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Entitled

MODULATION OF CHRONIC ALLERGIC CONTACT DERMATITIS THROUGH B CELLS REPROGRAMMING

by

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Abstract

Allergic contact dermatitis (ACD) is a common inflammatory disease caused by contact with an allergen and is a T-cell mediated type IV hypersensitivity reaction. Short chain fatty acids (SCFAs) showed the ability to modulate immune cells in case of inflammation. Pentanoate is one of the abundant SCFA that showed therapeutic ability by inducing IL-10 secretion in lymphocytes. IL-10 producing regulatory B cells have shown to decrease different inflammatory responses; however, overall role of B cells in ACD is not well studied. This project aims to explore the role of B cells during chronic ACD and their modulation with the SCFA pentanoate. A chronic CHS mouse model was established in vivo by the hapten TNCB and studied for allergic inflammatory responses by measuring ear swelling, cellular analysis and histology. The B and T cell subsets were analyzed by flow cytometry. The data showed a significance increase in tissue resident memory-like T cell subsets along with the central memory T cell subset and the total CD4+ T cells. Simultaneously, a significant decrease in regulatory T cell subsets, regulatory B cells and naïve T cells was observed. In vitro stimulation of B cells with pentanoate showed a significant increase in IL-10 and STAT3 gene expression which confirmed the modulation of B cells into IL-10 producing regulatory B cells. After the adoptive transfer of these stimulated B cells followed by ACD induction showed a significant decrease in the inflammatory response of the acute CHS response and a mild effect on chronic CHS response. Our data showed that CHS responses can be modulated through pentanoate. Our research highlights the role of B cells in ACD and the therapeutic potential of the pentanoate.

Keywords: allergic contact dermatitis, B cells, IL-10, pentanoate.