

The Role of *Propionibacterium acnes* in the Pathogenesis of Acne

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Although *Propionibacterium acnes* has been associated with acne vulgaris for almost 90 years and there is considerable circumstantial evidence to suggest its importance, its role in acne is still not totally understood.

This subject¹ and the current status of acne research² have been reviewed recently, but many philosophical questions remain concerning the relationship between clinical acne and *P. acnes* (this name is used herein to include also *P. granulosum*, unless a more specific name is used).

Clinical Findings

P. acnes produces free fatty acids (FFA) and glycerol from triglycerides, and is, in turn, inhibited by the presence of those certain FFA, perhaps as a natural method of controlling growth of the organism.³ There are group differences in skin surface lipids obtained from children, adolescents with and without acne, young adults, and elderly persons, and there is growth enhancement of *P. granulosum* by skin lipid (sebum) extracts from acne patients and young adults.⁴ The cytotoxic effect of FFA and other comedonal materials on leukocytes is also postulated to induce inflammation in acne lesions.⁵

Considerable immunologic evidence links clinical acne with *P. acnes*, a potent stimulator of the reticulo-endothelial system. There is a relationship between the severity of acne and antibody levels and delayed hypersensitivity to *P. acnes*. *P. acnes* is also known to activate complement through both the classical and alternate pathways. The cell walls of the organism are mitogenic, suggesting that *P. acnes* has the ability to produce nonspecific reactions in both T and B lymphocytes. Voss⁶ postulated a possible role in comedo formation by

stimulating keratinization. While no general abnormalities have been found in the immunologic responsiveness of patients with acne, there is a depressed cell-mediated immune response in patients with acne conglobata. Whether this is the cause of, or the result of their disease, remains to be determined. Overall, acne patients seem to have elevated serum antibodies to *P. acnes* and an increased hypersensitivity reaction shown by intradermal injection. Release of lysosomes from polymorphonuclear (PMN) leukocytes seems to occur as a response to *P. acnes* only when serum antibodies (Ab) are present.

The cytotoxic property of *P. acnes*, its metabolites, and other comedonal contents are probably very important.⁶ The initial event in acne is felt to be a hormonally induced increase in lipid production, followed by comedo formation, and an increase in the number of bacteria. Neutrophilic leukocytes move toward chemotactic substances within the follicle, aiding rupture of the follicle wall and extrusion of comedonal and other follicular contents into the surrounding dermis. Complement is present in inflammatory acne lesions, but not in comedones, which probably means that it occurs as a later event. Numerous follicular components are chemotactic, but a dialyzable, small molecular weight chemotaxin derived from *P. acnes*, possibly the lipase, is likely the most important. Cytotaxins from *P. acnes* vary in vitro with the strain of bacteria and even with the substrate upon which the organisms are grown.⁶ When cultured on callus, for example, organisms produce far more cytotoxins than when grown on standard media.

It is not known whether substances applied to the skin surface can alter the number or strain of *P. acnes* except through comedogenesis, but there is evidence to suggest that it is possible. Kishishita⁷ has found that strains of *P. acnes* that do not ferment sorbitol are more commonly isolated from the forehead region than from areas around the mouth. Since sorbitol is found in toothpaste, sugarless gum, and certain natural foods, it would be

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