Possible role of autophagy in sclerodermat

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Sclerodermat (SSc) is a connective tissue disease characterized by dermal and/or visceral fibrosis. Accumulating evidence has suggested that the pathogenesis of SSc involves impaired matrix turnover and chronic inflammation. Amongst the major features of the disease is the fibrotic transformation of the skin, which is often accompanied by skin damage.

The aim of this study was to investigate the role of autophagy in the pathogenesis of SSc. We hypothesized that autophagy is involved in the fibrotic transformation of the skin by regulating the CXCL5 pathway. Our results revealed that autophagy is upregulated in skin samples from SSc patients compared to healthy controls, and that autophagy plays a crucial role in the fibrotic transformation of the skin by regulating the CXCL5 pathway.

Hyaluronan oligosaccharides induce suppressive effect to chronic allergic dermatitis

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Hyaluronan (HA) has been implicated in wound healing and inflammation, and its biological role has been shown to be dependent on its molecular size. Recently, it has been shown that HA oligomer administration in vivo modulates contact hypersensitivity responses.

In a previous study, we demonstrated that HA oligomer administration in vivo modulates contact hypersensitivity responses in mice. In the current study, we investigated the role of HA oligomer administration in the modulation of contact hypersensitivity responses in vitro.

In vitro, HA oligomer administration significantly suppressed the increase in ear thickness, ear swelling, and inflammatory cell infiltration in mice with contact hypersensitivity reactions. These results suggest that HA oligomer administration in vivo modulates contact hypersensitivity responses in vivo.

Metabolomic profiling of psoriasis skin reveals localized cortisol deficiency resulting in maintenance of inflammatory state and disruption of epidermal barrier repair

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Psoriasis is an autoimmune skin disease characterized by chronic inflammation and hyperproliferation of the epidermis. The pathogenesis of psoriasis involves the activation of the immune system and dysregulation of the epidermal barrier repair. The aim of this study was to investigate the role of cortisol in the pathogenesis of psoriasis.

Cortisol levels were significantly decreased in psoriatic skin compared to healthy skin. The reduction in cortisol levels was associated with an increase in pro-inflammatory cytokines, including IL-17A and CXCL10. These results suggest that cortisol deficiency plays a crucial role in the maintenance of the inflammatory state and disruption of epidermal barrier repair.

Adaptive and Auto-Immunity | ABSTRACTS

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