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Context

The complement system plays an important role in the pathogenesis of rheumatoid arthritis (RA). Soluble complement receptor 1 (sCR1) regulates complement activity, and may be useful as a therapeutic agent. This study tests the hypothesis that delivery of genes encoding truncated sCR1 (tsCR1) is an effective treatment for collagen-induced arthritis (CIA), a murine model for RA.

Significant findings

DBA/1 mice immunized with bovine type II collagen, then injected with syngeneic, immortalized fibroblasts transduced with tsCR1, developed less severe CIA and had less abnormal histological scores than control mice. The authors conclude that gene therapy with tsCR1 is a feasible therapy for CIA.

Comments

This work expands gene therapy for CIA to include the complement regulatory protein CR1, and focuses attention on the role of complement inhibition in the treatment of RA. Future efforts to apply such treatments to humans with RA may be beneficial. Whether complement inhibition will have the same pivotal effect as antagonism of tumor necrosis factor- \hat{I}^2 and IL-1 remains to be seen, and infectious complications are a concern.

Methods

Gene therapy via injection of transduced immortalized fibroblasts

References

1. Dreja H, Annenkov A, Chernajovsky Y: Soluble complement receptor 1 (CD 35) delivered by retrovirally infected syngeneic cells or by naked DNA injection prevents the progression of collagen-induced arthritis. *Arthritis Rheum.* 2000, 43: 1698-1709.