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Abstract

RA (rheumatoid arthritis) is a chronic progressive polygenic multifactor autoimmune disease with a high socioeconomic burden. The regulation of cell death and proliferation is vital for homeostasis, but the mechanisms that coordinate these two events in rheumatoid arthritis (RA) remains largely unknown. In RA, the synovial lining increases through decreased cell death and/or enhanced proliferation. The aberrant decrease in apoptosis and/or increased cell cycle activity of the fibroblast-like and macrophage-like synoviocytes is responsible for the synovial hyperplasia and contributes to the destruction of cartilage and bone. Besides synovial cells, T cells, and mast cells also increase in number and life span, resulting in the increase of macrophage activation, and production of autoantibodies. Recently, numerous molecules that modulate apoptosis and cell cycle have been implicated to play a role in RA. This review will describe the current understanding of the molecular mechanisms that govern apoptosis and cell cycle, their relationship to RA pathogenesis, and the potential intervention in the treatment of RA.

Keyword: potential, intervention, apoptosis, cell, cycle, Rheumatoid, Arthritis,

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