Special Issue

The Pathogenesis of Rheumatoid Arthritis—Breakthroughs in Molecular Mechanisms

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.

Guest Editor

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The International Journal of Molecular Sciences (*IJMS*, ISSN 1422-0067) is an open access journal, which was established in 2000. The journal aims to provide a forum for scholarly research on a range of topics, including biochemistry, molecular and cell biology, molecular biophysics, molecular medicine, and all aspects of molecular research in chemistry. *IJMS* publishes both original research and review articles, and regularly publishes special issues to highlight advances at the cutting edge of research. We invite you to read recent articles published in *IJMS* and consider publishing your next paper with us.

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