

The use of animal models in rheumatoid arthritis research

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The pathological hallmark of rheumatoid arthritis (RA) is a synovial pannus that comprises proliferating and invasive fibroblast-like synoviocytes, infiltrating inflammatory cells, and an associated neoangiogenic response. Animal models have been established to study these pathological features of human RA. Spontaneous and induced animal models of RA primarily reflect inflammatory aspects of the disease. Among various induced animal models, collagen-induced arthritis (CIA) and collagen antibody-induced arthritis (CAIA) models are widely used to study the pathogenesis of RA. Improved transplantation techniques for severe combined immunodeficiency (SCID) mouse models of RA can be used to evaluate the effectiveness of potential therapeutics in human tissues and cells. This review provides basic information on various animal models of RA, including CIA and CAIA. In addition, we describe a SCID mouse coimplantation model that can measure the long-distance migration of human RA synoviocytes and cartilage destruction induced by these cells.

Keywords: Animal models; Cell movement; Rheumatoid arthritis; Severe combined immunodeficient mice; Synoviocytes

Introduction

Rheumatoid arthritis (RA) is a common autoimmune disorder that affects approximately 1% of the global population. It is characterized by tumor-like expansion of the synovium, angiogenesis, and destruction of the articular cartilage and bone [1]. Despite the advent of anticytokine therapies that ameliorate the inflammatory manifestations of RA, there is no curative treatment and the pathogenesis of the disease is not fully understood. In RA joints, various cell populations, including innate immune cells, adaptive immune cells, endothelial cells, and fibroblast-like synoviocytes (FLSs), are activated [1]. Resident synoviocytes participate in the chronic inflammatory response and joint destruction in RA, and in fact,

these cells represent the major cell population in the invasive pannus [2]. RA-FLSs have the potential to secrete matrix-degrading enzymes (matrix metalloproteinase 3 [MMP3] and MMP9), pro-inflammatory cytokines (interleukin 1 [IL-1] and IL-6), chemokines (IL-8 and C-C motif chemokine ligand 2), and angiogenic factors (vascular endothelial growth factor and placental growth factor) [2]. Moreover, although RA synoviocytes are primary cells, they proliferate abnormally and display resistance to Fas-mediated apoptosis, similar to cancer cells [2,3]. During RA development, the phenotype of RA-FLSs is altered to an invasive and aggressive behavior, with increased migratory ability and reduced attachment-dependent growth, leading to articular cartilage destruction [2,4]. RA-FLSs can spread by migrating from the af-

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