

## Association between Obesity and Acne Vulgaris Development

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### Abstract

**Background:** Numerous factors have been linked to the common dermatological disorder acne vulgaris. Excessive production of sebum and its composition plays a crucial part in the complicated and multifaceted pathophysiology of acne. Peripheral hyperandrogenism and insulin resistance are common conditions associated with obesity, and they may be linked to elevated production of sebum and the emergence of severe acne. Body mass index (BMI) is a number that's frequently used to determine obesity and overweight. Long-standing theories suggest a link between BMI and acne, and in recent years, relevant research has emerged, albeit with mixed findings. **Objectives:** The purpose of this review is to comprehend the relationship between fat and acne. **Conclusion:** Acne vulgaris development is associated with obesity. It was observed that Acne vulgaris is more common in people with higher BMIs.

**Key Words:** Acne Vulgaris; Body Mass Index; Serum Lipids.

### Introduction

Acne vulgaris (AV) affects a significant section of the global population, many of whom are teenagers [1]. Persistent inflammation of the pilosebaceous units is characteristic of acne vulgaris (AV). Seborrhea, papules, comedones, pustules, pseudocysts, nodules, and maybe scarring are its defining features. The pathognomonic lesions of AV are called comedones [2]. The fundamental cause of AV is excessive androgen-induced stimulation of sebaceous glands, which results with hyperkeratinization and Cutibacterium acnes infection of the pilosebaceous follicle [1].

The face, upper anterior trunk, and posterior trunk are areas with a high density of sebaceous glands where AV is more likely to occur [3,4]. The etiology of AV is complex and multifaceted. Propionibacterium acnes (P. acnes) activity, inflammation, follicular epidermal hyperproliferation and increased sebum production are the four primary variables that are crucial in the pathogenesis of AV [5]. A familial history of AV, an elevated body mass index (BMI), psychological stress, and an oily skin type are some of the risk factors for AV [6].

Overabundance of maladaptive adipose tissue may cause lipid peroxidation, which impacts innate immunity's processes of inflammation as part of the body's reaction to the pathophysiology of acne vulgaris [7]. Certain studies suggesting a diet heavy in fat and carbs exacerbate acne have shown a link between food and acne [8]. Obesity appears to have an impact on acne vulgaris occurrences, and there is a correlation between obesity (BMI >30) and immunearbitrated processes and hormones that enhance the inflammatory response [9]. Body weight reduction reduces bacterially-induced face lipolysis and enhances the therapy [10].

### Acne vulgaris:

A major inflammatory condition that affects the pilosebaceous unit is acne. Increased sebum production, hyperkeratinization of the follicular infundibulum, inflammation, and Cutibacterium acnes (previously Propionibacterium acnes) are the four main factors involved in the multifactorial pathogenesis [11]. Research using twins has demonstrated that 81% of the population diversity in acne is explained by genetic variables, indicating that acne is highly heritable [12]. When it comes to sebum-excretion rates and acne prevalence, monozygotic twins exhibit higher concordance than dizygotic twins [13].

### Epidemiology

Males are more likely to have more severe acne vulgaris, while girls are more likely to have it after the age of twenty [14]. A comprehensive epidemiologic analysis revealed that there is disagreement on the topic of acne prevalence, despite earlier assessments suggesting that acne affects women more often than men [15, 16, 17]. In a cross-sectional epidemiologic investigation, it was shown that 20.8% of girls and 27.9% of males had teenage acne [18]. Conversely, adult acne is more commonly seen in women. Eighty-two percent of the 280 individuals in the research who had adult acne were female. In another study, late-onset acne was identified in 97.3% of women [19].

### Etiopathogenesis

The pathophysiology of acne vulgaris in both adolescent and adult acne is largely determined by abnormal follicular keratinization, which is not always associated with increased sebum production. Other important factors include the existence of Cutibacterium acnes (C. acnes), inflammation, and qualitative differences in sebum composition, such

as squalene, triglycerides, and wax/cholesterol esters. The absence of hypercolonization of *Candida albicans* in acne patients is noteworthy; nonetheless, there are specific variations in *Candida albicans* phylotypes between skin types that are acne-prone and those that are not, such as type IA and IC strains found using multilocus sequence typing [20, 21].

Acne in adolescents and adults is not different in terms of bacterial colonization; however, adult inflammatory lesions may result from long-term activation of the innate immune system by resistant strains of *Candida acnes* [22, 23, 24, 25]. Hormonal considerations may be the cause of adult acne in women more often than in males. While some research indicates that adult female acne is more frequently associated with endocrinologic disorders such as hirsutism, premenstrual aggravation, and androgenetic alopecia. In laboratory testing, only few people have high levels of hyperandrogenism indicators [19, 26]. However, it has been shown that DHEA-S may activate the Th1 system by stimulating the synthesis of IL-2, even at the top limits of normal concentrations [22].

In adult female acne, the prevalence of polycystic ovaries has been reported to be as high as 52%–82%; nevertheless, the hormonal profile is frequently incompatible with PCOS [27]. There is debate over the precise role that hormonal variables play in adult acne. Apart from research findings that indicate elevated amounts of testosterone and Dehydrotestosterone (DHT) in the serum, there are also studies that indicate normal hormonal levels [28, 29]. Among the potential mechanisms are potent androgen metabolites in the skin and an enhanced sensitivity or intracrine metabolism of androgens in the sebaceous glands [28, 30].

An enzyme mechanism found in keratinocytes and sebocytes allows them to create DHT and testosterone. Enzymes that catalyze the metabolism of androgenic hormones, such as 3-beta-OH steroid dehydrogenase, 5-alpha reductase, and 17-OH steroid dehydrogenase, exhibit hyperactivity and aberrant activity. The production of more powerful androgenic hormones (DHT and testosterone) is facilitated by these enzymes by increasing the peripheral turnover of pro-hormones (DHEAS, androstenedione, and testosterone) [31, 32]. DHT is five to ten times more effective than testosterone in this situation [33]. Neuropeptides, stress, and other hormones including melanocortins and Corticotropin-Releasing Hormone (CRH) can also cause the production of sebum [34]. It has been established that histamine, vitamin D, retinoids, and Insulin-like Growth Factor 1 (IGF1) are variables that regulate the production of sebum [35].

### Classification

Within itself, adult acne is divided into three categories. "Persistent acne" is defined as having started throughout adolescence and continuing,

whereas "late-onset acne" refers to acne that initially appears beyond the age of 25. Last but not least, acne that heals and recurs in attacks from childhood to adulthood is referred to as "relapsing acne." [35, 36]. Nonetheless, chronic acne and late-onset acne are the two categories of acne that have been identified in several investigations. According to reports, the most prevalent kind of adult acne, accounting for 73.2%–82% of instances, is persistent acne [37, 26]. Compared to teenage acne, adult acne involves a variety of pathophysiological causes, distinct clinical characteristics, and therapeutic modalities [14].

### Triggering factors

Numerous internal and external variables can impact acne. The pathophysiology of acne is influenced by a number of variables, including diet, premenstrual aggravation, hyperhidrosis, stress, smoking, genetic predisposition, medications, cosmetics as moisturizers, seasonal factors and hormonal problems [26, 37, 38,39, 40]. Protective face masks have recently been linked to facial acne, or maskne. [14].

### Obesity:

Obesity has serious negative effects on one's health, society, and economy on a national and international level. The body mass index (BMI), which is calculated by dividing a person's weight in kilograms by their height in meters squared, is a measure of obesity. Adults with a BMI between 25 and less than 30 kg/m<sup>2</sup> are considered overweight, and those with a BMI between 30 and greater are considered obese [41]. In the USA, obesity is highly prevalent and is still rising [42]. In 2011–2012, obesity afflicted 16.9% of children and 34.9% of adults [43], while in 2015–2016, the prevalence increased to 18.5% in children and 42.4% in adults [44]. By 2030, 48.9% of US people are predicted to be obese, according to predictive modeling [45]. The majority of those affected by this pandemic are underrepresented groups and socially disadvantaged people, such as women, those from lower socioeconomic origins, and members of ethnic and racial minorities [42].

A significant worry for medical professionals who treat patients who are fat or overweight is the increased likelihood of coexisting conditions including diabetes, cancer, and cardiovascular disease (CVD). Finding these comorbidities and the negative effects they cause is a crucial task for primary care doctors. Understanding which diseases have the highest correlations with obesity is crucial for both early detection and treatment of these illnesses as well as for determining which individuals have the best chance of improving their weight. This will make it possible to identify and evaluate risk early on and to put the right actions in place to lower risk and mortality [46].

### Obesity and acne vulgaris

Numerous studies have linked Western diets and insulin resistance to the occurrence and exacerbation of acne [38, 41]. Despite the fact that the majority of research suggested that Body Mass Index (BMI) could play a part in the etiopathogenesis of acne, the findings on this matter are inconsistent [38, 47, 48]. It was discovered in a sizable population-based study that there is a dose-dependent negative relationship between obesity and overweight and acne [49]. Additionally, a different study discovered a negative correlation between the BMI and the number of acne lesions in adult female acne patients [50]. However, other research revealed that being overweight and obese may increase a person's chance of developing acne vulgaris in their teens and early adulthood [47, 51], but some studies were unable to show a connection between acne and BMI [38, 52].

Prior research has demonstrated that insulinotropic milk and dairy products, as well as a glycemic load comprising of hyperglycemic carbohydrates, enhance insulin/IGF-1 signaling while lowering Insulin-like Growth Factor Binding Protein 3 (IGFBP-3) levels [53, 54].

IGFBP-3 binds to the retinoid X receptor to suppress apoptosis and promote cell proliferation [55, 56]. However, IGF-1 reduces the nuclear levels of the transcription factor 1 (FoxO1), the metabolic forkhead box class O, which activates the mammalian Target of Rapamycin Complex 1 (mTORC1). Acne is caused by increased mTORC1 expression, which also increases lipid production, keratinocyte hyperplasia and sebaceous gland hyperproliferation [57].

### Conclusions

Our analysis revealed that there is a correlation between elevated BMI and a higher incidence of acne, offering insight into the link between the two conditions. Future research and meta-analyses are required to validate this finding when additional literatures becomes available.

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