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(REVIEW ARTICLE)

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Cutibacterium acnes (*Propionibacterium acnes*): Review Article

Ahmed Sami Salman [1,*], Mohammed M. Alkhuzaie [2]

[1] *Department of Pathological analysis, College of science, Al-Qasim Green University, Iraq.*
[2] *Department of Pathological analysis, College of science, University of AL Qadisiya, Iraq.*

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Abstract

P. acnes or short for *Propionibacterium Acnes*, is a bacterium that is natural on human skin more or less on everyone. It is a ubiquitous and fascinating wrinkle in our largest organ that merely scratching the skin's surface gives an impression of. While it's commonly associated with pimples, the truth is quite diverse, as it plays far more roles than only causing acne. P. acnes is an anaerobic bacterium, which should suggest that it practices in oxygen-scarce environments such as the hair follicles and sebaceous glands, where it indeed feeds on sebum, or skin oil, however, *P. acnes* does not solely cause this inflammation; the glands have additional friends that contribute to the formation of pimples, papules, and pustules characteristic of acne. Other contributing factors are hormonal, genetic, and immune systems, as they also account for acne manifestations. Second, it is worth pointing out that not all *P. acnes* strains are alike. Of these, some may be more irritable than others, with a greater impact on acne severity. This has given researchers a cue to look for precise treatments that will focus on the worst-causing strains, thereby giving hope to improved acne treatment. However, different from what it was hitherto associated with, *P. acnes* is currently being researched for what it can do. Some of the proposed benefits are that it might act as an immunomodulator; it helps us control the immune system, which prevents infections; and it may be involved in the healing process, especially in burns, thus, it was found that *P. acnes* and our skin are intertwined in a mutually wakeful and poorly understood connection. It may be bad for acne-prone skin, but it may be good for something in our bodies. It is imperative to fully understand this intricate relationship in order to derive new tactics for halting the occurrence of acne and promoting proper skin health.

Keywords: Acne; Skin; Inflammation; Sarcoidosis; Prostate Cancer

1. Introduction

New information about acne motive and *P. acnes*, the principal pimple germ, has grown. This post will let you know what we know now about *P. acne's* position in acne and the way this could have an effect on treatment. Recent studies show *P. acne* attacks skin, hair, and oil glands, inflicting zits. It's additionally very seasoned-inflammatory. A higher hold on its process would possibly result in new methods to treat acne. New remedies may want to target things like germ-killing peptides, protease-activated receptors, and Toll-like receptors that start immune responses [1,2] .

What role does *Propionibacterium Acnes* play in causing zits, and what are these bacteria? Most microorganisms at the pores and skin floor are in three main categories. Keeping pores and skin wholesome depends on how these microorganisms interact. One of those bacteria is *Propionibacterium Acnes*. It lives in hair follicles on the pores and skin and protects it from harmful germs. It survives without air and likes a positive pH degree to grow nicely [3,4].

* Corresponding author: Ahmed Sami Salman

Figure 1 *Propionibacterium Acnes* under a microscope

2. Acne germ's genes

All genes discovered in *Propionibacterium Acnes* have been examined. These genes make enzymes for buying electricity from oil in hair sacs and for thriving in locations with less oxygen. Some genes additionally wreck down pores and skin. Five of those genes can cause pollution that creates holes in pores and the skin's masking. Inflammation happens while pollution is made [5]

The first of those divides the population into two phenotypes, Type I and Type II, based on the idea of gene collection. According to the recent division that was derived from the collection of sequencing, the TL gene, a house responsibility gene that encodes a hemolysin/cytotoxin, and the RecA gene, a non-ribosomal gene, were mainly used [6].

The second sort of feature is the biological feature, which consists of a correlation with the degree of lipid solubility that influences the improvement of pimples by this bacterium. To date, there are 5 kinds of *P. acnes*, and B3, also referred to as kind I, is the most important biotype. It produces more propionic and butyric acids than the other biotypes of *P. acne.* This may be due to this reason. This approach suggests that one-of-a-kind strains of *P. acnes* may additionally produce one-of-a-kind portions of the described proteins or possess extraordinary seasoned-inflammatory outcomes at the host and consequently show off specific capabilities to manipulate innate immunity [7,8].

Acnes secretes quick-chain fatty acids, in particular propionic acid, which performs the function of keeping the skin pH and renders the skin the required shield to enhance skin microbiota stability. Sebaceous glands are likewise capable of secreting enzymes that are capable of hydrolyzing the triglycerides at the floor of the pores and skin. Yet, it is a reality proven with the aid of severe studies that *P. acne* has a connection with acne vulgaris, but it is lively simplest inside the inflammatory segment of the technique [9,10].

3. Acne: What's it?

Acne vulgaris entails infection of the pilosebaceous unit; this has an effect mainly on the face, although it could arise in alternative regions of the frame, which include the back, sides, or even the chest. They are known as the sebaceous glands, which can be narrow and not as thick in contrast to the frame hair devices that compose the skin’s skin region. These are organs that secrete sebum, a fluid that is effortlessly rendered out onto the floor of the skin through a hole known as a pore, supplying the skin with its silkiness and elasticity [1,2,3].

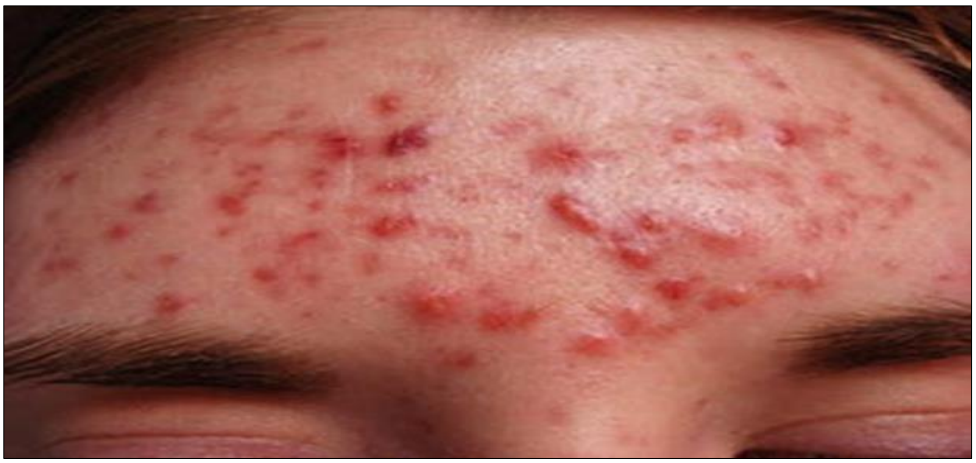

Figure 2 Acne caused by *P. acnes*

4. Inflammation and *P. acnes*

Leprobacterium acnes colonization. On the component of palatability, the pilosebaceous unit offers excessive palatability for *P. acne* because the surroundings created inside have a lipid, alkaline base, which is favorable to the growth and productivity of *P. acne.* Additionally, the paleo intestine, including the anaerobic intestine, favors the increase of *P. acne.* Nevertheless, any interpretation of *P. acne* determined inside the comedowns goes to purpose infection due to the aspect in which many elements released by this bacterium are worried within the pathophysiology of the acne for the reason that sebaceous secretion is going to restrict the outflow of sebum certainly. Among all the systems that might be fundamental to the decomposition of hyaluronate, the enzyme that is most important and has the capability to decompose connective tissue matrix proteoglycans with a glycosaminoglycan is hyaluronate lyase. Nevertheless, the microorganism is capable of engaging TLRs, a receptor that is present on the floor of host cells and mediates innate immunity, which contains first-line protection against the knock of a foreign invader. Especially for TLR 2 and TLR 4, it was discovered that their existence confirmed that the increase in the expression of those receptors is in the epidermal layer of pores and skin [11,12].

Studies have therefore proven that a few proteins in *P. acne* cause a heightened expression of TLR-2 and TLR-4 by the keratinocytes contained in the pores and skin. This results in the production of cytokines, which might be popularly known as interleukin-eight, or IL-8, and interleukin-12, or IL-12, involved in the control of irritation. Noteworthy in this respect is that P. acnes secretes a previously diagnosed enzyme denoted as e-lip tricellular lipase, which catalyzes the additional breakdown of necessary sebum triglycerides into glycerol. This glycerol is used to feed on the other varieties of microorganisms and on the fatty acids that create comedowns and which constitute nearby irritation [13].

Cytokines and chemokines, inflammatory mediators, produce irritation. NPs and Cs develop following encroachment of the inflammatory system, in addition to Acne, which is very intense, reaches a stage called inflammation, and inside the ultimate level, the follicular wall explosion occurs. Lipids, fatty acids, and the organisms that input the pores and skin cause irritation of surrounding tissue that is deep-seated and extends some distance past the website of the pustule [14].

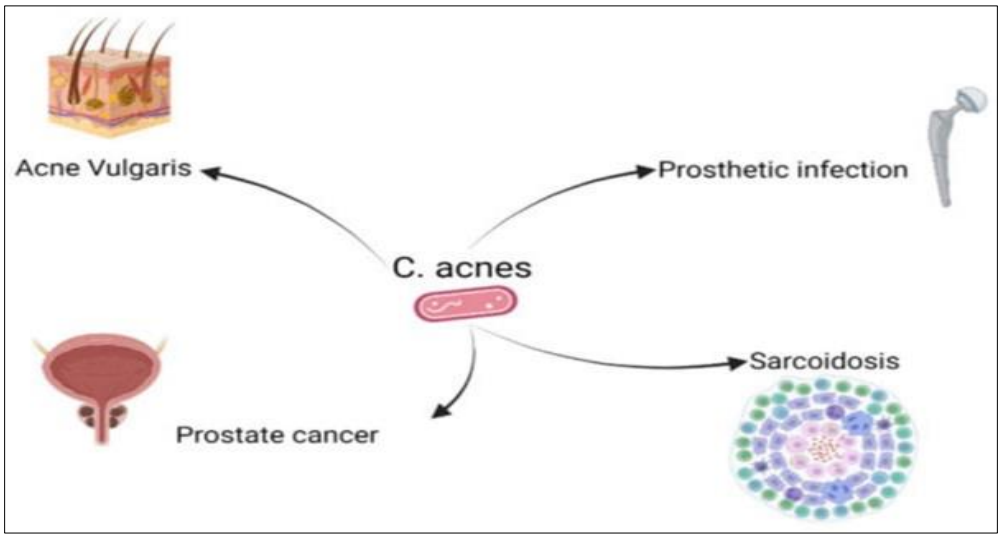

Figure 3 Infection sites infected by P. *acnes*

5. Biofilms are a crucial part of the pathogenicity of bacterial species, which include *S. aureus*.

Since *Propionibacterium Acnes* biofilm, which constitutes an adhesive that complements corneocyte cohesiveness and is implicated in the formation of comedones, there has been a perception that the capability of *P. acnes* microorganisms on the formation of biofilms was first defined in 2007. In addition, bacteria can adapt to intense situations and keep away from host defense mechanisms, as affirmed by the formation of a protective layer once they turn into biofilms; they may be more resistant to antimicrobial compounds than planktonic (free-living) cells, and they bring higher ranges of lipase and ac The mean values of bacterial biofilms for *P. acne* are acknowledged to be lower when cultured from everyday skin than from different diseases. Based on his professional affiliations and rankings, one would have anticipated P to be extra careful in his use of capital [15].

6. Pathogenesis of *Propionibacterium Acnes*

According to many faculties of thought, it's a harmless resident flora of the human frame and is no longer considered an infective agent. However, over time, it has additionally been assumed that C. pimples is related to some diseases that can be referred to as pores and skin sicknesses, namely Acne vulgaris. Even though *C.* acnes formation has no longer been thoroughly demonstrated, it has been cautioned that its miles related to Acne vulgaris. Candida zits is a part of everyday plant life in human pores and skin and consequently plays a full-size function in keeping pores and skin host protection and skin homeostasis and to beat back invasion through other pathogenic species. This remark implies that the variety of *C. acne* inside the skin that lacked acne vulgaris became just as numerous as in the pores and skin with zits vulgaris and its lesions [16].

7. Intervention

Erythromycin, tetracycline, and nutrition A derivatives, benzoyl peroxide, isotretinoin, pseudo-ephedrine, and aspirin are the not unusual topical and systemic medications used within the remedy of acne as they work to reduce inflammation and comedomal formation and to lower the manufacturing of sebum. A systemic remedy with antibiotics can be an alternative if the patient has excessive acne; otherwise, GPs prescribe low-dose antibiotics [17].

This is most commonly finished by using chemicals that can be deposited on the skin with the purpose of killing or treating the microorganism, in particular antibiotics, including tetracyclines, which are utilized in excessive amounts. Topicals, which include clindamycin or antimicrobials together with benzoyl peroxide, in the range of 5–10% are administered subsequent. Not only does this mixture serve well as an anti-septic and a cleanser that helps take away the debris that clogs the follicles accordingly, preventing sebum from escaping the pores, but it also has a very effective anti-bacterial action [18].

The second modality is the incorporation of retinoids or vitamin A derivatives that helps with the boom of the penetration of different anti-pimples marketers in pilosebaceous follicles in which P. acnes lays. Several types of P. acnes

are surely proof against wonderful antimicrobials, most commonly used in topical therapy, together with clindamycin in addition to BP; at the same time as the former is a bactericide, the latter is related to infection reduction [19].

8. Development of vaccinations

However, the look at preventing infections with *P. acne* is now in development. There are a number of barriers to immunizing against P. acne exocellular enzymes, wall fractions, or membrane-bound proteins; this is via engineering herbal antibodies against *P. acne.* One way is through azelaic acid, which inhibits the enzyme CAMP factor, a secretory cytotoxic protein of keratinocytes as well as macrophages, or a specific protein of P acnes. A great deal of decrease in the cytotoxicity of *P. acnes* was proven when the aggregate of CAMP and ubiquitous phosphodiesterase expressed inside the host were changed [20].

9. Other routes

Other pathways, including anti-CAMP monoclonal antibodies that would be administered locally, are currently being explored. Unfortunately, in all non-medical vaccine studies, zits lesions reappeared no matter the era of anti-*P. acne* antibodies. This is potentially due to inadequate production of shielding antibodies in opposition to key virulence factors. Although there are several crucial questions about vaccine sensitivity, vaccines against *P. acne* have crucial benefits: preserving the microbiome to prevent the development of resistance as a consequence of long-term antibiotic use and restricting scarring to govern inflammation [21].

10. Conclusion

Certainly, a higher knowledge of the position of *P. Acnes* opens new views for the development of the latest remedies, which include targeted healing procedures for molecules implicated in innate immunity or topical antimicrobial peptides as an opportunity for topical antibiotics that reduce bacterial resistance brought on by means of topical macro lipids. Vaccines can also offer promising answers. However, for these, the most appropriate applicants nonetheless want to be selected.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

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