INTRODUCTION

Skin as an outer layer of the body and a main connection between the body and the environment is a way that pathogens access the body; therefore, it has a crucial protecting role for the body because of obstacle functioning and microbiota. Factors that seem to commonly contribute to the disease are follicular hyperkeratinization, propionibacterium acnes, sebum production, and inflammation; however, pathogenesis of acne vulgaris as an inflammatory skin disorder is still poorly understood. Factors that seem to commonly contribute to the disease are follicular hyperkeratinization, propionibacterium acnes, sebum production, and inflammation; however, pathogenesis of acne vulgaris as an inflammatory skin disorder is still poorly understood. Factors that seem to commonly contribute to the disease are follicular hyperkeratinization, propionibacterium acnes, sebum production, and inflammation; however, pathogenesis of acne vulgaris as an inflammatory skin disorder is still poorly understood. Factors that seem to commonly contribute to the disease are follicular hyperkeratinization, propionibacterium acnes, sebum production, and inflammation; however, pathogenesis of acne vulgaris as an inflammatory skin disorder is still poorly understood. Factors that seem to commonly contribute to the disease are follicular hyperkeratinization, propionibacterium acnes, sebum production, and inflammation; however, pathogenesis of acne vulgaris as an inflammatory skin disorder is still poorly understood. Factors that seem to commonly contribute to the disease are follicular hyperkeratinization, propionibacterium acnes, sebum production, and inflammation; however, pathogenesis of acne vulgaris as an inflammatory skin disorder is still poorly understood. Factors that seem to commonly contribute to the disease are follicular hyperkeratinization, propionibacterium acnes, sebum production, and inflammation; however, pathogenesis of acne vulgaris as an inflammatory skin disorder is still poorly understood. Factors that seem to commonly contribute to the disease are follicular hyperkeratinization, propionibacterium acnes, sebum production, and inflammation; however, pathogenesis of acne vulgaris as an inflammatory skin disorder is still poorly understood. Factors that seem to commonly contribute to the disease are follicular hyperkeratinization, propionibacterium acnes, sebum production, and inflammation; however, pathogenesis of acne vulgaris as an inflammatory skin disorder is still poorly understood. Factors that seem to commonly contribute to the disease are follicular hyperkeratinization, propionibacterium acnes, sebum production, and inflammation; however, pathogenesis of acne vulgaris as an inflammatory skin disorder is still poorly understood. Factors that seem to commonly contribute to the disease are follicular hyperkeratinization, propionibacterium acnes, sebum production, and inflammation; however, pathogenesis of acne vulgaris as an inflammatory skin disorder is still poorly understood. Factors that seem to commonly contribute to the disease are follicular hyperkeratinization, propionibacterium acnes, sebum production, and inflammation; however, pathogenesis of acne vulgaris as an inflammatory skin disorder is still poorly understood. Factors that seem to commonly contribute to the disease are follicular hyperkeratinization, propionibacterium acnes, sebum production, and inflammation; however, pathogenesis of acne vulgaris as an inflammatory skin disorder is still poorly understood. Factors that seem to commonly contribute to the disease are follicular hyperkeratinization, propionibacterium acnes, sebum production, and inflammation; however, pathogenesis of acne vulgaris as an inflammatory skin disorder is still poorly understood. Factors that seem to commonly contribute to the disease are follicular hyperkeratinization, propionibacterium acnes, sebum production, and inflammation; however, pathogenesis of acne vulgaris as an inflammatory skin disorder is still poorly understood. Factors that seem to commonly contribute to the disease are follicular hyperkeratinization, propionibacterium acnes, sebum production, and inflammation; however, pathogenesis of acne vulgaris as an inflammatory skin disorder is still poorly understood.
Studies on the influence of diet in acne formation are unconvincing and usually focused chiefly on specific foods such as milk that is considered artificial in diet category \(^2,^3\). In this study, we reviewed the literature focusing on the association between diet and BMI in acne vulgaris.

**MATERIALS AND METHODS**

We reviewed English articles reported in the literature from 2008 to 2019 in order to find associations between diet and BMI in acne vulgaris. Google Scholar and PubMed databases were searched, using the key words zinc, diet, vitamin D, BMI, and acne vulgaris.

**RESULTS AND DISCUSSION**

The first studies about the effect of milk have been conducted many years ago; however, there has been no conclusive evidence that milk and dairy products have comedogenic effects \(^3\). The probable cause of possible comedogenic effects of milk and its products is the hormonal content produced by cows during pregnancy, and it is claimed that the constituent of milk which most stimulates the pilosebaceous unit is insulin-like growth factor 1 (IGF-1), whose concentration in the blood varies depending on the severity of acne \(^2,^3\). Acne is affected not only by hormones but also by some biochemical associations between the hormones and the pilosebaceous unit. Hence, addition of exogenous hormones derived from the diet to the pool of endogenous hormones may have a significant effect \(^3\).

Chocolate has usually been considered as a factor that may relate to exacerbation of acne, but there are few evidences supporting this negative impact on the skin, and dermatologists have frequently observed that patients have new acne lesions few days after ingestion of products containing chocolate \(^3\). However, there is no data and evidence about the type of chocolate consumed by subjects and the cocoa percentage in consumed samples which may affect the results. Dark chocolate contains more antioxidants derivatives than milk chocolate, which would imply that it may have less comedogenic effects. The question of whether chocolate aggravates acne lesions, is yet to be clear \(^3\).

Reactive oxygen species produced by leukocytes contribute to inflammatory progression of acne and reactive oxygen species that are normally removed by cellular antioxidants such as glucose-6-phosphate dehydrogenase and catalase, both of which are presented in little amounts in acne patients. It has been suggested that oxidative stress may be implicated in the origin of acne, and antioxidant drug effects (or antioxidant supplements) may be valuable adjuvant in acne treatment \(^3\). Studies on this matter are crucial to support the theory of the positive role of antioxidants in acne therapy, but effects of such substances in the course of acne are not yet fully explored.

Zinc is a micronutrient that is necessary for functioning and development of the human skin tissue. It has been also shown to be bacteriostatic against Propionibacterium acnes, to inhibit chemotaxis, and to reduce production of pro-inflammatory cytokine – tumor necrosis factor \(\alpha\) (TNF-\(\alpha\)) \(^3\). Some studies have established that patients with acne often are zinc-deficient, and the oral supplementation would have positive effects on the treatment of acne vulgaris \(^3\).

Retinol and vitamin A derivatives are found principally in products with animal origin, while products of plant origin contain mainly provitamin products. The main sources for it are the milk and dairy products, liver, eggs, and oils derived from them. Vitamin A is a fat-soluble vitamin stored in the liver and may be effective in prevention of acne in high-dosages \(^3\).

Vitamin D regulates the immune system and also proliferates and differentiates keratinocytes and sebocytes. The effect of vitamin D is not only limited to calcium homeostasis, but it is also important in the regulation of the cell growth, immune system, and cell differentiation. Human sebocytes and keratinocytes are some target cells for biologically active vitamin D metabolites by nuclear vitamin D receptors. Hence, it has anti-comedogenic and antioxidant properties, and vitamin D deficiency may facilitate the pathogenesis of acne and may play a potential role in pathogenesis of acne vulgaris, or acne vulgaris may have a negative effect on vitamin D synthesis \(^4,^5\).

Regarding body mass index (BMI) and acne severity, there are many controversies based on different geographic populations as well as age and gender related categories. However, this correlation has not been completely supported
by many articles in spite of some correlations in many sub-group analyses.

High prevalence and great burden of acne makes it as one of the most important areas of scientific research especially regarding its correlations, therapies, and complications like acne scars. In this review, we briefly focused on some associations of inflammatory acne.

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REFERENCES