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"Acne biofilms," temperature, and microenvironments

To the Editor: I note that a February 2004 article by Dahl et al[1] entitled "Temperature regulates bacterial protein production: Possible role in roasacea" appeared in a section marked "Ideas." However, this article offers three essential observations that could form a framework for future scientific analyses of acne and its related disorders.

First, the various proteins secreted by bacteria play a central role in acne and rosacea pathogenesis. These bacterial products include lipases, proteases, hyaluronidases, and chemotactic factors.[2] The inflammatory response initiated by these extracellular enzymes affects humoral- and cell-mediated immunity, complement activation, and cytotoxin production.

Second, the authors acknowledge that the microorganisms in the pilosebaceous unit exist there not as plankton, or free-floating bacteria in suspension, but as a community of bacteria. This supports the microbiologic principle of biofilms, which provides a theoretical structure for understanding the physical, chemical, and biologic interactions of bacteria within microenvironments. In an "acne biofilm," the microorganisms encase themselves in an extracellular polysaccharide that they secrete after adherence to a surface.[3] This glycocalyx polymer acts like a protective exoskeleton and physical barrier, limiting the antimicrobial concentrations that reach the bacteria within the biofilm. Bacteria in the protected microenvironment of a biofilm are 50 to 500 times more resistant to antibacterial therapies than planktonic organisms.[4]

Additionally, the authors note that alterations in the microenvironment, such as temperature, can alter production of extracellular enzymes by bacteria. Within biofilms in the pilosebaceous units, the milieu and the enzyme activities are constantly changing and evolving depending on various environmental factors. The microenvironment surely plays a major role in the amount of exoenzymes produced. In vitro, *Propionibacterium acnes* growth can be altered by factors such as pH and oxygen tension. Thus, treatments directed toward altering the microenvironment in which biofilms exist, thereby altering the enzymes secreted by those bacteria, are not intellectual whims, but could be a future direction of acne and rosacea therapeutics.

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HLA-DRB1*04 is associated with the genetic susceptibility to develop vitiligo in Mexican patients with autoimmune thyroid disease

To the Editor: Putative associations of vitiligo with human leukocyte antigen (HLA) genes suggests a role of these in the genetic susceptibility to develop vitiligo in several ethnic groups. We aimed was to study the association between HLA class II alleles and vitiligo in patients with or without autoimmune thyroid disease.

One hundred and twelve patients with the diagnosis of vitiligo were studied. Forty-six patients had autoimmune thyroid disease. The HLA-DRB1 allele typing was performed by polymerase chain reaction using sequence-specific oligonucleotide (PCR-SSO) reverse dot blot and polymerase chain reaction sequence-specific primer (PCR-SSP). A statistically increased frequency of the HLA-DRB1*04 allele was found in patients with vitiligo and autoimmune thyroid disease as compared to healthy controls ($P_{corrected}$ = .02; OR = 2.26; 95% CI, 1.29-3.97) (Table I). Phenotype analysis showed a significantly increased gene frequency in the homozygous state of HLA-DR4/DR4 in patients with vitiligo and autoimmune thyroid disease as compared to patients with only vitiligo ($P_{corrected}$ = .02; OR = 3.2; 95% CI, 1.1-10.43) and healthy controls (P = .01; OR = 7.28; 95% CI, 1.25-54.7).

On the other hand, a significant decreased frequency of HLA-DRB1*08 allele was observed in the group of patients with vitiligo and autoimmune thyroid disease as compared to healthy control