# Surface Bacteria Maintains Skin's Healthy Balance

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On the skin's surface, bacteria are abundant, diverse and constant, but inflammation is undesirable. Research at the University of California, San Diego School of Medicine now shows that the normal bacteria living on the skin surface trigger a pathway that prevents excessive inflammation after injury.

"These germs are actually good for us," said Richard L. Gallo, MD, PhD, professor of medicine and pediatrics, chief of UC San Diego's Division of Dermatology and the Dermatology section of the Veterans Affairs San Diego Healthcare System.

The study, to be published in the advance on-

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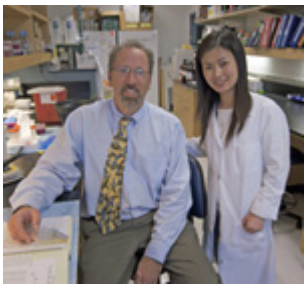

richard Gallo, MD, PhD, & Yu Ping Lai

ne edition of Nature Medicine on November 22, was done in mice and in human cell cultures, primarily performed by post-doctoral fellow Yu Ping Lai .

"The exciting implications of Dr. Lai's work is that it provides a molecular basis to understand the 'hygiene hypothesis' and has uncovered elements of the wound repair response that were previously unknown. This may help us devise new therapeutic approaches for inflammatory skin diseases," said Gallo.

The so-called "hygiene hypothesis," first introduced in the late 1980s, suggests that a lack of early childhood exposure to infectious agents and microorganisms increases an individuals susceptibility to disease by changing how the immune system reacts to such "bacterial invaders." The hypothesis was first developed to explain why allergies like hay fever and eczema were less common in children from large families, who were presumably exposed to more infectious agents

than others.  It is also used to explain the higher incidence of allergic diseases in industrialized countries.

The skin's normal microflora – the microscopic and usually harmless bacteria that live on the skin – includes certain staphylococcal bacterial species that will induce an inflammatory response when they are introduced below the skin's surface, but do not initiate inflammation when present on the epidermis, or outer layer of skin.

In this study, Lai, Gallo and colleagues reveal a previously unknown mechanism by which a product of staphylococci inhibits skin inflammation.  Such inhibition is mediated by a molecule called staphylococcal lipoteichoic acid (LTA) which acts on keratinocytes – the primary cell types found on the epidermis.

The researchers also found that Toll-like receptor 3 (TLR3) activation is required for normal inflammation after skin injury.

"Keratinocytes require TLR3 to mount a normal inflammatory response to injury, and this response is kept from becoming too aggressive by staphylococcal LTA," said Gallo. "To our knowledge, these findings show for the first time that the skin epithelium requires TLR3 for normal inflammation after wounding and that the microflora helps to modulate this response."

Additional contributors to the paper include Yu Ping Lai, Anna Di Nardo, Teruaki Nakatsuji, Anna L Cogen, Chun-Ming Huang and Katherine A. Radek, UCSD Division of Dermatology and the VA San Diego Healthcare System; Anke Leichtle and Allen F. Ryan, UCSD Department of Surgery/Otolaryngology and the VA San Diego Healthcare System; Yan Yang and Zi-Rong Wu, School of Life Science, East China Normal University, Shanghai; Lora V Hooper, Howard Hughes Medical Institute and University of Texas Southwestern Medical Center, Dallas; and Richard R Schmidt and Sonja von Aulock, University of Konstanz, Germany.

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