Synopsis of Original Research Paper

Involvement of staphylococcal immune-distubing proteins in atopic dermatitis

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Staphylococcus aureus (*S. aureus*) colonizes approximately 30% of the population asymptomatically but causes various diseases such as food poisoning, suppurative disease, sepsis, or systemic infections. S. aureus is associated with atopic dermatitis (AD); it frequently colonizes in the skin of atopic dermatitis. S. aureus is notorious for producing a series of exotoxins and immune disturbing proteins, however none of the staphylococcal exotoxins except with δ-toxin and phenol soluble modulins had been reported to activate mast cells and basophils, critical immune cells involved in the development of Th2 cell-mediated allergic inflammation such as atopic dermatitis. In this study, we aimed to identify staphylococcal exotoxins that activate mast cells and basophils. We found that α-hemolysin, a principal small β-barrel pore-forming toxin, did not activate mast cells individually, but it augmented the degranulation induced by other stimuli. We showed that among 14 members of staphylococcal superantigen-like protein, SSL12 induced the degranulation and cytokine production of mast cell in an IgE-independent manner, and SSL12 evoked local inflammation in vivo. We also showed that SSL12 induced the production of IL-4 in bone marrow derived basophils and freshly isolated murine basophils in bone marrow cells. These results propose the novel immune regulatory activity of α-hemolysin and SSL12 by activating mast cells and basophils that contributes to the development of allergic inflammation disorders such as atopic dermatitis.