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Air Pollution, Cigarette Smoking, Diet, and Acne: Do Environmental and Habitual Factors Affect Acne?

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Abstract

Acne vulgaris is a common skin condition that is mostly seen in young adults. It has always been an enigma for physicians and patients if diet or environmental factors are responsible for developing acne. This article presents the relationship between air pollution, cigarette smoking, dietary factors and acne through the pathogenesis of acne.

Keywords: Acne vulgaris; Acne; Air pollution; Cigarette smoking; Diet; Nutrition

Introduction

Acne vulgaris is a common skin condition presenting as chronic inflammation involving the pilosebaceus unit. It is mostly seen in 15-17 age groups and the prevalence is 85% of young adults aged 12–24 years [1,2]. The chronic course of acne causes psychological and economic burden for patients and it causes significant impairment in the quality of life (QoL) of patients [3,4]. The steps of pathogenesis include sebum production, follicular keratinization, Propionobacterium acnes colonization and inflammation [3]. Besides, the processes of the pathogenetic pathways of acne could be affected by numerous external factors via metabolic alterations in the body. For instance, sebum production levels are elevated when dietary elements raise glycemic index levels [5-7]. Additionally, in the literature, there are studies questioning the effects of air pollution and cigarette smoking on acne as external factors [8-19].

The purpose of this article is to present the effects of environmental, habitual, and dietary factors on acne by understanding acne's pathophysiology. In the research process, Medline and Google scholar articles were searched using acne, acne vulgaris, air pollution, cigarette smoking, diet, and nutrition.

Pathogenesis of Acne

The pathogenesis of acne has multifaceted processes, and these involve production of sebum, keratinization in the follicles, colonization of p. acnes and inflammation. P. acnes, also known as Cutibacterium acnes or Corynebacterium acnes play a crucial role in the inflammatory process of acne [3]. In addition to colonizing the pilosebaceous unit for the metabolism of lipid-rich sebum, P. acnes specifically grows when excessive sebum production is present [3,20-22]. The mechanisms through which p. acnes stimulates the innate immunity include the expression of protease activated receptors (PARs), tumour necrosis factor (TNF)-a, and toll-like receptors, as well as the production of interferon (INF)-c, interleukins (IL-1, IL-8, IL-12), and matrix metalloproteinases (MMPs). Activation of these inflammatory mediators results in hyperkeratinization of the pilosebaceous unit [20,21]. Moreover, abnormal differentiation of follicular epithelial cells with tightened intracellular adhesions leads to easy shedding, which results in new hyperkeratotic plugs or microcomedones. These microcomedones may develop into non-inflammatory open or closed comedones [3]. When follicles rupture, pilosebaceous unit structure deteriorates into the dermis and causes inflammation, which manifests as papules, pustules, nodules, and cysts [23].

Sebum/lipid oxidation promotes inflammation via complement activation. Proinflammatory mediators cause the proteases, and neutrophil-attracting chemotactic factors such as IL-1 and leukotriene-B4 (LTB-4) that contribute to inflammation. In sebum, specifically MMPs have an active role in the inflammation process, cell proliferation and deterioration in the dermal matrix. Pilosebaceous vascularization is stimulated by CD4+ lymphocytes and macrophages that trigger inflammation [20,21].

Clinical Presentation of Acne

Acne is typically present on body parts with sebaceous glands, including the face, neck, chest, upper back, and upper arms. As a

result of sebum secretion, those body areas may have oily and shiny skin [23]. Human beings have sebaceous glands because of an evolutionary adaptation in nature. As only human babies are at risk of dystocia during birth, their face, neck, chest, upper back, and upper arms produce increased levels of sebaceous glandsin order to protect them from dystocia. In this hypothetical scenario, only humans have a higher risk of dystocia and acne [24].

Initial acne lesions are microscopic lesions called microcomedones, which may not be visible during the clinical examination. These microcomedones can grow to non-inflammatory forms, including closed and open comedones, followed by inflammatory lesions that include superficial or deep lesions such as papules and pustules and deep pustules or nodules. Diagnosis of acne is based on clinical examination and the classification of acne can be ordered as comedonal, papulopustular, nodular, nodulocystic or conglobate acne [25].

Air Pollution and Acne

In recent years, the correlation between air pollution and skin diseases has gained more interest; however, few studies have been carried out about air pollution's effects on dermatologic conditions, so air pollution is rarely investigated in relation to skin diseases. According to the literature, air pollutants like particulate matter (PM2.5 and PM10), nitrogen dioxide (NO2), sulfur dioxide (SO2), and ground level ozone are associated with various skin conditions [9,26]. Negative effects of air pollution on skin have not yet been explicitly explained. Recent studies indicate that aggravation of eczema in children has been linked to air pollution as well as high concentrations of ozone in the ground are linked to an increased incidence of urticaria cases [26,27]. NO2 exposure was found to be related to pigmentation on the cheek area of the face in Caucasian and Asian women. Thus, limited data is available to demonstrate the relation between acne and air pollution. However, it was reported at the International Dermatology Conference in Beijing that, via personal communication, dermatologists presented that 67% of responders agreed that acne prevalence increases with pollution [9].

During a 2-year time period study, rising concentrations of air pollutants as NO2, PM2.5, and PM10 were significantly associated with higher numbers of outpatient visits for acne vulgaris in dermatology clinics [10]. Also it was reported that heavily polluted metropolitan areas were associated with increased production of sebum, which is a known pathogenic factor for acne vulgaris. In the same manner, biochemical features of sebum demonstrated qualitative changes in the people living in polluted areas [11]. Therefore, it is assumed that increased levels of carbonylated proteins in the stratum corneum and sebum modifications may explain the relation between acne and air pollution [12].

Notably, the alterations of skin microflora via air pollution are reactive oxygen species production, aryl hydrocarbon receptor activation, and inflammatory cascade induction. Pollution particles plug the pores physically and lead to p. acnes flourishing in the anaerobic microenvironment. Pollutants also induce the inflammatory cascade in skin cells through proinflammatory factors such as IL-1α, IL-6, IL-8, and TNF-α that cause inflammatory changes in the skin [8]. Moreover, air pollution induces oxidative effects by decreasing vitamin E levels and increasing levels of oxidized squalene in the sebum. These findings reveal that air pollution may worsen acne vulgaris [9]. Accordingly, a study revealed that acne patients exposed to air pollution by living in proximity to an airport or an industrial site had more acne exposomes [28]. On the other hand, in an epidemiologic study, no significant association was found between PM10 and outpatient visits for acne; however, the increase of SO2 and NO2 concentrations indicated significant correlation with the rise in the number of outpatient visits for acne [13]. Another study, which did not evaluate acne results, demonstrated that the prevalence of oily skin in a metropolitan area was 25.6% higher than in participants in a non-polluted area [14].

Cigarette Smoking and Acne

Cigarette smoking causes exposure to thousands of chemical substances, including reactive oxygen species, carbon monoxide, reactive nitrogen species and electrophilic aldehydes [15]. It is reported in a study that acne was significantly more prevalent in smokers as 102 smokers compared with 184 non-smokers. On the other hand, if the same study evaluated only the 15–40-year-old group, no association was found between acne and smoking [16]. Similarly, a study compared 293 acne patients and 301 patients with other dermatological conditions about smoking, but no statistically significant correlation was found [17]. In a large cohort study of young men, active smokers showed a significantly lower prevalence of severe acne than non-smokers. According to this result, it was suggested that nicotine could be used as a topical treatment agent in acne disease [18]. Similarly to this study, Rombouts et al demonstrated smoking may show protective effects in the development of inflammatory facial acne especially in girls; on the other hand, smoking is not advisable for acne prevention. Hence, there was no relation in the boys' acne group with cigarette usage [19]. In conclusion, these results lead us to understand that cigarette smoking does not cause acne to worsen, however it increases the production of the proinflammatory cytokine IL-1α in the inflammation cascade of acne [8]. Cigarette smoking oxidizes squalene, producing peroxidized forms that are comedogenic and trigger acne inflammation [15]. Additionally, polycyclic aromatic hydrocarbon (PAHs) called benzo(a)pyrene (BaP) is a compound of cigarette that triggers the secretion of IL-8 from the epidermal keratinocytes which is related to inflammatory acne [8].

Diet and Acne

The carbohydrate content induces glycemic and insulinemic responses which result in acne progression [6,29]. The association between high glycemic load (excess carbohydrate intake) and the presence of acne was reported in the literature. Smith et al showed that the low glycemic load diet (LGLD) reduced acne lesion counts and improved insulin significantly comparing with a high glycemic

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load diet (HGLD) after 12 weeks [30]. Further, Penso et al.'s study evaluated 24,452 participants and concluded that the hypothesis about Western diet (rich in animal products, fats and sugars) is related to acne in adulthood [1]. On the other hand, 49 acne patients and 42 healthy controls were interviewed about their dietary routine. No significant relation between acne and serum glucose, insulin, leptin levels, indeed glycemic index, or dietary glycemic load was reported [31].

There are studies showing chocolate consumption is a relevant factor for causing acne [7,32]. Halvorsen et al demonstrated an association between acne and a low intake of raw vegetables and a high intake of chocolate/sweets and potato chips. According to this study, a LGLD is beneficial in preventing acne [32]. Moreover, LGLD reduces sebum secretion and acne numbers compared to HGLD in observational studies [6]. However, in another study, Ismail et al didn't find any relation between chocolate and acne. The results remained unclear regarding chocolate's role as an etiologic factor in acne. Chocolate's ingredient heterogeneity presents ambiguous reasons to affect the acne pathogenesis [33].

In a study investigating hormones such as serum androgen, insulin and IGF (insulin-like growth factor)-1, elevated levels of concentration were demonstrated to be related to acne in women [34]. HGLDs induce IGF-1 activity which stimulates androgen production that activates the proliferation of keratinocytes and sebocytes. In this context, there is a positive correlation between dairy product consumption and acne. Milk increases insulin and IGF-1 levels as a HGLD. Bovine IGF-1 in cow milk can bind to human IGF-1 receptors [6]. Hypothetically milk contains hormonal factors such as estrogens, progesterone, the androgen precursors as androstenedione, dehydroepiandrosterone-sulfate, 5a-reduced steroids like 5a-androstanedione, 5a-pregnanedione, and dihydrotestosterone. Progesterone and dihydrotestosterone precursors in milk are suspected of comedogenic effects. Also some bioactive molecules such as glucocorticoids and IGF-1 in the milk are responsible for sebum production [7]. Accordingly, whey and casein proteins present in milk and may aggravate acne; casein increases IGF-1 concentrations while whey protein promotes hyperinsulinemia by stimulating secretion of beta cell insulin [35]. After industrial milk processing, levels of IGF- 1, 5α- reduced steroids, and α-lactalbumin hormones remain high in milk left over. These hormones may affect the pilosebaceous glands, causing them to produce more sebum. In addition to containing carbohydrates, milk contributes to the effect of a high glycemic index/glycemic load diet that leads to acne progression [30]. In a meta-analysis, 14 observational studies were evaluated and a positive relationship between acne and total milk, low-fat milk, and skim milk intake was found. Low fat milk and skimmed milk exhibit more inducible effects on acne pathogenesis than whole milk [36]. These results could be explained as fat reducing processes enhance the insulin and IGF-1 promoting elements of milk [6]. Despite milk consumption being associated with acne incidence, no correlation was found between yogurt consumption and acne incidence [7]. Yoghurt has lower levels of IGF-1 due to probiotic bacteria during fermentation [37]. Furthermore, fermented milk enriched with lactoferrin has been shown to reduce skin surface lipid triglycerides, resulting in reduced sebum production, preventing acne [38].

In acne pathogenesis, vitamin D also has immune regulatory functions as well as anti-inflammatory and antimicrobial effects. It had been shown that, vitamin D treatment decreased the expression of inflammatory biomarkers, such as interleukin IL-6, IL-8, and MMP-9 in cultured sebocytes. In addition, vitamin D treatment results in a decrease in the levels of IL-17 which is an inflammatory cytokine, in acne patients [39]. Moreover, it has antimicrobial effects by inducing antimicrobial peptides such as LL-37 in human sebocytes [40]. Accordingly, a controlled study found that vitamin D deficiency was more common in acne patients [38]. Furthermore, in a randomized controlled study, vitamin D supplementation showed improvement in acne patients due to its anti-inflammatory effect [41].

Conclusion

There are many external factors that contribute to the etiopathogenesis of acne. Observational studies demonstrated a relationship between acne and air pollution. According to these studies, pollutants alter skin flora, modify sebum production, and increase inflammatory mediators. Cigarette smoking is also a factor contributing to inflammation. In order to understand the specific mechanisms involved in the pathogenetic pathways, further clinical research is required. Nonetheless, dietary habits have been linked to acne; specifically, milk consumption and acne are clearly associated via hormonal mechanisms. In addition, HGLDs may aggravate acne, whereas LGLDs may prevent it. Currently, the relationship between chocolate consumption and acne is ambiguous due to the heterogeneity of ingredients. Besides, vitamin D intake could be supportive for acne treatment due to its anti-inflammatory effects. Frankly, to demonstrate the relations between air pollution, cigarette smoking, diet and acne, further clinical and translational research is still needed.

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