

Th2 Cytokines Increase *Staphylococcus aureus* Alpha Toxin-Induced Keratinocyte Death through the Signal Transducer and Activator of Transcription 6.

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Original Article

Th2 Cytokines Increase *Staphylococcus aureus* Alpha Toxin-Induced Keratinocyte Death through the Signal Transducer and Activator of Transcription 6 (STAT6)

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Atopic dermatitis (AD) is an inflammatory skin disease characterized by increased T-helper type 2 (Th2) cytokine expression. AD skin lesions are often exacerbated by *Staphylococcus aureus*-mediated secretion of the lytic virulence factor, alpha toxin. In the current study, we report that alpha toxin-induced cell death is greater in the skin from patients with AD compared with controls. Furthermore, we find that keratinocyte differentiation and Th2 cytokine exposure influence sensitivity to *S. aureus* alpha toxin-induced cell death. Differentiated keratinocytes are protected from cell death, whereas cells treated with Th2 cytokines have increased sensitivity to alpha

toxin-induced lethality. Our data demonstrate that the downstream effects mediated by Th2 cytokines are dependent upon host expression of STAT6. We determine that Th2 cytokines induce biochemical changes that decrease levels of acid sphingomyelinase (SMase), an enzyme that cleaves sphingomyelin, an alpha toxin receptor. Furthermore, Th2 cytokines inhibit the production of lamellar bodies, organelles critical for epidermal barrier formation. Finally, we determine that SMase and its enzymatic product, phosphocholine, prevent Th2-mediated increases in alpha toxin-induced cell death. Therefore, our studies may help explain the increased propensity for Th2 cytokines to exacerbate *S. aureus*-induced skin disease, and provide a potential therapeutic target for treatment of AD.



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