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## Whole genome expression of epidermis infected with common skin dwellers

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C kin is the first line of defense against physical, chemical as well as the biological environment. A number of the antibiotics have been Oproduced; however, continuous emergence of resistant strains is a big challenge for human health. Skin is an ideal organ to study molecular responses to biological infections by virtue of diverse skin cells specialized in immune responses. Comparative analysis of skin responses to pathogenic, non-pathogenic and commensal bacteria would help in the identification of disease-specific pathways for drug targets. In this study, we investigated human breast reduction skin responses to Propionibacterium acnes, Staphylococcus aureus, Staphylococcus epidermidis and TLR1/2 agonist using Affymetrix microarray chips. The Pam3CSK4 solution and bacterial cultures were prepared and inoculated in steel rings that were placed on the acetone-treated epidermis in a petri dish. After 24 hrs incubation, 8mm punch biopsies were taken from the center of the ring and RNA was extracted. The genome-wide expression was then analyzed using Affymetrix HG-133A gene chip microarray. The bacteria checked cause skin to boost the production of extracellular matrix components and attenuate the expression of differentiation markers. The above responses are mediated via the TLR2 pathway. Skin also responds to S. aureus and P. acne by inducing the genes of the cell cycle machinery; this response is not TLR2-dependent. S. aureus induces, whereas P. acnes suppresses the genes associated with apoptosis; this is also not TLR2-dependent. The pathogenic behavior of non-resident bacteria is principally defined by their ability to induce host immunity while evading the host immune bactericidal mechanisms through either upregulation of normal cellular processes in infected cells or directly interfering apoptotic process. The commensals have also evolved strategies to survive skin immunity, however, they lack the ability to initiate immune processes until and unless other factor set a pre-infection environment for them.

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