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lS100-A7 protein protects the skin from fungal infections

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Despite the permanent exposure and colonization by various filamentous fungi and yeasts, human skin is rarely infected. Plants and insects seem to be well protected by antimicrobial peptides, the effector molecules of the innate immune system, such as RsAFP1 and drosomycin. However, it is largely unknown, how human body surfaces resist to fungal infection. Here, we show that the keratinocyte-secreted protein, S100 calcium-bind protein A7 (S100-A7), is the principle antifungal factor of human skin. It kills various potentially pathogenic fungi, including *Aspergillus, Malassezia, Microsporum, Rhizopus* and *Trichophyton* species. To kill these pathogens, the protein needs to change its shape, i.e., the oxidized protein to the reduced protein. Site-directed mutagenesis and derivatization revealed that the cysteine thiols of S100A7 are essential for its antifungal activity. Sensitivity to Zn^{2+}-pretreatment, ultrastructural immuno-gold-analyses and functional analyses suggest that S100A7 protein kills fungi by inducing apoptosis via intracellular Zn^{2+}-depletion. S100A7 is found on the skin and the mucosa of the respiratory, aerodigestive and genital tract. *In vivo,* in a lethal mouse *Aspergillus* lung infection model, treatment with S100A7 prevented death from *Aspergillus* infection in immuno-compromised mice. Thus, S100A7 may represent a key component of the human innate epithelial defense system in the control of a wide range of fungal pathogens.

Biography

Kyaw Zaw Hein has completed his medical degree from University of Medicine Mandalay in 2008 and expects to graduate his Ph.D. from Shimane University Faculty of Medicine in 2014. His current research specializes on the epithelial immune system and anti-microbial peptides.

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