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Troubled Skin, Inflammation, and the Immune Response: Is targeting C. acnes Enough?

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Historically, treatments for acne have focused on a known causal microbial agent, *Cutibacterium acnes* (*C. acnes*) that is known to exist in high numbers in acne lesions. It has been suggested that *C. acnes*, which populates the human skin microbiome typically as a non-pathological entity, becomes pathological in acne outbreaks. However, the fundamental question of whether the *C. acnes* causes acne outbreaks, or whether it is simply responding to a modification of the skin's immune response has not been adequately answered. What is known is that most FDA-approved treatments for acne address the high levels of *C. acnes* which seems to effectively control acne outbreaks. In 2002, researchers discovered the NOD-Like Receptor Proteins (NLRP Inflammasomes). These skin cell Terracotta Warrior-like sentinel proteins are one of the keys that starts the engine of innate inflammation. Recently, the role of *C. acnes* in being the primary source of acne lesions has come into question, and it has been suggested that the cosmetic and therapeutic industries need to change the focus from this microbe as the cause of acne and troubled skin and focus instead on how inflammation initiates the onset of acne. So, the problem with troubled skin may not rest solely with control of the skin commensal, *C. acnes,* but may lie in understanding and controlling the skin's inflammation response driven, in part, by the presence of pathological *C. acnes* and its interaction with the skin's immune response. This talk will focus on the skin's immune response and on how certain well-known acne actives, like salicylic acid and azelaic acid, may do more than just kill *C. acnes*.

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