Small peptide ameliorates autoimmune skin blistering disease in mice

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Pemphigus vulgaris is a life-threatening autoimmune skin disease that is occurs when the body's immune system generates antibodies that target proteins in the skin known as desomogleins. Desmogleins help to form the adhesive bonds that hold skin cells together and keep the skin intact. Currently, pemphigus vulgaris is treated by long-term immune suppression; however, this can leave the patient susceptible to infection.

In this issue of the Journal of Clinical Investigation, researchers led by Jens Waschke at the Institute of Anatomy and Cell Biology in Munich, Germany, report on a small peptide that blocked antibody recognition of desmogleins. Importantly, the peptide could prevent antibody-mediated skin blistering when applied topically to mice. At the cellular level, the peptide improved cell-cell adhesion and attenuated signaling pathways that are activated by antibody binding.

These results suggest that this peptide could serve as a treatment option for pemphigus vulgaris.

More information: Peptide-mediated desmoglein 3 crosslinking prevents pemphigus vulgaris autoantibody-induced skin blistering, *Journal of Clinical Investigation*, 2013. doi:10.1172/JCI60139

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