Modulation of Protein Tyrosine Phosphatase Receptor Type A (PTPRA) to Treat Arthritis

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BACKGROUND

Fibroblast-like synoviocytes (FLS) in the intimal lining of the joint synovium control the composition of the synovial fluid and extracellular matrix (ECM) of the joint lining. In rheumatoid arthritis (RA), FLS become aggressive and invasive, contributing to many aspects of RA pathology. FLS produce matrix metalloproteinases (MMPs) that break down the ECM, directly invade and digest the articular cartilage, promote bone erosion, and promote inflammation through secretion of interleukin 6 (IL-6), chemokines, and other inflammatory mediators. FLS are highly sensitive to the inflammatory environment present in rheumatoid joints. Growth factors, especially platelet-derived growvth factor (PDGF), stimulate FLS invasiveness. Inflammatory cytokines, particularly tumor necrosis factor-alpha (TNF) and interleukin-I (IL-1), enhance FLS aggressiveness, pro-inflammatory features and MMP production. Targeting of molecules that control FLS invasiveness and inflammatory output is being considered an option for development of new therapies for RA.

Many signaling pathways controlling FLS behavior rely upon phosphorylation of proteins on tyrosine residues, which results from the balanced action of protein tyrosine kinases (PTKs) and phosphatases (PTPs). We found that a protein (PTPRA) belonging to a novel and currently untapped class of drug targets is present at high levels in cells lining the joints of RA patients, where we believe it promotes the aggressive behavior of these cells in joint inflammation and destruction.

TECHNOLOGY DESCRIPTION

Researchers at UC San Diego have developed a patented method of inhibiting PTPRA protein activity in a cell, using an effective amount of a PTPRA antisense oligonucleotide thereby inhibiting the expression of the phosphatase.

APPLICATIONS

The PTPRA antagonist is designed to be used in the treatment of a subject with autoimmune disease in need of therapeutic administration of the PTPRA antagonist for decreasing inflammation in the synovium.

ADVANTAGES

This represents a novel method of treating autoimmune disease in RA patients.

STATE OF DEVELOPMENT

Ptpra KO mice are protected from inflammation during K/BxN passive transfer arthritis.

INTELLECTUAL PROPERTY INFO

The technology is available for licensing and has an Issued patent US20170247469A1

PATENT STATUS

Country	Type	Number	Dated	Case
United States Of America	Issued Patent	10,604,585	03/31/2020	2015-127

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OTHER INFORMATION

KEYWORDS

Receptor tyrosine-protein phosphatase alpha (PTPRA), rheumatoid arthritis, inflammation, Fibroblast-like synoviocytes, antagonist, RA, protein tyrosine kinases, signal transduction cascades

CATEGORIZED AS

- Medical
  - Disease: Autoimmune and Inflammation
  - Therapeutics

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