An excess of free radicals leads to oxidative stress, which accelerates the degradation of collagen and elastin and triggers inflammatory processes in the skin. As the skin ages, its intrinsic ability to produce antioxidants diminishes, increasing its susceptibility to oxidative damage.[23] Furthermore, telomeres, which are structures located at the ends of chromosomes, progressively shorten in cells. These structures are essential for protecting DNA from damage during cell division. As telomeres shorten, it triggers apoptosis and leads to a decline in the cells' proliferative capacity.[20]

Protein glycation

Glycation is a non-enzymatic process involving the covalent attachment of sugar molecules to proteins, lipids, and nucleic acids, leading to the formation of advanced glycation end products (AGEs). These molecules induce the stiffening and structural impairment of key proteins such as collagen and elastin, resulting in reduced skin elasticity and integrity. Diets high in simple sugars and processed foods promote glycation and AGEs accumulation, accelerating cutaneous ageing.[24] In contrast, diets enriched with antioxidants and anti-glycation agents, such as carnosine, may attenuate AGE formation and protect skin function.[25]

Degradation of the Extracellular Matrix

Matrix metalloproteinases (MMPs) are enzymes responsible for the degradation of extracellular matrix (ECM) components such as collagen and elastin. The activity of MMPs is elevated by factors such as UV radiation, inflammation, and oxidative stress.[26] When MMP activity becomes excessive, it leads to the breakdown of collagen and elastin fibers, which contributes to the loss of skin firmness and elasticity, and accelerates the visible signs of aging. MMP activity is regulated by natural inhibitors, known as tissue inhibitors of metalloproteinases (TIMPs). These inhibitors help protect the ECM by regulating MMP activity. However, with aging, the production of TIMPs tends to decrease, which results in an increase in MMP activity and a faster rate of ECM degradation. This imbalance between MMPs and TIMPs is a key factor in skin aging and the degradation of other tissues [27]

Hormone Management

Estrogens are essential hormones for maintaining both the health and appearance of the skin. They stimulate the production of key components such as collagen, elastin, and hyaluronic acid, which are crucial for preserving the skin's moisture balance and firmness. Estrogens also promote epidermal thickness by increasing the number and size of keratinocytes, as well as enhancing the integrity of the dermal-epidermal junction. In addition to their structural benefits, estrogens play a vital role in skin repair processes and possess antioxidant properties, offering protection against free radical damage and oxidative stress. A reduction in estrogen levels can disrupt melanin production, leading to pigmentation irregularities such as age spots. Moreover, lower estrogen levels slow down regenerative processes, resulting in longer healing times for wounds and increased susceptibility to skin infections.[22]

Impact of Lifestyle and Environmental Factors

Ultraviolet radiation (UV)

Ultraviolet radiation is a key factor in skin aging, with both short-term and long-term effects on the skin. The UV spectrum that reaches the Earth's surface is divided into three main segments: UVC (100–290 nm), UVB (290–320 nm), and UVA (320–400 nm). UVC radiation, the most harmful type, is almost entirely absorbed by atmospheric ozone and water vapor, preventing it from reaching the Earth's surface. Approximately 95% of the UV radiation reaching the Earth is UVA radiation, while UVB radiation constitutes the remaining 4–5%. UV

exposure triggers several mechanisms that influence skin aging. These mechanisms include DNA damage, oxidative stress, collagen degradation, pigmentary inflammation, and immunosuppression. [4] UVB and UVA radiation differ in both their depth of penetration and their effects on the skin. UVB radiation is directly absorbed by DNA in epidermal cells, leading to characteristic DNA mutations. The resulting DNA damage products, particularly pyrimidine dimers, activate melanogenesis in the skin, providing protection against further UV-induced damage.[28] In contrast, UVA radiation, which constitutes over 80% of daily UV radiation, penetrates deeper into the skin, reaching the dermal-epidermal junction and dermis. This penetration leads to the formation of reactive oxygen species (ROS), such as singlet oxygen, hydrogen peroxide, and hydroxyl radicals.[29] These ROS can cause lipid peroxidation, protein oxidation, and damage to nucleic acids and cell organelles, all of which are crucial in the photoaging process. Additionally, UVA radiation activates the transcription factor AP-1, which increases the production of matrix metalloproteinases (MMPs), enzymes that degrade collagen.[30] Furthermore, UVA exposure inhibits collagen production by blocking transforming growth factor beta (TGF-β) and reducing the expression of type I and III procollagen genes. Consequently, long-term UV exposure results in collagen loss, both due to degradation and reduced production.[31] UV radiation also triggers inflammatory responses in the skin, leading to the release of primary pro-inflammatory cytokines such as tumor necrosis factor alpha and interleukin 1. This results in the activation of enzyme systems, including cyclooxygenase and lipoxygenase, which increase the production of pro-inflammatory prostaglandins and leukotrienes.[30] Additionally, UV radiation can cause immunosuppression, which exacerbates skin damage and impairs the recognition and elimination of cancerous and precancerous lesions.[32] UVA and UVB radiation also differ in their effects on skin pigmentation. Exposure to UVA leads to immediate pigment darkening, resulting from the photo-oxidation of melanin, while UVB induces a delayed tanning response through the activation of melanogenesis. Immediate pigment darkening fades quickly, but after a sufficient dose of UVA (>10 J/cm^2), permanent pigmentation may occur. This poses a significant concern for darker-skinned populations due to their higher susceptibility to hyperpigmentation.[33]

Visible light

Visible light covers the wavelength range from approximately 400 to 700 nm, is visible to the human eye, and constitutes a significant portion of the solar radiation reaching the Earth's surface. Although less energetic than UV radiation, visible light also plays an important role in skin aging. It is a natural component of solar radiation that we regularly encounter in everyday life. Visible light can affect the skin through several mechanisms, including the induction of oxidative stress, activation of the inflammatory response, and effects on melanin production.[34] Within the skin, endogenous chromophores—compounds that absorb visible light—include melanin, flavins, and porphyrins. In these chromophores, electron transitions from the ground state to the excited state occur, causing photosensitization of cells and leading to the generation of reactive oxygen species. [35]

Infrared radiation (IR)

Infrared radiation (IR) spans the wavelength range from 800 nm to 1 mm and is categorized into three types based on increasing wavelengths: IR-A (700-1400 nm), IR-B (1400-3000 nm), and IR-C (3000 nm-1 mm). Approximately half of the solar energy reaching the Earth's surface is in the IR range, indicating that infrared radiation can have a significant biological effect on human skin. In direct sunlight, the temperature of human skin rises to approximately 40°C due to the conversion of absorbed IR radiation into heat. This temperature increase is more pronounced in darker skin compared to fair skin. While IR-B and IR-C radiation do not penetrate deeply into the skin, they still cause an increase in skin temperature. In contrast, more than 65% of the more energetic IR-A radiation reaches the dermis and subcutaneous layer without raising its temperature. IR-A radiation contributes significantly to extrinsic skin aging through the induction of oxidative stress, promotion of metalloproteinase production, and alterations in the extracellular matrix.[36] Similar to ultraviolet radiation, IR-A leads to collagen degradation by increasing the expression of the collagen-degrading enzyme matrix metalloproteinase 1 (MMP-1) and reducing the synthesis of new collagen. Skin exposure to infrared radiation induces the formation of intracellular reactive oxygen species that activate protein kinase signaling pathways, leading to enhanced MMP-1 expression. [37] Paradoxically, low-level IR-A radiation can stimulate cellular mechanisms and even promote collagen production through a process known as photobiomodulation. Low-level IR-A exposure also offers therapeutic benefits and applications in prevention, preparing the skin for future damage or injury.[38,39]

Ozone

Ozone (O_3) is considered one of the most toxic environmental agents to which humans are constantly exposed. The average ozone concentration in the troposphere is typically less than 0.08 ppm, which is much lower than in the stratosphere (10 ppm). However, in large metropolitan areas such as Mexico City, Rome, Milan, and Paris, O_3

concentrations can reach significantly high toxic levels, especially during the summer.[40] Although ozone cannot penetrate the skin, the damage caused by O_3 primarily results from its ability to induce oxidative stress on the skin surface, through the formation of lipid and protein peroxidation products. Exposure to high levels of atmospheric ozone leads to the depletion of antioxidants and the activation of markers of stress and inflammation, not only in the outer layers of the skin but also in the fluids lining the respiratory tract.[41] Ozone induces cytotoxicity, inhibits cell proliferation, and increases the formation of HNE protein adducts. It also reduces the activation of the transcription factor NF-κB, which is involved in the transcription of pro-inflammatory cytokines.[42] Furthermore, there is a correlation between ozone exposure and the exacerbation of various skin diseases. These conditions include urticaria, eczema, dermatitis, rashes, other non-specific eruptions, skin infections, and other dermatological disorders.[43]

Particulate Matter

Particulate matter (PM), also known as airborne particulate matter, ranges in size from 10 μm to 2.5 μm. A distinction is also made for ultrafine particles, which are smaller than 100 nm. These particles primarily result from industrial and road pollutants, as well as from activities such as cooking with impure fuels, inefficient technologies, and the burning of candles and firewood in confined spaces. Despite their small size, these particles have a large surface area, allowing them to absorb organic and inorganic compounds such as trace metals and polycyclic aromatic hydrocarbons.[4] Air pollutants induce oxidative stress in the deeper layers of the skin through the activation of the aryl hydrocarbon receptor [44]. As a result of the altered redox status, increased nuclear translocation of NF-κB has been observed following exposure to high levels of particulate matter, as well as elevated levels of cyclooxygenase 2 and cytochrome P450, both of which may be involved in the inflammatory response triggered by PM. Particulate matter can also induce apoptotic processes in the skin, increase the formation of lentigines, and exacerbate various skin diseases. [45] All of these effects compromise the viability of surface cells, leading to visible skin deterioration.

Cigarettes

Smoking constitutes a significant risk factor for numerous diseases, particularly cancer and cardiovascular disorders. Its impact on the skin is multifaceted, involving both the direct effects of tobacco smoke and the biological influence of nicotine. Tobacco smoke contains over 4,000 chemical compounds, many of which have been identified in studies as carcinogenic, teratogenic, and mutagenic. Notably harmful components include nicotine, ammonia, hydrogen cyanide, and carbon monoxide, alongside irritants such as acrolein and formaldehyde. Moreover, potent carcinogens such as benzene and polycyclic aromatic hydrocarbons further contribute to the detrimental health effects. Understanding the intricate mechanisms through which these substances affect both systemic and dermatological health is crucial for developing effective public health interventions and therapeutic strategies.[46] Nicotine, the primary alkaloid in tobacco, contributes significantly to skin remodeling by enhancing the activity of cell cycle regulators, apoptosis-related proteins, and the synthesis of collagen types I and III, elastin, and matrix metalloproteinase-1 (MMP-1). Additionally, nicotine disrupts extracellular matrix metabolism and alters nicotinic receptor expression, thereby compromising the skin's structural integrity and contributing to increased laxity and sagging. Tobacco smoke is a potent source of free radicals, which induce oxidative stress in the skin and are widely recognized as major contributors to the acceleration of skin aging.[47-49] Furthermore, smoking depletes essential antioxidants such as vitamins C and E, which are critical for protecting the skin from oxidative damage. Vitamin C deficiency, in particular, impairs collagen synthesis and regeneration, resulting in weakened, fragile skin that is more susceptible to infection. [50] Fibroblasts are highly sensitive to the toxins in tobacco smoke [51]. Direct contact of the skin with cigarette smoke decreases moisture levels in the stratum corneum and induces a mild inflammatory response, contributing to skin dryness and the accelerated formation of wrinkles. Additionally, the repetitive contraction of the orbicularis oris muscle around the mouth results in the formation of vertical lines, commonly referred to as "smoker's wrinkles," which contribute to the visible signs of premature skin aging. Over time, this leads to thinning of the skin, reduced elasticity, and the development of deep lines and numerous fine wrinkles, particularly on the cheeks and jawline.[52] Smokers typically exhibit more pronounced signs of aging compared to non-smokers, with the risk of wrinkle formation increasing in proportion to the number of cigarette packs consumed. Women are generally more susceptible to these effects than men.[53] These mechanisms culminate in a thinner, uneven skin surface that adopts a dull, greyish hue. Long-term smoking is also linked to chronic skin hyperpigmentation, particularly in areas such as the gums and oral mucous membranes. Furthermore, smoking is associated with a higher incidence of precancerous lesions and squamous cell carcinoma, particularly on the oral mucosa and lips. It also exacerbates numerous dermatological conditions, impairs wound healing, and elevates the risk of postoperative infections compared to non-smokers[54]. Smoking

accelerates premature greying of hair and contributes to androgenetic alopecia. Reduced peripheral circulation and decreased activity of hair growth hormones compromise hair follicle integrity, exacerbating hair loss.[55]

Alcohol

Alcohol consumption has a complex impact on skin health, contributing to the development of wrinkles, puffiness, loss of facial volume, and the appearance of visible blood vessels. Reducing alcohol intake, particularly by avoiding excessive consumption, may help delay these signs of skin aging.[56] Alcohol functions as a diuretic, promoting fluid loss and depleting essential vitamins and minerals. As a result, the skin becomes dry and dull, accelerating the formation of wrinkles. Dehydrated skin also loses elasticity, making it more vulnerable to damage. [57] Concurrently, alcohol-induced dilation of peripheral vessels, including facial capillaries, can result in uneven skin tone, persistent redness, the formation of telangiectasias (vascular spider veins), and swelling under the eyes.[58] Furthermore, alcohol places a significant burden on the liver, the primary organ responsible for detoxification. Impaired liver function can lead to toxin accumulation, which damages skin cells and accelerates premature aging. [59] Chronic alcohol consumption also promotes inflammation and reduces the effectiveness of the body's antioxidant defense mechanisms. By increasing free radical production, alcohol damages skin cells, including fibroblasts, which are essential for producing collagen and elastin, key proteins in maintaining skin firmness and elasticity.[60]

Physical activity

Physical activity is an essential element of a healthy lifestyle, offering numerous benefits, including positively influencing skin aging. Regular exercise improves the function of the entire body, particularly benefiting the skin's condition. [57] By enhancing blood circulation, exercise facilitates the delivery of oxygen and essential nutrients to skin cells, supporting healthier skin. Improved circulation also aids in the removal of toxins and metabolic byproducts, contributing to skin detoxification. Additionally, physical activity stimulates the production of endogenous antioxidants, such as glutathione, which neutralize free radicals, thus protecting skin cells from oxidative damage. [61] Furthermore, exercise promotes collagen production, a crucial protein in maintaining skin firmness and elasticity. It also accelerates tissue regeneration, aiding in skin repair and helping to delay the onset of wrinkles. [57]

Diet

In the context of skin aging, diet plays a pivotal role as a source of energy and essential nutrients. Nutritional status has a direct impact on skin health, with deficiencies in key nutrients, such as vitamins and minerals, contributing to skin deterioration, reduced elasticity, and accelerated aging processes. [62]A low-glycemic diet, characterized by low intake of refined carbohydrates and processed foods, and high intake of vegetables and lean proteins, helps to maintain stable blood sugar levels. Diets high in sugars and saturated fats can promote the formation of advanced glycation end products, which are chemical compounds that damage collagen and elastin fibers in the skin, leading to increased stiffness and loss of elasticity. Elevated blood sugar levels can also exacerbate skin inflammation and contribute to the development of acne[63]. In 1989, Dr. Stephen DeFelice introduced the term "nutraceuticals" by combining the words "nutrition" and "pharmaceutical." This concept describes bioactive dietary components derived from natural food sources that are believed to confer medical or health benefits. Nutraceuticals include a diverse range of substances, such as vitamins, amino acids, polyphenols, unsaturated fatty acids, and minerals.[64] Vitamins C and E, obtained from dietary sources, are well-known for their potent antioxidant properties and their ability to neutralize free radicals. These vitamins are crucial for protecting cellular components, including tissues, membranes, and DNA, from oxidative damage. Moreover, they serve as essential cofactors in collagen hydroxylation, a process critical for the maturation of intracellular and extracellular collagen.[65] Both vitamins also exhibit photoprotective effects, increasing the threshold radiation dose required to induce skin erythema.[66] Dietary sources rich in vitamin C include raw red and green peppers, oranges, grapefruits, kiwis, broccoli, strawberries, and Brussels sprouts. In contrast, vitamin E is primarily found in high concentrations in seeds such as sunflower seeds, peanuts, almonds, walnuts, pecans, chia seeds, and sesame seeds, with smaller amounts present in certain fruits and vegetables[62]. N-acetylcysteine, an amino acid with significant influence on skin appearance, is a precursor in the synthesis of glutathione, the most abundant endogenous intracellular antioxidant. Glutathione plays a pivotal role in maintaining cellular redox balance and protecting against oxidative stress, which is a key contributor to skin aging and other dermatological conditions.[67] Plant-derived compounds, such as carotenoids and polyphenols, constitute a major class of bioactive substances with potent antioxidant properties. Polyphenols, including curcumin and epigallocatechin gallate (EGCG), are secondary metabolites predominantly found in green tea, fruits, vegetables, and cereals.[62] Furthermore, they reduce reactive oxygen species (ROS) production,

inhibit lipid peroxidation, and suppress theThese compounds exhibit a broad spectrum of biological activities, including antimutagenic, anticancer, anti-inflammatory, and antioxidant effects. Mechanistically, polyphenols inhibit cancer cell proliferation and induce apoptosis by modulating key molecular pathways, such as p53 expression, and suppressing the activity of nuclear factor kappa B [68]. release of pro-inflammatory cytokines, contributing to their protective effects on skin and overall health.[69] Carotenoids, another important group of antioxidants, are primarily found in green leafy vegetables, root vegetables, yellow and orange citrus fruits, eggs, and tomatoes. These compounds support skin health by mitigating radiation-induced and singlet oxygen-mediated lipid peroxidation. Additionally, carotenoids protect against sunburn and photoimmunosuppression, thereby preserving skin integrity and immune function. Their role as natural photoprotective agents underscores their importance in preventing photoaging and other adverse effects of UV exposure.[62] Unsaturated fatty acids are crucial components of a balanced diet, with significant roles in maintaining skin health. These fatty acids are abundant in oils such as linseed oil, rapeseed oil, and hemp seed oil, as well as in cold-water fish like salmon and trout, and various nuts and seeds. Their anti-inflammatory properties are mediated through the suppression of pro-inflammatory cytokines, including prostaglandins, leukotrienes, tumor necrosis factor-alpha (TNF-α), interferon-gamma (IFN-γ), and interleukins 12 and 6 (IL-12 and IL-6). [70] Additionally, in the stratum corneum, unsaturated fatty acids contribute to the lipid matrix that forms a protective barrier, supporting skin hydration and defense against environmental stressors.[71] Copper, an essential trace element, is found in nuts, seeds, seafood, meat, and cereals. It serves as a vital cofactor in enzymatic processes crucial for skin structure and function. For instance, copper is required for collagen cross-linking, facilitated by lysyl oxidase, which enhances the structural integrity of the dermal extracellular matrix. Furthermore, it plays a role in melanin synthesis through tyrosinase activity, influencing skin pigmentation. Copper also supports the proliferation of keratinocytes and fibroblasts, essential for tissue repair and regeneration.[72,73] Selenium, primarily sourced from animal-based proteins such as meat and seafood, is a critical micronutrient for maintaining genomic stability and cellular health. It plays a central role in DNA synthesis and repair, regulation of apoptosis, and protection against oxidative stress. Selenium's antioxidant activity is particularly beneficial in mitigating oxidative damage in the skin, which contributes to aging and photo-induced cellular damage.[74] Zinc is another indispensable mineral, predominantly found in whole grains, red meat, and seafood. The epidermis harbors the majority of the body's zinc stores, underscoring its importance in skin health. Zinc supports epidermal proliferation, keratinocyte differentiation, and wound healing.[75] It exerts anti-inflammatory effects by inhibiting the expression of intercellular adhesion molecule-1 in keratinocytes and reducing nitric oxide production, both of which are associated with inflammatory skin conditions. Additionally, zinc is a critical cofactor for enzymatic activities that protect against lipid peroxidation, UV-induced cytotoxicity, and oxidative stress, reinforcing its role in maintaining skin integrity and resilience.[74]

Photoprotection

Photoprotection constitutes a pivotal strategy in mitigating the deleterious effects of solar radiation on human skin. Historically, the primary objective of sunscreens and skincare products has been the prevention of acute damage, such as erythema (sunburn), and long-term sequelae, including photocarcinogenesis and photoaging.[76] Photoprotection methodologies are typically categorized into physical and chemical approaches, each employing distinct mechanisms to safeguard the skin against ultraviolet (UV) radiation. Physical photoprotection is achieved through mechanical barriers such as tightly woven textiles, wide-brimmed hats, and sunglasses equipped with UV filtration, all of which effectively minimize exposure to both UV and visible light. Additionally, the avoidance of direct solar exposure during periods of peak radiation intensity, typically between 10:00 AM and 4:00 PM, is strongly advised. [77] Chemical photoprotection, in contrast, relies on topical applications of sunscreens formulated with UV filters, which are classified into physical (mineral) and chemical types. Physical filters, such as titanium dioxide and zinc oxide, provide a protective barrier by reflecting and scattering incident UV radiation. Conversely, chemical filters, including compounds like avobenzone, octocrylene, and oxybenzone, absorb UV radiation, subsequently converting it into heat that is dissipated from the skin. [78] Modern sunscreen formulations often incorporate adjunctive components such as antioxidants, which neutralize free radicals generated by UV exposure, and emollients and humectants, which enhance skin hydration and barrier function. Optimal use of sunscreen necessitates its application on a daily basis, irrespective of weather conditions, as UV radiation can traverse cloud cover. It is recommended that sunscreen be applied at least 20 minutes prior to sun exposure, with reapplication within one hour or after activities such as swimming or excessive perspiration. [79] The combined use of physical and chemical photoprotection measures remains essential in mitigating UV-induced dermal damage. This underscores the imperative for continued research and public health initiatives aimed at optimizing photoprotective formulations and promoting adherence to evidence-based photoprotection practices.

Conclusion:

This study provides an in-depth analysis of the multifaceted processes driving skin aging and the significant role environmental factors play in accelerating these changes. Aging, as a natural biological process, manifests through structural and functional transformations not only in the skin but also in the underlying adipose, muscle, and bone tissues. These alterations lead to visible signs such as wrinkles, loss of elasticity, discoloration, and impaired regenerative capacity, all of which affect both physical appearance and quality of life. The findings highlight that while intrinsic factors like genetics are unavoidable, extrinsic factors including UV radiation, air pollution, smoking, alcohol consumption, and an unhealthy diet substantially influence the rate and extent of aging. Mechanisms such as oxidative stress, glycation, hormonal changes, and the degradation of the extracellular matrix are central to these processes. Key environmental influences discussed include UV radiation as a primary driver of photoaging, causing DNA damage, oxidative stress, and the breakdown of collagen and elastin, leading to premature skin aging. Air pollution and particulate matter induce oxidative stress and inflammatory responses, damaging skin integrity and exacerbating visible aging symptoms. Smoking and alcohol consumption contribute to oxidative damage, nutrient depletion, and impaired collagen synthesis, accelerating skin deterioration. Diet also plays a crucial role, as nutritional imbalances, particularly diets high in sugars, promote glycation, which stiffens collagen and elastin fibers. In contrast, antioxidant-rich diets can protect against oxidative damage and slow aging processes. Physical activity enhances circulation, promotes detoxification, and supports the production of antioxidants, contributing to healthier and more resilient skin. By understanding these mechanisms and their interplay with environmental factors, this study underscores the potential for preventive strategies that mitigate the adverse effects of extrinsic aging. Adopting a healthy lifestyle, including a balanced diet, regular physical activity, and sun protection, not only preserves skin health but also promotes overall well-being. The integration of these findings into clinical and everyday practices can empower individuals to maintain skin vitality and delay the visible and functional effects of aging. Future research should continue to explore innovative interventions and lifestyle modifications to further enhance our ability to combat the effects of environmental factors on skin aging.

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Author's contribution:

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