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The Pathogenesis of Rheumatoid Arthritis—Breakthroughs in Molecular Mechanisms

Guest Editor:

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Message from the Guest Editor

The pathogenesis of rheumatoid arthritis (RA) consists of the formation of synovial villi, inflammation, immune abnormalities, and bone–cartilage destruction. According to these pathogenesis findings, conventional therapy was empirically performed using conventional synthetic disease-modifying antirheumatic drugs (csDMARDs). However, since the 1990s, pathogenesis investigations have advanced to include the cloning of IL-6, clarifying the role of IL-17 • Th17 in bone destruction; the cloning of RANKL; anti-RANKL Ab in the therapy of RA in Japan; and the introduction of “Osteoimmunology”. In addition, therapies using biological DMARDs have resulted in breakthroughs in pathogenesis investigations; the inhibition of the function of a specific molecule by an antibody has clarified its function in vivo, such as “knock-out in vivo in human”. Recently, inflammatory cell states have been clarified in RA joint synovial tissues by integrating single-cell transcriptomics and mass cytometry. In this Special Issue, both original and review papers present important advances in molecular investigations of RA pathogenesis.

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Message from the Editor-in-Chief

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