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In vivo expression of recombinant pregnancy-specific glycoprotein 1a inhibits the symptoms of collagen-induced arthritis.

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Abstract

PROBLEM:

The contribution of Pregnancy-specific glycoproteins (PSG), the major variant of PSG released into the circulation during pregnancy, to the pregnancy-dependent improvement of rheumatoid arthritis (RA) has still not been elucidated.

METHOD OF STUDY:

Collagen-induced arthritis (CIA) was used to test the hypothesis that PSG1a when released into circulation has a modulatory role on the Th1-pathogenic response, thus improving the CIA symptoms. In vivo expression of PSG1a was induced by injection of the vaccinia (Vac)-based expression vector harboring the complete open-reading frame of PSG1a cDNA.

RESULTS:

In vivo PSG1a expression during the induction of CIA ameliorated the clinical symptoms, thereby reducing the arthritis score and incidence. Significantly lower levels of IL-17, IL-6, and IFN-γ, but higher levels of TGF-β and IL-10 were secreted by collagen type II-stimulated spleen mononuclear cells from Vac-PSG1a-treated mice compared with control mice. Moreover, Vac-PSG1a treatment promoted the increase in splenic CD4+CD25+Foxp3+ Treg cells.

CONCLUSION:

Pre-clinical Vac-PSG1a treatment suppressed the Th1- and Th17-type-specific responses, leading to an increase in splenic Treg cells as well as IL-10- and TGF-β-secreting cells, with the CIA symptoms being ameliorated.

KEYWORDS:

Collagen induced arthritis; immunoregulation; pregnancy specific glycoproteins; regulatory T cells; rheumatoid arthritis

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