

Mathematical modeling of immunopathogenesis of rheumatoid arthritis

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Rheumatoid arthritis is a systemic autoimmune disease characterized by the joint inflammation and the cartilage destruction. Autoreactive B lymphocytes represent integral elements of the pathophysiology of rheumatoid arthritis. Immune balance between the effector and the regulatory T cell subsets guide the production of autoantibodies by B lymphocytes and, therefore, play a cardinal role in disease severity. While targeted therapeutic approaches are successfully emerging in medical practice, refined personalized analyses of T and B lymphocyte subsets in patients with rheumatoid arthritis are critically needed for rigorous disease management.

Mathematical models of immune mediated disorders provide an analytic framework in which we can address specific questions concerning disease immune dynamics and the choice of treatment. Herein, we present a novel mathematical model that describes the immunopathogenesis of rheumatoid arthritis using non-linear differential equations. The model explores the functional dynamics of cartilage destruction during disease progression, in which a system of differential equations deciphers the interactions between autoreactive B lymphocytes and T helper cells. Immunomodulatory relation between pro-inflammatory and regulatory T lymphocyte subsets is also solved in these equations. Of importance, our model provides a mechanistic interpretation of targeted immunotherapy which deals with the intervention of pathophysiological immune processes in rheumatoid arthritis.

In conclusion, we propose a novel mathematical model that best describes the immunopathogenic dynamics in patients with rheumatoid arthritis and, therefore, may take a rapid pace towards its implementation in biomedical and clinical research.

References:

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