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The Impact of Excessive Sugar Consumption on Skin Health: Analysis of Biological Mechanisms and Dermatological Effects

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Abstract

Introduction and purpose:

Excessive sugar consumption is one of the main dietary problem of the modern Western diet. Numerous studies have shown that a high intake of simple sugars is associated with an increased risk of developing various diseases such as obesity, type 2 diabetes, metabolic syndrome, and cardiovascular diseases. Moreover, the consumption of large amounts of sugar also affects skin condition, accelerating its aging and exacerbating the course of certain dermatological conditions. This study aims to present the underlying mechanisms of these processes and demonstrate the impact of a low-glycemic index diet on skin health.

Description of the state of knowledge:

The skin is the largest organ of the body, and its condition is closely linked to diet. Excessive sugar consumption can contribute to biochemical changes such as skin protein glycation, increased production of reactive oxygen species (ROS), and inflammation. The glycation process leads to damage of collagen and elastin fibers, which reduces skin elasticity and contributes to the formation of wrinkles. Elevated levels of MMP-1/MMP-2 and LOX in the skin can affect collagen fragmentation and disrupt its structural integrity. Studies have also shown that a high-glycemic index diet can exacerbate inflammatory conditions such as acne and psoriasis and promote hormonal imbalances.

Conclusion:

In summary, excessive consumption of simple sugars significantly affects skin condition through mechanisms such as glycation, inflammation induction, and hormonal disturbances. Therefore, a low-glycemic index diet may be an effective tool in the prevention and treatment of skin diseases. Future studies should focus on gaining a more precise understanding of these relationships and developing dietary recommendations to improve skin health.

Key words: "skin health," "protein glycation," "skin aging," "glucose and skin," "healthy diet"

INTRODUCTION AND OBJECTIVE

It is widely recognized that high sugar consumption is a characteristic feature of the Western diet, with the intake of sugar-sweetened beverages and processed foods increasing significantly in recent years [1,2]. Increasing evidence suggests that excessive consumption of processed foods containing simple sugars is closely linked to the development of obesity [3,4], type 2 diabetes [5,6], metabolic syndrome [7], and cardiovascular diseases [8]. The rise in sugar consumption also has a significant impact on the skin, accelerating its aging process and contributing to the exacerbation of numerous skin conditions [2,9,10]. The escalation of sugar intake in recent years has led to numerous studies indicating a causal relationship between a diet high in simple sugars and skin condition. Therefore, due to the relevance and prevalence of the issue, this study aims, based on a review of available literature, to outline the mechanisms underlying this process and to demonstrate the significance of a low-glycemic index diet in the prevention of skin diseases.

Materials and Methods

A literature review was conducted using medical databases such as PubMed and Google Scholar. Articles were retrieved in English using the following keywords: "skin health," "protein glycation," "skin aging," "glucose and skin," and "healthy diet" in appropriate combinations.

DESCRIPTION OF THE STATE OF KNOWLEDGE

The skin is the largest organ of the human body, and diet has a substantial impact on its condition. Excessive sugar consumption can lead to skin dysfunction, accelerated aging, and the exacerbation of many skin conditions through various mechanisms, including biochemical processes associated with protein glycation, inflammation, and hormonal disturbances.

With age, the skin undergoes aging processes, causing thinning of the epidermis, dermis, and extracellular matrix, leading to increased fragility, reduced collagen and elastin content, and resulting in the formation of fine wrinkles. Clear vascular changes disrupting thermoregulation and nutrient delivery have also been observed.[11] Skin aging is a complex and not fully understood process resulting from numerous biological, biochemical, and

physical interactions. Both endogenous (genetic) and exogenous factors (UV radiation, pollution, lifestyle) contribute to this process.[12] Diet significantly affects the health and appearance of the skin, and the social obsession with maintaining a youthful appearance has led to numerous studies exploring this impact. Researchers, based on data from the first National Health and Nutrition Examination Survey, which examined the correlation between nutrient intake and skin aging in 4,025 women (aged 40-74), discovered that lower fat and carbohydrate intake was associated with better skin appearance, while higher fat and carbohydrate intake was linked to an increased likelihood of wrinkles and skin atrophy, regardless of age, race, education, sun exposure, income, menopausal status, body mass index, supplement use, physical activity, and energy intake.[13]

In a study using skin samples from people with type 2 diabetes, elevated levels of MMP-1/MMP-2 and LOX were observed in the dermis, which may contribute to increased collagen fragmentation, cross-linking, and consequently, changes in the mechanical properties of the dermis. The accumulation of fragmented and cross-linked collagen fragments over the years may thus contribute to skin aging in diabetic patients. Furthermore, researchers' data indicate that impaired structural integrity and abnormal collagen microenvironment could have a significant impact on the development of skin diseases in diabetic patients.[14]

In another study involving mice, researchers observed that the skin of mice on a high-sugar diet tended to have a reddish, yellow, dark, and deep color. Additionally, the epidermis was thin and irregular, with visible abnormalities, and the dermis lost its normal structure and exhibited vacuolar changes. Researchers also reported a downregulation of the ECM-receptor interaction pathway, significant upregulation of AGEs expression, and significant downregulation of COLI, FN1, LM5, and TNC expression levels. This confirmed that a high-sugar diet causes skin damage associated with aging by accumulating AGEs, disrupting the expression of ECM proteins and their receptors, and reducing the activity of the ECM-receptor interaction pathway, which affects cell proliferation, migration, and adhesion, as well as the normal structure of skin tissue.[15]

One of the causes of aging is the accumulation of advanced glycation end products (AGEs) over a lifetime. The accumulation of AGEs in the body has two main sources. The first comes from endogenous production through the glycation process. Glycation is a non-enzymatic

process of covalently binding proteins, lipids, or nucleic acids with sugar molecules, usually glucose or fructose.[16]

Exogenous sources of AGEs include the consumption of refined and simple carbohydrates and foods subjected to thermal processing, such as grilling, frying, or baking. Excessive alcohol consumption, smoking, and physical inactivity can also contribute to AGE production. Other external factors that can increase AGE levels in the skin include pollution, metabolites present in tobacco smoke, UV radiation, and dihydroxyacetone (DHA) contained in self-tanning products. [20,21] Proteins with a long biological half-life are more prone to glycation and AGE formation than other proteins. The half-life of collagen varies depending on the tissue and is approximately 10 years in the skin, making it highly susceptible to glycation and AGE formation.[18] AGEs significantly impact collagen and elastin fibers, which provide skin elasticity and resilience. Glycation leads to intermolecular collagen cross-linking, increasing its stiffness and sensitivity to mechanical stimuli.[17,19] The formation of AGEs on collagen side chains also affects the protein's charge and disrupts its active sites, thereby impairing its ability to interact properly with surrounding cells and matrix proteins.[25] Other matrix proteins such as elastin and fibronectin can also be affected by glycation, further deteriorating skin function.[21,22,23,24]

Glycated collagen and other proteins exhibit high resistance to degradation by matrix metalloproteinases (MMPs). This further delays collagen turnover and its replacement with functional proteins.[26,27] Glycation is also closely linked to oxidation and inflammation. Glycation increases reactive oxygen species (ROS) levels, accelerating oxidative damage.[21,28] Moreover, AGEs can bind to specific receptors on the surface of cells known as receptors for AGEs (RAGE). Upon activation, RAGE triggers several cellular signaling pathways, further promoting inflammation and altered cytokine expression.[11,16,21,22]

Numerous studies indicate that excessive sugar intake in the diet can cause metabolic disorders and induce an increase in inflammatory mediators and certain pro-inflammatory cytokines in various tissues, leading to insulin resistance and chronic low-grade inflammation. [2, 29]

Chronic low-grade inflammation may be caused by factors secreted by adipose tissue, inflammatory factors secreted by liver tissue, and increased gut permeability, which can ultimately lead to the development of cardiometabolic diseases. [30]

A study aimed at assessing the impact of consuming small and moderate amounts of sugar-sweetened beverages over a 3-week period on LDL particle distribution and other glucose and lipid metabolism parameters, as well as inflammatory markers in healthy young men, revealed an increase in fasting glucose and an increase in high-sensitivity C-reactive protein, as well as a negative impact on LDL particles, confirming the harmful effects of even low intake of sugar-sweetened beverages. [32]

Another study evaluating the impact of a diet high in sucrose or artificial sweeteners on inflammatory markers such as CRP, haptoglobin, and transferrin in overweight individuals reached similar conclusions. The group with high consumption of sugar-sweetened foods and beverages showed increased levels of haptoglobin, transferrin, and CRP. [33]

Excessive sugar intake promotes inflammation in several ways. It has been shown that dietary fructose metabolism promotes de novo synthesis of free fatty acids (FFA), whose metabolites, by triggering inflammatory processes such as CRP, IL-6, and IL-1RA, the formation of ROS, and apoptosis, can contribute to the progression of non-alcoholic fatty liver disease (NAFLD) to non-alcoholic steatohepatitis (NASH). [31] Increasing evidence suggests a link between visceral obesity and inflammation and cortisol activation induced by fructose. Visceral adipose tissue is metabolically active and involved in producing numerous inflammatory cytokines, including TNF-α and IL-6, which in turn can induce CRP release in the liver. Moreover, other animal studies suggest that fructose leads to bacterial overgrowth in the gut and increased intestinal permeability, allowing endotoxin levels to migrate and activate Toll-like receptor 4 in Kupffer cells of the liver, whose activation leads to the release of several cytokines, primarily TNF-α. [31,34,35] Chronic inflammation induced by a diet high in simple sugars is a significant etiopathogenic factor in many skin diseases such as psoriasis, atopic dermatitis, hidradenitis suppurativa, or acne. [36,37]

Recent studies indicate that dietary components such as simple sugars and fats exacerbate psoriasis. Researchers found that the Western diet activated the interleukin 23 (IL-23) signaling pathway and production of IL-17A in γδT cells upon IL-23 stimulation. IL-17A is essential for the comprehensive development of skin inflammation. Meanwhile, IL-23 expression resulted in reduced microbiological diversity and marked dysbiosis in mice fed a Western diet. In one study, it was shown that in mice switched from a Western diet to a

standard diet, skin inflammation was reduced. [41,42,44] In another study, patients with psoriasis who consumed a Mediterranean diet had lower inflammatory markers. [43]

Scientific research also shows that high sugar intake can negatively impact hormonal balance, contributing to skin problems such as acne. A high-glycemic-load diet has been shown to increase circulating IGF-1 and insulin levels, stimulating rapamycin 1 activity. Rapamycin 1 promotes cell proliferation and inhibits apoptosis, increasing oxidative stress and inflammation, thereby promoting acne development. Elevated IGF-1 levels also stimulate androgen production, which is associated with sebum production and thus acne development. [38,39,40]

Excessive sugar intake is associated with an increased risk of obesity. [45] The accompanying increase in visceral fat in obesity is characterized by low-grade inflammation with the production of several cytokines and chemokines, such as beta-defensins 2/3, CXCL8/10, and CCL20. These changes affect both homeostasis and immune cell activation, promoting chronic inflammatory diseases such as psoriasis and delayed wound healing. [49,50]

Obesity also significantly impacts Hidradenitis Suppurativa (HS), also known as inverse acne. It is a chronic inflammatory disease localized in the axillary, inguinal, perineal, gluteal, pubic, and breast areas, i.e., areas rich in apocrine glands. Obesity leads to insulin resistance with increased insulin and IGF-1 levels and hyperandrogenism. This stimulates androgen receptors, which increase intraductal keratinocyte production. Hyperkeratinization causes follicular obstruction, rupture, inflammation, and skin damage. This, in turn, increases susceptibility to infection by commensal bacteria, leading to fistula formation and scarring. [51,52] Additionally, some foods, such as dairy products and simple sugars, have been observed to exacerbate the symptoms of the disease. A study evaluating the role of diet or nutrient supplementation on the course of the disease found that a significant number of individuals who changed their eating habits, including reducing simple sugar intake, experienced symptom improvement. [53]

Furthermore, patients with abdominal obesity have elevated cortisol levels. [46] High cortisol levels negatively affect the skin, worsening existing skin problems such as acne, atopic dermatitis, or psoriasis. [47,48]

Like other inflammatory diseases, metabolic syndrome is associated with the development of alopecia areata (AA). [54] Excessive sugar intake is also linked to androgenetic alopecia (AGA). [55] A study examining the relationship between sugar-sweetened beverage consumption and male pattern hair loss in young men found that high sugar-sweetened beverage intake was associated with a higher risk of androgenetic alopecia. [56] The high sugar content in beverages leads to increased serum glucose, activating the polyol pathway in the liver, leading to liver steatosis and impaired synthesis of sex hormone-binding globulin. [57] The scalp phenotype in alopecia is characterized by overactive PPAR-γ receptors, increased fatty acid synthesis, enlarged sebaceous glands, and increased sebum synthesis. Studies have shown that glucose utilization in the polyol pathway reduces the glucose available to keratinocytes in the outer root sheath of hair follicles, and the gluconeogenesis process is further hindered by depletion of ATP and phosphate levels. Lack of energy in outer root sheath keratinocytes is considered a possible cause of AGA development. Furthermore, hair loss due to excessive sugar consumption may be further exacerbated by the presence of chronic diseases such as diabetes, hypertension, hyperglycemia, thyroid dysfunction, and anemia or emotional problems. [55,56]

SUMMARY

Sugar consumption has significantly increased over the last 30 years and now poses a serious threat to human health by contributing to an increased incidence of many non-communicable diseases, including obesity, cardiovascular diseases, metabolic syndrome, and type 2 diabetes (T2D), and worsening skin health and condition. [2] Excessive sugar intake deteriorates skin quality through several mechanisms, including skin protein glycation, inflammation induction, and hormonal disturbances, as well as contributing to obesity and related skin dysfunctions. Dietary strategies supported by research include a low glycemic index diet that focuses on low-glycemic foods and portion sizes. The authors, through the analysis of 55 studies involving 77,557 patients, including 4,534 with psoriasis, found high-quality evidence supporting weight loss through a low-calorie diet as an adjunct to standard medical therapy for overweight or obese adults (body mass index (BMI) $\geq$ 25) with psoriasis. This specifically refers to a low-calorie diet with a daily caloric intake ranging from 800 to 1,400 kcal, lasting from 16 weeks to 6 months, with consistent improvement in disease severity, dermatological quality of life index (DLQI), and weight loss. [55] Improving glycemic control is also a key strategy in reducing AGE production. In one experimental study, improved glycemic control

in volunteers over a 4-month period significantly reduced new collagen glycation. [22,58] Studies have shown that AGE consumption is not only associated with sugar content in food but also depends on the cooking method. AGE formation is accelerated by increased protein turnover, hyperglycemia, temperatures above 120°C (248°F), and the presence of oxygen, reactive oxygen species, or active transition metals. Therefore, limiting grilling, baking, and frying, which can increase AGE levels by 10 to 100 times, and opting for boiling or steaming, as well as cooking at lower temperatures and for shorter periods, can provide many benefits for the skin. Additionally, various dietary compounds show positive effects on the skin due to their antioxidant and anti-inflammatory properties. [22,59] As can be seen, diet plays a significant role in skin disease prevention; however, further research on dietary manipulation and the impact of dietary components, including sugars, on skin diseases is necessary to better understand the pathomechanism and develop appropriate treatment strategies for patients.

Author Contributions:

Conceptualization, supervision and project administration, methodology, software, validation, formal analysis, investigation, resources, writing original draft preparation, writing review editing and visualization: Natalia Dolata, Bartosz Balcer, Paweł Liszka, Mateusz Pakuła, Maja Weimann, Adrianna Kruczkowska, Aleksandra Stosiek, Agnieszka Cebula, Emil Bulzacki, Wojciech Urbański

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The data presented in this study is available upon request from the corresponding author.

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Conflict of Interest Statement

All authors declare that they have no conflicts of interest.

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