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The story of Copaxone (glatiramer acetate) in the treatment of multiple sclerosis and its new applications

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Multiple sclerosis (MS) is currently recognized as complex diseases in which inflammatory autoimmune reactivity in the central nervous system (CNS) results in demyelination, axonal and neuronal pathology. Novel treatment strategies aim to reduce the detrimental inflammation and induce neuroprotective repair processes. The synthetic copolymer Copaxone (glatiramer acetate, GA), an approved drug for the treatment of MS, is the first and so far the only therapeutic agent to have a copolymer as its active ingredient. Using the animal model of MS experimental autoimmune encephalomyelitis (EAE), the immunomodulatory mechanism of action of GA was elucidated. It was found that GA treatment induces immunomodulatory shift from the inflammatory towards the anti-inflammatory pathways. Furthermore, recent studies revealed neuroprotective and repair consequences of GA treatment in the CNS. These include elevation in neurotrophic factors expressions, remyelination and neurogenesis. Based on its immunomodulatory mode of action, additional potential applications of GA were investigated, such as prevention of immune rejection, improvement of stem cells engraftment and amelioration of inflammatory bowel diseases (IBD).

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Increases in group-2 innate lymphoid cells are inhibited by glucocorticoid treatment in allergic airway inflammation in human

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Background: Group-2 innate lymphoid cells (ILC2s) was closely associated with the human allergic disease such as asthma and allergic rhinitis. However, the effects of ILC2s to the severity of illness and the correlation between ILC2s and glucocorticoid treatment, is not well understood.

Objective: The purpose of this study was to investigate the effects of glucocorticoid treatment on ILC2 levels in asthma patients.

Methods: The patients of asthma, asthma with allergic rhinitis were received the treatment of glucocorticoid for 3 months. The peripheral blood monocytes (PBMCs) from the subjects including healthy subjects were detected directly or cultured with IL-2, IL-25 and IL-33 and then detected for ILC2s using flow cytometry. The ILC2s were sorted and stimulated with the glucocorticoid. ELISA was used to measure the cytokines of IL-5 and IL-13 in the plasma or cell-free supernatant.

Results: The levels of ILC2s in blood were greater in the asthma group and asthma with allergic rhinitis patients compared to healthy subjects. The ILC2 levels significantly decreased at 1 or 3 months after glucocorticoid treatment. There were dramatically production of IL-5 and IL-13 in patient PBMCs cultured with IL-25 and IL-33. Furthermore, glucocorticoid significantly inhibited the function of ILC2s *in vitro*. The PBMCs from patients produced dramatic production of IL-5 and IL-13 responded to IL-2, IL-25 and IL-33 compared to healthy subjects.

Conclusion: ILC2 levels in asthma and asthma and allergic rhinitis were decreased with the treatment of glucocorticoid.

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