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## The Role of the Skin and Gut Microbiota in Acne Vulgaris: Current Insights and Future Directions - Review

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### Abstract

### Introduction

Acne vulgaris is the most common chronic inflammatory skin disease, affecting up to 85% of young adults. It can lead to scarring and significantly reduce the quality of life. Traditionally, acne pathogenesis has been linked to excess sebum, follicular hyperkeratinisation, *Cutibacterium acnes* colonisation, and inflammation. However, an increasing role has been attributed to changes in the microbiota. Additionally, the gut-skin axis is a modulating factor in acne. Understanding these microbiota interactions offers opportunities for new treatment strategies.

## **Aim of study**

This work aims to evaluate the current evidence regarding the role of the microbiota in the pathogenesis, progression, and treatment of acne vulgaris.

## **Materials and methods**

Articles for this review were retrieved from multiple scientific databases, including PubMed, Google Scholar, and other scientific resources. The search methodology incorporated the terms “acne vulgaris” or “acne” AND “microbiome” or “microbiota” or “gut-skin axis”, along with variations of these terms.

## **Conclusion**

Acne vulgaris is a complex disease. The skin and gut microbiota play a crucial role in its pathogenesis and treatment. Disruption of *Cutibacterium acnes* phylotype diversity, alongside its interactions with *Staphylococcus epidermidis* and *Malassezia*, contributes to its pathogenesis. Modern therapies, like probiotics and bacteriophages, may provide new treatment opportunities, but further research is needed in this area.

## **Keywords**

acne vulgaris, skin microbiome, gut microbiota, gut-skin axis, *Cutibacterium acnes*, dysbiosis

## **Introduction**

Acne vulgaris is the most common chronic inflammatory skin disease. It affects about 9% of the world's population and up to 85% of young adults [1,2]. It is characterised by skin lesions in the form of blackheads, papules, pustules and nodules, mainly located on the face and trunk. These lesions may subside, leaving scars [2,3]. By directly affecting appearance, it is also a frequent cause of reduced quality of life, diminished self-esteem and dissatisfaction with life [4].

The traditional model of acne pathogenesis involves four main factors: overproduction of sebum, hyperkeratinisation of hair follicles, colonisation by *Cutibacterium acnes* and subsequent inflammation. Nowadays, increasing importance is placed on the role of changes in

the microbiota in both the onset and progression of acne. It is believed that not the excess of *Cutibacterium acnes*, but a change in its resident strains underlies the disease. Additionally, the gut-skin axis, which describes the relationship between gut health and skin health, has gained significant attention as a modulating factor in acne. Gut dysbiosis, influenced by factors such as diet, stress and antibiotic use, potentially exacerbates acne severity by promoting systemic and cutaneous inflammation. Understanding the interaction between the microbiota and acne pathogenesis opens up new treatment options that extend beyond traditional therapies, focusing on modulating the microbiota to restore skin and systemic homeostasis.

### **Aim of study**

This study aims to assess the influence of the skin microbiota on the development and progression of acne vulgaris. Modern methods of treatment of this disease were explored, based on the latest scientific reports.

### **Materials and methods**

Articles for this review were retrieved from multiple scientific databases, including PubMed, Google Scholar, and other scientific resources. The search methodology incorporated the terms “acne vulgaris” or “acne” AND “microbiome” or “microbiota” or “gut-skin axis”, along with variations of these terms. The selected articles were limited to those published in English. Moreover, the lists of references of the scientific papers were reviewed to find further relevant works. Publications issued before 2015 and case reports were excluded. The last search was conducted on June 30, 2025.

## **Main Body**

### **1. The Skin and Gut Microbiota**

#### **1.1. Skin Microbiota**

The skin is the largest human organ, with a total surface area, including its appendages, of about 25 m<sup>2</sup> [1,5]. It also serves as a habitat for many microorganisms (bacteria, fungi and viruses) that form a diverse ecosystem, called the microbiome [6,7,8]. The skin microbiota is considered the second-largest microbial community in the human organism after the microbiota of the gastrointestinal tract [9]. Healthy skin is mostly inhabited by commensal microorganisms that support skin homeostasis and prevent colonisation by pathogens [10].

The microbiome's composition depends on many factors, such as body area, diet, environmental factors, antibiotic use and others [1,11]. In seborrhoeic areas (glabella, chest and back), lipophilic bacteria such as *Cutibacterium*, among others, are predominant (Table 1). In moist skin areas (the bends of the elbows and knees, lower buttocks), *Staphylococcus* and *Corynebacteria* are the most common (Table 1). The most prevalent genera in dry areas are *Micrococcus*, *Streptococcus*, *Cutibacterium* and *Corynebacterium* (Table 1) [6,8,11].

The diversity of microorganisms, such as *Cutibacterium acnes* and *Staphylococcus epidermidis* (*S. epidermidis*), and their interactions play a major role in acne formation [12,13]. Dysbiosis in the skin microbiome can disrupt the skin barrier's integrity, contributing to increased transepidermal water loss (TEWL) and altered pH levels [10].

Skin area	Type of typical bacteria
Seborrhoeic areas	<i>Cutibacterium</i>
Moist areas	<i>Staphylococcus, Corynebacteria</i>
Dry areas	<i>Micrococcus, Streptococcus, Cutibacterium and Corynebacterium</i>

Table 1. Skin areas and the characteristic bacterial flora.

## 1.2. Gut Microbiota

It is estimated that approximately  $10^{14}$  microbial cells inhabit the human intestine, making it the largest microbial community in the body. As with the skin, the gastrointestinal microbiome is very diverse, which is important for overall health [1].

The composition of the gut microbiome is individual and varies from person to person. The main bacterial taxa in the gut profile of healthy adults are *Firmicutes*, *Bacteroidetes*, *Actinobacteria*, *Proteobacteria*, *Fusobacteria* and *Verrucomicrobia*, with up to 90% of the microbiota consisting of *Firmicutes* and *Bacteroidetes* [14]. One of the most significant variables in the gut microbiome is the ratio between *Firmicutes* and *Bacteroidetes* genera, as higher levels of *Firmicutes* have been reported in obesity [15].

The interplay between the gut microbiome and the skin is called the gut-skin axis. The gut microbiome exhibits neuroendocrine and immunological functions, making a healthy gut microflora a huge contributor to skin health [2,8]. Metabolites produced by gut bacteria can reach the skin via the bloodstream, affecting epidermal differentiation and skin barrier integrity [16]. It is believed that dysbiosis in the gut can contribute to diseases such as psoriasis, atopic dermatitis and acne [3,17]. Emotional stress can affect gut microbiota composition, leading to systemic and cutaneous inflammation and exacerbation of acne [13].

## **2. Microbiota in Acne Pathogenesis**

### **2.1. *Cutibacterium acnes* – not all strains are equal**

It has long been widely known that *Cutibacterium acnes* (*C. acnes*) is a bacterium associated with acne. It influences the body's immune responses, affects skin physiology by modulating keratinocyte differentiation and influences lipid production in sebocytes [9]. For a long time, it was believed that an overabundance of *C. acnes* bacteria was the cause of acne. However, studies have shown that the density of *C. acnes* does not significantly differ between individuals with acne and healthy controls. The diversity of *C. acnes* phylotypes, however, does differ. It consists of six main phylotypes. These are IA1, IA2, IB, IC, II and III. There are also ten principal ribotypes, from RT1 to RT10 [18]. A predominance of phylotype IA1, especially ribotypes such as RT4 and RT5, has been observed in people with acne. [9,11,12] This phylotype produces significant amounts of substances such as triacylglycerol lipase, porphyrins, and hyaluronate lyase. This results in elevated levels of IFN- $\gamma$  and IL-1, which contribute to increased inflammation. [7,18] In addition, strain IA1 has been shown to have the greatest potential for biofilm production, making it much more difficult to treat [18,19]. On the other hand, other strains of *C. acnes*, including ribotype RT6, appear to be relatively protective and associated with healthy skin [7,10].

### **2.2. Skin microbial interactions**

The skin microbiota is constantly undergoing diverse interactions between its components. *Staphylococcus epidermidis* (*S. epidermidis*) is present on the skin of both healthy individuals and those with acne. The incidence of *S. epidermidis* in acne-prone skin was found to be inversely correlated with the incidence of *P. acnes* [19]. *S. epidermidis* competes with *C. acnes*, inhibits its proliferation and suppresses the inflammation induced. It facilitates the fermentation

of glycerol produced by the skin and by releasing succinic acid, which limits the growth of *C. acnes* [12,19]. In addition, it can inhibit biofilm formation and destroy biofilm through the production of bacteriocins. Maintaining a balance between *S. epidermidis* and *C. acne* appears to be crucial for skin health [18].

Another type of bacteria worth mentioning is *Staphylococcus capitis*. Its strain E12 shows particular activity against *C. acne*, inhibiting its growth while sparing other bacteria. According to some sources, its activity against *C. acnes* is even greater than that of traditional antibiotics [18].

Another microorganism that may be associated with the pathogenesis of acne is *Malassezia*, the most prevalent fungus in the human skin. As it is unable to synthesise fatty acids, *Malassezia* relies on external sources and is concentrated in sebaceous regions [18]. Its lipase activity is estimated to be 100 times that of *C. acne*. As such, attention is being drawn to its potential role in acne. While its precise function remains unclear, research suggests a possible role in treatment-resistant acne [7,9,12].

### **2.3. Impact of the gut microbiome and diet on acne**

The role of diet in acne development remains a subject of debate. Diet is one of the main factors influencing the composition of the gut microbiome. The Western diet is characterised by a high intake of highly processed foods, saturated fats and sugars. Such a diet disrupts the balance of healthy intestinal microbiota, leading to increased inflammation and negatively affecting skin condition [2,12,17]. According to the American guidelines, there is insufficient evidence to recommend diets such as a low dairy diet, a low whey diet and chocolate restriction to patients for the treatment of acne [20]. A diet that seems to have a positive effect on acne patients is

plant-based. A healthy plant-based diet low in processed foods is rich in antioxidants and fibre, which has a positive impact on the gut microbiota and reduces inflammation [13,16,21]. Studies are showing a correlation between gut dysbiosis and acne vulgaris. Several studies have shown that the gut microbiota of people with acne differs from that of healthy people, showing lower diversity and disturbed microbial ratios [14,15]. However, more research is needed to understand these correlations in detail.

### **3. Acne Treatment and the Microbiota**

#### **3.1. Recommended treatments**

Current recommended treatments for acne include topical therapy for mild acne and systemic therapy in combination with topical therapy for moderate to severe acne. Among the topical medications recommended are antibiotics, benzoyl peroxide (BPO), retinoids and azelaic acid. For more severe lesions, general treatment in the form of antibiotics or isotretinoin is recommended. The use of antibiotics in monotherapy is not recommended [20,22].

The European Academy of Dermatology and Venereology (EADV) guidelines distinguish various recommendations for different types of acne. For comedonal acne (a subtype of acne in which most lesions are comedones), topical retinoids are the recommended treatment. Topical antibiotics and systemic treatment are not recommended. As for papulopustular acne, in the case of mild to moderate lesions, topical treatment is recommended and in severe cases, isotretinoin. For conglobate acne, the guidelines recommend isotretinoin in monotherapy [22].

In contrast, the American guidelines depend on the severity of the acne. For mild lesions, multimodal topical treatment with topical antibiotic & BPO, topical retinoid & BPO, or topical antibiotic & retinoid is recommended. For moderate to severe acne, in addition to topical



therapy, systemic therapy is recommended. The systemic therapy includes doxycycline, isotretinoin or intralesional corticosteroids if there is a risk of scarring of large lesions [20].

### **3.2. Traditional treatments – impact on microbial balance**

Among the recommended acne treatments, effects on the microbiota have been shown for antibiotics, BPO and isotretinoin [23]. Supramolecular salicylic acid (SSA) may also influence the skin microbiome [10,19].

Antibiotics are widely used in the treatment of acne. Despite their high efficacy, their use also has its drawbacks due to the production of antibiotic resistance and their non-selective effect on the skin and gut microbiota [24].

Topical antibiotics have antibacterial as well as anti-inflammatory effects. They are particularly effective in inflammatory lesions. In combination with topical retinoids or BPOs, they are one of the main treatment options [13,20]. The systemic antibiotic recommended as a first-line treatment by both the EADV and American guidelines is doxycycline. Other recommended antibiotics include Minocycline, Sarecycline, and Lyme cycline [20,22]. European guidelines recommend limiting the duration of systemic antibiotic therapy to three months.

In addition to the increasing resistance of bacteria, another concern raised is their impact on the skin and gut microbiota. The use of antibiotics, such as doxycycline or minocycline, appears to reduce the dominance of *C. acnes* and increase the diversity of the skin microbiota [10,19]. On the other hand, however, oral antibiotics negatively affect the intestinal flora, reducing the population of *Lactobacillus* and *Bifidobacteria* [3,19].

Benzoyl peroxide is another of the essential drugs in the treatment of acne. Unlike antibiotics, it does not cause bacterial resistance. To date, no *C. acnes* strains have been identified that demonstrate resistance to BPO [20]. Studies have shown that it is equally effective against both

antibiotic-sensitive and antibiotic-resistant bacteria [9,23]. Furthermore, when combined with antibiotics, they act synergistically, increasing their penetration and concentration in lesions [3]. Adding BPO to antibiotic therapy can reduce the development of bacterial resistance [20,25]. Several studies have also reported the inhibitory potential of BPO in biofilm formation by *C. acne* [25]. It also significantly increases the diversity of the skin's microflora [10]. Despite its many benefits, its use is limited by side effects such as dryness, erythema, pain, flaking, irritation, fabric staining and bleaching. In cases of poor tolerance, products with lower concentrations of BPO or reduced frequency of application may be recommended [13,20].

Isotretinoin is the primary drug for the systemic treatment of severe acne. Although it has no direct antimicrobial properties, it acts on the microbiome indirectly by reducing sebum production, decreasing inflammation and inhibiting comedogenesis by normalising follicular keratinisation. In this way, it reduces the population of *C. acnes* while increasing the population of other bacterial genera such as *Streptococcaceae*, *Pasteurellaceae*, and *Corynebacteriaceae* and simultaneously increasing the diversity of the microbiome [9,10,19].

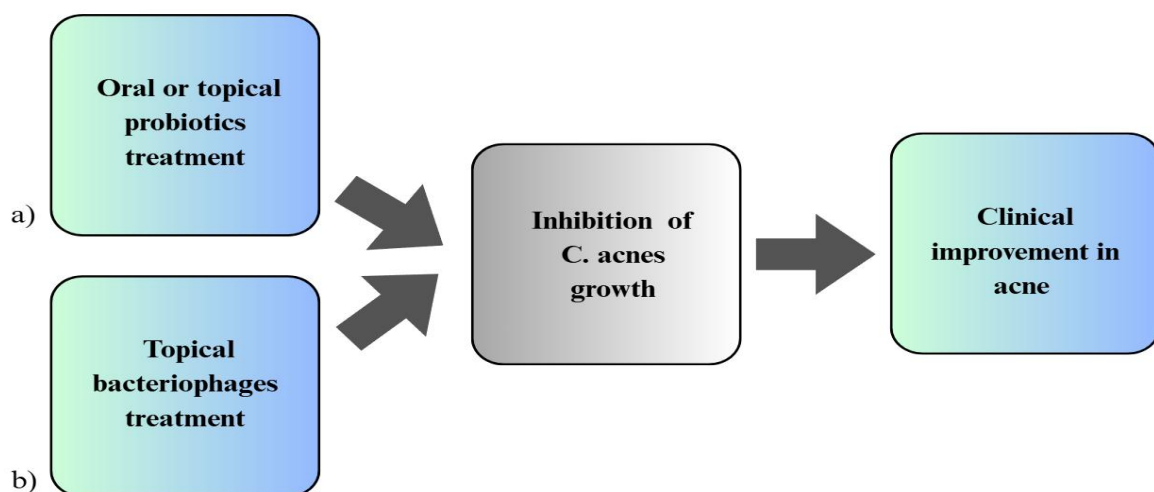
#### **4. Future Perspectives**

In addition to traditional methods, new promising therapies focusing on the skin microbiome continue to emerge. Examples of such acne therapies include probiotics and phage therapy.

Probiotics are live microorganisms that, when introduced into the body, can influence its microbiome by regulating dysbiosis. The probiotics used in acne can be divided into oral and topical. Probiotic treatment is considered safe and with very limited side effects, especially compared to traditional therapy [3,8]. The most common strains used as probiotics are *Lactobacillus* and *Bifidobacterium*. They can inhibit the growth of *C. acnes*, reducing sebum secretion and enhancing the skin's immune response, leading to clinical improvement in acne (Figure 1) [3,9,14,26]. However, many other types of bacteria are also being studied. Some

probiotics, such as *Streptococcus thermophilus*, among others, increase the production of ceramides, thereby having an anti-inflammatory effect and strengthening the skin barrier against pathogens [2,3,8]. An interesting possibility for the use of probiotics is to support antibiotic therapy. Several studies have shown the superior efficacy of such a combination therapy for acne relative to antibiotics alone [2,14]. Furthermore, such treatment could reduce the negative effects of antibiotics on the microbiome. Despite being a promising therapeutic option, there is little research to date regarding the effects of probiotics on acne-prone skin [2,14].

Bacteriophages, also known as phages, are viruses that infect and kill bacteria. They offer an innovative new way to treat acne by directly targeting *C. acnes* [13]. In addition to simply reducing *C. acnes* populations, they may also be a potential solution to the problem of increasing antibiotic resistance. They can be used to restore the susceptibility of resistant *C. acnes* strains, by spreading susceptibility genes among the bacteria [23,27]. In addition, they can penetrate biofilms [27]. Their use appears to be a promising therapeutic option, but further clinical studies are needed to confirm this phenomenon (Figure 1) [13].



*Figure 1. Future perspectives on treatment. a) Oral or topical probiotics treatment. b) Topical bacteriophages treatment.*

## **Conclusion**

Acne vulgaris is a multifactorial disease with significant physical and mental health implications, and the role of the microbiota in its pathogenesis, progression and treatment is increasingly recognised. The traditional view of *Cutibacterium acnes* overgrowth as a causative factor in acne has changed, emphasising the importance of disrupting diversity between its different phylotypes, in particular the predominance of phylotype IA1 with ribotypes RT4 and RT5. Moreover, the interaction between *C. acnes* and other skin microbiota, such as *Staphylococcus epidermidis* and *Malassezia*, is receiving increasing attention. Furthermore, the gut microbiota, through its role in immune modulation and systemic inflammation, appears to be an important factor influencing skin health through the gut-skin axis. Among current acne treatments, antibiotics, benzoyl peroxide and isotretinoin have a proven impact on the diversity of the skin microbiota. With the increasing resistance of bacteria to antibiotics and the growing importance of microbiome diversity, new therapies such as probiotics and bacteriophage therapy are emerging. These focus on reducing the population of pathogenic *C. acnes* strains on the skin. In the future, they may become a promising alternative or complement to traditional treatments, while offering fewer side effects. Although these therapeutic options are promising, there is still not enough research on the effect of the microbiota on acne. Further clinical studies are required to determine the most effective strategies for the treatment of acne.

## **Disclosure**

### **Author's contribution**

Conceptualization: Monika Grochowska-Rak, Paweł Racisz

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### **Conflict Of Interest**

The authors declare no conflict of interest.

**All authors have read and agreed with the published version of the manuscript.**

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