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Targeted therapy of autoimmune diseases using monoclonal antibodies

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Autoimmune diseases are chronic immune-mediated disorders characterized by loss of tolerance to self-antigens and persistent inflammation that leads to progressive tissue damage. Rheumatoid arthritis, multiple sclerosis, and Crohn's disease belong to the most clinically significant autoimmune conditions because of their chronic course, relapsing activity, and major impact on quality of life. Over the last decades, monoclonal antibodies (mAbs) have become one of the most important classes of biological therapeutics in modern medicine. Their clinical value is based on high specificity toward defined molecular targets and the possibility of selective modulation of pathogenic immune pathways rather than nonspecific immunosuppression. European Medicines Agency (EMA) recognizes mAbs as a large and important class of therapeutic biologicals with a very broad range of clinical indications, while also emphasizing that many mAbs are associated with unwanted immunogenicity that may impair response or contribute to adverse reactions. The therapeutic significance of mAbs in autoimmune diseases is determined by their ability to interfere with key pathogenic mechanisms.

In rheumatoid arthritis, one of the central molecular drivers is tumor necrosis factor alpha (TNF-α), which sustains synovial inflammation, pannus formation, and progressive joint destruction. Anti-TNF mAbs such as adalimumab and infliximab substantially reduce inflammatory activity and slow structural damage. In addition, targeting the interleukin-6 receptor with tocilizumab provides another effective strategy because IL-6 contributes to synovial inflammation, systemic manifestations, and chronic immune activation. B-cell depletion with rituximab is clinically relevant in patients with severe or refractory disease, confirming the pathogenic role of B cells in autoantibody production and antigen presentation.

In multiple sclerosis, the modern mAb-based therapeutic strategy is especially strongly associated with B-cell-directed treatment. Anti-CD20 mAbs therefore became one of the most important advances in disease-modifying therapy. In the OPERA I and II trials, ocrelizumab was associated with lower disease activity and less progression than interferon beta-1a in relapsing multiple sclerosis, while in the ORATORIO trial it reduced progression compared with placebo in primary progressive disease.

In Crohn's disease, targeted therapy with mAbs has transformed the treatment of moderate-to-severe inflammatory bowel disease. TNF-α is one of the pivotal cytokines in intestinal inflammation, and infliximab became one of the landmark antibodies proving that selective biologic intervention can induce and maintain remission. Clinical studies showed that infliximab supports sustained response and steroid-sparing effects when used as maintenance therapy. Adalimumab later extended this anti-TNF strategy. Another important direction is blockade of leukocyte trafficking: vedolizumab targets α4β7 integrin and limits migration of inflammatory cells into gut mucosa, thereby reducing tissue damage with a more gut-selective mechanism. The pivotal studies of infliximab in Crohn's disease established anti-TNF antibodies as a cornerstone of targeted therapy in this indication.

mAbs represent a key strategy in the targeted treatment of autoimmune diseases by enabling precise intervention in specific immunopathological pathways. The analysis of rheumatoid arthritis, multiple sclerosis, and Crohn's disease demonstrates that different molecular targets, including TNF-α, interleukins, CD20, and integrins, determine the effectiveness of therapy depending on disease pathogenesis. The success of monoclonal antibody therapy highlights the transition from nonspecific immunosuppression to mechanism-based treatment approaches. However, issues such as immunogenicity, variability of clinical response, and safety concerns remain significant challenges. Thus, future development of monoclonal antibodies should focus on improving selectivity, reducing adverse effects, and optimizing individualized treatment strategies, which will further strengthen their role in modern targeted and personalized therapy.