

BIAŁOSZYCKA, Żanna, BIAŁOSZYCKA, Monika, PACHEVSKA, Alisa, ISTOSHYN, Valerij and BIŁOSHYTSKA, Alina. Skin aging - the role of nutrition and sugar. Journal of Education, Health and Sport. 2025;80:58368 eISSN 2391-8306.
<https://doi.org/10.12775/JEHS.2025.80.58368>
<https://apcz.umk.pl/JEHS/article/view/58368>

The journal has had 40 points in Minister of Science and Higher Education of Poland parametric evaluation. Annex to the announcement of the Minister of Education and Science of 05.01.2024 No. 32318. Has a Journal's Unique Identifier: 201159. Scientific disciplines assigned: Physical culture sciences (Field of medical and health sciences); Health Sciences (Field of medical and health sciences).

Punkty Ministerialne 40 punktów. Załącznik do komunikatu Ministra Nauki i Szkolnictwa Wyższego z dnia 05.01.2024 Lp. 32318. Posiada Unikatowy Identyfikator Czasopisma: 201159. Przypisane dyscypliny naukowe: Nauki o kulturze fizycznej (Dziedzina nauk medycznych i nauk o zdrowiu); Nauki o zdrowiu (Dziedzina nauk medycznych i nauk o zdrowiu). © The Authors 2025;

This article is published with open access at Licensee Open Journal Systems of Nicolaus Copernicus University in Torun, Poland Open Access. This article is distributed under the terms of the Creative Commons Attribution Noncommercial License which permits any noncommercial use, distribution, and reproduction in any medium, provided the original author (s) and source are credited. This is an open access article licensed under the terms of the Creative Commons Attribution Non commercial license Share alike.

(<http://creativecommons.org/licenses/by-nc-sa/4.0/>) which permits unrestricted, non commercial use, distribution and reproduction in any medium, provided the work is properly cited.

The authors declare that there is no conflict of interests regarding the publication of this paper.

Received: 30.01.2025. Revised: 28.02.2025. Accepted: 22.03.2025. Published: 15.04.2025.

Skin aging - the role of nutrition and sugar

Żanna Białoszycka St. Vincent de Paul Hospital, ul. Wójta Radtkego 1, 81-348 Gdynia,
Poland

<https://orcid.org/0000-0002-3136-7687>

dr.zannabialoszycka@gmail.com

Monika Białoszycka School of Medicine, Collegium Medicum, University of Warmia and
Mazury Aleja Warszawska 30, 11-082 Olsztyn, Poland

<https://orcid.org/0009-0005-9371-0398>

monika.margaretka@gmail.com

Alisa Pachevska Department of Pediatric Stomatology of National Pirogov Memorial
University st. Pirogova 56, 21018 Vinnytsya, Ukraine

<https://orcid.org/0000-0002-6041-3814>

alisa.paczewska@gmail.com

Valerij Istoshyn Department of Biochemistry of National Pirogov Memorial University
st. Pirogova 56, 21018 Vinnytsya, Ukraine
<https://orcid.org/0000-0002-1857-3195>
walery.istoszyn@onet.pl

Alina Biloshytska Department of Medical Biology of National Pirogov Memorial
University st. Pirogova 56, 21018 Vinnytsya, Ukraine
<https://orcid.org/0000-0003-2790-060X>
alina.biloszycka@gmail.com

Corresponding author:

Żanna Białoszycka St. Vincent de Paul Hospital, ul. Wójta Radtkego 1, 81-348 Gdynia,
Poland
<https://orcid.org/0000-0002-3136-7687>
dr.zannabialoszycka@gmail.com

Abstract

Introduction

The skin is the largest organ of the human body, covering almost the entire surface. It performs numerous functions essential for life and normal functioning. The condition of the skin is influenced by various factors, including race, gender, age, and general health. Over time, the skin becomes thinner and weaker, necessitating proper care. Skin aging is a complex process involving both intrinsic and extrinsic changes. It can be classified into intrinsic and extrinsic aging, both of which are influenced by environmental factors. Among these, sun exposure is undoubtedly the most significant and primary exogenous factor, with its effects depending on intensity and duration. Additionally, glycation has emerged as one of the most studied mechanisms underlying skin aging. The accumulation of advanced glycation end products (AGEs) significantly affects skin aging, as these compounds primarily accumulate in the skin, especially in areas exposed to sunlight.

Aim of the study: This review aims to present and summarize the roles of key factors influencing skin aging, emphasizing the importance of healthy nutrition as a method to slow down the aging process.

Materials and Methods: A literature review was conducted using the PubMed database to identify relevant studies from the past twenty years. The most significant findings were compiled in this review. The literature was selected using keywords such as "skin aging," "skin and effects of sugar," and "skin and glycation."

Results

The skin, as the body's largest organ, covers almost its entire surface and fulfills numerous essential functions. Skin aging is a complex process involving intrinsic and extrinsic changes, which are most visibly noticeable on exposed areas such as the face, neck, and hands. Among extrinsic factors, sun exposure is the most significant. Clinical signs of photoaging include dryness, wrinkles, uneven pigmentation, loss of elasticity, telangiectasia, and areas with purpura. Other external factors include air pollution, cigarette smoke, dietary habits, temperature, stress, lack of sleep, physical activity, and lifestyle choices.

Recently, the importance of water in preventing nutrition-related non-communicable diseases has gained attention, as sugary and caloric beverages increasingly replace pure water in the diet. Water is essential for the proper and healthy functioning of the skin, especially its outermost layer, the stratum corneum. Proteins are vital components of all tissues and organs, playing roles in tissue formation and repair, mediating physiological functions, and providing energy. Vitamins, particularly those with antioxidant properties, are mainly obtained through diet, and their content is closely linked to the skin's physiological functions. Active metabolites of vitamin D3 and lumisterol (L3) have diverse anti-aging and photoprotective effects on the skin. Other micronutrients closely associated with skin physiology and biochemistry include iron, iodine, zinc, and copper. Diets high in fats delay skin healing, promote oxidative stress, and induce inflammatory reactions in the skin. Excess dietary fats can also lead to morphological changes and impaired matrix remodeling in the skin.

Among the mechanisms underpinning skin aging, glycation is one of the most extensively studied. Glycation is a non-enzymatic process where proteins (including collagen and elastin), lipids, and nucleic acids covalently bind with sugar molecules, typically glucose or fructose. An intriguing discovery is that AGE accumulation is not only linked to dietary sugar content but also depends on cooking methods.

Conclusion: Various exogenous factors influence the skin's condition, accelerating aging, contributing to skin diseases, and increasing carcinogenesis risk. Evidence supports the effectiveness of sunscreen use and limiting sun exposure to prevent skin aging. Furthermore, a balanced diet rich in antioxidants, with reduced free sugars and carbohydrates, can lower AGE production in the skin.

Keywords: skin aging, skin and sugar, skin and glycation, photoaging, advanced glycation end products (AGEs)

Introduction

The Skin: Structure and functions

The skin is the largest organ of the human body, covering almost the entire surface. It performs numerous functions essential for life and normal functioning. These include: protection against microorganisms (its primary role), participation in thermoregulation, production of vitamin D3 and melanin, maintenance of overall body homeostasis.

Embryologically, the epidermis and its appendages develop from the superficial ectoderm, while the dermis and subcutaneous tissue originate from the mesoderm. The skin consists of three layers: the dermis, subcutaneous tissue, and epidermis. It also houses sweat and sebaceous glands, a rich network of blood and lymphatic vessels, and nerve endings. The skin on the scalp and torso differs from the epidermis on the hands and feet, as the latter lacks subcutaneous tissue, hair, and glands, making it thicker.

The Dermis

The dermis is the middle layer, with a thickness ranging from 1 to 3 mm. It is durable and elastic, containing sweat glands, blood vessels, receptors, hair roots, and nerve endings that enable the perception of external stimuli like temperature, pain, and pressure. Its structure is dominated by fibrous connective tissue and includes mast cells and various types of fibers. Subcutaneous tissue (hypodermis)

The hypodermis plays a critical role in thermoregulation and serves as a reserve energy source. It consists of adipose tissue, which contains fat cells, receptors, glands, and the lower portions of hair roots, along with connective tissue. It also includes blood vessels, nerves, and fibers. Although not part of the skin, it is closely associated with it. The thickness of the hypodermis is influenced by anatomical and individual characteristics, reflecting a person's nutritional status.

The Epidermis

The epidermis, the outermost skin layer, serves a protective function. It provides the skin and hair with individual color due to the presence of melanin pigment.

Anatomically, the epidermis includes five layers of epidermal cells: basal, spinous, granular, lucid, and stratum corneum.

The skin is considered one of the most important organs due to its numerous vital functions:

- 1. Protection: Acts as a barrier between the body and external factors, including chemicals, microorganisms, burns, and injuries.**
- 2. Melanin production: Responsible for skin and hair pigmentation.**
- 3. Vitamin D3 synthesis.**
- 4. Allergen recognition and classification.**
- 5. Defense against pathogenic microorganisms.**
- 6. Thermoregulation.**
- 7. Perception of external stimuli.**
- 8. Increased absorption of certain substances.**
- 9. Regulation of water-electrolyte, fat, and vitamin metabolism.**

The skin's condition is determined by various factors, including race, gender, age, and overall health. Over time, the skin becomes thinner and weaker, requiring proper care [1].

Skin Aging

Skin aging is a complex process involving intrinsic and extrinsic changes, often most visible in exposed areas such as the face, neck, and hands. It manifests as skin laxity, deeper wrinkles, uneven pigmentation, age spots, and noticeable vascular changes that impair thermoregulation and nutrient delivery.

Chronological (intrinsic) aging is an inevitable process influenced by factors such as time, genetics, and hormones. Oxidative processes also play a significant role, linked to reduced antioxidant capacity and increased production of reactive oxygen species. These can damage lipids, proteins, nucleic acids, and organelles, leading to cellular aging—a key mechanism of skin aging [2].

Mechanisms of skin aging

Sun exposure is the most significant exogenous factor in skin aging. Clinical signs of photoaging include dryness, wrinkles, uneven pigmentation, loss of elasticity, telangiectasia, and areas of purpura. UV radiation stimulates fibroblasts to release melanogenic growth factors such as hepatocyte growth factor (HGF), keratinocyte growth factor (KGF), and stem cell factor (SCF). These factors directly and indirectly act on melanocytes through keratinocytes, contributing to hyperpigmentation seen in solar lentigines [3].

The impact of photoaging depends on UV wavelength, categorized as UVA, UVB, and UVC. UVA: accelerates collagen hydrolysis, promotes the production of matrix metalloproteinases (MMPs), which leads to tissue destruction and degeneration of the thermal extracellular matrix [6, 7]. UVA inhibits the synthesis of hyaluronic acid and therefore changes the composition of skin proteoglycans [4]. UVB: affects mainly keratinocytes in the epithelial layer of the skin, inducing DNA damage and mutations in keratinocytes, release of cytokines from keratinocytes, and leads cells to aging, inflammation, apoptosis and carcinogenesis [7, 8].

The following is a classification of photodamage developed by Dr Richard Glogau. This scale is used by dermatologists and plastic surgeons to determine skin type and choose therapy (Table 1). [9]

<https://sfderm.com/glogau-wrinkle-scale/>

Table. 1 - Glogau's photoaging classification

Type 1 - No wrinkles Patient age: 20s to 30s	Early photo-aging Mild pigment changes Minimal wrinkles No "age spots"
Type 2 - Wrinkles in motion Patient age: 30s to 40s	Early to moderate photo-aging Appearance of lines only when face moves Early brown "age spots" Skin pores more prominent Early changes in skin texture
Type 3 – Wrinkles in rest Patient age: 50s and older	Advanced photo-aging Prominent brown pigmentation Visible brown "age spots" Prominent, small blood vessels Wrinkles now present with face at rest
Type 4 – Only wrinkles Patient age: 60s or 70s	Severe photo-aging Wrinkles everywhere, at rest or moving Yellow-gray skin color Prior skin cancers Precancerous skin changes (actinic keratosis)

Other external factors contributing to skin aging include polluted air, cigarette smoke, dietary habits, temperature, stress, lack of sleep, physical activity, and overall lifestyle. External aging depends on the intensity and duration of environmental exposure as well as the type of skin.

The epidermis, dermo-epidermal junction, and dermis become thinner over time. The dermis becomes atrophic, with a reduction in the number of fibroblasts [10]. A decrease in the surface area of the dermo-epidermal junction leads to increased skin fragility, fewer cells, and reduced nutrient exchange between the dermis and epidermis, ultimately causing wrinkles. Slower cellular turnover in the epidermis contributes to delayed wound healing, inefficient desquamation, and reduced barrier function.

Photoaging is histologically characterized by elastosis, the accumulation of elastin material below the dermo-epidermal junction. Epidermal atrophy and fragmentation of collagen and elastic fibers are also consequences of photoaging [11]. Aging affects collagen

by reducing the quantity and diameter of collagen fiber bundles, impairing type I collagen synthesis, and increasing the proportion of type III collagen [12]. Chronic sun exposure and aging also decrease the number of Langerhans cells, which have antigen-presenting properties. This may facilitate the development of skin carcinoma in elderly individuals with sun-damaged skin [13].

Telomeres, located at the ends of chromosomes, are essential for maintaining chromosome integrity and controlling the cell cycle. Epithelial stem cells exhibit low proliferative capacity [14]. UV radiation damages cellular DNA in the skin, accelerating telomere shortening and contributing to intrinsic cellular aging [15].

With age, melanocyte density decreases by approximately 6–8% per decade in sun-exposed skin compared to non-exposed skin. This explains the general increase in pigmentation observed during aging [16].

The accumulation of AGEs, which result from excess sugar binding to proteins, significantly impacts skin aging. AGEs are products that are formed after the combination of excess sugar and protein formed as a result of synthesis in the body and food consumption. AGEs accumulate in the skin, particularly in sun-exposed areas, affecting protein function in the dermis and accelerating skin aging [17, 18].

Chronic UV radiation causes oxidative stress in cells, leading to damage, lipid oxidation, and ultimately inflammation. When the extent of inflammation surpasses macrophages' clearing capacity, they begin secreting pro-inflammatory factors and reactive oxygen species (ROS), exacerbating cellular damage [19, 20].

The only proven ways to prevent photoaging are avoiding sun exposure and using sunscreens. Retinoids are effective in suppressing collagenase synthesis and promoting collagen production. Antioxidants, such as vitamins E and C, coenzyme Q10, glutathione, alpha-lipoic acid, and others, help neutralize free radicals, reducing signs of aging. Other methods, including stem cell transplantation, hormone therapy, and telomerase modification, have been explored in medical research. However, some of these approaches come with significant drawbacks. For instance, hormone therapy increases the risk of breast cancer. Therefore, the most acceptable strategy for improving skin health is adhering to a balanced diet and engaging in moderate physical activity [20].

The impact of nutrition on skin health

Nutrition forms the foundation of human vitality, providing essential nutrients for growth, proper functioning, and energy. In developed Western countries, vitamin, microelement, and protein deficiencies are rare. However, unbalanced or inadequate nutrition can lead to diseases, aging, and adverse effects on skin health [21].

Role of specific nutritional elements

Water is essential for maintaining tissue function and balance. It acts as a nutrient, solvent, transporter, body volume maintainer, and regulator of body temperature. Its role in preventing diet-related non-communicable diseases has gained attention, as populations increasingly replace water with sugary, calorie-rich beverages. Adults can regulate their water balance more effectively than young children and the elderly, who are at higher risk of dehydration [22, 23].

Proper hydration is crucial for healthy skin function, particularly the outermost layer, the stratum corneum. Water influences its maturation and desquamation. Increased transepidermal water loss disrupts enzymatic functions necessary for normal skin shedding, leading to dry, flaky skin [24]. Restoring water balance is a key step toward maintaining healthy skin.

Proteins are vital components of all tissues and organs. They build and repair tissues, mediate physiological functions, and supply energy. Adequate protein intake supports tissue renewal, including the skin. Protein deficiency or excess can result in metabolic disorders and impact physical health [25]. Studies show that sufficient protein intake aids in healing pressure ulcers in rats, whereas protein deficiency or excess hinders wound healing. Dietary protein supplements enhance cellular protein synthesis and metabolism [26].

Hypovitaminosis. Skin-related antioxidant vitamins primarily come from food, making dietary vitamin content closely linked to skin's physiological functions.

Active metabolites of vitamin D3 (D3) and lumisterol (L3) exhibit anti-aging and photoprotective effects. These include anti-inflammatory properties, keratinocyte proliferation regulation, differentiation programs for forming the epidermal barrier, and antioxidant responses. They suppress DNA damage, induce repair mechanisms, and mitigate premature aging and carcinogenesis [27]. Vitamin D deficiency negates these protective effects. Vitamin C deficiency manifests as scurvy, tender skin, and impaired wound healing [28].

Other micronutrients that are closely related to the skin and its physiological and biochemical processes are: iron, iodine, zinc, copper [29]. They are associated with skin immunity, inflammatory processes, and the homeostasis of some of these trace elements can play an important role in the treatment of skin diseases, such as psoriasis [30].

Copper supports extracellular matrix formation, protein stabilization, and angiogenesis. Studies show copper improves skin elasticity, reduces fine lines and wrinkles, and enhances wound healing [31].

Iron acts as a biooxidation catalyst. Studies have shown that the skin is a cellular store of iron; under the influence of UV radiation, the iron content in the skin cells of postmenopausal women increases rapidly, reducing the antioxidant capacity and leading to accelerated skin aging [32, 33].

Selenium deficiency in the diet reduces the antioxidant function of the skin of mice exposed to UV radiation, and this leads to skin sensitivity, greater vulnerability to oxidative stress [34]. Selenoproteins, proteins containing selenium in the form of the amino acid selenocysteine, have also been found to be important antioxidants in the skin and play a role in the growth and viability of keratinocytes [35].

Diets high in fat slow skin healing, exacerbate oxidative stress, and trigger inflammatory reactions. Excessive dietary fats can also cause skin morphology changes and disrupt matrix remodeling [36].

So, first of all, a balanced diet, correction of deficiencies, and maintenance of the body's water balance are the first steps towards healthy skin and slowing down its aging.

Sugar, glycation and their effects on skin condition

Due to a lack of a balanced diet, poor eating habits, lack of sleep, stress and other individual factors, people feel a constant urge to eat something sweet. There are two reasons why this happens. The first one is psychological: sweets reward us and give us a quick brain satisfaction, which is what we really want during times of stress. The second is that the sensation of sweetness changes with its consumption: if you consume more, you will feel less of a taste and then you will want more stimulus. [37].

Among the many mechanisms believed to underlie the aging process, glycation has become one of the most studied processes. Glycation is a non-enzymatic reaction of proteins, including collagen and elastin, lipids and nucleic acids, which covalently bind to sugar molecules, usually glucose or fructose. Thus, after binding, proteins and lipids lose their

functionality, and this process is called ‘sweet old age’. The lack of enzymatic mediation is the key difference between glycation and glycosylation. Glycation occurs at random molecular sites and usually results in the inhibition of the target molecule's ability to function. The products that result from glycation are called advanced glycation end products (AGEs) [38].

It has been found that dermal glycation usually occurs after the age of 35 and then rapidly increases with intrinsic aging. An increase in glycation has been observed under the influence of solar radiation [39]. If you abuse the amount of simple sugars and carbohydrates, consuming them in large quantities or too often, or the body cannot cope with too high glycaemic peaks, protein glycation begins.

AGEs are pro-inflammatory molecules and are involved in the development of many pathologies, including diabetic complications, atherosclerosis, end-stage renal disease, and chronic obstructive pulmonary disease [40]. Glycation is more common in old skin and leads to structural, morphological and functional changes in the skin (Fig. 1). [41]

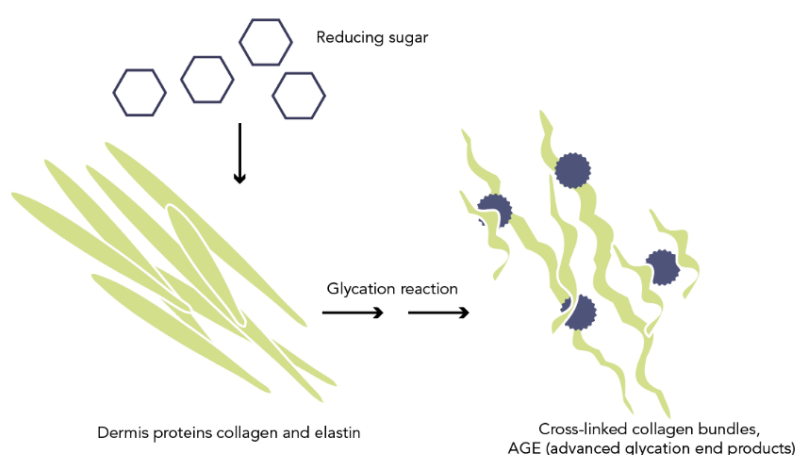


Fig 1. Glycation process <https://mibellebiochemistry.com/not-so-sweet-process-glycation>

AGEs comprise a heterogeneous group of molecules, the first and most famous AGE described was glycated haemoglobin (haemoglobin A1C), which is now widely used in the diagnosis of diabetes and glycaemic control. However, the most common AGE in the human body is carboxymethyllysine (CML). In the skin, CML is found in normal epidermis, old and diabetic dermis, and it has been shown that diabetes is characterised by accelerated chemical aging of long-lived tissue proteins and photoaging with actinic elastosis [42, 43, 44]. Glycation affects the extracellular matrix proteins elastin and fibronectin, which further

exacerbates dermal dysfunction, as cross-linked collagen, elastin, and fibronectin cannot be restored like their primary analogues [45,46].

Other AGEs that have been found in the skin include pentosidine, glyoxal, methylglyoxal, glucosepane, fructoselysine, carboxyethyl-lysine, glyoxal-lysine dimer, and methylglyoxal-lysine dimer. [47].

More than half a century ago, it was discovered that blood and skin sugar levels decrease with a low-sugar diet. It is now clear that food, as a source of monosaccharides, which in large quantities affect increased production of AGEs (Advanced Glycation End Products) in the body [48, 49].

An interesting discovery was that AGE consumption is not only linked to the sugar content in food but also to cooking methods. Grilling, frying, and deep-frying significantly increase the AGE levels in food. Such cooking methods often enhance flavor and impart an appealing brown color to the food. Effective strategies to limit AGE formation include avoiding foods like donuts, fried meats, and dark-colored soft drinks. Cooking methods involving water, such as boiling and steaming, result in lower AGE production [50]. A low-AGE diet correlates with reduced biomarkers of inflammation (TNF- α , IL-6, CRP) in diabetic patients, improved wound healing, and other benefits observed in diabetic mice [51]. Interestingly, the relatively youthful appearance of elderly Asian populations has been linked to their characteristic water-based cooking methods, which are common in Asian cuisine.

Another noteworthy finding is that certain culinary herbs and spices can suppress endogenous AGE production. These include cinnamon, cloves, oregano, and allspice. Other dietary compounds shown to inhibit AGE formation in in vitro studies and animal models include ginger, garlic, α -lipoic acid, carnitine, taurine, carnosine, flavonoids, benfotiamine, α -tocopherol, niacinamide, pyridoxal, sodium selenite, selenium-enriched yeast, riboflavin, zinc, and manganese [50-52].

Since active oxygen species contribute to glycation, antioxidants are thought to be effective in limiting new AGE production. In one human study, AGE-targeted therapy using L-carnitine over six months successfully reduced skin AGE levels in patients undergoing hemodialysis [53].

Conclusions

Many exogenous factors affect skin health, accelerating aging, causing skin diseases, and even contributing to carcinogenesis. Among these, solar radiation and diet are the most

significant, as reviewed in this article. The effectiveness of sun protection using sunscreen and limiting sun exposure is well-established as a foundation for daily routines, particularly during summer. This not only prevents skin aging but also reduces the risk of skin cancer. Maintaining a balanced diet with sufficient antioxidants and minimizing free sugars and carbohydrates is crucial to reducing AGE production in the skin.

In summary, the most effective strategy to slow the overall aging process of the skin is a balanced diet and limiting UV exposure to counteract AGEs accumulation in the human body.

Author's contribution:

Conceptualization Żanna Białoszycka, Monika Białoszycka, methodology Żanna Białoszycka, Alisa Pachevska, Alina Biloshytska, software Monika Białoszycka, Valerij Istoshyn, check Żanna Białoszycka, Alisa Pachevska, Alina Biloshytska, formal analysis Monika Białoszycka, Valerij Istoshyn, investigation Alisa Pachevska, Valerij Istoshyn, Alina Biloshytska, resources Żanna Białoszycka, Monika Białoszycka, data curation Żanna Białoszycka, Alisa Pachevska, Valerij Istoshyn, writing-rough preparation Monika Białoszycka, Alina Biloshytska, writing review and editing Alisa Pachevska, Alina Biloshytska, visualization Żanna Białoszycka, Valerij Istoshyn, supervision Alisa Pachevska, Valerij Istoshyn, Alina Biloshytska, project administration Alisa Pachevska, Valerij Istoshyn, Alina Biloshytska.

The authors have read and agreed with the published version of the manuscript.

Funding Statement

The Study Did Not Receive Special Funding.

Institutional Review Board Statement

Not Applicable.

Informed Consent Statement

Not Applicable.

Data Availability Statement

Not Applicable.

Acknowledgments

Not applicable.

Conflict Of Interest:

The authors declare no conflict of interest.

References

1. Kovacs D, Cardinali G, Aspite N, et al. Role of fibroblast-derived growth factors in regulating hyperpigmentation of solar lentigo. *Br J Dermatol*. 2010;163(5):1020-1027. doi:10.1111/j.1365-2133.2010.09946.x
<https://doi.org/10.1111/j.1365-2133.2010.09946.x>
2. Bernerd F, Asselineau D. An organotypic model of skin to study photodamage and photoprotection in vitro. *J Am Acad Dermatol*. 2008;58(5 Suppl 2):S155-S159. doi:10.1016/j.jaad.2007.08.050
[https://www.jaad.org/article/S0190-9622\(07\)02414-0/abstract](https://www.jaad.org/article/S0190-9622(07)02414-0/abstract)
3. Imokawa G, Ishida K. Biological mechanisms underlying the ultraviolet radiation-induced formation of skin wrinkling and sagging I: reduced skin elasticity, highly associated with enhanced dermal elastase activity, triggers wrinkling and sagging. *Int J Mol Sci*. 2015;16(4):7753-7775. Published 2015 Apr 8. doi:10.3390/ijms16047753
[https://www.jaad.org/article/S0190-9622\(07\)02414-0/abstract](https://www.jaad.org/article/S0190-9622(07)02414-0/abstract)
4. Tigges J, Krutmann J, Fritsche E, et al. The hallmarks of fibroblast ageing. *Mech Ageing Dev*. 2014;138:26-44. doi:10.1016/j.mad.2014.03.004
<https://doi.org/10.1016/j.mad.2014.03.004>
5. Bernerd F, Marionnet C, Duval C. Solar ultraviolet radiation induces biological alterations in human skin in vitro: relevance of a well-balanced UVA/UVB protection. *Indian J Dermatol Venereol Leprol*. 2012;78 Suppl 1:S15-S23. doi:10.4103/0378-6323.97351
<https://ijdv1.com/solar-ultraviolet-radiation-induces-biological-alterations-in-human-skin-in-vitro-relevance-of-a-well-balanced-uva-uvb-protection/>
6. Fagot D, Asselineau D, Bernerd F. Matrix metalloproteinase-1 production observed after solar-simulated radiation exposure is assumed by dermal fibroblasts but involves a paracrine activation through epidermal keratinocytes. *Photochem Photobiol*. 2004;79(6):499-505. doi:10.1562/yg-03-11-r1.1

<https://doi.org/10.1562/YG-03-11-R1.1>

7. Khavkin J, Ellis DA. Aging skin: histology, physiology, and pathology. *Facial Plast Surg Clin North Am.* 2011;19(2):229-234. doi:10.1016/j.fsc.2011.04.003

<https://doi.org/10.1016/j.fsc.2011.04.003>

8. Baumann L. Skin ageing and its treatment. *J Pathol.* 2007;211(2):241-251. doi:10.1002/path.2098

<https://doi.org/10.1002/path.2098>

9. Lovell CR, Smolenski KA, Duance VC, Light ND, Young S, Dyson M. Type I and III collagen content and fibre distribution in normal human skin during ageing. *Br J Dermatol.* 1987;117(4):419-428. doi:10.1111/j.1365-2133.1987.tb04921.x

<https://doi.org/10.1111/j.1365-2133.1987.tb04921.x>

10. Thiers BH, Maize JC, Spicer SS, Cantor AB. The effect of aging and chronic sun exposure on human Langerhans cell populations. *J Invest Dermatol.* 1984;82(3):223-226. doi:10.1111/1523-1747.ep12260055

[https://www.jidonline.org/article/S0022-202X\(15\)43327-5/pdf](https://www.jidonline.org/article/S0022-202X(15)43327-5/pdf)

11. Cao C, Xiao Z, Wu Y, Ge C. Diet and Skin Aging-From the Perspective of Food Nutrition. *Nutrients.* 2020;12(3):870. Published 2020 Mar 24. doi:10.3390/nu12030870

<https://doi.org/10.3390/nu12030870>

12. Kosmadaki MG, Gilchrest BA. The role of telomeres in skin aging/photoaging. *Micron.* 2004;35(3):155-159. doi:10.1016/j.micron.2003.11.002

<https://doi.org/10.1016/j.micron.2003.11.002>

13. Gilchrest BA, Blog FB, Szabo G. Effects of aging and chronic sun exposure on melanocytes in human skin. *J Invest Dermatol.* 1979;73(2):141-143. doi:10.1111/1523-1747.ep12581580

[https://www.jidonline.org/article/S0022-202X\(15\)45561-7/pdf](https://www.jidonline.org/article/S0022-202X(15)45561-7/pdf)

14. Farrar MD. Advanced glycation end products in skin ageing and photoageing: what are the implications for epidermal function?. *Exp Dermatol.* 2016;25(12):947-948. doi:10.1111/exd.13076

<https://doi.org/10.1111/exd.13076>

15. Radjei S, Gareil M, Moreau M, et al. The glyoxalase enzymes are differentially localized in epidermis and regulated during ageing and photoageing. *Exp Dermatol.* 2016;25(6):492-494. doi:10.1111/exd.12995

<https://doi.org/10.1111/exd.12995>

16. Handoko HY, Rodero MP, Boyle GM, et al. UVB-induced melanocyte proliferation in neonatal mice driven by CCR2-independent recruitment of Ly6c(low)MHCII(hi) macrophages. *J Invest Dermatol*. 2013;133(7):1803-1812. doi:10.1038/jid.2013.9
[https://www.jidonline.org/article/S0022-202X\(15\)36341-7/fulltext](https://www.jidonline.org/article/S0022-202X(15)36341-7/fulltext)
17. Zhuang Y, Lyga J. Inflammaging in skin and other tissues - the roles of complement system and macrophage. *Inflamm Allergy Drug Targets*. 2014;13(3):153-161. doi:10.2174/1871528113666140522112003
<https://www.eurekaselect.com/article/60630>
18. Popkin BM, D'Anci KE, Rosenberg IH. Water, hydration, and health. *Nutr Rev*. 2010;68(8):439-458. doi:10.1111/j.1753-4887.2010.00304.x
<https://doi.org/10.1111/j.1753-4887.2010.00304.x>
19. Jéquier E, Constant F. Water as an essential nutrient: the physiological basis of hydration. *Eur J Clin Nutr*. 2010;64(2):115-123. doi:10.1038/ejcn.2009.111
<https://www.nature.com/articles/ejcn2009111>
20. Verdier-Sévrain S, Bonté F. Skin hydration: a review on its molecular mechanisms. *J Cosmet Dermatol*. 2007;6(2):75-82. doi:10.1111/j.1473-2165.2007.00300.x
<https://doi.org/10.1111/j.1473-2165.2007.00300.x>
21. Bellizzi V, Calella P, Carrero JJ, Fouque D. Very low-protein diet to postpone renal failure: Pathophysiology and clinical applications in chronic kidney disease. *Chronic Dis Transl Med*. 2018;4(1):45-50. Published 2018 Mar 8. doi:10.1016/j.cdtm.2018.01.003
<https://doi.org/10.1016/j.cdtm.2018.01.003>
22. Strasser B, Volaklis K, Fuchs D, Burtscher M. Role of Dietary Protein and Muscular Fitness on Longevity and Aging. *Aging Dis*. 2018;9(1):119-132. Published 2018 Feb 1. doi:10.14336/AD.2017.0202
<https://www.aginganddisease.org/EN/10.14336/AD.2017.0202>
23. Bocheva G, Slominski RM, Slominski AT. The Impact of Vitamin D on Skin Aging. *Int J Mol Sci*. 2021;22(16):9097. Published 2021 Aug 23. doi:10.3390/ijms22169097
<https://doi.org/10.3390/ijms22169097>
24. Ellinger S, Stehle P. Efficacy of vitamin supplementation in situations with wound healing disorders: results from clinical intervention studies. *Curr Opin Clin Nutr Metab Care*. 2009;12(6):588-595. doi:10.1097/MCO.0b013e328331a5b5
https://journals.lww.com/clinicalnutrition/abstract/2009/11000/efficacy_of_vitamin_supplementation_in_situations.6.aspx

25. Mehri A. Trace Elements in Human Nutrition (II) - An Update. *Int J Prev Med*. 2020;11:2. Published 2020 Jan 3. doi:10.4103/ijpvm.IJPVM_48_19
https://journals.lww.com/ijom/fulltext/2020/11000/trace_elements_in_human_nutrition_ii_an_update.2.aspx
26. Chen W, Zhou X, Zhu W. Trace Elements Homeostatic Imbalance in Psoriasis: a Meta-Analysis. *Biol Trace Elem Res*. 2019;191(2):313-322. doi:10.1007/s12011-018-1626-1
<https://link.springer.com/article/10.1007/s12011-018-1626-1>
27. Borkow G. Using Copper to Improve the Well-Being of the Skin. *Curr Chem Biol*. 2014;8(2):89-102. doi:10.2174/2212796809666150227223857
<https://www.eurekaselect.com/article/65514>
28. Reelfs O, Eggleston IM, Pourzand C. Skin protection against UVA-induced iron damage by multiantioxidants and iron chelating drugs/prodrugs. *Curr Drug Metab*. 2010;11(3):242-249. doi:10.2174/138920010791196265
<https://www.eurekaselect.com/article/15751>
29. Pelle E, Jian J, Zhang Q, et al. Menopause increases the iron storage protein ferritin in skin. *J Cosmet Sci*. 2013;64(3):175-179.
<https://pubmed.ncbi.nlm.nih.gov/23752032/>
30. Zhu X, Jiang M, Song E, Jiang X, Song Y. Selenium deficiency sensitizes the skin for UVB-induced oxidative damage and inflammation which involved the activation of p38 MAPK signaling. *Food Chem Toxicol*. 2015;75:139-145. doi:10.1016/j.fct.2014.11.017
<https://doi.org/10.1016/j.fct.2014.11.017>
31. Sengupta A, Lichti UF, Carlson BA, et al. Selenoproteins are essential for proper keratinocyte function and skin development. *PLoS One*. 2010;5(8):e12249. Published 2010 Aug 18. doi:10.1371/journal.pone.0012249
<https://doi.org/10.1371/journal.pone.0012249>
32. Rosa DF, Sarandy MM, Novaes RD, da Matta SLP, Gonçalves RV. Effect of a high-fat diet and alcohol on cutaneous repair: A systematic review of murine experimental models. *PLoS One*. 2017;12(5):e0176240. Published 2017 May 11. doi:10.1371/journal.pone.0176240
<https://doi.org/10.1371/journal.pone.0176240>
33. Nguyen HP, Katta R. Sugar Sag: Glycation and the Role of Diet in Aging Skin. *Skin Therapy Lett*. 2015;20(6):1-5.

<https://pubmed.ncbi.nlm.nih.gov/27224842/>

34. Jeanmaire C, Danoux L, Pauly G. Glycation during human dermal intrinsic and actinic ageing: an in vivo and in vitro model study. *Br J Dermatol*. 2001;145(1):10-18. doi:10.1046/j.1365-2133.2001.04275.x

<https://doi.org/10.1046/j.1365-2133.2001.04275.x>

35. Van Puyvelde K, Mets T, Njemini R, Beyer I, Bautmans I. Effect of advanced glycation end product intake on inflammation and aging: a systematic review. *Nutr Rev*. 2014;72(10):638-650. doi:10.1111/nure.12141

<https://doi.org/10.1111/nure.12141>

36. Kawabata K, Yoshikawa H, Saruwatari K, et al. The presence of N(ε)-(Carboxymethyl) lysine in the human epidermis. *Biochim Biophys Acta*. 2011;1814(10):1246-1252. doi:10.1016/j.bbapap.2011.06.006

<https://doi.org/10.1016/j.bbapap.2011.06.006>

37. Dyer DG, Dunn JA, Thorpe SR, et al. Accumulation of Maillard reaction products in skin collagen in diabetes and aging. *J Clin Invest*. 1993;91(6):2463-2469. doi:10.1172/JCI116481

<https://www.jci.org/articles/view/116481>

38. Gkogkolou P, Böhm M. Advanced glycation end products: Key players in skin aging?. *Dermatoendocrinol*. 2012;4(3):259-270. doi:10.4161/derm.22028

<https://doi.org/10.4161/derm.22028>

39. Nowotny K, Grune T. Degradation of oxidized and glycoxidized collagen: role of collagen cross-linking. *Arch Biochem Biophys*. 2014;542:56-64. doi:10.1016/j.abb.2013.12.007

<https://doi.org/10.1016/j.abb.2013.12.007>

40. URBACH E, LENTZ JW. Carbohydrate metabolism and the skin. *Arch Derm Syphilol*. 1945;52:301-316. doi:10.1001/archderm.1945.01510290006001

<https://jamanetwork.com/journals/jamadermatology/article-abstract/521127>

41. Danby FW. Nutrition and aging skin: sugar and glycation. *Clin Dermatol*. 2010;28(4):409-411. doi:10.1016/j.clindermatol.2010.03.018

<https://doi.org/10.1016/j.clindermatol.2010.03.018>

42. O'Brien J, Morrissey PA. Nutritional and toxicological aspects of the Maillard browning reaction in foods. *Crit Rev Food Sci Nutr*. 1989;28(3):211-248. doi:10.1080/10408398909527499

<https://doi.org/10.1080/10408398909527499>

43. Vlassara H, Striker GE. AGE restriction in diabetes mellitus: a paradigm shift. *Nat Rev Endocrinol*. 2011;7(9):526-539. Published 2011 May 24. doi:10.1038/nrendo.2011.74
<https://www.nature.com/articles/nrendo.2011.74>
44. Dearlove RP, Greenspan P, Hartle DK, Swanson RB, Hargrove JL. Inhibition of protein glycation by extracts of culinary herbs and spices. *J Med Food*. 2008;11(2):275-281. doi:10.1089/jmf.2007.536
<https://doi.org/10.1089/jmf.2007.536>
45. Tarwadi KV, Agte VV. Effect of micronutrients on methylglyoxal-mediated in vitro glycation of albumin. *Biol Trace Elem Res*. 2011;143(2):717-725. doi:10.1007/s12011-010-8915-7
<https://link.springer.com/article/10.1007/s12011-010-8915-7>
- (50)46. Fukami K, Yamagishi S, Sakai K, et al. Potential inhibitory effects of L-carnitine supplementation on tissue advanced glycation end products in patients with hemodialysis. *Rejuvenation Res*. 2013;16(6):460-466. doi:10.1089/rej.2013.1459
<https://doi.org/10.1089/rej.2013.1459>
47. Peppas M, Brem H, Ehrlich P, et al. Adverse effects of dietary glycotoxins on wound healing in genetically diabetic mice. *Diabetes*. 2003;52(11):2805-2813. doi:10.2337/diabetes.52.11.2805
<https://doi.org/10.2337/diabetes.52.11.2805>
- (tabl 1) 48. <https://sfderm.com/glogau-wrinkle-scale/>
- (tabl 2) 49. <https://mibellebiochemistry.com/not-so-sweet-process-glycation>