How eating sweets affects the skin – the impact of high glycemic index diet on acne formation: A review

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Abstract:

Introduction and purpose

Acne is one of the most common dermatological conditions, especially in adolescents. The etiology of acne is complex, and many factors influence the formation of lesions. Local inflammation, a proliferation of Propionibacterium acnes, hormonal influences with particular importance of androgens, hyperkeratinization, and excessive sebum production are essential in the pathogenesis. The factors stimulating these processes are complex and not always clear. Therefore, in our work, we focused on investigating the connection between a high-glycemic diet and the formation of acne lesions.

The state of knowledge

The link between diet and acne has been only mentioned in the literature for many years. However, the development of science has made it possible to understand the exact pathogenesis of acne and the relationship of acne lesions to specific dietary components. Of all the diet products, a high glycemic index diet stands out as the one with the most significant impact. Foods with a high glycemic index, i.e. those with a high amount of simple sugars, through an increase in the expression of factors such as insulin, IGF-1 cause excessive sebum production in the sebaceous glands and even an increase in inflammatory infiltration, which further exacerbates acne lesions. In the following review paper, we have looked at the metabolic pathways and hormonal changes that link the consumption of high glycemic index foods to the formation of acne lesions and described several original papers that unequivocally demonstrate the critical role of this diet on acne formation.

Summary

Based on the literature, we emphasize that in acne treatment, it is necessary not only to focus on medications but, above all, to look at the patient holistically, modifying the lifestyle with particular importance on modifying the patient's diet by lowering its glycemic index.

Introduction and purpose

Acne vulgaris, a prevalent dermatological disease, mainly affects people in adolescence or early adulthood. It afflicts up to 9.4% of the world's population, with a staggering 85% of teenagers being affected [1]. The physical manifestations of acne, including scars, significantly impact self-image, often leading to a negative influence on the quality of life [2, 3]. This chronic, local inflammatory process is characterized by excess sebum production, and proliferation of Propionibacterium acnes (P. acnes), hormonal factors also play a significant role [4, 5]. Acne lesions can be divided into inflammatory (pustules, papules, cysts and nodules) and non-inflammatory lesions (comedones) [6].

Of the many factors influencing acne formation, diet is of particular interest due to the relative ease of modifying eating habits. Several dietary components are mentioned in scientific papers, such as high glycemic index food, dairy products, fat food, chocolate, or others [7, 8]. However, the scientific evidence for a high glycemic index diet is the most convincing [9, 10]. Therefore, we decided to focus on a high-GI diet in the following review.

Foods with a high glycemic index are rich in carbohydrates and are characterized by rapid absorption and an immediate and rapid increase in blood glucose, leading to large insulin release [11]. The effect of a high-GI diet on the onset of acne is still under research. Therefore, the following review summarizes the recent evidence on the influence of a high-GI diet on acne formation.

The state of knowledge

1.Pathogenesis of Acne Vulgaris

The pathogenesis of acne vulgaris is multifactorial, with increased sebum production, hormonal factors such as androgen influence, hyperproliferation of P. acnes, hyperkeratinization, chronic and local inflammation, or genetic impact such as DNA methylation [4, 12].

The main pathological processes are hyperseborrhea, increased sebum production in the hair follicles, and dysseborrhea, a change in the quality of sebum [13, 14]. These conditions promote P. acnes proliferation and lead to impaired follicular barrier, which induces acne lesion formation [15].

Androgens also play a crucial role in acne pathogenesis. These hormones promote the proliferation and differentiation of sebocytes with increased lipogenesis. Other hormones in the endocrine system are also involved. This includes corticotropin-releasing hormone (CRH) or α-melanocyte-stimulating hormone (α-MSH). CRH influences acne formation through steroidogenesis induction, testosterone, and growth factor interaction. On the other hand, α-MSH induces lipogenesis and plays a role in sebocyte differentiation [13, 16, 17].

Another critical factor is hyperkeratinisation. Increased cellular deposition leads to the accumulation of irregular exfoliated corneocytes in hair and sebaceous follicles [4, 18].

P. acnes hyperproliferation, currently known as Cutibacterium acnes (C. acnes), plays a key role in acne pathogenesis. These bacteria colonize hair and sebaceous follicles. They secrete lipase, which metabolizes sebum to glycerol and fatty acids. This leads to local inflammation, which results in comedone formation [19].

Local inflammation leads to increased infiltration of immune cells, such as neutrophils, lymphocytes, and macrophages, causing hair follicle damage and inflammatory lesions formation (nodules, papules, cysts, or pustules) [4, 20].

2.High Glycaemic Index Diet and The Mechanism in Which It Influences Acne Lesions Formation

For many years there has been a strong common belief that diet, especially tasty and greasy, has a significant effect on the development of acne [21]. Especially Western diet, popular in highly developed countries that contains fair amount of milk and dairy products, meat, chocolate, saturated fatty acids and has high glycemic index is believed to aggravate acne vulgaris [22-24]. In populations consuming foods with a low glycemic index and no milk or dairy products, acne is much less common [24, 25] – to mention just a few: none of the 1200 examined Kitavian subjects from Papua New Guinea or 155 examined Ache subjects from Paraguay had acne lesions [26].

The best example of the link between the Western diet and the development of acne comes from studies of populations that have migrated to developed countries. Both Inuit and Irish populations developed acne lesions after emigration and the associated changes in diet [27, 28].

Nowadays, thanks to the development of science we can know in detail the pathways and mechanisms behind the formation of acne lesions after consumption of certain foods.

High glycemic index (GI) diet, on which we focus in this paper, induces significant insulin output causing hyperinsulinemia that aggravates acne in different mechanisms. The first one is increased androgen secretion, which contributes to formation of acne lesions trough the stimulation of sebum production [29]. What is more, hyperinsulinemia disrupts the balance between IGF-1 and insulin-growth factor binding protein (IGFBP-3) levels [30]. Overall IGF-1 level increases, which results in higher keratinocytes proliferation and, acting through the mediation of other hormones (IGF-1 influenced androgens, growth hormone and glucocorticoids), increased sebum production [15, 30-32]. What is important, increased IGF-1 level promote cell proliferation by IGF-1 receptor (IGF1R) activation, which can lead to the hyperproliferation of keratinocytes of the sebaceous glands [33]. Furthermore, insulin and IGF-1 lead to PI3K/Akt pathway activation, causing higher forkhead box protein O1 (FoxO1) expression, a key factor in acne pathogenesis [13]. It is also important to emphasize, that IGF-1 increases lipogenesis in sebaceous glands through activation of sterol response element

binding protein-1 (SREBP1), leading to increase of total amount of sebum consisted of monounsaturated fatty acids [24]. This leads to acne exacerbation.

Furthermore, there is evidence that a high glucose concentration also upregulates microRNA-21 in macrophages, which leads to proinflammatory M1 macrophage polarization. M1 macrophages by IL-1β production stimulate inflammation [34, 35]. Therefore, it is a possible mechanism that could worsen acne lesions' inflammation.

A review of the literature provides numerous examples to support the mechanisms in which high glycemic index contributes to acne lesions formation [9, 36].

Smith et al. [37] in their randomized, investigator-masked, controlled trial compared for the first time the impact that low (LGL) and high glycemic diet (HGL) has on acne vulgaris. In low glycemic diet group after 12-week dietary intervention there was significant reduction of total acne lesion counts and inflammatory counts compared to the control group (p=0.01 and p=0.02 respectively). Considering hormonal variables, in LGL group the reduction of free androgen index and an increase of IGFBP-1 were observed.

Another study conducted by Smith et al. [38] was based on the administration to patients LGL or HGL diet. After only 7-day-long dietary intervention SHBG levels significantly decreased in the HGL group. They also observed an increase of the free androgen index in HGL group. The observed changes in the LGL group are increase in IGFBP-1 and IGFBP-3 levels.

The importance of such short interventions is also confirmed by the work of Burris et al. [39], in which a two-week change to a low GI and GL diet in 34 of the 66 people studied had a beneficial effect in decreasing IGF-1 levels.

Kwon et al. [40] in their 10-week dietary intervention also got promising results. In LGL group there was significant improvement of all (inflammatory and non-inflammatory) acne lesions compared to HGL group. What is interesting, they examined the skin at the histopathological level noticing the reflection of clinical changes - reduction of sebaceous glands and lesser inflammation in the LGL group.

Zapała et al. [41] examined the impact of reduced glycemic load diet on acne being one of the clinical hyperandrogenism manifestations. The 30 women of age 18–49 were subjected to a low glycemic index diet with anti-inflammatory elements and compared to 14 controls. After sixty days in the study group there was a decrease in total testosterone levels, decrease in the free androgen index and an increase in SHBG levels. The mean presence of acne lowered pre- and post-intervention respectively. There was almost no change observed in the control group.

Looking at the topic from another point of view we see similar dependencies. Jung et al. [42] divided patients with acne to two groups: acne patients aggravated by food (AF) and acne patients not aggravated by food (NAF). Among the AF patients, a higher level of IGF-1 and lower level of IGFBP-3 were observed compared to the NAF patients.

Even though the harmful effect of high glycemic diet could be widely spread in public campaigns, it doesn't necessarily mean that people will change their eating habits. In Kostecka et al. [22] study the participants' awareness of acne aggravation by a high glycemic index diet didn't prompt them to give up such products.

Conclusions

The aim of this review is to emphasis the fact that the treatment of acne vulgaris should include not only medications but also complex lifestyle interventions. Due to the evident impact of a high glycemic index diet on acne formation, people being treated for acne should receive dietary treatment added to conventional drug treatment, with special attention paid to eliminating high-GI products. It means that only a holistic view will allow for the best therapeutic outcome. To develop the best-balanced diet for acne patients, it is important to continue the research focused on the mechanisms of acne, hormonal and metabolic factors and the most importantly various factors presented in food and their role in acne aggravation.

Institutional Review Board Statement
Not applicable.

Informed Consent Statement
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Data Availability Statement
Not applicable.

Conflict of Interest Statement

Authors declare no conflicts of interest.

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