Synopsis of Original Research Paper

Elucidation of the onset mechanism of allergy mediated by neuropeptide signals and the relieving effect on dermatitis

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Not a few people are suffering from allergic skin diseases such as atopic dermatitis and psoriasis. With the development of medicine, patients with skin diseases are relieved by the treatment with anti-inflammatory or steroidal drugs. However, there are some patients having not only side effects such as drug allergies and anaphylaxis but also refractory patients. Therefore, it is important to elucidate the onset and aggravation mechanisms of skin diseases from a new perspective. In the previous studies, we revealed that a neurokinin A receptor, NK2R-mediated neuropeptide signals, activated in a STAT1-dependent manner, augmented antigen presentation ability of dendritic cells and induced allergen-specific T cell immune responses.

In this study, we investigated the involvement of STAT1-mediated neuropeptide signals in the onset and clinical condition of skin diseases by using a psoriasis mouse model. We established a psoriasis model by the treated with imiquimod (IMQ) to the skin and found that body weight loss scale formation and thickness of skin in STAT1-deficient mice were significantly reduced compared to wild-type mice. Ly6C-expressing myeloid cells were transiently induced in the IMQ-treated wild-type mice were significantly lower in the STAT1-deficient mice. Furthermore, we confirmed that IL-17A and NK2R gene expression levels were down-regulated in the spleen cells of IMQ-treated STAT1-deficient mice compared to that of wild type mice. These data suggested that STAT1 activation were closely related to the pathogenesis of in the IMQ-induced psoriasis model mice.

Base on the present findings, we speculate that STAT1-mediated neuropeptide signaling cascade and the related molecules may be promising drug targets to improve skin diseases such as psoriasis.