

Role of a Novel Bacterial Lipopeptide from *Staphylococcus epidermidis* in Treating Atopic Diseases in Human

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One of the most common inflammatory skin diseases is Atopic disease (AD) and has life time prevalence of 20%. AD is characterized by pruritus and chronic and relapsing skin inflammation. Defensive and protective abilities of epidermis provide protection against radiation and microbial invasion [1]. Microbial invasion in skin is prevented by production antimicrobial peptides (AMPs) also commonly known as host peptides (HDPs). [2,3]. These HDPs play an important role in inducing pro-inflammatory and anti-inflammatory responses and are largely located in skin [2-4]. Human have small (3.5 - 6 kDa) specific HDPs known as defensins. Based on cystine bridges, defensins are of two types α and β defensins. Protein crystal structures of the defensins have already been already solved and these cationic soluble mediators provide protection against the viral bacterial and fungal pathogen that enters body through skin [5]. Among other HDPs- cathelicidin and Psoraisin also play a key role against the invading pathogenic skin micro-organisms [6,7]. Skin also allows mutual survival of symbiotic microbial organisms. Among them, one of the most commonly known symbiotic colonizers is *Staphylococcus epidermidis*. Recent studies suggested this microorganism produces a < 10 kDa lipopeptide (DIISTIGDLVKWIIDTVIIDATE) which triggers the TLR-2 pathway that in turn activates the beta defensins (hBD-2) through CD-36-p38-NF- κ B pathway [8]. The major difference from its close orthologue *Staphylococcus aureus* which is pathogenic to human skin is that *S. aureus* induces expression of hBD-1 and hBD-3 which inhibits their colonization and prevents the infections. On the other hand lipopeptide of *S. epidermidis* induces the hBD-2 that induces activation of antimicrobial defensins from keratinocytes ensuring not only its colonization within the epidermis but also eradication of pathogenic organisms [9]. Specific way how this < 10 kDa peptide induces the hBD-2 is unclear. Recent studies have shown that mixture of secreted lipopeptides in medium form *S. epidermidis* and *Pseudomonas aeruginosa* are even more effective for eradication of pathogenic organism related to AD [10]. Structural efforts deciphering the details of the induction of TLR-2 by this lipoprotein will prove vital for understanding the exact sequence of events that controls the expression of hBD-2. Atomic details of lipopeptide will be important in understanding the way it specifically allows to symbiotically colonize the bacteria during such skin infection. Synthetic peptides mimicking this natural lipoprotein might open new ways for peptide therapeutics to treat Atopic Diseases (ADs) like psoriasis where defensins are produced less.

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