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Determining the mechanism and scope of a novel immunomodulatory peptide (C1) in Rheumatoid arthritis

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Introduction: Rheumatoid arthritis (RA) is an autoimmune disease of unknown aetiology. The initial event is T-cell driven followed by macrophage infiltration leading to cytokine secretion, chronic inflammation and joint destruction. Most recent therapies therefore aim to neutralize key cytokines such as TNF or IL-6 and or inhibition of tyrosine kinases associated with T-cell signaling. We have developed a novel approach to inhibit the T-cell antigen mediated receptor (TCR) signaling by means of peptides. The initial key peptide termed "core peptide, CP" derived from the transmembrane region of TCR-a chain that inhibits T-cell mediated responses *in vitro* and *in vivo*. To enhance the usage of CP as a therapeutic, D-amino acids and cyclization of peptide was carried out. The new peptide C1 was found to be more effective in treating arthritic rats than CP.

Aim: To determine the effect of C1 on cytokine expression profile.

Methodology: To understand the role of C1 in cytokine regulation we stimulated 2B4.11 hybridoma T-cell line and LK35.2, antigen presenting cells with pigeon cytochrome C (in an antigen presentation assay) in presence and absence of peptides. Cell free supernatants were assessed for secreted cytokine levels.

Results & Conclusion: We observed that C1 and CP inhibited IL-2 production, key cytokine required for T-cell activation and proliferation. Along with this we also observed down regulation of inflammatory cytokines IL-6, TNF-a, and CCL5. However an interesting phenomenon observed was induction of IL-10 (regulatory cytokine) as a result of CP treatment; thereby suggesting a potential role of C1 and CP as new class of small molecule for treating inflammation. These are exciting possibilities with significant biological and therapeutic potential worthy of further investigation.

Characterization of the interactions between fish serum proteins and pathogenic bacteria by using label-free quantitative proteomics

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The objectives of this study are to systematically characterize the serum proteins from fish that stably bind to bacteria and to investigate how bacteria respond to these interactions. Serum from turbot, but not the heat inactivated control, significantly decreased the number of viable *E. tarda* bacteria. By conjugating fish serum proteins with fluorescent dyes, we showed that *E. tarda* were coated with multiple labeled proteins before lysis. In order to systematically identify these bacteria binding proteins, we used living *E. tarda* cells to capture turbot serum proteins and subjected the samples to shotgun proteomic analysis, followed by label-free quantitation. A total of 76 fish proteins have been identified, including known antimicrobial proteins such as immunoglobins, plasminogen, complement components and Wap65-2.34 proteins with no previously known immunological function were also identified to bind to *E. tarda*. This approach also allowed the study of the proteomic changes in *E. tarda* exposed to turbot serum. Our data indicated a rapid decrease of translation factors and an up regulation of membrane components in response to serum treatment. Interestingly, fish serum induced a rapid suppression of the expression of bacterial antioxidant enzymes like catalase and led to an accumulation of reactive oxygen species, suggesting that fish serum can override a bacterial self defense mechanism. Taken together, this work offers a comprehensive view of the interactions between fish serum proteins and pathogenic bacteria and reveals previously unknown factors and mechanisms in fish innate immunity.