

The stimulatory effect of autoreactive marginal zone B-cell on the cytokine production in T-cells

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Our immune system's purpose is to defend our body from foreign pathogens while not attacking body's own cells termed as tolerance. Autoimmune diseases results when this tolerance is broken and immune system starts attacking one's own body. B-cells are a type of immune cells that are involved in many autoimmune diseases such as rheumatoid arthritis.

When being attacked by a foreign pathogen B-cells secrete small molecules called antibodies that mediate clearance of these pathogens. Spleen is involved in blood filtration and also a site for immune reaction in case of invading pathogens. Two types of B-cells are found in the spleen - marginal zone B-cells (MZB) and follicular B-cells (FOB). MZB cells located in the marginal zone of spleen were found to initiate autoreactive reaction against collagen in mouse model of rheumatoid arthritis - collagen induced arthritis. T-cells are another type of immune cells that helps the B-cells in producing antibodies. Antibodies produced by B-cells with T-cell help are specific for the pathogen leading to rapid clearance and production of memory cells for protection against future attack. B-cells and T-cells also produce small messenger molecules known as cytokines that relay messages between different cells. Cytokines can also enhance breakage of tolerance and induce autoimmunity.

We were interested to know if MZB cells can stimulate T-cells to produce specific cytokines that aid in breakage of tolerance and induction of inflammatory response in mouse model of rheumatoid arthritis. We were able to show that MZB cells modulate the production of inflammatory cytokines. The varying number of T and B-cells had an effect on cytokine production. Higher number of B-cells suppressed many of the cytokines investigated. B-cells did not produce any cytokines when cultured alone but did so when cultured with T-cells. This finding needs to be reconfirmed by further experiments. The results might illuminate the breakage of tolerance by B-cells and their role in mediating an autoimmune reaction which might help in finding some answers for disease treatment.