**Endogenous Protein Protects Skin From E. coli**

**BY BRUCE JANCIN**

Denver Bureau

VIENNA — Healthy skin secretes an antimicrobial protein called psoriasin that is a potent *Escherichia coli*–killing compound, Regine Glaser, M.D., said at the annual meeting of the European Society for Dermatological Research.

Psoriasin appears to be the principal reason that cutaneous *E. coli* infection is rare despite the bowel bacterium’s ubiquitous presence in daily life, according to Dr. Glaser of the department of dermatology at the University of Kiel (Germany).

Danish investigators first described psoriasin in psoriatic lesions in the late 1990s. Its function was unknown. But in recent studies, Dr. Glaser and her co-workers showed that psoriasin’s main function appears to be to protect the skin from *E. coli* infection.

Reasoning that healthy skin’s high degree of resistance to *E. coli* infection might be due to some innate defensive factor, Dr. Glaser looked for candidate compounds in human stratum corneum extracts. The 11-kd S-100 protein psoriasin emerged as the top candidate.

Moving on to in vivo work, Dr. Glaser used an antipsoriasin monoclonal antibody to show that high concentrations of psoriasin were produced selectively by keratinocytes located in the upper, more differentiated epidermal layers of the nose, the anogenital area, the armpits, and the sebaceous glands—all sites with high bacterial colonization rates. In contrast, psoriasin staining was patchy in skin on the extremities and other areas where bacterial colonization is less common.

Psoriasin’s antimicrobial spectrum showed a strong preference for *E. coli*. The compound’s mechanism of killing *E. coli* appears to involve sequestration of zinc, which deprives the microorganism of an essential metal ion.

Dr. Glaser and her co-investigators are exploring the potential utility of a psoriasin-based topical therapy.

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**Atopic Dermatitis More Common in Very Clean Home**

**FLORENCE, ITALY —** A case-control study conducted in Greece lends support to the theory that a ‘superclean’ environment during infancy and early childhood may predispose children to atopic dermatitis.

Penny Emmanouil, M.D., and associates in the department of dermatology at Pentelis Children’s Hospital in Athens, Greece, studied home hygiene, standards of living, exposure to infections, and vaccination rates among 150 children aged 28 days to 3 years who were seen for atopic dermatitis (AD) symptoms at an outpatient clinic. These results were compared with data from a group of 150 children aged 35 days to 3 years who had no atopic symptoms during the same period.

Significant differences were seen in the two groups of children. For instance, nearly half of children with AD had their own bedrooms, while those without AD symptoms tended to share living space with parents and siblings. Those with AD were more likely to live in larger, cleaner, more well-to-do households with fewer children.

No relationships were seen between vaccinations or infections and AD. More work must be done to tease out risk factors that may be responsible for the development of AD in early childhood, Dr. Emmanouil said.

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No relationships were seen between vaccinations or infections and AD. More work must be done to tease out risk factors that may be responsible for the development of AD in early childhood, Dr. Emmanouil said.

However, she hypothesized that exposure to microbes might be restricted in those households that practice meticulous hygiene

“As a result, the immune system in infancy and early childhood is restricted, and the switch from the TH2- to TH1-mediated immune response is impaired,” she said.

—Betsy Bates