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BIFUNCTIONAL PEPTIDE INHIBITORS FOR CONTROLLING AUTOIMMUNE DISEASES

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Bifunctional peptide inhibitors (BPI) are conjugates between antigenic peptides and cell adhesion peptides or protein. The hypothesis is that BPI molecules simultaneously bind to MHC-II and ICAM-1 on antigen-presenting cells (APC) to inhibit the formation of the immuno¬logical synapse at the interface between T cells and APC followed. Therefore, this inhibition induces selective alteration of T-cell differentiation from inflammatory to regulatory responses. Our results showed that BPI molecules suppressed experimental autoimmune encephalomyelitis (EAE) disease in mice significantly better than antigenic peptide (i.e., PLP peptide) or PBS. BPI molecules have been shown to suppress rheumatoid arthritis in collagen-induced arthritis mice significantly better than antigenic peptide (i.e., Collagen-II peptide) or PBS. In EAE mice, BPI molecules suppressed the production of inflammatory cytokines and induced the production of regulatory and/or suppressor cytokines. We have also shown that BPI molecules suppressed EAE in antigen specific manner. Currently, we are working on understanding the mechanisms of action of BPI molecules in suppressing autoimmune diseases in animal models.

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