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INTESTINAL DYSBIOSIS AND ACNE: INVESTIGATING THE INTERCONNECTION BETWEEN THE INTESTINAL MICROBIOME AND SKIN HEALTH

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RESUMO

Introduction: The balance between intestinal microbiota and skin is essential for health, including acne prevention and understanding these interactions is role for developing targeted acne therapies. **Objective:** This literature review aims to elucidate the interconnection between the intestinal microbiome and health, focusing on the etiology and intensity of acne, and examining modifications in intestinal microbiota and their effect on acne pathogenesis. **Results and discussion:** Intestinal dysbiosis, Western diet, *Cutibacterium* acnes, and microbiota-host interactions are key elements in acne pathogenesis, affecting sebum production and triggering inflammatory responses. The diet, especially the Western one, influences both intestinal microbiota and acne, highlighting the relationship between gastrointestinal and skin health. *C. acnes* plays a essential role in acne by stimulating sebum production and triggering inflammatory responses in the skin. **Conclusion:** Intestinal microbiome, skin microbiota, diet, dysbiosis, and *C. acnes* are key research areas for understanding and treating acne, emphasizing the importance of diet in skin health. Dietary interventions may benefit acne patients, leading to more effective and personalized therapeutic approaches.

Keywords: Dysbiosis; *Cutibacterium* acnes; Diet; Skin microbiota.

ABSTRACT

Introdução: O equilíbrio entre a microbiota intestinal e a pele é essencial para a saúde, incluindo a prevenção da acne e compreender essas interações é fundamental para o desenvolvimento de terapias direcionadas à acne. **Objetivo:** Esta revisão de literatura busca elucidar a interconexão entre o microbioma intestinal e a saúde, com foco na etiologia e intensidade da acne, além de examinar as modificações na microbiota intestinal e seu efeito na patogênese da acne. **Resultados e Discussão:** Disbiose intestinal, dieta ocidental, *Cutibacterium* acnes e interações microbiota-hospedeiro são elementos fundamentais na patogênese da acne, afetando a produção de sebo e desencadeando respostas inflamatórias. A dieta, especialmente a ocidental, influencia tanto a microbiota intestinal quanto a acne, destacando-se a relação

entre a saúde gastrointestinal e especificamente. *C. acnes* desempenha um papel fundamental na acne, estimulando a produção de sebo e desencadeando respostas inflamatórias na pele. **Conclusão:** Microbioma intestinal, microbiota da pele, dieta, disbiose e *C. acnes* são áreas-chave de pesquisa para entender e tratar a acne, ressaltando a importância da dieta na saúde da pele. Intervenções dietéticas podem ser benéficas para pacientes com acne, levando a abordagens terapêuticas mais eficazes e personalizadas.

Palavras-chaves: Disbiose, *Cutibacterium acnes*, dieta, microbiota da pele.

1 INTRODUÇÃO

The adult human intestine harbors approximately 10^{14} bacterial cells, comprising over 1000 distinct bacterial species. The normal intestinal microflora includes as many beneficial bacteria as those environmental to the host. Under safe conditions, there is a mutual interaction and regulation between the host and the intestinal microbiota, maintaining a homeostatic balance of bacteria that promotes gastrointestinal health, while preventing the overgrowth of bacteria with potential pathogenicity (BIEN et al., 2013; LI et al., 2015). However, any alteration in microbial composition leading to a significant imbalance between beneficial and pathogenic environmental bacteria can render the intestine susceptible to pathogenic disturbances and changes in the intestinal microbiota. This imbalance, known as “dysbiosis,” is characterized by a disruption in the homeostasis of the intestinal microbiota, resulting in changes in bacterial flora, functional composition, metabolic activities, or local distribution (BIEN et al., 2013; SHANAHAN, 2013; KNIGHTS et al., 2013).

Acne is an inflammatory condition that affects the pilosebaceous unit and can affect up to 90% of adolescents. Severe forms of acne can lead to disfigurement and scarring, resulting in low self-esteem, difficulties in social interaction, and psychological distress (SALVUCCI, 2016). Increased sebum production, skin-mediated inflammation, and follicular keratinization of pilosebaceous ducts are believed to contribute to the development of acne. Although colonization by *Cutibacterium acnes* (*C. acnes* - formerly known as *Propionibacterium acnes*) is recognized in acne patients, its role is not fully understood due to its ubiquitous presence in the sebaceous layers of healthy skin since puberty. As part of the growing interest in the human microbiome, studies are beginning to elucidate the role of skin microorganisms in health and disease, including acne (LEE et al., 2019; HOOPER et al., 2012).

The pathogenesis of acne is influenced by several factors, including excessive sebum secretion, follicular hyperkeratinization, inflammatory responses, and the role of bacteria (STEENSEL, 2019). These factors are interconnected; for instance, increased sebum production promotes the proliferation of many lipophilic bacteria, such as *C. acnes*. These bacteria produce free fatty acids through the enzyme lipase, which stimulates cells to secrete IL-1 β and other cytokines, triggering inflammatory responses and contributing to acne formation (OCHSENDORF, 2019). Although previous studies have primarily attributed acne to the presence of *C. acnes*, recent research indicates that dysbiosis of the facial microbiota plays a significant role in acne pathogenesis (RAMASAMY et al., 2019; DRÉNO, 2020).

Studies suggest that dietary factors, such as the Western diet, may influence the development of acne. A typical Western diet, rich in foods with a complex mixture of fats (e.g., red meat), high glycemic index, and dairy, can exacerbate acne by increasing levels of insulin-like growth factor-1 (IGF-1) and insulin (ADEBAMOWO et al., 2008; AGAMIA et al., 2016; KIM et al., 2017). Furthermore, diet can shape the intestinal microbiota. There is a significant body of evidence indicating that a Western diet low in

fiber and high in fat can cause fundamental changes in the intestinal microbiota, leading to metabolic and inflammatory skin diseases (BOWE et al., 2014).

Considering the relationship between the intestinal microbiome and skin health, we hypothesize that intestinal dysbiosis may play a substantial role in the manifestation and severity of acne. In this context, our aim is to elucidate, through a comprehensive literature review, the interconnection between the intestinal microbiome and skin health, focusing on the etiology and intensity of acne, as well as examining modifications in the composition of the intestinal microbiota and their potential effect on acne pathogenesis.

2 METHODOLOGY

The methodology employed in this bibliographic research integrates qualitative and quantitative approaches to examine the implications of changes in the intestinal microbiome on acne, investigating the potential link between intestinal dysbiosis and skin problems. Data collection involved searches across various scientific platforms such as PubMed, Web of Science, SCIELO, and relevant electronic journals. In the qualitative analysis, we thoroughly reviewed studies, reviews, and critical analyses on the role of the intestinal microbiome in skin health. For the quantitative research, we utilized specific keyword combinations to gather data on intestinal dysbiosis and acne, such as “*intestinal dysbiosis*,” “*intestinal dysbiosis and acne*,” “*intestinal dysbiosis and dermatology*,” “*intestinal microbiome*,” “*intestinal microbiome and microbiota*,” while evaluating articles for their relevance and quality. The selection of articles considered established inclusion criteria, prioritizing pertinent and high-quality works contributing to understanding the relationship between intestinal dysbiosis and skin health.

3 RESULTS AND DISCUSSION

Based on the results of the research parameterized with keywords found in PubMed, we observed a significant trend in the number of documents related to intestinal dysbiosis over the years (Fig. 1). From 2014 to 2024, there was a gradual increase in the total number of documents, reaching a peak in 2021, with 29,085 documents, followed by a decrease in 2022 and 2023, and a more significant reduction in 2024, with only 8,136 documents. Regarding the relationship between intestinal dysbiosis and acne, we observed a limited number of documents over the years, with only two documents in 2020 and 2021, followed by a reduction to one document in 2022 and 2024, and no documents in other years.

Regarding intestinal dysbiosis and dermatology, there was a steady increase in the number of documents from 2015, peaking in 2022 with four documents, followed by a reduction to one document in 2024. Concerning intestinal microbiota, there was a steady increase in the number of documents until 2021, with a peak of 11 documents in that year. However, a significant decrease was observed in 2024, with only two documents.

Finally, the relationship between intestinal dysbiosis and microbiota showed variation over the years, with an increase in the number of documents until 2023, reaching a peak of four documents in that year, followed by a reduction to no documents in 2024. These results suggest a growing attention to the relationship between intestinal dysbiosis and various aspects of health, such as acne, dermatology, and intestinal microbiota, although there are variations in interest over the years, indicating the need for more research to better understand these relationships and their impact on human health.

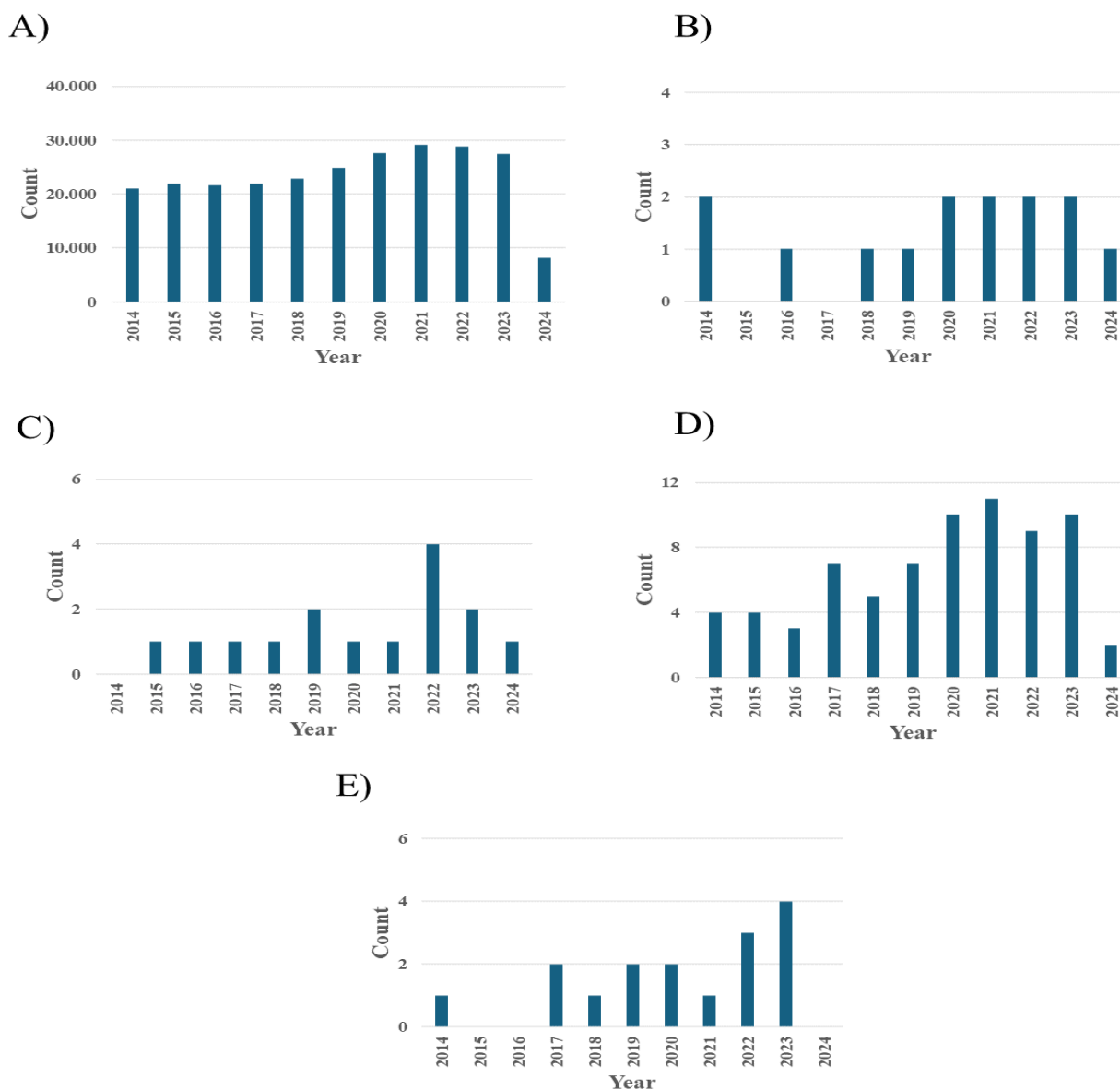


Figure 1: Results of the research parameterized with keywords found in PubMed: A) Intestinal dysbiosis, B) Intestinal dysbiosis and acne, C) Intestinal dysbiosis and dermatology, D) Intestinal microbiota, and E) Intestinal dysbiosis and microbiota.

3.1 Intestinal Microbiota

The intestinal microbiome is a complex system composed of about 100 trillion organisms and their genetic material (LAWLEY & WALKER, 2013). The various components of this ecosystem are called microbiota, including bacteria, viruses such as phages, fungi, eukaryotes, and archaea (LAWLEY & WALKER, 2013; AMON SANDERSON, 2016; THURSBY & JUGE, 2017). Most research so far has focused on the bacterial microbiota and its role in the microbiome, due to its predominance over other microorganisms (LAWLEY & WALKER, 2013).

The composition of the intestinal microbiome undergoes changes from birth and is influenced by various factors such as diet (WEXLER & GOODMAN, 2017), environment (BUFORD, 2017), age

(BUFORD, 2017), and medications. Therefore, each individual has a unique intestinal microbiome, akin to a fingerprint (SHAHINAS, et al., 2012; MCBURNEY, et al., 2019). However, there are composition characteristics that can distinguish a “healthy” microbiome from an “unhealthy” one, mainly related to patterns among commensal bacteria (LAWLEY & WALKER, 2013).

Bacteria in the intestinal microbiome can be defined at different levels of detail using taxonomy (RINNINELLA et al., 2019). The kingdom of bacteria is described through taxonomic units such as phylum, class, order, family, genus, species, and strain. Although these units may vary depending on the study, it is common to analyze the bacterial microbiome at the phylum level. The most prevalent phyla include Firmicutes, Bacteroidetes, Actinobacteria, and Proteobacteria, as these four phyla contain up to 99% of the intestinal microbiota in healthy adults (LAWLEY & WALKER, 2013). Firmicutes and Bacteroidetes, in particular, appear to be the most predominantly collectively, representing up to 90% (AMON SANDERSON, 2016; RINNINELLA et al., 2019).

Changes in the microbiota can be triggered by exposure to various environmental factors, including diet, toxins, medications, and pathogens. Among these factors, enteric pathogens present the greatest potential to cause microbial dysbiosis (KAMADA et al., 2013). This is observed in experimental animal models, where foodborne viral pathogens can trigger local and systemic inflammation, resulting in altered microbiota composition and barrier function. This can serve as a mechanism for the development of autoimmunity, as evidenced in type 1 diabetes, involving T cell-mediated destruction of insulin-producing pancreatic β cells (KAMADA et al., 2013; TANOUE, 2010; WEN, 2008).

The state of symbiosis is attributed to the intestine, as it is inhabited by a diverse community of microorganisms, and the host tolerates these commensal bacteria and associated benign antigens. The immune system's ability to develop tolerance to these benign antigens is acquired over time, through ontogeny and attenuation of inflammatory responses (MAHMUD et al., 2022). This balance is sustained through immunities, which prevent the establishment of an exacerbated inflammatory response. This inflammatory response is triggered by the release of a variety of proteins, called cytokines, whose production is induced by the presence of microorganisms or other antigens (MATSUOKA & KANAI, 2015).

Recent advances in sequencing methodologies have enabled autonomous analysis of the composition of the intestinal microbiota, revealing that imbalance therein, termed dysbiosis, plays a significant role in the pathophysiology of various diseases, in detriment to the presence of specific pathogens (MATSUOKA & KANAI, 2015)). Dysbiosis is characterized by an imbalance between the population of bacteria with protective properties and those with aggressive potential, resulting in a condition where the gastrointestinal tract becomes more susceptible to toxin proliferation, triggering of inflammatory processes, and suppression of the immune system (PEREIRA & FERRAZ, 2017).

The documentation of historical dysbiosis has relied on classical microbiological techniques and the ability to cultivate pure isolates for identification and classification, which is limited to “cultivable” microorganisms. However, the advent of high-throughput DNA-based pyrosequencing technology enables the classification of bacteria and archaea through individual sequences of 16S rRNA directly from human samples, usually of fecal origin, without a need for culture. This approach offers a rapid and detailed means of profiling complex communities of microorganisms. Since its first application, it has become evident that the composition of the intestinal microbiota varies significantly among individuals (MAHMUD et al., 2022).

The intestine hosts as many protective bacteria as some that may be atmospheric to the host (Fig. 2). In secure individuals under normal conditions, there is an interaction and cross-regulation between the host and the intestinal microbiota, resulting in a homeostatic balance of bacteria that keep the gastrointestinal tract healthy and protected from the overgrowth of potentially pathogenic bacteria. The microbiota establishes a commensal relationship with the host, where bacteria thrive in the rich environment of the intestine, while the host benefits from the multiple functions provided by these bacteria.

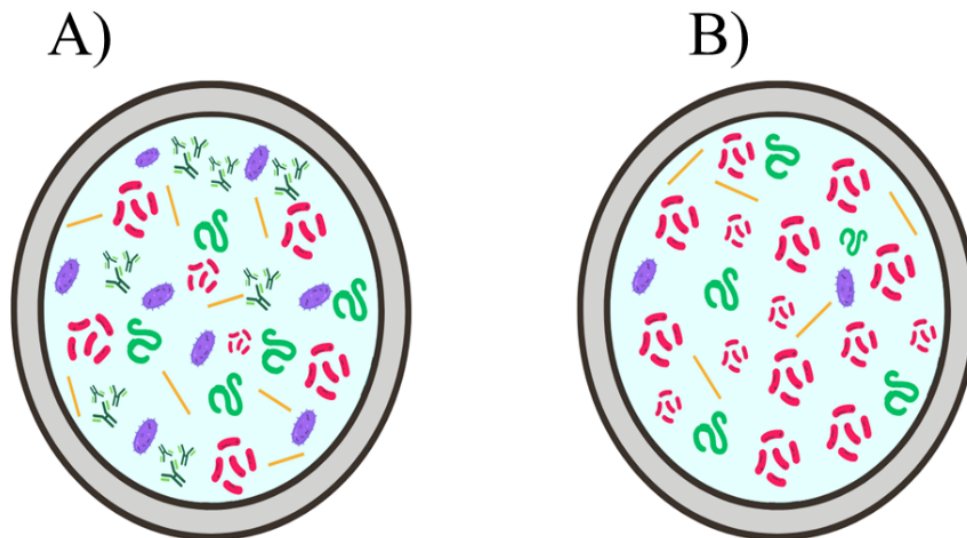


Figure 2. Normal and Dysbiotic Intestinal Microbiota: A) In healthy individuals, the intestines are colonized by a wide variety of bacteria, totaling more than 1000 species. In this state, there is a homeostatic balance between commensal bacteria and potentially pathogenic ones, resulting in an intestinal tract that does not exhibit excessive growth of pathogenic bacteria. B) When intestinal bacterial homeostasis is disrupted, dysbiosis occurs. This is characterized by an imbalance in bacterial composition, alterations in bacterial metabolic activities, or changes in bacterial distribution in the intestine. The three main types of dysbiosis are: 1) Loss of beneficial bacteria, 2) Overgrowth of potentially pathogenic bacteria, and 3) Reduction in overall bacterial diversity.

3.2 Skin Microbiota

The human skin, covering an area of approximately 2 m² in adults, constitutes the largest organ of the body and represents the first line of defense against external agents. Therefore, skin microbiota plays a fundamental role in human health, and dysbiosis is believed to cause or exacerbate skin diseases (DRÉNO et al., 2020). Advances in sequencing technology, such as sequencing of the 16S ribosomal RNA (16S rRNA) gene, have provided important insights into the human microbiome.

The composition of the microbiota exhibits significant variability among individuals, influenced by genetic, environmental, and behavioral factors. Differences in sebaceous gland density, skin moisture, exposure to external agents such as chemicals and cosmetics, as well as diet, can affect the composition and microbial diversity of the skin. This diversity has substantial implications for skin health and the development of specific conditions such as acne, dermatitis, and eczema (GRICE et al., 2009).

The colonization of the human body by microorganisms is not uniform, as each region has its own microbiota characterized by microorganisms with different properties. This microbiota can be divided into two main groups: transient, composed of microorganisms that remain in the body for a short period without establishing significant colonization, and resident, formed by microorganisms that colonize the body in

symbiosis with the host, maintaining a steady presence (SIVIERI et al., 2017).

Despite their potential to cause disease, skin microorganisms also play beneficial roles in health. In addition to protecting against pathogens, they assist in maintaining the skin pH balance, producing antimicrobial substances, and modulating the local immune response. This complex interaction between microorganisms and the skin is crucial for effective homeostasis and the integrity of the local immune system (GRICE et al., 2009).

As the primary inhabitants of the skin, bacteria have been the most studied components of cutaneous microbiota. Most commensal skin bacteria are categorized into the following four phyla: Actinobacteria (e.g., *Corynebacterineae*, *Propionibacterineae*), Proteobacteria, Firmicutes (e.g., *Staphylococcaceae*), and Bacteroidetes (DRENO et al., 2017). Bacterial composition varies from person to person and also according to the body region (LEYDEN et al., 1975; MARPLES, 1982; GIACOMONI et al., 2009). Additionally, environmental factors such as soap use, cosmetics, antibiotics, occupation, temperature, humidity, and exposure to ultraviolet radiation (FAERGEMANN & LARKO, 1987) also influence microbial colonization (FIERER et al., 2008; MCBRIDE et al., 1977).

3.3 Skin Microbiota and Acne

A healthy cutaneous microbiome is characterized by a balanced diversity of microorganisms that interact harmoniously with the host. However, hormonal changes, genetic predisposition, diet, lifestyle, and the use of described products can disrupt this balance, resulting in changes in the composition of specific microbiota and increasing the risk of acne development (SANFORD, et al., 2013). Acne is one of the most common dermatological conditions and affects millions of people worldwide. Although previously believed to be predominantly caused by factors such as sebum overproduction, pore clogging, and inflammation, research has emphasized the crucial role of skin microbiota in acne pathogenesis (COATES, et al., 2019).

Acne is a complex condition influenced by various factors, including skin microbiota. Excessive sebum production, follicular interference, and inflammation are key elements in its origin, and certain skin microorganisms may contribute to these processes. Some microorganisms can stimulate sebum production or trigger inflammatory responses, exacerbating acne. These interactions between skin microbiota and skin physiological processes have a significant impact on the development and severity of acne (SANMARTÍN et al., 2023).

The pathogenesis of acne is attributed to four primary factors: excessive sebum production, *C. acnes* (formerly known as *Propionibacterium acnes*) overgrowth, hyperkeratinization of pilosebaceous follicles, and inflammatory mechanisms (KIRCIK, 2016; ZAENGLEIN, et al., 2016). Excess sebum production results from increased androgen hormone activity and insulin-like growth factor 1 (IGF-1) (KUCHARSKA, et al., 2016). Insulin-like growth factor 1 (IGF-1) has been shown to reduce nuclear levels of forkhead box O1 (FoxO1) transcription factor, leading to mammalian target of rapamycin complex 1 (mTORC1) activation (MELNIK, 2016) (Fig. 3).

In the context of acne, imbalances in the microbiota can contribute to the development and worsening of the condition. Thus, better understanding the interactions between specific microbiota and acne is essential for the development of more effective and targeted therapeutic strategies aiming to restore microbiological balance and promote healthy skin (SANTANA, 2023). *C. acnes* is the primary occupant of

the pilosebaceous unit, representing up to 90% of the microbiota in sebum-rich areas such as the scalp, face, chest, and back (GRICE, et al., 2009). The scalp and face have the highest density of *C. acnes*, followed by the upper limbs and trunk, with the lower limbs presenting less *C. acnes* (GRICE, et al., 2009; PATEL, et al., 2009). The abundance of *C. acnes* also changes with age. *C. acnes* is sparse on the skin in childhood, gradually increases from puberty to adulthood, and decreases after the age of 50.

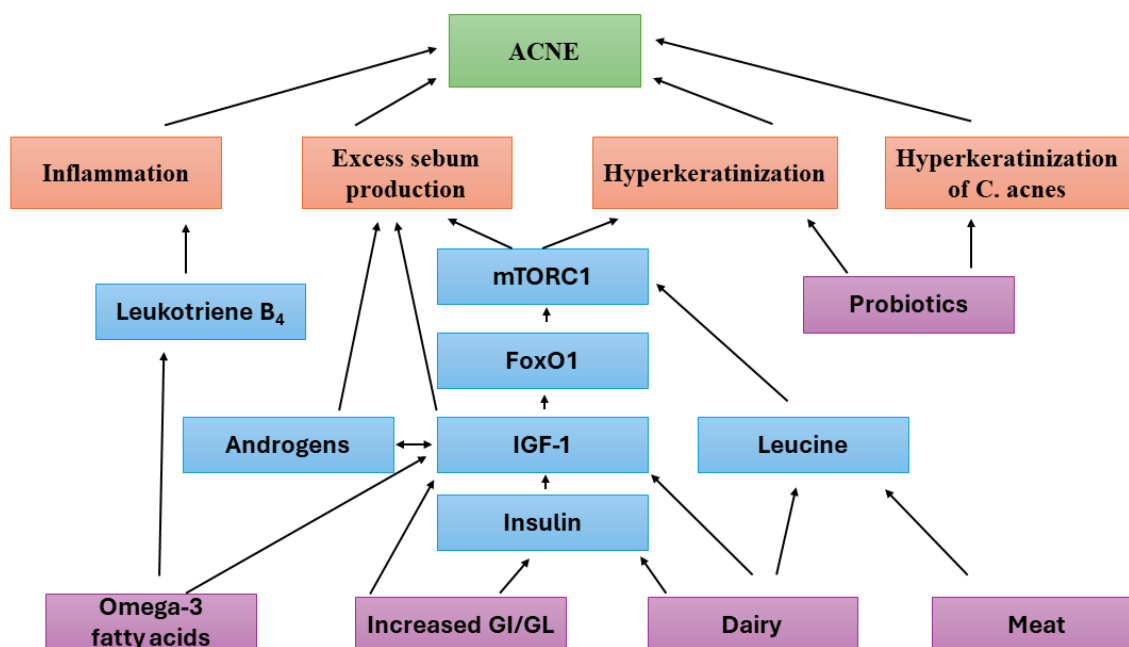


Figure 3: The pathogenesis of acne is related to four main factors: excess sebum production, hyperproliferation of *Cutibacterium acnes* bacteria (formerly known as *Propionibacterium acnes*), hyperkeratinization of pilosebaceous follicles, and inflammatory processes.

3.4 Acne Classifications

Acne is divided into two main types: non-inflammatory acne and inflammatory acne. Non-inflammatory acne occurs in the absence of inflammation, while inflammatory acne is categorized into five grades, taking into account the intensity, quantity, and characteristics of the lesions (PIMENTEL, 2011):

a) Grade 1 Acne (Mild): Characterized mainly by comedones and a few papules and pustules. Generally, there is not much evident inflammation (BARROS et al., 2020).

b) Grade 2 Acne (Moderate): More inflammatory lesions are present, such as papules and pustules. Inflammation is more prominent, and there may be a greater quantity of comedones (BARROS et al., 2020).

c) Grade 3 Acne (Moderately Severe): In addition to the lesions present in previous grades, nodules may occur. Inflammation is more significant, and there is a considerable quantity of lesions (BARROS et al., 2020).

d) Grade 4 Acne (Severe): At this stage, there is a substantial amount of inflammatory lesions, including nodules and cysts. Inflammation is severe and can lead to scarring (BARROS et al., 2020).

e) Grade 5 Acne (Very Severe): This is the most severe stage of acne, with a large quantity of inflammatory lesions, including deep nodules and cysts. Inflammation is intense, and scarring is common (BARROS et al., 2020).

3.5 Cutibacterium Acnes: The Acne Culprit

C. acnes is a Gram-positive bacterium that is part of the normal flora of the skin, oral cavity, large intestine, conjunctiva, and external auditory canal (ADEBAMOWO et al., 2008). Although primarily recognized for its role in acne, *C. acnes* can also trigger a range of postoperative and device-associated infections, such as bone and prosthesis infections (BPI) (AGAMIA et al., 2016), as well as infections related to cerebrospinal fluid shunts. This commensal bacterium can transform into an opportunistic pathogen, although the underlying processes are not fully understood. Infections by *C. acnes* often have a prolonged incubation period (occurring 3 to 24 months or more after the installation of the medical device) (KIM et al., 2017).

When hair follicles are obstructed, *C. acnes* finds a conducive environment for its routine. This bacterium feeds on sebum, a nutrient-rich substrate, and dead epithelial cells present in the follicle. This metabolic process results in the production of free fatty acids as byproducts, which can damage the follicle, triggering a local inflammatory response (COSTA et al., 2008). The interaction between *C. acnes* and the immune system plays a fundamental role in the pathogenesis of acne, where the release of pro-inflammatory cytokines is essential to initiate and maintain the inflammatory response in acne lesions. This process involves the activation of immune cells such as macrophages and lymphocytes in response to the presence of bacteria on the skin, resulting in the production of pro-inflammatory mediators that exacerbate local inflammation and symptoms for the formation and progression of acne lesions (MIAS et al., 2023).

Studies have shown that metagenomics has brought new insights into the differences in *C. acnes* strain level in health and disease, particularly in acne. Johnson et al. (2016) identified that strains associated with acne produced more porphyrin, a substance that generates reactive oxygen species (ROS) and can trigger inflammation in keratinocytes (MARCASON, 2010). Tomida et al. (2013) compared complete DNA sequences of *C. acnes* strains and found that the non-essential genomic sector of acne-related strains carries additional virulence genes compared to healthy strains. Strains associated with acne are also known to induce an inflammatory response in sebocytes, keratinocytes, and peripheral blood mononuclear cells, while non-acne strains do not exhibit this effect (MORALES et al., 2016; PITT, 2016).

The clinical manifestations of low-grade *C. acnes* infection are generally nonspecific, including chronic pain, recently observed fever, and an increase in systemic biological markers of inflammation (KIRBY, 2018). These infections do not induce a clear inflammatory response, indicating that this microorganism may evade the immune system. A study conducted by Hudek et al. demonstrated that *C. acnes* was immunohistochemically detected persisting intracellularly within stromal cells and macrophages in the shoulders' joints of asymptomatic patients (SPULBER et al., 2009).

3.6 Interaction between Gut - Acne - Diet

Recent evidence shows a link between dietary factors, especially those found in the Western diet, and the development of acne. Frequent consumption of specific foods from the Western diet, such as high-fat red meats, high glycemic index foods, and dairy, can worsen acne by increasing levels of insulin-like growth factor 1 (IGF-1) and insulin (ADEBAMOWO et al., 2008; AGAMIA et al., 2016; KIM et al., 2017). Additionally, this diet influences the composition of the gut microbiota. Studies indicate that a Western diet low in fiber and high in fat alters the gut microbiota in a way that promotes metabolic and inflammatory conditions in the skin (BOWE et al., 2014).

The gut microbiota plays a crucial role in regulating the host's immune system, contributing to tolerance to dietary and environmental antigens as well as defense against pathogens (SALEM et al., 2018). In recent years, there has been growing interest in the role of environmental factors, particularly the Western diet, in the pathogenesis of acne. This diet, characterized by the consumption of dairy, organic carbohydrates, chocolate, and saturated fats, is associated with a higher incidence of acne, activating nutrient-derived metabolic signals (MELNIK, 2015; MARCASON, 2010). Evidence also suggests that the gut microbiota influenced by the Western diet contributes to inflammatory skin conditions. Diets high in fat have been associated with reduced diversity of the gut microbiota and increased systemic inflammation, impairing epithelial integrity and colonic barrier function, and increasing immunity to pro-inflammatory cytokines (DENG et al., 2018; MORALES et al., 2016).

Current dietary trends can be further explored by outlining the fundamental principles of each: Mediterranean diets, such as the Pioppi diet, promote low carbohydrate intake, high consumption of oils and vegetables, and a moderate amount of fish; the ketogenic diet is characterized by high fat consumption and significant carbohydrate restriction; while the Paleo diet generally emphasizes high meat consumption and substantial reduction of dairy and sugars (PITT, 2016; KIRBY, 2018; SPULBER et al., 2009).

Comparatively, the typical Western diet has a higher ratio of omega-6 to omega-3 fatty acids, contrasting with hunter-gatherer diets and other similar dietary patterns, which are rich in fish, wild meats, and plants (ADEBAMOWO et al., 2008). Some diets, like the Mediterranean, encourage fish and olive oil intake to ensure adequate intake of essential vitamins (LOGAN, 2003). On the other hand, vegetarian and vegan diets involve a significant or complete reduction in the intake of all types of meat products (including fish) and dairy. Meat and dairy-based diets tend to have higher levels of leucine. As leucine is an activator of mTORC1 (MELNIK, 2012). Minerals such as calcium, magnesium, zinc, iron, and aluminum can interfere with the absorption of tetracyclines, resulting in reduced absorption and consequently reduced effectiveness (LEYDEN, ROSSO, 2014).

4 CONCLUSION

The intestinal microbiome and skin microbiota play crucial roles in skin health and pathology, including acne. Intestinal dysbiosis, characterized by an imbalance in the composition and function of the intestinal microbiota, emerges as a potentially contributing factor to the development and exacerbation of acne. Studies indicate that diet, especially Western diets, can influence both intestinal microbiota and acne manifestation, highlighting the interconnectedness between gastrointestinal and cutaneous health. Furthermore, evidence suggests that specific microorganisms, such as *Cutibacterium acnes*, play a crucial

role in acne pathogenesis, stimulating sebum production and triggering inflammatory responses in the skin. Understanding these complex interactions between the gut, skin, and diet is essential for the development of more effective and targeted therapeutic strategies for acne treatment.

CONFLICT OF INTEREST

The authors affirm that they have no conflicts of interest.

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