Study Sheds Light on Atopic Dermatitis Infections

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VIENNA — Levels of some of the skin's key innate antimicrobial peptides are abnormally low in atopic dermatitis patients, perhaps accounting for the high rate of bacterial and viral superinfections in this population, Jurgen Harder, M.D., said at the annual meeting of the European Society for Dermatological Research.

The explanation for the low skin levels of antimicrobial peptides may lie in the high levels of Th2 cytokines typically present in atopic skin. These cytokines appear to sharply inhibit secretion of the antimicrobial peptides by keratinocytes, added Dr. Harder of the University of Kiel (Germany). He presented evidence from in vitro studies done in Kiel showing that high concentrations of the Th2 cytokines interleukin-4 (IL-4), IL-10, and IL-13 greatly dampen the normally robust induction of an important antimicrobial peptide in normal skin, human β-defensin-2, by *Pseudomonas aeruginosa*.

Expression of human β-defensin-2 was reduced in response to *P. aeruginosa* by 80%-90% in keratinocytes exposed to high levels of IL-4, by 70%-80% with IL-13, and by 20%-30% with IL-10.

These laboratory findings are consistent with the hypothesis that bacterially mediated induction of human β-defensin-2 and other inducible antimicrobial proteins is disrupted in atopic dermatitis patients and that the culprit is high levels of the Th2 cytokines.

These studies also suggest a novel potential strategy for prevention of cutaneous superinfections in patients with atopic dermatitis: treatments aimed at decreasing the elevated Th2 cytokine levels, Dr. Harder said.

Treatments aimed at decreasing the elevated Th2 cytokine levels are a novel potential strategy for for fighting superinfections in this population.

It's worth noting, he added, that levels of innate antimicrobial proteins are high in the skin of psoriasis patients. This might explain their relatively low rate of bacterial superinfections despite the often severe damage to the skin's barrier function. ■

Quick Fungal Dx With In Situ Hybridization

BOSTON — In situ hybridization may provide a much faster method of diagnosing dimorphic fungal infections than tissue culturing, Jared J. Abbott, M.D., reported at the annual meeting of the American Society of Dermatopathology.

Although tissue culturing is considered the preferred diagnostic method in current practice, it takes time to complete. Often, tissue cultures are not done, said Dr. Abbott, a resident in pathology at the Mayo Clinic, Rochester, Minn.

Cutaneous fungal infections can be the presenting sign of a systemic infection, and thus the skin infection can be secondary. The prognosis of cutaneous fungal infections is heavily dependent on timely diagnosis and treatment, he said in a poster presentation.

Dr. Abbott and his colleagues diagnosed cutaneous infections of dimorphic fungi (histoplasmosis, blastomycosis, coccidiomycosis, or cryptococcosis) in five patients, four of whom were immunocompromised. Within 36-48 hours, in situ hybridization gave the same results as tissue culturing for all patients.

The investigators determined the presence of dimorphic fungi in all of the skin sections via morphology, but none of the organisms were diagnostically discernible.

The in situ hybridization method used by Dr. Abbott and his associates detected fungal ribosomal RNA elements in paraffin-embedded tissue using labeled, single-stranded DNA oligonucleotide probes.

"In situ hybridization has a potential role in distinguishing between organisms of similar morphology in cutaneous fungal infections," the investigators concluded.

—Jeff Evans