SKIN MICROBIOM: FORMATION AND CHANGE

Yehorova Svitlana,
Ph.D., Associate Professor
Department of microbiology, virology, immunology,
epidemiology and Biomedical physics and informatics,
Dnipro State Medical University

The skin is the human body's largest organ, colonized by a diverse milieu of microorganisms. The primary role of the skin is to serve as a physical barrier, protecting our bodies. The skin is also an interface with the outside environment and, as such, is colonized by a diverse collection of microorganisms (bacteria, fungi and viruses). Colonization is driven by the ecology of the skin surface, which is variable depending on endogenous host factors and exogenous environmental factors (genes, sex, age, diet, air pollution, exposure to UV light, etc.). Age has a great effect on the microenvironment of the skin and on the colonizing microbiota. In utero, fetal skin is sterile, but colonization occurs immediately after birth. During puberty, changes in sebum production parallel the levels of lipophilic bacteria on the skin [1].

In addition to being a physical barrier, the skin is an immunological barrier. The cutaneous innate and adaptive immune responses can modulate the skin microbiota; the microbiota also functions in educating the immune system. Keratinocytes continuously sample the microbiota colonizing the skin surface through pattern recognition receptors, such as Toll-like receptors (TLRs) and mannose receptors. The activation of keratinocytes initiates the innate immune response, resulting in the secretion of antimicrobial peptides, cytokines and chemokines. *Staphylococcus epidermidis*, a commensal bacterium, has been demonstrated to modulate the host innate immune response. Phenol-soluble modulins produced by *S. epidermidis* can inhibit skin pathogens, such as *S. aureus* and group A *Streptococcus* [2]. Also, *S. epidermidis* triggers keratinocyte expression of antimicrobial peptides through a TLR2-dependent mechanism [3].

References:

1. Grice EA, Segre JA. The skin microbiom. Nat Rev Microbiol. 2011; 9(4):244–253.

2. Cogen AL, et al. Selective antimicrobial action is provided by phenol-soluble modulins derived from *Staphylococcus epidermidis* a normal resident of the skin. J. Invest. Dermatol. 2010;130:192–200.

3. Lai Y, et al. Activation of TLR2 by a small molecule produced by *Staphylococcus epidermidis* increases antimicrobial defence against bacterial skin infections. *J. Invest. Dermatol.* 2010;130:2211–2221.