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Anti-inflammatory activity of a CD44 variant-specific antibody and the CD44 variant derived peptide

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We identified a CD44 variant (designated CD44vRA), which is expressed mostly, if not exclusively, in the joint inflamed cells of rheumatoid arthritis (RA) patients. The CD44vRA variant was used to generate anti-CD44vRA monoclonal antibody (mAb) and CD44vRA-derived 5-mer peptide obtained from the functional pathological sequence of this CD44 variant. Both the anti-CD44vRA mAb and the 5-mer peptide showed an efficient anti-inflammatory response in collagen-induced arthritis (CIA) mouse model, as they renewed the normal anatomy and function of the damaged tissue. Injection of the antibody or the peptide after the onset of the disease substantially reduced the inflammation as indicated by blind analysis of footpad swelling and histopathology of joint sections. The anti-inflammatory effect of these reagents was autoimmune-specific (influence on the normal immune response was not detected) and injection of the peptide did not generate neutralizing antibodies. Mass Spectrometry analysis revealed a protein, serum amyloid A (SAA), as a potential target for the anti-inflammatory activity of the 5-mer peptide. The target protein, SAA, which was *in vitro* neutralized by the peptide, is highly involved not only in the pathology of rheumatoid arthritis but also in the pathologies of Alzheimer's disease, cancer diseases and cardiovascular diseases. This finding provides additional indications for the therapeutic potential of the antibody and peptide. The target protein supports cell proliferation in the RA model, explaining why the 5-mer peptide is effective in the inhibition of joint inflammation, as cell proliferation is an essential element of the inflammation cascade.

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Antibodies are not required to a protective immune response against dengue virus elicited in a mouse encephalitis model

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Neutralizing antibodies have been considered prerequisite in the control of Dengue virus (DENV) infection. However, T-lymphocytes have also been shown to be important in a protective immune state. To investigate the contribution of both, humoral and cellular immune responses in DENV immunity, we used an experimental model in which a non-lethal DENV2 strain (ACS46) is used to prime Balb/C mice which develop protective immunity against a lethal DENV2 strain (JHA1). Primed mice generated envelope-specific antibodies and CD8+ T cell responses targeting mainly non-structural proteins. Immune sera from protected mice did not confer passive protection to naive mice challenged with the JHA1 strain. In contrast, depletion of CD4+ and CD8+ T-lymphocytes significantly reduced survival of ACS46-primed mice challenged with the JHA1 strain. Collectively, results presented in this study show that a cellular immune response targeting non-structural proteins are a promising way in vaccine development against dengue.

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