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HIGHLIGHTS

▸ Dysbiosis in RA is characterized by expansion of Prevotella copri and depletion of beneficial commensals such as Lactobacillus and Bifidobacterium. ▸ Increased intestinal permeability facilitates translocation of microbial antigens, triggering systemic and synovial inflammation. ▸ Th17/Treg imbalance, molecular mimicry and reduced SCFA production are key immunological mechanisms linking gut dysbiosis to RA. ▸ The gut–joint axis integrates intestinal microbial

disturbances with joint inflammation through metabolic and immunological pathways. ▸ Dietary interventions, probiotics and fecal microbiota transplantation show therapeutic potential but require further clinical validation.

NARRATIVE REVIEW

Intestinal Microbiota and Rheumatoid Arthritis: From Pathogenesis to Potential Therapies

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ABSTRACT

BACKGROUND: Rheumatoid arthritis (RA) is a chronic autoimmune disease characterized by progressive joint inflammation and multifactorial pathogenesis. Growing evidence indicates that the intestinal microbiota plays a significant role in disease development and progression.

AIM: This review aimed to summarize key mechanisms linking gut microbiota with RA and to evaluate emerging therapeutic strategies targeting intestinal dysbiosis.

MATERIALS AND METHODS: A narrative literature review was conducted based on scientific publications indexed in PubMed between 2013 and 2025.

RESULTS: Gut microbiota contributes to immune homeostasis, while dysbiosis — defined as altered microbial composition — is associated with reduced diversity, expansion of pro-inflammatory taxa such as Prevotella copri and depletion of beneficial commensals. Key mechanisms include increased intestinal permeability, immune dysregulation with Th17/Treg imbalance, molecular mimicry and altered production of microbial metabolites, particularly short-chain fatty acids. These processes are integrated within the concept of the gut–joint axis.

CONCLUSIONS: Therapeutic strategies targeting gut microbiota, such as dietary interventions, probiotics and fecal microbiota transplantation, show promise but require further validation before routine clinical application.

KEYWORDS: intestinal microbiota; rheumatoid arthritis; dysbiosis; gut–joint axis; Prevotella copri; intestinal permeability

PLAIN LANGUAGE SUMMARY

Rheumatoid arthritis (RA) is a chronic disease in which the immune system attacks the joints, causing pain, swelling and progressive damage. Recent research shows that the trillions of bacteria living in our intestines — collectively called the gut microbiota — may play an important role in triggering and sustaining this disease. In people with RA, the balance of gut bacteria is often disturbed: harmful bacteria such as Prevotella copri become more abundant, while beneficial bacteria decrease. This imbalance can weaken the gut wall, allowing bacterial products to enter the bloodstream and activate the immune system in ways that promote joint inflammation. This review explains how the gut and the joints communicate through the "gut–joint axis" and discusses how restoring a healthy gut microbiota through diet, probiotics or fecal microbiota transplantation might help manage RA. While these approaches are promising, more research is needed before they can be recommended as standard treatments.

1. INTRODUCTION

Rheumatoid arthritis (RA) is a chronic autoimmune disease characterized by progressive joint inflammation. The pathogenesis of the disease is heterogeneous. Risk factors for RA include genetic factors, with the presence of the HLA-DRB1 allele, epigenetic factors including histone modifications and DNA methylation, and environmental factors such as smoking, obesity and the mucosa-associated microbiota (Alivernini et al., 2022; Finckh et al., 2022). Mucosal sites, such as the oral cavity, respiratory tract and intestines are lined by mucous membranes and are thought to be the origin of early autoimmune processes in RA (Gravallese & Firestein, 2023). Current treatment of RA is based on early initiation of disease-modifying antirheumatic drugs (DMARDs) and a treat-to-target strategy aimed at achieving remission or low disease activity. Conventional synthetic DMARDs, particularly methotrexate, remain the first-line therapy, while patients with inadequate response may receive biologic agents targeting cytokines or immune cells, or targeted synthetic DMARDs such as tofacitinib. These therapeutic advances have significantly improved disease control and long-term outcomes in patients with RA (Di Matteo et al., 2023; Gravallese & Firestein, 2023).

The effectiveness of therapy in RA varies considerably among patients. According to analyses using the EULAR criteria (DAS28), only about one-quarter of patients achieve a full response, nearly half experience a moderate improvement, and roughly one-third show little to no response to treatment. These findings highlight the importance of individualized therapeutic approaches and the ongoing need to develop more effective treatment strategies (Prasad et al., 2023).

2. CHARACTERISTICS OF GUT MICROBIOTA

The gut microbiota is a complex ecosystem of microorganisms inhabiting the human gastrointestinal tract, including bacteria, viruses, fungi and archaea (Thursby & Juge, 2017). The composition of gut microbiota is highly dynamic and influenced by numerous factors such as age, diet, environmental exposure, medication use and host genetics (Rinninella et al., 2019). Healthy gut microbiota is dominated by a few major bacterial phyla, especially Firmicutes and Bacteroidetes, with smaller proportions of Actinobacteria and Proteobacteria (Fujisaka et al., 2022; Rinninella et al., 2019). Gut

microorganisms contribute to host metabolism by fermenting non-digestible carbohydrates and producing short-chain fatty acids (SCFAs), which serve as energy substrates for intestinal epithelial cells (De Vos et al., 2022). Furthermore, the gut microbiota participates in vitamin synthesis (Kumar et al., 2025), bile acid metabolism and protection against colonization by pathogenic microorganisms through competitive exclusion mechanisms (Yoo et al., 2020).

Beyond metabolic activity, the gut microbiota exerts a crucial regulatory influence on both innate and adaptive immune responses. Commensal microorganisms are involved in the maturation of gut-associated lymphoid tissue, modulation of dendritic cell activity and differentiation of regulatory T cells and T helper 17 cells (Ding et al., 2024). Through these mechanisms, the intestinal microbiota contributes to the maintenance of immune tolerance and prevention of excessive inflammatory responses. Alterations in microbial composition, commonly referred to as dysbiosis, may disrupt intestinal barrier integrity, increase epithelial permeability and promote translocation of microbial antigens into systemic circulation. These processes can subsequently activate pro-inflammatory signaling pathways and have been implicated in the pathogenesis of several autoimmune disorders, including rheumatoid arthritis (Paul et al., 2025; Wasim et al., 2025). Because the microbiota is highly responsive to diet, drugs and other environmental inputs, dysbiosis is not only a marker of disturbed homeostasis but also a potential therapeutic target, motivating interventions such as diet modification, prebiotics, probiotics and fecal microbiota transplantation aimed at restoring or maintaining a health-associated microbial ecosystem (Shen et al., 2025).

3. ALTERATIONS OF INTESTINAL MICROBIOTA IN RHEUMATOID ARTHRITIS

RA is consistently associated with characteristic alterations in the intestinal microbiota. These disturbances, commonly referred to as dysbiosis, are characterized by reduced microbial diversity and changes in the relative abundance of specific bacterial taxa involved in immune homeostasis and inflammatory regulation. Increasing evidence suggests that such alterations may occur not only during established disease but also in early stages, potentially preceding clinical manifestations (Lin et al., 2023; Romero-Figueroa et al., 2023).

Prevotella copri expansion and loss of beneficial commensals form a characteristic RA-associated pattern, especially in newly diagnosed and untreated disease (Scher et al., 2013). Studies indicate that new-onset, untreated RA is associated with a marked expansion of Prevotella copri, detected in

approximately 75% of patients compared with roughly 20% of healthy controls (Bernard, 2013). RA patients generate both T-cell and B-cell responses to P. copri antigens (e.g. Pc-p27), characterized by Th1/Th17 cytokine production and disease-specific IgA and IgG antibodies, demonstrating immunological relevance at both mucosal and systemic levels (Ajith & Anita, 2025). Together, these responses suggest that P. copri may actively contribute to immune dysregulation and systemic inflammation in RA.

Human cohort studies and reviews also consistently report a reduction in beneficial commensal taxa, including Bifidobacterium, Bacteroides, Lactobacillus and other SCFA-producing bacteria (Sun et al., 2023). These microorganisms normally support epithelial barrier integrity and anti-inflammatory immune pathways, such as regulatory T cell induction, interleukin-10 production and SCFA generation. Their depletion has been linked to increased gut permeability, reduced SCFA availability and systemic inflammation, suggesting a mechanistic connection between gut dysbiosis and the pathogenesis of RA (Ajith & Anita, 2025; Sun et al., 2023).

4. MECHANISMS LINKING INTESTINAL MICROBIOTA AND RHEUMATOID ARTHRITIS

The relationship between the intestinal microbiota and rheumatoid arthritis is mediated through immunological and metabolic pathways that together promote chronic inflammation and autoimmunity. Current evidence suggests that gut dysbiosis may influence disease development by altering intestinal barrier integrity, modulating immune cell differentiation, promoting molecular mimicry and affecting the production of microbial metabolites (Xu et al., 2022).

A key pathway connecting the gut microbiota with RA involves impairment of the intestinal epithelial barrier, commonly referred to as increased intestinal permeability or "leaky gut". In healthy conditions, tight junctions between epithelial cells restrict the passage of luminal antigens and microbial products into the circulation, maintaining immune homeostasis (Longo et al., 2024). Gut dysbiosis can impair tight junction integrity and mucus layer composition, which facilitates the translocation of bacterial components such as lipopolysaccharide (LPS), peptidoglycan and microbial extracellular vesicles into the bloodstream (Longo et al., 2024; Xu et al., 2022). Once these components reach the systemic circulation, they activate innate immune pathways through receptors such as Toll-like receptors, stimulate pro-inflammatory cytokine production and contribute to both

systemic and synovial inflammation, potentially preceding and promoting the development of arthritis (Romero-Figueroa et al., 2023). Experimental studies suggest that barrier disruption can occur before clinical arthritis and that interventions restoring barrier function can reduce disease severity (Lv et al., 2025).

Another important mechanism involves immune system modulation. In RA, microbial dysbiosis is associated with an imbalance between pro-inflammatory T helper 17 (Th17) cells and regulatory T (Treg) cells (Qi et al., 2025). The shift favours expansion of Th17 cells, which enhances secretion of key inflammatory mediators such as interleukin-17, interleukin-6 and tumour necrosis factor alpha — cytokines directly involved in synovial inflammation, osteoclast stimulation and progressive joint damage (Wasim et al., 2025). At the same time, reduced Treg number or function weakens peripheral immune tolerance and supports the persistence of autoreactive T and B cells (Lv et al., 2025). In addition, altered microbial composition may affect dendritic cell signaling, macrophage activation and B-cell responses, while also modifying the balance between T follicular helper and T follicular regulatory cells, contributing to abnormal autoantibody production (Cai et al., 2025).

Molecular mimicry represents another key mechanism through which the microbiota may drive RA. Certain bacterial antigens share structural or sequence similarity with self-proteins, including citrullinated epitopes relevant to RA (Reyes-Castillo et al., 2020). As a result, immune responses initially directed against bacterial components may cross-react with self-antigens, promoting the generation of autoantibodies such as anti-citrullinated protein antibodies (ACPAs) and rheumatoid factor (Abebaw et al., 2025). Microbial enzymes, including peptidylarginine deiminases, may also contribute to the post-translational modification of host proteins, increasing their antigenicity and further disrupting self-tolerance (Romero-Figueroa et al., 2023).

Another important mechanism involves the production of bioactive microbial metabolites. Short-chain fatty acids (SCFAs) such as acetate, propionate and butyrate are crucial mediators of communication between the gut microbiota and the host immune system. Produced by bacterial fermentation of dietary fiber, SCFAs support epithelial barrier function, modulate epithelial metabolism and promote anti-inflammatory immune pathways (Xu et al., 2022). SCFAs promote differentiation of regulatory T cells, limit Th17 cell expansion and regulate macrophage and B-cell function through signaling pathways involving G-protein-coupled receptors and epigenetic

regulation (Lv et al., 2025). In RA, reduced abundance of SCFA-producing bacteria and altered metabolite profiles have been consistently reported, indicating that reduced availability of these metabolites may contribute to chronic inflammation, disturbed Th17/Treg balance and progressive bone damage (Wasim et al., 2025). Other microbial metabolites, including bile acid derivatives and tryptophan catabolites, also participate in maintaining immune homeostasis and may exert anti-arthritic effects when dysbiosis is corrected (Xie et al., 2025).

The concept of the gut–joint axis describes the complex relationship between intestinal microbial disturbances and joint inflammation, combining the mechanisms described above into a single biological framework. Changes in gut microbiota composition can weaken intestinal barrier function, alter immune regulation, promote cross-reactive immune activity and affect the production of microbial metabolites, leading to systemic inflammatory effects (Zaiss et al., 2021). Microbial products, inflammatory mediators, activated immune cells, autoantibodies and metabolites derived from the intestinal environment may enter the circulation and reach synovial tissues, where they can sustain local inflammation and contribute to structural joint damage (Romero-Figueroa et al., 2023). Findings from both animal and human studies support the relevance of this interaction and suggest that modulation of the gut microbiota may have therapeutic value (Xu et al., 2022).

5. GUT MICROBIOTA AS DIAGNOSTIC AND PROGNOSTIC BIOMARKERS IN RHEUMATOID ARTHRITIS

Gut microbiota shows significant potential as a source of diagnostic and prognostic biomarkers in RA. Studies have consistently described dysbiosis in RA patients, with reduced microbial diversity and characteristic shifts in bacterial composition, including increased abundance of Prevotella copri and expansion of less common taxa such as Collinsella, Eggerthella and Faecalibacterium (Abebaw et al., 2025). Several of these microbial changes have been linked to disease presence and clinical severity. Machine learning approaches based on gut microbiota profiles have shown good diagnostic performance, with reported AUC values above 0.88 and identification of bacterial genera that differentiate RA patients from healthy controls (Wang et al., 2025).

Beyond taxonomic composition, microbiota-derived metabolites such as short-chain fatty acids, L-arginine, phosphorylcholine and arachidonic acid may also be relevant, as they influence immune regulation and appear to correlate with disease activity and treatment response (Lu et al., 2024).

Longitudinal studies suggest that higher baseline gut microbial diversity and specific taxa are associated with better clinical outcomes, including minimum clinically important improvement (MCII) in disease activity (Gupta et al., 2021). Although these findings highlight the potential of microbiota-based biomarkers in RA, further validation in larger and more diverse populations is required before they can be incorporated into clinical practice (Zhao et al., 2022).

6. THERAPEUTIC STRATEGIES TARGETING INTESTINAL MICROBIOTA

Therapeutic approaches targeting the intestinal microbiota are increasingly being explored as supportive strategies in rheumatoid arthritis. Since the gut microbiota plays an important role in maintaining immune balance, interventions aimed at correcting dysbiosis may help reduce inflammatory processes and improve disease control.

Among these approaches, dietary modification is considered one of the most practical ways to influence the gut microbiota. The most commonly recommended pattern is the Mediterranean diet, which is rich in vegetables, fruits, legumes, whole grains, nuts, olive oil and fish. This type of diet provides fiber and polyphenols that beneficial gut bacteria ferment into short-chain fatty acids, especially butyrate, which has anti-inflammatory effects and helps maintain gut barrier integrity (Kupczyk et al., 2025). Patients with high adherence to the Mediterranean diet were found to have lower disease activity, as measured by DAS28, and reduced levels of C-reactive protein (CRP) compared with those exhibiting low or moderate adherence (Diamanti et al., 2020).

A high-fiber diet is also important because dietary fiber is a substrate for beneficial intestinal bacteria. Foods such as oats, beans, lentils, vegetables and whole grains can increase the abundance of bacteria associated with immune regulation and lower inflammatory signaling (Häger et al., 2019). Fermented foods may also be useful because they naturally contain live microorganisms. Products such as yoghurt, kefir, sauerkraut and kimchi may support microbial diversity and complement probiotic interventions, although their effects vary between individuals. This aligns with observations from the TASTY trial, suggesting that microbiota-oriented dietary patterns may contribute to improved inflammatory profiles in RA (Charneca et al., 2025).

Omega-3-rich foods, particularly fatty fish such as salmon, sardines and mackerel, are often recommended because omega-3 fatty acids may reduce inflammatory cytokine production and may

also influence gut microbial composition (Tański et al., 2022). At the same time, reducing highly processed foods, excess sugar and saturated fats is considered beneficial, since these dietary patterns are associated with reduced microbial diversity and increased pro-inflammatory bacterial species (Bolte et al., 2021). Some studies also examine vegetarian or plant-forward diets because they tend to increase beneficial microbial metabolites (Bostan et al., 2024). Results from the NutriFast Study indicate that short-term dietary interventions, such as fasting followed by plant-based nutrition, may lead to temporary improvements in disease activity and inflammatory markers, although their long-term sustainability requires further investigation (Hartmann et al., 2022).

Probiotics have received attention because of their potential to restore microbial balance and modulate inflammation. Some probiotic strains may suppress the growth of harmful bacteria, increase the production of short-chain fatty acids and influence immune regulation through effects on inflammatory cytokines and regulatory T cells (Opoku et al., 2022). However, reported benefits are not always consistent, as treatment outcomes appear to depend on the bacterial strains used, treatment duration, dosage and individual patient characteristics (Li W. et al., 2024). Prebiotics may complement these effects by selectively stimulating the growth of beneficial gut bacteria, supporting microbial diversity and improving the production of metabolites involved in immune regulation, especially when combined with probiotics (Li J. et al., 2024).

Antibiotics have also been considered as a way to alter gut microbial composition in RA, but their use remains limited because of potential negative effects on microbial diversity and the risk of antibiotic resistance (Fishbein et al., 2023). Fecal microbiota transplantation (FMT) is another emerging approach that aims to restore a healthy intestinal microbial community through the transfer of donor microbiota. While FMT is already established in recurrent Clostridioides difficile infection, its use in RA is still experimental. Early studies suggest possible benefits for immune regulation, but important issues such as donor selection, safety, standardization and long-term outcomes still need further investigation (Sahle et al., 2024). New microbiota-based therapies are also being developed, including engineered probiotics, bacteriophage therapy and precision microbe inter

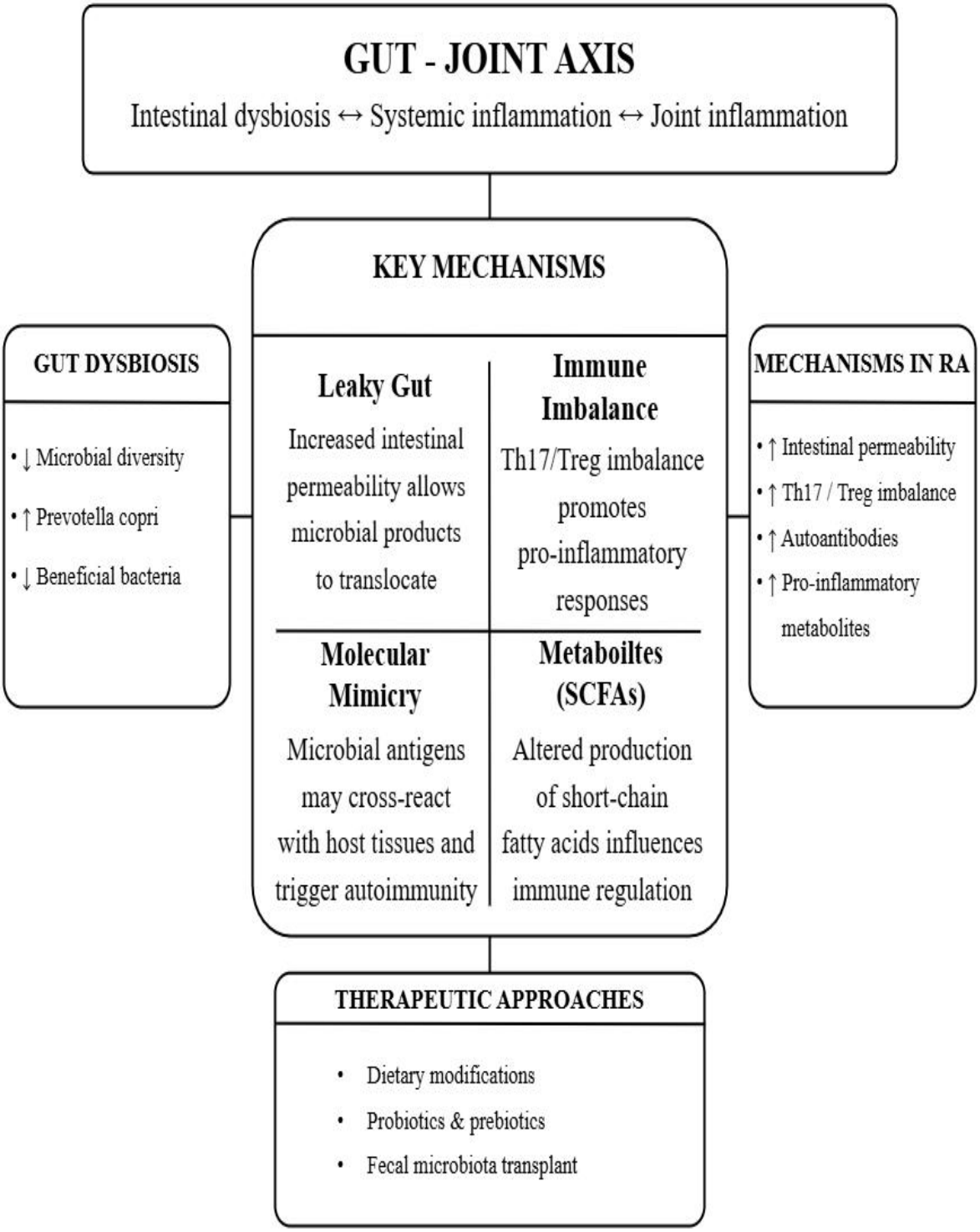

Figure 1. Conceptual overview of the gut–joint axis in rheumatoid arthritis.Created in draw.io.

7. CONCLUSIONS

The intestinal microbiota plays an important role in the pathogenesis of rheumatoid arthritis, contributing to immune dysregulation and chronic inflammation. Dysbiosis, characterized by reduced microbial diversity, expansion of Prevotella copri and depletion of beneficial commensals, may occur early and influence disease development. Key mechanisms include increased intestinal permeability, Th17/Treg imbalance, molecular mimicry and altered microbial metabolite production. The gut–joint axis provides a unifying framework linking intestinal disturbances with systemic and synovial inflammation. Gut microbiota also shows promise as a source of diagnostic and prognostic biomarkers, as well as predictors of treatment response. Therapeutic strategies targeting the microbiota — including dietary interventions, probiotics and fecal microbiota transplantation — represent emerging supportive approaches in RA management. However, further large-scale, well-designed clinical studies are needed to confirm their efficacy, safety and clinical applicability before routine implementation.

DISCLOSURE

Author Contributions

Conceptualization: WZ, IT · **Methodology:** KK, JP · **Data collection:** ZW, NP, AB2, AB1 · **Data analysis:** VR, US, ZJL · **Writing – original draft:** WZ, ZJL, AB1, AB2, US · **Writing – review & editing:** KK, ZW, IT, VR · **Visualization:** NP, JP · **Supervision:** WZ · **Funding acquisition:** Not applicable

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Declaration of AI Usage

During the preparation of this work, the authors used ChatGPT to improve language and readability. After using this tool, the authors reviewed and edited the content as necessary and take full responsibility for the content of the publication.

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