



Current Perspectives and Emerging Trends in Rheumatoid Arthritis Therapeutics

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ABSTRACT

Rheumatoid arthritis (RA) is a chronic, progressive, systemic autoimmune disease characterized by persistent synovial inflammation, joint destruction, disability, and multiple extra-articular complications. The disease affects approximately 0.5–1% of the global population and occurs two to three times more frequently in women than in men. RA pathogenesis involves a complex interaction between genetic susceptibility, environmental exposures, immune dysregulation, and chronic inflammatory pathways. Important genetic risk factors include female gender, family history, and HLA genotypes, whereas environmental triggers include smoking, silica exposure, periodontal disease, infections, pesticides, and chemical exposure. Disease progression may begin years before clinical symptoms appear due to asymptomatic synovitis and autoantibody production. Clinically, RA commonly presents as symmetric polyarthritis involving small joints accompanied by morning stiffness, pain, fatigue, swelling, reduced mobility, and chronic functional impairment. Extra-articular manifestations may involve cardiovascular, pulmonary, gastrointestinal, ocular, psychological, and skeletal systems, significantly reducing quality of life and increasing mortality risk. Early diagnosis and prompt therapeutic intervention are essential to prevent irreversible joint destruction and disability. Current treatment strategies emphasize a treat-to-target approach aimed at achieving remission or low disease activity. Conventional therapies include nonsteroidal anti-inflammatory drugs, glucocorticoids, and conventional synthetic disease-modifying antirheumatic drugs such as methotrexate, sulfasalazine, hydroxychloroquine, and leflunomide. Biologic DMARDs targeting tumor necrosis factor-alpha, interleukin-6, B cells, and T-cell co-stimulation pathways have significantly improved outcomes in refractory patients. Targeted synthetic DMARDs, particularly Janus kinase inhibitors, provide additional oral therapeutic options for disease control. Emerging therapies including liposomal drug delivery systems, mesenchymal stem cell therapy, precision medicine, and gut microbiota modulation show promising future potential. Complementary approaches such as dietary modification, physiotherapy, acupuncture, moxibustion, and Traditional Chinese Medicine may further support symptom control and functional improvement. Despite major advances, multidisciplinary and patient-centered management remains essential for improving long-term outcomes and quality of life in patients with rheumatoid arthritis.

Keywords: Rheumatoid arthritis, Synovial inflammation, Autoimmune disease, Biologic DMARDs, Janus kinase inhibitors, Liposomal drug delivery systems.

INTRODUCTION

Rheumatoid arthritis (RA) is a chronic, inflammatory, systemic autoimmune disorder that leads to progressive disability, systemic complications, and an increased risk of early mortality. RA is marked by synovial inflammation and hyperplasia, the production of autoantibodies such as rheumatoid factor (RF) and anti-citrullinated protein antibody (ACPA), as well as deformities in cartilage and bone. Additionally, it presents systemic features that can affect cardiovascular, pulmonary, psychological, skin, and skeletal health. Most epidemiological research on RA has been carried out in Western nations, revealing a prevalence of RA between 0.5% and 1.0% in the United States.¹

Rheumatoid arthritis (RA) is recognized as an autoimmune condition with a global prevalence of roughly 0.5% to 1% among the adult population.² Women are 2 to 3 times more likely to develop RA compared to men, with the cumulative lifetime risk of adult-onset RA estimated at 3.6% for women and 1.7% for men.¹ When RA is not effectively managed, patients may suffer from joint deterioration, significant

disability, a reduced quality of life, comorbid conditions, and an increased risk of premature death.²

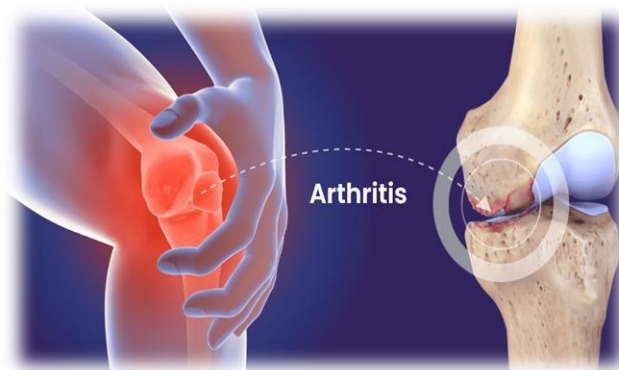


Figure 1: Arthritis

(Source: <https://kansaspainmanagement.com/conditions/arthritis/> accessed on 28-05-2026)

Pathogenesis and risk factors of rheumatoid arthritis

The etiology and pathophysiology of rheumatoid arthritis (RA) remain incompletely elucidated; however, it seems to involve an autoimmune assault on the joints of bone and



cartilage. RA predominantly impacts the synovial joint membranes, resulting in disability, premature mortality, and a significant socioeconomic impact.³ Both genetic predispositions and environmental influences play a role in the development of RA, with multiple risk factors potentially necessary to reach the threshold that triggers the condition. Key environmental risk factors for RA include smoking, exposure to silica, and periodontal disease.¹

Tobacco use is recognized as one of the principal established environmental risk factors, as it induces oxidative stress that can lead to the onset of rheumatoid arthritis.⁴ Additionally, exposure to pesticides, solvents, and chemical fertilizers has been linked to the progression of the disease.³ Genetic contributors encompass female sex, a family history of the condition, older age, and specific HLA genotypes.⁴ Furthermore, infections from *Mycoplasma*, Epstein–Barr virus, cytomegalovirus, rubella virus, and *Porphyromonas gingivalis* may also play a role in the initiation of the disease.^{4,6} The progression of the disease can include asymptomatic synovitis and the spread of autoimmunity years prior to the manifestation of clinical symptoms.¹

Clinical manifestations and complications of rheumatoid arthritis

Rheumatoid arthritis (RA) is a damaging form of symmetric polyarthritis that primarily targets the small joints, resulting in a decline in physical function and chronic pain, which adversely affects the quality of life. The clinical features of RA include morning stiffness, discomfort in the shoulder, neck, and pelvic girdle, reduced mobility accompanied by fever, fatigue, malaise, weight loss, and the formation of rheumatoid nodules.³

Symptoms associated with the condition encompass pain during passive movement, swelling, a sensation of heat, and firmness in the mornings that persists for more than an hour. Rheumatoid nodules are frequently found on the scalp, back, feet, hands, buttocks, knees, heart valves, lungs, and spleen.⁴ Extra-articular manifestations may involve cardiovascular diseases, gastrointestinal disorders, infections, depression, strokes, Raynaud's phenomenon, and ocular issues such as scleritis.^{2,4}

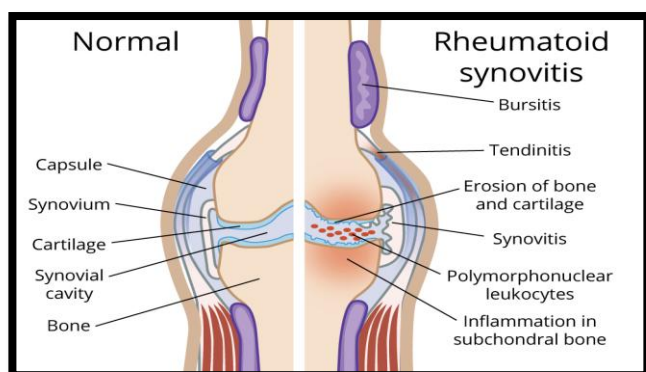


Figure 2: Rheumatoid arthritis

(Source: <https://orthoinfo.aaos.org/en/diseases--conditions/rheumatoid-arthritis/> accessed on 28-05-2026)

RA is classified into two categories: seropositive and seronegative, which are determined by the presence or absence of elevated serum levels of rheumatoid factor and anti-citrullinated protein antibodies.⁵ Accurate differential diagnosis is crucial, as the symptoms of RA can overlap with those of other conditions such as lupus, psoriatic arthritis, gout, and Sjögren's syndrome.⁶

TREATMENT OF RHEUMATOID ARTHRITIS

Treatment of Rheumatoid Arthritis has evolved significantly over recent decades with advances in understanding immune mechanisms and inflammatory pathways.^{7,8} Modern therapeutic approaches focus on reducing inflammation, preventing joint destruction, maintaining functional ability, and improving quality of life.^{9,10} Current management strategies emphasize early diagnosis and prompt initiation of therapy because irreversible joint damage may occur during the early stages of disease.⁹ The treat-to-target approach, which aims for remission or low disease activity, has become a standard principle in RA management.^{8,9} Treatment selection depends on disease severity, prognostic factors, patient preference, comorbidities, and response to previous therapies.^{9,11}

Nonsteroidal anti-inflammatory drugs (NSAIDs)

Nonsteroidal anti-inflammatory drugs are frequently prescribed to relieve pain, swelling, and stiffness in RA patients.^{10,12} These drugs inhibit cyclooxygenase enzymes and reduce prostaglandin-mediated inflammation. Commonly used NSAIDs include ibuprofen, naproxen, diclofenac, and celecoxib.¹⁰ Although NSAIDs provide symptomatic relief, they do not alter disease progression or prevent structural joint damage.¹² Long-term NSAID therapy may cause gastrointestinal ulceration, bleeding, renal impairment, and cardiovascular complications.¹⁰ Because of these risks, NSAIDs are generally used at the lowest effective dose for the shortest possible duration.⁹

Glucocorticoids

Glucocorticoids remain important anti-inflammatory agents in RA treatment.^{9,12} Corticosteroids rapidly suppress synovial inflammation and reduce pain and swelling.¹⁰ They are commonly used as bridging therapy while waiting for DMARDs to achieve therapeutic effects.⁹ Prednisone and methylprednisolone are frequently prescribed systemic corticosteroids in RA management.¹⁰ Intra-articular corticosteroid injections may also provide local symptom control in inflamed joints.¹² Short-term corticosteroid use is effective in controlling acute disease flares.⁹ However, prolonged corticosteroid therapy is associated with adverse effects including osteoporosis, hypertension, diabetes mellitus, cataracts, adrenal suppression, and increased infection risk.¹⁰ Current treatment recommendations emphasize minimizing long-term steroid exposure whenever possible.⁹

Conventional synthetic disease-modifying antirheumatic drugs (csDMARDs)

These remain the cornerstone of RA therapy.^{7,10} These agents suppress immune activity and slow disease progression.¹⁰

1. Methotrexate

Methotrexate is considered the anchor drug because of its proven efficacy, affordability, and favorable long-term safety profile.^{9,10} Methotrexate inhibits folate metabolism and suppresses inflammatory immune responses.¹⁰ The drug effectively reduces synovial inflammation, decreases radiographic progression, and improves functional outcomes.⁷ Methotrexate may be administered orally or subcutaneously depending on patient tolerance and response. Folic acid supplementation is commonly prescribed to reduce methotrexate toxicity. Potential adverse effects include hepatotoxicity, gastrointestinal symptoms, bone marrow suppression, pulmonary toxicity, and teratogenicity.¹⁰ Regular monitoring of liver function tests and blood counts is therefore essential during therapy.⁹

2. Sulfasalazine

Sulfasalazine is another csDMARD frequently used in RA treatment. The drug possesses anti-inflammatory and immunomodulatory properties.¹⁰ Sulfasalazine is often used in combination with methotrexate for enhanced therapeutic efficacy.⁷ Common side effects include gastrointestinal upset, rash, headache, and reversible oligospermia.¹⁰

3. Hydroxychloroquine

Hydroxychloroquine is considered a relatively mild csDMARD and is commonly used in patients with early or less severe disease. The drug interferes with antigen presentation and immune activation. Hydroxychloroquine is generally well tolerated and may improve fatigue and constitutional symptoms. Retinal toxicity is a rare but important complication requiring periodic ophthalmologic monitoring.¹⁰

4. Leflunomide

It inhibits pyrimidine synthesis and suppresses lymphocyte proliferation.¹⁰ It is an effective alternative for patients who cannot tolerate methotrexate.⁷ Leflunomide improves clinical symptoms and delays structural joint damage. Common adverse effects include hepatotoxicity, hypertension, diarrhoea, and teratogenicity.¹⁰

Combination csDMARD therapy is frequently recommended in patients with moderate to severe disease activity.^{7,9} Triple therapy using methotrexate, sulfasalazine, and hydroxychloroquine has shown substantial efficacy in clinical trials.⁷ Early aggressive use of csDMARD combinations may improve long-term outcomes and reduce disability.⁹ Biologic DMARDs have revolutionized the management of RA by specifically targeting inflammatory cytokines and immune pathways.^{7,8} These agents are

generally recommended for patients with inadequate response to csDMARD therapy.⁹ Biologics significantly reduce disease activity, inhibit radiographic progression, and improve quality of life.⁷

Tumor necrosis factor-alpha (TNF- α) inhibitors

These were the first biologic agents approved for RA treatment.⁷ TNF- α is a major inflammatory cytokine involved in synovial inflammation and joint destruction.^{1,7} Blocking TNF- α reduces inflammatory cell recruitment and suppresses osteoclast activation.⁷ Common TNF inhibitors include etanercept, infliximab, adalimumab, certolizumab pegol, and golimumab.^{7,8} Clinical studies have demonstrated substantial improvements in pain, physical function, and radiographic outcomes with TNF inhibitor therapy.⁷ Combination therapy with methotrexate often enhances the efficacy of TNF inhibitors.⁹ Despite their effectiveness, TNF inhibitors increase susceptibility to infections including tuberculosis and opportunistic infections.⁷ Screening for latent tuberculosis is recommended before initiating biologic therapy.⁹

Interleukin-6 (IL-6) inhibitors

IL-6 inhibitors represent another important class of biologic agents. IL-6 contributes to synovial inflammation, systemic symptoms, anaemia, and bone destruction in RA. Tocilizumab is a monoclonal antibody targeting the IL-6 receptor. The drug effectively reduces disease activity and improves physical function in moderate to severe RA. Tocilizumab may be administered intravenously or subcutaneously. It is effective both as monotherapy and in combination with methotrexate. Patients with inadequate response to TNF inhibitors may benefit from IL-6 inhibition.^{7,13} Potential adverse effects include neutropenia, elevated liver enzymes, hyperlipidemia, and increased infection risk.¹³

B-cell depletion therapy

It has become an important option for patients with refractory RA.¹⁴ B cells contribute to disease pathogenesis through autoantibody production and cytokine secretion.^{1,14} Rituximab is a monoclonal antibody directed against CD20-positive B cells. The drug reduces autoantibody levels and suppresses inflammatory activity. Rituximab is particularly effective in seropositive RA patients who fail TNF inhibitor therapy. Combination therapy with methotrexate enhances treatment outcomes. Infusion reactions and infections are among the major adverse effects associated with rituximab therapy.¹⁴

T-cell co-stimulation modulation

It is another therapeutic strategy in RA treatment. Abatacept inhibits T-cell activation by blocking the interaction between CD80/CD86 and CD28. Suppression of T-cell activation reduces inflammatory cytokine production and synovial inflammation. Abatacept is effective in patients with inadequate response to csDMARDs or TNF inhibitors. The drug generally demonstrates a favorable safety profile



with lower rates of serious infections compared with some biologic agents.⁷

Targeted synthetic DMARDs

These have expanded therapeutic options for RA management. Unlike biologic agents, these drugs are small molecules administered orally. Janus kinase (JAK) inhibitors interfere with intracellular cytokine signaling pathways. JAK inhibitors reduce inflammatory responses by blocking signal transduction mediated by multiple cytokines. Common JAK inhibitors include tofacitinib, baricitinib, and upadacitinib. These agents provide rapid symptom relief and improved patient convenience because of oral administration. Clinical studies have demonstrated efficacy in patients refractory to methotrexate and biologic therapies. JAK inhibitors may improve pain, physical function, and radiographic progression. However, concerns remain regarding adverse effects including infections, herpes zoster reactivation, venous thromboembolism, cardiovascular events, and malignancy risk.⁸ Careful patient selection and monitoring are therefore necessary during therapy.⁹

Nanotechnology-based drug delivery systems

These are being explored to improve RA treatment efficacy. Liposomes are phospholipid vesicles capable of encapsulating therapeutic agents. Liposomal drug delivery may enhance bioavailability and reduce systemic toxicity. Inflamed synovial tissues demonstrate increased vascular permeability, allowing preferential accumulation of liposomal formulations. Liposome-encapsulated corticosteroids and methotrexate have shown promising anti-inflammatory effects in experimental studies. Targeted liposomal systems may improve therapeutic outcomes while minimizing adverse effects. Although further clinical studies are needed, liposome-based therapies represent a promising future direction in RA management.¹⁵

Patient-centered care

Patient-centered care has become increasingly important in RA treatment. Patients differ considerably in treatment goals, risk tolerance, and preferences regarding route of administration. Some patients prioritize pain reduction and functional improvement, whereas others focus on minimizing adverse effects. Treatment adherence may be influenced by medication cost, injection frequency, convenience, and perceived efficacy. Shared decision-making between physicians and patients improves treatment satisfaction and clinical outcomes. Educational interventions also help patients better understand disease progression and treatment expectations.¹¹

Dietary interventions

This may provide supportive benefits in RA management.¹⁶ Chronic inflammation in RA may be influenced by dietary habits and gut microbiota composition.^{16,17} Omega-3 fatty acids found in fish oil possess anti-inflammatory properties. Supplementation with omega-3 fatty acids may reduce joint tenderness, morning stiffness, and NSAID requirements. The Mediterranean diet emphasizes fruits, vegetables, fish,

olive oil, and whole grains rich in antioxidants and anti-inflammatory compounds. Studies suggest that adherence to the Mediterranean diet may improve disease activity and cardiovascular health in RA patients. Vitamin D supplementation may also support immune regulation and bone health.¹⁶

The gut microbiota

The gut microbiota has emerged as an important factor in RA pathogenesis and treatment. Alterations in intestinal microbial composition may contribute to immune dysregulation and chronic inflammation.¹⁷ Probiotics and dietary interventions aimed at restoring microbial balance may exert beneficial immunomodulatory effects.^{16,17} Traditional Chinese Medicine (TCM) may also influence gut microbiota composition and inflammatory pathways.¹⁷

Acupuncture and moxibustion

Acupuncture and moxibustion are complementary therapies increasingly used by RA patients. Research suggests that acupuncture may regulate cytokine production and immune responses. Acupuncture may reduce pain perception and improve joint function. Moxibustion involves thermal stimulation of acupuncture points and may exert anti-inflammatory effects. Although some studies report beneficial outcomes, further high-quality randomized controlled trials are required to establish efficacy and safety.¹⁸

Traditional Chinese Medicine includes herbal formulations, acupuncture, and holistic therapeutic approaches. Certain herbal compounds may demonstrate anti-inflammatory and immunomodulatory properties. TCM approaches may reduce inflammatory cytokine production and regulate immune cell activity. Combination of TCM with conventional therapy may provide additional symptomatic benefits in some patients. However, variability in herbal formulations and limited standardization remain important challenges.¹⁷

Lifestyle modification

It is an important component of comprehensive RA management.⁹ Smoking cessation is strongly recommended because smoking contributes to disease progression and reduced treatment response.¹ Weight management may reduce joint stress and improve overall health.⁹ Cardiovascular risk reduction is particularly important because RA patients have increased risk of cardiovascular disease.^{9,10}

Emerging therapies

It will may further improve future RA management. Mesenchymal stem cell therapy demonstrates immunomodulatory and regenerative potential. Stem cells may suppress inflammatory responses and promote tissue repair. Chimeric antigen receptor T-cell (CAR-T) therapy is being investigated for selective targeting of autoreactive immune cells. Bruton's tyrosine kinase inhibitors may suppress B-cell signaling and inflammatory pathways. Precision medicine approaches aim to individualize



treatment according to genetic, molecular, and clinical characteristics. Biomarkers predicting therapeutic response may improve treatment selection and reduce unnecessary drug exposure.⁸

Despite therapeutic advances, RA management remains challenging.^{7,8} Some patients fail to achieve remission despite multiple therapies.⁸ Long-term immunosuppression increases susceptibility to serious infections and malignancies.⁷ High treatment costs may limit access to biologic and targeted therapies in many healthcare systems.⁹ Psychological factors including depression,¹ chronic pain, and fatigue significantly affect patient quality of life.¹⁰

Multidisciplinary management involving rheumatologists, physiotherapists, nurses, dietitians, and mental health professionals is therefore essential for optimal patient care.^{9,10}

CONCLUSION

In conclusion, rheumatoid arthritis (RA) remains a major chronic autoimmune disorder associated with progressive joint destruction, systemic complications, disability, and reduced quality of life. The disease arises through a multifactorial interaction between genetic susceptibility,² environmental triggers, immune dysregulation, and inflammatory pathways. Advances in understanding RA pathogenesis have significantly transformed therapeutic strategies and improved disease outcomes over recent decades. Early diagnosis and timely intervention are now recognized as essential components in preventing irreversible joint damage and long-term functional impairment. Conventional treatment approaches, including nonsteroidal anti-inflammatory drugs, glucocorticoids, and conventional synthetic disease-modifying antirheumatic drugs, continue to play an important role in disease management. Among these agents, methotrexate remains the cornerstone of therapy because of its efficacy, affordability, and long-term clinical benefits. The introduction of biologic and targeted synthetic DMARDs has further revolutionized RA treatment by specifically targeting inflammatory cytokines and intracellular signaling pathways. These therapies have improved remission rates, reduced radiographic progression, and enhanced patient quality of life, particularly in patients with refractory disease. Emerging therapeutic approaches including liposomal drug delivery systems, stem cell therapy, precision medicine, and gut microbiota modulation represent promising future directions in RA management. Liposomal formulations may improve drug targeting to inflamed synovial tissues while reducing systemic toxicity, thereby enhancing therapeutic efficacy. Complementary interventions such as physiotherapy, dietary modification, acupuncture, moxibustion, and lifestyle changes may also contribute to better symptom control and functional improvement when combined with conventional therapy. Despite substantial progress, several challenges remain, including treatment resistance, adverse drug reactions, infection risk, high healthcare costs, and psychosocial

burden. Long-term immunosuppression and variability in treatment response continue to complicate disease management. Therefore, multidisciplinary and patient-centered care involving rheumatologists, physiotherapists, dietitians, nurses, and mental health professionals is essential for optimal outcomes. Continued research into novel therapeutic targets and individualized treatment approaches may further improve disease control, reduce disability, and enhance the overall quality of life in patients with rheumatoid arthritis.

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